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Victoria Hendrick, MD
Editor-in-Chief

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Workplace Violence Prediction and Prevention

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Violence Prevention in Acute Psychiatric Settings: Staying Safe in Challenging Environments

Victoria Hendrick, MD. Chief, Inpatient Psychiatry, Olive View-UCLA Medical Center; Editor-in-Chief, The Carlat Hospital Psychiatry Report.

Dr. Hendrick has no financial relationships with companies related to this material.

Working in psychiatric emergency rooms and inpatient units, you may encounter aggressive or violent behavior, as it's not uncommon in these high-acuity settings. That said, there's a growing recognition that violence is not something staff should accept as "just part of the job." With this in mind, many hospitals now offer training programs like AVADE, CPI, MOAB, or PMAB to improve workplace safety and support staff in managing high-risk situations (see "Violence Prevention Training Acronyms" box on page 4). These programs vary in focus. AVADE, MOAB, and PMAB incorporate physical self-defense or intervention techniques for situations where de-escalation fails, while CPI emphasizes verbal de-escalation and nonviolent approaches. Any of these or similar training programs, regardless of their specific emphasis, will equip you with tools to defuse aggression and protect yourself and others. If you have access to such programs, be sure to take advantage of them. Their role-playing scenarios are especially useful for gaining valuable hands-on experience. But even without formal training, you can take significant steps to keep yourself and others safe by adopting the strategies I'll outline here.

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Q&A With the Expert

Violence Prediction and Prevention Tools
Heba Mesbah, MD, PhD

Assistant Professor of Emergency Medicine, Baylor College of Medicine, Houston, TX.

Dr. Mesbah has no financial relationships with companies related to this material.

CHPR: Please start by telling us a little about yourself.
Dr. Mesbah: I'm an assistant professor of emergency medicine at Baylor College of Medicine in Houston, Texas. My research focuses on behavioral health patients—specifically, predicting aggression and improving safety for patients in the emergency department (ED). I recently published a systematic review evaluating all the validated tools used to predict violent behavior in EDs (Mesbah H et al, *Am J Emerg Med* 2024;80:44–50), and my team is currently working on developing and validating new tools for predicting aggression.

CHPR: What prompted you to study this area?
Dr. Mesbah: Two reasons. First, the ED is a unique, fast-paced environment with high patient volume. It's critical for ED staff to identify patients who are at high risk for aggression, not only for the patient's safety but also for the safety of other patients and staff. We know that mental health presentations now

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make up more than 12% of all adult ED visits in the US, which highlights how common and pressing this issue is (Peters ZJ et al, *National Health Statistics Reports* 2023;181). Second, there's no consensus on the most reliable screening tool for predicting aggressive behavior, so my team wanted to explore this question.

CHPR: Can you tell us about the tools that are available?

Dr. Mesbah: In our review, we identified 10 tools. The Brøset Violence Checklist (BVC) was the most promising tool we found for EDs (Woods P and Almvik R, *Acta Psychiatr Scand Suppl* 2002;412:103–105). It's a six-item checklist of observable behaviors, like confusion, irritability, and verbal or physical threats, and it was designed for 24-hour observation. Its specificity is extremely high at over 99%. But on the downside, its sensitivity is under 50%, meaning it misses a substantial number of patients who later become aggressive. Still, when the BVC does flag someone, it's very meaningful, as patients who score 3 or more are over 70 times more likely to become violent within 24 hours (Partridge B and Affleck J, *Australas Emerg Care* 2018;21(1):31–35). In Australia, the BVC has been incorporated into a structured decision-making tool called the Occupational Violence Assessment (OVA) score. It uses the same items but adds action steps (eg, "If score is 1, do X; if 2, notify the doctor; if 3, call security") (Senz A et al, *Emerg Med Australas* 2021;33(4):665–671).

"The tools offer a structured way to support clinical judgment and create a shared language among staff. Especially in EDs, where we often don't have time to build rapport or gather a detailed history, having a brief, structured score at hand can make that judgment more efficient and more consistent across providers."

Heba Mesbah, MD, PhD

CHPR: Is the OVA used in the US also?

Dr. Mesbah: While the OVA itself is not routinely used in the US, it illustrates how a brief risk score can be paired with clear response protocols—an approach we could incorporate into our own agitation or workplace violence policies.

CHPR: And can you tell us about some of the other tools you reviewed?

Dr. Mesbah: Another tool is the Dynamic Appraisal of Situational Aggression (DASA). It's essentially the BVC's more sophisticated cousin. While the BVC gives you six observable behaviors and a straightforward score, the DASA adds a seventh item—impulsivity—and has been validated across more settings, including forensic units, general inpatient psych, and even medical-surgical floors. Research from the original validation study found that for each one-point increase in DASA score, the odds of aggression within 24 hours increased roughly threefold. What makes the DASA clinically useful is that it doesn't just predict violence; it gives you a dynamic score that changes shift to shift, which means you can track whether de-escalation efforts are actually working. That said, in a busy ED where you have five minutes to triage a patient, the BVC's simplicity often makes it more practical as the DASA requires more clinical judgment on some items, like "sensitivity to perceived provocation," which can be difficult to assess during rapid turnover. There's also the Behavioral Activity Rating Scale (BARS), which measures a patient's current agitation level, from difficult-to-rouse to violent (Swift RH et al *J Psychiatr Res* 2002;36(2):87–95). It's not predictive, but it is useful for assessing real-time agitation.

CHPR: Are there tools that incorporate more historical risk factors?

Dr. Mesbah: Yes. The Aggressive Behavior Risk Assessment Tool (ABRAT) is a brief checklist that combines behavioral items plus historical factors like prior incidents of aggression as well as psychiatric diagnoses. It was originally developed to predict violence in medical–surgical units, but it's also been tested in EDs, where it had a sensitivity of 84% and specificity of 95% (Kim SC et al, *J Am Coll Emerg Physicians Open* 2022;3:e12693). (*Editor's note: For a quick guide to the various tools Dr. Mesbah mentions, see "Violence Risk Screening and Assessment Resources" table on page 3).*

CHPR: Do the tools predict violent behavior across different populations like adolescents and older adults?

