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Victoria Hendrick, MD
Editor-in-Chief

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IN THIS ISSUE

Focus of the Month:
Catatonia

Learning Objectives

After reading these articles, you should be able to:

1. Diagnose and manage psychogenic polydipsia.
2. Identify when ECT may be indicated for catatonia and other psychiatric disorders.
3. Determine appropriate next steps upon learning of sexual contact between patients on the unit.
4. Summarize current research findings on psychiatric treatment.

Management of Psychogenic Polydipsia

Susie Morris, MD. Assistant professor of psychiatry and forensic psychiatrist, UCLA. Los Angeles, CA.

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

Highlights From This Issue

Psychogenic polydipsia is common and potentially life-threatening. We examine how to manage this challenging condition.

When patients with catatonia fail to respond to benzodiazepines, there are additional treatments we can try, argues Dr. Stephan Heckers.

Dr. Stephen Seiner reviews advances in ECT that have made the procedure more effective and less likely to produce cognitive impairment.

How do you handle sexual encounters between patients on the unit? Dr. Husna Najand walks us through steps to address these incidents.

Your new patient came from jail and has a history of schizophrenia. In jail, he was prescribed chlorpromazine and was observed to drink water compulsively, even from his cell toilet. On interview, he seems confused and disoriented, and he urinates on himself. The nurses tell you he has been complaining of thirst and needs to urinate frequently. You order an electrolyte panel and note that his serum sodium is 126 mEq/L.

Psychogenic polydipsia (PP), also known as primary polydipsia and potomania, was first described in the 1930s. It was mainly observed in patients with psychotic disorders who drank too much water, leading to frequent urination and low sodium

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Q&A With the Expert

Assessing and Treating Catatonia
Stephan Heckers, MD

William P. and Henry B. Test Professor Chair, Department of Psychiatry and Behavioral Sciences, Vanderbilt University Medical Center. Psychiatrist-in-Chief, Vanderbilt Psychiatric Hospital. Nashville, TN.

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

CHPR: Welcome, Dr. Heckers. You recently published a review of the management of catatonia (Heckers S and Walther S, *JAMA Psychiatry* 2021;78(5):560–561). What is catatonia and how do you diagnose it?

Dr. Heckers: Catatonia is a psychomotor syndrome. Some patients cannot move or respond normally. Patients often stare blankly, remain motionless for long periods, and don't talk or respond to questions. These presentations are typical of the akinetic form of catatonia, which is the most common. This form is sometimes referred to as retarded, inhibited, or stuporous catatonia, or as the Kahlbaum type of catatonia *(Editor's note: Karl Ludwig Kahlbaum was the first to describe catatonia in 1868).*

CHPR: Are there other subtypes of catatonia?

Dr. Heckers: Yes, there's also an excited, hyperkinetic

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subtype of catatonia, where patients are hyperactive and restless. The third subtype is malignant catatonia, which looks very similar to neuroleptic malignant syndrome (NMS). It can be life-threatening, but fortunately it's very rare.

CHPR: How do you distinguish excited catatonia from the agitation of psychosis or mania?

Dr. Heckers: They can appear similar, but hyperactive catatonic patients typically engage in odd, purposeless, disorganized behaviors.

CHPR: To make sure we don't miss the diagnosis, is it helpful to use a rating instrument?

Dr. Heckers: Yes. We use the Bush-Francis Catatonia Rating Scale (Bush G et al, *Acta Psychiatr Scand* 1996;93(2):129–136). In fact, we use it so frequently that it's now part of our electronic medical record. It measures the degree of immobility and mutism, as well as other features of catatonia, like grimacing, posturing, odd or stereotyped behaviors, hyperactivity, and waxy flexibility *(Editor's note: See the Bush-Francis Catatonia Rating Scale at www.thecarlatreport.com/bushfrancis)*. We also use the Bush-Francis scale to monitor patients' response to treatment.

CHPR: What are the most common underlying causes of catatonia?

Dr. Heckers: Catatonia is most common in mood disorders. But it also occurs in psychotic disorders, neurodevelopmental disorders, and various medical conditions. The excited form of catatonia is more typical of a patient with bipolar disorder than schizophrenia, but catatonia presentations generally appear similar, regardless of the underlying diagnosis.

CHPR: But one difference is that patients with underlying schizophrenia are less likely to respond promptly to benzodiazepines, compared to patients with underlying mood disorders, right?

Dr. Heckers: Most patients with catatonia respond to benzodiazepines, even patients with schizophrenia. More than two-thirds of all patients respond promptly even after a single administration, so benzodiazepines are the treatment of choice even if the patient has a diagnosis of schizophrenia. But patients with schizophrenia, particularly disorganized schizophrenia, often need an antipsychotic to fully recover.

CHPR: Do you worry that antipsychotic-induced extrapyramidal symptoms might worsen the catatonic symptoms or mask any improvement?

Dr. Heckers: Yes. We usually stop the antipsychotic and treat the patient with just a benzodiazepine, but around 20% of patients with catatonia also have a psychotic disorder that will not get better without adding an antipsychotic. So, we begin with a benzodiazepine and, if the patient is getting better but still has significant psychosis in addition to the catatonia, then we add an antipsychotic.

CHPR: I've seen that many psychiatrists are hesitant to use antipsychotics for patients experiencing catatonia.

Dr. Heckers: I understand that concern. Antipsychotics can lead to NMS and muddy the diagnosis: Does a patient have NMS or a severe form of catatonia? Also, antipsychotic-induced extrapyramidal symptoms can exacerbate a catatonic patient's muscle rigidity and reduced movement. But these concerns miss the fact that some psychotic symptoms in catatonic patients—disorganized thought, for example—will not get better without an antipsychotic. We start with a low dose and monitor closely for any new onset or worsening of muscle rigidity.

CHPR: Are patients in catatonic states at greater risk of developing NMS than other patients taking antipsychotic medications?

Dr. Heckers: Yes. Some patients with catatonia can have extremely elevated creatine kinase values, sometimes in the hundreds or thousands. These are typically patients with prominent psychomotor presentations, who posture and are hyperactive. They might have frank muscle damage because they are so hyperactive, and they are the ones most likely to develop severe NMS if you initiate an antipsychotic.

