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Editorial

Mapping the Human Connectome

Rajiv Radhakrishnan, M.B.B.S., M.D.
Senior Deputy Editor

Although it has long been recognized that mapping the connections of the brain could help us better understand how it works, it is only in the past few years that this has become feasible. The early neuroanatomists Carl Wernicke, Paul Flechsig, Joseph Jules Dejerine, and Theodor Maynert recognized the importance of anatomical connectivity and the significance of white matter tracts in brain function but were limited by the technology available to them (1). Subsequent studies, such as those mapping neuronal connections in the nematode *Caenorhabditis elegans* (2) and those examining the relationship between neuronal organization in the macaque visual cortex and properties such as segregation and integration of visual processing (3), as well as the landmark analysis of the regional connectivity of the macaque visual cortex (4), which showed that specific cortical regions have specific inputs and outputs that form unique "fingerprints" that could distinguish one cortical region from the other, paved the way for studies on cortico-cortical connectivity. These investigations revealed that the brain is not just a network of neurons but that it is also organized hierarchically, with some regions showing greater connectivity than others.

The term "connectome" was first used by Olof Sporns (5) in a paper published in 2005 in which he outlined a detailed proposal for compiling the human connectome (which he defined as "a comprehensive structural description of the network of elements and connections forming the human brain") using a combination of structural and functional mapping techniques.

In 2010, the National Institutes of Health (NIH) awarded a total of $40 million in grants to the Human Connectome Project to map the connections of the human brain. The Human Connectome Project consists of two consortia: 1) the WU-Minn Human Connectome Project consortium, led by Washington University and the University of Minnesota, which was funded $30 million to characterize the relationship between structure and function using resting-state functional MRI (fMRI), task-based fMRI, and diffusion MRI in 1,200 healthy adults (consisting of monozygotic and dizygotic twin pairs and their siblings from 300 families); and 2) the MGH-UCLA Human Connectome Project consortium, led by Massachusetts General Hospital and UCLA, which was funded $8.5 million to optimize diffusion MRI technology for imaging the brain's structural connections.

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The Human Connectome Project consortia have thus far made remarkable technological advances in speed and spatial resolution of fMRI and diffusion MRI data acquisition such that whole brain coverage can now be accomplished in 0.7 seconds for fMRI and diffusion MRI, revealing intricate details of white matter connectivity. It is hoped that further advances in speed and spatial resolution would make it possible to capture real-time network dynamics.

The current methods, however, are far from perfect and have multiple technical limitations (6). Nevertheless, it is an exciting time for psychiatry as we wait to see if the Human Connectome Project, along with other connectome projects (such as 1,000 Functional Connectomes, the Brainnetome Project from China, the CONNECT Project in Europe, the Brain Initiative of NIH, and the Human Brain Project in Europe) are able to provide insight into the complexity of the human brain.

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Attending to Safety in the Psychiatric Interview

Tobias D. Wasser, M.D.

Learning how to safely conduct a psychiatric interview is imperative for the psychiatric trainee. However, residency programs generally do not provide thorough training in this area. In a national survey of psychiatric residents, one-third reported receiving no training in safety and violence management, and another third described training as inadequate (1). This is particularly concerning given that 36% of these residents reported having been physically assaulted by a patient (1). However, most individuals with mental illness will not act out violently (2), and the severely mentally ill are more likely to be victims of violent crime than perpetrators (3, 4). Nevertheless, given the high rates of resident assaults and insufficient training in safety and violence management, it is critical to refocus our attention on this area. The present review is intended to serve as a pragmatic guide for the psychiatric trainee on how to safely conduct a psychiatric interview.

Conducting the Interview

Attending to Safety

The resident should always make his or her own safety the first priority. An appropriate psychiatric evaluation cannot take place if the trainee is more concerned with being harmed than with the information the patient is communicating. The interview should not move forward until measures have been taken to ensure that the resident feels safely able to complete the task at hand. Implicit in this is the message that the interviewer must be cognizant of his or her own emotions during the interview and take action to change the situation if he or she feels frightened.