Dr. Mesbah: We didn't find major differences based on the population studied, but we did see differences across settings. Some tools perform best in inpatient units, while others are more useful in busy EDs. For example, some of the ABRAT items require information that's hard to obtain in an ED, such as detailed psychiatric history, so it generally ends up being a better fit for use in

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inpatient psychiatric or medical–surgical units than in general EDs. The setting matters more than the patient's age in terms of how well a tool performs.

CHPR: It sounds like some of these tools could be useful for inpatient psychiatric units.

Dr. Mesbah: While my clinical focus is in emergency medicine rather than inpatient psychiatry, many of these tools, including the DASA, ABRAT, and BVC, were originally studied in inpatient settings. And they fit well where teams can reassess behavior over time. The same caveat applies: They support, but don't replace, clinical assessment and good team communication.

CHPR: Is there any value in using more than one tool?

Dr. Mesbah: We didn't find evidence supporting the use of multiple tools. Using more than one can be confusing and may produce conflicting results. A single tool that's simple to use and accurate is preferable.

CHPR: How do these tools compare with clinical judgment?

Dr. Mesbah: A tool should be used together with clinical judgment, not in place of it. The tools offer a structured way to support clinical judgment and create a shared language among staff. Clinicians still need to use their judgment when interpreting scores and choosing interventions. And especially in EDs, where we often don't have time to build rapport or gather a detailed history, having a brief, structured score at hand can make that judgment more efficient and more consistent across providers.

CHPR: What are the implications of false positives? Could staff overreact?

Dr. Mesbah: False positives aren't a failure—they're a form of prevention. If we believe a patient may become aggressive, we can take early steps: moving them to a quieter room, increasing observation, involving de-escalation teams, offering medication proactively, or alerting security. These measures help prevent escalation.

CHPR: Have these tools been shown to reduce adverse outcomes beyond violent incidents, like lowering the rates of staff injuries?

Dr. Mesbah: We found that when EDs used the OVA tool—which is just the BVC with some action steps attached—security callouts dropped significantly (Senz et al, 2021). That's the kind of outcome that matters day-to-day in an ED: Having to hit the panic button fewer times means things are getting de-escalated earlier, which probably means fewer staff getting hurt, though the review didn't track injuries directly. In a separate ED project that embedded the DASA into routine rounding protocols, staff injuries decreased by 68% after the DASA was introduced (Olshan-Perlmutter M et al, *Standardizing Behavioral Health Emergency Department Nursing Rounds Incorporating Diagnostic Sensitivity of the Dynamic Appraisal of Situational Aggression to Improve Workplace Violence* [Poster presentation]. Advocate Health Midwest Region Nursing Research & Professional Development Conference 2024; virtual.).

CHPR: When someone is identified as moderate or high risk, what steps should staff take?

Dr. Mesbah: The OVA is currently the only tool that builds in specific action steps. When a patient scores in the moderate-risk range, the OVA recommends moving the patient to a quieter or more observable room, and adjusting the environment by removing any objects that the patient could use as a weapon. It also calls for an increase in nursing observation and to notify the physician so the team can begin verbal de-escalation and offer medication if appropriate. Security should then be alerted to be on standby, and the psychiatric team should be contacted to expedite evaluation or disposition when possible. Taken together, these proactive steps can significantly reduce the likelihood of a violent incident occurring.

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Violence Risk Screening and Assessment Resources

Tool	Setting	Resource
Brøset Violence Checklist (BVC) Six-item checklist predicting violence within 24 hours. High specificity but lower sensitivity—some high-risk patients may be missed.	EDs; inpatient psychiatric units	www.tinyurl.com/yspzxjze
Occupational Violence Assessment (OVA) BVC-based decision framework with built-in action steps. Improves consistency but limited validation outside Australia.	EDs with protocolized workflows	Senz A et al, *Emerg Med Australas* 2021;33(4):665–671
Dynamic Appraisal of Situational Aggression (DASA) Seven-item tool for repeated dynamic risk assessment. Well validated but less suited to rapid emergency department (ED) screening.	Inpatient psychiatric units; forensic settings	www.tinyurl.com/22mha2xu
Aggressive Behavior Risk Assessment Tool (ABRAT) Combines behavioral cues with historical risk factors. More sensitive than observational tools but requires background information.	Medical–surgical units; inpatient psychiatry; select EDs	Kim SC et al, *J Am Coll Emerg Physicians Open* 2022;3(2):e12693
Behavioral Activity Rating Scale (BARS) Single-item agitation severity scale. Useful for real-time management, not future violence prediction.	EDs; inpatient units (agitation assessment)	Mesbah H et al, *Am J Emerg Med* 2024;80:44–50

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CHPR: Might flagging a patient as high risk bias how staff approach them?

Dr. Mesbah: Formalizing violence risk with tools like the BVC and DASA actually helps counteract bias by replacing subjective "gut feelings" with objective, observable behavioral criteria. These tools don't create a permanent label—they are dynamic assessments that track a patient's current state. As scores drop and the patient stabilizes, staff receive clear data to scale back precautions and reduce restrictive measures. Rather than labeling a "dangerous person," the goal is to trigger specific clinical supports. In practice, studies show these approaches can lead to less restrictive care, including a 55%–75% reduction in restraints and a 68% decrease in staff injuries (Senz et al, 2021; Olshan-Perlmutter et al, 2024).

CHPR: Do staff need any special training to use these tools?

Dr. Mesbah: Training is helpful so that everyone can interpret behaviors consistently. Some checklist items are subjective, like "boisterous" or "verbally threatening," so it's important that all nurses, doctors, and other staff members are on the same page. Formal training programs that specifically focus on the use of violence prediction tools are not yet universal, but they are becoming increasingly common. In some hospitals, tools like the BVC are incorporated into nursing orientation and reinforced through periodic refreshers. Some institutions also incorporate simulation-based training in aggression recognition and de-escalation, allowing staff to practice applying risk assessments in realistic scenarios and receive structured feedback.

CHPR: Might more experienced staff interpret behaviors differently than newer clinicians?

Dr. Mesbah: We're in fact studying how a person's level of experience and background might affect how they interpret these scores, such as whether a staff member has personally experienced being assaulted by a patient. Some research is now using AI and machine learning to pull charts—like millions of charts—to give us more information about the factors that predict violent behaviors.