CHPR: And how do we distinguish NMS from malignant catatonia?

Dr. Heckers: Both can present with changes in vital signs (increased heart rate, blood pressure, and body temperature) and high creatine kinase levels. In NMS you're more likely to see lead-pipe rigidity *(Editor's note: See* CHPR*, Jan/Feb/Mar 2021 for more on NMS)*. The patient's medication history aids the diagnosis since patients with NMS will have been taking antipsychotic medications. Also, patients with catatonia are more likely to display waxy flexibility, where you can move a patient's limbs to a new position and they stay in that position.

CHPR: Should we preferentially use any specific antipsychotic medications in catatonia?

Dr. Heckers: We try to avoid the high-potency first-generation antipsychotics, so we use second-generation or third-generation partial

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EDITORIAL INFORMATION

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agonists like aripiprazole. But few studies have compared medications for catatonia, so there isn't compelling evidence that one is better or safer than the other.

CHPR: Please walk us through your treatment steps for catatonia.

Dr. Heckers: Most cases of catatonia get diagnosed in an emergency setting. In fact, catatonia is one of the more common reasons why a person presents to a psychiatrist in an emergency room. We treat with lorazepam (Ativan) 2 mg IM or IV, and patients typically respond very well. The excited form of catatonia often gets misinterpreted as agitation or mania, but when you realize that the patient is in a hyperactive state of catatonia—for example, demonstrating undirected, nonpurposeful motor activity—and treat with lorazepam, they respond quickly. Oral lorazepam is not as effective, and patients are often not able or willing to take PO medication.

CHPR: How quickly do you see a response to IM or IV lorazepam?

Dr. Heckers: Typically in five to 10 minutes—it's the fastest response that we have in clinical psychiatry, akin to stopping a status epilepticus. I have seen patients with waxy flexibility, completely rigid, have a complete resolution with 2 mg of IV lorazepam and become capable of interacting normally.

CHPR: Why do you choose lorazepam vs another benzodiazepine?

Dr. Heckers: IM or IV lorazepam has a fast onset of action, within a few minutes. Case reports have described successful treatment with other benzodiazepines, like midazolam and clonazepam, but most studies of benzodiazepines for catatonia have used lorazepam.

CHPR: What do you do if the patient does not respond to lorazepam?

Dr. Heckers: If they don't improve within half an hour, we add another 2 mg of lorazepam. And if they still don't respond, we add an additional 2 mg 30 minutes later. We call this the Ativan challenge test. Most people respond to 6 mg of lorazepam, but we sometimes need to reach high doses—12, 16, even 20 mg—and might need to treat for two to three days before seeing a response.

CHPR: Are there any risks to this much lorazepam?

Dr. Heckers: We might encounter problems with the patient's therapeutic window, which refers to the amount of lorazepam that will resolve the catatonia versus cause excessive sedation. If the therapeutic window is narrow, patients will fall asleep before we have a chance to see whether the medication is effective. We don't worry about the concerns that come up with phenobarbital and other barbiturates, such as suppression of breathing.

CHPR: What's your next step if the benzodiazepine doesn't work?

Dr. Heckers: When benzodiazepines are ineffective, electroconvulsive therapy (ECT) is our next option. If ECT is not available, alternative treatment options include glutamate antagonists, like amantadine 100–600 mg daily or memantine 10–20 mg daily, and valproic acid 500–1500 mg daily (Beach S et al, *Gen Hosp Psychiatry* 2017;48:1–19). If those still don't work, then we must ask ourselves whether we're missing something.

"Patients with akinetic catatonia often stare blankly, remain motionless for long periods, and don't talk or respond. There's also an excited, hyperkinetic subtype of catatonia, where patients are hyperactive and restless. The third subtype is malignant catatonia, which looks very similar to neuroleptic malignant syndrome. It can be life-threatening, but fortunately it's very rare."

Stephan Heckers, MD

CHPR: Do you change your management if the catatonia is mild vs severe, or akinetic vs hyperkinetic?

Dr. Heckers: Less severe forms of catatonia usually improve with lower doses of benzodiazepines compared to more severe forms, but our management otherwise remains basically the same. This remains true for akinetic vs hyperkinetic forms of catatonia. They both respond to benzodiazepines, but hyperkinetic manifestations may require higher doses.

CHPR: And what's on your differential?

Dr. Heckers: In addition to mood and psychotic disorders, we consider delirium, various systemic and toxic conditions, and neurologic illnesses like strokes or brain-occupying lesions. Patients with autoimmune encephalitis—particularly anti-NMDA-receptor encephalitis—can present with catatonia, usually before they develop more severe forms of encephalopathy.

CHPR: What tests would you want to obtain in your workup?

Dr. Heckers: Depending on the patient's clinical presentation, we might want to obtain brain imaging. We'll also want a toxicology screening and routine blood tests like a comprehensive metabolic screening and CBC. A lumbar puncture will be necessary if encephalitis is a possibility.

CHPR: For catatonic patients who don't respond promptly, are there medical sequelae we should watch for?

Dr. Heckers: Most cases with catatonia are not so severe that patients would develop a deep venous thrombosis, a pulmonary embolism, pressure ulcers, or severe dehydration, but these do happen. Therefore, we hold that a patient who is not moving needs to be treated like a patient who is unable to move for neurological reasons to prevent the consequences of prolonged immobility.

CHPR: And once the patient's catatonic state has resolved, how long do you maintain the lorazepam?

Dr. Heckers: If it's an acute episode of catatonia that responds well to a benzodiazepine, we stop

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the benzodiazepine at the end of the hospitalization. Sometimes we discharge the patient with a taper over the course of a month, during which we slowly take them off the benzodiazepine. But some patients will need to be maintained for several months on a slow taper of lorazepam. We might discharge the patient on 2 or 3 mg, typically broken down into one or two doses over the day, and then very slowly taper it off over several months.

CHPR: How do you know which patients will need a taper?

Dr. Heckers: We'll do what's referred to as the negative challenge test: We take the patient off the benzodiazepine while they're still on the inpatient unit. If we see a rapid resurgence of the catatonic presentation, that indicates that the patient will need maintenance or a very slow outpatient taper.