The next consideration should be for the safety of the physical space where the interview will occur. First, a clear route of egress from the room must be ensured (5). The next issue to consider is positioning in relation to the door. Patients prone to paranoia should be given easy access to the door, and the clinician should not place him or herself between the patient and the door (6). The safest choice is to arrange seating such that the interviewer and the patient have equal access to the door, without either acting as a barrier to the other's exiting (e.g., directly across from each other with equal access to the exit).

In outpatient work, any office is only as safe as the nearest available help. Ideally, other staff should be in the building, within earshot and aware that the clinician will be with a patient (6). If particularly worried, the clinician can arrange to keep the door partially open (6). Factors linked to higher rates of inpatient assault include times of transition (e.g., shift change), increased staff-patient interaction (e.g., medication administration), crowded/high-traffic areas, and decreased staff:patient ratios (7–9). In the emergency department, patients must be searched before meeting with the clinician (5). Additionally, the clinician should know how to urgently communicate the need for security personnel and should never hesitate in using such services to maintain safety.

Establishing Boundaries

Boundaries establish the limits of what will occur during the interview, both the physical interaction and the content of the discussion. Ideally, the interview should take place with both participants at the same eye level to reduce any perceived disparities in power (both sitting or standing) (10). The interviewer should be positioned closely enough to communicate easily but at least two arm's length away so that he or she is not immediately within striking distance should the patient become aggressive.

It is often helpful to explicitly state the goals of the interview at the outset to establish pertinent areas for discussion. An intake interview in the emergency department will be quite different from an initial visit for psychodynamic psychotherapy. Making clear the goals of the interview will help direct the patient to discuss content that will be appropriate and necessary for the situation without provoking unnecessary anxiety or arousal. One should also try to be cognizant of the impact the interaction is having on the patient. Particularly given the increase in the use of electronic medical records, the resident must be careful not to get so absorbed by checklists on the computer screen as to overlook signs that the patient is escalating.

Another way to establish boundaries is to engage the patient around the topic of safety. This provides an opportunity for collaboration around strategies for de-escalation should the patient become agitated. If during an interview you become concerned about your own safety, ask the patient if there is sufficient reason for you to be worried. This can be done by raising a concern about the patient's current level of volatility and your safety in his or her presence. One can then try to negotiate an agreement on how or whether to further conduct the interview (6).

Recognizing Signs of Escalation

The first sign that something is amiss often comes from within the clinician themselves, as one's own internal state is often the best tool available to determine whether a situation is safe. However, despite recognizing that something is not quite right, trainees will often suppress their own anxiety in unsafe situations. There are many possible explanations, most related to the systems within which residents work (e.g., being overworked and insufficiently supervised), rather than with the individual trainee. Another contributing factor is that trainees may respond psychologically to the fear of being harmed by repressing or denying this consciously intolerable experience, and, as a result, may even ignore data indicating a higher risk of violence (10).

To counteract this tendency, one should attempt to be more aware of one's own internal state, since there is evidence that psychiatrists who are able to acknowledge such fear but also express a desire to help are less likely to be injured by a threatening patient (11).

Recognizing the signs of escalation begins with having an awareness of the process in the room. A patient who is engaged in the interview and increasingly organized by questions is less of a risk, whereas a patient with poor compliance with the interview or who becomes increasingly agitated by questions is at higher risk (12). Aggression rarely occurs suddenly and unexpectedly. There is often a prodromal syndrome preceding violent behavior, consisting of increasing tension, verbal volume (as well as abuse/threats), motor activity (e.g., pacing), and clenching of the fists (10, 13).

