Editor: Eleni K. Maneta, MD

Ambushed by Memories of Trauma: Memory-Processing Interventions in an Adolescent Boy with Nocturnal Dissociative Episodes

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Keywords: dissociation, functional neurological symptoms, memory processing, non-epileptic seizures, PTSD, structural dissociation of the personality

CASE HISTORY

At presentation to the Accident and Emergency Department, BJ was a 16-year-old boy in eleventh grade, with a one-month history of unusual sleep-related behaviors. As the latest example, BJ had awakened one morning to find a painful cut on his arm and a bloodied knife in the kitchen; he had no memory of what had happened. BJ was admitted to the hospital, jointly, under a neurologist (RW), a sleep-medicine physician (KW), and a psychiatrist (KK).

Background

BJ lived with his biological mother, his stepfather of ten years, his full biological younger brother, aged 14, and his half-brother, aged 7. BJ's biological parents acrimoniously separated when BJ was 4 years old, in the context of domestic violence. Following the separation, the Family Court mandated that BJ and his brother have regular visits with their father. The visits continued for seven years, until BJ was 11. During that seven years, the father subjected BJ—and to a lesser extent his younger brother—to physical and emotional abuse, but neither of the children told their mother about it. BJ later reported

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Supplemental digital contents are available for this article. Direct URL citations appear in the printed text and are provided in the HTML and PDF versions of this article on the journal's Web site (www.harvardreviewofpsychiatry.org).

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DOI: 10.1097/HRP.0000000000000195

that he had thought, as a boy, that his father had the right to do such things.

At 11 years of age, BJ was diagnosed with posttraumatic stress disorder, for which he received therapy. His therapist recalled BJ as a hypervigilant boy who showed a heightened startle response to touch and who suffered from nightmares, anticipatory anxiety, and panic attacks. At the age of 13, after a prolonged and acrimonious family court case, BJ was placed in full custody of his mother and ceased having any further contact with his father. Although BJ's overall functioning improved over time, he continued to experience intermittent panic attacks and fears of being kidnapped by his father.

BJ's medical history was significant for type 1 diabetes (first diagnosed at age 4) and for long-standing hyperhidrosis and fainting episodes associated with orthostatic stress and hot weather. Some of these faints were followed by tonic/clonic-like movements, consistent with seizure-like movements that occur in the context of hypoxia. He also experienced some atypical fainting episodes that were followed by sustained periods of unconsciousness lasting up to 20 minutes. BJ's neurologist hypothesized that these episodes were stress related—that is, psychogenic non-epileptic seizures.

During primary school and the early years of high school, BJ had trouble making friends and was a frequent target of bullying, in part because of his diagnosis of diabetes but also because of his sensitivity to interpersonal situations in which he perceived himself or other children to be victimized. By middle adolescence, BJ had grown into an outspoken student with a close circle of friends.

Three months prior to the onset of the nocturnal episodes, BJ experienced a number of stressful events at school. His relationship with staff and the school administration had become increasingly adversarial over the issue of accommodating his medical needs. Due to BJ"s hyperhidrosis, he needed an airconditioned room at school, and as his fainting episodes

became more frequent, his medical absences from school increased. In addition, he had been caught trying to cover up for several students who had been playing a game involving the use of mobile phones. The students ended up in trouble anyway, and BJ's teachers turned on him, too. BJ said that "they were all against me, and the more I tried to do, the worse it got." He tried not to burden his mother with these difficulties and was left trying to manage them alone. He was unable to confide in his stepfather because their relationship was distant, despite living in the same house. BJ had felt so helpless and frustrated that he had considered dropping out of school altogether.

History of Presenting Illness

In the month prior to admission, there were four episodes of unusual sleep-related behavior. During the first episode, BJ had shaved a portion of his scalp with a shaving razor. Although he could not remember shaving his head, he did recall a nightmare in which he needed to enter the witness protection program after testifying against his father in court. Prior to the second episode, BJ had fallen asleep at his computer. He then awoke in an altered state, during which he looked at his mother with "an intense stare" but did not appear to recognize her. He searched frantically for his mobile phone, shouting that his phone had been moved. His mother found the phone, spoke to him calmly, and placed the phone on his bed. BJ then went back to sleep. During the third episode BJ shaved a second portion of his scalp. The fourth episode pertained to the laceration to his wrist (mentioned above) and his presentation to the emergency department.