CHPR: You mentioned your team is developing a new tool. Can you say more?

Dr. Mesbah: We want to expand beyond just observable behaviors. We're looking at factors like prior aggression, psychiatric diagnoses, substance use, and arrival circumstances (such as whether the police were involved). Patient characteristics like homelessness or insurance status may also contribute. We're seeking funding to develop and validate a tool that incorporates these variables.

CHPR: So perhaps we'll eventually see a Mesbah violence prevention tool?

Dr. Mesbah: Not necessarily with my name on it!

CHPR: Either way, we look forward to seeing what your team develops. Thank you for your time, Dr. Mesbah.

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Violence Prevention in Acute Psychiatric Settings: Staying Safe in Challenging Environments

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This article focuses specifically on nonpsychopharmacologic approaches to managing aggression. Of course, psychopharmacology plays a critical role in inpatient settings, especially for patients in acute agitation or psychosis, and there are well-established protocols for rapid tranquilization, PRN use, and longer-term medication strategies. We've covered those approaches in a previous issue (see "Medications to Rapidly Treat Psychotic Agitation," *CHPR* Oct/Nov/Dec 2021), so they're not the focus here.

Violence Prevention Training Acronyms

AVADE: Awareness, Vigilance, Avoidance, Defense, and Escape

CPI: Crisis Prevention Institute

MOAB: Management of Aggressive Behavior

PMAB: Prevention and Management of Aggressive Behavior

Screen for risk: Who's at risk and why

Reducing the risk of violent incidents begins with identifying potential threats and risk factors. Gather information about a patient's prior violent behavior, substance use history, or psychiatric conditions linked to impulsivity or aggression, like borderline personality disorder, bipolar disorder during manic episodes, intermittent explosive disorder, or psychotic disorders with paranoia or command auditory hallucinations.

It's also helpful to assess the *type* of violence that a patient may be at risk for:

- Impulsive violence is reactive and emotionally driven, often triggered by frustration, provocation, or perceived threats. It's common in patients who have limited emotional regulation or low frustration tolerance.
- Psychotic violence is driven by delusions or hallucinations, especially paranoid delusions or command auditory hallucinations.
- Predatory violence is rare but serious—premeditated, goal directed, and not emotionally driven. It may involve planning and targeting and often requires closer supervision and firm boundaries.
- Cognitive impairment–related violence can occur in patients with dementia, delirium, or brain injury. It's often due to confusion, misinterpretation of staff intentions, or fear during personal care.
- Substance-induced violence may stem from intoxication, withdrawal,

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or chronic effects of substance use. It can present as impulsive, psychotic, or disinhibited aggression.

Use this knowledge to take precautions, like assigning additional staff, initiating early calming interventions, placing patients in high-visibility rooms, and making sure staff have access to emergency response tools like panic buttons. Consider using validated tools like the Brøset Violence Checklist (Almvik R and Woods P, *Int J Psychiatr Nurs Res* 1999;4(3):498–505; www.tinyurl.com/yc4ttvaz).

Create a safer, calmer environment

Make sure your environment promotes safety. As much as possible, arrange the interview space to allow for clear sight lines, accessible exits, minimal blind spots, and adequate lighting. When working with patients who are agitated or have a history of aggression, choose an open space, ensure security is nearby, and stop the interview if the patient begins to escalate.

Some units have adopted features from EmPATH models in an effort to reduce environmental stimuli that can contribute to agitation. Popular features include quieter spaces with ample room, soothing colors, and staff trained to engage patients early, before escalation occurs (see our interview with Kimberly Nordstrom on EmPATH units in *CHPR* Jul/Aug/Sept 2022).

Enhance security measures

A visible and responsive security presence acts as both deterrence and support. If these measures aren't already in place at your hospital, consider requesting:

- Wearable alert buttons
- Panic buttons for high-risk areas
- Security cameras
- Metal detectors in the ED

Recognize early warning signs

An essential aspect of violence prevention is spotting risks before they have a chance to escalate.

- Watch for behavioral clues, like pacing, clenched fists, or tense posture.
- Listen for verbal signals like raised tones or angry muttering.
- Identify patient-specific triggers. Some patients are particularly sensitive to triggers like personal space violations, feeling ignored, or sensory overload. Proactively address these to prevent escalation.

Use de-escalation techniques

If you find yourself in an increasingly heated situation, creating a stable, low-stimulation environment can help you reduce tension (see "Principles of Verbal De-Escalation" box below).

- Use non-threatening, reassuring language—something simple like, "I hear you. Let me help," can go a long way in defusing frustration.
- Minimize overstimulation by reducing loud noises, bright lights, or other disruptive environmental factors.
- Be mindful of your body language: Maintain an open posture, stand at a slight angle to avoid appearing confrontational, speak with a calm, steady voice, and keep an arm's-length distance.
- Practice active listening by acknowledging the patient's emotions and concerns.

Principles of Verbal De-Escalation

1. Respect the patient's personal space.
2. Don't be provocative.
3. Establish verbal contact.
4. Be concise.
5. Identify wants and feelings.
6. Listen closely to what the patient is saying.
7. Agree to disagree.
8. Lay down the law and set clear limits.
9. Offer choices and optimism.
10. Debrief the patient and staff.

Source: Richmond JS et al, *West J Emerg Med* 2012 Feb;13(1):17–25; and *CHPR* Jan/Feb/Mar 2022.

When violence escalates: Defensive strategies

While the goal is always to prevent escalation, there may be rare situations where violence occurs despite your best efforts. If neither escape nor calling for help is an option, you may need to rely on self-defense techniques to stay safe. Role-playing scenarios, offered during training programs such as the ones discussed at the start of this article, can help you develop these skills. If you're unfamiliar with self-defense techniques, learning even basic tips can help you stay safe. Explore resources available from vetted sources (www.tinyurl.com/bdes8rba). Remember:

- Deflect attacks with blocking techniques
- Escape holds through disengagement maneuvers; practice these maneuvers to break free safely
- Physical restraint should be a last resort and should only be performed by trained staff

Strengthen team protocols and prevention plans

Clear guidelines and strong teamwork are essential for reducing workplace violence. All staff should be familiar with documentation and reporting protocols, which support effective follow-up and quality improvement. Hold regular multidisciplinary meetings, including representatives from security, psychiatry, and nursing, to review incidents and strengthen prevention strategies. After an incident, debrief as a team to reflect on what went well, identify what could be improved, and provide peer support for anyone affected emotionally or physically by the event.