How to Address the Escalating Patient

From a practical standpoint, an escalating patient should not be approached from behind because this may be perceived as threatening (10). The clinician should also be mindful never to turn his or her back to the patient (14). Volume, tone, and rate of speech should be decreased, while being careful not to be perceived as insulting (14). Maintain active eye contact; however, use caution because prolonged or intense eye contact can be perceived as threatening (15, 16). Using active listening techniques, such as briefly paraphrasing, convey an attempt to understand the patient's experience (10). Providing the patient with choices may help to increase his or her sense of control over the situation and, if possible, one can offer to accompany the patient to a calmer, less stimulating space (17).

An intensely angry patient may intimidate the resident and cause the resident to respond logically or rationally (10), which unfortunately may serve to further inflame the situation and should, ideally, be minimized (13). One de-escalation strategy is affect management, in which the primary goal is to teach the patient to reduce his or her internal state of tension by verbalizing feelings without resorting to violent confrontation (10). This involves the clinician allowing the patient to ventilate his or her affect, acknowledging it and validating it (when appropriate), as well as encouraging the patient to further discuss his or her feelings (13, 16). The clinician should focus less directly on the content of the patient's speech and, instead, repeatedly validate the patient's perceived emotional experience (18). However, one must first be sure the patient is capable of hearing and responding to these interventions.

If the above interventions are ineffective, it may be necessary to set limits with the patient. Utilized properly, limit setting can be therapeutic and avert violent behavior. The goal is to contain and counteract maladaptive behavior that threatens safety (19). However, responding with punitive threats in an attempt to set limits is not helpful, and doing so may actually increase the risk of violence by evoking feelings of impotence or humiliation (10). Effective limit setting involves clear identification of the specific behaviors that need to be altered and precise articulation of the potential consequences (19). The clinician must again assess whether the patient is capable of responding to this intervention (10).

If all else fails, the trainee should not hesitate to end the interview. In the outpatient setting, it may be necessary to facilitate the patient's transfer to a setting capable of treating psychiatric emergencies. Then, if necessary, mechanical restraints and/or medications can be utilized to help maintain safety.

Conclusions

Despite consistent research findings demonstrating low rates of violence among the mentally ill, a significant proportion of psychiatric residents are the victims of assault by their patients. Having an appreciation for how to safely conduct psychiatric interviews should help residents decrease their chances of being physically harmed by their patients.

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Family History of an Alcohol Use Disorder and Ketamine's Antidepressant Efficacy in Major Depression

Mark J. Niciu, M.D., Ph.D.

The *N*-methyl-D-aspartate receptor antagonist ketamine has rapid and relatively sustained antidepressant effects in major depressive disorder and bipolar depression. Family history of an alcohol use disorder in a first-degree relative has been identified as a positive predictor of ketamine's antidepressant efficacy. Glutamate neurotransmission (1) and glutamatergic mechanisms of disease in major depressive disorder (2) have been reviewed in the literature.

Subanesthetic Ketamine in the Treatment of Major Depressive Disorder and Bipolar Depression

Berman et al. (3) made the initial serendipitous discovery that a single subanesthetic (0.5 mg/kg) dose of ketamine rapidly improved depressive symptoms in a small (N=7) combined sample of unipolar and bipolar depression patients. This initial discovery was then extended to larger samples with treatment-resistant symptoms with both single (4–6) and repeated (7) dosing. Ketamine also has rapid antidepressant effects in major depressive disorder patients resistant to ECT and rapidly resolves suicidal ideation, including explicit and implicit measures of suicidal cognition. This latter finding has also been observed in acutely suicidal patients in an emergency psychiatric milieu. Nonintravenous ketamine preparations (oral, intramuscular, intranasal) have shown antidepressant potential. Intranasal ketamine is particularly appealing due to its high CNS penetrance and relative ease of administration.