CHPR: So, some patients basically need to be on maintenance treatment to avoid this relapse?

Dr. Heckers: I'm taking care of patients who need long-term maintenance with benzodiazepines. Some need maintenance ECT because when we stop the treatment, the patient will have a relapse of catatonia.

CHPR: What do we know about the pathophysiology of catatonia?

Dr. Heckers: Very little. It's striking because catatonia is one of the most treatment-responsive conditions in psychiatry. There are two theories. The first focuses on the motor circuit in the brain and makes the case that catatonia is an abnormality of motor planning. The second centers on the affective charge and the heightened anxiety often seen in patients with catatonia, akin to a severe panic attack, with the idea being that people are frozen out of fear. This theory proposes that the limbic system, the orbitofrontal cortex, and the amygdala are driving a person into a severe form of anxiety, which in turn produces the catatonia. From a pathophysiologic perspective, these are two very different mechanisms and very different brain regions. Neuroimaging has not provided a definitive answer. We have animal models for some features of catatonia, but they have not provided new clues for interventions yet. The pathophysiology is remarkably unclear.

CHPR: You made a point in your paper about mild forms of catatonia often going unrecognized. Can you say a little more about this?

Dr. Heckers: A mild form of catatonia is negativism, which Herman Melville succinctly captured in the phrase "I would prefer not to." It is willful avolition, not quite the same as the lack of will and motivation that we recognize as a negative symptom of schizophrenia. Many psychiatrists reserve the diagnosis of catatonia for the complete inability to engage, but that is a misconception.

CHPR: Any final thoughts?

Dr. Heckers: I'll end by saying that I find Eugen Bleuler's concept of ambivalence to be very relevant to catatonia. Among Bleuler's "four A's" of schizophrenia, Autism, Affect, and Association made perfect sense, but the fourth—Ambivalence—eluded me for many years, until I realized that Bleuler was referring to catatonia. He gives an example in one of his books: *Dementia Praecox or the Group of Schizophrenias* (Madison, CT: International Universities Press; 1950). He goes on his morning rounds and sits down at a patient's bed and addresses her, saying "I am Dr. Bleuler. How are you doing?" The patient says nothing and doesn't move. But when he leaves the room, the patient says "Dr. Bleuler, I want to talk with you." He describes it as an example of ambivalence: When you engage, the patient doesn't respond, but when you turn away they give you a sign, either saying or doing something to indicate that they're willing to engage. This ambivalence, I've realized more and more, can indicate a subtle form of catatonia.

CHPR: Thank you for your time, Dr. Heckers.

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Management of Psychogenic Polydipsia

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levels. The cause is unknown, but these patients may have an acquired defect in hypothalamic thirst regulation. Several psychotropic medications are believed to cause and/or exacerbate PP, possibly triggered by their anticholinergic effects that produce an uncomfortable sensation of dry mouth. These drugs include phenothiazines like chlorpromazine, SSRIs, carbamazepine, oxcarbazepine, haloperidol, MAOIs, amitriptyline, and valproate (Dundas B et al, *Curr Psychiatry Rep* 2007;9(3):236–241).

Diagnosing PP

PP is surprisingly common, with a prevalence of 3%–25% in institutionalized patients (Iftene F et al, *Psychiatry Res* 2013;210(3):679–683). It is most commonly associated with schizophrenia, with an incidence of 11%–20%, but it also occurs in patients with other psychotic, mood, and anxiety disorders (Sailer C et al, *Swiss Med Wkly* 2017;147:w14514). PP has been observed in restrictive eating disorders, most likely from poor nutrition. Inadequate nutrition also places patients with substance use disorders at risk for PP. For instance, "beer potomania," aptly named for drinking to the exclusion of proper nutrition, results in low sodium and other electrolyte abnormalities typical of PP. Some users of MDMA or ecstasy can also develop PP.

Two other conditions also cause polydipsia and polyuria and should be ruled out before diagnosing PP: diabetes mellitus and diabetes insipidus. Here are the differences in pathophysiology:

- In PP, the primary problem is that the patient is drinking too much water. This leads to a dilution of the blood, and a chemistry panel will show low sodium (< 135 mEq/L) as well as low serum osmolality. In addition, the urine will be diluted, with a urine osmolality < 100 mOsmol/kg

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and a low urine sodium (*see "Differential Diagnosis of Polyuria and/or Hyponatremia" table below for more information*).

- In diabetes mellitus, the primary problem is hyperglycemia, which leads to polyuria because excess glucose is being dumped into the urine, drawing excess water along with it via osmotic diuresis. The excess thirst is a result of the dehydration caused by the polyuria. The key diagnostic features, predictably, are hyperglycemia and glucosuria (glucose in the urine)—neither of which are expected in PP.
- In diabetes insipidus, the primary problem is neither excess water intake nor hypoglycemia, but lowered secretion of, or lowered response to, ADH (antidiuretic hormone). In nephrogenic diabetes insipidus, ADH secretion from the brain is normal, but the kidneys are less sensitive to ADH. It is sometimes caused by long-term lithium use and consequent chronic kidney disease. As in PP, you'll see a dilute urine, but unlike PP, serum sodium will be high as the serum is concentrated from free water loss.

Finally, it's important to keep one other condition in mind, because it occurs frequently in elderly patients taking psychiatric drugs: syndrome of inappropriate antidiuretic hormone secretion (SIADH). In SIADH, the primary problem is too much ADH in the system, caused by various medications including oxcarbazepine, carbamazepine, and serotonergic antidepressants. The kidneys are being told to absorb excessive water, so the serum is dilute, but the urine is concentrated (unlike PP). Risk factors include being elderly, female, or underweight. If a patient is on an SSRI, switch to a non-serotonergic antidepressant like bupropion or mirtazapine.

Managing PP

The most important treatment strategy for PP is fluid restriction (1000–1500 mL/day), which sounds simple in theory but is more complex in practice. Theoretically, you can write an order in the chart to limit a patient's fluid intake to a certain amount, but that's difficult to enforce on a busy inpatient unit since patients will have access to a bathroom. Some patients may find surreptitious ways of drinking water (eg, from the toilet or sink). If you suspect this is occurring, they may need 1:1 supervision. Have nursing attendants monitor patients by keeping the door ajar when they use the bathroom.

