Cannabidiol as an Adjunctive Treatment for Schizophrenia

Anahita Bassir Nia, MD

Cannabis is a complex plant with more than 100 types of cannabinoids. Its main psychoactive compound is δ-9-tetrahydrocannabinol (THC), which activates cannabinoid receptors to produce its “feeling high” effects. Cannabidiol (CBD) is another cannabinoid that has attracted growing attention recently. Unlike THC, CBD does not bind to cannabinoid receptors and has shown different, sometimes counteractive, effects. Currently, there are more than 100 clinical trials registered on the ClinicalTrials.gov website on the potential therapeutic effects of CBD.

The FDA has recently approved the first cannabis plant-derived medication, Epidiolex (an oral solution of pure CBD), for treatment of seizures associated with Lennox-Gastaut syndrome and Dravet syndrome in patients aged 2 years and older.1 Consequently, DEA scheduled Epidiolex in Schedule V of the Controlled Substances Act (CSA), the least restrictive schedule.2 Though Epidiolex is only approved for the above rare seizure disorders, physicians may recommend it off-label for other conditions, based on their own judgment. It is important to note, however, that the only approved form of CBD is Epidiolex and off-label recommendation of other forms of CBD does not follow the same rules.

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Reefer Madness
Continued from cover

Berenson claims that cannabis use leads to psychosis and violence. He states his thesis just a few pages into the book, claiming that “whether marijuana is dangerous to the brain and can ultimately cause violence is a scientific question, with a hard yes or no answer. We have that answer.”

This absurd promise is the fatal flaw of Berenson’s book. To date, research has not demonstrated a simple connection—let alone a causal connection—between cannabis and violence.

A disclosure: We are physicians who support the legalization and effective regulation of cannabis, based on principles of public health and social justice. We strive to follow the science, even when the science contradicts our conclusion that the harms of cannabis prohibition are far worse than the harms of cannabis use.

In that spirit, we acknowledge one key point of agreement with Berenson: Research does show that cannabis can trigger or worsen psychosis in predisposed individuals. Indeed, we have spent years educating the public about this risk.

But most of Berenson’s assertions are unsupported by science. For example, a recent study showed a decrease in domestic violence among cannabis-using couples. This doesn’t prove that cannabis reduces violence, but it certainly suggests that any relationship between cannabis and violence is complicated and influenced by a host of factors. Berenson conveniently dismisses studies like this that are in conflict with his narrative.

Reaction to the book has been swift. Dr Ziva Cooper, a co-author of the 2017 National Academy of Medicine report on cannabis upon which Berenson relies, tweeted: “[W]e did NOT conclude that cannabis causes schizophrenia.” RAND Drug Policy Research Center co-director Dr Beau Kilmer was unequivocal in a recent tweet about RAND’s interpretation of the literature from 2001 to 2011: “Marijuana use does not induce violent crime.”

Perhaps the greatest tragedy of Tell Your Children has nothing to do with cannabis. Berenson’s insidious association of psychosis with violence unfairly stereotypes people living with psychosis, very few of whom are ever violent, evoking the bias that has long plagued this vulnerable population.

Lest we be misunderstood, we’re not claiming cannabis use is risk-free, but we believe the book is a distraction from a serious discussion of the other risks of cannabis use. Studies show that you shouldn’t drive a car while under the influence, that underage recreational use is harmful, and that some people use cannabis problematically. However, cannabis use causes no lasting harm to most healthy, non-pregnant adults.

Berenson discusses cannabis as if it were uniquely dangerous. Many foods, drugs, and activities like motorcycle riding carry a risk of injury or death to people who indulge in them. As Alcohol Prohibition taught us, the government must not lightly wield the blunt instrument of criminal justice to stop consenting adults from engaging in risky behaviors.

Prohibition has not solved any problems of cannabis use, despite more arrests annually for cannabis possession than for all violent crimes combined. In fact, it is a failure of historic proportions, documented in the government’s own statistics.

Here is where we get into why we believe cannabis prohibition creates more harm than cannabis use: Black people are almost four times more likely to be arrested for cannabis possession than whites despite the fact that black people and white people use cannabis at roughly the same rate. This racial disparity worsens the economic plight of already struggling communities of color. The resulting poverty limits access to education, jobs, rehabilitation, and health care.

That is only one of the ways cannabis prohibition undermines public health. With underage use increasing from the 1960s until legalization began, prohibition has failed in its most basic intent, which was to prevent access to minors.

But far from the apocalyptic predictions of opponents of legalization, underage cannabis use has not increased in states that have legalized cannabis. We are not surprised. Regulation prevents youth access at licensed retailers—who check for ID—and creates a legal distinction between underage and adult use that minors can understand and respect.

We appeal to government officials, the public, and our fellow physicians to support the sensible regulated adult use of cannabis as a more humane and cost-effective alternative to prohibition. Our nation must embrace science, reject misguided moralism around cannabis use, and recognize that books like Berenson’s are exactly what he inadvertently entitled his own: Reefer Madness.

Former Surgeon General Joycelyn Elders is an honorary board member of Doctors for Cannabis Regulation (DFCR.org), which is the first and only national physicians’ association dedicated to the legalization and effective regulation of cannabis in the United States. Dr David L. Nathan and Dr Bryon Adinoff are board members of DFCR.

The authors report no conflicts of interest concerning the subject matter of this article.

Cannabidiol
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The evidence for cannabidiol
The association between cannabis use and psychosis is well-known in epidemiological studies, and a dose-response relationship is consistently reported with an odds ratios of 3.90 (95% CI, 2.84 to 5.34) for the risk of schizophrenia in heavy cannabis users.1 However, use of cannabis strains with high CBD content has been associated with fewer psychotic symptoms.2 Whereas THC produces acute psychotic-like symptoms in healthy volunteers, pre-treatment with CBD decreases the THC-induced psychotic symptoms and cognitive impairments.3-7

The potential beneficial effects of CBD on cognition in patients with schizophrenia have critical importance, since cognitive deficits are common in schizophrenia (up to 75%-85% of patients), usually precede other symp-

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TABLE. Studies on the clinical administration of CBD to treat psychosis in individuals with schizophrenia or non-affective psychosis

<table>
<thead>
<tr>
<th>Study</th>
<th>N</th>
<th>Dose of CBD</th>
<th>Assessments of symptoms</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zuardi et al.8</td>
<td>1</td>
<td>Up to 1500 mg/d in 2 divided doses for 26 days</td>
<td>BPRS</td>
<td>Improvement of symptoms, no adverse effects</td>
</tr>
<tr>
<td>Zuardi et al.9</td>
<td>3</td>
<td>Up to 1280 mg/d for 4 weeks</td>
<td>BPRS</td>
<td>Mild improvement only in one subject, no adverse effects</td>
</tr>
<tr>
<td>Zuardi et al.10</td>
<td>6</td>
<td>Up to 600 mg/d for 4 weeks</td>
<td>BPRS, PPQ, CGI</td>
<td>Improvement of symptoms, no adverse effects</td>
</tr>
<tr>
<td>Leweke et al.11</td>
<td>42</td>
<td>Up to 800 mg/d for 4 weeks in 3-4 divided doses</td>
<td>BPRS, PANSS</td>
<td>CBD was as effective as amisulpride for improvement of symptoms; CBD had superior adverse effect profile</td>
</tr>
<tr>
<td>McGuire et al.12</td>
<td>88</td>
<td>1000 mg/d in 2 divided doses for 6 weeks</td>
<td>PANSS, BACS, GAF, CGI-I, CGI-S</td>
<td>CBD group had lower levels of positive psychotic symptoms and subjects were more likely to have been rated as improved and as not severely unwell by their clinician; they also showed greater improvements in their cognitive processing speed and in overall functioning; the rate of adverse events was not significantly different with placebo</td>
</tr>
<tr>
<td>Boggs et al.13</td>
<td>36</td>
<td>600 mg/day in 2 divided doses for 6 weeks</td>
<td>PANSS, MCCB</td>
<td>There was no significant effect of CBD on interaction in either cognitive performances or psychotic symptoms; the CBD group showed greater sedation compared with placebo</td>
</tr>
</tbody>
</table>

BPRS, Brief Psychiatric Rating Scale; PPQ, Parkinson Psychosis Questionnaire; PANSS, Positive and Negative Syndrome Scale; BACS, Brief Assessment of Cognition in Schizophrenia; GAF, Global Assessment of Functioning scale; CGI-I and CGI-S, improvement and severity scales of the Clinical Global Impressions Scale; MCCB, MATRICSConsensus Cognitive Battery.
toms, and respond minimally to the available pharmacological treatments.

The very first case report on the use of CBD as an antipsychotic medication was published by Zuardi and colleagues in 2006 (Table). In this study, a 19-year-old female patient with schizophrenia was treated with CBD up to 1500 mg daily for 4 weeks, which resulted in improvement of acute psychotic symptoms. Findings from a study in 2006 that looked at the effects of CBD as monotherapy for treatment-resistant schizophrenia in three individuals show that improvement was seen in only one patient (Table). A later study on the antipsychotic effects of CBD (at flexible doses up to 400 mg/d) on 6 individuals who had Parkinson disease showed improvement of psychotic symptoms over the course of 4 weeks.

Since then, the antipsychotic properties of CBD have been investigated in three clinical trials with mixed results (Table). In 2012, Leweke and colleagues published the first double-blind randomized controlled trial on the therapeutic effects of CBD (600-800 mg/d) compared with amisulpride on acute psychosis in individuals with schizophrenia (N = 42). The study concluded that CBD is as effective as amisulpride in treating psychotic symptoms and has fewer adverse effects, including less extra pyramidal symptoms and weight gain.

More recently, the effects of CBD on psychosis were explored in two double-blind randomized placebo-controlled clinical trials. McGuire and colleagues used CBD as an adjunctive medication in the treatment of acute psychosis in individuals who had schizophrenia or other non-affective psychotic disorders. Participants (N = 88) received either CBD 1000 mg daily (in two divided doses) or placebo in addition to their routine antipsychotic medications (continued unchanged during the study) for 6 weeks.

Compared with the placebo group, the CBD group showed greater improvement of positive psychotic symptoms over the course of the treatment. Mean improvement of PANSS positive score was 3.2 (SD 2.60) in the CBD group compared with 1.7 (SD 2.76) in the placebo group. Moreover, by the end of the treatment, more patients in the CBD group were rated as “improved” on the CGI-I scale compared with those in the placebo group (78.6% and 54.6%, respectively). Patients who received CBD also showed a trend-level improvement in their cognitive functioning, and a significant improvement of their motor speed compared with controls.

In a similar study, Boggs and colleagues investigated the therapeutic effects of adjunctive CBD 600 mg daily (in two divided doses) compared with placebo in a 6-week double-blind placebo-controlled randomized clinical trial, in individuals with chronic schizophrenia (N = 36). However, their results showed no significant differences between CBD and placebo on psychotic symptoms or cognitive performances.

Mechanism of action
The exact mechanism of action is still unknown for CBD’s potential anti-psychotic properties. Unlike other antipsychotic medications, CBD does not greatly affect dopaminergic neurons, and unlike THC, it does not bind to cannabinoid receptors. However, CBD reportedly increases the CSF levels of anandamide in the brain, and it does not bind to cannabinoid receptors. This may suggest that anandamide levels are negatively correlated with severity of psychotic symptoms, whereas increased anandamide levels in psychotic patients treated with CBD are correlated with clinical improvement. This may suggest that CBD contributes to amelioration of psychosis by increasing the synthesis or reducing the degradation of anandamide.
endogenous levels of anandamide. However, further studies are needed to confirm this.

The current pharmacological treatment for schizophrenia is only partially effective and mainly for positive symptoms. This has led investigators to investigate new pharmacological targets and the endocannabinoid system has been one of the newest ones. Over the past few decades, increasing evidence has shown the presence of endocannabinoid system abnormalities in schizophrenia. However, the current studies on the potential therapeutic effects of CBD are not conclusive and the mechanism of action is poorly understood. The discrepancies in clinical results could be related to different doses of CBD, stages of psychosis, or possibly heterogeneity of schizophrenia itself.

Conclusion
In addition to the potential therapeutic effects of CBD for schizophrenia, CBD may also have a role in preventing or treating the psychosis related to recreational use of cannabis in vulnerable individuals. Cannabis continues to be the most commonly used illicit drug in the US, and with the spreading legalization for medical and recreational purposes, a lower proportion of people perceive the risk associated with regular cannabis use. At the same time, there is a decreasing ratio of CBD-to-THC in street cannabis from 1:14 in 1995 to 1:80 in 2014. Low CBD content may affect the overall impact of frequent cannabis use on mental health, which may become evident in the future. When discussing the medicinal use of cannabis, it is important to distinguish CBD, with its potential beneficial effects, from THC, with its controversial adverse effects, especially on individuals with psychiatric disorders.

In addition to the potential therapeutic effects of CBD for schizophrenia, CBD may also have a role in preventing or treating the psychosis related to recreational use of cannabis in vulnerable individuals.

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Dr Bassir Nia reports that she has no conflicts of interest concerning the subject matter of this article.

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Is It Time to Legalize Cannabis?

John J. Miller, MD | Editor in Chief

The controversial question of whether to legalize cannabis has emerged front and center as an important issue that deserves a serious and thoughtful dialogue. Cannabis is the general term for substances derived from the plant Cannabis sativa, with varying psychoactive and pharmacological properties that are found in 3 organic products: marijuana, hemp, and cannabinoids. With each passing year more states legalize cannabis—currently 10 states—and a much larger number of states pass legislation to decriminalize cannabis, make it legal for medical use, or both.

This creates increasing confusion across the country, as the legal ramifications of possessing and using cannabis in each state becomes increasingly complex. Thoughtful arguments can be made supporting or decrying the legalization of cannabis based on an array of issues: medical risks and benefits, psychiatric risks and benefits, the possibility of easier access by vulnerable populations, legal consequences, strong-held cultural beliefs, political ideologies, potential for tax revenue, and the fostering of outdated myths about cannabis.

In this month’s issue, Former Surgeon General Joycelyn Elders and colleagues make yet another compelling argument for the legalization of cannabis. Having cannabis classified as a Schedule 1 drug (classifying cannabis as illegal, with no clinical benefits) has stymied research into this complex portfolio of over 100 cannabinoid molecules—the 2 most common being delta-9-tetrahydrocannabinol (THC) and cannabidiol (CBD). Since cannabis is illegal under federal law, researchers face significant restrictions and regulation. As a result, research has been slow, and the endogenous presence of a cannabinoid receptor in the human brain was not confirmed until 1990.

FDA approval of cannabidiol

Significantly, in June 2018 the FDA approved one of the primary components of cannabis, CBD (brand name Epidiolex), for the treatment of seizures in Dravet syndrome and Lennox-Gastaut syndrome in children and adults. As a result of this FDA approval, the Drug Enforcement Administration reclassified CBD from a Schedule 1 drug (illegal), to a Schedule 5 “controlled substance” (in the same class as pregabalin).

A rapidly growing body of double-blind placebo-controlled studies are demonstrating clinically effective properties of CBD. It is not euphorogenic and demonstrates clinical effects that are opposite to THC. CBD has demonstrated anti-inflammatory and antioxidant effects, and findings suggest benefits of its treatment for seizures, psychosis, anxiety, multiple sclerosis, and movement disorders.

The extensive amount of information now available on cannabis is exemplified by the 2017 publication The Health Effects of Cannabis and Cannabinoids: The Current State of Evidence and Recommendations for Research.1 All relevant publications on cannabis were reviewed for this document and the document nicely describes the degree of evidence supporting the risks and benefits of cannabis use. The following possible or established benefits are listed: anti-emetic effect in chemotherapy-induced nausea/vomiting; increased appetite and decreased weight loss in individuals with HIV/AIDS; improved clinician-measured and patient-reported spasticity in multiple sclerosis; improved short-term sleep in individuals with a documented sleep disturbance; improved symptoms in some anxiety
disorders; decrease in some types of chronic pain in adults; and improved symptoms in Tourette syndrome.

Neurobiology of cannabis

After discovering the cannabinoid receptor in 1990, a plethora of basic science studies has taught us a great deal about the neuropharmacology of the two most active ingredients in cannabis—THC and CBD—which are pharmacologically complementary. In the early 1960s, when cannabis was widely used by our country’s youth, the leaves from the marijuana plant were derived from the original naturally occurring plants that had been harvested since approximately 3000 BC—Cannabis sativa.

In the original cannabis plant, the THC content was roughly 2%, with relatively equal amounts of CBD.

Once THC was identified as the cannabinoid associated with the “high,” marijuana plant breeders genetically enriched subsequent generations of plants to have increased THC—up to 30%—and decreased CBD. The most potent form of THC is called “wax,” which is 50% pure THC. A 2016 article documented that the ratio of THC to CBD of illegally sold cannabis in the US increased from 14:1 to 80:1 between 1995 and 2014.

Following the discovery of the first endogenous cannabinoid receptor in 1990, a second receptor was found; aptly named cannabinoid 1 receptor (CB1R) and cannabinoid 2 receptor (CB2R). CB1R is the primary receptor in the mammalian brain and is present in high concentrations in the hippocampus, hypothalamus, amygdala, basal ganglia, substantia nigra, and cerebellum. There are some CB2Rs in the brain as well, although their primary locations include the gastrointestinal system, the spleen, and immune cells such as the macrophages.