In major depressive disorder, ketamine has a moderate-to-large antidepressant effect size, with response rates of approximately 70% and remission rates of approximately 30% at 24-hours postinfusion. In the aforementioned randomized controlled trials, ketamine's antidepressant efficacy lasted, on average, 1 week. In the only randomized placebo-controlled extension trial after an open-label single infusion, approximately 27% of ketamine responders maintained response during the 28-day follow-up period (mean time to relapse=13.2 days [SEM=2.2]) (8). Given the growing evidence for ketamine's potent antidepressant efficacy, interest in sustaining this response has naturally increased. Several alternative strategies have been attempted to augment and/or extend ketamine's efficacy but with only modest results (e.g., augmentation with the glutamatergic modulator riluzole and ECT). Future studies should explore alternative relapse prevention strategies, including traditional antidepressants, mood stabilizers (both ketamine and lithium are glycogen synthase kinase-3 inhibitors), antipsychotics (due to their efficacy as monotherapy in bipolar depression and as augmenting agents in major depressive disorder), and evidence-based psychotherapies. Yet the most obvious method of maintaining response is repeated administration, which is efficacious in some patients. This has led some experts to propose ketamine maintenance therapy and/or "boosters" upon early detection of clinical deterioration, similar to maintenance therapy in ECT.

Family History of an Alcohol Use Disorder as Moderator of Ketamine's Antidepressant Efficacy

Although many depressed patients have a robust antidepressant response to ketamine, not all patients have a positive experience. As shown in a recent trial for treatment-resistant obsessive-compulsive disorder, two out of 10 patients with a comorbid lifetime history of major depressive disorder experienced worsening anxiety, depression, and suicidal ideation postketamine infusion (9). As a result, the identification of baseline predictor and treatment response biomarkers has been a major focus of our research group, as well as others (for a review of ketamine-related biomarkers in mood disorders, see reference 10). Several studies have shown that positive family history of an alcohol use disorder is a key predictor of ketamine's antidepressant efficacy. Indeed, in both major depressive disorder (11) and bipolar depression (12, 13), subjects with a family history of an alcohol use disorder had a more robust and sustained antidepressant response to ketamine. This finding is also consistent with the differential effects of ketamine in persons with alcohol dependence who are recently detoxified (14) and in otherwise healthy volunteers with a positive family history (15). We recently pooled subject-level data from our completed ketamine depression studies of both unipolar and bipolar depression (total patients, N=108) and correlated ketamine's hyperacute (230 minutes postinfusion), acute (1 day postinfusion), and sustained (7 days postinfusion) antidepressant response with numerous clinical and demographic characteristics (16). In this combined sample, positive family history was again highly correlated with ketamine's acute and sustained antidepressant efficacy; at day 7 postinfusion, positive family history was the strongest identified predictor variable and alone accounted for 22% of the antidepressant improvement.

Although it is presently unknown why a positive family history of an alcohol use disorder improves ketamine's antidepressant response, we hypothesize that genetic polymorphisms and/or epigenetic modifications in glutamatergic and related neuroplasticity genes contribute to this effect. Several glutamatergic genes have been associated with depression and alcohol use disorders in a genome-wide association study from a comorbid sample (17). Due to our small sample to date, we currently lack sufficient power

to reliably assess the role of glutamatergic genes on ketamine's antidepressant response. Additionally, many of the polymorphisms identified in previous studies are not functional.

Positive family history of alcoholism is also associated with several alcohol-related phenotypes and neuroimaging parameters that overlap with major mood disorders. Schuckit et al. (18) were the first to demonstrate that subjects with an alcohol-dependent father had lower levels of response to alcohol than those without a father with an alcohol use disorder, and this low level of response correlated with the subsequent development of alcohol dependence in the index subject. Several genetic loci have been associated with a low level of response to alcohol. Although not all studies have identified differential effects of alcohol based on familial risk, a positive family history of an alcohol use disorder correlates with decreased body sway, decreased plasma prolactin and cortisol, and altered saccadic latencies on alcohol challenge. Moreover, during an intravenous alcohol challenge, subjects with a positive family history display acute tolerance (improved performance), while subjects without a family history of an alcohol use disorder display acute sensitization (impaired performance). Positive family history has also been studied in the context of neuroimaging parameters. Males with a positive family history increase functional MRI (fMRI) blood-oxygen-level-dependent signal in the left anterior insula and left frontal gyrus during successful inhibition on the go/no-go task, indicative of increased impulsivity. fMRI studies have revealed impaired nucleus accumbens activation during reward processing tasks and at rest. Adolescents with a positive family history also display altered resting state functional connectivity with the nucleus accumbens and amygdala. Both impulsivity and aberrant reward processing are present in bipolar disorder and major depressive disorder, respectively, indicating further neurobiological overlay between major mood disorders and familial risk for an alcohol use disorder.

