Battered Woman Syndrome
Is It Enough for a Not Guilty by Reason of Insanity Plea?

Renée Sorrentino, MD, Meghan Musselman, MD, and Lauren Broderick, MD

Today, 25 years after the Lorena Bobbitt trial, the case has gained a renewed interest. The #MeToo and Time’s Up movements have begun to identify the widespread problem of sexually abusive behaviors in both social and occupational arenas. In light of these crusades, filmmaker and producer Jordan Peele (Get Out, BlacKkKlansman) has created a four-part docuseries reexaming the Lorena Bobbitt case. A look back at the case is a reflection of how far we have—and haven’t—come in conceptualizing sexual violence, including gender bias in the determination of culpability as examined in Bobbitt’s not guilty by reason of insanity (NGRI) acquittal.

Research Sheds Light on Two Types of Treatment for ADHD

Marilyn Griffin, MD, and Elizabeth Harari, MD

New research highlights the potential benefits and detriments of treatment strategies for ADHD, which include both pharmacological and nonpharmacological options (Table).

Is psychosis a concern?
Stimulants are the first line of treatment for ADHD and are typically well tolerated. Although there are potential adverse effects associated with stimulant administration, the most common are usually transient or dose related. One of the more serious effects is stimulant-induced psychosis.

To explore this risk, Moran and colleagues examined the records of 337,919 young persons aged 13 to 25 who had an ADHD diagnosis and were newly prescribed a stimulant medication. The researchers found 343 episodes of new-onset psychosis among the 221,846 study-matched population over an 11-year period. The median time from which the first stimulant was dispensed to the psychotic episode was 128 days.

The researchers concluded that the risk of new-onset psychosis was...
Revisiting Bobbitt

Continued from cover

Twenty-five years ago, after she took a knife to her husband’s genitals, Lorena Bobbitt became a household name. At her trial, she and others testiﬁed that she had been trapped in an abusive marriage with a husband who raped, beat, and belittled her. The media, at that time, adopted comedic commentaries and headline puns in covering the story, depicting Lorena as a crazed woman who falsely claimed rape.

After the trial, Lorena’s popularity dissipated, and she faded into a quiet life in Manassas, Virginia. Her ex-husband, John Wayne Bobbitt, remained in the spotlight, starring in adult ﬁlms, giving interviews on talk shows, and getting paid to appear at events around the country. He was subsequently charged, on more than one occasion, with battery.

The not guilty by reason of insanity plea

On the evening of June 23, 1993, Lorena Bobbitt cut off her husband’s penis with a kitchen knife as he slept. She left the house, appendage in hand, and subsequently threw the penis out of her car window, near a 7-Eleven, and called 911. Lorena later told police that John was sexually, physically, and emotionally abusive. She reported that just before she cut her husband, he raped her. John’s version of events was that he had come home after an evening of drinking with a friend and went to sleep. He stated that Lorena attempted to make sexual advances toward him and that there was “some petting,” but they did not have sex. Lorena was charged with malicious wounding, and John was charged with marital sexual assault.

John was tried ﬁrst and was acquitted. At Lorena’s trial, she testified to the years of abuse that occurred in her marriage, including being forced by her husband to undergo an abortion. She discussed in detail the rape on the evening of the incident for which she was charged. She testiﬁed that following the rape, she went to the kitchen to get a glass of water and attempt to calm down. While there, she had memories of the traumatic events she had undergone at the hands of her husband. Lorena said she did not remember cutting her husband. Both the defense and prosecution offered psychiatric testimony. While both psychiatrists agreed that Lorena suffered from a mental illness, their opinions differed regarding Lorena’s criminal responsibility.

Dr Susan Fiester, the defense expert, made a diagnosis of MDD, PTSD, and panic disorder. She described Lorena as a classic example of a “battered wife,” weakened by years of abuse and fearful of leaving her husband. She testiﬁed that John’s assault on Lorena on the evening of June 23 led to “an acute deterioration of her mental state,” and she “became psychotic.” Dr Fiester further opined that Lorena did not have control over her actions when she assaulted her husband.

Dr Miller Ryans, the prosecution expert, testiﬁed that although he did not doubt Lorena was raped, he did not detect any evidence that she was “out of touch with reality or experiencing any delusions” at the time of the assault.

The battered woman syndrome has been used as a defensive strategy to justify a broad array of behavior, ranging from self-defense and provocation, to insanity and diminished capacity.

The idea of an “irresistible impulse” was raised in the 1843 trial of Daniel M’Naghten, who shot and killed the secretary of Sir Robert Peel, while believing that he was being persecuted by Sir Robert Peel and the Tory Party. In his successful defense of M’Naghten, defense attorney Alexander Cockburn described M’Naghten’s murderous act as an “irresistible impulse.” Following this trial, multiple states added an irresistible impulse rule to their insanity standards, and by 1944, 17 states included an irresistible impulse rule. Practically, the difﬁculty in applying this defense is in establishing the difference between an irresistible impulse and an impulse not resisted. This proved to be a key issue in the Bobbitt trial, and where Dr Fiester and Dr Ryans differed in their opinions.

Irresistible impulse or impulse not resisted

No state relies on irresistible impulse as the only test of insanity, largely because of the difﬁculty in distinguishing an “irresistible impulse” from an “impulse not resisted.” This means that those individuals, such as Lorena Bobbitt, who are found NGRI based on the volitional prong would have been found guilty in most other states. The irresistible impulse test was ﬁrst used successfully in the English case of Regina v Oxford in 1840. In this case, Edward Oxford was indicted for treason for attempting to shoot Queen Victoria. His defense was on the grounds of insanity, largely relying on a family history of insanity and his own prior “insane acts.” At the trial, Justice Denman charged, “If some controlling disease was . . . the acting power within him which he could not resist, then he will not be responsible.”

...the basic premise of such a defense [temporary insanity] would be that the defendant, at the time of the killing, suffered from severe stress and an impaired mental state as a result of the battering relationship, and that this impaired mental state caused her to kill the batterer. The causal link between the woman’s impaired mental state and the killing can be established by showing that the woman viewed her predicament from a psychologically distorted perspective and thus was unable to perceive her options accurately, or by showing that the “woman was driven to the breaking point by the circumstances of her situation and therefore was substantially unable” to conform [her] conduct to the requirements of law.

In contrast to the successful use of battered woman syndrome in the Hughes’ defense, the case Ibn-Tamas v United States in 1979 highlighted early conﬂict about the validity of the diagnosis. Following years of spousal abuse, Beverly Tamas shot her husband and was charged with second-degree murder while armed. After a guilty verdict, the case was taken to appeal, claiming the court had erred in excluding the expert testimony by Dr Lenore Walker on battered woman syndrome. Following the trial and associated media coverage, based on the pioneering work of Dr Lenore Walker, battered woman syndrome provided a rubric for understanding the complex, often countervuitive, responses of domestic violence victims.

The battered woman syndrome has been used as a defensive strategy to justify a broad array of behavior, ranging from self-defense and provocation, to insanity and diminished capacity. The 1977 case of Francine Hughes subsequently charged with first-degree murder. Following a trial by jury that included expert witness testimony, Hughes was found not guilty by reason of temporary insanity. In 1987 Cipparone wrote:...
Words have power. These include the power to heal, especially words spoken by physicians, mental healers in particular—psychiatrists. I’ll start here.

People approach you and say, “You’re a psychiatrist . . . what’s Trump’s diagnosis?” Their reason for asking seems beside the point to them as they offer none, their interest clearly unrelated to treatment or healing. Something else is on their mind, something they want to know. Or need to hear. Their subtext is hidden, possibly even from themselves, but we’re not unfamiliar with hidden subtexts. What is unfamiliar is being blind-sided and questioned in a public place, like during a lunch break or at a party among friends making small-talk.

So, how do we respond? How ought we respond? Need we even respond?

You’ve no time to think, plus it’s complicated and a medical diagnosis is out of the question. Mr Trump has neither a mental illness nor dysfunctional personality disorder, so the question boils down to the generic: What’s with him? What’s going on with him? Our elected president clearly marches to a different drummer and, like all of us (and that question), there must be multiple motives behind his behavior, motives not necessarily known even to him. Happily, once again, we’re not strangers to queries with multiple and possibly concealed motives. What rattles us is the cheeky expectation that we are able and willing to provide an impromptu public sound-byte about a touchy and seriously contentious topic.

Clearly, words have the power to derail. But words also have the power to heal. Remember the mantra, “Sticks and stones may break my bones but names will never hurt me.” We were taught as children to repeat this reprieve and laughter, fostered relief as if emitting healing energies. It’s the same for words we seek today to master something that confuses, devils, or scares us. We’re groping for words with similar “healing powers.” Mr Trump’s behavior, so unpredictable and inconsistent and with troublesome consequences, generates an anxiety we react to. Our seeking relief is automatic, instinctive.

We look for the right name, the “diagnosis,” because it just might be the antidote for our anxiety. A primitive survival instinct has just kicked in. (That smile and chuckle that sometimes accompanies the query is very much part of this.)

Why pick on a psychiatrist? Aren’t there enough words already out there, enough “names” in the public domain that accurately describe and account for Mr Trump’s behavior? Don’t we already have a pretty good handle on “what’s going on with him”? Yes, but such words aren’t emitting potent enough healing energies, apparently. Unlike the usual individual who fits this profile, the immature, uneducated, disrespectful, unreliable, and insecure sad sack, Mr Trump is no sad sack. He’s our president. Our customary reaction of labelling and dismissing simply doesn’t cut it here; something stronger is needed, a medicine with ju-ju. And our psychiatrist...
Optimizing Brain Health

John J. Miller, MD | Editor in Chief

When I began medical school in 1982, the brain was often referred to as a “black box,” a description meant to convey how little we knew about brain function and circuitry. We have learned much since then, and many of the scientific publications on brain-related research that flood my inbox each month have words in the title that I have never heard of before. I try to keep up, but I often feel as though I am trying to bail out the Titanic with a cup. Amidst this information explosion, there remain core principles that are well established and can help facilitate the health of our brain. On my website, Brain-Health.co, I have a link to a page where I list “Ten Ways to Grow a Healthy Brain.” I do my best to follow these myself, and I routinely discuss these with my patients. I invite feedback to lengthen this list and encourage all of us to work toward continued brain health.

Sleep
Arguably one of the most important requirements for good brain health is a good night’s sleep. Sleep is a primary mammalian function that has been evolutionarily conserved, and ideally each 24-hour day includes a defined period for sleep.

Learning new information in areas that were previously not known seems to facilitate synaptogenesis, which helps promote improved brain functioning and cognitive reserve.

There is tremendous individual variation in the number of hours a person requires to achieve the benefits of a good night’s sleep, ranging from 5 to 10 hours every night. During sleep, the brain’s energy utilization decreases, and the brain assumes a restorative posture, removing unwanted waste products and replacing the adenosine triphosphate (ATP) energy supply used up during the day. Sleep is a time for health promoting immune processes to occur, and for the endocrine system to recalibrate.

Significantly, the information and experiences of the day are consolidated in the various specialty areas of the brain’s cortical regions from the brain’s hippocampus, where they are stored temporarily during the day. Throughout the day, the hippocampus serves a function much like the random access memory (RAM) of a computer—without the storage of the day’s events in the cerebral cortex that occurs during sleep, the memory of these events will be impaired and less enduring.

To continue our computer analogy, our daily experiences and learning is temporarily saved in RAM, with the expectation that we will “save” this information to the hard drive before we shut off our computer. If we forget to save the information, it disappears from RAM after the computer is powered off. Hence the inefficiency of studying all night for an exam—we may pass the test, but we will not retain much of the material.

Sleep disorders are very common. Approximately one-third of the US population report symptoms of insomnia. Over the past 30 years, sleep studies have improved greatly, and it has become routine to diagnose and treat sleep disorders like sleep apnea—everyone knows anticipation. Although we have quite an armamentarium of sleep hypnotics, the most effective treatment we have to offer as clinicians is education about good sleep hygiene.

The most commonly utilized over-the-counter sleep aid in the US is diphenhydramine (Benadryl®). Most products sold at your local pharmacy that promote improved sleep contain diphenhydramine as one of the ingredients. Diphenhydramine has two pharmacological properties that can cause significant morbidity if taken daily for prolonged periods of time—antihistamine and anticholinergic. The antihistamine effect is what causes sedation; however, over time the histamine receptor de-sensitizes, so it becomes less effective. Moreover, chronic use of an antihistamine causes weight gain in many individuals. The chronic anticholinergic effect increases cognitive impairment, constipation, dry mouth, and urinary retention.

Aerobic exercise
As a general rule, lifestyle recommendations that make for a healthy heart also make for a healthy brain. Aerobic exercise has consistently shown to have healthy effects on the brain. Increasing blood flow promotes the delivery of oxygen and the removal of CO2 and waste products. Numerous studies demonstrate the health promoting effects of aerobic exercise, including:

- Decreased depression
- Increase in energy level

Of course, before starting or increasing aerobic exercise, get the green light from your health care provider.

Social connectedness
We are, by nature, social animals. There have been several studies of communities around the world where individuals commonly live to 100 years of age. National Geographic writer Dan Buettner1 has studied these communities extensively. The Barbaga region of Sardinia is home to the largest population per capita percentage of male centenarians. The Aegean Island of Ikaria, Greece, has been found to have the world’s lowest incidence of dementia, and low rates of middle-age mortality.

Buettner and his team of researchers extracted nine evidence-based common attributes found in these two regions, along with three other regions that share the properties of having the highest life expectancy and highest per capita centenarians. These attributes include:

- A sense of purpose
- Daily practices to reduce stress
- Belonging to a larger community
- Making the family needs priority number 1
- Being a member of a social circle that reinforces healthy behaviors

These communities where individuals often live long and healthy lives are called “Blue Zones.”

Daily practice to reduce stress
Chronic stress is a highly destructive physiological state. As our physical bodies evolved in a physically dangerous world, the fight-flight-freeze response served as a biological defense to increase our survival. Now that the primary causes of stress in the US are non-physical (work stress, financial stress, relationship stress, political stress), the evolutionary defenses of increased blood pressure, increased heart rate, increased respiration, re-distribution of blood to our muscles, pulses of epinephrine from
Healthy and diverse diet

A phrase that we often hear, and which contains a great deal of wisdom, is “you are what you eat.” The field of nutrition, and our growing understanding of the long-term effects of a health-promoting diet in contrast to an unhealthy diet (disease promoting) has expanded exponentially in the past 50 years. Common components of a healthy diet include whole grains, fresh fruits and vegetables, fresh fish, nuts, and unsaturated fats (including omega-3-fatty acids). Strawberries, blueberries, and blackberries contain anthocyanins (a type of polyphenol) that have antioxidant and anti-inflammatory properties that can be protective of the brain. Year after year the “Mediterranean diet” is promoted as a healthy and attainable diet for the average person. Of course, each person may have specific and unique dietary requirements for fluid intake.

Drink a lot of water

Maintaining good hydration, ideally with pure and unadulterated water, goes a long way towards both good physical health and brain health. Our culture has done a great job marketing soft drinks and other beverages, often containing sugar, artificial sweeteners, and caffeine, that are frequently more accessible than a glass of pure water. At airports around the country a bottle of pure water often costs more than an equal sized non-alcoholic beverage.

ments. In my experience, my body feels good after a healthy meal and feels tight and tired after a meal of “fast food” or other food products that I know are not so healthy for me.

The more your brain is challenged with new tasks, and the more complex these tasks are, the healthier your brain will be.
Congratulations and kudos to Dr Richard M. Berlin, whose poem “Eye Contact” was chosen to be included in the 2019 Hippocrates Prize Anthology. The poem was one of 22 selected by the Hippocrates Society for Poetry and Medicine, an international group that fosters the interface between poetry and medicine.