Two endogenous cannabinoids bind to these CB receptors, anandamide (AEA) and 2-arachidonoylglycerol (2-AG). Interestingly, unlike most neurotransmitters in the brain, AEA and 2-AG are synthesized post-synaptically “on demand” and diffuse upstream, across the synapse, and bind to presynaptic CB1 and CB2 receptors. This ultimately results in inhibition of the presynaptic release of glutamate, GABA, dopamine, norepinephrine, serotonin, and acetylcholine. (For more details on this pathway see Atakan, and Maccarrone et al.)

Benefits of legalization

One of the most compelling arguments for the legalization of cannabis is the ability to regulate its production, composition, and distribution. Illegally obtained cannabis has unpredictable content: concentrations of THC and CBD can vary widely; and there is no quality control to inform the user of the potency and ratios of THC to CBD. When purchased illegally, the “cannabis” may actually contain “cannabinimetic agents.” Two well-known examples are K-2 and Spice, which are sprayed onto dry plant matter. These synthetic chemicals mimic THC but bind much more potently to CB1R and can cause serious adverse effects, including psychosis.

Cannabis sold at state certified dispensaries is quantitatively assayed for contents, including defined percentages of CBD versus THC. This allows for a high degree of predictability of the cannabis product being purchased, and the possibility should not be used until the brain is fully developed, somewhere between the ages of 21 and 25. Moreover, based on multiple, large epide-miological studies, we have learned that 10% of THC users will become addicted, with a well-defined withdrawal syndrome upon acute discontinuation.

With our current understanding of the neurobiology of the endocannabinoid system, it is not surprising that constant agonism at the CB1R during brain development affects brain function in significant ways. The brain structures that are rich in CB1R, and the impact of CB1R agonism on neurotransmitters are essential for optimal neuronal circuitry function.

THC appears to accelerate the onset of a first psychotic episode by 2 to 3 years in individuals who are at risk. In addition, an exposure dependent effect has also been demonstrated—frequent cannabis use and more potent THC levels increase the risk of psychosis. Finally, ongoing cannabis usage after a first psychotic episode is correlated with an increased risk of relapse, as well as a higher severity of positive symptoms and a greater decline in overall functioning. Abstinence reduces the relapse risk.

A recent study by McGuire and colleagues demonstrated that individuals with schizophrenia who were maintained on their baseline antipsychotic medication while augmenting with 1000-mg CBD daily experienced improvements. After 6 weeks of augmentation, the CBD group demonstrated significant improvement over the placebo group on measures of positive psychotic symptoms, the clinicians’ impressions of improvement and illness severity, as well as a non-statistically significant improvement in cognitive and overall functioning.

There is a solid body of prospective studies that report a significant irreversible decline in cognitive functioning in adolescents who regularly use cannabis. In his editorial, Harvey Nicely summarizes our current understanding of the effect of cannabis on cognition. He references a seminal paper by Meier and colleagues who followed 1037 individuals in Dunedin, New Zealand born in 1972 or 1973. This cohort was evaluated every 2 years from birth up to age 38, with 95% retention. Cannabis use was monitored, and IQ testing was performed at ages 8, 11, 13, and 38. Individuals with persistent cannabis use that began during the adolescent years lost an average of 8 IQ points. In contrast, individuals who began using cannabis as adults had no decline in their IQ score. These data support the likelihood of a neurotoxic effect with the regular use of cannabis in the developing brain, resulting in an enduring decline in cognitive function.

Conclusion

So, the debate goes on about whether to legalize cannabis nationally. Both sides of the debate can present arguments to support their position. As a father, psychiatrist, and scientist, in my opinion it is time to re-schedule cannabis as a legal, but controlled substance. This would create increased safety and predictability in the production, distribution, and individual use of cannabis. And, the legal status of cannabis would be nationally cohesive. Research on the basic science of cannabinoids, as well as novel clinical applications would increase, and funding would be more available for public education about the evidence-based facts of the risks and benefits of cannabis use.

References

Meet & greet.

Come meet the editor-in-chief of Psychiatric Times at the APA Annual Meeting! John J. Miller, MD will be available on Sunday, May 19, 2019, from 11 AM to 1 PM in our booth, #1424.
Neurobiology and Clinical Management of Childhood Onset Schizophrenia

Niitin Gogtay, MD

Although psychotic illnesses are fortunately rare in children, contrary to common belief, psychotic symptoms can be fairly common in very young healthy children. As a symptomatic child grows older, these experiences tend to become much less frequent but, if symptoms persist, they can be indicators of increased risk for more serious mental illness and poorer outcomes later in life.

Given the reasonably common occurrence of hallucinations in otherwise healthy children it is critically important that the symptoms be carefully characterized and a diagnosis such as schizophrenia be made with extreme caution in children. For example, an “imaginary friend,” or hallucinations while transitioning either into asleep (hypnogogic) or wakefulness (hypnopompic) are common and should not be considered problematic in healthy children. The important challenge comes when hallucinations or other psychotic experiences are more pervasive, serious, and pose a diagnostic and treatment challenge.

History of childhood onset schizophrenia

Although clinicians were already aware by the early 20th century that schizophrenia could manifest very early in childhood, the diagnostic criteria were unclear. This was probably because the term “psychosis” was often loosely used in children to describe a range of symptoms from behavioral disturbances to those on the autism spectrum; a problem that has continued to date. To complicate things further, psychotic disorders in children often have a complex and severe presentation that makes it harder to tease apart the origins of symptoms.

In 1990, the National Institute of Mental Health (NIMH), launched a study on childhood onset schizophrenia (COS). This study, the only one of its kind in the world, was prospective and had a comprehensive design that encompassed clinical diagnosis, risk factors, neuropsychological and brain development, and genetic underpinnings. The effort continued for 25 years and generated the largest cohort of well-characterized children with COS in the world. The insights gained were critical both for the understanding of childhood psychotic illnesses as well as for schizophrenia in general and comprise most of the literature in the neurobiology and clinical course of COS.

Diagnosis and differential diagnoses

For COS diagnosis, the NIMH COS study required the onset of psychosis to be before age 13, premorbid IQ of 70 or above, and absence of a neurological disorder. The screening process was extremely thorough: it included inpatient observation with a complete medication washout in most cases. A startling diagnostic observation was that almost 30% of children who met the screening criteria for suspicion of COS (and who were treated as such) had other diagnoses (such as anxiety disorders, mood disorders) after careful observation and medication washout. The diagnosis remained stable at long-term follow up, which highlights the importance of spending the time and effort for diagnostic characterization.

Clinically, COS resembles severe, chronic, poor outcome adult onset schizophrenia (AOS) cases. However, the psychosis of COS is usually severe, pervasive, and remarkably “non-episodic and unremitting” from the initial presentation. A detailed history usually reveals poorer premorbid functioning in social, motor, and language domains, learning disabilities, and disruptive behavior disorders, which suggests widespread brain dysfunction from the get-go. Commonly associated are transient autistic symptoms such as hand flapping and echolalia and upon careful evaluation almost 29% show comorbid autism spectrum disorder.13 Careful evaluation of differential diagnosis is critical because, as learned from the imaging analyses from the NIMH COS study, the underlying brain pathology is highly diagnostically specific. Several childhood conditions can manifest with psychotic symptoms and/or deterioration in function. Pediatric mood disorders, such as pediatric bipolar disorder or MDD can present with psychotic symptoms, although they tend to be mood congruent unlike those in COS where the psychosis is much more pervasive.

Autism spectrum disorders and childhood disintegrative disorder have severe impairment in reciprocal communication, social interactions, and odd stereotyped behaviors and thus can be mistaken for the severe psychosis of COS. Behavioral disturbances such as conduct disorder can be associated with hallucinations and psychosis. They may also be an outward manifestation of medical conditions such as diseases due to metabolic errors; neurological illnesses, such as epilepsy; or substance use disorders. A carefully collected patient history and good medical and laboratory work up as well as observations in a neutral setting, and preferably without medications, can usually distinguish these other conditions from the pervasive psychosis of COS.

A group of unique patients who often pose a common diagnostic dilemma is worth special mention. In the NIMH COS study, a subgroup of patients had real but transient psychotic symptoms. The main presentation for these children was severe, daily periods of emotional/mood lability disproportionate to the precipitating triggers. Characteristically these children had difficulty forming relationships and multiple developmental deficits (eg, ADHD, learning disabilities, auditory processing abnormalities).

A careful interview revealed lack of formal thought disorder and the psychotic symptoms were transient at best. For lack of anything better, the DSM diagnosis was multidimensionally impaired (MDI). The MDI cohort, not uncommon and often mislabeled, appeared to have a distinct long-term clinical course, with none progressing to schizophrenia.14

While considering the differential diagnosis for schizophrenia in a child with psychosis, it is important to recognize that COS could co-exist with many comorbid psychiatric disorders. Because symptom manifestations can also be part of (or masked by) the symptoms of schizophrenia, a diagnosis of an independent Axis I condition can often be ignored. The most frequent comorbid diagnoses in the NIMH COS cohort were depression (54%), followed by OCD (21%), generalized anxiety disorder (15%), and ADHD (15%). The rate of any one anxiety disorder (generalized anxiety disorder, OCD, separation anxiety, PTSD, panic disorder combined) at screening was 42%. Furthermore, comorbid diagnoses (particularly depression) correlated with poorer overall functioning and continued comorbidity, which suggests either a refractory nature of these conditions, or a close association with core schizophrenia pathology. Importantly, there were no significant associations between comorbid diagnoses and IQ, familiarity, medication status, premorbid functioning, or age of psychosis onset.

Neurobiological underpinnings

The NIMH COS study spent significant effort in establishing the neurobiological continuity of COS and...
more typical AOS. All published neurobiological risk factors were examined carefully in patients with COS. As expected, COS showed a similar yet exaggerated pattern of risk factors that may have reflected greater impairment in early brain development, but also supported the continuity between COS and AOS. These observations included higher rates of early developmental abnormalities (language, social, and motor domains), larger effect sizes for smooth pursuit eye movement abnormalities, and higher incidence of familial schizophrenia spectrum disorders.

Neuropsychological function in COS warrants special mention as it is commonly evaluated in clinical practice. This has been well studied in the NIMH COS cohort as well as by some other groups studying COS or adolescent onset schizophrenia. Analyses on the NIMH COS cohort showed a clear decline in IQ post-onset of psychosis, but more importantly, this drop in cognitive function did not continue even after 8 years. The incidence of neuropsychological deficits for COS were not significantly higher than for AOS per se, but healthy siblings of children with COS also shared some neuropsychological deficits, suggesting a more salient genetic/familial link in COS.

Prospective brain imaging studies in COS patients and their siblings have contributed to the understanding of abnormal brain development in COS and have also provided critical insights into brain development in schizophrenia in general.2,4,5 Most of these studies have originated from the NIMH COS cohort.

In addition to replicating the brain abnormalities seen in AOS (eg, large ventricular volumes, smaller gray matter volumes), the finer brain mapping studies in COS showed that there is profound gray matter loss in COS during adolescence that mimics the normal cortical gray matter maturation pattern, which suggests that the healthy pruning process in normal maturation may be exaggerated in COS. This gray matter loss, however, stabilizes over time and the pattern merges with that seen for AOS, thus establishing biological continuity between the two.

Young healthy COS siblings share this gray matter deficit pattern during their younger years, but the deficits normalize by late adolescence, which suggests that the gray matter loss in COS is an age-dependent trait marker. More importantly, the profound gray matter abnormalities are diagnostically specific, unrelated to medication exposure, and appear proportional to the degree or severity of psychosis. The abnormalities are not limited either to cortex or only to gray matter as white matter and deeper cortical structures also share the deficits and the deficit pattern. Connectivity analyses using structural, functional, and diffusion tensor imaging, show specific neurocircuitry abnormalities in circuits relevant to psychosis.1

As was expected, COS shows more salient genetic underpinnings. The patients in the NIMH COS study had a significantly higher incidence of velo-cardio-facial syndrome (with spontaneous 22q11.2 deletion) than AOS patients or than any clinical population. Patients with COS also had a higher incidence of cytogenic abnormalities with two agents. The only two randomised controlled trials (RCTs) established superiority of typical antipsychotics over placebo in COS, and a single trial demonstrated superiority of clozapine over haloperidol. Subsequently, results from a relatively small double-blind RCT showed that clozapine was better than olanzapine in alleviating negative symptoms. These data and the observations made during the NIMH study have led to the understanding that clozapine is the best option for this extremely ill cohort.

Unfortunately, as anticipated, clozapine is associated with more adverse effects, including risk of agranulocytosis, significant weight gain, akathisia, enuresis, tachycardia, orthostatic hypotension, and seizures. Managing the adverse effects when treating with clozapine is a major challenge where the observations from the NIMH COS study provide critical insights.

The most significant adverse effect of clozapine is neutropenia that can lead to agranulocytosis. The FDA requires weekly blood monitoring with neutropenia, often resulting in discontinuation of clozapine with potentially disastrous clinical worsening. This can be successfully managed by the addition of lithium.

Weight gain is pronounced with clozapine particularly during childhood. Along with significant psychoducation for life-style changes, the antidiabetic medication metformin (which improves peripheral insulin sensitivity) showed some success when started with clozapine. Akathisia, seen rarely in adults taking clozapine, is surprisingly common in children and can manifest as worsening of psychotic symptoms or agitation in children. This often results in dose increments that further worsen the akathisia. Akathisia is responsive to adjunctive beta blocker (eg, propranolol) treatment.

Cardiac adverse effects such as orthostatic hypotension and tachycardia need regular monitoring and can be managed by adjusting the dose, fluid intake, activity, or medications such as beta blockers. Incontinence is not uncommon with clozapine but although it is responsive to vasopressin, it also tends to be better with longer treatment. Drooling also gets better with time, but children need to be taught to manage it daily.

Finally, clozapine can lower the seizure threshold and can induce seizures. Regular EEG monitoring and prophylactic antiepileptic medication such as gabapentin has helped manage this adverse effect. Metabolic changes from chronic clozapine use (such as hyperlipidemia, hyperglycemia) should be monitored regularly so that appropriate treatment can be initiated. Clozapine may be the best medication available to treat COS symptoms and improve functionality, but careful monitoring and managing adverse effects is a challenge.

Dr Gogtay is Director, Office of Clinical Research, National Institute of Mental Health, Rockville, MD.

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The psychosis of COS is usually severe, pervasive, and remarkably “non-episodic and unremitting” from the initial presentation.
Recognizing and Treating Trauma in the Inpatient Psychiatric Setting

Rachel B. Nowlin, MS and Sarah K. Brown, DrPH

Compared with the general population, patients with serious mental illness are more likely to:

- Experience a traumatic event and have a higher number of overall trauma exposures
- Meet criteria for a diagnosis of PTSD during their lifetime and in the previous 12 months
- Report greater impairment in general functioning due to trauma
- Have increased use of health care services related to trauma

Previous research suggests that comorbid mental illness and post-traumatic stress interact to produce worse outcomes in patients, making trauma an important facet of a patient’s overall psychiatric care. However, diagnosis rates of PTSD in psychiatric inpatient treatment settings are consistently lower than expected given the rate of trauma exposure. It seems inpatient psychiatric treatment may focus more on primary psychiatric symptoms (eg, psychosis) that brought the patient in, while identifying and treating PTSD may be secondary.

Identifying trauma symptoms and psychological distress

Inpatient psychiatric outcomes data provide a unique opportunity to examine the presence of PTSD symptoms and diagnoses for patients with mental illness and how PTSD symptoms are related to overall psychiatric symptoms and functioning.

Data were analyzed for 4126 patient records with an Abbreviated PTSD Checklist (PCL-C-6) completed at admission to an inpatient psychiatric treatment program. The average admission severity for patients exceeded the suggested cutoff, indicating the likelihood of PTSD (patient average was 17.5 compared to cutoff of 14). A subset of these patients (29.6%) also had admission scores on the Behavior and Symptom Identification Scale (BASIS-32), a measure of general functioning useful for monitoring behavioral health outcomes. Patients who scored at or above the cutoff on the PTSD screener had significantly higher levels of dysfunction on the BASIS-32 compared with patients who scored low (below the cutoff) on the screener.

Does routine trauma screening identify trauma in inpatient units?

Although the majority of patients (65.7%) had high scores on the PCL-C-6, only 7.6% of these patients also had a primary or secondary PTSD diagnosis. Those who were diagnosed may have just been the most severe: patients with PTSD diagnoses had higher PCL-C-6 scores and showed greater distress overall on the BASIS-32 compared with patients with high PCL-C-6 scores but no PTSD diagnosis. These findings suggest PTSD may be underdiagnosed in these populations, especially when symptoms of serious mental illness are at the forefront.

Clinical lessons

Our data support the presence and role of trauma in this population, and we see evidence of the need for trauma-informed care. Regardless of diagnosis, patients with serious mental illness and post-traumatic stress symptoms would likely experience benefit in both areas from trauma-informed care.

References
Heartbeat

SERIES EDITOR,  
H. Steven Moffic, MD

Anandhi Narasimhan, MD

Medical school was rough. From a young age I always knew I would enjoy working more than I would enjoy school, and that has turned out to be true. The confines of exams and deadlines were stifling. After an anxious, anticipated beginning in residency training, I got used to the scheduling demands. Eventually, I would finish residency and fellowship and start employment as a child psychiatrist.

For many people in medicine, the academic and professional journey advances in a cookie-cutter fashion—you know what is next and what comes after that. I thought life would unfold for me in a similar fashion. You may struggle, deal with rejection, but eventually the fairytale ensues. I did not realize how tumultuous the journey would be for me.