CARLAT VERDICT

When you work with patients in acute states of agitation or paranoia, aggression is sometimes unavoidable. That said, you can lower the risk by screening patients, recognizing early warning signs, using de-escalation techniques, and improving security measures. If your hospital offers violence prevention training or role-playing exercises, be sure to take advantage of them to build your confidence and readiness. Even if you've already had training, consider a refresher, as these skills can fade over time.

Q&A With the Expert

Ketamine Treatment in Inpatient Psychiatry: What Clinicians Need to Know

Benjamin D. Brody, MD

Associate Professor of Clinical Psychiatry; Service Chief, Acute Care Services, Department of Psychiatry, Weill Cornell Medicine, New York, NY.

Dr. Brody has no financial relationships with companies related to this material.

CHPR: Please begin by telling us what inspired you to research the use of ketamine on the inpatient unit.

Dr. Brody: The ketamine story goes back to 2000 and even earlier, when John Krystal's group published the landmark paper showing ketamine's rapid-acting antidepressant effects (Berman RM et al, *Biol Psychiatry* 2000;47(4):351–354). When I started working in inpatient psychiatry, I kept running into a familiar dilemma: We admit patients for depression and start an antidepressant, knowing that while some benefit can appear within the first couple of weeks, the full therapeutic effect often takes six to eight weeks. That timeline can feel painfully long on an inpatient unit.

CHPR: Right—it's frustrating that standard treatments can take so long to work.

Dr. Brody: We have ECT, of course, but for antidepressants the time course doesn't align with a typical inpatient stay. What drew me to ketamine was its rapid onset—hours to days. I remember thinking it might be a perfect fit for inpatient psychiatry, where patients tend to have more severe depression and need faster-acting interventions. By 2017, two developments convinced me we should pursue this on our unit at Weill Cornell. First, a couple of studies showed ketamine wasn't just rapidly acting for depression—it also appeared to reduce suicidal ideation. One study showed that a single dose led to marked reductions in suicidal thoughts that lasted at least six weeks (Grunebaum MF et al, *Am J Psychiatry* 2018;175(4):327–335). That's how long it takes for a standard antidepressant just to start working. Second, the American Psychiatric Association (APA) published what was essentially a position paper outlining how ketamine could be used safely and responsibly (Sanacora G et al, *JAMA Psychiatry* 2017;74(4):399–405). Having formal guidance helped make the case to hospital leadership that this wasn't fringe work. Interestingly, we launched our program in 2019, around the same time intranasal esketamine was approved for treatment-resistant depression.

CHPR: What does ketamine administration actually look like on the inpatient unit?

Dr. Brody: It's fairly straightforward. We use the original protocol described by Krystal and colleagues: IV ketamine at 0.5 mg/kg infused over 40 minutes. It produces a subanesthetic blood level—roughly 10% or 20% of what you'd see with an anesthetic dose.

CHPR: Why not just go with intranasal esketamine?

Dr. Brody: There are clear advantages to IV ketamine. We can titrate the dose precisely based on body weight. That differs from esketamine, which comes in fixed-dose intranasal formulations and requires judgment about which dose fits best. And with an infusion, if someone has an adverse reaction, we can simply stop. The drug level falls quickly, and symptoms resolve. That level of control has made IV ketamine practical and manageable in the inpatient setting. Another advantage of IV ketamine is cost. Racemic ketamine has been around since the 1970s, it's off-patent, and a dose costs just a few dollars. So obviously that's an advantage on the inpatient side if hospitals already have it on formulary or can add it inexpensively. Esketamine (Spravato), in contrast, is substantially more expensive. But the flip side is that it's commonly covered by insurance for outpatients, so that's decisive for many patients.

> **"The dissociative experience deserves attention. Most patients find it neutral, odd but not troubling. Some find it pleasant, which explains its misuse potential. But a minority, maybe 2%–5%, find it frightening. They may say, 'I feel out of control' or 'This is scary.'"**
>
> Benjamin D. Brody, MD

CHPR: I see. Do you have anesthesiologists involved in the process of administering IV ketamine?

Dr. Brody: We don't. When I launched the program, I worked with the anesthesiology department, and they credentialed me by assisting with the first few infusions and reviewing our protocol. But these are subanesthetic doses. Patients remain arousable. They may get drowsy, but they're not unconscious. That distinction was key to demonstrating that psychiatrists could safely administer ketamine without anesthesia staff. This is consistent with national guidance. The APA's recommendations do not call for anesthesiology involvement for subanesthetic ketamine. Our setup is simple: We have one physician present, and a nurse who administers the infusion and monitors vitals. We also follow standard medical monitoring protocols and ensure staff are prepared to manage uncommon but recognized effects such as transient hypertension, tachycardia, or respiratory slowing.

CHPR: How quickly do you see benefits?

Dr. Brody: When ketamine works, it works fast, sometimes within a day or two. On the unit, we give twice-weekly infusions for a couple of weeks, as long as the patient remains hospitalized. If they're discharged sooner, we refer them to an outpatient program for IV ketamine or esketamine, depending on what's available to the individual in the community and covered by their insurance. If a patient hasn't improved after about four doses, that's our cue to pivot, most commonly to ECT. And that's really one of the reasons I became so interested in ketamine in the first place: If it's going to help, it tends

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to help quickly. In terms of time course, it's much closer to benzodiazepines or stimulants than to our traditional antidepressants or antipsychotics. The onset is just dramatically faster than what we're used to seeing with standard medications.

CHPR: You also start patients on antidepressants to maintain the gains, correct?

Dr. Brody: Yes. Most inpatients we treat with ketamine have treatment-refractory depression, and we use ketamine as an augmentation strategy combined with an antidepressant. If you stop dosing ketamine, the benefit fades pretty quickly. Patients often relapse within a couple of weeks, so they need a maintenance plan. Ketamine can be part of that, but it's not ideal as long-term monotherapy. That said, its cousin, intranasal esketamine, recently received an FDA indication as monotherapy for treatment-resistant depression (www.tinyurl.com/fjjcjabp).