Inspired by a portrait of 16th century physician/anatomist Andreas Vesalius standing with a dissected cadaver and looking into the viewer’s eyes, Dr Berlin “felt like I was in the presence of Vesalius himself, making eye contact with a colleague from 500 years ago.” The piece was originally invited by Richard Ratzan, MD, as part of an anthology sharing reflections on Vesalius’ De Humani Corporis Fabrica, the first comprehensive textbook on anatomy that revolutionized medicine’s understanding of the human body.

The poem was Commended in the Hippocrates Poetry and Medicine Awards Health Professional category. The Society received about 1000 entries from all over the world to be considered for its awards. The 2019 Hippocrates Anthology that features his poem became available at the 10th annual Hippocrates Awards Ceremony in May.

“The poems which caught my eye—and my heart—were both arresting and illuminating,” shared Kate Addie, one of this year’s judges. “I was listening to those who have a deep insight into the human condition—not in a contemplative, detached way, but deeply involved and at times feeling a huge responsibility about the health and state of mind of others.”

Dr Berlin’s Poetry of the Times column has been a monthly feature in Psychiatric Times since 1997. Author of three poetry collections, Dr Berlin has previously received the Pearl Poetry Prize, the Pushcart Prize, the John Ciardi Poetry Prize, and best poetry book by USA Book News. He also fosters creative writing in the health sciences, having established a creative writing prize for medical students, nursing students, resident physicians, and doctoral students at the University of Massachusetts Medical School.

“Eye Contact” is included in the 2019 Hippocrates Prize Anthology, which can be ordered at http://hippocrates-poetry.org/order-2019-hippocrates.html.

**Historical note:** Prior to Andreas Vesalius (1514-1564), the understanding of human anatomy was derived from the work of Galen who died in 200 AD. Even though Galen’s findings were based on dissections of Barbary macaques, not humans, his authority was so strong that for 1400 years a succession of anatomists claimed confirmation of Galen’s inaccurate assertions until Vesalius demonstrated they were clearly false. (I refer to some of Vesalius’ findings in my poem, and to various images in Fabrica). Vesalius’s novel practice was to perform human dissections in the presence of others who could directly observe his findings and replicate them. His new knowledge was subsequently preserved and widely distributed in dramatic and detailed images in Fabrica. The name of the artist (or artists) who created the woodcut images is unknown.

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**Psychiatrist**

Continued from page 2

“diagnosis” might prove just the ticket. Our psychiatrist words might be just strong enough as a word-cudgel-proxy to subdue our nagging anxiety.

Unfortunately, while we psychiatrists feel flattered by this unexpected power-role, and rightly so, that flattery carries the risk of a serious, albeit unintended, consequence.

This observation first. That query about what’s going on with Trump is ours, too. We, too, have wanted to ask that question and likely have already. Over and over. Republicans and Democrats, both. We identify with the person asking, our only differences being the intensity of our anxiety and the importance and priority of addressing it. As for the serious consequence, our responding to that query and the connection we feel with the person asking inevitably invites a pleasant bond over our shared concern, a relaxed mood that comes with the connecting and rapport with a soul brother or sister followed by a gentle pull to get into, and then even off on, this bond. We start sharing personal sensibilities along with their emotional coloring. We are now a heartbeat away from ranting and venting, of sharing our favorite toxic quips and disrespectful names. We are about to indulge and abet the very thing we’ve long claimed to condemn, the divisive toxicity in our civil discourse, be it inter-party or intra-party.

I’ve been a chronic complainer about the toxic level of our civil discourse and only recently became aware that my habits of bonding and blaming to the point that ranting and venting were fueling that toxicity. I’ve since become mindful of that lure, that pull to rant, vent and, of course, laugh. But it’s not from hate or anger, distrust or fear, or even self-righteousness. It’s from anxiety. It’s always from anxiety. (But we already know that.)

**Dr Climo** is the author of Psychiatrist on the Road: Encounters in Healing and Healthcare, an account of his Locum Tenens experience.

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**Eye Contact**

Richard M. Berlin, MD

*From the portrait of Andreas Vesalius in De Humani Corporis Fabrica (The Structure of the Human Body), 1543.*

A ghostly glow frames the face of a man with nothing to hide, his vision honed on the graceful heft of charnel house bones poached from the Cemetery of the Innocents and the hangman’s noose. His fierce eyes lock on ours, confident as we stand beside him witnessing the dissected truth of a sternum’s three bones, a sacrum’s six, the singular presence of a ductus arteriosus. With nostrils lumbering death and a flayed cadaver at hand, his eyes compel us to plumb The Fabrica’s woodcut skeletons dressed in flesh flaunting deltoids gaudy as epaulet, each sartorius ribboning a lusty thigh of pained, praying souls that mirror his heart and mind, men muscled hard as gods whose beauty turns a blind eye toward death.

*Come closer, his gaze* commands. *Abandon fear and Galen’s dogma.*

*Confirm my work to find in a body’s design the naked truth your own keen eyes can see.*

Dr Berlin is Instructor in Psychiatry, University of Massachusetts Medical School, Worcester, MA.
Work Stress Takes a Toll on Physicians’ Health

Julie Bowen

A survey of British physicians found that 44% binge drink and 5% meet the criteria for alcohol dependence.

A survey of British physicians found that 44% binge drink and 5% meet the criteria for alcohol dependence. The findings were recently published in BMJ Open.

The survey was conducted to examine the effects of occupational distress (including burnout, depression, and maladaptive coping strategies) on substance abuse and other health problems. Researchers analyzed data from 417 physicians in the United Kingdom. The mean age of the participants was 47 years, and 52% were women.

“Our research shows that 55% of doctors have burnout, and this has real health consequences,” said study author Caroline Kamau, PhD, at Birkbeck, University of London, UK.

About 34% of physicians in the survey reported that they drank alcohol to cope with work-related stress, and 22% used substances to deal with stressful events. Less experienced physicians and those who worked in hospitals were more likely to engage in binge drinking. Binge eating was another coping strategy reported by physicians: up to 29% experienced negative emotions after overeating and 8% had a binge eating disorder.

Reference

New Treatment Reduces Levels of Protein Linked to Huntington Disease

Julie Bowen

The study
The phase 1/2a trial included 46 adults with early Huntington disease. Thirty-four patients received HTT, in doses ranging from 10 to 120 mg, while 12 participants were given placebo. Each participant received a total of four bolus intrathecal injections, which were administered at 4-week intervals.

The researchers found dose-dependent reductions in the concentration of mutant HTT protein in the cerebrospinal fluid (CSF) of patients who received HTT. Specifically, there were reductions of 20%, 25%, 28%, 42%, and 38% in those given doses of 10 mg, 30 mg, 60 mg, 90 mg, and 120 mg, respectively. In contrast, patients who received placebo had an increase of 10% in CSF levels of mutant HTT.

No serious adverse events were reported in the patients who received HTT. Most of the events (83%) were mild, and the remainder were moderate in severity. The most common adverse events in those who received HTT were procedural pain and post–dural puncture headache.

After the study ended, participants were given the opportunity to enroll in a 15-month, open-label extension trial to assess the effects of intrathecal administration of 120 mg of HTT monthly or every other month.

Clinical benefits still unclear
Questions remain, however, about whether the reduction in CSF levels of mutant HTT will translate into clinical benefits. The researchers themselves noted the limitations of their trial: “Functional, cognitive, psychiatric, and neurologic clinical outcomes were generally unchanged at the dose-group level during the trial, and no meaningful differences were observed between patients who received placebo and patients who received HTT, regardless of the dose level.”

“Larger studies of greater duration will be needed to determine whether HTT-mediated reduction of the concentration of mutant HTT in CSF is associated with a treatment effect on the disease course, which is typically slow, with changes on standard outcomes generally occurring over a period of years,” the authors concluded.

This study was funded by Ionis Pharmaceuticals and F. Hoffmann–La Roche.

References
Things Are Seldom What They Seem

Sharon Packer, MD

A s Gilbert and Sullivan wrote, “Things are seldom what they seem,” and skim milk masquerades as cream. Medical disorders may also masquerade, not as cream but as psychiatric disorders, as occurred in Maureen’s case.

Maureen had every reason to be depressed. Her live-in boyfriend left after 5 fitful years. Since then, her energy waned. She rarely slept through the night and couldn’t make it to morning team meetings.

Not surprisingly, her job was in jeopardy. Worse yet, her company was also in jeopardy, for the fashion industry had been floundering ever since “fast fashion” priced out their designer dresses. Her once-prestigious brand would be “cutting the fat” to stay afloat. They hinted at lay-offs to come.

Even when she was in time for the morning meetings, Maureen couldn’t concentrate. She made careless and costly errors. She received a formal warning from her boss, who said that her “presenteeism” was worse than her absenteeism. He referred her to HR, expecting them to do the dirty work of dismissing her.

Maureen left a voice message for me, saying that she had explained her situation to Eileen, the head of HR, who suggested a medical leave of absence, rather than the immediate termination recommended by her supervisor. She received my name from the company’s Employee Assistance Program (EAP). To me, this sounded like a generous offer, especially since it came with short-term disability benefits; however, there was one caveat. Maureen needed to seek psychiatric treatment while on leave. Maureen was told in no uncertain terms that she needed a psychiatrist to “sign off” on the disability and to prescribe medications as needed. HR would not accept a note from the texting therapist whom Maureen had found through a subway ad.

I knew from the get-go that there would be plenty of papers to sign, and annoying insurance forms to complete, along with regular clinical care, should I schedule her appointment. The EAP staff knew that I would not endorse questionable disability papers, and I made sure that Maureen knew that I could promise her an evaluation but could not promise anything more before completing that evaluation.

Maureen arrived at her appointment, looking pale and haggard for someone so young. She had neglected her grooming, which didn’t go unnoticed in the fashion industry. Her nails were chipped. Three inches of dark roots told me that her self-care had lapsed 6 months earlier, since hair grows half an inch a month.

It would have been easy enough to diagnose MDD, recurrent, fill out her forms, start an SSRI, set up follow-up appointments, touch bases with her therapist, and leave it at that. But there was no way that she would leave my office without reviewing her medical history as much as her psychiatric and social history. A “review of systems” is a standard part of any initial evaluation. Obliged to avoid certain medications in potentially pregnant patients. She scribbled a series of question marks after that question. I asked her to elaborate.

Upon questioning, Maureen mentioned that her periods often lasted so long that she wasn’t sure when they began or ended. She recalled that her college gynecologist attributed her anemia to heavy periods but did not recommend treatment or further work-up. Maureen added that her previously mild menstrual pain had become major pain. When asked about her last primary care or gynecology visit or recent laboratory tests, she looked up sheepishly and said that she hadn’t seen a doctor for 2 or 3 years.

I needed more data about Maureen’s medical status, since any number of medical conditions can affect mood and cognition, even when someone has obvious social stressors that can precipitate depressive symptoms. So, I bargained with Maureen and told her that I would sign her disability papers a week at a time, with the proviso that she consult a primary care physician and a gynecologist in the interim. Again, the ever-efficient EAP head had those doctors on direct-dial. To expedite matters, I handed Maureen a slip, knowing that basic lab results would return electronically the following day, but thyroid functions would follow in a few days. Those tests would be ready by the time she scheduled her PCP appointment.

Maureen was started on sertraline, but I emphasized that her unexplained medical problems could also cause lassitude. Her gynecology appointment was arranged quickly, and the gynecologist took the time to call and say that she felt large masses during the office examination. She suspected fibroids but needed imaging studies and a biopsy to support her suspicions and to rule out something more serious. In the meantime, Maureen’s lab tests returned with strikingly low hemoglobin levels. The repeat lab test ordered by the gynecologist later that week showed even lower levels. The gynecologist recommended iron supplements in the interim and explained the treatment options to Maureen.

The biopsy report came in. Luckily, there were no cancerous cells.
Goldilocks and the Opioids

Steven A. King, MD

Everyone knows the story of Goldilocks and her visit to the home of the three bears. Where she was seeking the right balance between the things she discovers to attain the maximum comfort.

Medical professionals face something similar when it comes to prescribing opioids. We don’t want to prescribe too many pain killers, which may contribute to the epidemic of opioid misuse, yet we don’t want to have patients needlessly suffering who might benefit from opioids for pain management. Two recent publications highlight both the concerns regarding this issue and also the limitations in our knowledge regarding how best to achieve the ideal.

The first is a letter signed by over 300 medical professionals to the Centers for Disease Control and Prevention (CDC) regarding its guideline on prescribing opioids for chronic pain issued in 2016. The guideline strongly recommends limiting the use of opioids for the management of chronic pain, noting the lack of research to support their use for this and highlighting the potential benefits of using non-pharmacologic treatments and non-opioid medications.

Although the letter doesn’t dispute the CDC guideline recommendations, it expresses concerns that within a year of its publication, “there was evidence of widespread misapplication of some of the Guideline recommendations” including concerns about exceeding its recommended opioid daily dosage limitations.

According to the letter, the guideline has resulted in barriers to patients obtaining needed opioids including amount and dosage limitations set by insurance companies, limitations in dispensing the medications by pharmacies, and doctors themselves limiting or even refusing to prescribe opioids out of fear of legal action if they consider prescribing them in a manner that is different from what the guideline recommends. The letter also states:

Patients with chronic pain, who are stable and arguably benefiting from long-term opioids, face draconian and often involuntary rapid dose reductions. Furthermore, the guideline has created barriers that have resulted in not only unnecessary suffering because of patients being unable to obtain the opioids they require to control their pain but also other problems. In desperation, patients in pain are turning to suicide or illicit drug use or being forced to turn to invasive treatments such as spinal injections that often provide little benefit.

In the letter from the health professionals, they note, appropriately, that there is little objective evidence for their statements and that the letter is mostly based on anecdotes. In fact, the one point that the letter makes is that anyone who does pain management can attest to without any doubt is that the non-medication treatments recommended by the CDC, which the letter doesn’t name but includes treatments such as cognitive-behavioral therapy and acupuncture, may not be covered by insurance thus limiting access to them.

The letter expresses concerns about the CDC guideline recommendations. It is worth noting that even when the number of opioid prescriptions was increasing during the last decade of the 20th century and the first decade of the 21st, there was no apparent reduction in the number of unnecessary treatments such as spinal injections despite limited evidence of their efficacy. This suggests that whether opioids are liberally or conservatively prescribed has little to do with the fre-
frequency of the provision of such treatments.

The letter calls on the CDC to evaluate the impact its recommendations have on patients and caregivers and that it clarify its recommendations on opioid taper and discontinuation. I agree with the need for ongoing research about the effects of the CDC guideline, but I am troubled by what the letter ignores.

Although the letter indicates that physicians may have misread the guideline as being a requirement, I question how frequent this is. Considering the number of guidelines that have been issued for many different disorders and problems, I hope that health care professionals are aware that guidelines are not laws. As far as I’m aware, none, including the CDC, require or expect that every patient be treated according to a specific guideline.

Based on my experience, I believe that most physicians who refuse to continue treatment with opioids or to initiate them based on the CDC guideline may be using it as an excuse, not a reason. It is far easier for physicians to say they are prevented from prescribing an opioid or are required to limit dosage or discontinue it, than to tell patients they (the prescribers) are making the decision. It may take considerable time and effort to explain to patients the reasons for the decision.

There are two other points that the letter overlooks. The first is the reasons why physicians may seek to reduce and possibly discontinue opioids. It is widely thought that the only reason for this is fear of addiction. It is true that this is a problem, and evidence has shown that a significant number of patients taking opioids for chronic pain end up misusing these medications. However, of equal importance is the issue of hyperalgesia where extended use of opioids can actually lower the pain threshold and thereby exacerbate the pain. Although the correct treatment for this is to reduce and discontinue the opioid, the worsening of the pain is often misinterpreted as being due to tolerance or worsening of the condition, which results in an increase rather than a decrease in the dose.