I wanted to get married to the man of my dreams before my dad passed away. That didn’t happen. The former US Surgeon General Dr Vivek Murthy said that loneliness is America’s biggest health problem. For all intents and purposes, I had made it professionally, but it was the loneliness that made living in a big city daunting, despite being surrounded by millions of people. I made friends, but a partnership seemed elusive.

Recently, I got married—15 years later than I had envisioned. Being fiercely independent for so many years, I no longer feel lonely. The juxtaposition of loneliness with independence as a single person sometimes can feel both confusing and crushing.

Many professionals envision some version of personal goals that include financial stability, work-life balance, and family. As I started writing this, I entered my tenth week of pregnancy. At my first prenatal appointment, we found out the fetus was 2 weeks behind in size and the heart rate was low. My husband and I were told to wait things out to see if the fetus would develop, but I knew I would not be confident about this pregnancy. After she left the room, we both cried.

As a child psychiatrist I have borne witness to the ramifications of abuse, neglect, and genetic/environmental influences on children. I was under no illusion of the inherent fallacies of bringing a child into this world. Most of us in this field would state emphatically that some people are not capable of parenting. Whether it is addiction, mental health issues, or an emotional disconnection from their offspring, it is a simple fact that not all children are brought into this world wanted and loved.

An older mother, I would like my child to have plenty of autonomy (with appropriate supervision). External pressures and expectations can kill a child’s curiosity. I have always felt that a child must develop a strong will. Still, you can do everything right as a parent, but your child may still deal with suicidal thoughts or develop a substance abuse problem. Your child may lose motivation to do something purposeful. I see all of this in my career. So, I decided to approach this pregnancy with objectivity.

At 41, I knew the rates of miscarriage were higher, so although I was not surprised when the obstetrician expressed concern about whether this would be a viable pregnancy. However, objectivity went out the window when I saw the heartbeat. I felt a deep innate desire to protect this little life that was struggling to grow. Until then I had been telling myself if this baby has a will to live, it has to do so of its own accord. I have experienced grief before, and I have shed tears over losses. Losses are an inevitable part of life and having a partner does soften the blow.

What is the answer in our culture isolated environment? Meetup groups, rock climbing clubs, or poker tournaments? Reading, writing, meditating, and self-care? Networking events can be exhausting and seldom result in more than small talk. Maybe balance comes with trying to connect, even when your heart is not in it. We may look for that connection our whole life, whether with a family member, a partner, or a friend, only to have it taken away at the end. There is simple beauty in appreciating what is given to us.

I am grateful for this pregnancy and the glimpse of a life beginning inside of me.

For centuries, women before me have endured slavery, oppression, and abuses while going through pregnancies and miscarriages. Brave women have fought for the survival and evolution of the human race. When I think about other minority women and the hardships they have faced, it makes my personal struggle seem more manageable.

We know that other mammals such as elephants grieve in an expressive fashion when they lose their young. Is there anything that can be learned from this?

We know that other mammals such as elephants grieve in an expressive fashion when they lose their young. Is there anything that can be learned from this? It is okay to be interdependent as a species. I used to fault myself for not being stronger emotionally when I was alone and for not being more positive and present in the moment. For not being able to fix things. For a psychiatrist, that lesson poses a tremendous conflict of interest.

On December 27, I learned that this pregnancy will not come to fruition with a baby for me. The healing process begins for me as it has for countless women before me.

Dr Narasimhan is in private practice in Los Angeles, CA. □
In 1994 I wrote an article for *Psychiatric Times*, “Traumatic Brain Injury: Its Psychiatric Manifestations and Management.” A quarter of a century ago, few clinicians, and certainly the public, had little concern or understanding of the consequences of traumatic brain injury (TBI). That article provided the perspective that psychiatrists have an important role in the treatment of patients who have had a TBI. Since that time, it is now recognized that disorders such as anxiety, depression, and posttraumatic stress disorder are significant contributors to increased symptoms and disability. This Special Report addresses several specific areas of concern that are of importance to psychiatrists: Can depression be prevented after TBI? What are the risks of suicide? Are there special issues in the elderly (the most rapidly growing population who experiences TBI)? What are the guidelines for the use of medications? What do we do about the post-TBI psychosis in patients?

With the increased interest in mild TBI (of which concussion is the mildest severity), practitioners are seeing many more individuals who have experienced an episode. To determine the natural history and consequences of TBI we need long-term prospective studies. Several of these are currently underway and comprise athletes, individuals who present to emergency departments, and the military. Findings from these studies will be invaluable in understanding selection bias that all studies (and individual practices) have.

The most important development in the treatment of individuals with TBI is the recognition that symptoms that persist are non-specific, and patients require a targeted and focused physical examination to determine etiology and treatment. We cannot adequately assess and treat our patients by relying solely on the use of rating scales and cognitive tests. This includes evaluation of visual problems (including symptom exacerbation with eye movements and assessment of convergence insufficiency); balance problems; careful assessment of neck issues, including manual examination for trigger points; and assessment of exercise tolerance with tests such as the Buffalo Concussion Treadmill Test (BCTT). It is also apparent that exercise may be an effective treatment, and rest (except for decreasing the risk of another TBI) is detrimental. Many patients may have depression or anxiety because no one has ever conducted a proper physical examination. Having persistent symptoms that have been “unexplained” has emotional consequences.

So, I invite you to explore the topics discussed in this Special Report, and the others mentioned in this introduction. For those interested, these topics (and many others) are discussed in detail in the *Textbook of Traumatic Brain Injury*.

Dr Silver is Clinical Professor of Psychiatry, New York University School of Medicine, New York, NY.

Dr Silver reports that as senior editor he receives royalties for the 3rd edition of *Textbook of Traumatic Brain Injury*.

**References**

Pharmacological Management of the Psychiatric Aspects of Traumatic Brain Injury

> Yani Rao, MD, and Sandeep Vaishnavi, MD, PhD

Traumatic brain injury (TBI) is a public health epidemic. Approximately 2.8 million people sustain a TBI annually; of these, approximately 50,000 die, 282,000 are hospitalized, and 2.5 million are discharged from an emergency department.1 Mild TBI or concussion accounts for the majority of TBIs. Falls are the most common cause of TBI, followed by assault and motor vehicle accidents. According to recent statistics, during a 6-year period from 2007 to 2013, rates of TBI-related ER visits increased by 47%, but rates of hospitalization and death decreased by 2.5% and 5% respectively, underscoring the importance of managing TBI morbidity.1

Psychiatric disturbances are the most common long-term sequelae of TBI. In a recent article on the epidemiology and natural history of psychiatric disorders after TBI, Ponsford and colleagues2 noted that compared with the general population, patients with TBI have increased incidence of depressive disorder, anxiety disorder, and PTSD; depressive disorders are likely to be chronic and persistent.

In this article, we define TBI psychiatric disturbances as the development of psychiatric symptoms (emotional, behavioral, or cognitive) after the occurrence of a single or multiple TBIs. It may not always be possible to establish a causative link between TBI and the onset of psychiatric problems. When not possible, TBI should be considered as a treatment-informing medical comorbidity rather than an etiologic factor.

Appropriate management of psychiatric disturbances (of TBI) can result in better outcomes, improved quality of life, and decreased societal impact.2

When examining psychiatric symptoms after TBI, it is important to consider a differential diagnosis, including other diagnostic categories that present with symptoms that may be confused with psychiatric disturbances after TBI.3

Emotional problems

TBI-associated depression. TBI-associated depression is characterized by prolonged, persistent sadness associated with other symptoms such as anhedonia, lack of motivation, decreased self-care, variable sleep and/ or appetite pattern, feelings of hopelessness, and/or suicidal thoughts. These symptoms may last for a couple of weeks to months (major depressive episode) or persist in a milder form for two or more years (dysthymia).

SSRIs are often considered first-line agents because of their benign side effect profile. Sertraline, citalopram, and escitalopram are often favored because of their limited drug-drug interaction. Most clinical trials have focused on sertraline, but results have been inconsistent. In a trial comprising 15 patients with mild TBI 87% had a significant response and 67% achieved remission with sertraline.4 In a follow-up study of patients with TBI-associated depression no significant differences were seen between sertraline and placebo in depression severity, response, or remission rates.4 Similarly, Ashman and colleagues5 found no statistically significant difference between sertraline and placebo in a group of 52 patients with TBI-associated depression. However, the small sample size (N=11) and the high rate of discontinuations may have contributed to the lack of difference between the medication and placebo groups.

SNRIs have also been used with fair success and no significant adverse effects, but they have not been systematically studied. In a small study (N=10) the tricyclic antidepressant (TCA) desipramine was shown to reduce depressive symptoms.6 However, because of anticholinergic adverse effects and risk for seizures, TCAs are less favored. Nortriptyline in the treatment of TBI depression.

Methylenedate and other stimulants may be used to augment the effect of antidepressants, especially when there is evidence of fatigue, apathy, or executive function deficits. Lee and colleagues7 compared the efficacy of sertraline, methylenedate, and placebo in a study of 30 patients with mild to moderate TBI. Both methylenedate and sertraline significantly improved symptoms of depression, but only methylenedate improved cognitive function. Patients treated with methylenedate also had diminution in daytime sleepiness.

Electroconvulsive therapy (ECT) may be safe and effective in patients with TBI. Jorge and Arciniegas8 have suggested some common-sense approaches to using ECT in patients with TBI: use low energy levels to generate a seizure of about 20 seconds, use pulsatile currents, increase between-treatment time intervals to 2 to 5 days, and keep the number of treatments to a minimum. Non-dominant unilateral ECT has a decreased risk of cognitive adverse effects compared to bilateral ECT and may be preferable in TBI patients.

**TBI-associated suicide. In a review of 48 studies, Simpson and Tate9 concluded that the risk of suicide, suicide attempts, and suicidal ideation is increased in TBI survivors compared with the general population, even after adjusting for psychiatric comorbidities. Mackelprang and colleagues10 also noted that 25% of their sample of 560 patients with all severities of TBI reported suicidal ideation during the first year following a TBI, a number much higher than 3.7% reported for the general population.

In other cases of suicidal ideation, the most important factor in the management of TBI-associated suicide is to maintain safety. Immediate hospitalization should be considered for patients with active suicidal thoughts with intent or plan to die. Management of suicidal thoughts associated with psychiatric disturbances after TBI should focus on the psychiatric disturbances themselves. Extra caution to maximize safety is needed not only in the inpatient setting but also in the outpatient setting.**
ensuring that the patient has consistent outpatient care and a strong supportive network.

Post-TBI mania. The incidence of bipolar spectrum disorders is low in the TBI population, with a range of 2% to 9% in the first year after injury. Findings indicate the estimated lifetime relative risk for bipolar and related disorders to be 1.1, similar to the lifetime risk of bipolar disorders in the general population. TBI mania is characterized by changes in mood, sleep, and activation, which may manifest as irritability, euphoria, insomnia, agitation, aggression, impulsivity, or even violent behavior. There is scant literature on the pharmacological management of TBI mania.

Based on our clinical experience and case reports, we recommend use of quetiapine as a first-line and risperidone as a second-line agent for acute mania; we recommend valproate as first-line and carbamazepine as a second-line agent for maintenance. Although many psychopharmacologists might argue that lithium is the gold standard for the treatment of idiopathic bipolar disorder, we are concerned about the CNS and motor adverse effects in persons with TBI.

TBI-associated anxiety disorders. A wide range of anxiety disorders may occur after TBI including generalized anxiety disorder, agoraphobia, social phobia, panic disorder, and obsessive-compulsive disorder. Results from a meta-analysis of the prevalence of generalized anxiety disorder and self-reported TBI showed that 11% had a diagnosis of GAD, and 37% reported clinically significant levels of anxiety. Commonly used medications for treatment of anxiety include sertraline, escitalopram, citalopram, and venlafaxine at doses similar to the treatment of TBI-associated depression.

PTSD. In a study of 1084 patients with mild TBI, 13% of the patients had PTSD. Higher incidence has been seen in military studies. Carlson and colleagues found that 64% of 836 veterans from Iraq/Afghanistan had PTSD. A recent review confirmed that PTSD occurs after TBI, but is more common after mild TBI. Although rates of TBI vary in the civilian population, they are higher in the military population, and the risk increases with the severity of TBI. The researchers concluded that the risk of PTSD varies according to the context in which the TBI occurred, the psychological trauma experienced at the time of TBI, and pre-TBI history of trauma or PTSD.

Sertraline is the first-line option for treating comorbid PTSD and TBI. Citalopram is a second choice, but caution is recommended above 40 mg daily because of potential cardiac adverse effects. Second-line agents include fluoxetine and mirtazapine, although the latter may be associated with mild anticholinergic adverse effects. SNRI antidepressants can also be used safely. Tricyclic antidepressants are third-line options. Prazosin may be used as adjunctive treatment for persistent nightmares. However, there were no statistically significant differences in sleep quality between prazosin and placebo in a recent 10-week study. Extreme caution should be used with benzodiazepines for treatment of anxiety disorders or PTSD. In general they should not be prescribed for more than 2 weeks because longer use increases risk for dependence. TBI patients may have increased susceptibility to the adverse effects of benzodiazepines, including drowsiness, ataxia, slurred speech, memory impairment, psychomotor impairment, and possibly disinhibition. Moreover, benzodiazepines may aggravate the fear response in persons with PTSD.

TBI-associated psychosis. Psychotic symptoms are not uncommon after TBI. There are predominantly 2 types of TBI-related psychosis: delusional disorders and schizophrenia-like psychosis. As far as we know, there are no clinical medication trials on the management of TBI psychosis. Case reports suggest efficacy for second-generation antipsychotics. Based on our clinical experience, we recommend second-generation antipsychotics (risperidone, 0.25-4 mg; quetiapine, 25-200 mg; lurasidone, 20-80 mg) over first-generation antipsychotics because the latter may in-
Behavioral disturbances

Apathy. Apathy refers to a syndrome of disinterest, disengagement, inertia, lack of motivation, and absence of emotional responsivity. Although there are also emotional and cognitive aspects of apathy, we have chosen to include apathy under behavioral disturbance, as the disengagement and lack of involvement is most distressing for caregivers. Apathy can be a symptom of depression, which is the most common presentation, or occur as an isolated syndrome. As with most psychiatric disturbances in TBI, only reviews, anecdotal reports, and small case series are available to guide management. Most of the literature favors methylphenidate. Based on this and on our clinical experience, we recommend methylphenidate as a first-line agent.

Sleep disturbances. Sleep disturbances are common after TBI and can occur in isolation or as a symptom of a psychiatric disorder. Insomnia is the most common sleep disturbance, seen in about 50% of patients with TBI, although other disturbances such as hypersomnia, sleep apnea, and sleep-walking may also be present. Treatment of TBI sleep disturbance varies according to the type of sleep disorder and related comorbidities. Comprehensive medical evaluation including an overnight sleep study can help with the diagnosis of sleep disturbance. Maintenance of sleep hygiene is always first in the management of sleep disturbances. When sleep disturbance is comorbid with a psychiatric disturbance, the treatment of the psychiatric condition may also help with the sleep disturbance.
disorder, it is important to treat the underlying psychiatric disorder.

When insomnia occurs in isolation, consider use of a non-benzodiazepine hypnotic such as zolpidem for a short period. Other agents such as melatonin, amitriptyline, lorazepam, and zopiclone may also be considered. Benzodiazepines should be avoided secondary to risk for addiction, motor and cognitive adverse effects, and paradoxical rage outbursts. In patients with TBI who have excessive daytime sleepiness, sleep apnea should first be ruled out. Modafinil (100–400 mg) or armodafinil (150–300 mg) can be considered in patients with persistent and unexplainable daytime sleepiness.

Other behavioral disturbances. Other common behavior disturbances include impulsivity, aggression, and disinhibition. As with other psychiatric disturbances, management of post-TBI behavior problems should always be multifaceted. This includes a combination of environmental modification strategies, behavioral therapy, supportive therapy, family therapy, and pharmacotherapy. Medications should be based on presence of comorbid neuropsychiatric disturbances.

Cognitive deficits

The constellation of cognitive impairments following TBI is variable and depends on the severity of the location of the injury on the brain. TBI can affect every cognitive domain, including attention, memory, visual-spatial processing, language, social cognition, and executive functioning.

The core intervention for cognitive impairments is cognitive rehabilitation, which includes use of remediation techniques and external strategies to help individuals compensate for the deficits. In addition to cognitive rehabilitation, there are some pharmacologic options for augmenting cognitive functioning. The three main classes of pharmacological agents utilized to treat the cognitive sequelae of TBI are psychostimulants, cholinergics, and NMDA receptor antagonists.

Psychostimulants. Psychostimulant medications are commonly used to improve arousal/attention and related neurobehavioral symptoms after TBI. Methylphenidate is the first-line agent and most commonly used (5–40 mg/d). Methylphenidate may improve arousal, attention, and processing speed and general cognitive function in the subacute and chronic phase of injury. Lee and colleagues have also noted improvement in cognitive function and reduction in daytime sleepiness in addition to improvement in depression in persons with depression following TBI.

Cholinesterase inhibitors. Donepezil and rivastigmine have been studied for cholinergic augmentation following TBI. Donepezil is a centrally selective acetylcholinesterase inhibitor that has been may improve attention and memory during the subacute and chronic phase of injury and enhance sensory gating during the chronic TBI period. Rivastigmine, which inhibits both acetylcholinesterase and butyrylcholinesterase improved scores on the Hopkins Verbal Learning Test and the Cambridge Neuropsychological Test Automated Battery Rapid Visual Information Processing mean latency, but only in patients with severe memory impairment at baseline.