Assessment Phase

On admission, BJ's general physical examination, neurological examination, bedside cognitive testing, basic lab work, video electroencephalogram (vEEG), and brain MRI were all normal. Percutaneous PCO2 readings done during the routine vEEG showed a skewed hyperventilation-challenge profile characterized by an exceptionally low trough-level PCO2 (22 mm Hg after three minutes of hyperventilation, and 17 mm HG after five minutes), followed by a failure to recover homeostasis (PCO2 ≥ 36 mm Hg), suggesting that hyperventilation-induced changes in brain physiology could be contributing to BJ's clinical presentation.^{2,3} Further laboratory work, including inflammatory markers, an autoimmune screen, thyroid function tests, and morning cortisol were all within the normal range. Serial hemodynamic parameters showed frequent mild tachycardia, no orthostatic hypotension, and mild hypertension. Initial diagnostic considerations focused on three broad diagnostic categories: a parasomniarelated disorder,* an epileptic illness, or a psychiatric illness.^{4,6,7}

PSYCHIATRIC ASSESSMENT The family and individual assessments took place while medical investigations were in process. BJ presented as warm, talkative, and forthcoming. He described himself as sensitive, gentle, and caring. He recounted how he

avoided physical sports because he did not like violence, and was proud that he had "never hit anyone." BJ's mother likewise impressed upon the team that BJ was "nothing like his father." BJ's role as a caregiver to others emerged strongly in the family story, where he described how he had tried to protect his younger brother from his father.

During the process of sharing the family story, BJ and his brother recalled incidents of abuse that had occurred during visits with his father; examples included being strangled, being thrown down stairs, and being forcibly held under water in a swimming pool. At various difficult points in the story, the tension and emotion in the room was palpable, and family members became teary or emotional.

BJ and his mother could not explain his nighttime episodes. BJ said that he was puzzled that his father still appeared in his dreams because "I never even think of him anymore." BJ's arm-cutting episode at night did not make sense to him or his mother because both thought he had been upbeat and optimistic for the past year. For BJ and also for his mother, the most frightening aspects of the episodes were the loss of control and the fear that BJ might hurt himself or a family member.

From the point of view of the psychological medicine team, BJ presented as an adolescent with a significant history of trauma, much of which remained unprocessed, as evidenced by the intense affect and palpable physiological arousal during assessment. In this context, a referral was made to a therapist (LM) who specialized in eye movement desensitization and reprocessing (EMDR), radical exposure tapping, and progressive counting ^{8–10}—forms of memory processing that use exposure (see Supplemental Text Box, available online at http://links.lww.com/HRP/A71). ^{9–17} The first appointments with LM took place while the medical assessment was being completed.

Neurological and sleep-medicine assessments The results of a sleep-deprivation sleep study¹⁸ were equivocal. Neither sleep deprivation nor auditory stimuli induced parasomnic events, though BJ did have four awakenings out of slow-wave sleep, a number considered as weakly positive for a possible parasomnia disorder.¹⁹

A trial of clonazepam (1 mg at bedtime)—the empirical treatment for parasomnias²⁰—showed a worsening of symptoms, suggesting that BJ did not suffer from a parasomnia. Whilst medicated with clonazepam, BJ experienced an increased number of nocturnal episodes: punching the wall with his fist; staring at staff intensely; clenching his fists as if