The second overlooked issue is how we should be evaluating the efficacy of opioids or any other treatment for chronic pain. From the letter it would be easy to conclude that measurement of the pain itself is of most importance. However, it is generally agreed that a far better measure of efficacy and one that can be more objectively observed than pain level is the level of functioning. I have treated many patients with chronic pain over the course of my career with opioids and all have had one thing in common: there is evidence that they are functioning better because of the medications. When these patients are in a fairly stable condition, I try to reduce dosage and, if this is possible, to consider discontinuation. I have never just started a patient with chronic pain on an opioid and then not made an attempt to see if it is still required over time.

The letter calls on the CDC to clarify its recommendations on the tapering and discontinuation of opioids, and I believe this is appropriate. This is another area of pain management with limited research, which the CDC notes. We have very little guidance as to the best ways to taper doses of opioids either with the goal of reducing dosage or discontinuing the medications. A recently published statement calls attention to concerns over rapid and forced tapering for outpatients. This is something that seems to be more for the benefit of health care providers who no longer wish to prescribe opioids than of patients.

Medication management of opioid use disorder: what we know and what we don’t

The National Academies of Sciences (NAS) recently issued a report on the use of medications for opioid use disorder that highlights the efficacy of the three currently available medications for the management of opioid use disorder: methadone, buprenorphine, and extended-release naltrexone. The report emphasizes the problem of the underutilization of these medications.

I am in complete agreement but have one major problem with the report. There is virtually no attention given to the issue of iatrogenic opioid use disorder where patients are appropriately treated with opioids for a legitimate pain problem and end up misusing them. The report briefly mentions the problem of opioid use disorder among patients with chronic pain. However, it primarily addresses the treatment of opioid use disorder in people with comorbid chronic pain, and it notes the limited research on the efficacy of medication treatment for opioid use disorder. However, there is no specific mention of iatrogenic opioid use disorder and what recommendations there are for its management or the need for future research.

Although the evidence for the existence of iatrogenic opioid use disorder goes back almost 30 years, we still know very little about it. We don’t know if either the physiology or psychology of it is the same or differs from opioid use disorder resulting from the non-medical use of opioids much less whether the treatment for both should be the same.

If methadone and buprenorphine are the appropriate treatment for iatrogenic opioid use disorder, they should also be the first-line treatment for pain because they are just as effective for pain management as other opioids. We would then have something unique in medicine: the same medications that cause the problem are also the appropriate treatment for it.

Why is iatrogenic opioid use disorder so ignored by the NAS report? I can’t be certain, but I believe that an important factor is those involved in writing the report. These are substance abuse experts who probably have limited experience with iatrogenic opioid use disorder and may not even be aware of it. The fact that substance abuse experts involved in the development of DSM-IV and DSM-5 failed to include a separate iatrogenic opioid use disorder diagnosis suggests that they may have limited knowledge about it or think it’s so incredibly rare that it doesn’t warrant a separate diagnosis.

I should note that the letter critical of the CDC guideline also suffers a similar shortcoming. The primary authors of the letter appear to be experts in substance abuse but not in pain management. Many of the over 300 who endorsed the letter are pain specialists and many are addiction specialists, but there are very few who can be considered experts in both and might be expected to be better able to weigh the risks and benefits of opioids.

We need doctors who have training in pain management and addiction medicine. Patients who appear to be receiving only limited benefit from opioids still often refuse to discontinue them out of fear that they will be left to suffer even more than they already are. Patients need to be taught other methods for managing pain apart from opioids for there to be any reasonable expectations that they will agree to reduce the dose or discontinue use.

There are two ways we can address the lack of doctors with expertise in pain management and opioid misuse. Because pain management falls under anesthesiology in most medical schools and hospitals, we could require that anesthesiologists receive training in recognizing and managing substance abuse. Conversely, substance abuse primarily falls under psychiatry, but most psychiatrists receive limited training in pain management.

Although it is doubtful that anesthesiology programs will ever provide much training on substance abuse, I still hope that psychiatry will recognize the important role it can play in pain management. Someone with training in both pain management and substance abuse would be better able to weigh both the benefits and risks associated with the use of opioids in patients with chronic pain and tailor treatment to the needs of individual patients.

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References
Although a biological basis for psychiatric disorders has now been firmly entrenched into psychiatric training, research, and clinical care, a strong understanding of the neuropsychiatric syndromes—both psychiatric manifestations of neurological diseases, and functional neurological disorders—has eluded standard psychiatric practice. The ACGME (Accreditation Council for Graduate Medical Education) sought to rectify this with the psychiatry milestones project, which identifies neuropsychiatry and neuroscience as among the score of knowledge and skillset for psychiatrists.\(^1\)

Unfortunately, our burgeoning understanding of neuropsychiatric disorders has escaped a generation. Moreover, even though neuropsychiatry is now recognized as a core of psychiatric residency training, most programs lack the faculty and resources for basic neuropsychiatric education. This Special Report goes some way to rectify these deficiencies by providing state of the art overviews of some of the common neuropsychiatric syndromes. Unfortunately, many important neuropsychiatric topics such as autoimmune limbic encephalitis, behavioral variant frontotemporal dementia, the neuropsychiatry of epilepsy, rapidly progressive dementias, movement disorders and so on, could not be included. The neuropsychiatry of TBI was previously featured as its own special report.\(^2\)

The recent discoveries of a variety of antibodies such as the NMDA-R antibody that can cause neuropsychiatric symptoms, and the various neuropsychiatric symptoms including delusions, hallucinations, and mania associated with the C9orf72 hexanucleotide repeat expansion, have focused our attention on the need to identify neurological causes of “psychiatric presentations.”\(^3,4\) As neurobiological research into psychiatric disorders has become the predominant model of inquiry, there have been calls for psychiatry to be reformulated as a “clinical neuroscience discipline” while some of my colleagues argue that all psychiatry is neuropsychiatry.\(^5,6\) I disagree. To be sure, clinical neuroscience is a fundamental discipline for psychiatry. However, the social, cultural, political, and economic context is just as important to psychiatry. Furthermore, not all of the problems that psychiatrists treat are best explained in a neurobiological paradigm. Indeed, we are now learning that even for neuropsychiatric disorders, a narrow focus on the brain alone does not yield the secrets to our conundrums.
pathology can exist in the context of normal cognition. Furthermore, it is clear that brain pathologies cannot explain the whole picture, and even hitherto undiscovered pathologies are unlikely to explain much of the variance in cognitive decline. Instead, it appears that social and psychological factors, nutrition, and other environmental contributors may provide important risk and resilience factors that confer vulnerability and protection to pathology.8

Neuroscience and neuropsychiatry provide a fundamental foundation to psychiatry. The reviews of neuropsychiatric syndromes featured in this Special Report including chronic traumatic encephalopathy, HIV-associated neurocognitive disorder, catatonia, tinnitus, autism spectrum disorder, psychogenic non-epileptic attacks, and tardive dyskinesia attacks an excellent update on these disorders for the general psychiatrist. It may be tempting to view these syndromes as further evidence that psychiatry is merely a clinical neuroscience. While psychiatrists must be able to detect and manage these conditions that exist in the hinterland between neurology and psychiatry, it is precisely because our training privileges the social, cultural, and psychological dimensions of care—in addition to the neurobiological—that psychiatrists are best placed to manage such syndromes. As Leon Eisenberg noted with concern: “The very elegance of research in neuroscience has led psychiatry to focus so exclusively on the brain as an organ that the experience of the patient as a person has receded below the horizon of our vision.”9p5

As we discover new diseases of the brain that present with psychiatric symptoms and clarify the neural underpinnings of well-described syndromes, it remains incumbent upon us to remember the experience of patients and their families in the context of society at large.

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Neuropsychiatry of Catatonia Clinical Implications

Catatonia is a neuropsychiatric syndrome in which the cluster of psychomotor signs and symptoms results in aberrations of movement and behavior. DSM-IV included new criteria for mood disorders with catatonic features, and for catatonic disorder secondary to a general medical condition. In DSM-5, catatonia is recognized as due to a medical or psychiatric condition, or unspecified, as, for example, in recurrent idiopathic catatonia. Mood disorders such as MDD and bipolar disorder are now recognized as more commonly associated with catatonia.

The diagnosis of catatonia relies on the recognition of its sometimes unusual symptoms. Three subtypes of catatonia are conceptualized in Figure 1: stuporous, excited, and malignant. Catatonic withdrawal or stuporous catatonia, includes motoric immobility, staring, mutism, rigidity, withdrawal and refusal to eat, along with more bizarre features such as posturing, grimacing, negativism, waxy flexibility, echopraxia, stereotypy, verbigeration, and automatic obedience. Excited catatonia is characterized by purposeless and excessive motor activity that includes disorganized pressured speech, flight of ideas, verbigeration, disorientation and/or confusion, and confabulation.

Catatonic signs of malignant subtype are accompanied by fever and dysautonomia. Malignant catatonia is associated with increased morbidity and mortality. A specific example of malignant catatonia is neuroleptic malignant syndrome, induced by dopamine-2 blocking agents or withdrawal of a dopamine or gamma-aminobutyric acid (GABA) agonist. Another variant, known as manic delirium or delirious mania, exists with features of both excited and malignant catatonia. Periodic catatonia can present with alternating stuporous and excited forms.

The prevalence of catatonia among psychiatric patients ranges from 7.6% to 38%.1 (See Table 1 for risk factors for catatonia.)

Pathophysiology
Catatonia has attracted neuropsychi
tists interested in brain mediation of motivation and movement, leading to attention on neurotransmitters and circuits. Several models for the neuropsychopharmacology of catatonia focus on neurotransmitters. In 1985, it was hypothesized that pharmacotherapies for catatonia act on dopamine-GABA connections in the mesostri
tum and mesocorticolimbic systems and in the hypothalamus.2 A restitutive dopamine system seeks re
dance through GABA-α-mediated up
and down-regulation of dopamine.

When the restitutive system is dysfunctional, a vulnerability to catatonia emerges from dopamine antagonists. Carroll1 has described a “universal field theory” encompassing diverse neurotransmitter changes for catatonia. Their model postulates a reduced GABA activity in the frontal cortex, increased N-methyl-d-aspartate glutamatergic activity in the posterior parietal cortex, and a dampening of dopaminergic action in the basal ganglia.

Approaching catatonia from a circuits perspective, C. Miller Fisher4 proposed that catatonic depression emerged from connection disturbanc
es in the same mesencephalofrontal system (brainstem to basal ganglia to limbic system and cerebral cortex) that also leads to akinetic mutism. Nortoff5 used neuroimaging in pa
tients recovering from catatonia to uncover trait features through provoc
tive testing of response to fearful fac
es and situations. He described a re
gion of interest in the orbitofrontal cortex that overreacts in these provoc
tive situations, which suggests dys
functional responsiveness.

Other functional imaging studies have shown altered activity in orbito
frontal, prefrontal, parietal, and motor cortical regions.6 Recently, T1-weighted MRI imaging in catato
nia secondary to schizophrenia spec
trum disorders showed reduced cor
tical thickness in frontoparietal regions as well as hypergyrification in the anterior cingulate cortex and medial orbitofrontal cortex compared with controls.7 The basal-gan
glia-thalamo-cortical loop system has been implicated in the patho
physiology of catatonia, with inter
ruption at various nodes leading to specific symptoms of catatonia.8 For

SIGNIFICANCE FOR PRACTICING PSYCHIATRISTS
Catatonia is a syndrome with multiple medical and neuropsychiatric etiologies, which is associated with significant morbidity and mortality especially when malignant. Becoming proficient at diagnosis and management is important for all psychiatrists, since catatonia occurs in both medical and psychiatric settings and in all age groups.

- Catatonia is a distinctive syndrome with a variety of motivational and movement deficits.
- Catatonia is associated with significant morbidity and mortality.
- Catatonia is highly responsive to treatment with lorazepam and ECT.
Differential diagnosis
Catatonia as a syndrome may arise from multiple etiologies and can lead to medical complications that result in significant morbidity and mortality, making rapid diagnosis and treatment a priority. Medical complications abound, and the mortality rate for malignant catatonia despite better recognition and treatment is still 9% to 10%.

For a list of potential medical complications, see Table 2.

Catatonia has several mimics, which must be ruled out before making a diagnosis. Locked-in syndrome, linked to pontine lesions, can be distinguished from catatonia because patients will generally attempt to communicate with their eyes. Patients in a persistent vegetative state may also appear to be catatonic. Stiff person syndrome is an autoimmune disorder that presents during severe stress with intense lower extremity spasmatic stiffness that may appear like catatonic posturing, but these patients speak and complain about their pain.

Some patients suspected of being in a catatonic state may have an extrapyramidal parkinsonism. These can have a distinctive tremor but are not negativistic and lack bizarre catatonic psychomotor symptoms. Nonconvulsive status epilepticus can also produce a catatonic-like state; electroencephalography is essential for accurate diagnosis and prompt management may minimize cognitive damage.

Several additional syndromes show clinical overlap with catatonia. Like Fisher, we believe akinetic mutism, an extreme form of the abulia syndrome caused by neurologic injury, to be a neurologic version of catatonia. Hypoactive delirium may overlap or coexist with catatonia. It is extremely important to confirm a delirious patient is not also suffering from a catatonic episode, because neuroleptics can worsen simple catatonia, resulting in malignant catatonia. Decreased eyelash and resistance to eye and mouth opening may hint at a catatonia nestsed in delirium.

To assess catatonia, a few steps can be routinely followed (Table 3). Once the diagnosis of catatonia is made, a definable etiology must be sought. In addition to mood disorders and schizophrenia-spectrum disorders, catatonia can be seen in up to 20% of patients with autism-spectrum disorders. Case reports have described catatonia secondary to obsessive compulsive disorder, PTSD, and personality disorders.

Up to 50% of cases of catatonia may be due to a host of neuromedical syndromes. These include paraneoplastic and limbic encephalitidites (especially anti-NMDA receptor antibody encephalitis), ictal and post-ictal states, posterior reversible encephalopathy syndrome, and lupus. Substances associated with catatonia include dopamine-blocking agents, tacrolimus, disulfiram, and phencyclidine, among others. Finally, catatonia can occur as an isolated clinical syndrome without an obvious underlying cause; this phenomenon is known as recurrent idiopathic catatonia.

Management and treatment
With severe threat, challenges to brain function can emerge via circuit disconnection or modulatory dysfunction and result in catatonia. This view may underlie the effectiveness of benzodiazepines, in particular lorazepam, of NMDA-R antagonism, and of electroconvulsive therapy (ECT). These treatments show effects on GABA, dopamine, acetylcholine, and glutamate within cortico-striato-thalamo-cortical loops. (For an outline of a proposed catatonia treatment algorithm, please see Figure 2.)

All treatments for catatonia are off-label uses, as there is no FDA-approved drug indicated for catatonia. In 1983, lorazepam was described as a successful treatment for catatonia and later became the first-line treatment for the catatonic syndrome.

A lorazepam challenge (using 2-mg intravenous [IV] lorazepam) can be a helpful diagnostic test. Although a negative response does not rule out catatonia, many patients will show improvement with a single dose. Following the challenge, a standing dose of 2 mg every 4 to 6 hours is typical; some patients may require titration to as much as 30 mg daily, especially in cases with malignant features.

Doses should be held only for respiratory depression due to oversedation and not for sedation alone, as the regularity of dosing is important for full lysis. IV lorazepam is preferred over other routes or benzodiazepine types because of its quick onset, preference for the GABA-A receptor, and long duration of effect.

Approximately two-thirds of patients will respond to treatment with benzodiazepines, although those with catatonic schizophrenia show reduced responsiveness. Catatonia sometimes re-emerges during transition from IV lorazepam to oral dosing. This may require reinstitution of...
When benzodiazepines are ineffective or contraindicated and ECT is unavailable or refused, second-line treatments include glutamatergic agents, such as memantine (5-10 mg twice daily) or amantadine (400-600 mg daily in divided doses). These agents appear to be safe, well-tolerated, and effective in case reports, as monotherapy or in combination with a benzodiazepine. Other options include valproate or carbamazepine, which may be particularly useful if the catatonia is related to underlying mania. Valproic acid has demonstrated some effectiveness in excited catatonia.

