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Bachaar Arnaout, MD
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IN THIS ISSUE

Focus of the Month:
Traumatic Brain Injury and Addiction

Learning Objectives
After reading these articles, you should be able to:

1. Identify ways for clinicians to assess and treat patients with co-morbid addiction and traumatic brain injury (TBI).

2. Describe some of the challenges in treating substance misuse in patients with co-occurring TBI.

3. Summarize some of the current findings in the literature regarding psychiatric addiction treatment.

Traumatic Brain Injury: Definition, Classification, and Management

We hear a lot about traumatic brain injury (TBI) nowadays: among NFL players (as in the movie 'Concussion'), and as a signature diagnosis among recent combat veterans. What doesn't get as much press coverage is the impact of TBI on those suffering from addiction. Having an alcohol or other substance use disorder greatly increases the risk of TBI. But what is TBI? How do I diagnose it? How does it manifest? How do I manage it?

Defining TBI
Although there is no universally accepted definition for TBI, recently updated guidelines from the Department of Veterans Affairs (VA/DoD Clinical Practice Guideline for the Management of Concussion-mild Traumatic Brain Injury, 2016; https://tinyurl.com/y8e6owdx)

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In Summary

- The severity of a TBI is determined by the symptoms immediately following the injury.
- Most mild TBI cases resolve within 30 days without any intervention; patients with ongoing symptoms may benefit from a targeted referral.
- Medication treatment can help manage neuropsychiatric symptoms after a TBI which can also be attributed to a comorbid, major depressive disorder or substance use disorder.

Q&A With the Expert

Assessing Traumatic Brain Injury in Patients with Substance Use Disorders

John D. Corrigan, Ph.D, ABPP

Professor at Ohio State University and Director of the Ohio Valley Center for Brain Injury Prevention and Rehabilitation. Editor-in-Chief, Journal of Head Trauma Rehabilitation.

Dr. Corrigan has disclosed that he has no relevant financial or other interests in any commercial companies pertaining to this educational activity or other interests in any commercial companies pertaining to this educational activity.

CATR: A lot of patients struggling with addiction have a history of head trauma. How would I go about knowing whether the person had an actual traumatic brain injury (TBI)?
Dr. Corrigan: The only way to know is to ask in a systematic way. You can't count on behavioral manifestations or on seeing a scar on the head. It might be a quite remote injury; maybe one that was experienced in childhood, so there is really no substitute for asking. And it makes a difference how you ask. There have been several attempts in research and clinical practice to use questions like "Have you ever had a traumatic brain injury?" which is essentially asking the person to self-diagnose.
CATR: What approach do you recommend instead?
Dr. Corrigan: There are standardized measures that you can use, including one we have developed called Ohio State University TBI Identification Method (OSU TBI-ID). It is available for free and includes online training

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Traumatic Brain Injury: Definition, Classification, and Management

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state that a TBI is an injury to the brain caused by an external force accompanied by one of several clinical signs following the event). These signs can be an intracranial lesion, loss of consciousness, amnesia, confusion, slowed thinking, muscle weakness, sensory loss, or another neurological deficit. The severity of a TBI (mild, moderate, or severe) is determined by the symptoms immediately following the injury (see the VA TBI severity table on page 3. If the patient meets different ratings for the different criteria, go with the more severe rating. The lay term "concussion" equates to a mild TBI. In addition to the neurological symptoms, patients may experience cognitive problems affecting their attention, memory, processing speed, and executive function. Mental health effects include irritability, impulsivity, depression, and anxiety. However, these symptoms can be an effect of the TBI or part of a comorbid, major depressive disorder, posttraumatic stress disorder (PTSD), or substance use disorder.

Assessment and treatment

But what symptoms should I really be concerned about? For any TBI that is associated with progressively declining neurological function or worsening headache, pupil asymmetry, seizures, intractable vomiting, ongoing disorientation or neurological deficit, slurred speech, or new bizarre behavior, you should immediately refer for emergency evaluation.

The good news is that the vast majority of mild TBI cases resolve without any intervention. It's important for the physician to provide education and reassurance to the patient and family. Any interventions should be tailored to the specific symptoms while reinforcing good sleep hygiene, relaxation techniques, and limiting use of caffeine, tobacco, and alcohol. Return to normal functioning at work or school should be encouraged in a gradual, monitored fashion. Patients with a TBI who report ongoing symptoms need appropriate referral and a comprehensive treatment plan (Silver JM et al, *Textbook of Traumatic Brain Injury,* American Psychiatric Publishing, Inc; 2nd ed;2011).