In addition to fMRI, amino acid neurotransmitter levels have been examined through proton MR spectroscopy in response to ketamine in both major depressive disorder patients and healthy volunteers. Changes in glutamate, its precursor glutamine, and GABA did not correlate with ketamine's antidepressant efficacy in the occipital cortex in a goup of patients with major depressive disorder (5). In the prefrontal cortex of major depressive disorder patients, pretreatment glutamate and GABA levels also did not correlate with ketamine's antidepressant response (19). However, the baseline detectable glutamine + glutamate/glutamate ratio was lower in depressed patients with greater antidepressant response, and pretreatment glutamate levels were increased in depressed patients whose anxiety symptoms improved with ketamine (19). In a group of healthy volunteers, subanesthetic-dose ketamine caused no acute changes in glutamine + glutamate or glutamate. Acute changes in glutamate (i.e., the hypothesized "glutamate surge") have been postulated to underlie ketamine's antidepressant response, and it is presently unknown whether a family history of an alcohol use disorder correlates (either at baseline or with alcohol and/or ketamine challenge) with differential levels of cortical amino acid neurotransmitters, since none of the aforementioned MR spectroscopy studies stratified by familial risk. Interestingly, acute alcohol administration decreased both GABA and glutamate levels in 11 healthy social drinkers (20), but it remains to be seen how alcohol and/or ketamine affects glutamate and other amino acid neurotransmitter levels in depressed patients. Our research group is currently investigating the neurobiological mechanisms responsible for the enhanced antidepressant efficacy of a positive family history of an alcohol use disorder in major depressive disorder: ClinicalTrials.gov identifier: NCT02122562.

Conclusions

In summary, subanesthetic dose ketamine has rapid and sustained antidepressant effects in major depressive disorder and bipolar depression, and a positive family history of an alcohol use disorder is one of the most robust identified predictors of ketamine's antidepressant response. Because of the overlapping glutamatergic effects of both ketamine and alcohol, a neurobiological understanding of a positive family history may improve the existing descriptive and heterogeneous nosology of major depression in the latest iterations of DSM-5 and ICD-10. The identification of familial risk may also provide quantifiable means of assessing the probability of ketamine's antidepressant efficacy. Nevertheless, this promising predictor of treatment response requires replication/validation by other groups and in larger prospective clinical trials.

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For one of the first ranomized controlled trials studying ketamine for treatment-resistent depression, see the article by Murrough et al. in the October 2013 issue of the American Journal of Psychiatry, *but also see cautionary guidance provided by both A. John Rush, M.D., and Alan F. Schatzberg, M.D.*

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Case Report

A Unique Differential Diagnosis in a Case of Abnormal Motor Movements

Adriana de Julio, M.D., M.S.P.H.
Sarah Hutton, M.D.

Psychogenic nonepileptic seizure is now the academically accepted term to describe a pseudoseizure, which is a subtype of conversion disorder (1). Physicians have documented cases of conversion disorder since the time of Charcot (1825–1893). Explanations for the neurologic phenomenon have ranged from theories on dissociation to repressed memory. It is now known that up to 16% of patients with psychogenic nonepileptic seizure have documented epileptic seizures (2). We report the case of a woman with a medically complicated history who began having abnormal motor movements while undergoing alcohol withdrawal.