CHPR: Why don't you like ketamine for long-term monotherapy?

Dr. Brody: I worry about safety issues. We do have some long-term data, including studies from the Janssen esketamine program showing safety and effectiveness out to about five years at standard doses. That's reassuring. But we also know that if the total amount of ketamine gets too high, it can become neurotoxic, and we don't yet know exactly where that cutoff is—when it stops helping and starts to carry risk. That uncertainty makes it hard to recommend ketamine as a long-term monotherapy. There are also practical issues. I'm opposed to the at-home model where companies mail ketamine to patients, usually as lozenges or oral tablets. I don't think that's safe. It's inconsistent with APA recommendations and completely inconsistent with the Risk Evaluation and Mitigation Strategy (REMS) program for esketamine. The FDA has warned against this practice. Safe treatment requires monitoring, which means patients have to come into clinic, and that adds a layer of logistics for long-term use. By contrast, traditional antidepressants have been used since the 1950s. They're not perfect, but they're broadly safe over the long term. For all of these reasons, ketamine monotherapy is likely to remain uncommon, even with the new esketamine monotherapy indication.

CHPR: Can you say more about neurotoxicity? That's not something many clinicians are aware of.

Dr. Brody: A recent review examined this across animal and human data (Li WS et al, *Am J Psychiatry* 2025;182:903–912). At very high or very frequent doses, far above anything we use clinically, you can start to see problems. In animals, repeated high-dose exposure can overstimulate glutamate pathways—a process called excitotoxicity, where brain cells get "over-revved" to the point of injury—and can cause long-term cognitive impairment. In humans, heavy recreational users, in some cases taking a gram or more a day, show memory, attention, and executive functioning deficits (Morgan CJA et al, *Addiction* 2010;105(1):121–133). But at therapeutic doses used in clinical practice, especially in the esketamine trials, the picture is quite different. Large, long-term studies of intranasal esketamine—up to 84 mg weekly or every other week for years—show stable or slightly improved cognition in adults with depression (Wajs E et al, *J Clin Psychiatry* 2020;81(3):19m12891). So, when we talk about neurotoxicity, we're really talking about exposures far outside therapeutic use.

CHPR: What are the typical adverse reactions you watch for?

Dr. Brody: The most common issues are transient increases in blood pressure and pulse; nausea happens occasionally. Hypertension is by far the most frequent. When that occurs, we pause the infusion for 10 or 15 minutes, the blood pressure usually settles, and we finish the treatment.

CHPR: Are there any other potential side effects we should know about?

Dr. Brody: The dissociative experience deserves attention. Most patients find it neutral, odd but not troubling. Some find it pleasant, which explains its misuse potential. But a minority, maybe 2%–5%, find it frightening. They may say, "I feel out of control" or "This is scary." In a paper we published, one patient even became suicidal during the experience (Brody BD et al, *J Clin Psychiatry* 2025;86(3):25com15946). That's why close monitoring is essential, with regular assessment of mental status, anxiety, and suicidality during and after dosing, and staff should be prepared to provide reassurance, grounding, or intervention if distress escalates. The good news is ketamine's half-life is short. Dissociation resolves as the drug is metabolized, usually within hours. But you can't predict who is going to have that experience, so monitoring is critical.

CHPR: Are there exclusion criteria, particularly for people with cognitive impairment?

Dr. Brody: Yes. We generally avoid ketamine in people with dementia or significant cognitive disorders co-occurring with depression, as well as in people with psychosis. For patients with cognitive impairment, the concern is that the dissociative experience can be disorienting, frightening, or difficult to interpret, and may worsen confusion or behavioral symptoms. They can also complicate the diagnostic picture by making it less clear whether we're really dealing with major depressive disorder vs a primary neurocognitive disorder. For patients with psychosis, there's a separate concern about exacerbating psychotic symptoms.

CHPR: What about comorbid substance use disorders?

Dr. Brody: That's a thornier area. A history of ketamine use disorder—or really any psychedelic use disorder—is a contraindication, but other substance use disorders are more complicated. Several studies suggest ketamine may have a role when it's paired with psychotherapy. For example, one randomized trial combined ketamine with motivational enhancement therapy for alcohol use disorder (Dakwar E et al, *Am J Psychiatry* 2020;177:125–133), and another paired infusion with mindfulness-based behavioral modification for cocaine dependence (Dakwar E et al, *Am J Psychiatry* 2019;176:923–930). And this work goes back even further. In the 1980s, a psychiatrist in Russia used ketamine combined with psychotherapy to treat alcohol and heroin dependence, and he published a substantial body of work that was really ahead of its time (Krupitsky EM and Grinenko AY, *J Psychoactive Drugs* 1997;29:165–183).

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CHPR: Are there medications that need to be discontinued before infusion?
Dr. Brody: The main one is benzodiazepines. They can blunt ketamine's antidepressant effects, likely because of how they affect GABAergic interneurons that modulate glutamate circuits. We taper or minimize them when possible. The other is naltrexone, since it blocks ketamine's therapeutic effect and makes the experience unpleasant (Williams NR et al, *Am J Psychiatry* 2018;175(12):1205–1215).
CHPR: If a clinician wants to begin using ketamine, what would you advise?
Dr. Brody: Ketamine is used all across medicine—emergency, anesthesia, pain medicine—so there's a lot of expertise out there. Having an anesthesiologist join you for the first few cases is exactly how I learned, and it's a sensible way to get comfortable. The APA has clear guidance on how to administer ketamine safely (Sanacora et al, 2017) and the REMS program for esketamine is also very straightforward.
CHPR: Are there any final things we should know?
Dr. Brody: I'd emphasize that inpatient psychiatry is uniquely suited for ketamine. Units already have nurses, the ability to give IV fluids, and the capacity to monitor vitals and post-infusion recovery. And we have social workers who can connect patients to outpatient ketamine or esketamine programs, which is essential because ketamine's benefits are transient. The "expertise" in ketamine isn't just giving the drug. It's understanding the literature, patient selection, risks, and the importance of follow-up. Ketamine is in its own therapeutic domain and requires real familiarity to use safely and effectively.
CHPR: Thank you for your time, Dr. Brody.