^{* &}quot;Parasomnias are sleep disorders characterized by undesirable behavioral or experiential phenomena occurring during NREM, REM or during transitions to and from sleep." ^{4(p-13)} Studies using stereo-electroencephalography suggest that they involve a dissociation between brain areas underpinning mental arousal and those underpinning motor arousal—in particular, a sleep EEG pattern in some brain regions and a wakefulness pattern in others (e.g., motor and cingulate cortices). Migratory birds are the animal model for these brain changes; during long flights, they induce sleep in one hemisphere but not the other, enabling them to sleep (half a brain at a time) during long flights. ⁵

to strike and shouting "get away from me"; stomping through the ward and throwing a microwave on the floor; and smashing his insulin pump. After each episode, BJ would fall asleep and wake in the morning bruised and with no recollection of events. There were also two episodes of prolonged unresponsiveness—one following a wall-banging episode during the night and another following a faint during the day. On both occasions, he was unresponsive to noxious stimuli (sternum rub) and, upon regaining consciousness, described the sensation of being unable to move his body, suggesting a period of motor immobility. Nursing observations showed mild tachycardia but no orthostatic hypotension. The clonazepam was discontinued.

During ambulatory EEG monitoring on the ward, a nurse found BJ asleep with the taps in his bathroom running, his room flooding, and the portable EEG machine soaking in the sink. The recovered trace of the EEG at the time of its disruption was consistent with BJ being in a waking state (sustained EEG wakefulness).

The converging data suggested that BJ was not suffering from a parasomnia. Relevant in this context were the failure of sleep deprivation to precipitate events, the EEG finding of wakefulness at episode onset, the complexity of behaviors during the various episodes (including the presence of self-harm, which would be highly unusual for a parasomnia), the increased frequency of episodes since commencing clonazepam, and the intrusion of episodes into the daytime. The absence of epileptic activity during the recorded event also argued strongly against the events being epileptic.

HOSPITAL COURSE The risk that BJ posed to staff and other patients was increasingly a matter of concern, and a request was made for a bed in the mental health unit. Quetiapine 50 mg at bedtime was started, and increased to 200 mg, as a means of containing the nighttime episodes. This intervention, combined with BJ's distress about the prospective move to the mental health ward, shifted the timing of BJ's episodes from the night to the day:

- BJ fell asleep on the game-room sofa. Nursing staff witnessed him rouse from sleep, pick up the television, and throw it to the ground.
- A nurse observed BJ in a state of panic, hyperventilating at a breathing rate of 40 per minute. (Of note, BJ's automatic hyperventilation in response to trauma-related flashbacks of his father was much more vigorous than the intentional hyperventilation he induced during the vEEG.) After a few minutes, he collapsed from a sitting position to the ground, where he remained unresponsive for 30 seconds. Rising with an intense stare, he briefly looked around and ran out of his room, off the ward and out of the hospital, to a grassy embankment, where he

- collapsed in exhaustion. When he came back to himself, some minutes later, he was confused and disoriented, and could remember nothing of what had happened.
- After seeing a photograph of his biological father on Facebook, BJ hyperventilated and fainted. When he came to, he threw a chair, charged out of the hospital classroom, ran out of the hospital, and hid cowering and shaking in a corner of the parking lot.
- When BJ tried to explain to the child psychiatry fellow what had happened in the classroom, he hyperventilated, fainted, came to, surveyed the room, looked at the fellow with an "intense stare," stormed out of the room, and ran out of the hospital to the railway station, where he banged his forehead violently against a brick wall. When the train arrived, BJ boarded and continued banging his head against the train interior. After ten minutes, he collapsed and came to in a confused and distressed state with no memory of what had just occurred. After collecting himself, he was escorted back to the hospital by the fellow, who had been able to follow him onto the train.

Formulation and Treatment

FORMULATION The team met with BJ and his mother and stepfather to discuss the medical findings and the working formulation. We explained that BJ, rather than sleepwalking, was probably experiencing dreams that involved his father, from which he awoke "in flashback mode," thereby responding to the material in the dream as if the events were happening in the here and now. As the nighttime episodes were increasingly controlled through sedation, the episodes (flashbacks) shifted to the daytime. The flashbacks were inducing episodes of "fainting" (see below), from which he "awoke" in flashback mode, enacting three different behavioral scenarios: (1) a state of "fight" (clenched fists, throwing objects), in which BJ attempted to protect himself; (2) a state of "flight," in which BJ tried to protect himself by running away and hiding; and (3) a state of collapsed immobility, in which he was unresponsive for a long period of time, itself an innate mechanism that functioned to protect BJ from overwhelming arousal, fear, anger, or feelings of entrapment.²¹ All these innate defensive states—fight, flight, and collapsed immobility commonly occur in PTSD, and BJ's PTSD seemed to have been reactivated in the context of recent stressors at school, including the feelings of powerlessness that BJ had so clearly described in relation to his teachers. Because similar feelings of powerlessness had pervaded his childhood relationship with his father, they may have triggered past memories of him.