Cognitive rehabilitation therapy (CRT)

You may have heard of cognitive rehabilitation therapy (CRT) as a treatment for TBI. But what exactly does it involve? And does it work? After a TBI there may be functional deficits that are both physical and mental in nature. CRT is a therapeutic process structured to improve the patient's functioning in their daily lives. Patients are first guided through recognizing their strengths, weaknesses, and what deficits they want to improve. Then techniques are relearned when possible (solve the problem), or compensatory strategies are identified (work around the problem). The last step is to incorporate these relearned or new skills into daily life. This process can be applied to both physical and cognitive deficits that arise from a TBI.

CRT sessions should be tailored to the individual but most incorporate memory compensation techniques. Such techniques include having the patient write down at each session what was important to them, then reviewing their notes and memory of what was said during the next session. This method not only increases their participation in the therapy sessions but teaches them how to use the memory compensation techniques in their daily lives.

The evidence for CRT after stroke and moderate to severe TBI has long been established, showing improvement in the domains of memory, attention, and communication (Cicerone KD et al, *Arch Phys Med Rehabil* 2005;86(8):1681-1692). However, for mild TBI, CRT remains more controversial as there isn't strong evidence for improved functional outcomes. The 2016 VA clinical guidelines recommend short-term CRT for moderate to severe TBI and discourages prolonged treatment courses without measurable improvements.

Sometimes the most concerning symptoms the patient will come to us for are the cognitive deficits and they may press for neuropsychological (NP) testing early. However, NP testing should not be done in the first 30 days. Most cognitive deficits of mild TBI will improve within this time period. And if the problems last longer than 30 days, NP testing may be helpful. Whenever referring for NP testing, be specific in why you are making the request. A targeted referral allows the NP examiner to choose the right tests to provide the most useful information.

Pharmacologic treatment

When considering medication treatment for symptoms following a TBI, there are several general guidelines to follow (Silver JM et al, *Neurology*

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Traumatic Brain Injury: Definition, Classification, and Management

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Classification of TBI Severity
(If a patient meets criteria in more than one category of severity, the higher severity level is assigned)

Criteria	Mild	Moderate	Severe
Structural imaging	Normal	Normal or abnormal	Normal or abnormal
Loss of Consciousness (LOC)	0-30 min	>30 min and <24 hours	>24 hours
Alteration of consciousness/ mental state (AOC)[1]	up to 24 hours	>24 hours; severity based on other criteria	
Posttraumatic amnesia (PTA)	0-1 day	>1 and <7 days	>7 days
Glasgow Coma Scale (GCS) (best available score in first 24 hours)[2]	13-15	9-12	<9

[1]Alteration of mental status must be immediately related to the trauma to the head. Typical symptoms would be: looking and feeling dazed and uncertain of what is happening, confusion, difficulty thinking clearly or responding appropriately to mental status questions, and being unable to describe events immediately before or after the trauma event.
[2]In April 2015, the DoD released a memorandum recommending against the use of GCS scores to diagnose TBI. See the memorandum for additional information. Department of Defense Instruction. DoD policy guidance for management of mild traumatic brain injury/concussion in the deployed setting. 6490.11: Department of Defense; September 18, 2012. Updated April 2015
Source: https://www.healthquality.va.gov/

2006;67(5):748-755.2011). Again, most symptoms of a mild TBI will abate within a month, so watchful waiting and reassurance are important. Symptom improvement may continue throughout the first year as the brain continues to heal, so be sure to reassess the need for the medication intervention. Many times, the neuropsychiatric symptoms after a TBI can be complicated by concurrent major depressive disorder, PTSD, or a substance use disorder. Untreated depression can be the root cause of cognitive problems, irritability, sleep disturbance, fatigue, and headache. Be sure to perform a thorough psychiatric assessment so that you can tailor the treatment plan accordingly. Target specific symptoms or concurrent conditions with your medication choices. After a TBI the brain can be more susceptible to side effects of medications, underscoring the importance of "starting low, and going slow."