At any rate, if water is successfully restricted, serum sodium will improve rapidly. If the patient's sodium remains low, you can supplement with sodium chloride tablets, 1–3 g daily.

Differential Diagnosis of Polyuria and/or Hyponatremia

Diagnosis	Primary Problem	Thirst	Serum	Urine	Management
Diabetes insipidus	Less ADH	Elevated	Normal or elevated Na+	Low osmolality	Desmopressin
Diabetes mellitus	Hyperglycemia	Elevated	Hyperglycemia Na+ can be low, normal, or elevated	Glucosuria	Diabetes medication
Psychogenic polydipsia	Increased water intake	Elevated	Low Na+ Normal glucose	Dilute, osmolality < 100 mOsmol/kg Urine Na+ < 10 mEq/L	Fluid restriction
SIADH	Too much ADH	Normal	Low Na+ Osmolality low, < 275 mOsmol/kg	Osmolality concentrated, > 100 mOsmol/kg Urine Na+ > 20 mEq/L	Fluid restriction, stop responsible medication

You should also try to determine if any of the patient's medications might have triggered or worsened PP—these are typically anticholinergic antipsychotics that cause dry mouth, such as chlorpromazine, haloperidol, or tricyclic antidepressants.

In severe cases of PP—when serum sodium drops to the low 120s or below—patients may present not only with delirium, but also with seizures and obtundation. Serious cases of PP can be fatal, due to cerebral edema and central herniation. Patients with sodium levels < 120 mEq/L and/or serious symptoms require transfer to a medicine unit for closely monitored sodium repletion using intravenous saline (a 3% saline solution).

To prevent recurrent episodes of hyponatremia, patients may need to have sodium levels checked at regular intervals depending on the severity of their PP; you can prescribe daily sodium supplements as needed. Also, behavioral interventions and group therapeutic strategies have shown promising results in preventing relapses (Sailer et al, 2017). Nicotine dependence, a common comorbidity in patients with schizophrenia, can place patients at risk for PP, so smoking cessation can be helpful. Small studies have reported that the opioid antagonist naltrexone, used 50 mg daily as an adjunct to an antipsychotic, reduces compulsive drinking (Rizvi S et al, *Cureus* 2019;11(8):e5320).

Your patient's urine does not show evidence of glucosuria, further confirming your diagnosis of PP. You stop the patient's chlorpromazine and restrict his water intake by withholding water overnight. When you recheck his sodium in the morning, it's risen to 133 mEq/L.

CHPR VERDICT: Low sodium and dilute urine almost always indicate psychogenic polydipsia. Restrict water overnight, stop any potentially offending agents, provide sodium chloride supplements, and recheck serum sodium in the morning. Some patients require 1:1 supervision to restrict water consumption. In severe cases, transfer your patient to the medical unit for IV saline repletion. The opioid antagonist naltrexone may help reduce compulsive water intake.

Q&A With the Expert

Electroconvulsive Therapy: A Primer

Stephen J. Seiner, MD

Director of neurotherapeutics, McLean Hospital. Belmont, MA.

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

CHPR: Welcome, Dr. Seiner. Can you tell us what led to your interest in electroconvulsive therapy (ECT) and neurotherapeutics?

Dr. Seiner: I went to the University of Michigan and got a degree in chemical engineering, but I decided I didn't want to fractionate oil for a living, so I went to medical school thinking I would be an internist. While in medical school, I absolutely fell in love with psychiatry, but I had a hard time giving up the white coat. I did a psychiatry residency here at McLean Hospital and then a fellowship in geriatric psychiatry, which included ECT training. There was a big need for ECT, so I stayed on and became the director of McLean's neurotherapeutics program. Our program has expanded to include TMS, ketamine infusions, and esketamine and is now one of the busiest neurotherapeutics programs in the country.

CHPR: How has the use of ECT changed in the last few years?

Dr. Seiner: ECT is moving away from small, community-based hospitals and toward academic centers and larger programs. Many smaller programs have closed, but academic centers like ours are busier than ever. When I started doing ECT treatments around the year 2000, we performed 2,500 treatments annually, and now we're doing about 10,000. When you get large enough, you can offer more outpatient ECT, and ECT is moving much more to the outpatient world.

CHPR: Which patients are the best candidates for ECT?

Dr. Seiner: Many severely ill patients, like patients with psychotic depression or catatonia, respond well to ECT. ECT for catatonia works 80%–90% of the time. In psychiatry we don't have a lot of 80%–90% anything, so I think ECT will be around for a long time because it's so effective for the most severely ill patients. Also, ECT works well for treatment-resistant depression, schizoaffective disorder, bipolar depression, and delirious mania.

CHPR: For depressed patients, how do you decide whether to use ECT vs TMS?

Dr. Seiner: For moderately to severely depressed but treatment-resistant patients, ECT is still the gold standard, but TMS is a reasonable alternative. Some patients prefer ECT because it works more quickly, but others prefer TMS because they're scared of ECT or don't like going under anesthesia. And patients who work might not be able to take the time off for ECT. TMS, in contrast, is very convenient because it's a half-hour treatment and patients can come during their lunch break or at the beginning or end of the day.

CHPR: Where does ketamine fit in?

Dr. Seiner: There's a really good case to be made for patients who are severely ill and suicidal to try ketamine first. It'll either work quickly, within a couple of treatments, or it won't, in which case they can move on to ECT.

CHPR: Is ECT more effective than ketamine?

Dr. Seiner: Not necessarily; some patients respond better to one, some better to the other. Overall, response rates seem similar, but they've never been compared head-to-head. There's a big multinational study going on, the ELEKT-D study, which is comparing both treatments and is expected to wrap up later this year (Mathew SJ et al, *Contemp Clin Trials* 2019;77:19–26). However, ketamine is not considered appropriate at this time for psychotic depression, catatonia, or delirious mania. So, we would still send those patients right to ECT, which seems to work well for those illnesses. Active substance abuse is still a concern for ketamine treatment as well, so those patients probably would be better served by ECT or TMS.