The linguistic problem of what to call the episodes when talking about them to other health professionals was also discussed with the family. Because BJ had a previous diagnosis of PTSD, and we could construe his episodes as a response to flashbacks, and because PTSD is well understood by medical professionals, we opted to retain this label. Also, discussed with the family, however, were the alternate diagnostic options including dissociative identity disorder and

 $^{^\}dagger$ This small dose of quetiapine was prescribed at night—rather than orally disintegrating olanzapine, which was used PRN—because it has less weight-gain potential.

unspecified dissociative disorder.^{22–26} We subsequently found historical case histories[‡] and more contemporary case histories (with concurrent data documenting EEG wakefulness) of individuals with presentations that were similar to that of BI.^{27–29}

Because BJ's mother and stepfather were very concerned about BJ's fainting episodes, we also discussed in more detail the biological mechanisms—known or hypothesized—that were likely to underpin those episodes (Supplemental Text Box 2, available online at http://links.lww.com/HRP/A71). 1,2,30–37 The discussion included: fainting in response to heat; fainting as a consequence of hyperventilation-induced cerebral hypoxia; and fainting as part of the innate defense response of collapsed immobility.

We also suggested to the family that their collective efforts to leave the family trauma story in the past, together with BJ's determination and his mother's expectation that BJ not be like his father, may have prevented BJ from expressing angry emotions and from processing memories associated with anger. In addition, BJ's need to take on caregiver roles and to distance himself from any strong negative feelings—powerlessness, fear, anger, and desire for revenge—may have made it difficult for him to address the trauma memories. In any case, the in-hospital admission presented an opportunity for BJ to process previously unprocessed memories and for him to come to accept all aspects of the self, both those that were friendly and sociable, and those that were frightened or angry with regard to earlier life events.

TREATMENT PLAN The suggested treatment plan was as follows. In hospital, BJ would be treated by the psychological medicine team, with the neurologist staying involved as needed. The psychological medicine team would use medication in an effort to keep things safe. Quetiapine and clonidine (titrated up to 50 mcg) would be used at night to keep BJ sedated and to preempt the extreme physiological arousal and fear that he experienced when waking from a nightmare. We would also trial fluoxetine 20 mg/day because it has been found to be potentially helpful for both PTSD³⁸ and fainting. ³⁹ In addition, antidepressants, which are known to have a neurogenesis effect, ^{40–42} could potentially support the reprocessing of trauma-related memories. The key interventions that would help BJ get well, however, involved the processing of trauma-related memories^{8,9,15,16,43} and the use of mind-body interventions with the psychological medicine team, who would provide BJ with strategies for managing body arousal;^{21,44} with time, he would become able to monitor and control that arousal with either lower doses of medication or even no medication at all. As previously noted, the referral for trauma processing had already been made, and the therapy began in parallel to the hospital intervention.

Nursing management in the acute mental health ward In the mental health ward, BI was nursed in a single room, separate from the main body of rooms and visible from the nursing station. The aim was to mimic the home environment and to practice "sleeping"—in a locked room, as it would occur at home. Although BI "hated" the mental health ward, he was settled there and went home daily for afternoon leave. The nursing staff were baffled as to the need for a high-security ward until an incident occurred. BJ had taken a shower and had come out anxious and hyperventilating, reporting that he was experiencing flashbacks of his father. A few minutes into the hyperventilation, BJ fainted but soon regained consciousness in an altered state. He ran into the nurse's station via an open door and attempted to activate the emergency button. He then broke through the locked door and, in the foyer, started banging his head against the wall. Following this "breakout" episode, BI began to access new memories, as we describe in detail below.