Here are a few specific medication recommendations to target neuropsychiatric symptoms (Silver JM et al, 2011). For improving processing speed, methylphenidate has the most evidence. Donepezil and rivastigmine also may have some utility for treating memory impairment. When targeting depression and anxiety, SSRIs are first-line and choose a specific SSRI based on side effect profile and limiting medication interactions (sertraline, citalopram, and escitalopram are favorable choices) (Salter DL et al, *J Head Trauma Rehabil* 2016;31(4):E21-32). Be cautious with bupropion due to increased seizure risk. Caution is also advised with typical antipsychotics as they may inhibit neuronal recovery, and also benzodiazepines due to the memory impairment effects. For controlling mania or irritability, valproate is preferred due to its anti-seizure effect as well as having less cognitive side effects in long term treatment than other mood stabilizers (carbamazepine or lithium). Atypical antipsychotics may also be helpful in controlling irritability especially when combined with psychosis, and are preferred over typical antipsychotics. More recent research shows beneficial effects of amantadine in treating aggression from TBI even 6 months post-injury and more studies are evaluating its use in the acute phase after a severe TBI (Hammond FM et al, *J Head Trauma Rehabil.* 2017;32(5):308-318).

CATR VERDICT: When treating patients with TBI, always remember that the brain has a great capacity for plasticity and recovery. Encourage patients to see their treatment as a process and journey. Take care to evaluate for comorbid mental health disorders, and handle accordingly. Those with substance use disorders, whether existing pre-TBI or newly occurring, should be encouraged to enter into treatment promptly. With the right combination of cognitive rehabilitation, pharmacotherapy, and a good therapeutic alliance, your patients can make great strides in recovery after a TBI.

Expert Interview

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(See: http://ohiovalley.org/tbi-id-method/).What we first do is ask about a lifetime history of injuries by reminding people about hospitalizations; emergency room visits; and times they fell, were in a fight, or in a car crash. All this to remind them of ways they might have gotten a TBI. And if they say, "Oh yeah, I had one of those injuries." Then you say, "Now remember the bike crash you said you had in 8th grade, were you knocked out or did you lose consciousness from that?" So, you systematically interview to get at the data. It does sound a little long, but a typical administration of the OSU TBI-ID is about three to five minutes. So the bottom line is that you need to ask, and the way you ask makes a difference in terms of what you are going to find out.

CATR: What other sources of information do you find useful?

Dr. Corrigan: Well, if you have collateral input from someone who knows the person then you can include them in the interview. And, of course, you can review medical records. You cannot trust medical records, however, because there are so many TBIs that are untreated or undocumented. And what we're learning is that you should think about TBI less like a broken bone and more like lead paint. It is lifetime exposure that makes the difference, and we don't really have medical records that go for your entire lifetime.

CATR: What about imaging or neuropsychological testing?

Dr. Corrigan: They can be useful, but the thing to remember is that they have attenuated sensitivity. So a "yes" on one of those methods is a "yes." but a "no" is actually a "maybe." In other words, you can have a history that you should be concerned about along with a perfectly clean-looking MRI, fMRI, or even neuropsychological assessment. They are sensitive to more severe or specific kinds of injuries, but they don't get the full lifetime exposure. The starting point really needs to be a systematic interview.

Expert Interview
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CATR: Could you tell us about the intersection with addiction—how common is TBI in people who misuse substances?
Dr. Corrigan: We don't have definitive general population rates, but several statewide surveys suggest that perhaps 20% of adults have had at least one TBI with loss of consciousness in their lifetime. In studies done in addiction treatment settings, that number is more like 50%. And the prevalence is greater among clients in higher levels of care. We did a large study in dual-diagnosis programs and found that 80% of those individuals had a history of TBI with loss of consciousness (McHugo GJ et al, *J Head Trauma Rehabil* 2016). And if you consider moderate and severe TBI, so at least 30 minutes loss of consciousness, then you'd expect a prevalence of 2% to 3% in the general population and about 20% in treatment settings—that's one in five with at least moderate to severe TBI. Another issue is that if you have both addiction and TBI, then you can expect to have some other psychiatric disorders as well, which makes treatment even more complicated.

"It simply is going to take longer before you can be confident that the person with TBI is not going to relapse because their insight alone will not assure abstinence. Their conscious commitment to sobriety may be just as good as the person without TBI, but they are going to be more vulnerable to relapse and have more problems staying sober for a longer time."