Another approach is to use cognitive brain training. There are ongoing studies to see if intensive cognitive games can help in TBI. Elbogen and colleagues have studied a mobile cognitive training app (CALM) in TBI; they found that emotional regulation improved; however, executive functioning did not.

Conclusion

Psychiatric symptoms after TBI are common and troublesome. As TBI rates continue to remain high, there are many patients with long-term sequela such as mood, behavioral, or cognitive symptoms.

Treatment of TBI symptoms is in line with treatment of symptoms in idiopathic psychiatric disorders; however, dosing may vary because of the potentially heightened sensitivity of the traumatized brain. There are also some medications used in TBI patients that are not used very often in general psychiatry.

Treatment of TBI is in line with treatment of symptoms in idiopathic psychiatric disorders although some medications, such as amantadine, are not used very often in general psychiatry.
Preventing Suicide When Caring for Patients With a History of TBI

The Current State of the Science and Strategies for Intervention

Lisa A. Brenner, PhD, Riley P. Grassmeyer, MS, and James P. Kelly, MD

The association between traumatic brain injury (TBI) and negative psychiatric outcomes, including suicide, has a relatively long history. However, a focus on TBIs sustained by patients who served in recent conflicts in Iraq and Afghanistan, as well as by those who play sports has resulted in a resurgence of interest in acute and post-acute sequelae. According to the Centers for Disease Control and Prevention, a TBI is a “disruption” in normal brain function secondary to “a bump, blow, or jolt to the head.”

The vast majority of TBIs sustained are mild in nature. That is, the disruption of brain functioning at the time of injury is relatively brief (eg, loss of consciousness less than 30 minutes). The severity of injury (mild [concussion], moderate, severe) is determined based on the disruption of brain functioning at the time of injury and is associated with physical and psychological outcomes. Patients with more significant disruptions of brain functioning at the time of injury (eg, loss of consciousness greater than 24 hours) are at increased risk for long-term functional impairment.

Although psychiatrists are intimately familiar with the terms suicidal ideation, attempt, and suicide, clinicians, researchers, and policy makers have long struggled with the lack of universally agreed upon definitions. In light of this, both the Departments of Veterans Affairs and Defense adopted the Self-Directed Violence Classification System (SD-VCS). Within the system, key terms that are related to the basic clinical phenomena of suicide and suicide-related behaviors are defined. The use of operationalized systems helps to facilitate communication regarding both level of risk and treatment planning.

**Epidemiology**

Seminal epidemiological work in the area of suicide and TBI was conducted by Teasdale and Engberg,2 who used a Danish population register of hospital admissions to determine rates of suicide among those with concussions, cranial fractures, and cerebral contusions/traumatic intracranial hemorrhages. Their findings suggest increased risk among members of all three groups, when compared with members of the general population (3.0, 2.7, 4.1). Of note, the researchers did not adjust for history of psychiatric condition.

A similar study found that history of TBI was associated with an increased risk for death by suicide; however, the hazard ratios identified suggest a more modest relationship (any TBI 1.55, concussion/fracture 1.98, and cerebral contusion/traumatic intracranial hemorrhage 1.34).3 Findings also highlight the key role that co-occurring psychiatric conditions play in increasing risk for suicide among those with a history of TBI.

**TABLE 1. Acute therapeutic risk management**

<table>
<thead>
<tr>
<th>Acute Therapeutic Risk Management</th>
<th>HIGH ACUTE RISK</th>
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<tbody>
<tr>
<td><strong>Essential Features</strong></td>
<td>Suicidal ideation with intent to die by suicide</td>
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<tr>
<td></td>
<td>Inability to maintain safety, independent of external support/help</td>
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<tr>
<td><strong>Common Warning Signs</strong></td>
<td>A plan for suicide</td>
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<tr>
<td></td>
<td>Recent attempt and/or ongoing preparatory behaviors</td>
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<tr>
<td></td>
<td>Acute major mental illness (eg, MDD episode, acute mania, acute psychosis, recent/current drug relapse)</td>
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<tr>
<td></td>
<td>Exacerbation of personality disorder (eg, increased borderline symptomatology)</td>
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<tr>
<td><strong>Common Risk Factors</strong></td>
<td>Access to means</td>
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<tr>
<td></td>
<td>Acute psychosocial stressors (eg, job loss, relationship dissolution, relapse on alcohol)</td>
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<tr>
<td><strong>Action</strong></td>
<td>Typically requires psychiatric hospitalization to maintain safety and aggressively target modifiable factors.</td>
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<tr>
<td></td>
<td>These individuals need to be directly observed until on a secure unit and kept in an environment with limited access to lethal means (eg, kept away from sharps, cords/tubing, toxic substances).</td>
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<td></td>
<td>During hospitalization, co-occurring psychiatric symptoms should also be addressed.</td>
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<tr>
<th>INTERMEDIATE ACUTE RISK</th>
<th><strong>Essential Features</strong></th>
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<tbody>
<tr>
<td><strong>Suicidal ideation to die by suicide</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Ability to maintain safety, independent of external support/help</strong></td>
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<tr>
<td>These individuals may present similarly to those at high acute risk, sharing many of the features. The only difference may be lack of intent, based upon an identified reason for living (eg, children), and ability to abide by safety plan and maintain their own safety. Preparatory behaviors are likely to be absent.</td>
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<tr>
<td><strong>Action</strong></td>
<td></td>
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<tr>
<td>Consider psychiatric hospitalization, if related factors driving risk are responsive to inpatient treatment (eg, acute psychosis).</td>
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</tr>
<tr>
<td>Outpatient management of suicidal thoughts and/or behaviors should be intensive and include:</td>
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<tr>
<td>• Frequent contact</td>
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<tr>
<td>• Regular re-assessment of risk</td>
<td></td>
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<tr>
<td>• A well-articulated safety plan</td>
<td></td>
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<tr>
<td>Mental health treatment should also address co-occurring psychiatric symptoms.</td>
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<tr>
<th>LOW ACUTE RISK</th>
<th><strong>Essential Features</strong></th>
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<tr>
<td><strong>No current suicidal intent AND</strong></td>
<td></td>
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<tr>
<td><strong>No specific and current suicidal plan AND</strong></td>
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<tr>
<td><strong>No preparatory behaviors AND</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Collective high confidence</strong> (eg, patient, care provider, family member) in the ability of the patient to independently maintain safety</td>
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<tr>
<td>Individuals may have suicidal ideation, but it will be with little or no intent or specific current plan. If a plan is present, the plan is general and/vague, and without any associated preparatory behaviors (eg, “I’d shoot myself if things got bad enough, but I don’t have a gun.”). These patients will be capable of engaging appropriate coping strategies, and willing and able to utilize a safety plan in a crisis situation.</td>
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<tr>
<td><strong>Action</strong></td>
<td></td>
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<tr>
<td>Can be managed in primary care.</td>
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<tr>
<td>Outpatient mental health treatment may also be indicated, particularly if suicidal ideation and psychiatric symptoms are co-occurring.</td>
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Source: Rocky Mountain MIRECC.
Using nationwide registries from Denmark, Madsen and colleagues found that persons with TBI were at increased risk for suicide compared with the general population. Having access to pre- and post-injury records of all individuals living in Denmark allowed the researchers to examine several key areas. They found that those with a history of severe TBI had a higher risk for suicide than individuals with a mild TBI. In addition, pre- and post-history of psychiatric illness increased risk for death by suicide after a TBI.

Fralick and colleagues conducted a systematic review and meta-analysis to examine the risk of post-concussion suicide. Their findings suggest that compared with persons with no history of TBI, sustaining a concussion was associated with a two-fold increased risk for suicide. The authors also highlighted challenges associated with quantifying the “typical” time between injury and suicide, as well as identifying specific cohorts, among those with a history of TBI, who may be at heightened risk for death by suicide.

**Long-term negative outcomes**

Acute sequelae are common among patients with a history of all severity levels of TBI. Depending on the severity of injury, mechanisms underlying symptoms are believed to vary and may include inflammation, as well as structural damage to the brain. Most individuals who sustain a mild TBI can expect to return to baseline functioning. However, those with moderate to severe TBIs are likely to have long-term impairment, which often impacts psychosocial functioning.

This simplified explanation of injury severity and sequelae begins to highlight clinical challenges associated with understanding increased risk related to injury history. For example, it may be that an injury (exposure) in persons with mild TBI results in heightened inflammation, and when coupled with other exposures known to be associated with inflammation (eg, poverty, acute stress, cardiovascular disease), a threshold is reached that places individuals at increased risk for death by suicide (see Brundin et al). This is consistent with conclusions asserted by Teasdale and Engberg, who in 2001 wrote “the increased risk of suicide among patients who had a mild traumatic brain injury may result from concomitant risk factors such as psychiatric conditions and psychosocial disadvantage.”

Conceptualizing risk among patients with more severe injuries also requires clinicians to consider multiple factors including neuroanatomic and cognitive dysfunction, and psychosocial impairment. In developing a neurocognitive model underlying suicidal processes for members of the general population, Jollant and colleagues highlighted several key areas of impairment that are frequently observed among those with moderate to severe TBI including impaired decision making and problem-solving abilities.

Moreover, findings indicate that patients with a history of a suicide attempt and moderate to severe TBI are not able to respond to task demands. Based on the results, Brenner and colleagues suggest that during a suicidal crisis this response pattern may result in impulsive decisions aimed at relieving current distress (ie, engaging in suicidal behavior), without considering alternatives (ie, alternate coping strategies).

Work by Simpson and Tate has focused on increased risk for hopelessness among those with moderate to severe TBI. In addition to being a strong predictor of suicide among those without a history of TBI, hopelessness has been widely observed among patients with histories of TBI, with rates of moderate to severe hopelessness as high as 35%. Evaluation of suicide risk among patients with TBI requires the clinician to consider injury severity and the potential impact of multiple exposures as well as psychiatric history and past and present psychological stressors. Wortzel and colleagues advocate for therapeutic risk management, a patient-centered approach that emphasizes augmenting evaluations with structured instruments, stratifying risk in terms of severity (high, intermediate, low) and temporality (acute, chronic) in addition to developing and documenting a safety plan.

### TABLE 2. Chronic therapeutic risk management

<table>
<thead>
<tr>
<th>HIGH CHRONIC RISK</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Essential Features</strong></td>
<td>These individuals are considered to be at chronic risk for becoming acutely suicidal, often in the context of unpredictable situational contingencies (eg, job loss, loss of relationships, and relapse on drugs).</td>
</tr>
<tr>
<td>Common Warning Sign</td>
<td>Routine mental health follow-up</td>
</tr>
<tr>
<td>Chronic suicidal ideation</td>
<td>A well-articulated safety plan, including means safety (eg, no access to guns, limited medication supply)</td>
</tr>
<tr>
<td>Common Risk Factors</td>
<td>Routine suicide risk screening</td>
</tr>
<tr>
<td>Chronic major mental illness and/or personality disorder</td>
<td>Coping skills building</td>
</tr>
<tr>
<td>History of prior suicide attempt(s)</td>
<td>Management of co-occurring psychiatric symptoms</td>
</tr>
<tr>
<td>History of substance abuse/dependence</td>
<td></td>
</tr>
<tr>
<td>Chronic pain</td>
<td></td>
</tr>
<tr>
<td>Chronic medical condition</td>
<td></td>
</tr>
<tr>
<td>Limited coping skills</td>
<td></td>
</tr>
<tr>
<td>Unstable or turbulent psychosocial status (eg, unstable housing, erratic relationships, marginal employment)</td>
<td></td>
</tr>
<tr>
<td>Limited ability to identify reasons for living</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>INTERMEDIATE CHRONIC RISK</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Essential Features</strong></td>
<td>These individuals typically require:</td>
</tr>
<tr>
<td>These individuals may feature chronicity similar to those at high chronic risk with respect to psychiatric, substance abuse, medical, and painful conditions.</td>
<td>Routine mental health care to optimize psychiatric condition and maintain/enhance coping skills and protective factors.</td>
</tr>
<tr>
<td>Protective factors, coping skills, reasons for living, and relative psychosocial stability suggest enhanced ability to endure future crisis without resorting to self-directed violence.</td>
<td>A well-articulated safety plan, including means safety (eg, no access to guns, limited medication supply)</td>
</tr>
<tr>
<td>Stressors historically have typically been endured absent suicidal ideation.</td>
<td>Management of co-occurring psychiatric symptoms</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>LOW CHRONIC RISK</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Essential Features</strong></td>
<td>Appropriate for mental health care on an as-needed basis, some may be managed in primary care settings. Others may require mental health follow-up to continue successful treatments.</td>
</tr>
<tr>
<td>These individuals may range from those with little or no mental health or substance abuse problems, to those with significant mental illness associated with relatively abundant strengths/resources.</td>
<td></td>
</tr>
<tr>
<td>The following factors will generally be missing</td>
<td></td>
</tr>
<tr>
<td>History of self-directed violence</td>
<td></td>
</tr>
<tr>
<td>Chronic suicidal ideation</td>
<td></td>
</tr>
<tr>
<td>Tendency towards being highly impulsive</td>
<td></td>
</tr>
<tr>
<td>Risky behaviors</td>
<td></td>
</tr>
<tr>
<td>Marginal psychosocial functioning</td>
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</table>

Source: Rocky Mountain MIRECC.
Stratification of risk by severity and temporality facilitates clinical decision making (Tables 1 and 2). Safety planning, a collaborative process to identify coping strategies and sources of support for individuals to use during a crisis, should be considered a best-practice for patients at risk for suicide. Findings from Barnes and colleagues13 suggest that use of safety plans is feasible among those with moderate to severe TBIs. Repetition of content during sessions, as well as inclusion of members of the individual’s support system in the process is encouraged (see Department of Veterans Affairs14).

**Intervention strategies**

In addition to a source of support during times of crisis, a safety plan can be used as a starting point for treatment. That is, in completing the initial safety plan, patients and clinicians can identify areas for intervention including drivers of suicidal thoughts (warning signs), as well as internal and external sources of support.

Although drivers of suicidal thoughts should be addressed (eg, depression) using evidence-based strategies (medications, psychotherapy) the clinician and patient should also focus on immediate strategies to reduce imminent risk. For example, lethal means safety counseling is a process by which providers engage patients and their family members in a discussion regarding ways (eg, firearms, medications) that can be used to engage in suicidal self-directed violence, as well as strategies to promote safety, particularly during periods of increased risk. This includes psychosocial education regarding tangible strategies to facilitate safety including firearm locking devices and medication disposal kits (see Department of Veterans Affairs14).

Although an in-depth discussion of psychopharmacologic and psychotherapeutic interventions within this article is not feasible, it is important to note that individuals with moderate to severe TBI can benefit from traditional treatments. See Jorge and Arciniegas15 for information regarding prescribing for more severe injuries. In addition, the results for Window to Hope, an intervention for moderate to severe TBI and hopelessness, have been positive. The findings show that patients with cognitive impairments can benefit from psychological interventions aimed at preventing suicide.16

**Conclusions**

As with most things involving the brain, the relationship between TBI and suicide is complicated. Nonetheless, it is vital that clinicians, particularly, those who are less familiar with working with patients living with TBI, increase their level of comfort in terms of asking about injury history, and incorporating such findings into the conceptualization of suicide risk. Doing so requires neither an overemphasis on TBI history (eg, promoting the connection between chronic traumatic encephalopathy and suicide), nor a lack of appreciation for how a TBI history can influence acute or long-term factors known to increase death by suicide.

**References**


**SPECIAL REPORT**

**TRAUMATIC BRAIN INJURY**

**Aging with a TBI**

Most recent research that examines the effect of TBI on the aging process takes a cross-sectional approach; individuals of different ages are compared rather than following the same individual across time. This lack of longitudinal investigation may be precluding valuable insights into clinical trajectory post-TBI.

In a subset of individuals, it has been found that cognitive and other neuropsychiatric symptoms manifesting acutely after TBI may persist throughout life.1 Conceptually, and making the assumptions that the brain has a finite capacity for recovery and adaptation and that both TBI recovery and aging utilize the same plasticity mechanisms, a brain after TBI may have fewer resources to devote to the aging process.

**Overview of geriatric TBI**

Across all age groups, an estimated 5.3 million Americans are living with a TBI-related disability.1 Many of these individuals will live to be older adults (here defined as ≥65 years of age). From 2009 through 2010, approximately 39 million older adults were evaluated for a new TBI in US emergency departments (EDs). This represents a 61% increase from prior year estimates.7 In addition to rising numbers of ED visits, older adults have rising levels of hospitalizations and death following TBI. In fact, as of 2013, adults 75 years and older sustained the highest number of TBIs—exceeding that of infants.8

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plasticity mechanisms, a brain after TBI may have fewer resources to devote to the aging process. It is important to think of this as an alteration in how the brain handles “normal” aging rather than the onset of a new neurodegenerative process.