CHPR: You mentioned that ECT scares some patients. Is this because of the worry about memory impairment?

Dr. Seiner: That's the concern that gets the most press. Other people are frightened by the ECT procedure itself. They have disturbing memories of Jack Nicholson in *One Flew Over the Cuckoo's Nest*. But many people now, like many in Gen Z, have no familiarity with that movie, so we're finally starting to be able to put it behind us.

CHPR: That movie unfortunately really stigmatized ECT.

Dr. Seiner: Right, and there's still a lot of stigma and misinformation, especially on the internet. We have a group here at McLean for anyone who wants to know more about ECT, and it includes people who've undergone the procedure. Our philosophy is that we don't downplay anything; we just want people to see what we do. ECT is a procedure like any other procedure, with side effects and risks, but with a lot of benefits, too. We do everything we can to make it comfortable and minimize the side effects. Kitty Dukakis, the wife of former Massachusetts governor Michael Dukakis, also runs a group here in Massachusetts called "A Light in the Darkness." It started off as a support group and now focuses on ECT advocacy. These groups help patients feel more comfortable about proceeding with ECT.

CHPR: Can you tell us about recent innovations in ECT?

Dr. Seiner: The biggest change in the field is the way we administer the electrical stimulus. We do

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a lot more unilateral placement of the electrodes than we did 20 years ago—typically on the right side of the brain, which is the nondominant side for most people—because unilateral placement produces fewer cognitive side effects. We still use other techniques like bitemporal or bifrontal placement of electrodes, but we use them primarily when we're not getting a sufficient clinical response with the unilateral technique, or when we need a quick response such as with a patient who is catatonic or not eating and drinking.

CHPR: Have there been any other innovations?

Dr. Seiner: We've also found that ultra-brief pulses, which are very tiny pulses, produce fewer side effects. Optimal depolarization time of a neuron in the brain is about 0.1–0.2 ms, and the ultra-brief pulse lasts about 0.3 ms, so it's very close to that, as opposed to the 1 ms that we had been using earlier. A 2008 study showed that the ultra-brief pulses resulted in significantly less retrograde memory loss in patients (Sackheim HA et al, *Brain Stimul* 2008;1(2):71–83). That finding took the field by storm. All of us sent our machines in to get retrofitted.

CHPR: Do the ultra-brief pulses work as well as the regular pulses?

Dr. Seiner: The ultra-brief pulses are a milder treatment, and about 40%–50% of patients don't respond fully. For those patients we have to make adjustments, like increasing the pulse width or switching to bifrontal or bilateral.

CHPR: Have there been any other changes?

Dr. Seiner: The other major development is that we've learned better ways to prolong ECT's benefits (Brown ED et al, *J ECT* 2014;30(3):195–202). The two main options are: 1) a medication regimen to keep people well after ECT; and 2) a slow, gradual taper of ECT to prolong the benefit. With either option alone, there's about a 50% relapse rate at six months, but by combining them, the relapse rate is lower and the response is often more robust. For example, a study of geriatric patients found that 13% of patients receiving ECT plus medications experienced relapses compared to 20% of patients on medications alone. Also, the patients receiving medications plus ECT remained well for longer durations (Kellner CH et al, *Am J Psychiatry* 2016;173(11):1110–1118).

"The biggest change in electroconvulsive therapy is the way we administer the electrical stimulus. We do a lot more unilateral placement of the electrodes than we did 20 years ago—typically on the right side of the brain, which is the nondominant side for most people—because unilateral placement produces fewer cognitive side effects."

Stephen J. Seiner, MD

CHPR: How long does continuation ECT go for?

Dr. Seiner: Patients usually taper from three times a week to twice a week to once a week. The first month or two after ECT is when the relapse rate is highest, so we watch people closely over that time. If they're doing well, we taper and discontinue the ECT more quickly, but some patients might require maintenance ECT. Overall we like to follow patients for about six months, but by the end we may be treating them every six weeks or so.

CHPR: What's the difference between continuation ECT and maintenance ECT?

Dr. Seiner: Continuation ECT lasts for up to six months before we taper off. Maintenance ECT goes beyond six months. Most patients are able to taper it off eventually, but that can sometimes take a year or two in tougher cases.

CHPR: Do any patients continue for longer than that?

Dr. Seiner: Yes, some patients remain in long-term maintenance for years. These tend to be patients with severe and chronic mental illnesses, like schizophrenia, and ECT can really improve their quality of life.

CHPR: You alluded to patients with catatonia earlier; for these patients, how quickly do you see an improvement with ECT?

Dr. Seiner: Catatonia usually is pretty sensitive to ECT, so sometimes after a single procedure you'll see the patient chatting up the nurses in the recovery room. But often they quickly slip back into a catatonic state, so it can take a few treatments, typically at least five or six, before they really start to improve. You have to be careful with catatonia because patients can look like they've recovered but then slide back very quickly, especially if you taper the benzodiazepines too fast.

CHPR: You keep the patients on benzodiazepines when they go for ECT? Don't the benzodiazepines interfere?

Dr. Seiner: We would love to taper the benzodiazepines, but they are often what's keeping the patients eating, ambulating, and not completely catatonic between treatments. For young healthy people, young women in particular, their seizure threshold is so low that you can usually produce a seizure even with high doses of benzodiazepines. And if you can't get a good seizure, you can give flumazenil, which is a benzodiazepine reversal agent, right before the treatment. It's well tolerated, and many places use it routinely for anybody who's on a benzodiazepine. We only use it if we absolutely need it in catatonia, because nobody's studied the effect of reversing benzodiazepines in somebody who's struggling with catatonia.

CHPR: Can you say a little more about memory loss with ECT?

Dr. Seiner: There are two types of memory loss that we see with ECT. The first type affects anterograde memory, which refers to the ability to store new memories. A meta-analysis found there's no evidence of any long-term effect of ECT on anterograde memory (Semkovska M and McLoughlin DM, *Biol Psychiatry* 2010;68(6):568–577). In fact, that meta-analysis found that if you wait a few weeks after ECT, patients do better on cognitive testing after the ECT than they did before. It's not that we're making them smarter; it's just that we underestimate how debilitating depression is on cognition.