Case

"Mrs. B" is a 48-year-old married, Caucasian female retired social worker. She presented to the emergency department accompanied by her mother for increasing depression, nausea, vomiting, and heavy alcohol use for 2 weeks. She had a history of major depressive disorder and anxiety and had previously been treated with mirtazapine, escitalopram, and aripiprazole. She was currently being treated by a psychiatrist with trazodone, which she took nightly. Additionally, she had a history of alcohol use but no history of alcohol withdrawal seizures, encephalitis, meningitis, or tremors. Emergency department evaluation revealed that the patient had tachycardia, was tremulous, and had a blood alcohol level of 286 mg/dL. She was administered lorazepam, intravenous fluids with thiamine and dextrose and admitted to the general medical floor for alcohol detoxification using the Clinical Institute Withdrawal Assessment.

Psychiatry was consulted on hospital day 3, and a full psychiatric examination was completed. The patient was generally anxious, tearful, and disheveled but maintained good eye contact and an open rapport. She explicitly stated that she had been secretively drinking heavily, since her husband was a recovering alcoholic, for the past 9 years. On average, the patient drank two to five bottles of wine daily. Alcohol had been an issue at age 20, but at the urging of her husband, she discontinued drinking and remained abstinent until 9 years prior when her eldest son was diagnosed with a brain tumor causing seizures that had caused her to become increasingly depressed and anxious and led to her alcohol relapse. The patient reported that for 1 month before coming to the hospital, she had been severely depressed, had engaged in self-harm by cutting, and had plans to commit suicide by cutting her wrists. During the initial evaluation, she described visual hallucinations of shadows of people next to her and auditory hallucinations of her children's voices. By hospital day 4, she had complaints of "whole body shakes" and tremulousness.

On hospital day 5, she awoke with presyncopal complaints. A sitter was present and described how the patient laid down on her bed and started shaking gradually from head to toe. The shaking became more intense and then gradually tapered off after 5 minutes. During this episode of shaking, the patient remained awake and conscious of her surroundings and received diazepam. She had no amnesia prior to or during the event, and she reported that the diazepam had calmed her. There was no postseizure confusion or clouding of her sensorium. An EEG was ordered, but she refused the examination. Later that same day, she was transferred to the inpatient psychiatric unit where she experienced two more episodes, which were witnessed by the psychiatry team. These episodes of body shaking were stereotypic, beginning with a trembling chin and chattering teeth that gradually progressed to tremulous hands, then arms, and then her whole body. The episodes lasted between 2 and 5 minutes. Psychiatry worked alongside neurology, addiction medicine, and internal medicine to establish an initial differential diagnosis of these episodes that included alcohol withdrawal seizure, malingering, factitious disorder, simple partial seizure, epilepsia partialis continua, and psychogenic nonepileptic seizure.

Discussion

The psychiatry team had a low suspicion for alcohol withdrawal seizure in this case because the patient's blood pressure, heart rate, and overall sensorium were stable. Additionally, there was a high level of suspicion that the patient's symptoms were psychogenic in nature due to the odd and stereotypic movements witnessed multiple times by the psychiatry team. The psychiatry team began to conduct research to obtain information about conversion disorder and its epidemiology (3), as well as treatment modalities. The diagnostic criterion for conversion disorder was reviewed using DSM-5 (4). A literature review revealed that treatment with cognitive-behavioral therapy (CBT) (5) and selective serotonin reuptake inhibitors (SSRIs) (6) had the most evidence-based support. Expert opinions gleaned from the literature review overwhelmingly suggested that to make a diagnosis of psychogenic nonepileptic seizure, an EEG, specifically a 24-hour video EEG, should be

FIGURE 1. Checklist for Physicians Giving Diagnosis of Psychogenic Nonepileptic Seizures

These are genuine symptoms.
Real attacks—can be frightening or disabling.

Label
- Give a name for the condition.
- Give alternative names the patient may hear.
- Reassure that this is a common and recognized condition.

Case and maintaining factors
- Not epilepsy.
- Predisposing factors—difficult to find out causes.
- Precipitating factors—can be related to stress/emotions.
- Perpetuating factors—vicious cycle—worry leads to stress leads to attacks.
- Provide a model for the attacks—e.g., the brain becomes overloaded and shuts down.