When conceptualizing TBI in an aging individual, one should consider how this prior insult may affect the development and progression of neurodegenerative processes. In a recent study of 350,000 military veterans, even mild TBI without loss of consciousness was associated with more than a 2-fold increase in the risk of dementia diagnosis.5

Data from the National Alzheimer Coordinating Center suggest that TBI may be a risk factor for early-onset (<65 years) Alzheimer disease and may be a risk factor for early-onset coordinators suggest that TBI in older adults with more women height, are the leading mechanism of complications, there exists a nuanced and nature of onset and progression. Although not discussed here due to inconsistencies in the literature, a separate neurodegenerative process directly related to repetitive TBI—chronic traumatic encephalopathy—is also likely to exist.

New onset TBI in the aged

At every point along the arc of a newly sustained TBI, from pre-injury risk factors to acute symptoms and future complications, there exists a nuanced set of geriatric-specific factors to consider. Falls, largely from standing height, are the leading mechanism of TBI in older adults with more women affected.4 As even a single fall is a risk factor for subsequent falls, older adults may be at risk for repetitive TBI.

TBI's that would otherwise be classified as mild place older adults at risk for intracranial bleeding due to intracranial changes that occur with aging (eg, dura adherence to the skull, bridging vein fragility, cerebrovascular ath erosclerosis) and the increased use of anticoagulant medication. Following a blunt head trauma, older adults may produce a completely normal neurological examination, yet still have evidence of intracranial trauma on head computerized tomography.6 Older adults are also more likely to have pre-existing medical conditions that are associated with worse post-TBI outcomes.10

The cumulative effect of these factors is that, on average, older adults with TBI experience higher morbidity and mortality, slower recovery trajectories, and worse functional, cognitive, and psychosocial outcomes than younger individuals. Layering the variability of aging onto the already complex TBI equation makes outcome prediction all the more difficult.11

Global outcome measures indicate that older adults have greater functional dependence after TBI.4 Unfortunately, differences in cognitive and other neuropsychiatric symptoms between younger and older adults with new onset TBI are vastly understudied. There appears to be an increased dementia risk when new onset TBI is experienced at an older age compared with younger age, particularly for mild TBI (moderate-to-severe TBI more consistently associated at any age).12

Findings indicate that cognitive and other neuropsychiatric symptoms that manifest acutely after TBI may persist throughout life.

Evidence indicates that when it comes to preexisting psychiatric diagnoses, older adults experiencing TBI are less likely to have a previously diagnosed psychiatric disorder.11 TBI significantly increases the risk of new onset depression, anxiety, and/or PTSD in older adults, with evidence of under-recognition and undertreatment.14 On a more positive note, there appears to be a subset of older adults with TBI who achieve outcomes similar to younger patients, which indicates that chronological age and TBI severity are not the sole determinants of outcome.13

A focused approach to clinical care

In the era of individualized medicine, many fields are utilizing patient characteristics and clinically relevant biomarkers to guide care. The application of individualized medicine to the treatment of TBI is in its infancy; however, some groups are working diligently on its development. Recent advances include:

- Trauma-field triage criteria to optimally identify older adults with TBI who require emergent transfer to a trauma center
- Neurorehabilitation practices specific to older adults with a focus on removing “excess disability”
- Neurocritical care teams involving geriatricians
- Incorporating accreditation standards for geriatric trauma care in future editions of Resources for Optimal Care of the Injured Patient,17 and the Textbook of Traumatic Brain Injury18

This progress should be viewed as a success, although many challenges remain. One such limitation is that the measures used to diagnose TBI and evaluate its outcome were developed in younger cohorts; this makes it difficult to know how applicable these clinical practices are to older adults. As an example, the commonly used Glasgow Coma Scale (GCS) is a reliable predictor of morbidity and mortality in younger, but not older adults who have an abnormal GCS at baseline or an intact GCS despite ac cumulating intracranial hemorrhage.

Prognostic models for outcome prediction after TBI (eg, CRASH CT, IMPACT) show poor performance in older adults. This could be attributed to the models excluding key geriatric outcome predictors such as comorbidities, polypharmacy, baseline function, and frailty. There remains much work to be done to implement current evidence into widespread practice.

A geriatric approach to TBI clinical research

Many clinical studies on TBI implement upper age limits or exclude patients with preexisting conditions. Although this is done in an effort to study true cases of TBI, it excludes older adults and limits the generalizability of a given study. By combining methods commonly used in geriatric research with those already used in TBI studies, the challenges of including older adults in TBI research can be overcome. As an example, in the study of neurodegenerative diseases, a battery of neuroimaging- and blood-based biomarkers are used to supplement clinician evaluations and inform on diagnosis in challenging or ambiguous cases. More research, however, is needed to identify the optimal diagnostic biomarkers in TBI. To address preexisting conditions, geriatric studies often systematically measure and study, rather than exclude, preexisting conditions and disability. In this way, real-world generalizability can be improved.

Another research barrier is that frail older adults may be unable to complete outcome assessments. This has been overcome in other lines of geriatric research through reliance on proxy informants and study partners. Older adults with TBI are likely to have an informant (eg, spouse, caregiver) and a similar approach could be utilized.

Although a relatively new focus in all types of research, innovative follow-up methods, such as home and telemedicine visits, have the potential to increase follow-up rates and generalizability of results. Older adults with TBI may have physical disabilities, perhaps even from the same fall that led to TBI, and this makes involvement in research studies even more difficult. Only by combining methods commonly used in geriatric research with those already used in TBI studies can the research community achieve generalizability to real-world older adults with TBI, develop better diagnostic and prognostic tools to guide care, design inclusive trials, and optimize outcomes.

Conclusion

Working with older adults with TBI is extremely rewarding and is a critical area of study that will become more important as the population ages. That being said, many providers don’t know where to begin or how to unpack the multitude of medical comorbidities existing alongside the TBI. Also, many individuals with new onset TBI will have a history of remote TBI and it can be challenging to ascertain how these histories interrelate. As a result, it is important to distinguish an individual aging with a TBI versus an individual with a new onset TBI later in life. Add to that the variability that occurs with “normal” aging, and the possibility that a neurodegenerative process may develop, and it’s understandable that this population is viewed as daunting by many.

Clinical endeavors, such as comprehensive, multidisciplinary fall and TBI clinics, are invaluable and increasing in number. Geriatric research, particularly on Alzheimer disease and related dementias, has an extensive track record and application of similar techniques to research on older adults with TBI will help move the field forward. (CONTINUED ON PAGE 32)
Depression Following TBI
Can It Be Prevented?

Melissa Jones, MD, and Ricardo E. Jorge, MD

Every year, 1.7 million people in the US sustain a traumatic brain injury (TBI), and nearly 1.1% of Americans live with a disability related to TBI. Psychiatric disorders frequently complicate the course of recovery from TBI and occur at rates exceeding those of the general population. Major depression is the most common psychiatric disorder following TBI, affecting an estimated 29.4% of patients in the first year post-injury alone. TBI-associated depression contributes to higher suicide risk, altered executive function, poorer social reintegration and vocational outcomes, and decreased quality of life.

The chronic and relapsing course of TBI-associated depression poses a challenge to the management of afflicted patients. Two-thirds of patients depressed at 1-year post-injury remain so in the second year, and the risk of depression remains elevated for 20 to 30 years after the injury. In a small trial (N = 21) of citalopram’s efficacy to prevent relapse in patients with remitted TBI-associated depression, over half of the sample relapsed at a mean time of 6 months. The high prevalence, chronicity, and potentially irreversible consequences of post-TBI depression underscore the importance of developing interventions targeting this disorder.

Previous work in the field of TBI-associated depression has focused on treatment strategies. Conflicting results have been seen in randomized controlled trials (RCTs) examining the efficacy of pharmacotherapy for the treatment of TBI-associated depression. Results from a double-blind RCT of patients with TBI-associated depression who received 25 mg to 200 mg sertraline did not show a statistically significant difference in the severity of depressive symptoms compared with placebo after 12 weeks of treatment. RCTs of non-pharmacologic treatments have also yielded inconsistent results regarding their efficacy to treat TBI-associated depression.

In general, preventive strategies are more effective than treatment interventions to decrease the burden of a disease. Our group published preliminary evidence supporting the efficacy of sertraline for the prevention of TBI-associated depression but preventive strategies for TBI-associated depression remain underdeveloped.

Defining prevention
Clinicians may be most familiar with the concepts of primary, secondary, and tertiary prevention. Primary prevention refers to interventions protecting against a disease before its onset, whereas secondary prevention refers to early interventions aimed at preventing disease progression. Tertiary prevention focuses on strategies that reduce the morbidity of a disease after its onset.

Because of the challenges of applying this scheme to the prevention of psychiatric disorders, the Institute of Medicine Committee on the Prevention of Mental Disorders in 1994 recommended a new classification scheme encompassing universal, selective, and indicated preventive strategies. When applied to TBI-associated depression, this scheme emphasizes the prevention of a depressive episode before it begins.

Universal prevention refers to strategies targeting the entire population; in the case of TBI-associated depression, one example would be the enforcement of laws against driving under the influence to reduce the incidence of TBI and hence associated depression. Selective interventions target high-risk groups. Because patients with TBI are at risk for depression, administering an intervention to all patients with a TBI falls under this category. Finally, indicated prevention targets patients with early signs of a disorder, such as those with TBI who have early signs of mood disturbance.

Selective pharmacologic interventions have been shown to prevent depression in patients with other medical conditions conferring high rates of mood disorders. SSRIs have demonstrated efficacy in the prevention of depression following acute stroke and in patients with hepatitis C treated with interferon.

A meta-analysis of 32 randomized controlled trials comprising 6214 participants of diverse age ranges and comorbidities found a 21% decrease in the incidence of major depression for participants exposed to preventive therapies compared with controls. Unfortunately, preventive strategies for TBI are lagging relative to other medical conditions.

Preventive interventions for TBI-associated depression
Few researchers have examined the efficacy of preventive interventions for TBI-associated depression as the primary outcome. Given the tolerability of sertraline in patients with TBI-associated depression, our group conducted a double-blind, placebo-controlled RCT examining the efficacy of medication for the selective prevention of TBI-associated depression.

A unique feature of our study was the administration of a semi-structured interview to actively exclude participants with current depressive, anxiety, or psychotic disorders at the time of enrollment. Also, participants with histories of mood disorders were required to have been in remission for at least one year. Additional exclusion criteria included the use of antidepressants within 6 months prior to the injury; historical failure of an adequate trial of sertraline; or a history of adverse effects from sertraline that required its discontinuation.

Participants were randomized with a permuted blocks randomization scheme to sertraline, titrated over 10 days to 100 mg daily, or to placebo for 24 weeks. Participants were evaluated in-person at baseline, 2-weeks, 4-weeks, and every 4 weeks thereafter. A semi-structured interview was conducted over the phone at 6-, 10-, 14-, 18-, and 22-weeks. If a participant reported an anchor symptom for a depressive disorder in the previous 2 weeks, he or she was evaluated in-person with the same semi-structured interview. Participants ascertained to have a mood disorder by experienced psychiatrists were discontinued from the study and referred for routine psychiatric care.

The final randomized sample consisted of 94 participants, approximately two-thirds of whom had a mild-complicated or moderate TBI. The most common mechanisms of TBI in our sample were falls (48%) and car crashes (38%). Participants in the sertraline (n = 48) and placebo (n = 46) groups were similar in terms of demographics, injury severity, mechanisms of injury, cognitive functioning, disability, exposure to rehabilitation interventions, and measures of anxiety, apathy, and PTSD symptoms.

Over the course of the study, a depressive disorder developed in 3 par-

FIGURE. Sertraline versus Placebo

![Graph showing comparison between Sertraline and Placebo]
Participants (6.3%) in the sertraline group versus 10 participants (21.7%) (Figure). Intent-to-treat analysis revealed that the hazard of developing depression for participants receiving placebo was approximately 4 times that participants receiving sertraline (HR = 3.6, 95% CI, 1.1-16.2). Likelihood Ratio Test [LRT] χ² (1) = 4.6, P = .031. The number needed to treat (NNT) to benefit at 24 weeks was 5.9 (95% CI, 3.1-7.1). The treatment effect remained significant after controlling for Glasgow Coma Scale score, history of alcohol use disorders, and history of mood disorders.

All incident cases of depression had features of major depression. Although concurrent anxiety symptoms were common, these participants did not meet criteria for an anxiety disorder. Only one participant developed suicidal ideation in the placebo group. The odds of dry mouth (odds ratio, 2.2; 90% CI, 1.9-27.6; P = .01) and diarrhea (odds ratio, 2.3; 90% CI, 1.0-5.5; P = .10) were higher for participants receiving sertraline, but overall, sertraline was well-tolerated.

In a subgroup analysis of participants without an identified mood disorder (n = 67), sertraline did not significantly impact attention, working memory, episodic memory, executive control, inhibition, or processing speed compared to placebo. Interestingly, although SSRIs are thought to exacerbate apathy, the sertraline group showed a decrease in apathy symptoms as measured by the Apa-thy Evaluation Scale compared with the placebo group who showed an increase in apathy scores (F [1, 67] = 4.73; P = .033).

To our knowledge, the only other double-blind, placebo-controlled RCT of a pharmacologic intervention for the prevention of TBI-associated depression examined the effect of sertraline in a sample of patients with mostly severe TBI. In this study, participants were randomized to sertraline 50 mg (n = 49) or placebo (n = 50) for 3 months and were evaluated for depression at 3-, 6-, and 12-months post-enrollment. According to the intent-to-treat analysis, the cumulative incidence of depression (defined by a cutoff score of 6 on the 6-item version of the Hamilton Depression Scale) at 3-months follow-up was lower with sertraline compared with placebo (χ² 5.16, P = .024). However, this difference was no longer significant at 12-months follow-up (χ² 3.69; P = .055).

These discrepant findings may be explained by several methodological differences. For instance, the dose and duration of sertraline in the latter trial may have been insufficient. It is also plausible that the preventive effect of sertraline only occurs while taking the antidepressant. Consistent with our findings, however, was the lack of significant improvement on measures of concentration, processing speed, memory, or executive function in the sertraline group compared with placebo at 3 months.

Prospective, controlled studies of non-pharmacological interventions are lacking. In one study by Bombardier and colleagues, 7 telephone calls from a research care manager that addressed client-centered, real-time problems following discharge from inpatient rehabilitation significantly reduced depressive symptoms at 12-months relative to a control group. However, 29% of their sample had depression at baseline.

Scheen and colleagues compared a 5-session, cognitive behavioral therapy intervention to a telephone control in 84 patients with mild TBI and at least 3 post-concussive complaints. The intervention did not have a statistically significant effect on the primary outcome of return-to-work or on the severity of depression and anxiety symptoms at 6 or 12 months. Further studies of non-pharmacological interventions are warranted.

**Potential barriers to prevention**

The success of a preventive intervention depends on its ease of administration and acceptability to patients and clinicians. The acceptability of pharmacologic interventions may be hindered by concern about adverse effects. Results from our sertraline study showed modestly increased odds of dry mouth and diarrhea compared with placebo. However, to our knowledge, more serious adverse effects of SSRIs have not been reported in treatment and preventive studies of patients with TBI, including hyponatremia, gastrointestinal bleeds, falls, fractures, hypomania, or mania. The risks of SSRIs must still be considered on an individualized basis.

It is unclear how comorbid psychiatric, neurologic, and other medical complications may influence the pre-existing mood and anxiety disorders and antidepressant medications for the development of TBI-associated depression. In our study, 16% of patients screened for eligibility were excluded due to current mood or anxiety disorders or use of antidepressant medications within the 6 months prior to injury. However, 22% of our randomized patients had a remote history of a single episode of depression, and they too benefited from sertraline. Since safety and efficacy of this or other SSRIs. The development of feasible, non-pharmacologic interventions is greatly needed. At this time, there is insufficient evidence to recommend the implementation of any pharmacologic or non-pharmacologic intervention for the prevention of TBI-associated depression, and the safety of prophylactic SSRI therapy for high-risk patients must be considered on a case-by-case basis.

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The authors report no conflicts of interest concerning the subject matter of this article.

**References**

A Complicated Case of Psychiatric Disability

Barbara Long, MD, PhD, Andrew O. Brown, MD, Sean Sassano-Higgins, MD, David "Daven" E. Morrison, MD, and Ben Hunter, MD, for the Committee on Work and Organizations, Group for the Advancement of Psychiatry

Bob, aged 50 years, worked as a factory manager for a large corporation but had problems getting along with peers and managers. Ultimately, he was fired for initiating safety policies without consulting with his supervisor and mandating compliance in his subordinates, who complained, leading to his termination. In his spare time, Bob renovates and sells (i.e., flips) houses.

He went to his psychiatrist because of PTSD symptoms that he alleged were caused by the harassment he was subjected to and to wrongful termination by his employer. He wants his psychiatrist to complete his forms for disability. Bob had been discussing his problems about getting along with coworkers and superiors but said that he had finally “realized that the issue is my job. I’m always stressed and working full time has eliminated any space to work on myself and decompress. I just found out that I’m eligible for permanent disability with full pay based on my tenure—isn’t that wonderful?”

Countertransference

If this was so wonderful, then why was Bob’s psychiatrist filled with countertransferential dread? The concept of patient-defined disability seemed problematic. Work was indeed a source of ongoing angst, but Bob had been employed for years. Even if his current work environment was truly damaging, he was clearly capable of performing another substantial job, if not presently, then at some point in the future.

Patients who confess to struggling at work feel differently than patients who request disability. As the psychiatrist’s countertransference reaction revealed, the problem is one of role reversal between the treater and patient. Patients do not make functional assessments on themselves, professionals do.