A final option to consider is an atypical antipsychotic. These agents are the last step in the algorithm because of their potential to worsen catatonia or cause conversion to a malignant catatonia. Antipsychotics should be given in combination with a benzodiazepine, and low-potency atypical agents are preferred. Among antipsychotic agents, aripiprazole may be the safest choice given its partial-agonist action. If the catatonia occurs in the setting of clozapine cessation, re-initiation of clozapine should be a first-line strategy. High-potency typical antipsychotics should generally be avoided in catatonic patients.

Supportive management is essential in cases of catatonia. In addition to treatment of the catatonic symptoms, full resolution often requires treatment of the underlying disorder. This can be challenging in cases of psychosis, which may require a careful balance of antipsychotics and benzodiazepines.

References
Diagnostic Errors in Neuropsychiatry

**SIGNIFICANCE FOR PRACTICING PSYCHIATRISTS**

Making a diagnosis is a fundamental skill for all physicians. How do we do it? Driven by an effort to reduce the serious problem of diagnostic errors in medicine, a better understanding of the diagnostic process has emerged in recent years. In this article, psychiatrists will learn about how the diagnostic process works and where vulnerabilities lie, especially in the area of neuropsychiatry. Recommendations for how to reduce diagnostic error rates are highlighted.

**CASE VIGNETTE: Psychosomatic Concerns**

According to his primary care physician (PCP) who referred him, Mr O has a long history of psychosomatic complaints. Mr O is a 63-year-old accountant who recently retired. He's been sleeping restlessly and waking up depressed each morning. He jogs a few miles a day, most days, but says that lately he's been feeling stiff and somewhat unsteady on his feet. Also, he says he's "plagued by" constipation. The patient told the PCP that he can't tell whether these physical problems are "all in my head" or whether there's really something to worry about.

When you get a referral, you are presented with a great deal of information, and it isn’t clear which information will be relevant to your eventual understanding of the patient’s health problems. Some of the material that is contained in the referral will be explicit, some implied, details may be inaccurate, and surely, much will be left out.

Now consider that once you greet a patient in the waiting room, you will have even more information. You will notice how the patient is dressed and what he’s been doing while waiting; you will observe his facial expression, level of social comfort, his gestures and gait. And, before you even hear the patient’s voice or ask a single question, all of these experiences will affect you and have an influence on how you are inclined to think about the diagnosis. Diagnostic problem solving is extremely engaging for physicians, but it is not the defined type of problem solving one encounters with something like a crossword or Sudoku puzzle in which there are 26 letters or 9 numbers that are manipulated to form one unique solution. Making a diagnosis is complex problem solving in an uncertain domain. Any particular information or observation might be relevant; and any particular information or observation might be irrelevant (noise). The patient might leave out crucial parts of the illness story, perhaps not realizing that they are important. Or the physician–problem-solver may have insufficient knowledge, perhaps never having heard of the diagnosis that actually explains the patient’s symptoms. There also could be more than one diagnosis; and these diagnoses might be interacting with one another.

**System 1 and/or system 2 cognition**

The reigning theory about diagnostic problem solving is that it involves two interacting cognitive systems—system 1 and system 2.1

System 1 cognition is illustrated by the experience of seeing a patient for the first time and “knowing” immediately that the patient has, for example, schizophrenia. System 1 is based on mental pattern-recognition; it is intuitive, but honed and refined by experience; it is fast, effortless, and operates outside of awareness. While this system is extraordinarily efficient, processing large amounts of information very rapidly, it is also highly error-prone and open to biases (predispositions to think and act in a certain way) of which the clinician is generally not aware.

System 2 cognition is exemplified by the generation of a differential diagnosis list and examining the evidence for and against each diagnosis. System 2 utilizes explicit rules of logic; it is slow, effortful, entirely conscious, and analytical. Clinicians naturally favor the use of system 1 cognition because it is effortless. But the most effective clinical reasoning involves going back and forth iteratively between system 1 and system 2 cognition.

**Thinking about Mr O**

Now let’s return to the case vignette. The patient’s story is in your mind when you go into the waiting room. Depending upon your fund of knowledge and your experience, you might have what turns out to be an accurate diagnostic impression from the referral information. But perhaps you don’t. To get to the correct diagnosis, you might have to overcome the “framing effect” in which your cognition has been biased by the way the patient’s problem was first presented to you. In the case of Mr O, your intuition may have been biased toward thinking that the patient is depressed with somatic complaints precipitated by his recent retirement.

Now you escort the patient from the waiting room and by the time you and the patient have settled into the chairs in your office you have new information to include in your diagnostic formulation. Your system 1 cognition immediately registered that Mr O had parkinsonian features. His affect was somewhat flat and he walked slowly, with a diminished arm-swing.

Now you use system 2 logic in an effort to piece together the information you have so far. Does the narrative presentation fit with your observations? You interview the patient, carefully framing your questions to better understand the patient’s symptoms and the arc of his illness over time.

**The diagnosis of Parkinson disease**

Knowledge and experience are both crucial in making the diagnosis of Parkinson disease in Mr O’s case. In addition to the blunting of facial expression and “feeling stiff and somewhat unsteady on his feet,” you learn that Mr O has been worried about falling, especially when he walks down a staircase. This is consistent with a shift in the center of gravity and forward propulsion associated with Parkinson disease. Mr O also has had depression, prominent constellation, and fatigue—these symptoms are often prodromal in Parkinson disease, Lewy-body dementia, multiple systems atrophy and other, less common parkinsonian syndromes. If you know the symptoms for Parkinson...
Avoiding Diagnostic Error

What is a diagnostic error?
The Institute of Medicine (now the National Academy of Medicine) definition of diagnostic error: “a failure to establish an accurate and timely explanation of the patient’s health problem(s) and to communicate that explanation to the patient.” This definition crucially includes: the patient, the factor of time, and the notion that health problem(s) have complex “explanations,” and are not simply “diagnostic labels.”

What is the diagnostic process?
- The diagnostic process is a complex, information gathering and processing endeavor.
- It is collaborative, involving the patient, family, and other health care professionals.
- It is iterative, taking place over time.
- The diagnostic process occurs within a health care system.

Another common bias is a tendency to look for confirming evidence—“confirmation bias”—rather than disconfirming evidence, although disconfirming evidence is much more powerful. It turns out that Mr O’s symptoms of restless sleep, constipation, fatigue, and depression actually began before he retired, not after.

Furthermore, it is helpful for clinicians to be aware of the possibility of “premature closure,” the tendency to stop investigating once you have come upon a diagnostic hypothesis but before that hypothesis has been fully proven. This is an especially easy error to make when a medical condition has a long prodromal period without symptoms that are specific enough to make a clear medical diagnosis. As psychiatrists, we often encounter diseases of this type; Parkinson disease is but one example. Others include: multiple sclerosis, Wilson disease, Alzheimer disease, Cushing disease, fronto-temporal dementia, Huntington disease, and more. Periodically reviewing a patient’s condition while maintaining diagnostic uncertainty forces the clinician to rely more on system 2 cognition.

Non-specific behavioral and mood alterations often represent the very first and, occasionally, for prolonged periods of time, the one single and exclusive sign of an undetected physical illness. Flagrantly and convincingly “psychological” in nature on presentation, such masked physical conditions frequently mislead the examiner and obliterate any further medical consideration, resulting in misdiagnosis and thus, inevitably, in treatment gone astray.

Recent studies of diagnostic error have not focused on patients who are being treated in mental health clinics or inpatient psychiatric units; however, past studies of psychiatric patients found that, important medical diagnoses were missed in these patients as often as 40% of the time, depending upon the setting. Moreover, these medical diagnoses were not uncommon the sole cause or a significant contributing factor to the patient’s presentations.

What can you do?
How can you contribute to decreasing diagnostic error rates? Expert opinion emphasizes: expand your knowledge; cultivate good clinical habits of mind, including expanding your knowledge and experience, self-care, humility, persistence, and the use of feedback; and utilize the use of metacognition—thinking about thinking, and work to improve the institutions within which you work.

Expanding your knowledge
There is much for psychiatrists to learn in the realm of neuropsychiatric disease. Often patients with medical conditions will first present to psychiatrists thinking they have a psychological problem or a mental illness. Many will have common diseases: focal seizure disorders; neurodegenerative diseases in addition to Parkinson disease, such as Alzheimer disease, vascular dementia, or Lewy body dementia; the long-term consequences of traumatic brain injury; autoimmune diseases such as multiple sclerosis; or sleep disorders. Some patients will have diseases that are less common: anti-N-methyl-D-aspartate receptor encephalitis, Wilson disease, Huntington disease, frontotemporal dementia. Familiarize yourself with the clinical presentations of these and other neuropsychiatric conditions. And remember to keep this question in the front of your mind: Could this patient have a medical condition that I am missing?

Metacognition
Diagnosing is a high-level creative process; as such it relies on reflective practices that allow time for you to think about your reactions to patients and about your diagnostic process. Even with a few moments of mental stepping back to get perspective are important. In addition, actively employ strategies that force you to use the conscious, analytic system 2 cognition. Ask yourself, “What could this be?” Make a differential diagnosis list. Work against premature closure in the face of doubt and uncertainty. Remember that keeping a diagnosis “open” forces your “problem-solving mind” to keep working on what the diagnosis might be.

Improving health care systems in support of diagnosing
Psychiatrists rely on hospital systems to support the diagnostic endeavor. Health care institutions may provide effective communication systems that allow collaboration with the patient and his or her family, health information technology, reliable laboratory services, access to library resources, expert second opinions, collaboration across disciplines, and specialties, and a culture that values reduction of diagnostic error rates while, at the same time, avoiding shaming and blaming.

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Janice, aged 42 years, has been having seizure-like events for the past 4 years, uncontrolled by a series of anti-seizure medication trials. During the seizures her body shakes uncontrollably, her back arches painfully, and she frequently falls. Her surroundings appear blurry, and sounds are muffled or entirely inaudible. Afterwards, her memory of the seizure is patchy and incomplete, and she is sometimes entirely unaware she has had an event.

Janice has been unable to work and is on medical disability from her job as a home health aide. She left a physically abusive relationship 2 years ago and now spends much of her time alone. She has lost interest in visiting with friends and sleeps for much of the day. She attributes her depressed mood and increasing anxiety to the frequent seizures and to her doctors’ failed efforts to treat them.

Prompted by the failure of 5 different anti-seizure medications over 4 years, Janice’s neurologist refers her to a tertiary care epilepsy center. In the epilepsy unit she is continuously monitored with digital video-electroencephalogram (vEEG) and electrocardiogram for 5 days while her anti-seizure medications are withdrawn. Two typical seizure-like events are captured on vEEG, and based on normal brain activity and the clinical appearance on video review, the diagnosis is psychogenic nonepileptic seizures (PNES).

Janice is advised to obtain psychiatric care and given contact information for three psychiatrists: one is not accepting new patients, one did not accept Janice’s Medicaid insurance, and the third offers her an appointment in 6 months. When Janice finally presents for the appointment, she has a seizure in the waiting room, prompting the psychiatrist to summon paramedics who take her to a hospital emergency department. Janice is discharged with instructions to follow up with her neurologist. When she calls the neurologist, he tells her that she does not have a neurological disorder and that she should schedule another appointment with the psychiatrist. When Janice calls the psychiatrist, he tells her that she has a neurological problem and requires further neurological evaluation.

Finally, frustrated by the lack and forth, Janice’s primary care physician refers her to a second epilepsy center, where she is again evaluated with vEEG and confirmed to have cancels or reschedules nearly half her appointments, but she also recognizes the value of the skills she learns to identify and manage her stress before it builds to the point of a psychogenic attack. She begins to recognize the sadness and fear she still experiences because of the abuse she experienced and develops skills to manage these emotions.

The PNES gradually decrease and finally stop entirely. Her depressive symptoms also improve significantly with venlafaxine and CBT. Janice remains PNES-free and participates in psychotherapy every 2 weeks. She begins working outside the home for the first time in 4 years as a volunteer in an animal shelter.

**Overview of the disorder**

DSM-5 categorizes PNES as a functional neurological disorder or conversion disorder with seizures. In contrast to other functional neurological disorders, such as functional movement disorders and psychogenic paralyses, PNES are transient and paroxysmal, often triggered by a stressor. The majority of patients have a history of emotional, physical, or sexual trauma, and 94% have one or more psychiatric comorbidities including (in order of decreasing frequency) depression, anxiety, PTSD, and personality disorders. Other medically unexplained symptoms such as fibromyalgia, chronic fatigue syndrome, and irritable bowel syndrome are also disproportionately common among patients with PNES, and measures of alexithymia are elevated.

 Unlike factitious disorder and malingering, patients with PNES and other functional neurological disorders do not consciously produce symptoms and may experience symptoms even when there is no possibility of primary or secondary gain.

Early structural and functional magnetic resonance imaging (MRI) studies suggest that there are both structural and functional differences between the brains of patients with PNES and the brains of healthy controls. However the detected differences vary somewhat between studies, suggesting possible heterogeneity in underlying neuropathological correlates, and are not yet validated for diagnosing individual patients in a clinical setting.

PNES cause significant suffering and disability, with worse health care related quality of life than that associated with epileptic seizures. Most patients with undiagnosed and/or untreated PNES continue to have seizures and remain on disability. The majority of patients with PNES are misdiagnosed with epilepsy and are treated with at least one anti-seizure medication before the correct diagnosis is made.

Unlike epileptic seizures that are caused by abnormal hypersynchronous electrical discharges in the cortex of the brain, PNES are not associated with abnormal neuronal discharges. This means that PNES do not benefit from anti-seizure medications, and that they can be readily distinguished from most epileptic seizures by evaluation of brain activity during a typical event with an EEG. Fully a quarter of civilians and veterans evaluated for putative epileptic seizures in epilepsy monitoring units ultimately receive a diagnosis of PNES.

The average time from the onset of symptoms to the correct diagnosis is several years. Unfortunately a prolonged delay to diagnosis is a prognostic marker for poor treatment outcome.

**Diagnosis**

The gold standard for diagnosis of PNES entails capture of all habitual episode types on vEEG, with normal brain activity before, during, and following the event as well as semiology (signs and symptoms) consistent with PNES. While vEEG capture of
TABLE. Criteria for diagnosis of psychogenic nonepileptic attacks: history characteristics consistent with PNES

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<tr>
<th>Diagnostic level of certainty</th>
<th>Witnessed event</th>
<th>EEG</th>
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<tbody>
<tr>
<td>Possible</td>
<td>By witness or self-report</td>
<td>No epileptiform activity on routine interictal EEG</td>
</tr>
<tr>
<td>Probable</td>
<td>By clinician who reviewed video recording or in person</td>
<td>No epileptiform activity on routine interictal EEG</td>
</tr>
<tr>
<td>Clinically established</td>
<td>By clinician experienced in diagnosis of seizure disorders (on video or in person), while not on EEG</td>
<td>No epileptiform activity on routine or ambulatory ictal EEG during a typical event in which semiology would make epileptiform activity expected during an equivalent epileptic seizure</td>
</tr>
<tr>
<td>Documented</td>
<td>By clinician experienced in diagnosis of seizure disorders, showing clinical signs and symptoms of an ictal event typical of PNES, while on vEEG</td>
<td>No epileptiform activity immediately before, during, or after all typical events, with typical PNES semiology on vEEG</td>
</tr>
</tbody>
</table>

PNES, psychogenic nonepileptic attacks; vEEG, video-electroencephalogram. Adapted with permission from LaFrance et al.5

all typical events is necessary to make a diagnosis of PNES, the diagnosis may be made with lower levels of certainty as possible or probable PNES based on interictal EEGs and verbal descriptions or video recordings of typical seizures (Table). In some cases, where typical attacks occur rarely (less than once per 2 weeks), it may be impossible to capture events on vEEG, and a diagnosis of probable PNES made with smartphone video recordings of seizures may be the next best alternative. A promising alternative for such cases may be surface electromyography, which can be employed for weeks or months at a time and may be able to reliably distinguish PNES from epileptic seizures in the near future.