John D. Corrigan, Ph.D, ABPP

CATR: Is it fair to say that the relationship is bidirectional—that substance misuse can lead to trauma, and that, in turn, TBI is a risk factor for addiction?
Dr. Corrigan: Systematic reviews have typically concluded that substance misuse leads to TBI. That's because if you take a population of folks who are in treatment for TBI, there's a large number who have had substance misuse prior to that. And it's because intoxication leads to injury, and there is evidence that the more intoxicated the person is at the time of an injury, the more likely it is to involve a TBI (Savola O et al, *Alcohol Alcohol* 2005;40(4):269-273). So, no question, misusing substances leads to TBI. And we have some of the population data I mentioned comparing general population rates to persons in addiction treatment, and once you see substance use kicking in, the prevalence rates just skyrocket. So that direction is pretty much unequivocal.
CATR: What about the other direction—TBI leading to addiction?
Dr. Corrigan: The other direction has been harder to get our arms around. For one, there are folks who have a bad injury and stop drinking. They are scared out of it or have insight into what it can mean. Plus, you have folks whose injury is so devastating that they may be in an environment where they don't have easy access to alcohol or drugs. A very interesting part of this question involves looking at childhood TBI and whether or not that may predispose to adult substance misuse. This has been demonstrated in animal studies, and eventually led us to start looking at human data—and there are similar trends in some birth cohorts and large population studies that are finding this relationship between childhood injury and adult substance misuse.
CATR: Very interesting. Do we know the reasons for this predisposition?
Dr. Corrigan: There are a couple of plausible mechanisms. One is neuroinflammation, which is a natural and needed response to acute injury. But that heightened inflammatory state can persist beyond the short term. This is something that is observed in all neurotrauma, not just TBI, and it may be part of what triggers later consequences, like a predisposition to drinking alcohol, which then helps the inflammatory state persist (Weil ZM & Karelina K, *Front Behav Neurosci* 2017;11;135). So, it ends up being kind of a vicious cycle.
CATR: What about the neuropsychiatric consequences of TBI—can they also be a factor in why people misuse substances?
Dr. Corrigan: Yes. TBI can lead to the disruption of the dopaminergic circuitry that plays a part in the development of addiction. And there's also a mechanical effect. What is sometimes not known about TBI is that wherever the blow to the head comes, there typically is contusion in the frontal areas. And that's because once you get enough force that the brain is essentially jiggling within the cranial vault, there is a tendency to scrape across some of the bony ridges in the frontal areas. You also get heightened shearing and tearing in the dense circuitry going from the midbrain and basal ganglia to the frontal lobes. So the frontal lobes are particularly vulnerable, which has a lot to do with not being able to put the brakes on impulsive or disinhibited behavior, and may predispose the person to misuse substances.
CATR: It seems that this may impact treatment, which often is about restoring those brakes.
Dr. Corrigan: Yes, and one of the reasons to screen for TBI in addiction treatment is that it provides another hypothesis about possible causes of behaviors. So, if you know somebody has a history of TBI then you might be looking for problems around attention or processing speed or initiation or impulsivity. And one of my takeaways from years of working in addiction and TBI is that, compared to others in addiction treatment, patients with TBI have a greater disconnect between intention and actual behavior change. Obviously, there is a disconnect for everybody or we wouldn't have so many people in treatment for addiction. But the distance from the intent to change behavior and actual successful behavior change is a bigger for clients with TBI.
CATR: Sounds like this can explain why patients with TBI drop out of treatment.
Dr. Corrigan: True. And if you look at it from the providers' side they will say "They're not compliant." If you ask the patient, they will say, "They don't get me." And I think there are reasons for that. For one thing, we underestimate that treatment has a cognitive load. Whether that's something as simple as participating in groups where there are multiple people to pay attention to or being able to keep something in your head while somebody else is talking. And there are a lot of didactics in treatment, without much attention to the person's learning capacity.

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News of Note

The FDA Campaigns Against Flavored Tobacco Products

Electronic cigarette use, or vaping, is becoming increasingly common. Companies manufacturing e-cigarettes advertise them as alternatives to conventional cigarettes and even a pathway to smoking cessation. However, the popularity of vaping has sky-rocketed in high schools across the country with adolescents becoming addicted to nicotine daily. Monitoring the Future Survey (MTF) (http://monitoringthefuture.org; Miech et al, *N Engl J Med* 2018;379:623-632) results have just been released, and they are quite concerning.