If a psychiatrist determines that a patient is profoundly functionally impaired as demonstrated by the patient’s report of symptoms and the manifestations thereof, such as difficulty performing job tasks, evaluation for disability is the next step. The disability evaluation should be performed by a third-party mental health professional, as the treating psychiatrist has an inherent conflict of interest: his or her task is to advocate for the patient’s mental health, not disability compensation—to listen and understand, to demonstrate empathy, and to alleviate suffering. The task is also to instill hope and confidence, and to restore functioning. In many cases, deeming a patient “disabled” can be highly counter-therapeutic.

Knowing the consequences

But what to do when the patient says that he needs disabled status? When, despite your professional opinion, the patient is convinced that work is not, in fact, a source of meaning and value and an excellent venue for working through relationship and life issues? When the patient believes that he is incapable of working, but you believe otherwise? Disagree or refuse his request, and the therapeutic alliance will be severely strained if not broken. The patient very well may displace his sense of work-related victimization, helplessness, and rage onto the provider, which can prove damaging for the psychiatrist as well as the patient.

What if a psychiatrist agrees with the patient’s professed disability and submits the required paperwork? This situation is no less problematic. Regardless of explanations to the contrary, the psychiatrist communicates that the patient is sick, ineffective, and at least temporarily broken and that he or she, as the treating psychiatrist, is incapable of healing the patient and therefore has given up. In the case of long-term disability, as with the patient above, the statement is more profound given its implied permanence. Thus, treaters have the options of either opposing disability, thereby damaging the therapeutic alliance, or supporting disability thus professing pessimism about the patient’s odds of recovery and removing work and its inherent value from the patient’s life.

If a psychiatrist agrees with the patient’s request for disability, he or she, as the treating psychiatrist, is incapable of healing the patient and therefore has given up. In the case of long-term disability, as with the patient above, the statement is more profound given its implied permanence. Thus, treaters have the options of either opposing disability, thereby damaging the therapeutic alliance, or supporting disability thus professing pessimism about the patient’s odds of recovery and removing work and its inherent value from the patient’s life.

The only reasonable option, particularly for long-term disability assessments, is to refer a patient for a third-party assessment. Occupational or forensic psychiatrists are an excellent option because they understand psychopathology and the functional and prognostic implications of disease. Unfortunately, this was not possible in the case that led our discussion. The paperwork specifically required that the treating psychiatrist deem the patient disabled, to the point of stating that third-party assessment would not be acceptable. The psychiatrist was handcuffed.

A quandary resolved

The psychiatrist, who did not agree that PTSD was the appropriate diagnosis for the patient, elected to provide fair warning, making explicit the dangers of filling out the paperwork. He warned that he would, in essence, be admitting defeat, and that the patient’s sense of trust in his psychiatrist and the field would waver. He explained that the patient’s core sense of brokenness and impotence would be confirmed, by a professional no less. He told the patient that removing work would remove a source of pur-
pose, half of Freud’s “love and work” equation for mental health. Furthermore, he expressed confidence in his own ability to help the patient heal, his hope that this man would improve his maladaptive behavioral patterns and improve his ability to work effectively. He allowed the patient to make the decision, invoking informed consent.

What ensued was a therapeutic failure. The patient chose disability, and for 2 weeks, he felt significant relief. Shortly thereafter, he became crippledly anxious, unable to plan and utilize his newfound free time effectively. Every unfilled minute reinforced his avoidance. He felt more broken than ever, separated from the herd of functional peers, a weak man who had admitted defeat, a man who no longer contributed to his society, his family, or his own well-being. Profound shame overcame him, followed by extreme anger misdirected towards the people and system he perceived to be driving his pain. Foremost among these abusers? His psychiatrist, who was “fired” less than a month later in favor of a “more supportive” physician down the road. The psychiatrist, viewed by the patient as adversarial, now experienced a profound feeling of impotence and guilt.

Conclusions
Although in some cases, a determination of disability is justifiable, necessary, and humanitarian, far too often, disability requests end in shared grief between patient and treater. This is a cautionary tale, but an avoidable one. Treating psychiatrists must continue to advocate for boundaries and avoid assuming a dual role of treater/evaluator with its inherent conflict of interest. Employers and insurance companies that insist on treating providers serving as disability assessors must be challenged. Patients must be encouraged to return to work, to have faith in themselves and their ability to recover, and to trust the system.

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EXCLUSIVE CLIMATE CHANGE COVERAGE

Divestment in Fossil Fuels
A Preventive Public Health Strategy

American Psychiatric Association Assembly Passes Action Plan on Divestment of Fossil Fuels

Robin Cooper, MD

The profound effects of climate change have become increasingly more difficult to ignore or compartmentalize into the comfortable deep recesses of our minds. Whether it be the alarming reports of the Intergovernmental Panel on Climate Change, the 4th National Climate Assessment, the Lancet Countdown of 2018, or daily media reports showing the horrors of mega-storms, floods, wildfires, and droughts, the evidence everywhere indicates that climate change is happening at a pace and ferocity previously unanticipated.1,2

The American Psychiatric Association (APA) has recognized the profound effects of climate change on mental health. In March 2017 the APA adopted a position paper on Climate Change and Mental Health, which is the official stance guiding the organization’s actions and activities.3 The position paper underscores APA’s commitment to action on climate change by stating, “climate change poses a threat to public health, including mental health” and recommends “support and collaboration with patients, communities and other health care organizations engaged in efforts to mitigate adverse health and mental health effects of climate change.”

Rapidly growing numbers of health professionals have expressed profound concern and are grappling with how to appropriately and effectively respond directly with our patients, with the general public, and in relation to policy advocacy. Several earlier articles in this series have outlined the direct and indirect clinical impacts of climate disruption on mental health and public health.

The Climate Psychiatry Alliance (CPA) is a group of dedicated psychiatrists whose mission is to inform the psychiatric profession and the public of the severe impacts of climate disruption on mental health and to advocate for actions to mitigate and adapt to these impacts. (www.climatepsychiatry.org).

Building on the APA’s commitment to “engage in efforts to mitigate adverse health impacts,” CPA developed a proposal for the APA to divest from fossil fuel companies. We were inspired in part by the successful passage of a divestment plan by the American Medical Association. With a commitment to align APA’s financial activities with our mental health professions’ stated values and principles, the CPA worked with APA assembly member, Dr James Fleming (also a CPA member) to introduce an Action Paper in Fall 2018 that advocates for APA divestment from companies with substantial assets in fossil fuels.4

Over 125 APA members signed on as supporters and several committees and caucuses endorsed the Action Paper (a remarkable level of membership engagement and support). The Action Paper passed with 61% majority, a result stunning for both the margin of victory and speed with which it moved through the Assembly’s usually slow process. The Action Paper has now been referred for review of the financial implications. Hopefully, this will lead to approval by the Board of Trustees, which would make divestment the official policy of the organization.

Why divestment?
There is no doubt that fossil fuels are driving catastrophic levels of climate change and they are the biggest contributors to global warming. Invest-
ments in fossil fuel companies contribute to making profitable the continuation of reliance on fossil fuels and are completely incompatible with the clear need to transition to a “clean economy,” an economy based on renewable sources of energy.

Divestment of investments in companies that operate using morally, ethically, and politically objectionable activities have historically been important and effective strategies to influence policies. Most notably, divestment was one tool for progressive change during the period of international efforts against apartheid in South Africa.

Divestment is now a tool in the efforts to mitigate climate change. Since climate change is such a profound threat to both general health and mental health, divestment can be viewed as a preventive public health strategy that addresses the root causes of health threats. Divestment operates at multiple levels.

1 MORAL LEVERAGE: Establishing a moral foundation for financial operations, Nobel Laureate for Peace and prior UN Secretary General Desmond Tutu has said that “people of conscience need to break their ties with corporations financing the injustice of climate change.”

2 ECONOMIC LEVERAGE: Although any individual groups’ holdings may not substantially affect share prices, the collective efforts of many groups can influence the vitality of the marketplace.

3 POLITICAL LEVERAGE: Growth in collective divestment has a political impact on efforts to move toward a clean economy. The precedent of physician support for divestment from tobacco companies was an effective preventive public health strategy in thwarting the tobacco industry and is a model for action now.

4 PSYCHIATRIC PRINCIPLE OF FACING THE TRUTH: Embedded in our professional being is the principle of facing and tolerating the truth. Just as with the tobacco industry, the fossil fuel industry has perpetrated public misinformation, sown seeds of doubt, and contributed to public confusion regarding the scientifically based evidence for the causes of climate change. The fossil fuel industry has used its enormous political and economic leverage to influence policies that sustain their profitability while undermining public interests. Combined with the unconscious processes of psychological denial, the fossil fuel industry promotes and contributes to denial and psychological retreat.

Divestment is a smart economic strategy

Not only is withdrawal of investments in fossil fuel companies the morally right thing to do, it is also a wise financial strategy. Christiana Figueres, formerly executive secretary of the United Nations’ Framework Convention on Climate Change was an early advocate of divestment. She believes that investment decisions should reflect the latest scientific evidence for the dangers of climate change to both protect health and financial savings of ordinary citizens. She has said “The pensions, life insurances, and nest eggs of billions of ordinary people depend on the long-term security and stability of institutional investment funds,” and that “Climate change increasingly poses one of the biggest long-term threats to those investments and the wealth of the global economy.”

Divestment from fossil fuels is gaining traction. To name only a few, New York City pension funds and CalPERS and the California Teachers’ retirement plans (one of the largest public entity retirement plans in the US) have declared that investing in fossil fuel companies is incompatible with their public mission. Medical institutions and professional health organizations are joining the lead of the Canadian Medical Association, American Medical Association, British Medical Association, Royal College of General Physicians and numerous other medical organizations by adopting divestment plans.

The recent adoption of the APA Action Paper puts our organization in good company. This is just the first step in the APA process of adopting divestment as the organization’s guiding investment policy. The next step will be the approval by the APA Board of Trustees to divest from the fossil fuel industry. If approved, it will be a model for physicians and other health care professionals to align their financial practices with the mission to promote individual and public health and will be a small but important step on the path toward preventing, mitigating and reversing the most serious health crisis of our time.

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Dr Cooper reports no conflicts of interest concerning the subject matter of this article.

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The Week the EMR Went Down

Richard M. Berlin, MD

we fled the computer room like inmates after lightning fries the prison fence.

Then we rounded with nurses who knew the doses and what made patients moan, wrote SOAP notes with pens, our findings compressed like sonnets. We sat at bedsides and absorbed each story, comforted families, took time to teach residents and share their burdens. Elated to eat lunch, we compared cases with colleagues and gave curbside consults. Finished before sunset, charts and patients tucked in, we cruised home to partners eager to hear the stories, their mirrored smiles the notes our progress.

Dr Berlin is Instructor in Psychiatry at the University of Massachusetts Medical School, Worcester, MA

References

Psychosis After Traumatic Brain Injury: Conceptual and Clinical Considerations

Psychosis after traumatic brain injury (TBI) is a relatively uncommon condition that presents both clinical and conceptual challenges. DSM-5 criteria for Psychotic Disorder due to Another Medical Condition define psychotic disorder due to TBI as delusions or hallucinations that are direct physiologic consequences of TBI; are not better explained by another psychiatric illness or delirium; and cause clinically significant distress or impairment. The requirement for attributing psychosis directly to TBI is often difficult to meet, as the relationship between psychosis and TBI is often multifactorial and rarely permits simple causal attribution.

The cognitive deficits, comorbid medical and neurological problems, and complex pharmacotherapeutic needs and sensitivities of persons with TBI frequently complicate evaluation and treatment of psychosis in this population. In most cases, the development of psychosis in an individual with a history of TBI will be most usefully framed as a psychosis associated with, or after, TBI (ie, posttraumatic psychosis) rather than psychosis due to TBI. Framed as such, relevant differential diagnostic considerations, neurodiagnostic investigations, and evidence-informed management can substantially improve quality of life for patients with posttraumatic psychosis and for their families.

Characteristics of posttraumatic psychosis

DSM-5 guidelines suggest that psychosis due to TBI be considered in a patient with both a history of TBI and psychosis when there is a temporal association between TBI and psychosis and/or when there are atypical features of psychosis present (eg, atypical age of onset or non-auditory hallucinations). In practice, the sometimes-long latency between TBI and the subsequent development of psychosis makes the required temporal association for the diagnosis of psychosis due to TBI difficult to establish. In fact, posttraumatic psychosis demonstrates a bimodal latency period, with a relative minority of patients developing hallucinations and delusions symptoms in the early period after TBI and a larger subset first developing such symptoms years—sometimes decades—after injury, with the mean onset of psychosis after TBI being four to five years.

Delusions are a core feature of posttraumatic psychosis. The syndrome typically takes one of two forms: delusional disorder, in which delusions are the dominant feature, and schizophreniform disorder, in which delusions, hallucinations, and disorganized speech and behavior are prominent.

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sole psychotic feature; and schizophrenia-like psychosis, characterized by delusions and hallucinations. Delusional disorder occurs about twice as often as schizophrenia-like psychosis, tends to occur later, and often features misidentification themes. Schizophrenia-like psychosis after TBI typically features paranoid and persecutory delusions and hallucinations, with grandiose, religious, and external control-related delusions occurring less often. Hallucinations are usually auditory but can occur in other modalities and do so at rates greater than those seen in schizophrenia. Both subtypes are often preceded by a prodrome superimposed on other, usually present, sequelae of TBI; that prodrome is characterized by affective instability, bizarre behavior, performance decline at school or work, social withdrawal, and antisocial behavior. Formal thought disorder and negative symptoms such as affective flattening and avolition are uncommon.

**Epidemiology**

Epidemiologic data on posttraumatic psychosis are mixed. A landmark 1969 review concluded the incidence of psychosis after TBI to be two to three times that of the general population, and modern estimates suggest rates of 0.9% to 8.5%. A large 2011 meta-analysis estimated a 60% increase in the risk of schizophrenia after TBI compared with the general population. Other large-scale studies, however, have failed to show this association, or have shown an increased risk of psychosis that is eliminated after statistically controlling for comorbid substance use disorders. While a 2011 5-year prospective study showed increased rates of schizophrenia in patients with TBI, a similar 2016 study did not. The data are likely confounded by inconsistencies across studies with regard to the methods by and rigor with which TBI is assessed:

- Ascertainment bias, particularly ascertainment in clinical/specialty referral populations rather than in community-dwelling populations
- Whether schizophrenia (rather than individual psychotic symptoms) is used as an outcome
- The latency period of psychosis after TBI, which may evade even 5-year observational studies
- The use of family history of psychotic disorders as an inclusion or exclusion criterion
- The probable bidirectional relationship between TBI and psychosis, particularly in multiplex pedigree schizophrenia and bipolar disorder samples

**Pathophysiology**

The nature of the relationship between TBI and psychosis is complex and appears to be influenced strongly by patient-specific factors. In some cases, TBI may act as a stressor in a stress-diathesis model of schizophrenia, interacting with underlying genetic susceptibility to produce psychosis. Studies on family history of schizophrenia as a risk factor for psychosis after TBI support this model. Findings from other studies suggest that TBI may directly induce structural and/or functional brain changes in sensory- and other information-processing networks that manifest with hallucinations and delusions, respectively. Data that show lower rates of familial schizophrenia in patients with posttraumatic psychosis than among patients with schizophrenia without TBI support this model. Because psychosis itself may engender high-risk behaviors, a “reverse causality” model suggests that psychosis predisposes to TBI rather than vice versa. While pre-TBI rates of psychosis are low—with estimates ranging from 0% to 4.3% and largely accounted for by substance use disorders—an increased risk of TBI exposure in members of schizophrenia multiplex pedigrees has been seen. Higher rates of TBI and other accidental injury in the year preceding the first lifetime admission for schizophrenia suggest that incipient psychosis might predispose to trauma. The association between TBI and psychosis may be spurious, driven by independent “psychosis-proneness” factors (eg, perceptual aberrancies, schizophrenia spectrum disorders. Subtle neurological abnormalities are more common among children and adolescents with or at-risk for schizophrenia, even when compared with bipolar controls—potentially predisposing these individuals to accidental injury. Childhood head trauma, which occurs more frequently in schizophrenic patients than in depressive, bipolar, and non-psychiatric controls could be interpreted as a marker of impaired caretaking, with schizophrenic patients placed at higher risk of early TBI merely by virtue of being offspring of parents with schizophrenia.

### Table 1. Potential mimics of psychotic symptoms in persons with posttraumatic psychosis

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Must be distinguished from</th>
</tr>
</thead>
<tbody>
<tr>
<td>Confabulation</td>
<td>Delusions (eg, fixed false beliefs); confabulated beliefs are not fixed and typically occur in the setting of severe amnesia</td>
</tr>
<tr>
<td>Fluent (Wernicke) aphasia</td>
<td>Word salad (nonsensical speech due to severe psychosis); fluent aphasia is accompanied by impairment in language comprehension</td>
</tr>
<tr>
<td>Severe apathy or akinetic mutism</td>
<td>Catatonia</td>
</tr>
<tr>
<td>Severe behavioral disinhibition</td>
<td>Grossly disorganized behavior</td>
</tr>
</tbody>
</table>

### Table 2. Considerations in the differential diagnosis of posttraumatic psychosis

- Posttraumatic confusional state
- Delirium due to other causes (eg, substance intoxication or withdrawal, adverse effects of medications)
- Primary (idiopathic) schizophrenia spectrum disorder
- Manic episode with psychotic features
- Major depressive episode with psychotic features
- Posttraumatic stress disorder with severe intrusive memories, flashbacks, and dissociative symptoms
- Confabulation in association with severe posttraumatic cognitive impairment
- Peri-ictal and postictal psychoses

**Risk factors for posttraumatic psychosis**

Younger age at the time of TBI may increase risk of schizophrenia, though these data are mixed. Among individuals diagnosed with posttraumatic psychosis or psychosis due to TBI rather than schizophrenia the age at which TBI occurred does not appear to be a relevant risk factor. Men are at higher risk for posttraumatic psychosis, even when controlling for the baseline increased risk of TBI in men. Family history of psychotic disorders is a risk factor for receiving a diagnosis of schizophrenia after TBI, with the risk increased almost threefold in patients in multiplex schizophrenia pedigrees. However, posttraumatic psychosis is frequently observed in individuals without a family history of psychotic disorder.