STABILIZATION AND PROCESSING OF TRAUMA MEMORIES IN THE INPATIENT SETTING Although treatment protocols for PTSD and dissociative disorders typically emphasize implementation of a stabilization phase prior to memory processing, ^{45–47} BJ's frequent switches into the flashback-related state, coupled with the high risk of harm associated with these switches, led us to implement the two phases of treatment in parallel. In that way, the stabilization component, implemented by the child psychiatry fellow and the psychiatrist in daily sessions, supported the memory-processing component involving radical exposure tapping and progressive counting with the therapist (LM), which took place across five sessions spread over four months (for more details, readers are referred to Supplemental Text Box 3, available online at http://links.lww.com/HRP/A71).^{9,10}

The key aim of the stabilization component of treatment was to help BJ manage his arousal. In order for the memory-processing therapy to work, BJ would need to keep his arousal in the window of tolerance;⁴⁸ states of very high arousal (his panic attacks with hyperventilation) or very low arousal (syncope) would impede memory processing. BJ responded to traumatic flashbacks in a stereotyped way: he experienced a spike of arousal; he hyperventilated, with his breathing becoming loud, deep, and very rapid; he fainted and then roused in an altered state. Because intense arousal was the first step of BJ's typical response, the psychiatry fellow and psychiatrist met daily with BJ to help him learn and practice grounding interventions that would help him manage states of increasing arousal (see Supplemental Text Box 3, available online at http://links.lww.com/HRP/A71).44 BJ also had access, as needed, to olanzapine 5 mg for times when he assessed that he would be unable to contain his escalating arousal. In addition, in preparation for the fourth session of radical exposure tapping and progressive counting, BJ was prescribed propranolol (10 mg morning and afternoon) to reduce his arousal. Once on propranolol, BJ reported that it

[‡] A fourteenth-century chronicle by Jean Froissart described the sleep-fighting of the knight Sir Pierre de Béarn, who rose from sleep each night to draw his sword and fight an invisible foe.²⁷ An account is also available in Trembinski (2011).²⁸

was easier to utilize the grounding strategies and that his overall level of anxiety was lower. Because of its inhibiting effect on melatonin (released at night from the pineal gland), propranolol was not used at night;⁴⁹ instead, we prescribed a bedtime dose of clonidine 50 mcg.

The key aim of the memory-processing component of treatment was for BJ to process the traumatic memories that had activated his nocturnal episodes and the states of high arousal that caused him to hyperventilate and experience episodes of fainting and unresponsiveness. In the memory-processing sessions utilizing radical exposure tapping⁸ and progressive counting^{9,10} (see Supplemental Text Box 1, available online at http://links.lww.com/HRP/A71), BJ was able to process, with support from the therapist, several traumatic memories involving his father: BJ's father standing over him with his fist raised while BJ cowered on the couch; BJ being left in a hot car and being threatened by his father when he tried to get out; and a memory of his father sexually assaulting him (see Supplemental Text Box 3, available online at http://links.lww.com/HRP/A71).

Vivid, intrusive memories of his father during the day gave BJ plenty of opportunities to practice his grounding interventions in his normal awake state—that is, before he switched into "flash-back" mode and before he lost his connection with the here and now. Following the fifth (and final) session of the memory-processing intervention, BJ decided that no further therapy involving memory processing was needed.

DISCHARGE PROCESS The discharge process took place over a six-week period. Leave was now increased to trial overnight leave; BJ was transferred back to the adolescent medical ward; and he attended the hospital classroom that serviced that ward. Eventually, he became a day patient.