CHPR: And what is the second type?

Dr. Seiner: The second type affects retrograde memory. This refers to the loss of memories of things that

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Responding to Sexual Activity on the Inpatient Psychiatric Unit

Husna Najand, MD. Olive View—UCLA Psychiatry Residency Program.

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

We know sexual attraction occurs among individuals staying together in a psychiatric inpatient unit. The prevalence of sexual activity on acute units is as high as 5%—and up to 70% among chronically ill patients in long-term residence in state hospitals (Ford E et al, *Psychiatr Serv* 2003;54(3):346–350; Wright ER et al, *Journal of Ethics in Mental Health* 2012;7:1–5). However, there are no universal policies addressing sexual activity between patients on inpatient units, and Joint Commission standards do not include any discussion of this topic.

Hospital policies regarding physical contact

Many psychiatric hospitals lack specific policies on patient-to-patient physical contact. Perhaps these policies are absent because hospital administrators view such contact as obviously inappropriate, for a variety of reasons. Sexual contact elicits strong emotions, both positive and negative, that may distract from or interfere with treatment. If sexual contact is non-consensual or involves patients with reduced capacity, it can be both criminal and psychologically damaging.

What about more casual physical contact, such as holding hands, sitting very close to others, or hugging? Patients often make intense connections with others in inpatient units, given the encouragement to be honest and self-revealing in treatment groups. Touching may seem like a natural extension of emotional intimacy. Nonetheless, there is a slippery slope from friendly touching to sex, so most units err on the side of caution and completely prohibit physical contact between patients. Generally, we agree that such policies are prudent. We remind patients of these policies at the beginning of and during the course of their inpatient stays, particularly when they display hypersexual behaviors.

How to respond to sexual contact in the hospital

The nursing or security staff inform you that two patients on the unit have engaged in sexual activity. What do you do next? First, determine what type of sexual activity occurred. In most cases on inpatient units, sexual activity occurs between fully clothed individuals and involves kissing, hugging, and fondling. Actual sexual intercourse is rare. You will generally have a good idea of what happened from the reports of the unit staff who discovered the activity, but you should do your best to confirm this when you interview the patients yourself. Separate the patients and place them under 1:1 supervision while you evaluate each privately. Patients should not change clothing or take showers during this time, as physical evidence may be needed. Be sure to inform the hospital's risk management office of the incident.

Determine capacity to consent

Regardless of the nature of the activity, you should try to determine whether the patients had the capacity to consent to sexual activity. All individuals—including those with severe mental illness, as well as those with intellectual disabilities or dementia—have sexual consent capacity once they reach the age of consent as established by their state. Patients on psychiatric holds also retain sexual consent capacity; the exception is if a court previously determined a patient lacks capacity, such as in probate conservatorship. Assuming the patients involved are their own decision makers, how do you determine if they really have the capacity to consent to sexual activity? While each state has its own statutes, the following principles have typically been used to determine sexual consent capacity (Boni-Saenz AA, *Psychiatric Times* 2016;33(7)):

- **Knowledge:** Does the patient demonstrate basic knowledge and understanding of the sexual act in question?
- **Rationality:** Does the patient demonstrate reasoning ability, including weighing the risks and benefits of sexual activity and appreciating its potential consequences (eg, STI transmission, pregnancy)?
- **Voluntariness:** Is the patient able to decide to engage in sexual activity without coercion or undue influence? Does the patient understand they have the right to say no (withdraw consent) at any time during the sexual activity?

If a person does not meet these standards, their capacity to consent is most likely impaired. But capacity to consent to sexual activity is on a continuum. An individual may have the capacity to consent to activities with a low level of risk, like cuddling and kissing, but not intercourse, which carries a higher level of risk (Syme ML and Steele D, *Arch Clin Neuropsychol* 2016;31(6):495–505).

If both patients report the sexual activity was consensual and both have the capacity to consent to sexual activity, do you need to intervene any further? Generally not. The exception is if there was sexual intercourse, in which case you should inform the patients of the risks of unprotected sex and offer screening and prophylactic interventions *(see "Interventions Following Sexual Activity on the Inpatient Unit" table on page 9).*

Guidelines for managing non-consensual sexual incidents

What do you do if the patients claim the activity was consensual but one or both lack the capacity to consent to sexual activity? The surrogate decision maker for the patient(s) lacking capacity must be contacted immediately to determine the steps they wish to take. State regulations generally require you to report such incidents to a department of public health or department of mental health. The reporting follows the same procedures used for any so-called "serious reportable event," such as self-inflicted or physical assaults. Reach out to your

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Responding to Sexual Activity on the Inpatient Psychiatric Unit
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hospital's risk management team with any questions.

If a patient reports the activity was not consensual, this constitutes sexual assault. Ensure the patient is safe and is kept separated from the alleged perpetrator. If the patient wants to press charges, contact the police and arrange for a private meeting. Depending on the nature of the contact, a rape kit should be offered, which is handled by a hospital's sexual assault response team. If your hospital does not have such a service, the patient may be transferred briefly to another hospital. Offer the patient counseling, given the psychological impacts such activity may have (counseling can also help even in cases where the sexual activity was consensual). Watch for unconscious biases: We might tend to view a petite female patient as a victim and a large male patient as a perpetrator, but all patients on inpatient units are vulnerable adults, and we need to follow the same procedures for all cases.

Keep in mind that patients have the right to refuse the recommended examinations and interventions unless they lack capacity to make informed decisions. In those cases, identify a surrogate decision maker who can make decisions on the patient's behalf.

Document all details of the incident, clinical evaluations, and interventions in the medical record. Include information on the location, timing, individuals involved, witnesses, clinical evaluations (including for capacity), interventions offered and completed, follow-ups, and referrals. Finally, debrief with the team to review staff response and management of the incident, with the goals of minimizing future incidents and maintaining patient safety.