Nearly 14,000 8th, 10th, and 12th graders were surveyed about their use of various substances over the past thirty days. The annual survey extends back to 1975 for 12th graders; the younger ages were added more recently. Over the history of MTF, the single greatest absolute increase in any substance happened this past year—12th graders reporting use of vaped nicotine nearly doubled from around 11% in 2017 to 21% in 2018 (of note, it was only 1.5% in 2011). Overall, there are an additional 1.3 million high school students that vaped in 2018 compared to 2017. Reasons adolescents have given for starting to vape include the sleek, futuristic style of the devices themselves—some look like USB flash drives. Interestingly, when teens were asked what is in their e-cig, 66% said just flavoring, 14% didn't know, 13% said nicotine and the rest said marijuana or other (manufacturers are not required to report e-cig ingredients so users can't easily know what's actually in them). Most e-cigs contain 5% nicotine, roughly the equivalent of a pack of cigarettes with flavors appealing to young people such as crème, cotton candy, gummy bear and bubble gum.

The FDA was initially caught off-guard by the surge in e-cigarette use among high school students. In fall of 2018 the FDA proposed a ban on flavors in e-cigarettes to help curb adolescent vaping. However, the FDA fell short of enacting an all-out flavor ban. Instead, it has proposed requiring flavored vaping products sold in retail stores to be kept in a closed-off area. Vape companies like Juul (who controls over 70% of the e-cigarette market share) are also volunteering their own restrictions. Juul announced in November 2018 that it would curtail its social media presence and stop selling flavored e-cigarettes (except tobacco, mint, and menthol flavors) in retail stores, but they are still available from its website.

While adults may use e-cigarettes as a possibly safer alternative to smoking or a stepping stone to smoking cessation, vaping is proving to be a mixed blessing at best. Some authorities argue that vaping represents a harm reduction approach, relative to the high risk of cancer, lung, and cardiovascular disease associated with tobacco cigarettes. Others point out that the nicotine in vape is just as addictive as that found in regular cigarettes, and that vaping has led to a new generation becoming addicted to nicotine.

—Thomas Jordan, MD and Talia Puzantian, Pharm.D. Drs. Jordan and Puzantian have disclosed that they have no relevant financial or other interests in any commercial companies pertaining to this educational activity.

Expert Interview
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CATR: What are practical ways to address this problem?

Dr. Corrigan: One of the things that we do in our program is to insert people we are working with into the right levels of care and educate their clinicians about TBI. We will get a patient into an IOP, for example, and the second or third day they don't show up or show up late, and the staff ream them for not being motivated. Well, if you have a neurologic injury, missing or being late could be because you forgot to set an alarm, or you weren't organized enough to make your bus. In brain injury rehab, we expect people to miss appointments, so we look for ways that we can assure they attend, like using reminders or setting an alarm in a calendar on their phone. What we look at is as a neurologic issue. While in addiction treatment, it's often looked at as a psychological or motivational issue. This happens so frequently that we try to immunize staff against it. If we know that a patient is disorganized or has a tendency to be hyperverbose, we try to get staff ready for those behaviors, so they are treated as neurologic, not just psychologic issues.

CATR: That's very interesting. What techniques can clinicians use to work with these neurologic aspects?

Dr. Corrigan: We end up having the conversation so often that we've developed a reference booklet for addiction clinicians and any professionals working with persons with TBI (See: http://ohiovalley.org/informationeducation/accommodatingtbi/). Basically, it helps you recognize some of these executive function impairments and then gets you thinking about ways you might accommodate them, either in the relationship or in your treatment planning. For instance, some of the simple things we suggest the counselor try with clients who have attention problems is to use short communications, ask the client to summarize what they just heard, or use written cues. These accommodations add to the skill set of the provider to be able recognize some of these neurologic deficits and then have some ideas about how to compensate.

CATR: This is a very hands-on approach. Do you also recommend engaging the family and other supports to compensate for the patient's deficits?

Dr. Corrigan: Yes, definitely. One of my rules of thumb is that the more severe the brain injury, the less you can count on insight alone resulting in success, and the more you have to use other tools for that individual. So, the professional should think about environmental factors like family, living situation, and relapse prevention medications (though disulfiram is best avoided in people who might impulsively drink on it). Another part of this is that the more severe the brain injury the more time is required for cravings to subside. It simply is going to take longer before you can be confident that the person with TBI is not going to relapse. And, again, that's because their insight alone will not assure abstinence. Their conscious commitment to sobriety may be just as good as

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Research Updates

SMOKING

Does Augmenting Varenicline with Bupropion Work Better than Varenicline Alone?