During the discharge period, BI began to have conscious recollections of his experiences when he had been in "flashback mode." He now remembered that he had cut his arm because he imagined/remembered that he had been shackled by his father; the cutting was the means of removing the shackles. Likewise, he remembered why he had broken into the nurses' station, pressed the emergency alarm, and crashed through the locked door; it was an effort to escape from his father, given that every individual in the ward had taken on that identity. He also remembered that he had submerged the EEG machine, which he perceived as a bomb, planted by his father, that needed to be deactivated. Finally, BJ made sense of why he had repetitively banged his head when in "flashback mode." He said that, even though the memories seemed so real, some part of him recognized that the memories were in his head; the head banging was an attempt to dislodge the memory from that location.§

QUESTIONS TO THE CONSULTANTS

- 1. How can the treatment team position itself to best help the patient and family when the presentation does not neatly fit into known diagnostic categories and when the patient has no memory of the nighttime episodes? (Dr. Silberg)
- 2. Unusual presentations often necessitate clinical adjustments of one kind or another. Against that background, in what respects did the treatment team's approach in this case diverge from existing or emerging approaches to treating complex trauma? (Dr. Greenwald)
- 3. What is the role of medications in treating patients with complex trauma? (Dr. Ribeiro)

Joyanna Silberg, PhD

This case illustrates the overlap between the neurological considerations and posttraumatic psychological considerations that often come up when evaluators are faced with abnormal nighttime behaviors in maltreated children. There are few treatment centers familiar with how to respond to episodes of such great severity, intensity, and potential danger, and where the diagnosis appears so unclear.

At my practice at Sheppard Pratt Hospital in Towson, Maryland, patients with a similar presentation have been seen on many occasions. 50 We generally diagnose these kinds of waking or sleeping episodes of discontinuous consciousness as "dissociative episodes." Sometimes they may be posttraumatic flashbacks as they seem to be in this case, and at other times these episodes may represent behavioral enactments that are attempts to undo traumatic thoughts or memories but may not involve reliving episodes from the past. In one case I treated, a teenager woke up in a child-like state in which she recounted an abuse from her preschool years in a childlike voice, and had no memories of these nighttime episodes. This nighttime behavior was clearly not as dangerous or challenging as BI's nighttime reactions. Often our clients are able to identify descriptors of their actions—such as "fighting me" or "running me" or "scared me with nowhere to go." Even when they initially have no memory of the events, with gentle confrontation about what the family witnesses, often aspects of the experience return to them slowly, enabling them to describe it or name it in their own words. By having the client generate descriptive language about these behaviors—which mirror aspects of PTSD (fight, flight, or freeze), as the authors point out so astutely—the client can begin the process of more ownership of the behavior before traumatic reexposure.

In my practice, my emphasis with a client in this state would be on the mastery associated with the "undoing" of the traumatic experience, rather that exposure to the trauma per se.

While the client had been taught to stay within an appropriate level of tolerance to tolerate visualizing and experiencing the angry face of his father, I have found it more powerful, particularly with adolescent boys, to emphasize an active coping strategy rather than just the toleration of the image. For example, I might have the client learn to say self-empowering

[§] The case history was prepared by Lux Ratnamohan, MBBS, FRANZCP, Laurie MacKinnon, MSW, PhD, Melissa Lim, MPsyc(Clin), Richard Webster, MBBS, MSc, FRACP, Karen Waters, MBBS, FRACP, PhD, GCCM, and Kasia Kozlowska, MBBS, PhD, FRANZCP.

statements like the following: "I can now run away from you whenever I need to." "I was more powerful than you. You are not in our life anymore." "I survived your torture, and I am better than you because of it." "I am strong and capable of getting away from danger." The client would recite these while engaging in bilateral stimulation or self-soothing behaviors when confronted with a memory or image.

An additional intervention would be to identify the exact feeling and thought at the moment of perceived onset of the dissociative episode. In fact, BJ showed capacity to anticipate an episode and was told to ask for medication. He could also be taught to identify a thought and feeling associated with the prodromal experiences of an episode. Such a thought might be "being in this locked ward makes me feel so helpless like I did then" or "going to sleep makes me feel like I can be vulnerable and taken advantage of." It is hard to know what the precipitating feeling or thought might be, but many might have occurred while he was in the hospital, particularly on the locked ward. Then, in turn, he could have learned strategies to directly address those feelings and thoughts, which might avert the dissociative episode, without medication or perhaps supplemented with the medication. This exercise also helps the client learn that even vague reminiscences of feelings of helplessness or passivity might serve as triggers—such as when he was with his peers and felt isolated, or when he was blamed and not believed by the adults. Clearly, through his traumatic past he has learned to "avoid" or "dissociate" from the feelings often associated with a given experience, and this avoidance has, in the past, plunged him into dissociative reenactments of experiences related to those feelings. By learning what precursors there are, even if they are mild, the habit of feeling avoidance is reversed through this kind of work—which can then be generalized to other situations. Perhaps if he had more practice with immediate processing of feelings as they come up, he would have been able to tolerate the changing stressors in his life as time went on.