More severe injuries may confer higher risk of posttraumatic psychosis, although schizophrenia studies generally have not shown an association with injury severity. The apparent association of posttraumatic psychosis with more severe TBI and schizophrenia with all injury severities may reflect ascertainment biases; researchers may also be more willing to attribute psychosis to TBI when the occurrence and severity of injury is unequivocal. Left hemisphere—particularly left temporal—injuries are more commonly associated with the schizophrenia-like psychosis form of posttraumatic psychosis; right-sided injuries are more frequently associated with its delusional disorder form. A history of another pre-TBI neurologic disorder may also increase the risk of posttraumatic psychosis.
Nearly 90% of patients with posttraumatic psychosis have cognitive dysfunction attributable to TBI, most typically impairments of attention, language, memory, visuospatial function, and executive function. Magnetic resonance imaging (MRI) or computed tomography (CT) of the brain is typically abnormal in individuals with posttraumatic psychosis, with frontal and temporal lesions being the most common neuroimaging abnormalities. Electroencephalography (EEG) is abnormal in 70% to 87% of cases; focal temporal and/or frontal slowing is the most common finding, with epileptiform discharges occurring in 27% of schizophrenia-like psychosis and 14% of delusional disorder cases.

**Approach to evaluation**

To establish occurrence of a TBI and gauge its severity, inquiry focuses on event-related alterations in consciousness, impaired memory for the injury event (posttraumatic amnesia), and related changes in mental state. The DSM-5 description of Neurocognitive Disorder due to Traumatic Brain Injury provides guidance on the information needed to establish the occurrence of a TBI and gauge its severity (page 626). Readers are encouraged to review and utilize this guidance in their assessment of persons with suspected posttraumatic psychoses.

In addition to determining whether a TBI occurred and, if so, characterizing its severity, the evaluation should seek to identify pre-injury risk factors for posttraumatic psychosis, including neurodevelopmental and psychiatric history, substance use disorders, and family history of psychosis. Careful probing of the weeks and months prior to TBI is essential and can reveal prodromal psychotic symptoms that may have gone unrecognized. The presence of delusions, hallucinations (auditory and otherwise), and negative symptoms should be evaluated, being mindful that negative symptoms may be difficult to parse from post-TBI cognitive dysfunction.

Atypical age of onset and a temporal relationship of psychosis to TBI can be helpful in establishing posttraumatic psychosis. The illness tempo should be elicited, with a paroxysmal course suggesting the possibility of posttraumatic epilepsy. Obtaining collateral information from knowledgeable informants and from the medical record is essential, as posttraumatic cognitive dysfunction, impaired self-awareness, and psychotic symptoms may impede a patient’s ability to serve as a reliable historian.

Mental status examination should evaluate for hallucinations (without insight) and delusions; the character of these symptoms among persons with posttraumatic psychosis is often indistinguishable from those occurring among persons with primary psychotic disorders. The evaluation should also seek to identify formal thought disorder, affective flattening, and negative symptoms, the presence of which is uncommon among individuals with posttraumatic psychoses.

Assessment of attention, processing speed, language, memory, and executive function is essential, and findings must exclude delirium and other potential psychosis mimics (Table 1). Elemental neurological exam should seek to identify focal (especially asymmetric) cranial nerve, motor, sensory, and/or reflex findings consistent with structural brain lesions.

Routine serum laboratory testing should be done to exclude metabolic arrangements known to produce delirium or psychotic-like symptoms. Structural neuroimaging should be obtained in all cases of suspected posttraumatic psychosis. MRI is the preferred modality, with T1, T2, fluid-attenuated inversion recovery (FLAIR), and T2* gradient echo (or susceptibility-weighted) sequences (Figures 1 and 2). In most cases, but especially when the history suggests paroxysmal events, EEG should be performed, with consideration of prolonged and/or video-EEG monitoring if the suspicion for posttraumatic epilepsy is high. Neuropsychological testing, which commonly clarifies the nature and extent of cognitive impairments, may help to identify patterns of impairment typical for TBI versus schizophrenia and should also be part of most evaluations.

**Management**

When psychosis occurs in the setting of mood disorders, substance use disorders (especially substance intoxication or withdrawal), or posttraumatic epilepsy, these should be treated first, and the diagnosis of posttraumatic psychosis deferred until these conditions are effectively treated. If cognitive impairment is prominent and appears to be contributing to information processing abnormalities (eg, delusions), treatment with a cholinesterase inhibitor may improve cognition or produce frank delirium, both of which may be mistaken for posttraumatic psychosis. Finally, benzodiazepines may induce paradoxical behavioral disinhibition and delirium that can be mistaken for posttraumatic psychosis.

Epilepsy, including posttraumatic epilepsy, can present with ictal, post-ictal, or interictal psychosis. When psychosis occurs in association with posttraumatic epilepsy, a diagnosis of posttraumatic psychosis should be deferred until it is clear that psychotic symptoms persist despite effective seizure control.

Medication-induced psychotic symptoms should also be considered. For example, amantadine, commonly prescribed for cognitive enhancement following severe TBI, may produce hallucinations and delusions in some patients, as can the anticonvulsants levetiracetam and topiramate. Potent anticholinergic and antihistaminergic medications may impair cognition or produce frank delirium, both of which may be mistaken for posttraumatic psychosis. Finally, benzodiazepines may induce paradoxical behavioral disinhibition and delirium that can be mistaken for posttraumatic psychosis.

**Differential diagnosis**

Psychosis occurring within days or weeks of injury must be distinguished from the posttraumatic confusional state (or delirium), a period of fluctuating disorientation, cognitive impairment, psychomotor restlessness, and sleep/wake disturbance that appears early in the TBI recovery course and can involve transient psychotic symptoms. Delirium due to other causes as well as substance-use disorders (including substance intoxication or withdrawal) must also be excluded. (See Table 2 for a list of considerations in differential diagnosis.)

If mood symptoms are present, diagnoses of mania or depression with psychotic features should be considered, especially if there is a history of premorbid mood disorder. Posttraumatic stress disorder should be considered if the content of symptoms is specific to the trauma event, and careful discrimination between trauma-related symptoms (eg, intrusive thoughts and memories, flashbacks, avoidance) and psychotic symptoms (eg, hallucinations without insight, delusions) is essential.

Schizophrenia should be considered when negative symptoms or formal thought disorder are prominent and when the severity of TBI is mild enough (eg, single, uncomplicated, very remote concussion) so that psychosis is implausibly attributable to it. Isolated hallucinations after TBI are rare and their presence should prompt consideration of other etiologies such as peducnular hallucinosis due to brainstem injury or release phenomena due to vision or hearing loss.

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with movement disorders given its relatively lower likelihood of producing extrapyramidal effects and cognitive dysfunction, although orthostatic hypotension and, with overly rapid dose escalation, sedation can be treatment-limiting adverse effects. Risperidone is generally effective at doses of 2 to 4 mg, and olanzapine and aripiprazole are also commonly used at doses of 2.5 to 10 mg and 6 to 10 mg, respectively. Chlorpromazine was reported to worsen the condition and produce psychosis after TBI in one case, possibly due to its prominent anticholinergic properties.19

Conclusions
Psychosis after TBI has many potential causes, and the differential diagnosis must be thoroughly considered before attributing psychotic symptoms to TBI. Pharmacotherapy for all forms of psychosis after TBI should take cognitive dysfunction, comorbid illnesses, and the injured brain’s increased sensitivity to medication adverse effects into account, following a “start low, go slow, but go” approach.

References

Figure 2. Brain MRI of a 23-year-old woman presenting with new-onset paranoia four months after a motor vehicle collision in which she suffered significant cervical spinal injury but no obvious head trauma. Susceptibility-weighted imaging sequence revealed chronic microhemorrhages in the bilateral frontal lobes, bilateral anterior temporal lobes, and splenium of the corpus callosum consistent with prior trauma with diffuse axonal injury, confirming a diagnosis of traumatic brain injury.

TBI in Older Adults

Older adults with TBI deserve the same advocacy and focused study, as sports- and military-related TBI. As individualized medicine and precision medicine continue to be heralded as the path forward, a “one-size-fits-all” approach to the conceptualization, diagnosis, and management of TBI in older adults will be viewed as increasingly antiquated and unacceptable.

Dr Nargapreddy is a Neuropsychiatry Fellow, Ms Richie is Research Coordinator, and Dr Peters is Assistant Professor, Department of Psychiatry and Behavioral Sciences, Johns Hopkins University School of Medicine, Baltimore, MD.

The authors report no conflicts of interest concerning the subject matter of this article.

References
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Visit www.CHAproviders.org to learn more and apply through our secure candidate portal. CV and cover letter may be sent directly to Melissa Kelley, CHA Provider Recruiter via email at makelley@challiance.org. CHA's Department of Provider Recruitment may be reached by phone at (617) 665-3555 or by fax at (617) 665-3553.

CHA is an equal opportunity employer and all qualified applicants will receive consideration for employment without regard to race, color, religion, sex, sexual orientation, gender identity, national origin, disability status, protected veteran status, or any other characteristic protected by law.
Northwest Permanente, P.C. invites you to consider these opportunities with our physician-managed, multi-specialty group of over 1,500 physicians and clinicians who care for over 600,000 members throughout Oregon and Southwest Washington.

**BC/BE Adult Psychiatrists** Opportunities in the Portland Metro area to utilize a combination of face-to-face and the latest virtual modalities. Candidates should have experience in evidence-based, psychiatric treatments, including primary care consults, crisis interventions and medication consultation.

**BC/BE Psychiatrist – Virtual Care Lead** Opening in Portland, OR to work as our Virtual Care Lead on-site, alongside 12 virtual therapists. This team provides initial virtual consultations for patients across OR and WA to assist with case closures, triage patients and support technical difficulties. All candidates will need a strong interest in providing Telepsychiatry services and leadership.

**BC/BE Child Psychiatrist** Opening in Portland, OR.

**VISIT US AT BOOTH 604 AT THE APA ANNUAL MEETING, MAY 18-22, 2019 IN SAN FRANCISCO, CA!**

We offer a competitive salary, incentives such as student loan assistance and signing bonus, incredible retirement plans, including Defined Contribution, 401K, pension & more, annual educational leave and long-term, sabbatical benefits.

Apply at: https://nwp.kpphysiciancareers.com. Or call Jason Dulin at (503) 813-2242 or email Jason.R.Dulin@kp.org. EOE

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RECRUITING FULL TIME & PER DIEM PSYCHIATRISTS NEW YORK METRO AREAS

Northwell Health’s Behavioral Health Service Line strives to address the diverse mental health needs of the communities we serve by providing a continuum of accessible, high quality psychiatric and substance abuse services including emergency, crisis, inpatient, and outpatient programs for people of all ages. Northwell’s clinical programs are complemented by a robust education, training, and research enterprise, including the world-renowned Psychiatry Research Department at The Zucker Hillside Hospital, which has led cutting-edge investigations that have meaningfully influenced many lives.

**TO BOLSTER OUR NETWORK OF OUTSTANDING CARE PROVIDERS,**
**WE ARE RECRUITING BOARD ELIGIBLE/BOARD CERTIFIED PSYCHIATRISTS FOR THE FOLLOWING POSITIONS:**

**GERIATRIC OUTPATIENT PSYCHIATRIST**
The Zucker Hillside Hospital
Glen Oaks, NY

**ADULT INPATIENT PSYCHIATRIST**
South Oaks Hospital
Amityville, NY

**DIRECTOR, OUTPATIENT MENTAL HEALTH CLINIC**
Lenox Hillside Hospital (MEETH)
Manhattan, NY

**CONSULTATION LIAISON PSYCHIATRIST**
LUMC- Forest Hills Hospital, NY
Staten Island University Hospital, NY

**CONSULTATION LIAISON PSYCHIATRIST**
Northern Westchester Hospital
Mt. Kisco, NY

**CONSULTATION LIAISON PSYCHIATRIST**
Staten Island University Hospital
Staten Island, NY

**CONSULTATION LIAISON PSYCHIATRIST**
Huntington Hospital
Huntington, NY

**TELEPSYCHIATRIST**
Greenwich Village HealthPlex
Greenwich Village, NY

Benefits at Northwell Health include:

- Nationally competitive salaries
- Comprehensive benefits package
- Four weeks’ vacation plus paid conference/CME time

Qualified candidates should forward their CV to Lan Ma: OPR@northwell.edu

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The Department of Psychiatry at Rutgers New Jersey Medical School in Newark, New Jersey, is recruiting for full-time academic psychiatrists to join our team. Successful candidates will actively participate in clinical care, medical student and graduate medical education, and will have the opportunity to engage in research and other scholarly activities in an urban tertiary-care medical center.

We currently have several openings for full-time positions:

1. **Inpatient Psychiatry Unit**: Each attending on this unit leads a multidisciplinary team which includes a social worker, a nurse, a recreational therapist, a psychiatry resident, and rotating medical students. This is a high acuity unit with ample social work support, an experienced nursing staff, and an enthusiastic recreational therapy team. The workload allows plenty of time for teaching.

2. **Consultation-Liaison Psychiatry Service**: We are looking to fill two full-time positions. This is a dynamic service with a strong focus on teaching; there is a full-time psychiatry resident and a variety of other residents, medical students, and psychology interns who rotate through the service on a regular basis. We provide consultations to medically complex patients on medical, surgical and obstetrical inpatient units, including an active liver transplant service.

3. **Emergency Department**: We are looking for an attending to direct the psychiatric services in the ED. There are plentiful opportunities for teaching as there is a full-time psychiatry resident on service and a number of rotating medical students and residents on elective. We have a team-based approach with a strong team of mental health screeners who assist with clinical assessment, collateral-gathering, and disposition.

The positions above are an outstanding opportunity to be part of an innovative and thriving academic department. Our faculty positions are particularly well-suited for early career psychiatrists with an interest in academic psychiatry. We are proud of our culture of mentoring the new generation of leaders in our field and strongly support our faculty member’s professional development.

Candidates must be able to obtain a New Jersey medical license and be board certified or board eligible. Academic rank will be commensurate with experience.

Rutgers NJMS is located within an easy commute from Manhattan and many attractive New Jersey communities with excellent school systems. Our competitive compensation package includes comprehensive health care, generous retirement savings, and 22 paid vacation days.

Rutgers NJMS faculty may also join the outpatient faculty practice which can generate additional income.

Interested psychiatrists should send their CV to the attention of the Department Chair, Petros Levounis, MD, MA, at Petros_levounis@rutgers.edu. We are an AA/EEO employer.
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.

Through a partnership between Hackensack Meridian Health and Seton Hall University, the School of Medicine will redefine graduate medical education, research, and clinical practice; reverse the critical physician shortage in both the New York/New Jersey metropolitan area and the nation; and stimulate economic development in northern New Jersey.

The School of Medicine will be the anchor in the development of a comprehensive health sciences campus that will also include research facilities and biotechnology endeavors – all in service of educating tomorrow’s doctors, discovering novel therapies, and facilitating compassionate and effective healthcare that will meet the ever-changing needs of tomorrow’s patients.

The School of Medicine will be the cornerstone of a dynamic venue for the exchange of ideas, the development of healthcare and research thought leaders and practitioners, and the discovery of novel therapies to meet the medical challenges of the future.

“Ocean Medical Center’s psychiatry program will be a community-based program,” said Ramon Solikhah, M.D., program director for psychiatry as well as founding Chair of Psychiatry & Behavioral Health at the Hackensack Meridian School of Medicine at Seton Hall University. “Our new psychiatry residency program will improve clinical care and ultimately encourage future health care leaders to build practices in the Jersey Shore area.”

Child and Adolescent Psychiatrist – Outpatient Consultation Position Full Time * Multiple locations in New Jersey

Hackensack Meridian Health is seeking a Board Certified/Board Eligible Child and Adolescent Psychiatrist to join this growing team. With 4 hospitals in the top 10 ranking in New Jersey, this is an outstanding opportunity to join the area’s largest healthcare network.

Highlights:
• Academic Affiliations with the new Hackensack Meridian Health School of Medicine at Seton Hall University.
• Collaborations among multiple sites (statewide).
• Call is not required.
• Outpatient/Consultative setting.
• Competitive Salary.
• Comprehensive Benefits Package.

In addition to our collegial work environment, we offer a highly competitive compensation package which includes: medical/dental plans, 401(b) retirement plan, and relocation assistance.

For immediate consideration, please contact Renee Theobald, at: Renee.Theobald@hackensackmeridian.org or call: 732 781-3597

NATIONWIDE

Aligned Telehealth, Inc. – California
Our mission is to be the leader in innovative, high quality, accessible behavioral health solutions. Explore opportunities in multiple states.