REVIEW OF: Cinciripini PM et al, *Addiction* 2018;113:1673-1682

We have a good array of smoking cessation treatments to choose from, including nicotine replacement therapy (NRT), bupropion, and varenicline. Varenicline is the most effective monotherapy agent, somewhat better than bupropion and single-product NRT, and comparable to combination NRT. Theoretically, adding bupropion to varenicline would be even more effective. A couple of studies have tested this strategy with mixed results. This latest study attempted to further clarify the efficacy of this combination.

Researchers randomly assigned smokers (at least 1 pack per day) to three treatment arms: varenicline alone (n=166), varenicline plus bupropion (n=163), and placebo (n=56). All participants were also given behavioral therapy (13 in-person individual 15-minute visits for smoking cessation counseling and two brief supportive telephone sessions) for 12 weeks of active treatment. They were then followed for 12 months. The primary outcome measure was abstinence at 1 year, which was verified by measuring expired carbon monoxide. The majority of participants were male (58%), and the average age was 49.

After 12 months, the quit rates were similar in the two active treatment groups. Beginning with the last four weeks of treatment, participants on varenicline had a continuous abstinence rate of 22.29% vs 20.25% for the varenicline+bupropion group. Both of these were superior to placebo, with a continuous abstinence rate of 5.36%

As expected, the rate of adverse events was higher in the combination (98.1%) and varenicline only (95.78%) groups compared with placebo (89.29%, p<0.021). Specifically, varenicline+bupropion participants experienced decreased appetite, altered taste, and increased dry mouth, insomnia, creatinine and edema compared with placebo. Varenicline only participants had increased rates of abnormal dreams, diarrhea, and nausea compared with placebo.

CATR'S TAKE

While it's tempting to combine two effective treatments, it appears that adding bupropion to varenicline is no better than varenicline alone. While all agents can be used as first line treatment, in the Carlat Medication Fact Book, we lay out an approach to smoking cessation that starts with nicotine replacement therapy, and then moves on to either varenicline or bupropion. These results appear to be in line with that approach.

—Jessica Goren, PharmD. Dr. Goren has disclosed that she has no relevant financial or other interests in any commercial companies pertaining to this educational activity.

OPIOIDS

Switching from Buprenorphine to Extended-Release Naltrexone: Does it Work?

Review of: Solli KK et al, *Addiction* 2018;113(10):1840-1849

Extended-release naltrexone (Vivitrol) has had some good data, yet getting patients on it remains a challenge, because an opioid-free period is required before starting it. Understandably, practitioners get nervous when patients stabilized on buprenorphine ask to be transitioned to extended-release (XR) naltrexone. But, if needed, can this switch be made safely and effectively?

To answer this question researchers in Norway conducted an open-label continuation of a previously-reported 3-month controlled trial. In the original study, 159 patients were randomized to up to 24 mg of buprenorphine/naloxone daily or 380 mg of extended-release (XR) naltrexone injection monthly. At the end of three months, participants were offered the option of continuing on XR naltrexone, switching from buprenorphine to XR naltrexone, or treatment with buprenorphine at a program outside the study. Of the 122 participants who completed the first phase, 117 chose XR naltrexone, and five chose buprenorphine outside of the study. XR naltrexone was not commercially available in Norway, which may account for the large number of people choosing it over buprenorphine. The switch was carefully made during a detox admission, where XR naltrexone was initiated after a test dose of naloxone and a minimum of 72 following any opioid intake (which is a lot shorter than the commonly recommended washout period), and adjunctive medications were available to help relieve withdrawal symptoms. Participants were followed for another 9 months, and the primary outcomes were continuation of treatment and abstinence rates for those who remained on XR naltrexone (n=54) compared with those who initiated XR naltrexone (n=63).

Participants were men and women ages 18-60 years with opioid use disorder (DSM-IV opioid dependence) and without alcohol dependence or serious somatic or psychiatric comorbidities. Pregnant and nursing women were excluded. The majority of participants were men (75%) and the mean age was 35.6 years.

9 months later there were no significant differences in outcomes between participants who continued XR naltrexone and those who switched to it from buprenorphine. Twenty-eight (51.9%) participants who were originally on XR naltrexone and 30 (47.6%) who newly started on it completed 9 months of follow-up. Complete abstinence from opioids was self-reported by 53.7% of participants continuing XR naltrexone and 44.4% of those newly started. Adverse events were generally related to withdrawal symptoms. Two patients discontinued XR naltrexone due to serious injection site reactions requiring surgery, after which they recovered completely.