Treatment
- Antiepileptic drugs are not effective.
- Evidence that psychological treatment is effective.
- Talk to the patient about referral to a specialist.

Expectations
- Can resolve.
- Can expect improvement.

performed, but our patient refused this test. On the psychiatric unit, trazodone was continued, and treatment with escitalopram and gabapentin was initialized. The patient initially denied that these episodes were triggered by strong emotions, but as rapport improved, it became clear that specific topics, especially those regarding her son's brain tumor and her guilt over the effect of her alcohol use on her family, could trigger an episode. On hospital day 10, the psychiatry team communicated with the patient, using a 14-item checklist, that these episodes of body shaking were most likely nonepileptic in origin and part of a class of disorders called conversion disorders. The patient accepted this diagnosis and inquired about treatment and her prognosis, and CBT was initiated thereafter. By hospital day 14, she had an increased ability to halt these episodes at the first sign of trembling by using CBT techniques. On hospital day 16, she experienced two more psychogenic nonepileptic seizure episodes, but no further episodes were witnessed or reported thereafter, and she was discharged on hospital day 19 to a residential substance abuse facility. This case is unique in many ways. First, the presentation of a conversion disorder with psychogenic nonepileptic seizure while a patient is undergoing alcohol detoxification, to our knowledge, has never been reported in the literature. Second, the fact that the patient's child had seizures secondary to a brain tumor did suggest to the psychiatry team that the patient's seizures were most likely nonepileptic and stemming from a deeper psychopathology. Third, the patient's ready acceptance of the diagnosis of conversion disorder was unexpected, since oftentimes patients resist that their condition may be psychiatric.

Conclusions

In the 21st century, the term pseudoseizure is losing its pejorative hold, and psychogenic nonepileptic seizure is gaining recognition because it appears that psychogenic nonepileptic seizure belongs in the spectrum of disorders that bridges neurology and psychiatry (7). Although EEG and video EEG are recommended for diagnosis in psychogenic nonepileptic seizure, they have limitations, and it is necessary for psychiatrists and neurologists to identify signs and symptoms that differentiate psychogenic nonepileptic seizure from epileptic activity (7). Recent studies have shown that approximately 27% of patients with psychogenic nonepileptic seizures continue to have poor long-term outcomes with high rates of psychopathology and poor overall levels of functioning (8). A checklist (Figure 1) has been developed to help effectively give the diagnosis of psychogenic nonepileptic seizure, and prognosis may improve with effective communication and the implementation of CBT and SSRIs as part of the treatment plan (9).

At the time this article was accepted for publication, Drs. de Julio and Hutton were second-year residents in the Department of Psychiatry, Advocate Lutheran General Hospital, Park Ridge, Ill.

The authors thank Dr. Geoffrey Levin, Department of Psychiatry, and Suela Sulo, M.S., of the James R. & Helen D. Russell Institute for Research & Innovation, Advocate Lutheran General Hospital, Park Ridge, Ill., for their assistance with this article.

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Book Forum

How to Talk to Parents About Autism

by Roy Q. Sanders. New York, W.W. Norton & Co., 2008, 256 pp., $21.95.

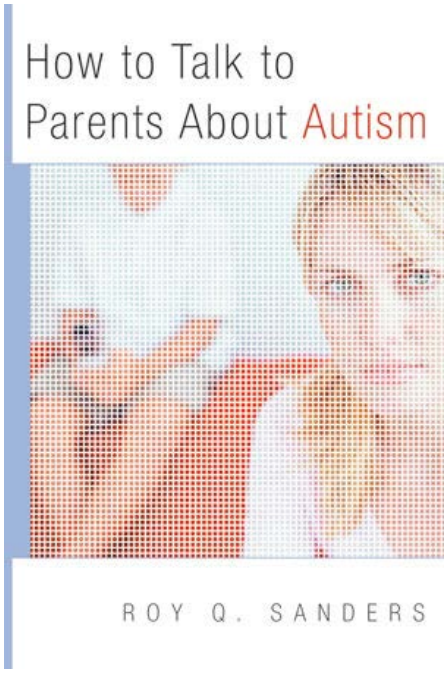
Panchajanya Paul, M.B.B.S., M.D.