Interventions Following Sexual Activity on the Inpatient Unit

Intervention	Notes
Pregnancy test Serum STI screening: • HIV • Hepatitis panel • Syphilis • Gonorrhea/chlamydia • Trichomoniasis	A repeat pregnancy test will need to be obtained two weeks following the sexual encounter If initial test results are negative but infection cannot be ruled out, repeat syphilis test at four to six weeks and at three months; repeat HIV test at six weeks and at three and six months
Postexposure prophylaxis for HIV within 72 hours	Consult with the medicine team for further guidance
HPV vaccination is recommended for females age 9–26 years and males age 9–21 years	
Postexposure hepatitis B vaccination	
Offer emergency contraception: • Ulipristal 30 mg: preferred treatment after 72 hours, effective up to 120 hours • Levonorgestrel 1.5 mg single dose, or 0.75 mg then repeated in 12 hours • Copper IUD	Ulipristal is more effective for overweight or obese women; levonorgestrel may be ineffective in this population
Empiric antibiotic treatment: • Chlamydia, gonorrhea: ceftriaxone 500 mg IM in a single dose plus doxycycline 100 mg two times/day orally for seven days (for persons weighing over 150 kg, administer 1 g of ceftriaxone) • Trichomoniasis: metronidazole 500 mg orally two times daily for seven days	

Sources: www.cdc.gov/std/treatment-guidelines/sexual-assault-adults.htm; www.uptodate.com/contents/evaluation-and-management-of-adult-and-adolescent-sexual-assault-victims

CHPR VERDICT: When a sexual incident occurs on the inpatient psychiatric unit, assess each patient privately to determine if the incident was consensual and if they each had the capacity to consent to sexual activity. Work with your hospital's risk management team to make sure all appropriate authorities are notified and to assist with next steps. Notify the police if the patient wants to press charges. Follow clinical guidelines regarding physical examinations, laboratory testing, and prophylactic treatment.

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Note From the Editor-in-Chief

For the first time in nine years, DSM-5 has undergone a text revision. DSM-5-TR, released in March 2022, contains updates and clarifications for the descriptive texts and criteria of most disorders. Extensive commentary now reviews racism's and discrimination's effects on the manifestation and diagnosis of mental disorders, and several wording updates have been made. For example, "experienced gender" replaces "desired gender" in the diagnosis of gender dysphoria. Other noteworthy changes in terminology include "antipsychotic medication or other dopamine receptor blocking agent" for "neuroleptic," "intellectual developmental disorder" for "intellectual disability," and "neurological symptoms disorder" for "conversion disorder." DSM-5-TR lists criteria for a newly recognized condition: prolonged grief disorder. Other notable changes include the addition of symptom codes for suicidal conduct and nonsuicidal self-injury, and the reinstatement of "unspecified mood disorder." DSM is the most widely used resource for mental disorders worldwide, so it's good to see updates that reflect advances in research and terminology.

Sincerely,
Victoria Hendrick, MD

Research Updates IN PSYCHIATRY

SCHIZOPHRENIA

Clozapine for Conduct Disorder in Schizophrenia

Anne Li, MD. Dr. Li has disclosed no relevant financial or other interests in any commercial companies pertaining to this educational activity.

REVIEW OF: Krakowski M et al, *Am J Psychiatry* 2021;178(3):266–274

STUDY TYPE: Randomized controlled trial

We often struggle to find effective interventions for aggressive behavior in patients with schizophrenia. For patients with a history of conduct disorder, the task is even more challenging. Patients with schizophrenia will often have shown symptoms of conduct disorder before age 15, although establishing the diagnosis sometimes requires digging in the history since the psychotic disorder may overshadow other symptoms. To make the conduct disorder diagnosis, look for a history of the following during childhood and adolescence: bullying or threatening behaviors, a chronic disregard for rules, criminal activity (eg, stealing, arson, sexual assault, battery), and a lack of empathy or remorse.

In a previous study, the authors demonstrated that clozapine is effective in reducing aggression and impulsive behavior in patients with schizophrenia (Krakowski M et al, *Arch Gen Psychiatry* 2006;63(6):622–629). But does clozapine also reduce aggression in patients who additionally have a history of conduct disorder?

The authors examined this question by enrolling 99 subjects with schizophrenia in a 12-week double-blind trial conducted in a psychiatric research unit. All subjects had a history of being physically assaultive, and about half (n = 53) had a history of conduct disorder prior to age 15. Subjects were randomly assigned to receive clozapine (n = 33), olanzapine (n = 34), or haloperidol (n = 22), and the frequency and severity of assaults were scored on the Modified Overt Aggression Scale (MOAS). The MOAS total score and the physical assault sub-score were compared across groups.

For all subjects, clozapine was superior to olanzapine and haloperidol in preventing violent behavior, and olanzapine was superior to haloperidol. However, for subjects with a history of conduct disorder, clozapine's efficacy in preventing violence was particularly high: These subjects were over three times as likely to have a lower MOAS score and four times as likely to have a lower physical assault score compared to subjects on haloperidol. Subjects without a conduct disorder history also benefited from clozapine, but not as dramatically: They were 1.9 times as likely to have a lower MOAS score and 2.7 times as likely to have a lower physical assault score compared to subjects on haloperidol.

CHPR'S TAKE

For patients with schizophrenia and a history of conduct disorder, clozapine appears significantly more effective in reducing violent behavior compared to olanzapine and haloperidol. Haloperidol seems to be less effective than clozapine or olanzapine for this patient population.

SIDE EFFECTS

Sublingual Atropine Drops and Clozapine-Induced Drooling

Sébastien Hardy, PharmD, BCPS. Dr. Hardy has disclosed no relevant financial or other interests in any commercial companies pertaining to this educational activity.

REVIEW OF: Mubaslat O and Lambert T, *Psychopharmacology (Berl)* 2020;237(10):2905–2915

STUDY TYPE: Randomized placebo-controlled trial

Excessive salivation or drooling is one of the more troublesome side effects of clozapine, occurring in up to 80% of patients. It is embarrassing for patients and is a factor in drug discontinuation. Aspiration pneumonia is a rare but serious complication.