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Genetic Conditions With Psychiatric Manifestations

Courtney S. Roberts, MD. Psychiatrist, Olive View-UCLA Medical Center, Los Angeles, CA.

Victoria Hendrick, MD. Chief, Inpatient Psychiatry, Olive View-UCLA Medical Center; Editor-in-Chief, The Carlat Hospital Psychiatry Report.

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Aiden is a 24-year-old man with autism and moderate intellectual disability (ID) admitted to the psychiatric unit after a sudden worsening of anxiety and behavior. He's always had trouble with loud noises and other sensory experiences, but things escalated recently after he witnessed a serious car accident. He's been pacing constantly, crying for hours, biting his nails until they bleed, and hitting his head hard enough to cause bruises. He's also been refusing most meals. His medical history includes mitral valve prolapse, and his family history includes a maternal uncle and cousin with ID and autism. Aiden has never had genetic testing.

After encountering a case like Aiden's, you might wonder whether a genetic condition could be contributing to his presentation. Many genetic neurodevelopmental disorders are identified in childhood, but not all. Some patients reach adulthood undiagnosed, particularly if earlier evaluations were limited or never pursued. In fact, if you test patients with autism, intellectual disability, or developmental delay, nearly one in five will have an identifiable underlying genetic cause (Muhle RA et al, *J Am Acad Child Adolesc Psychiatry* 2017;56(11):910–913). Identifying that cause can reshape your treatment plan, flag medical issues that need monitoring, and prompt evaluation of at-risk relatives.

When to suspect a genetic condition

A few red flags should prompt a deeper look. Autism, ID, or global developmental delay are the most common clues. Dysmorphic features or congenital anomalies, like atypical facial traits and cardiac or renal defects, add to the suspicion. A family history of ID or autism in multiple relatives is another strong signal. Repetitive or stereotyped behaviors like hand-flapping or rocking may point to specific syndromes, and new-onset cognitive or functional decline in adolescence or early adulthood can indicate a previously unapparent genetic condition.

Aiden has autism and ID—that's your first clue. He also has mitral valve prolapse, which is a known feature of fragile X syndrome. And his maternal uncle and cousin both have ID and autism, suggesting an X-linked inheritance pattern. These details together should put Fragile X near the top of your differential.

Syndromes psychiatrists are most likely to encounter

22q11.2 deletion syndrome (DiGeorge syndrome)

This is one of the most underrecognized genetic conditions in adult psychiatry. Up to 30% of individuals with this deletion develop psychosis, often resembling schizophrenia—making it one of the strongest known genetic risk factors for psychotic illness (Schneider M et al, *Mol Psychiatry* 2014;19(11):1205–1211). If you have a patient with treatment-resistant psychosis who also had a cleft palate repair as a child or a history of congenital heart disease (especially tetralogy of Fallot), think 22q11.2. Other clues include a history of hypocalcemia, immune deficiency, and subtle facial features like hooded eyelids or small ears. Anxiety and ADHD are also common. The deletion is found in roughly 1 in 4,000 live births, so it's not as rare as it sounds.

Fragile X syndrome

Fragile X is the most common inherited cause of intellectual disability, and it's the condition most relevant to Aiden's case. Males typically present with moderate to severe ID, autism (in 25%–50% of cases), ADHD, anxiety, and stereotypies like hand-flapping. The physical exam may show a long face and prominent ears. Post-pubertal males may also have macroorchidism (enlarged testes). Mitral valve prolapse and joint laxity are also common. An important detail for family counseling: Female carriers of the premutation may have milder symptoms like anxiety, attention problems, or learning difficulties, and they are at risk for fragile X–associated primary ovarian insufficiency (Hagerman RJ et al, *Curr Pediatr Rev* 2008;4(1):40–52).

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Prader-Willi syndrome
You're most likely to encounter Prader-Willi in the context of behavioral crises. Patients typically have mild to moderate ID, and the syndrome's hallmark is hyperphagia—an insatiable drive to eat that emerges in childhood and leads to severe obesity if food access isn't carefully managed. Psychiatrically, you'll see compulsivity (skin picking, hoarding), emotional dysregulation with intense tantrums, anxiety, depression, and in some cases psychosis. Excessive daytime sleepiness is also common. The behavioral profile is distinctive enough that if a patient with ID presents with both compulsive eating behaviors and skin-picking, Prader-Willi should be on your list.

Less commonly seen by psychiatrists

Several other genetic syndromes have psychiatric features but are generally diagnosed in childhood and managed primarily by other specialties. You're unlikely to be the first to identify these, but you may see these patients for comorbid psychiatric care. For a quick guide to key features, see the "Genetic Syndromes Relevant to Psychiatric Practice" table below. For a detailed supplement with resources, see: www.thecarlatreport.com/geneticconditions.

Genetic Syndromes Relevant to Psychiatric Practice

Down syndrome (trisomy 21) *Physical:* Characteristic facial features, congenital heart defects, mild–moderate intellectual disability (ID). *Psychiatric:* Depression, anxiety; elevated early-onset Alzheimer's risk.
Rett syndrome *Physical:* Normal early development, then regression with handwringing and seizures. *Psychiatric:* Severe ID; mainly supportive/behavioral interventions.
Turner syndrome (45,X) *Physical:* Short stature, webbed neck, ovarian insufficiency, congenital heart defects. *Psychiatric:* ADHD, anxiety, social difficulties.
Williams syndrome *Physical:* "Elfin" facial features, short stature, cardiovascular/renal abnormalities, mild ID. *Psychiatric:* Socially gregarious but anxious; ADHD, specific phobias, depression; strong affinity for music.

Understanding genetic testing

Genetic testing in this context isn't a single test. Instead, think of it as a toolkit with different levels of resolution. Chromosomal microarray (CMA) is the workhorse. It scans the genome for missing or extra segments of DNA and catches most of the deletions and duplications behind these syndromes. Fragile X testing is a separate, targeted test because the specific trinucleotide repeat expansion that causes fragile X doesn't show up on CMA. Karyotyping is an older test that examines whole chromosomes under a microscope; it's useful when you suspect something like Down syndrome or Turner syndrome, but it misses the smaller changes CMA can detect.