Treatment

Once the diagnosis of PNES is made, psychotherapy is the treatment of choice. Many approaches have been attempted to treat PNES, including brief interpersonal psychodynamic psychotherapy, acceptance and commitment therapy, mindfulness-based therapy, hypnosis, and psychosocial education. However, studies either did not compare treatment outcomes to those of a control group or were underpowered to demonstrate efficacy among patients with PNES.

There is a growing body of evidence, including two small randomized trials, demonstrating that weekly CBT informed regimens are effective in reducing PNES frequency and improving quality of life. The first trial, undertaken by Goldstein and colleagues,6 draws primarily on standard CBT principles and techniques. The second, undertaken by LaFrance and colleagues,7 is more eclectic. It incorporates psychodynamic, mindfulness, and interpersonal therapy theory and techniques into the CBT.

For both 12-session regimens, the primary goal of treatment is to reduce the frequency of psychogenic attacks. Both modalities teach patients to identify their environmental, physical, and emotional triggers and to use various relaxation and cognitive techniques to prevent negative thought patterns from progressing to psychogenic attacks.

The model used by LaFrance and colleagues includes additional sessions that identify motivations for behavior change and teach communication and conflict resolution skills. A session on medication management is also included.

SSRs have not shown a consistent benefit for PNES. Psychopharmacologic therapy is therefore recommended for treatment of psychiatric comorbidities but not for PNES itself. Rapid titration off all anti-seizure medications under vEEG monitoring at the time of diagnosis has been shown to result in improved seizure frequency and a greater internal locus of control for patients.8

Obstacles to care

While vEEG has become increasingly widespread, and awareness of the disorder is increasing, the majority of patients still do not receive a prompt diagnosis and evidence-based treatment. Obstacles to care include limited access to vEEG monitoring, stigma from clinicians and the public, a lack of training for clinicians, a short- or behavioral health specialists, restrictions on driving, and poor communication between the neuropsychologists who diagnose PNES and behavioral health specialists who provide treatment.

Even in circumstances where these systemic obstacles are successfully addressed, patient ambivalence about the condition and treatment may result in the majority of patients dropping out of treatment. Treatment nonadherence results in worse outcomes. Interventions such as motivational interviewing and improved communication between specialists can help to improve patient adherence with psychotherapy as well as treatment outcomes.

Direct verbal communication between neurologists and behavioral health specialists can be helpful in clarifying the diagnosis of PNES and aiding in diagnosing new somatic symptoms that frequently arise during the stresses of psychotherapy. It is essential that neurologists, psychiatrists, and other clinicians coordinate care to ensure that patients with PNES receive a prompt diagnosis and referral to psychotherapy.

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Catatonia

Continued from page 13

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SPECIAL REPORT
Vicarious Trauma in Clinicians: Fostering Resilience and Preventing Burnout

Gertie Quitangon, MD

Psychiatrists and other mental health clinicians have a distinct susceptibility to vicarious trauma from repeated exposure to aversive details of patients’ traumatic experiences. Understanding trauma has evolved since combat stress was first observed in soldiers returning from war, and the advent of the MeToo movement heightened awareness of the prevalence of trauma in the general population. The recognition of a wide range of traumatic experiences—physical or sexual assault, motor vehicular accidents, life threatening illness, unexpected death or serious injury to significant others, bearing witness to severe human suffering, natural disasters, war, terrorism—has implications for understanding the vulnerability to vicarious trauma inherent in a clinician’s practice.

Empathy in validating another’s suffering makes the clinician vulnerable. Vicarious trauma refers to negative changes in the clinician’s view of self, others, and the world resulting from repeated empathic engagement with patients’ traumatic thoughts, memories, and emotions. This construct is based on the Constructivist Self-Development Theory (CSDT) of personality that conceptualizes trauma as a disruption of human development and adaptation, specifically disruption of the basic human needs for safety, esteem, trust, control, independence, and intimacy. From the CSDT perspective, the clinician’s efforts to adapt to exposure to patients’ traumatic memories and emotional responses is disrupted. Empathic listening to story after story of human suffering challenges the clinician’s deeply held beliefs, assumptions, and expectations, which can manifest as intrusive thoughts and images as well as other emotional and behavioral manifestations.

Overlapping work-impact concepts: burnout, compassion fatigue, vicarious trauma

There is consensus in the professional community that secondary exposure to patients’ trauma has the potential to negatively affect quality of care and professional well-being. However, the overlapping theories and constructs are not commonly well understood by service providers. In reviewing the literature, the most frequent constructs used interchangeably with vicarious trauma are secondary traumatic stress, compassion fatigue, and burnout.

Compassion fatigue was originally referred to as secondary traumatic stress syndrome observed in caregivers and family members of trauma survivors who mirrored symptoms of PTSD but with lesser intensity. The term compassion fatigue, coined by Charles Figley, is thought to be a less stigmatizing characterization than secondary traumatic stress. Defined as empathic strain and general exhaustion resulting from caring for people in distress, compassion fatigue is most often associated with helping professions such as first responders, nurses, physicians, and disaster recovery workers. Similar to vicarious trauma, it involves empathic engagement and secondary trauma exposure, which could present as PTSD-like symptoms. Unlike vicarious trauma however, symptoms can manifest without cumulative secondary trauma exposure, and compassion fatigue is not associated with cognitive disruptions.

Unlike vicarious trauma, both compassion fatigue and burnout are not specific to clinicians who work with trauma survivors, but all three constructs describe manifestations of emotional and physical exhaustion. Burnout is defined as a persistent state of exhaustion, cynicism, and inefficacy as a result of work-related stress. The central domain of burnout is emotional exhaustion due to high work demands and often presents as frequent absenteeism, chronic tardiness, and underperformance on clinical and administrative responsibilities.

Secondary exposure to trauma is a job-related risk and if left unaddressed the resulting, vicarious trauma and burnout could progress to PTSD.

Is vicarious trauma a specific form of burnout?

Vicarious trauma is the only construct that specifically describes a cumulative, long-lasting impact on clinicians’ personal beliefs and world view. However, on closer examination, two of the three domains of burnout—cynicism or depersonalization and reduced sense of accomplishment or inefficacy—describe shifts in cognition, emotion, and behavior. Vicarious trauma and burnout have been considered conceptually distinct from the assumption that cognitive disruptions associated with burnout are limited to work conditions while the effects of vicarious trauma are wider in scope. There are more polemical arguments about all the overlapping concepts than evidence in the literature to support this assumption. Conducting more research has been widely recommend-

Can Psychiatric Association revised the PTSD diagnostic criteria DSM-5 and they added “repeated or extreme indirect exposure to aversive details of a traumatic event” as a qualifying stressor to meet criteria for diagnosis of PTSD. This criterion supports the assertion that secondary exposure to trauma is a job-related risk and suggests that if left unaddressed, vicarious trauma and burnout could progress to PTSD.

Studies have shown that integrating work safety and wellness programs is more effective in reducing chronic conditions. Wellness and self-care practices reduce individual risk-related factors while work safety reduces work-related risk factors. Examples of wellness programs are smoking cessation, weight control, healthy nutrition, physical activity, flu vaccination, meditation, and mindfulness. Combining wellness practices and psychological safety at work for clinicians is a comprehensive risk management approach to prevent vicarious trauma and burnout and foster resilience.

Meaningful action to foster resilience and prevent vicarious trauma and burnout

Integrate vicarious trauma education and training in curriculum

Residency training programs, internships/externships can adopt a primary prevention approach by integrating vicarious trauma and burnout in the academic curriculum. A course on trauma and PTSD is not complete without teaching vicarious trauma and a forum on burnout without discussing vicarious trauma is an oversight. FEMA and SAMHSA have recognized the need to better understand the negative mental health outcomes of disaster work by mandating programs to include training events and support services on compassion fatigue, secondary traumatic stress and vicarious trauma prior to deployment.

Screening and self-assessment

Screening is a secondary prevention strategy and many employers screen for workplace stress and offer health coaching, stress management and related programs when appropriate. Forensic workers responding to massive disasters undergo screening for stress-related disorders to monitor...
emergence of negative mental health outcomes.\textsuperscript{10,11} Similarly, offering self-assessment and screening tools for vicarious trauma raises awareness of personal strengths and vulnerabilities and establishes a baseline of symptoms that could be monitored over time.

There are a number of standardized tools that can assess symptoms of vicarious trauma (Table). Most of these standardized tools were developed to measure other work-impact concepts and has not been psychometrically validated to assess vicarious trauma. However, they have been adapted for research purposes and accepted as screening tools for vicarious trauma. They are not meant to be diagnostic tools. Rather these tools can be used to monitor changes in symptomatology longitudinally. Self-administered tools available electronically increases access and privacy and encourages staff participation.

**Enhance personal and professional supports**

Social support, both personal and professional, is a protective factor and has been associated with a decreased risk of vicarious trauma. Consultation with colleagues and peers has been shown to reduce feelings of isolation and increase feelings of efficacy.\textsuperscript{12,13} The role of managers and supervisors is key in enhancing staff support. Managers can use one-on-one supervision to provide support for challenging cases and manage caseloads, ensuring a balance of volume and complexity of trauma patients for each clinician. Supervisors can also assist staff in the development and implementation of self-care plans. A survey for therapists and hospital workers revealed that while the majority believed that self-care strategies can reduce the risk of vicarious trauma, very few actually reported using the strategies.

Research has shown an association between a high caseload of trauma victims and an increased risk of symptoms of vicarious trauma.\textsuperscript{12-14} Supervisors can also assist staff in the development and implementation of self-care plans. A survey of therapists and hospital workers revealed that while the majority believed that self-care strategies can reduce the risk of vicarious trauma, very few actually reported using the strategies. Dr Quitangon reports that she receives royalties from Routledge for her book. Vicious Trauma and Disaster Mental Health: Understanding Risks and Promoting Resilience.

**Dr Quitangon is Clinical Assistant Professor of Psychiatry, New York University School of Medicine and Medical Director, Community Healthcare Network, New York, NY.**

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**References**


**Helping Complex Patients NAMI as a Resource**

>>Ken Duckworth, MD and Christine Allen

The National Alliance on Mental Illness (NAMI) is a one-of-a-kind nonprofit that is here for your patients and their families, no matter where they are. As the largest grassroots organization in America for people experiencing mental illness, NAMI has a presence in every state, with hundreds of community affiliates nationwide.

NAMI was founded in 1979 by mothers who felt their children experiencing schizophrenia were not receiving proper care and support from the mental health care system. What started as a small, determined group of families gathered around a kitchen table has blossomed in the nation’s leading voice on mental health. Since its founding, NAMI has never stopped in its quest for better care, services, and research. This makes it a natural ally for any clinician seeking better outcomes for his or her patients.

**Peer connection**

Isolation is a common problem for people who have mental illness and support groups offer a safe space for like-minded peers to connect. Support groups are also a powerful way for your patients to get out, meet others, and discover different ways of approaching their challenges. NAMI has a variety of support groups that connect people with shared experiences; our members have “been there” and find a sense of purpose in helping individuals who are struggling.

In support groups like NAMI Connection and NAMI Family Support, your patients might also find resources for better housing, supported employment/education, or a recovery learning center they can join. These weekly meetings

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<tr>
<td>• Compassion Fatigue Self-Test (CFST)\textsuperscript{a}</td>
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<td>• Impact of Event Scale (IES)\textsuperscript{b}</td>
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<tr>
<td>• Professional Quality of Life Scale (ProQOL)\textsuperscript{c}</td>
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<tr>
<td>• Secondary Traumatic Stress Scale (STSS)\textsuperscript{d}</td>
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<tr>
<td>• Self-Care Assessment Scale\textsuperscript{e}</td>
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<tr>
<td>• Traumatic Stress Institute Belief Scale (TSI)\textsuperscript{f}</td>
</tr>
<tr>
<td>• Trauma and Attachment Belief Scale (TABS)\textsuperscript{g}</td>
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\textsuperscript{a}See table for notes.
might also lead to new friendships, which is itself an important outcome.

“So, what was NAMI, I wondered. I called and found out that there was a support group meeting near me in a few days, and that I was welcome to attend. I thought, ‘Why not go? What have I got to lose?’ Actually … I knew exactly what I had to lose if this didn’t work. So, I got up my courage, drove to the meeting location and walked into my first NAMI Connection meeting. It turned out to be the start of a new life for me.”

Fletcher M, South Carolina

Education for families

Families often find the mental health system difficult to navigate because mental illness and mental health care can be challenging to understand. Fortunately, there is a NAMI program that has been proven to be clinically effective to facilitate a better understanding of mental illness and mental health care. It’s called NAMI Family-to-Family.

NAMI Family-to-Family is a 12-session course for families, significant others, and friends of people with mental illness. The course is designed to provide a better understanding of the different conditions, increase coping skills and empowering participants to become advocates for their loved ones, if needed. Similarly, NAMI Basics is a 6-session course for parents and other family members who provide care for children and youth (aged 22 years or younger) who experience mental health symptoms.

“Every time I felt defeated and wondered if I should give up, the coping and problem-solving skills I learned in NAMI Basics came to my aid. Like many others, NAMI Basics saved my family. It gave us the tools to keep going and keep fighting for my son—his care, his happiness and his life.”

Anita H, North Carolina

NAMI Basics will be available online in late 2019. With this program, it won’t matter if you are referring a patient who lives in a rural community miles away from the nearest NAMI affiliate, or if you are referring a working parent of three with limited time to dedicate to an in-person class—soon, everyone will be able to access NAMI Basics on their own schedule.

Information and support

The NAMI National HelpLine fields thousands of calls a year, connecting concerned callers with mental health resources. NAMI HelpLine staff and volunteers are prepared to answer questions on a variety of issues including symptoms of mental illness, treatment options, local support groups and services, education programs, helping family members get treatment, programs to help find jobs, and much more.

“Almost every call ends with the caller sharing, ‘I can’t thank you enough. Before I called you, I didn’t know where to start. You’ve helped me so much.’ I cannot fully describe how good it feels to be able to speak with a caller—who at the beginning of a call feels helpless and lost—and help empower them with a sense of dignity, calm, and hope by the end of the call.”

Quinn Anderson, NAMI HelpLine Manager

Your patients can also visit NAMI.org where they will find up-to-date information, research and discussion boards. Of note, The College of Psychiatric and Neurologic Pharmacists (CPNP) posts medication fact sheets on our website for public use. These resources review a variety of topics, such as adverse effects, black-box warnings, and other considerations when taking psychiatric medications. CPNP has also developed resources on general issues related to medication delivery and action, including long-acting injectable, generic medications, and medication-induced weight gain. These fact sheets are a popular resource on our website and are available to you for share with your patients and their families.

NAMI also runs a series of educational webinars on topics of importance to our members. These webinars, called Ask the Expert, invite America’s leading health care professionals and researchers to cover topics such as the efficacy of ketamine, how to take care of one’s physical health while on atypical antipsychotics, and cognitive behavioral therapy for psychosis skills. Anyone can attend these webinars.

Advocacy and empowerment

Many of the best outcomes in terms of legislation and policy are based on first-person testimony. That’s because few things are more powerful than speaking your truth. NAMI can help guide your patients and their families in effectively fighting for better services and care, if they want to do so. NAMI Smarts for Advocacy is a hands-on advocacy training program that helps people with mental illness, and their families transform their lived experience into skillful grassroots advocacy.