As I read this case history I am deeply affected by the horrific experiences of this young man. The abuse by a father is only partially described in terms of unbearable pain and the fear of death (potential strangling); such an experience also embodies a profound betrayal. Therefore, working with the client on these feelings must involve more than just mastering the experience of tolerating the memory. There is a huge amount of processing to do about the meaning of this betrayal to him personally—by his father, his mother (for allowing this to happen for so long, despite her best efforts), and the court system. This profound betrayal will affect his view of life, relationships, and the safety of closeness. These issues need adequate attention in a nuanced therapy relationship where issues of attachment and trauma are directly addressed.

Ricky Greenwald, PsyD

The case presented here highlights many of the challenges faced in diagnosing and treating clients with complex trauma.

Despite the treatment team's success, several of the assumptions underlying the treatment approach are potentially open to dispute, especially in light of emerging findings.

MINIMUM LEVEL OF AROUSAL IS REQUIRED FOR TRAUMA PROCESSING The case history states that a certain minimum level of arousal is required in order for trauma memories to be processed, but that is not necessarily true. At least one animal study found that chemically blocking the trauma memory–related emotional arousal did not prevent successful treatment. Furthermore, several cases were recently reported that demonstrated the potential effectiveness of the flash technique, 2 a new trauma-resolution method requiring memory retrieval but virtually no memory-related emotional arousal.**

STABILIZATION AND COPING SKILLS SHOULD BE ESTABLISHED PRIOR TO TRAUMA WORK Although extant treatment guidelines recommend establishing stabilization and coping skills prior to trauma-resolution work, these recommendations have been challenged⁵³ as not being based on evidence. Since clients are destabilized because of their traumatization, resolving the trauma is a more effective stabilizer than the so-called stabilization interventions.⁵⁴ When intense work is possible, the trauma memories can be treated for many hours per day, to get the trauma-resolution job done quickly; even with a highly unstable patient, this method is feasible while in an inpatient setting. Intensive trauma-focused therapy has been found to yield just as good results as the same treatment when delivered in conventional format (weekly outpatient therapy sessions)—but, of course, much more quickly.^{55–57}

TREATMENT IS COMPLETED WHEN THE CLIENTS CAN COPE WITH THEIR TRAUMATIZATION The decision to discharge a client who is still frequently experiencing severe symptoms such as intrusive memories is problematic. In current clinical practice we have trauma treatments (such as eye movement desensitization and reprocessing, and progressive counting) that facilitate memory reconsolidation, effecting true healing of trauma memories. And we are now all too aware of the potential long-term consequences of allowing children to become adults who remain wounded by unresolved traumatic memories. The only question should be whether trauma-focused therapy should terminate as soon as the symptoms are gone, or should persist until all the trauma memories have been resolved, as a prophylactic against further vulnerability. 60,61

Monique Ribeiro, MD

The combination of psychotherapeutic (i.e. psychoeducation, cognitive, behavioral, dynamic, and family-based interventions) and psychopharmacological approaches as part of a multimodal plan of care is often utilized in managing patients like BJ with complex PTSD presentations. While the efficacy of different psychotherapies has been established through

^{**} The flash technique was not available at the time that BJ was treated.

randomized, controlled trials, the use of psychopharmacology is generally based on different clusters of symptoms (for instance, hyperarousal) or presence of psychiatric comorbidities. The selective serotonin reuptake inhibitors (SSRIs) paroxetine and sertraline have received Food and Drug Administration (FDA) approval for treating PTSD in adults, ⁶² but no specific agents have been approved for treating childhood PTSD. Nevertheless, given that comorbid disorders are common in children with PTSD and that effective treatment of a particular symptom (such as sleep difficulties, aggression, and irritability) can improve quality of life and the ability to engage in multimodal treatment, pharmacotherapy has often been used in this population. ⁶³