For most patients with unexplained ID or autism who haven't been tested, start with CMA plus fragile X testing. The American Academy of Pediatrics recommends this combination as the first-tier workup, and the same logic applies in adults who were never evaluated (Schaefer GB and Mendelsohn NJ, *Genet Med* 2013;15(5):399–407). If physical features point toward a specific whole-chromosome condition (eg, suspected Down or Turner syndrome), add karyotyping. If first-tier testing is negative but your clinical suspicion remains high, refer to a geneticist. More comprehensive sequencing tests that analyze many genes at once may be the next step. Coordinate the workup with a genetic counselor to ensure appropriate pre- and post-test counseling and interpretation of results.

Practical considerations

Insurance generally covers CMA and fragile X testing when the patient has documented developmental delays, autism, or ID. Preauthorization requirements vary by plan, so check before ordering. Out of pocket, CMA typically runs $500–$1,500, while fragile X testing is in the range of $300–$500. Results usually take two to four weeks. If testing can't be completed during an inpatient stay, initiate an outpatient referral. Don't let the logistics of hospitalization become a reason to skip it. You can help connect patients and families with genetic counselors through the National Society of Genetic Counselors at findageneticcounselor.nsgc.org.

Genetic testing can bring enormous relief to families who have struggled for years without answers, and a clear diagnosis often reduces guilt or self-blame. But testing can also uncover incidental findings—like a predisposition to BRCA-associated cancers—that raise new anxieties. Discuss these possibilities with patients and families before ordering tests so they're prepared for unexpected results.

Treatment approaches

You can manage most psychiatric symptoms in these patients with a combination of medications and behavioral supports, guided by the patient's specific symptoms, functional level, and medical comorbidities. SSRIs are typically first line for anxiety or obsessive-compulsive symptoms. Start at low doses (eg, escitalopram 5 mg, sertraline 25 mg). For acute anxiety or agitation, hydroxyzine 25–50 mg as needed is a reasonable option. For irritability or aggression, aripiprazole (starting at 2 mg) or risperidone (starting at 0.5–1 mg) are both FDA approved for irritability in autism. The general rule is to start low and go slow, as these patients are often more sensitive to side effects (for more treatment approaches, see: www.thecarlatreport.com/geneticconditions).

Establishing a genetic diagnosis does not usually change day-to-day psychiatric management. Treatment remains symptom-based: Anxiety is treated as anxiety, psychosis as psychosis, irritability as irritability. The value of identifying the genetic condition is that it can guide medical monitoring, clarify prognosis, reduce family uncertainty, and prompt testing of at-risk relatives. A diagnosis may also help clinicians anticipate vulnerabilities (eg, higher psychosis risk in 22q11.2 deletion) or medication sensitivities.

Behavioral strategies

In inpatient psychiatry, behavioral interventions are often the most effective tools for stabilization. Prioritize structure and predictability: Maintain consistent routines, use visual schedules, and minimize unstructured time. Identify sensory triggers (noise, lighting, crowding) and modify the environment as needed. Quiet spaces, reduced stimulation,

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or sensory supports (eg, weighted blankets, fidget tools) can significantly reduce agitation.

Use a functional approach to behavior. Try to figure out what the patient's behavior accomplishes—escape, attention, sensory input, or access to preferred items—and intervene at that level. Teach and reinforce replacement behaviors (eg, using a "break" request instead of self-injury). Keep communication simple and concrete, and rely heavily on positive reinforcement. For syndrome-specific risks (eg, food seeking in Prader-Willi), restrict unsupervised food access, use locked storage, schedule structured meals, and maintain consistent staff limit-setting.

During Aiden's hospitalization, you start escitalopram 5 mg daily for his anxiety and hydroxyzine 25 mg as needed for agitation. Structured routines and sensory supports reduce his pacing and self-injury. CMA and fragile X testing confirm fragile X syndrome. You discuss the diagnosis with his mother, explain the X-linked inheritance pattern, and recommend genetic counseling and testing for female relatives who may be carriers. The social worker connects the family with the National Fragile X Foundation and arranges in-home supportive services. You schedule follow-up with a clinical geneticist for long-term cardiac surveillance of Aiden's mitral valve prolapse.

CARLAT VERDICT

We often care for patients with developmental disorders, but it's easy to miss an underlying genetic diagnosis, especially in adults who weren't tested as children. Subtle physical features, family history patterns, or distinctive behaviors may point to a genetic syndrome. While establishing a diagnosis may not dramatically change immediate psychiatric treatment, it can clarify medical monitoring needs, guide family counseling, and anticipate long-term risks. If the picture seems suggestive, start with CMA and fragile X testing. Genetic counselors can help families navigate the diagnosis and coordinate ongoing care.

Research Updates IN PSYCHIATRY

ANTIPSYCHOTICS

Prenatal Antipsychotic Exposure: Reassuring Long-Term Data

Victoria Hendrick, MD.

REVIEW OF: Bruno C et al, *eClinical Medicine* 2024;70:102531; Straub L et al, *JAMA Intern Med* 2022;182(5):522–533; Swetlik C et al, *J Clin Psychiatry* 2024;85(1):23m14965

STUDY TYPE: Three cohort studies—one multinational registry study, one national birth registry, and one prospective clinical registry

Antipsychotics are widely used in reproductive-age women, and families want to know the long-term risks for kids. These three complementary cohorts point in the same direction.

Straub followed 3.4 million births from US claims (Medicaid + commercial) up to 14 years. After adjusting for maternal illness and other confounders, late-pregnancy antipsychotic exposure was not meaningfully linked to neurodevelopmental disorders (adjusted HR ≈ 1.08). A small statistical signal (meaning a possible association) surfaced for aripiprazole (HR ≈ 1.36) but needs replication.

Swetlik's prospective registry compared 178 children exposed to second-generation antipsychotics (mostly quetiapine, aripiprazole, and lurasidone) with 174 unexposed peers at preschool age. On validated parent-rated tools, global development and behavior were similar. An initial increase in abnormal communication scores disappeared after adjusting for maternal diagnosis and breastfeeding.