Non-drug treatment is a first step, such as chewing sugar-free gum and elevating/covering pillows with a towel. Anticholinergics, such as glycopyrrolate, trihexyphenidyl, and benztropine, are usually helpful but significantly exacerbate clozapine's powerful anticholinergic effects, placing patients at risk for constipation, impaction, and paralytic ileus. This has provoked interest in more localized treatments, such as the sublingual administration of atropine 1% ophthalmic solution. The clinical use of sublingual atropine has been guided by case reports and case series. Recently, Australian researchers investigated its efficacy for clozapine-induced drooling in a randomized controlled trial.

The researchers recruited inpatients (n = 23) from two major psychiatry centers in Sydney. Participants were randomized to receive two drops of sublingual atropine 1% (n = 11) or chloramphenicol 0.5% (n = 12); researchers used chloramphenicol as the placebo because it has a bitter taste similar to atropine. Medications were given at bedtime. The main outcome variable was saliva secretion, which was measured both at baseline and after medication by weighing dental cotton rolls and pads. Saliva secretion was reduced by 34% with sublingual atropine, while it was increased by 23% with placebo (p = 0.02). More atropine-treated participants had an improvement in pillow saliva wetness and sleep, though only 10 of the 21 participants were questioned about these issues.

CHPR'S TAKE

Sublingual atropine drops appear to be effective for clozapine-induced hypersalivation. A larger study would be helpful to better establish the safety and efficacy of this treatment compared to systemic drugs.

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This page is intended as a study guide. Please complete the test online at www.TheCarlatReport.com. Learning Objectives (LO) are listed on page 1.

1. Which psychiatric condition is most commonly associated with psychogenic polydipsia (LO #1)?
   - [ ] a. Major depressive disorder
   - [ ] b. Anxiety disorders
   - [ ] c. Schizophrenia
   - [ ] d. Bipolar II disorder

2. According to Dr. Seiner, there's a good case to be made for trying ketamine first, before electroconvulsive therapy (ECT), for patients with which psychiatric condition(s) (LO #2)?
   - [ ] a. Psychotic depression
   - [ ] b. Severe psychiatric illness and suicidality
   - [ ] c. Delirious mania
   - [ ] d. Catatonia

3. You are told that two patients in the psychiatric unit engaged in sexual activity. Other than educating the patients on unprotected sex and offering screening and prophylactic interventions, there is no need to intervene further when ____ (LO #3).
   - [ ] a. Both patients claim the activity was consensual and both patients have the capacity to consent to sexual activity
   - [ ] b. At least one patient claims the activity was consensual and both patients have the capacity to consent to sexual activity
   - [ ] c. Both patients claim the activity was consensual and both patients lack the capacity to consent to sexual activity
   - [ ] d. At least one patient claims the activity was consensual and both patients lack the capacity to consent to sexual activity

4. In a 2020 study of clozapine-induced drooling, what was concluded about the efficacy of sublingual atropine 1% ophthalmic solution for saliva secretion and sleep, compared to placebo (LO #4)?
   - [ ] a. Sublingual atropine improved sleep but had no effect on saliva secretion
   - [ ] b. Sublingual atropine improved saliva secretion and sleep
   - [ ] c. Sublingual atropine improved saliva secretion but had no effect on sleep
   - [ ] d. Sublingual atropine had no effect on saliva secretion or sleep

5. Which of the following about psychogenic polydipsia is true (LO #1)?
   - [ ] a. Nicotine dependence is a risk factor for psychogenic polydipsia
   - [ ] b. Behavior interventions do not significantly improve psychogenic polydipsia
   - [ ] c. Naltrexone alone significantly reduces compulsive drinking, compared to naltrexone plus an antipsychotic
   - [ ] d. Phenothiazines are superior to other medications for improving psychogenic polydipsia

6. According to Dr. Heckers, what is the first-line treatment for patients with catatonia (LO #2)?
   - [ ] a. ECT
   - [ ] b. Amantadine (100–600 mg/day)
   - [ ] c. Lorazepam (2 mg IM or IV)
   - [ ] d. Valproic acid (500–1500 mg/day)

7. Regarding sexual activity between patients in the inpatient psychiatric unit, when is it necessary to assess each patient's capacity to consent to sexual activity (LO #3)?
   - [ ] a. When one of the patients has a severe mental illness
   - [ ] b. When the activity was sexual intercourse
   - [ ] c. When one of the patients has dementia
   - [ ] d. All patients, regardless of clinical severity, must be assessed for sexual consent capacity when any sexual activity is performed

8. According to a 2021 study of schizophrenia, which of the following correctly ranks the effectiveness of the investigated antipsychotics, from most to least effective, for preventing violent behavior in subjects with and without comorbid conduct disorder (LO #4)?
   - [ ] a. Olanzapine > clozapine > haloperidol
   - [ ] b. Haloperidol > olanzapine > clozapine
   - [ ] c. Haloperidol > clozapine > olanzapine
   - [ ] d. Clozapine > olanzapine > haloperidol

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Expert Interview – Electroconvulsive Therapy: A Primer

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happened before ECT. Some patients lose memories and don't get them back. The highest risk for memory loss is the period three to four months before ECT, which is also when the patient was most depressed. Those memories are the most vulnerable and sometimes just get lost completely. In rare cases, patients report losing memories from years earlier. These cases of memory loss tend to occur in patients who 1) were very sick for a long time; 2) were on a lot of medications; and 3) went through multiple courses of ECT, particularly bilateral ECT. The most common things that people have trouble remembering—whether it's within the previous three months or in these rare cases of years in the past—tend to be things they did once and then didn't really think about again, like a trip, or a restaurant they once went to. Nobody forgets who their daughter is, but they might forget going to their daughter's piano recital. Of course, some people forget those things even when they don't have ECT.

CHPR: What do you think of the use of adjunctive medications to minimize memory loss?

Dr. Seiner: There aren't a lot of data about adjunctive medications, but cholinesterase inhibitors and memantine have been used since they are memory-enhancing agents. The other medication that has some data is thyroid supplementation. We don't use adjunctive medications because we focus on adjusting how we do the ECT to minimize memory impairment, and we work to optimize patients' pharmacologic regimen. So far I haven't been convinced by any of these augmentation strategies enough to implement them regularly.

CHPR: Thank you for your time, Dr. Seiner.