In the present case, several classes of psychotropics targeting different symptom clusters were used to achieve safety and to stabilize the patient for optimal engagement in memory-processing therapy. The alpha-2 agonist clonidine and the atypical antipsychotic quetiapine were dosed at night for sedation and to decrease physiological arousal. Quetiapine and olanzapine, which were used to contain agitation, also carry an additional benefit for mood stabilization. Propranolol, a centrally acting nonselective beta blocker, was also prescribed to decrease hyperarousal, and the SSRI fluoxetine was used to treat mood and anxiety symptoms.

Of the three symptom clusters in PTSD (reexperiencing, avoidance, and hyperarousal), the symptoms of hyperarousal may be the most amenable to pharmacologic intervention. Sleep disturbance, irritability, difficulty concentrating, hypervigilance, exaggerated startle responses, and outbursts of aggression represent states of increased physiological arousal.⁶⁴

In patients with PTSD, the trauma-mediated activation of the sympathetic nervous system, with subsequent release of catecholamines, is responsible for a "sympathetic arousal" state, contributing to mood dysregulation, anxiety, and deficits in working memory, thinking, and perception. Adrenergic agents, such as propranolol and clonidine, have been shown to decrease startle responses and physiologic lability, and to improve anxiety, concentration, mood, and behavioral impulsivity in children with PTSD.⁶⁴ Less sedating than clonidine, propranolol was used in this patient's case to decrease arousal during the day, whereas clonidine was dosed at night to assist with sleep. Both of these agents fulfilled a fundamental goal of psychopharmacology prescription: provide symptomatic relief in a way that facilitates engagement in other aspects of treatment—in BI's case, to make it easier for him to utilize the grounding strategies, and to decrease his overall level of anxiety.

Antipsychotics ameliorate psychotic symptoms and aggression in PTSD patients but do not seem to be useful for core PTSD symptoms. Previously, a number of small single-site studies suggested that atypical antipsychotics were an effective adjunctive treatment for patients with PTSD who had poor responses to first-line SSRIs or serotonin-norepinephrine reuptake inhibitors. ⁶⁵ However, a recent large-scale, multisite trial of risperidone as an adjunctive agent for poor/partial

responders to SSRIs showed no added benefit versus the control group. ⁶⁶ These agents are not FDA approved for child-hood PTSD and should be reserved for severe cases in which psychotic symptoms, severe aggression, or self-injurious behaviors complicate management. ⁶² In BJ's case, since the threat posed to himself, staff, and other patients was increasingly a matter of concern, the use of atypical antipsychotics was appropriate; they helped to contain his aggressive episodes in the context of dissociation, creating the relative safety that enables the treatment to progress.

Finally, the SSRI fluoxetine was used in BJ's case to address mood and anxiety symptoms. Serotonin's role in drive satiety, mood, aggression, anxiety, and impulsivity is well established, and, as such, it likely has an important role in mediating PTSD symptoms.^{67,68} Currently, two SSRIs, sertraline and paroxetine, are FDA approved for treating PTSD symptoms in adults, and favorable results have also been reported for fluoxetine, fluvoxamine, and citalopram. 38,69 The latter of these was also studied in a 12-week, open-label study in children and adolescents with moderate to severe PTSD and shown to be effective, with a 38% symptom reduction at the end of treatment. 70 While SSRIs seem to be less effective for avoidance or numbing symptoms, the frequent presence of associated mood and anxiety symptoms, coupled with the overall good tolerability of SSRIs, provides a reason for their use.62

BJ's case highlights that, once clear symptom targets have been identified, the use of medications can provide significant symptom relief, increased safety, and ability to adhere to psychotherapeutic interventions, maximizing the chances of positive outcomes.

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the article.

We thank BJ and his mother for allowing us to share BJ's story and his journey through the health system with other clinicians. Like BJ, we hope that BJ's story will help other clinicians recognize and treat this pattern of presentation in other young people.

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