Bruno pooled Nordic registry data (Denmark, Finland, Iceland, Norway, and Sweden) on 213,302 children of mothers with psychiatric diagnoses; 11,626 (5.5%) were exposed in utero, most often to quetiapine or olanzapine, with a median 6.7-year follow-up. They examined timing by trimester and found similar results whether exposure occurred early, late, or throughout pregnancy. Prenatal exposure didn't raise the already low risk of intellectual, speech/language, or learning disorders (composite aHR ≈ 1.06), and school performance was similar (math aRR 1.04; language aRR 1.00). Results were consistent regardless of the specific drug or timing of exposure. A small, uncertain chlorpromazine signal for speech/language disorders appeared but was based on few cases.

CARLAT TAKE

Across large national databases, a prospective registry, and a multinational Nordic cohort, prenatal antipsychotic exposure wasn't linked to later developmental, behavioral, or learning problems. Small possible increases in risk with aripiprazole and chlorpromazine are worth keeping an eye on, but for now the main message stands: Once maternal diagnosis and other confounders are accounted for, antipsychotic exposure itself does not show a meaningful independent association with adverse neurodevelopmental outcomes. Keep monitoring exposed children as a precaution, but you can reassure parents that long-term neurodevelopmental outcomes look good.

SUICIDALITY

Risk of Suicide Across Medical Conditions and the Role of Mental Disorder

Victoria Hendrick, MD.

REVIEW OF: Østergaard SD et al, *JAMA Psychiatry* 2024;81(12):1198–1206

STUDY TYPE: Retrospective cohort study

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CME Post-Test

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For those seeking ABPN Self-Assessment (MOC) credit, a pre- and post-test must be taken online at http://thecarlatcmeinstitute.com/self-assessment/

This page is intended as a study guide. Please complete the test online at www.TheCarlatReport.com.

1. A patient in the inpatient unit begins pacing and raising their voice. What is the most appropriate initial response based on evidence-based de-escalation principles?
   [ ] a. Move closer to the patient to demonstrate engagement and maintain eye contact
   [ ] b. Provide clear behavioral limits and outline consequences if escalation continues
   [ ] c. Maintain a calm tone, adopt a nonconfrontational stance, and respect personal space
   [ ] d. Offer immediate PRN medication to prevent further escalation

2. According to Dr. Mesbah, what is a limitation of the Brøset Violence Checklist when used in emergency departments (EDs)?
   [ ] a. It requires detailed psychiatric history unavailable in ED settings
   [ ] b. Its specificity is low, leading to frequent false positives
   [ ] c. Its sensitivity is under 50%, missing many patients who later become aggressive
   [ ] d. It cannot be completed within 24 hours

3. According to Dr. Brody, which of the following is a key practical advantage of IV ketamine over intranasal esketamine in the inpatient setting?
   [ ] a. It has a longer half-life, allowing once-monthly dosing
   [ ] b. It allows precise weight-based dosing and can be stopped quickly if adverse effects occur
   [ ] c. It does not require monitoring of vital signs
   [ ] d. It has demonstrated superior long-term monotherapy efficacy

4. In patients with autism or intellectual disability, approximately how many may have an identifiable underlying genetic condition on testing?
   [ ] a. 1 in 50 [ ] b. 1 in 20 [ ] c. Nearly 1 in 5 [ ] d. Nearly 1 in 2

5. Which of the following best reflects current evidence regarding prenatal antipsychotic exposure and long-term neurodevelopmental outcomes in children?
   [ ] a. Prenatal antipsychotic exposure substantially increases the risk of autism and intellectual disability
   [ ] b. After adjusting for maternal psychiatric illness and other confounders, prenatal antipsychotic exposure is not independently associated with adverse neurodevelopmental outcomes
   [ ] c. Children exposed in utero to second-generation antipsychotics have significantly worse developmental and behavioral scores at preschool age
   [ ] d. Antipsychotic exposure during the first trimester carries a higher risk of learning and language disorders than exposure later in pregnancy

6. On an inpatient psychiatric unit, a patient's Dynamic Appraisal of Situational Aggression score increases from 1 to 3 over 24 hours despite de-escalation efforts. Based on validation data, what is the most accurate interpretation of this change?
   [ ] a. The patient has crossed a high-risk cutoff threshold
   [ ] b. The patient's aggression risk has approximately doubled
   [ ] c. The patient's aggression risk has increased substantially, with each one-point rise associated with roughly triple the risk
   [ ] d. The score increase reflects subjective staff concern rather than a measurable change in risk

7. According to Dr. Brody, therapeutic doses of ketamine used in clinical practice have been shown to cause progressive cognitive decline over long-term follow-up.
   [ ] a. True [ ] b. False

8. A 32-year-old woman comes in for anxiety and long-standing attention problems. Her nephew has fragile X syndrome. What should you keep in mind during her psychiatric evaluation?
   [ ] a. Fragile X premutation carrier status can be associated with anxiety and attention symptoms
   [ ] b. Premutation carriers commonly develop early-onset schizophrenia
   [ ] c. Premutation carriers are generally protected from mood and anxiety disorders
   [ ] d. Psychiatric symptoms only occur in carriers after ovarian failure develops

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Research Updates

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Suicide prevention often focuses on psychiatric illness, but this large Danish study shows that medical conditions themselves can substantially raise suicide risk. Researchers analyzed data from over 6.6 million adults in Denmark (2000–2020), examining 31 medical conditions across nine categories. They also tested whether having a prior mental disorder compounded suicide risk. In total, 12,876 suicides were recorded.

Most medical conditions were linked to higher suicide rates, particularly gastrointestinal disorders (about 70% higher), cancers (50% higher), and blood-related illnesses such as HIV/AIDS (50% higher). The risk rose with increasing disability burden—a clear dose-response pattern. However, for patients with prior mental illness, additional medical conditions didn't further raise suicide risk, likely because their baseline risk was already high.

CARLAT TAKE

Serious medical illness alone elevates suicide risk, so screening shouldn't stop at psychiatric diagnoses. Stay alert for suicidality in patients with complex or disabling conditions, especially soon after diagnosis. For those with mental disorders, risk remains high regardless of physical comorbidity—another reason to maintain close monitoring and proactive safety planning.

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