This program is designed to develop the following skills:

- Telling a compelling story that makes an “ask” in 90 seconds
- Writing an effective email
- Making an impactful phone call
- Orchestrating a successful meeting with an elected official

“Like you, I am pained by our nation’s mental health crisis and how it affects the people I work with and serve. But I take comfort in knowing that mental health advocates across the country work tirelessly to advance research, mitigate the tragic consequences that stem from lack of services and promote programs that offer promise and hope to future generations. Together, we can create a brighter tomorrow.”

Mary Giliberti, CEO of NAMI

Every advocacy movement needs boots on the ground; NAMIWalks offer an avenue to connect with the mental health community while raising awareness and funds. More than 5000 people participated in the Boston NAMIWalks last year and the energy there was powerful. It is hard to feel alone with 5000 people around you, standing up for the same things you stand for.

Assistance for mental health professionals

NAMI is a participant of the SAMSHA/APA Clinical Support System for Serious Mental Illness project, otherwise known as SMI Adviser. This project offers education, data, resources, and consultations to clinicians (physicians, nurses, recovery specialists, peer-to-peer specialists, and others), so they can more effectively provide evidence-based care to people with serious mental illness. In collaboration with the APA, NMAI will be creating resources—fact sheets, webinars, podcasts, videos, and more—on topics of mutual interest for providers like you, as well as your patients and their families.

This project reaches deep within the underserved, misunderstood serious mental illness community to provide education and support that NAMI could never deliver on its own. We’re thankful to the APA and SAMSHA for including us in this vital and significant work, and we welcome you to keep up with our contributions at www.smiadviser.org.

What you can do

As a psychiatrist, you have a lot to offer the mental health community, as we all have a shared mission: patient health and wellness. So, start by connecting with and becoming a NAMI member at your local NAMI affiliate. See how you can get involved with NAMI activities. Maybe you could speak at a state convention. Whatever

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<td>Resource</td>
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<td>NAMI</td>
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<tr>
<td>Find Your Local NAMI</td>
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<tr>
<td>NAMI HelpLine (M-F, 10 AM – 6 PM ET)</td>
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<tr>
<td>NAMI Programs (eg, Family-to-Family, Connection, Ending the Silence)</td>
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<tr>
<td>Mental Health Medications</td>
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<tr>
<td>NAMI’s Ask the Expert</td>
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<td>NAMI Smarts for Advocacy</td>
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<tr>
<td>NAMIWalks</td>
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<td>SMI Adviser: A Better Tomorrow</td>
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SMI, serious mental illness.
MEDICATION TOO EXPENSIVE?
Megan Ehret, PharmD, MS, BCPP, President, College of Psychiatric and Neurologic Pharmacists

Patient assistance programs were created by pharmaceutical companies to provide free or discounted medications to patients who are unable to afford them. Each pharmaceutical company creates its own program with its own qualifying criteria to receive free medication. Many times, a quick internet search for drug-specific assistance programs will locate the criteria and application process. Here are three websites that can help even further.

1. Need Meds (www.needymeds.org) has a search tool that can help users locate various patient assistance programs and companies who can help complete the application.
2. Rx Assist Patient Assistance Program Center (www.rxassist.org) gives providers, advocates, consumers and caregivers comprehensive, up-to-date information on available patient assistance programs.
3. Rx Hope (www.rxhope.com) is a service free to prescribers and patients that allows prescribers or their office to complete applications on behalf of the patient.

You can also bring mental health awareness to your local school system. NAMI Ending the Silence is a presentation designed for middle and high school students, staff, and the parents or guardians of middle- or high-school-aged youth. Audiences learn about the signs and symptoms of mental health conditions, how to recognize early warning signs and the importance of acknowledging those warning signs.

Early intervention is essential to improving long-term outcomes for young people with serious mental illness—being exposed to this kind of information early is indispensable.

NAMI is devoted to improving the lives of people with serious mental illness and those who love them.

Dr Duckworth is Medical Director, The National Alliance on Mental Illness, Washington, DC; Ms Allen is Senior Content Strategist, The National Alliance on Mental Illness, Washington, DC. The authors report no conflicts of interest concerning the subject matter of this article.

Reference

EXCLUSIVE CLIMATE CHANGE COVERAGE

The Impacts of Extreme Heat on Mental Health

Robin Cooper, MD

It is hot out there! No doubt the climate is changing dramatically, and the evidence of profound heat waves and their impacts confront us in large and small ways on a regular basis. Record temperatures are reported year after year. 2016 set record high temperatures; then in 2017, despite La Niña’s cooling influence, temperatures nearly reached the 2016 records. 2018 was not much better, clocking in as the 4th hottest year globally with the US experiencing the hottest May in recorded history. Since the start of the 21st century, the annual global temperature record has been broken five times.

The beginning of 2019 has begun with weather extremes wreaking havoc with a severe “polar vortex” in North America while Australia baked in blistering heat. With global warming, extreme heat is becoming the new normal.

Extreme heat makes most of us cranky, more likely to have temper flares and feel agitated or listless. Yet heat waves are not benign uncomfortable periods but have profound health risks with potential for death. Heat waves are now considered the deadliest weather events, exceeding hurricanes, lightning, tornadoes, floods, and earthquakes combined. The death toll in India, a country with extreme poverty, experienced the fifth deadliest heat wave in history in 2015. That heat wave claimed 2500 heat-related deaths. By comparison, in the US where access to air-conditioning is more readily available, the CDC reported that between 1999 and 2010 there were an average of 618 deaths annually due to extreme heat.

Extreme heat can have significant effects on mental health and behavior. Given the predictable future of extreme heat waves potentially increasing the population exposure 4 to 6 times by mid-century, it is incumbent upon the psychiatric and mental health community to be knowledgeable about the specific impacts on behavior and psychiatric outcomes and to plan for ways to protect our patients and communities.

Impacts of extreme heat on behavior and psychiatric conditions

Violence. Evidence linking extreme heat and aggression confirms the general understanding expressed in our colloquial language of “hot head-
ed.” “so hot my blood boils.” One standard deviation of temperature increase leads to a 4% increase in interpersonal violence and 14% increase in group violence. This has significant implications for domestic violence and impacts on women and children. Burke and colleagues warn of the potential of climate change and global warming assuming no reduction in green-house gas emissions, it is estimated that by 2050 there may be between 9000 to 40,000 additional suicides in the US and Mexico during periods of 1 degree Celsius increase over average monthly temperatures. Projecting future impacts of climate change and global warming assuming no reduction in green-house gas emissions, it is estimated that by 2050 there may be between 9000 to 40,000 additional suicides in the US and Mexico. These rates are comparable to the effects on suicide incidence due to economic recessions and unemployment and offset gains in suicide prevention programs and gun control policies.

The negative effects of climate change are not equally distributed, and people with mental illness and substance abuse disorders are among the most vulnerable to the effects of extreme heat and other climate-change related events. Poverty, substandard housing, and lack of access to cool environments all contribute to this increased vulnerability. Homeless mentally ill patients have little control over their environments, limited ability to protect themselves from heat exposures, and therefore they are at extreme risk.

The most protective tool against heat stress/stroke is the availability of functioning air conditioners. Unfortunately, these kinds of cooling systems are out of reach for many people who live on the margins of society and need assistance in accessing public cooling respite centers.

As mental health care providers, we must do everything we can to protect our patients and our communities. Many communities have developed programs in partnership with public health, social services, and emergency response departments to respond and protect people from heat impacts. These entailed public education campaigns as well as providing services and cooling centers. As mental health providers, we must educate both our patients and colleagues, and collaborate with leaders in the public health delivery systems to design preventive and intervention strategies to protect our vulnerable patients and communities from the adverse effects of extreme heat.

The CDC has many available resources including a brochure to guide program development and patient interventions (see Infographic). However, we cannot stop there. We must go further and use our professional voices to advocate for policies that get at the root causes of global warming.

References
Reviewed by Lloyd I. Sederer, MD

I was a first-year psychiatric resident at Upstate Medical Center, at the SUNY in Syracuse. I would be dazzled and envious sitting in a seminar with the by then legendary, Dr Thomas Szasz. Everyone seemed to regard him in either a hallowed or contentious way since his book, *The Myth of Mental Illness*, was published almost 10 years earlier. My envy, by the way, came from wanting to have a mind like his: I was ready to make a deal with the devil if I were suddenly able to think as well as he did. His capacity to formulate, articulate, and argue ideas was as keen as was this lean, diminutive (in size) Eastern European who never lost his accent.

The seminar would end, as would my academic cocoon. It was time for me to leave the classroom and run a few blocks to the state psychiatric hospital, where my on-call would begin at 5 PM. Breathless, I arrived at the admitting office to find the Syracuse police waiting for me. They were surrounding a 250-pound agitated, psychotic man. Sometimes there would be two patients. However many, patients were generally exceptionally displeased to be held against their will. The local cops smiled and said, “Hey Doc, you’re late. Here’s your patient. We’re outta here.”

“Myth” certainly confronted the reality I experienced at that moment. As it did throughout my career as a psychiatrist. I have had to wrestle with Szasz’s ideas, especially during my years as Medical Director of McLean Hospital, Mental Health Commissioner for NYC, and, for the past 12 years, as Chief Medical Officer of the largest state mental health agency in the United States. I am grateful for Szasz’s influence on me, which has insisted that I take no ideas for granted, recognize the social power I can exercise, and try to do the right thing, and enduring, of his metaphors is the “myth of mental illness.” His work in the book so titled, and some 30 volumes and hundreds of papers that followed, brought him the mantle of anti-psychiatry. But he was not; he was a libertarian who held the importance of freedom over everything else (and he was clear to also say that with liberty came personal responsibility).

Haldipur and colleagues unbundle Szasz’s vast range of intellectual productions in 19 chapters divided into three book sections in *Intellectual Roots of Szasz’s Thought: The Concept of Mental Illness; and Szasz’s Larger Impact, as well as a superb summary epilogue by Dr Haldipur. The writings consider not just the material Szasz produced but the man and his compelling, often disruptive, rhetorical. The chapters draw from psychiatry, psychoanalysis (Szasz was formally trained as an analyst in Chicago before moving east), forensics, social contract theory, values, ethics, language, communications theory, and good old medical wisdom.

The contributors do not shy away from some of Dr Szasz’s most contentious claims, including: his highly vocal opposition to involuntary hospitalization and treatment of people with mental illness; his unwavering position that suicide was a right, not to be denied people under any circumstances (hard to bear since we know the compulsion to kill oneself is usually transient—not necessarily in thinking but in action, which can result in an irrevocable act—and that those who do not die are almost always grateful they survived); that “doctoring” was instrumental in helping people with “problems in living,” as was prescribing psychoactive medications; to allowing people with mental disorders to “die with their rights on”; and the inanity of the insanity defense.

Thomas Szasz was born in Budapest, Hungary. His educated, Jewish parents fled their native country to the United States in 1938, when Hitler invaded Austria. They understood that Hungary was next in the Nazi’s sights (as it later would be oppressed by Communism). Szasz was 18 and multilingual when he arrived in this country, speaking French and German in addition to his native Hungarian, but he spoke not a word of English. You would never know it because it was in the English language he immediately excelled in medical school and medicine.

Dr Szasz, whom I knew and learned from for 50 years, was a man whose persona was warm and engaging, except when publicly delivering his polemical arguments. He meant to abominate about the unintended consequences of would-be good intentions—that individuals needed to be protected from the power of the collective. His aim was to stir, not lull, the minds of all of us entrusted with the privilege of serving people with mental (and addictive) disorders. You may not have agreed with him, but he would surely put your assumptions and conventions to the test. There was no such thing as casual thinking or parroted instruction when in a conversation with him. That was a gift, whether you felt it or not, to all who knew, read, and debated him over the decades of his intellectual prowess.

Dr Thomas Szasz died in 2012 at the age of 92. I wonder what he would think of this comprehensive, intellectually challenging, carefully and clearly written analysis of his work? While I cannot imagine what he might say from a podium, I believe, privately, he would have appreciated the rigor, range, and intelligence brought to bear in this book. For those who knew or read this extraordinary man, your reading *Thomas Szasz: An Appraisal of His Legacy* will light up your remembrances. For those who barely know him or his work, you will surely be amazed by his contributions, whether you think them right or not (or some of both).

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Reference

**Book Review**

**Thomas Szasz: An Appraisal of His Legacy**

Reviewed by Lloyd I. Sederer, MD

*Thomas Szasz: An Appraisal of His Legacy* (Scribner, 2018), is now available in paperback.
The experts who evaluated Lorena Bobbitt agreed that she suffered from a mental illness at the time of the criminal offense. However, the use of the battered wife syndrome as a diagnosis was questionable in 1993 and remains so today. Although the definition of a mental illness or defect for the purpose of an insanity defense does not specifically reference DSM, most experts rely on DSM. The use of diagnoses not described in DSM raises questions about the scientific validity and acceptability of such diagnoses. The relevance of battered wife syndrome to the outcome of the Bobbitt case is unclear. Those who doubt the disorder speculate that battered wife syndrome was a way to recognize Lorena’s abuse and generate a narrative to address female victimhood and gender stereotypes. At the request of Lorena’s defense team, the judge instructed the jurors on irresistible impulse, requiring only that they find that Lorena’s mind was “so impaired by disease that she was unable to resist the impulse to commit the crime.” The jury found that Lorena met criteria for the volitional prong—namely that she cut off her husband’s penis because of an irresistible impulse fueled by her traumatic experiences.

Conclusion

Whether Lorena met criteria for an insanity defense may not be the most significant analysis of the case. Perhaps more important, and undeniably interwoven, is the impact this case has had on American society. Coverage of the Bobbitt case stimulated a discussion about whether describing women as “battered” fuels the stereotype of women as weak, helpless, or pathetic. Although some herald Lorena as “fighting back” against male aggression, her overall demeanor, including witness accounts, portrayed her as a fragile, “beaten” woman. Others identified Lorena as a symbol of female empowerment, resisting gender suppression.

Lorena’s acquittal may resonate with another societal misperception, that women are not perpetrators of violence, but rather victims. Women in general receive lighter sentences in the judicial system than their male counterparts. Models of sanity are gendered, and female sanity, or proper femininity, revolves around the belief that violent women are either seen as not sane or not women, because sane women are never violent.

ADHD

Continued from cover

approximately doubled for individuals who received amphetamine compared with those who received methylphenidate. This supported their hypothesis, which was based on the similarities between idiopathic psychosis and the biologic effects of amphetamine. Individu- als with primary psychosis have higher presynaptic dopaminergic capacity, which is an index of dopamine release.

Limitations of the study included: unmeasured confounding factors (ie, underreporting of substance use disorder), not knowing if the patients actually took the stimulant medication (there is a high rate of diversion, which is greater for amphetamine than methylphenidate), and the study findings, are not generalizable as the data were only from commercial insurance claims and did not take into account patients with no insurance or public insurance.

While the exact therapeutic mechanism of action of stimulants is unknown, they are thought to increase the availability of dopamine in the postsynaptic cleft by blocking the reuptake of dopamine. Amphetamine blocks the ability of the dopamine transporter to remove dopamine from the synapse, facilitates the reuptake of dopamine across the cell membrane, increases the release of vesicular dopamine (accumulates in the cytoplasm), and inhibits the degradative enzymes monoamine oxidase A and B. Methylphenidate binds to the dopamine transporter in the presynaptic cell membrane, blocking the reuptake of dopamine and, therefore, causing an increase in extracellular dopamine. The difference is that methylphenidate does not promote dopamine release from synaptic vesicles. Moreover, amphetamine induces the release of four times as much dopamine as methylphenidate. This increase of dopamine may contribute to adverse effects such as stimulant-induced psychosis.

Cases of stimulant-induced psychosis have been reported as early as 1938. However, the first case of a child who

Female sanity, or proper femininity, revolves around the belief that violent women are either seen as not sane or not women, because sane women are never violent.
experienced auditory, tactile, visual hallucinations, and paranoia after treatment with a therapeutic dose of D-amphetamine for hyperactivity was not published until 1967. The 8-year-old child’s symptoms of psychosis resolved after the medication was discontinued. A few years later, similar cases were reported by Lucas and Weiss after they observed “psychotic reactions” in two children following a short-term therapeutic dose and in an adolescent after ingestion of excess medication after long-term use of a therapeutic dose of methylphenidate, which had been prescribed for hyperactivity.