Hiring MULTIPLE Psychiatrists for Telemedicine and Onsite positions in the following states
CA, TX, NV, AZ, FL, OR, and Many other states. Full time and Part Time positions are available.

We offer competitive salaries and excellent benefits!

Immediate Need for BC Psychiatrist with CA license for several outpatient and inpatient sites

Contact Sandra Williams at 818-814-7790 or email me your current CV to swilliams@alignedth.com

To be considered for these great opportunities.

ARIZONA

Show Low, AZ!

Come practice in the beautiful White Mountains of Arizona! Enjoy a great quality of life, a thriving practice and a rewarding Medical Director position! Once you visit, you will want to stay! Horizon Health, in partnership with Summit Healthcare Regional Medical Center in Show Low, AZ, is seeking a Medical Director for a new 12-bed geriatric inpatient psychiatric program. The Medical Director will provide rounding and treatment on patients for the inpatient program, as well as program administration and oversight services regarding service line policies, practice, development, compliance, and performance improvement. Position will offer a substantial Base Salary, generous Medical Director’s Stipend and a full array of benefits!

For more information contact:
Mark Blakeney, Voice: 972-420-7473, Fax: 972-420-8233; email: mark.blakeney@horizonhealth.com

Cal_MEDICAL SCHOOL

California’s Newest ACGME Medical School
Arrowhead Regional Medical Center, a university affiliated tertiary care center with a burn unit and level 2 trauma center is now the location of California’s newest ACGME accredited medical school. The California University of Science and Medicine. We are seeking to hire a full time teaching attending in the Department of Psychiatry. The hospital is home to 180 residents from almost every specialty and subspecialty including a Psychiatry Residency Training Program. All faculty are actively involved in the education and training of our medical students and residents. An interest in research is a strong plus as the department is increasingly em-
With the continued growth of our Department of Psychiatry and our New General Psychiatry Residency Programs at Ocean Medical Center and Jersey Shore University Medical Center our vision for Behavioral Health is Bright.

As the area’s premier provider of psychiatric services, Hackensack Meridian Behavioral Health Services has provided comprehensive mental health and substance abuse services to the residents of Monmouth, Ocean, Middlesex, and Bergen Counties for over forty years. Due to continued growth and expansion, we are currently accepting applications for Psychiatrists to join our Mental Health and Addiction Interdisciplinary Teams in the following positions:

• Consultation Liaison Psychiatrists: Jersey Shore University Medical Center (Neptune, NJ) and Riverview Medical Center (Red Bank, NJ) and Hackensack University Medical Center (Hackensack, NJ)
• Staff Psychiatrist for Adult Inpatient Unit: Jersey Shore University Medical Center (Neptune, NJ) and Riverview Medical Center (Red Bank, NJ) and Hackensack University Medical Center (Hackensack, NJ)
• 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)
• Medical Director of Adult Inpatient Unit Riverview (Red Bank, NJ)
• Emergency Psychiatry: Raritan Bay Medical Center (Perth Amboy, NJ)
• Geriatric Psychiatry -- Hackensack University Medical Center (Hackensack, NJ)
• Outpatient/Consultation Liaison Psychiatrist -- JFK (Edison, NJ)
• Per Diem/Tele-psychiatry -- Hackensack University Medical Center (Hackensack, NJ)
• Staff Consultation Psychiatry -- Bayshore Medical Center, Holmdel (NJ)

In addition to our collegial work environment, we offer a highly competitive compensation package which includes: medical/dental plans, 403(b) retirement plan, and relocation assistance.

For immediate consideration, please contact Renee Theobald, at: Renee.Theobald@hackensackmeridian.org or call: 732 751-3597 HackensackMeridianHealth.org

Outpatient Adult and Child Psychiatrists are needed for Stanislaus County Behavioral Health & Recovery Services, in the Central Valley less than two hours from San Francisco and Yosemite.

Recovery-oriented treatment provided in a multidisciplinary setting with friendly and dedicated staff members. Recently revised rates with full malpractice coverage and pension plan (PARS) as a Personal Service contract with an income potential of over $325 K per year for adult psychiatrist and over $355 K per year for child psychiatrist for F/T work.

P/T options and the opportunity to combine Tele-Psych with limited onsite work are also available. Excellent work environment with NO Call Requirement, lower than average case load and comprehensive nursing & ancillary support makes this a very pleasant and rewarding opportunity. J1 applicants are welcome.

Fax CV to Bernardo Mora, MD at (209) 558-4326 or Email: bmora@stanbhrs.org

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 cosmopolitan city of Mexicali, Mexico population 1.2 million. San Diego State University maintains a satellite campus in 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.

J-1 applicants welcome.

For additional information, please contact:
Kristen Smith (442)265-1606 kristensmith@co.imperial.ca.us

Due to continued growth and expansion, we are currently accepting applications for Psychiatrists to join our Mental Health and Addiction Interdisciplinary Teams in the following positions:
Join our team!

Are you a psychiatrist looking for a team-oriented, collegiate practice supported by leading experts in psychopharmacology such as Stephen Stahl, MD., Ph.D.? Look no further than the California Department of State Hospitals. We operate the largest forensic psychiatry hospital system in the nation, offering an unparalleled quality of practice while providing care to some of the most complex patients found anywhere.

Email your curriculum vitae to DSH.Recruitment@dsh.ca.gov.

Practice and Benefits:

- Annual salaries to the high $200,000s
- Flexible workweek options may be available
- Voluntary paid on-call duty
- Substantial continuing medical education
- Generous defined-benefit pension
- Psychopharmacology support by leading experts and established protocols
- Medical, dental and vision benefits
- Private practice permitted
- Retiree healthcare
- Psychiatrist-led treatment teams
- Patient-centric, treatment first environment
- Relocation assistance may be available
- Telepsychiatry may be available at some locations

To find out more, please contact Juan Arguello, DO.

(916) 654-2609 • DSH.Recruitment@dsh.ca.gov

www.dsh.ca.gov

ASSOCIATION OF HOSPITALS, HOSPITAL EXPRESS

California Department of State Hospitals

Qualify for a Free Subscription Online @ www.psychiatrictimes.com
Be Part of the Most Noble Mission in Health Care.

The Bay Pines VA Healthcare System is seeking experienced Psychiatrists to join a dynamic team of dedicated, hardworking professionals in providing services in support of America’s Veterans. Full-time positions are available in Geropsychiatry and outpatient psychiatry. Locations include Bay Pines, Cape Coral, Naples, and Port Charlotte.

In addition to giving back to Veterans, you could enjoy:

- Competitive salary
- Work/Life Balance
- 10 Federal holidays, 13 sick days and 26 days of annual vacation
- Education Support
- Relocation/recruitment incentives
- Beautiful Southwest Florida location near some of the country’s top beaches
- No state income tax on earned income
- Relocation/recruitment incentives
- Work/Life Balance
- Professional, in participant psychiatry: commutable from downtown Chicago.

Regional Mental Health Center is a private non-profit mental health center that has successfully served Indiana for over 30 years. Experienced and collegial group of 12 most full-time psychiatrists, an extremely favorable malpractice environment. OP work, call q 12 wks. Regional is a leader in psychiatrist-directed integrated care services. Incentive bonus available, full benefits.

Please contact Kobie Douglas, MD, Kobie.Douglas@Regionalmentalhealth.org 219 736-7232

MISSOURI

MICHIGAN

Sparrow Psychiatry Position with Sparrow Medical Group

Sparrow Medical Group (SMG), a multi-specialty physician group and the premier physician organization of Sparrow Health System (SHS), located in Lansing, Michigan, is seeking a dynamic BC/BE psychiatrist for an adult inpatient position. Position is hospital-employed and offers excellent compensation and benefits including relocation assistance, 401(k) with matching funds, generous CME benefits and malpractice insurance that includes tail coverage.

Learn more about this position by contacting: Barbara Hilborn, Manager Provider Recruitment Office: 1.800.968.3225 Email: barbara.hilborn@sparrow.org

Visit our website at www.sparrow.org

More information on the Lansing area can be obtained at www.lansing.org

MEDICAL DIRECTOR - BRAND NEW ADOLESCENT 15-BED INPATIENT PSYCHIATRY UNIT OPENING IN 2019 - Small Town. Big Opportunity - Be in on the beginning of a new unit helping to mold and develop the program. Open to employment, or independent contractor arrangement. Located in southeast MO near Cape Girardeau, this is a low cost of living, low crime rate area but close to a local airport that has direct flights to Chicago. It’s also only two hours from Memphis and St. Louis. This designated underserved area is also located in the Delta Regional Authority so J1 Waivers can also be obtained through the DRA as well as the state. Position can be inpatient, or inpatient and outpatient.

Please contact Terry Good, Horizon Health, at 804-684-5661; terry.good@horizonhealth.com; Fax: 1-804-684-5663.

Compass Health Network, is a large non-profit health system delivering Behavioral Health services in multiple settings, both inpatient and outpatient in forty-nine Missouri counties. We have immediate openings for full and part-time Psychiatrists in multiple locations in Missouri. Candidates must have MD or DO degree, be ABPN board-certified or eligible in Psychiatry and possess or obtain a Missouri license. We offer a competitive compensation and benefit plan.

Apply online at www.compasshealthnetwork.org or send your CV to cgrigg@compasshn.org.

Candiates with J-1 or H1-b visa status are welcome to apply.

EOE

Texas Health Resources, North Texas behavioral health services, is currently recruiting board eligible/certified psychiatrists for inpatient and outpatient positions in both adult and child psychiatry. We are a growing, vibrant department in a strong academic medical center. We host specialty clinical and research programs, including research that crosses the translational spectrum. Our educational programs include adult psychiatry residency, child fellowship, psychology internship, externship and postdoctoral fellowships. We have a strong collaboration with basic and clinical science in other neuroscience disciplines across several Penn State campuses. With our clinical partner, the Pennsylvania Psychiatric Institute, the Department staffs several outpatient and partial hospital programs for children and adults, 89
Penn Medicine Lancaster General Health Physicians (LGHP) is actively seeking two BC/BE Psychiatrists to join our growing Behavioral Health program. One role is 100% outpatient-based with no night or weekend call responsibilities. Additional compensation will be offered for inpatient call coverage (if interested). This position provides outpatient care exclusively with an emphasis on collaborative care with a comprehensive team of behavioral health clinicians, including Psychiatrists, Psych-Certified Nurse Practitioners and Licensed Clinical Counselors. The Behavioral Health team is supported by a large primary care network. Experience practicing in an innovative outpatient care model is highly preferred. Those interested in contributing to the strategic growth of this model are encouraged to apply. The second position provides liaison duties for the ED and general medical/surgical units within the hospital. The schedule is Monday – Friday, 4pm – 12am plus two weekends per year and occasional holidays/call responsibilities. There will be no inpatient psychiatric unit responsibilities since we have relocated our mental health unit to the Lancaster Behavioral Health Hospital (joint venture with UHS).

COMMUNITY: Lancaster County is a growing and dynamic community. Lancaster offers the unique blend of access to major East Coast cities while providing a family friendly environment with excellent schools and easy access to the shore and mountain areas. There are five institutions of higher learning in the area to meet the needs of you and your family. The cities of Baltimore, Washington DC, Philadelphia, and New York are just 1.5 – 3 hours away.

REQUIRED QUALIFICATIONS:
- Medical Doctor (MD), Doctor of Osteopathy (DO), issued by the Accredited Council on Medical Education.
- Certified by the American Board of Psychiatry or Board eligible.
- Current Medical licensure issued by PA State Board of Medicine or the PA State Board of Osteopathic Medicine.
- Successful completion of Residency.
- A health care provider in good standing with Medicare, Medicaid, and other federal and state health insurance programs, i.e. not excluded from participation in Medicare, Medicaid, or any other federal or state health insurance program.

To Apply: Contact Stew Sampsel at Stewart.Sampsel@penmedicine.upenn.edu

Penn Medicine Lancaster General Health

Psychiatrist Opportunities in NYC!

Jacobi Medical Center (JMC) is a modern, state-of-the-art, Level 1 Trauma Center located in an attractive and safe residential Bronx neighborhood just 20 minutes north of Manhattan. It is a North Bronx Healthcare Network hospital affiliated with North Central Bronx Hospital and a teaching site and academic affiliate of the Albert Einstein College of Medicine. It offers a full continuum of Acute Care Inpatient and Outpatient services in diverse Medical and Surgical Specialties, including Psychiatry. The Department of Psychiatry has 89 Adult Acute Inpatient beds, a Comprehensive Psychiatric Emergency Program (CPEP), a Consultation-Liaison Service, an Adult Ambulatory Practice, and a Community-Based Assertive Community Treatment Program. The department employs evidenced-based best practices in providing the highest quality care to its patients, in a patient-centered approach that is respectful of their individuality, culture, and community.

North Central Bronx Hospital (NCBH) is a modern, state-of-the-art community hospital located in an attractive and safe residential Bronx neighborhood just 20 minutes north of Manhattan. It is a North Bronx Healthcare Network hospital affiliated with Jacobi Medical Center and a teaching site and academic affiliate of the Albert Einstein College of Medicine. It offers a full continuum of acute care inpatient and outpatient services in diverse Medical and Surgical specialties, including Psychiatry. The NCBH Department of Psychiatry has 70 Adult and Geriatric Acute Inpatient Beds, a Partial Hospital Program, Psychiatric Emergency Consultation-Liaison Service, an Adult Ambulatory Practice, and a community-based Assertive Community Treatment Program. The department employs evidenced-based best practices in providing the highest quality care to its patients, in a patient-centered approach that is respectful of their individuality, culture, and community.

Jacobi Medical Center & North Central Bronx Hospital are currently accepting applications and referrals for the following opportunities:

- Inpatient Attendings (JMC and NCB)
- Attending Psychiatrist E/PEP (JMC)
- Inpatient Unit Chief (JMC)
- Child Psychiatry CPEP
- Director of Psychiatry Emergency Services (NCB)

An academic appointment at Albert Einstein College of Medicine is offered. We offer a generous income package along with outstanding benefits, opportunities for advancement, retirement plans, malpractice, and much more!

For immediate confidential consideration, please contact: Carmen Velez – Office of Physician Recruitment: Velezcc@pagny.org 646-494-7559

www.pagny.org

We have opportunities in our inpatient setting for Psychiatrists for our Adult Admissions and Geriatric Units. These positions are employed positions offering a competitive salary with generous state benefits and paid malpractice insurance, loan repayment, CME stipend/leave, sign-on bonus, and relocation allowance–an on-call required, with compensated on-call available.

If you are licensed or eligible for licensure in Virginia, and have completed a psychiatric residency, please send your current CV to kim.sayers@dbhds.virginia.gov or you may contact a member of our Human Resources staff at 276-783-1204 to discuss this opportunity.

We invite you to join a team of dedicated physicians and loyal staff who are committed to promoting a life of possibilities for all Virginians.

For more information, please visit:

- www.swvnhm.dbhds.virginia.gov
- www.smithcounty.org
- www.abingdon-va.gov

TENNESSEE

EAST TENNESSEE STATE UNIVERSITY
QUILLEN COLLEGE OF MEDICINE
DEPARTMENT OF PSYCHIATRY & BEHAVIORAL SCIENCES

ADULT PSYCHIATRISTS, CHILD PSYCHIATRISTS

Two full-time positions available for Adult Psychiatrists who are BE/BC at time of hire, and/or Child Psychiatrists who are BE/BC at the time of hire in the subspecialty of Child and Adolescent Psychiatry. Positions may include inpatient and/or outpatient. Program activities include clinical care of patients combined with teaching and supervision of residents and medical students. Research and academic activities are strongly encouraged. Salary and academic rank are commensurate with experience and qualifications. Salary is competitive with funding available through the Medical School and other sources.

ETSU is located in Johnson City, TN, which has the perfect blend of four mild and beautiful seasons, gentle mountains, a local theater, and a symphony orchestra. Come explore this idea family location with college urban sophistication surrounded by national forests and beautiful parks. No state income tax, low cost-of-living, low crime rate, golf courses, and lakes.

Apply to the position at https://jobs.etsu.edu. Telephone inquiries should be made at (423) 439-2235 or e-mail at loveday@etsu.edu. AA/EOE.

WISCONSIN

PSYCHIATRIST

Clinical excellence and quality living! Winnebago Mental Health Institute (WMHI) is a 280 bed psychiatric facility associated with Medical College of Wisconsin’s North East Wisconsin Psychiatry Residency. We are seeking a Board Certified/Board Eligible Psychiatrist who wants to work with a Multidisciplinary Treatment Team to treat acutely ill Civil Patients and/or Forensic Patients. A strong commitment to excellence in clinical care and education of Residents, Medical Students and students/interns of all other clinical specialties makes WMHI a great place to practice. Excellent fringe benefit package, strong collegial support, paid call, and a beautiful campus enhance your work days.

WMHI is located near Oshkosh, Wiscon-sin, which is the center of the Fox River Valley, one of the fastest developing areas of Wis-con-sin. Four seasons with all the outdoor opportunities of each, cultural and sports venues, outstanding public and private schools and three universities in the area make this a great place to raise a family. In 1 1/2 hours you can be in Milwaukee, Madison, the Wisconsin Dells or “up north”.

Information on WMHI can be found at http://www.dhs.wisconsin.gov/ WMH_Winnebago/.

For application instructions, go to www.wisc.jobs and search for Psychiatrist (Job Announcement Code: 17-02966). EOE.