CATR'S TAKE

The results of the study imply that switching from buprenorphine to XR naltrexone may work as well as starting XR naltrexone from scratch. The study was not perfect—the design was open-label, there was no objective confirmation of abstinence, and the switch was carefully done on an inpatient unit, limiting our confidence that it

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CE/CME Post-Test

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Below are the questions for this month's CE/CME post-test. This page is intended as a study guide. Please complete the test online at www.carlataddictiontreatment.com. Note: Learning objectives are listed on page 1.

1. According to Dr. Corrigan, which of the following statements about substance misuse and traumatic brain injury (TBI) is true? (LO #2)

   a. TBI that involves the temporal lobe is more likely to predispose an individual to misuse substances
   b. The rate of adult substance misuse is 20% higher in individuals who've had childhood TBI
   c. Higher intoxication levels at the time of an injury raises the likelihood of a TBI
   d. Patients who were under 25 when their TBI occurred are less likely to have compliance issues in substance use treatment.

2. A 2016 study on dual diagnosis patients with substance use found that _____ of individuals had a history of TBI with loss of consciousness. (LO #2)

   a. 10% b. 40% c. 60% d. 80%

3. Individuals with TBI and co-occurring substance use disorder who present with cognitive deficits should be receive neuropsychological testing within the first 30 days of the injury. (LO #1)

   a. True b. False

4. Which of the following medications is an optimal first-line choice in treating aggression associated with TBI? (LO #1)

   a. Carbamazepine b. Bupropion c. Rivastigmine d. Amantadine

5. In a 2018 study comparing the effectiveness of varenicline and varenicline plus bupropion, patients taking varenicline alone experienced side effects such as decreased appetite, insomnia, and edema. (LO #3)

   a. True b. False

Expert Interview

Continued from page 5

the person beside them without TBI; but, they are going to be more vulnerable to relapse and have more problems staying sober for a longer time. The professionals must assist them to put more supports in place, like medication, family, living environment, and time.

CATR: Can this knowledge about TBI lower frustration among clinicians?

Dr. Corrigan: I think that when clinicians start to recognize that behavior can come from a neurologic basis, not just a psychological basis, their frustration level does go down. For instance, it's not uncommon that a person with a brain injury can be overly talkative, which can be a problem in a milieu-based treatment setting. Hyper-verbosity can bring out the worst in that environment. And not only will the therapist become frustrated because the client is dominating group time, but the other clients can become frustrated as well. Throw in a little social disinhibition and it's easy to see why clients with TBI often get into trouble in treatment settings. But just knowing that this behavior has a neurological source makes hyper-verbosity something to be dealt with therapeutically, not to become frustrated with. It's important that the professional know it is not going to offend a person by pointing out these behaviors—indeed, professionally delivered feedback is often welcome. It is a problem if professionals say nothing and come to the conclusion that the client is doing something intentionally to be irritating.

CATR: You mentioned that groups can be challenging in patients with TBI. Is there a way around this issue?

Dr. Corrigan: In this day and age, if you say no groups then you've really limited a person's access to treatment. There also are benefits of groups, such as hearing other peoples' stories and getting peer feedback. There are simple things you can do to accommodate the person with brain injury in group treatment. I generally recommend that there be some post-group processing, even brief, just to say to the client with a TBI, "Here are the big things I saw go on in the group today. What did you see?" And, if there's homework, seeing if they're using some compensatory strategy to remind themselves, like writing it down or putting a reminder in their phone. So, it doesn't take much, and these strategies are more about an informed and creative therapist who can do a lot for somebody with TBI without expensive bells and whistles.

CATR: Thank you for your time, Dr. Corrigan.

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This Month's Focus:
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can be done as safely and effectively in outpatient settings. Nonetheless, the naturalistic setting is similar to clinical practice, and the 50% self-reported abstinence rate is encouraging. Switching from buprenorphine to XR naltrexone can be attempted in select patients, but we recommend approaching switch requests with great caution. We continue to think of XR naltrexone as a second-line option for patients who cannot be on agonist treatment.

—Jessica Goren, PharmD and Bachaar Arnaout, MD. Drs. Goren and Arnaout have disclosed that they have no relevant financial or other interests in any commercial companies pertaining to this educational activity.