It was not until 2007 that the FDA mandated a drug label warning that stimulants may cause psychosis in patients with no prior history. There remains limited data on whether the risk of psychosis differs among the two classes of stimulants. While the incidence of new-onset psychosis with stimulant treatment for ADHD is small, this can be a frightening experience for the child as well as his or her parent and may negatively impact a parent’s decision to pursue stimulant treatment options. Thus, nonstimulant and non-pharmacological treatment options are often requested. Indeed, many parents continue to search for alternative treatment methods, especially for children with a newly established diagnosis. Until recently, there have been no viable non-pharmacological alternatives.

### Trigeminal Nerve Stimulation May Offer a Nonpharmacological Option

With concerns over potential adverse effects of medications, other strategies have been explored for treating ADHD. Preliminary studies using trigeminal nerve stimulation (TNS) to treat ADHD symptoms have peaked interest as a potential alternative or adjunctive treatment to stimulant and non-stimulant medications. TNS is a non-invasive, home administered, well-tolerated approach that has proven to be effective for epilepsy and MDD. Researchers became interested in TNS for ADHD after observing improvements in concentration and attention on mood disorder rating scales. The stimulation device is compact and worn on clothing during sleep. Thin wires from the stimulator are attached to adhesive electrode pads that are placed on the forehead bilaterally over the ophthalmic branch (V1) of the trigeminal nerve. A current is sent through the wires to activate the trigeminal nerve, which projects to the nucleus tractus solitarius activating the locus coeruleus and the reticular formation. These play a key component in cognitive functions, particularly in sustaining attention.

The recent double-blind sham controlled study by McGough and colleagues looked at cortical activation mechanisms using a quantitative electroencephalogram. The researchers found increased spectral power in the right frontal and frontal midline bands with active TNS, hypothesizing that activation of the frontotemporal ganglia network targets hyperactivity and impulsivity. This mechanism of action helps to support the overall findings for improvement in both the inattention and hyperactivity components of ADHD.

During trials, the pattern of improvement on ADHD Rating Scale IV (ADHD-RS) as well as on the Connor’s rating scale was comparable to that seen with non-stimulant medication. In this double-blind sham-controlled study by McGough and colleagues, symptomatic improvement was found to be highest after the first week of treatment; scores continued to improve over the next 3 weeks. While the sham group showed improvement over the first week, it was followed by a flattening response. One week after treatment discontinuation, rating scales were again administered in both the active and the sham groups. In both groups, scores from the ADHD-RS were found to decrease, indicating a recurrence of symptoms. This 4-week trial showed sustained improvement in both hyperactivity and inattention symptoms of ADHD with nightly active treatment.

Unfortunately, there are no long-term studies of mood or ADHD with TNS. Continued trials need to be undertaken to determine long-term outcomes as well as clear parameters for treatment.

The cost of the device used in the study, Monarch-TNS, is just over $1000; it is currently covered by insurance. Other available TNS devices range between $250 and $450; their efficacy, however, is unknown. It is difficult to compare the cost of TNS versus stimulant and non-stimulant medications, since there is such a wide range in cost. The monthly cost ranges from approximately $8 for a generic stimulant and may exceed $288 for a branded product. Most insurance companies will cover at least some medication costs.

TNS has been well tolerated with high safety and compliance rates. A long-term follow-up study for TNS use in epilepsy demonstrated skin irritation as the main adverse effect from treatment. Other commonly reported adverse effects of short-term TNS include headache and eye twitches. Based on the 4-week double-blind sham study, the manufacturer of the Monarch-TNS treatment device also noted drowsiness, increased appetite, trouble sleeping, and fatigue as possible side effects. No withdrawal effects have been observed.

Findings from a study by McGough and colleagues suggest that TNS may be effective for ADHD as an alternative treatment to stimulant and non-stimulant medications given symptomatic improvement in both inattention and hyperactivity symptoms of ADHD. Although direct comparison of TNS to pharmacological treatment has not been undertaken, treatment may be considered in children with parental preference against pharmacological treatment after providing psychoeducation or in children who cannot tolerate psychotropic medications. TNS as an adjunctive therapy may also be considered; however, adjunctive use has not been studied.

Currently, several limitations remain a concern. First, clear parameters (e.g., duration of treatment, number of treatments, the need for booster sessions) have not yet been identified. Second, there are no long-term studies for mood symptoms or ADHD, which limits the available data on long-term efficacy for TNS as a treatment. Third, the device itself is available but will come at a high out-of-pocket cost, thereby restricting treatment access. Based on these limited data, TNS appears to be an effective, safe, well-tolerated option with high compliance rates. Although more studies are needed, this treatment has high potential for use in managing the symptoms associated with ADHD.

### References

Things Are Seldom What They Seem

Continued from page 7

Unwilling to wait for a response to medical treatment when her hemoglobin was dropping, the gynecologist scheduled a myomectomy. By then, Maureen’s stomach pain had increased and interfered with traveling to our appointments. We decided to make do with telepsychiatry appointments until she recovered from surgery.

It was late in the day when Maureen telephoned me to say that her surgery took far longer than expected. The gynecologist called in a GI surgeon to assist with dissection of a bowel loop wrapped around the 5-pound fibroid. Maureen wasn’t sure of the specifics. What was certain is that Maureen’s worsening stomach pains were something other than the vague aches and pains of depression. The gynecologist warned her that she could have developed gangrene that required a bowel resection, or might even have died from sepsis, had the mucosal membrane ruptured, and had they not acted so quickly.

While she recovered, Maureen’s surgeon assumed responsibility for documenting her disability and our telepsychiatry appointments revolved around titrating her dose of antidepressants. I half-expected her to transfer all treatment to her PCP, but a few months later, Maureen scheduled an office appointment.

She bounced into the room, looking like a different person. Her cheeks were rosy; she had “pep in her step” or so she said. There were no signs or symptoms of depression. She said that this near brush with death made her appreciate life. I asked about the insurance paperwork, since insurance paperwork prompted her first appointment with me. Maureen said it was moot; her company collapsed since she left on leave, and her disability benefits disappeared. Yet she seemed happy. She received a severance package and paid COBRA benefits and now had renewed energy and enthusiasm to seek new work.

I, too, had renewed enthusiasm about the importance of medical training to the practice of psychiatry. I confess that I cringe when I hear people say that “psychiatrists prescribe medications” without considering the need for a differential diagnosis and the significance of the “big picture.” Maureen’s life was at stake; this was more than a matter of mood. Doing a review of systems was a rather simple matter and putting those pieces together could be done by any doctor. It wasn’t “rock-et science,” as they say—but it was a classic example of medical science and of why psychiatrists are physicians first.

That’s all well and good—but what happens when psychiatric physicians are not available? The supply is dwindling. There are some innovative solutions on the table, including “integrated care.” But that approach presupposes that patients already have PCPs that they trust and many younger people consult urgent care for minor problems without forging relationships with PCPs. Curiously, as per Peggy Drexler’s Wall Street Journal article on March 1, 2019,1 millennials are more comfortable with therapy, even texting therapy, and may bypass psychiatric assessment as well, as happened with Maureen.

Currently, pediatricians can fast-track into child psychiatry fellowships, but I don’t know of plans to fast-track PCPs into general psychiatry training. Perhaps that’s something to consider, especially for PCPs who miss the doctor-patient relationships that get lost in an increasingly impersonal and corporatized health care delivery system. Perhaps there is a solution in sight—but dismissing the value of medical training in providing psychiatric treatment is not the solution.

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Reference

Brain Health

Continued from page 4

Minimize “recreational” drugs

Although this recommendation sounds obvious, I am frequently surprised by the wide range of beliefs throughout the country as to what minimal, moderate, and problematic recreational drug use means. Some of my friends and patients will matter of factly state that they only drink three or four glasses of wine or five or six bottles of beer a day, with the implication that this is minimal and not problematic. The National Institutes of Health regularly updates its recommendations as to what a healthy amount of daily alcohol use is. For adults, both men and women, the current recommendation is one alcoholic beverage a day (one beer, one glass of wine, or one shot of hard alcohol).

If we ingest a substance that has a perceivable change in how our brain functions, that substance is changing the physiology of our brain. This topic is far too important and extensive to adequately address in this editorial, and so I will further explore the recreational use of drugs in the future.

Learn new information everyday

To date we have no adequate treatment for most of the common types of dementia. The annual international conferences on dementia often conclude with: the best treatment for dementia is prevention. The etiology of dementias is complex, with a range of genetic, medical, substance induced, brain trauma, lifestyle, and environmental risk factors. What we can say is that individuals with more education appear to have a greater brain “reserve” of healthy neurons, and that this is related to a later onset of dementia.

Learning new information in areas that were previously not known seems to facilitate synaptogenesis, which helps promote improved brain functioning and cognitive reserve. Simply put, keep a list of topics that you would like to learn about, and keep learning: art history, chemistry, astronomy, Russian literature, a new language, cooking, woodworking, statistics, particle physics... anything new will facilitate brain health.

Movement activities using all body parts

Our brains have two hemispheres, with complex connectivity that can send neurons from one side down the spinal cord to the same side of the body, or to the opposite side. Each of us is born with one hemisphere that is more dominant regarding language and handedness, although there is significant plasticity in this regard, especially early in life. Reflect on your first time learning a new activity—playing a guitar, driving a car, learning a new language. Initially it takes a great deal of attention, intention, repetition, and practice to learn any new activity. Eventually it becomes second nature.

The more your brain is challenged with new tasks, and the more complex these tasks are, the healthier your brain will be. Movements in dance and yoga often involve bilateral muscle groups throughout the body—these two activities have been shown to improve brain health. Someone once told me to get on all fours, and crawl like a baby backwards—this is something that most of us have never done, and initially it is challenging. However, as you crawl backwards new neurons are firing, and the brain is connecting in a new way with symmetrical circuits.

Comply with evidence-based treatments for health-related conditions

For any health-related condition that you have, after you feel confident that your health care providers have adequately and professionally diagnosed your condition, and after you have been informed of all treatment options available, do your best to comply with the treatment you choose. This last recommendation is a big one, and one that will be further explored in a future editorial.

Conclusion

Our brains are complex organs that are constantly rewiring in response to our behaviors, environment, stress, anti-stress, nutritional intake, sleep efficiency, social connectedness, medical conditions, intake of substances of all sorts, and numerous factors out of our control. We have learned a great deal to map out paths to support our brain health, much like we have previously achieved for heart health. Each moment of each day we make choices that can affect our brain health—our challenge is, without judgment, to increase the choices that promote a healthy brain.

References
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The most common movements associated with tardive dyskinesia (TD) are in the oral regions and include puckering, chewing, grimacing, and tongue protrusions. It is also common to see choreiform movements of the extremities, such as “piano playing” movements of the fingers.

Although other medications may be associated with TD, it is most often noted in patients treated with either typical or atypical antipsychotics, especially at higher doses. TD can be caused by additional medications that are dopamine D2 receptor antagonists, such as metoclopramide used in the treatment of gastrointestinal disorders. A list of medications with reports of associated TD and medications that can worsen TD are described in Table 1.

Pathophysiology

Although TD was identified over 50 years ago, its pathophysiology remains poorly elucidated and is likely multifactorial and complex. The most prominent theory involves postsynaptic dopamine D2 receptor upregulation with associated dopamine receptor supersensitivity due to chronic exposure to dopamine D2 receptor antagonists. This theory is supported by:

ACTIVITY GOAL

The goal of this activity is to provide a comprehensive understanding for the identification, assessment, and clinical management of tardive dyskinesia.

LEARNING OBJECTIVES

At the end of this CE activity, participants should be able to:

• Discuss what is known about the pathology of tardive dyskinesia as well as the risk factors for the disorder
• Assess a patient with movement disorder and make a diagnosis
• Identify FDA-approved pharmacotherapies for tardive dyskinesia

TARGET AUDIENCE

This continuing medical education activity is intended for psychiatrists, psychologists, primary care physicians, physician assistants, nurse practitioners, and other health care professionals who seek to improve their care for patients with mental health disorders.

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Melissa Palmer, PharmD, has no disclosures to report.

Austin R. Campbell, PharmD, reports that he is on the Speakers Bureau of Sunovion Pharmaceuticals.

Amber Finegan, PharmD, has no disclosures to report.

Leigh Anne Nelson, PharmD, reports that she has received research support from Neurocrine Biosciences and Teva Neuroscience; her husband is a shareholder in Neurocrine Biosciences.

Vivek Datta, MD (peer/content reviewer), has no disclosures to report.

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Worsening of TD symptoms when the offending medication antagonizing dopamine receptors is discontinued
Temporary improvement of symptoms seen upon increasing the dose of the offending medication

This theory can be challenged by the fact that TD may be irreversible in some cases when the offending medication is discontinued, even though downregulation of dopamine receptors would be expected. Neurotransmitter dysregulation and abnormalities in the striatal gamma-aminobutyric acid (GABA) neurons responsible for synaptic plasticity resulting in GABAergic hypofunction and degeneration of striatal cholinergic interneurons have been theorized as causes of TD. Glutamate and opioid neurotransmitter dysregulation has also been implicated. Another popular theory is the “neurodegenerative hypothesis” that suggests that medications that antagonize the dopamine D2 receptor increase lipid peroxidation and free radical formation leading to neuronal degeneration, damage, and structural changes in the brain. There is conflicting evidence for all of these theories, and the pathophysiology of TD remains poorly understood.

The risk for TD is significantly lower with atypical antipsychotics; the lowest risk is for patients who have never been treated with typical antipsychotics. A list of well-established risk factors for TD can be found in Table 2.

Diagnosis
When a patient presents with involuntary, repetitive movements, differential diagnoses must be considered including tics, drug-induced Parkinsonism, tardive syndromes, and other neurological causes. Table 3 describes the diagnostic criteria for TD based on DSM-5 and the Schooler-Kane Research Criteria.1 Although TD is irreversible in many cases, the manner and acuity of the movements can fluctuate over time.

Strategies for prevention
A number of preventive measures have been suggested to reduce TD risk. Patient and/or caregiver risk-prevention education is imperative before initiation of antipsychotic therapy. To minimize risk it is recommended to adhere to antipsychotic prescribing information regarding appropriate indications and dosing; avoid off-label use if possible. If dyskinesias are noted, consider an atypical antipsychotic with a lower risk of TD. Alternatively, a dose reduction of the antipsychotic can be considered.

In general, use the lowest effective dose for the shortest possible duration, particularly in the elderly. Keep in mind ongoing use with other medications that are dopamine D2 receptor antagonists (eg, metoclopramide, prochlorperazine). Use of these medications in combination with antipsychotics should be avoided. Regularly assess all patients chronically taking medications that antagonize the dopamine D2 receptor for any abnormal movements with the Abnormal Involuntary Movement Scale (AIMS). Table 4 summarizes TD prevention strategies.

Monitoring/assessment tools
The American Psychiatric Association (APA) recommends that all patients treated with an antipsychotic be regularly monitored for abnormal movements.1 Assess patients treated with a typical antipsychotic every 6 months; assess patients treated with atypical antipsychotics every 12 months. Consider more frequent monitoring for patients with TD or those who have TD risk factors (eg, every 3 months).

The AIMS was developed by the National Institute of Mental Health. It is widely used in clinical practice because it can be administered by any qualified health care professional—the examination and scoring procedures are explained in detail, and it takes approximately 10 minutes to administer. The 12-item assessment includes 10 items ranked on a 5-point scale from 0 to 4 that increase in severity (none, minimal, mild, moderate, severe). Various areas of the body are examined including a global assessment of severity and patient awareness of any abnormal movements. The last two items address any potential problems a patient currently has with his or her teeth and/or dentures that could affect assessment results.

Early recognition of abnormal movements is imperative to management of TD symptoms. Clinical utility of a movement disorder scale such as the AIMS can lead to timely identification and thus, improved outcomes.

Treatment strategies
Traditionally, the following are first steps in managing newly diagnosed TD:

1. Gradually taper the dose of the offending agent to reduce the risk of antipsychotic withdrawal dyskinesias.
2. Transition the patient from typical to atypical antipsychotic (other than clozapine), when applicable.
3. Discontinue anticholinergic medications.

In instances where an atypical antipsychotic is the causative agent, consider transitioning to a different atypical antipsychotic with lower risk for TD such as clozapine, olanzapine, or quetiapine. In addition to these standard approaches, there exists a myriad of additional treatment options with a variety of proposed mechanisms and varying levels of evidence. Treatment options considered to have at least a moderate level of evidence for use are reviewed in Table 5; see Table 6 for a comparison of FDA-approved treatments for TD.

Ginkgo biloba
The benefits of ginkgo biloba in the treatment of TD are attributed to its antioxidant effects and possible free-radical scavenging. In an effort to compare ginkgo biloba extract with placebo, Zheng and colleagues1 undertook a meta-analysis of three 12-week randomized controlled trials in 299 Chinese patients with schizophrenia. Patients taking ginkgo biloba experienced a 2.3 point reduction on AIMS total score; the discontinuation rates were similar to those for placebo. While it appears somewhat effective and well tolerated, more studies are needed in non-Chinese populations before the results can be extrapolated to the general population.

Amantadine
Amantadine is an anti-Parkinson disease medication that is a noncompetitive N-methyl-D-aspartate (NMDA) receptor antagonist and dopamine agonist that is FDA approved for the treatment of drug-induced extrapyramidal symptoms. While its use in managing Parkinson disease-related dyskinesias has been established, evidence for use in TD is much more limited.5

In a randomized double-blind controlled trial, 22 patients were treated with up to 400-mg amantadine or placebo for 2 weeks.6 The amantadine group experienced a 3 point (22%) reduction on AIMS total score. These results duplicate the find-
ings of an earlier study by Angus and colleagues, whose findings showed a 15% mean reduction in AIMS scores. Despite these results, the use of amantadine remains somewhat contentious because of the overall paucity of evidence specific to TD and the potential to worsen psychosis.

**Clonazepam**

Like all benzodiazepines, clonazepam potentiates the inhibitory effects of GABA, which may be of value in some patients with TD. Several small studies have shown short-term benefit of clonazepam in reducing dyskinetic movements associated with TD. However, due to the limitations of these trials, the development of tolerance in some study subjects, as well as general concerns for adverse effects, abuse, and dependence, clonazepam is not a first-line treatment for TD.

**Tetrabenazine**

Tetrabenazine, the first vesicular monoamine transporter 2 (VMAT2) inhibitor, is FDA approved for the treatment of chorea in Huntington disease. The drug’s benefits in TD are attributed to its reduction of synaptic monoamines, particularly dopamine, reducing neuronal stimulation and hyperactivity. A more recent review of the evidence for tetrabenazine in TD yielded a series of four open-label studies, two small randomized controlled trials, and two retrospective reviews. The results of these trials demonstrated a consistent improvement in TD symptoms in 41% to 100% of participants—the majority of studies showed moderate or better improvement. While these results are encouraging, tetrabenazine has several limitations associated with its use. The medication’s short half-life necessitates 2 to 3 times daily dosing, while adverse effects such as somnolence, parkinsonism, and akathisia limit dose titration and tolerability. The medication also carries a boxed warning for depression and suicide risk.

**Deutetrabenazine**

While similar to its parent compound, deutetrabenazine incorporates deuterium onto the primary site of drug metabolism. This minor structural change yields an increased half-life and lower Cmax, translating to less frequent dosing and improved tolerability. Like tetrabenazine, deutetrabenazine carries an indication for treatment of chorea associated with Huntington disease; however, in August 2017 it became the second drug to receive FDA approval for treatment of TD. The evidence behind this approval comes primarily from four randomized double-blind placebo-controlled trials and one long-term open-label extension study. While smaller-dose finding studies did not separate from placebo, two larger 6-week trials demonstrated significant improvements in AIMS total scores.

The second study included 298 patients randomized to receive placebo or deutetrabenazine 12 mg, 24 mg, or 36 mg daily in divided doses. Patients who received the 24 mg and 36 mg daily doses experienced significantly greater reductions in AIMS scores; one-third of these patients had a 50% or greater reduction of symptoms. Furthermore, rates of discontinuation and psychiatric adverse effects were low.

**Valbenazine**

In April 2017, valbenazine became the first FDA-approved treatment for TD. Another VMAT2 inhibitor, valbenazine and its two active metabolites are all highly selective reversible binders. The safety and efficacy of valbenazine was determined from four randomized double-blind placebo-controlled trials and one long-term open-label extension study. While smaller-dose finding studies did not separate from placebo, two larger 6-week trials demonstrated significant improvements in AIMS total scores.

The first study randomized 89 patients with moderate to severe TD to placebo or 25-mg valbenazine daily, which could be increased by 25 mg daily every 2 weeks up to 75 mg daily. The least-squares mean change in AIMS scores from baseline was -2.6 for valbenazine and -0.2 for placebo, with roughly one-half of patients experiencing a 50% or greater reduction in AIMS total scores. Of note, 76% of patients reached 75 mg daily; there were no discontinuations due to adverse events.

The second study was a randomized, double-blind, fixed dose study that compared placebo with either 40-mg or 80-mg valbenazine in 234 patients. At the end of 6 weeks both valbenazine treatment groups experienced significant reductions in AIMS scores. The least-squares mean changes from baseline were -3.2 for valbenazine 80 mg, -1.9 for valbenazine 40 mg, and -0.1 for placebo. A greater than 50% reduction in AIMS scores was observed in 40% and 23.8% of patients taking valbenazine 80 mg and 40 mg, respectively, but only 8.7% of those who received placebo.

In a 52-week open-label extension study, continued improvements were observed for 48 weeks, which worsened and trended back to baseline when valbenazine was discontinued.

**Conclusion**

TD remains a clinically important problem associated with antipsychotics and other medications that antagonize the dopamine D2 receptor. Regular screening with a standardized tool such as the AIMS is of utmost importance for early detection of TD. Until recently, treatment of TD was challenging because there were no FDA-approved treatments. Two newer medications with a novel mechanism of action, deutetrabenazine and valbenazine, were approved by the FDA for the treat-

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**Table 3. Schooler-Kane and DSM-5 criteria for diagnosis of tardive dyskinesia**

**Schooler-Kane**

- Abnormal involuntary movements of either moderate severity in one or more body regions; mild severity in two or more body regions
- Movements assessed using a standardized tool such as the AIMS or ESRS
- History of at least 3 months’ cumulative neuroleptic exposure
- Absence of other conditions that might produce abnormal involuntary movements

**DSM-5**

- Involuntary athetoid or choreiform movements (lasting at least a few weeks) generally of the tongue, lower face and jaw, and extremities (sometimes involving the pharyngeal, diaphragmatic, or trunk muscles)
- Develop in association with the use of neuroleptic medication for at least a few months
- Symptoms may develop after a shorter period of medication use in older persons
- Neuroleptic withdrawal-emergent dyskinesia (dyskinesia occurring after discontinuation or dosage adjustment of a neuroleptic that is time limited, lasting less than 4-8 weeks) needs to be ruled out; dyskinesia that persists beyond this window is considered to be tardive dyskinesia

**Table 4. Tardive dyskinesia prevention and harm reduction strategies**

- Avoid off-label use of antipsychotics
- Perform regular screening assessment such as the AIMS
- Educate patient and/or caregiver(s)
- Use lowest effective dose for shortest duration possible
- Dose reduction of offending medication
- Switch to a SGA

Table 3 includes AIMS, Abnormal Involuntary Movement Scale; ESRS, Extrapyramidal Symptoms Rating Scale.

Table 4 includes AIMS, Abnormal Involuntary Movement Scale; SGA, second generation antipsychotic.

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The second study included 298 patients randomized to receive placebo or deutetrabenazine 12 mg, 24 mg, or 36 mg daily in divided doses. Patients who received the 24 mg and 36 mg daily doses experienced significantly greater reductions in AIMS scores; one-third of these patients had a 50% or greater reduction of symptoms. Furthermore, rates of discontinuation and psychiatric adverse effects were low.

Valbenazine

In April 2017, valbenazine became the first FDA-approved treatment for TD. Another VMAT2 inhibitor, valbenazine and its two active metabolites are all highly selective reversible binders. The safety and efficacy of valbenazine was determined from four randomized double-blind placebo-controlled trials and one long-term open-label extension study. While smaller-dose finding studies did not separate from placebo, two larger 6-week trials demonstrated significant improvements in AIMS total scores.

The first study randomized 89 patients with moderate to severe TD to placebo or 25-mg valbenazine daily, which could be increased by 25 mg daily every 2 weeks up to 75 mg daily. The least-squares mean change in AIMS scores from baseline was -2.6 for valbenazine and -0.2 for placebo, with roughly one-half of patients experiencing a 50% or greater reduction in AIMS total scores. Of note, 76% of patients reached 75 mg daily; there were no discontinuations due to adverse events.

The second study was a randomized, double-blind, fixed dose study that compared placebo with either 40-mg or 80-mg valbenazine in 234 patients. At the end of 6 weeks both valbenazine treatment groups experienced significant reductions in AIMS scores. The least-squares mean changes from baseline were -3.2 for valbenazine 80 mg, -1.9 for valbenazine 40 mg, and -0.1 for placebo. A greater than 50% reduction in AIMS scores was observed in 40% and 23.8% of patients taking valbenazine 80 mg and 40 mg, respectively, but only 8.7% of those who received placebo.

In a 52-week open-label extension study, continued improvements were observed for 48 weeks, which worsened and trended back to baseline when valbenazine was discontinued.

**Conclusion**

TD remains a clinically important problem associated with antipsychotics and other medications that antagonize the dopamine D2 receptor. Regular screening with a standardized tool such as the AIMS is of utmost importance for early detection of TD. Until recently, treatment of TD was challenging because there were no FDA-approved treatments. Two newer medications with a novel mechanism of action, deutetrabenazine and valbenazine, were approved by the FDA for the treat-
References


Table 5. Evidence-based treatments for tardive dyskinesia

<table>
<thead>
<tr>
<th>Agent</th>
<th>MOA</th>
<th>Evidence in TD</th>
<th>N</th>
<th>Recommendations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Valbenazine</td>
<td>VMAT2 inhibitor</td>
<td>Multiple larger RCTs (level 1)</td>
<td>323</td>
<td>Grade A (FDA approved)</td>
</tr>
<tr>
<td>Deutetrabenazine</td>
<td>VMAT2 inhibitor</td>
<td>Multiple larger RCTs (level 1)</td>
<td>415</td>
<td>Grade A (FDA approved)</td>
</tr>
<tr>
<td>Tetrabenazine</td>
<td>VMAT2 inhibitor</td>
<td>Small RCTs, open-label studies, retrospective review (level 3)</td>
<td>250</td>
<td>Grade C</td>
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<tr>
<td>Ginkgo biloba</td>
<td>Antioxidant</td>
<td>Multiple small RCTs, and meta-analysis (level 1)</td>
<td>299</td>
<td>Grade B (population unknown)</td>
</tr>
<tr>
<td>Amantadine</td>
<td>NMDA antagonist/dopamine agonist</td>
<td>Small RCTs (level 2 and 3)</td>
<td>38</td>
<td>Grade C</td>
</tr>
<tr>
<td>Clonazepam</td>
<td>GABA potentiation</td>
<td>Small RCTs (level 1 and 2)</td>
<td>40</td>
<td>Grade B (short-term trial)</td>
</tr>
</tbody>
</table>

Table 6. Comparison of FDA-approved treatments for tardive dyskinesia

**Deutetrabenazine**

- Starting dose and titration: 6 mg twice daily and titrate weekly by 6 mg daily
- Maximum recommended dose: 24 mg twice daily
- Dietary administration recommendation: Take with food
- Half-life (hours): 9-10
- Primary metabolism: CYP2D6
- Hepatic impairment: Contraindicated
- Renal impairment: Has not been studied
- Other considerations: Warning for depression and suicide

**Valbenazine**

- Starting dose and titration: 40 mg once daily for 1 week then increase to 80 mg
- Maximum recommended dose: 80 mg once daily
- Dietary administration recommendation: May be taken with or without food
- Half-life (hours): 15-22
- Primary metabolism: CYP3A4
- Hepatic impairment: Child-Pugh class B or C: 40 mg daily
- Renal impairment: Not recommended for CrCl < 30 mL/min
- Other considerations: No box warning

CrCl, creatinine clearance.

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Department of Psychiatry

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Hackensack Meridian Health is a leading not-for-profit health care network in New Jersey offering a complete range of medical services, innovative research, and life enhancing care aiming to serve as a national model for changing and simplifying health care delivery through partnerships with innovative companies and focusing on quality and safety.

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- **Outpatient Child & Adolescent Psychiatrist:** Jersey Shore University Medical Center (Neptune, NJ) and Hackensack University Medical Center (Hackensack, NJ)
- **Medical Director/Section Chief, Child & Adolescent Psychiatry:** Jersey Shore University Medical Center (Neptune, NJ)
- **Outpatient General Psychiatrist:** Jersey Shore University Medical Center (Neptune, NJ), Riverview Medical Center (Red Bank, NJ), and Raritan Bay Medical Center (Perth Amboy, NJ)
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San Joaquin County Behavioral Health Services is seeking to fill Outpatient Adult [General], and Sub-Specialty Psychiatry (Child Psychiatry, Geriatric, Forensic, Addiction and Psychosomatic Medicine) positions in a multidisciplinary, recovery-oriented clinical setting. Services are provided either on-site or using a hybrid model of on-site and tele-psychiatry practice. The positions offer a very competitive salary with a guaranteed base, plus incentive opportunities, board certified Psychiatrists have the potential to fully earn over 300K+ a year; comprehensive health insurance; up to three retirement and pension programs; 35 days of vacation and CME time that increase with tenure. Signing and moving bonuses are also available. Interested J-1 and H-1B candidates are welcome to apply.

Contact Khurram Durani, MD at: kdurani@sjcbhs.org,
Fax CV to 209-468-2399, EOE.

PSYCHIATRIST POSITION

J-1 Visa Opportunity in California
Imperial County Behavioral Health Services is currently recruiting for a full time psychiatrist. Imperial County is located 90 miles by freeway to the city of San Diego to the west, and 90 miles to Palm Springs to the north. Located in a rich farming area, Imperial County has a population of 180,000 and borders with Yuma, Arizona and with the Mexican border. Imperial County Behavioral Health Services is located in El Centro, CA and our state capital of Baja California Norte. Imperial County has a population of 180,000 (2016) and public universities located in Mexicali, Calexico and there are a number of private and public universities located in Mexicali, the state capital of Baja California Norte. Imperial County’s location and diversity make it the perfect place for a psychiatrist to relocate under the J-1 Visa program or for any reason.

The position pays a highly competitive salary, including health benefits for you and your family, and requires no hospital work and minimal after hours work freeing you up for more leisurely activities.

The successful candidate diagnoses and treats patients with mental, emotional, and behavioral disorders. Qualified candidate must have CA medical license or ability to obtain.

Send CV to Imperial County Behavioral Health Services, 202 North 8th Street, El Centro, CA 92243.

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Interested applicants should submit a letter of interest and curriculum vitae addressed to:
Alan P. Brown, MD
Vice Chairman of UMMS Department of Psychiatry for BH Integration and Population Health
Clinical Professor of Psychiatry, Family Medicine and Community Health

Jessica Saintelus, Physician Recruiter
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