During my training as a child and adolescent psychiatry fellow, I was frequently faced with or was asked about the myriad of practical challenges and concerns that families and caregivers of children of autism face daily. Fortunately, my program director referred me to this excellent book.

The author, Dr. Roy Q. Sanders, is both a clinician (with over 30 years of experience in treating children with autism) and also the parent of a child with autism. His book takes a fluid chronological approach to addressing issues as they often arise over time, including questions frequently asked at first diagnosis; how to navigate school systems; handling matters of puberty, relationships, romance, and sex; and planning for adulthood.

The book is easy to read for both professional and lay parents alike. Each chapter includes a question-and-answer narrative at the end, but the real gem of the book is how the author incorporates and shares his own family's experience with autism.

Dr. Sanders succinctly summarizes the available research on autism and uses his own experience to convey clear and concrete answers. He also deals candidly with often difficult and painful questions, such as addressing issues of intellectual disabilities. In this regard, Dr. Sanders even challenges practitioners to confront their own assumptions and prejudices relating to intellectual and cognitive function.



Perhaps most importantly of all, *How to Talk to Parents About Autism* emphasizes the need for practitioners to be supportive and encouraging to families of children with autism. Dr. Sanders admonishes health care professionals to help parents recognize their child's potential to grow and to learn and to experience a full and meaningful life. He also calls upon us to help parents rejoice and celebrate the gifts their child brings to the world and how to help their child share those gifts with others.

I highly recommend this book, which delivers exactly what its title promises: practical guidance for talking to parents about autism. The only limitation is that the book was published in 2008, and the landscape surrounding autism has changed, including research and, perhaps most notably, changes in diagnostic criteria and nomenclature. The core value of the book, however, remains the same. *How to Talk to Parents About Autism* provides the reader—whether a clinician, parent, teacher, or anyone living or working with a child with autism—with meaningful and practical guidance to better understand and talk about autism.

At the time this article was accepted for publication, Dr. Paul was a fifth-year resident in the Department of Child and Adolescent Psychiatry, Emory University, Atlanta.

Letter to the Editor

Are We Hastily Embracing Biomarkers?

To the Editor: Dr. Tannu's article, "Biomarkers in Psychopharmacology: The Present and Future," which appeared in the July issue of the *Residents' Journal* (1), made an interesting and thought-provoking read. Understandably, the unique nature of our specialty, which is based on subjective observations and patients' verbal reports, calls for a need for biomarkers. However, it is important to take a particular stand as to what purpose biomarkers must serve–diagnosis, assisting in diagnosis, confirmation, treatment, or prognosis. Moreover, at what stage can a biological measure be regarded as a full-fledged biomarker (2)?

The biggest danger with biomarkers is potential misdiagnosis of psychiatric diseases, as these illnesses depend substantially on environmental factors that contribute to disease manifestation (3). Psychiatric diseases are already stigmatized. Would it be fair to "label" a person with a mental disorder if he or she has a certain gene or neuroanatomic abnormality with no symptoms? Falsely diagnosing people with behavioral problems may expose them to discrimination and unnecessary stress, which could be an impetus for future stress-related illnesses.

As evident from a previous experience, biomarkers can be incorrectly correlated with a particular medical illness. For example, in the past, premature ventricular contractions were postulated as biomarkers for sudden cardiac death. This led to Food and Drug Administration-approved prescriptions aimed at suppressing premature ventricular contractions with an ultimate goal to curb sudden cardiac deaths. Unfortunately, this incorrect presumption cost the lives of several patients who were prescribed these drugs over a period of 10 years (4). Learning from past mistakes, we shouldn't rush into adopting biomarkers without empirical evidence. Hastily embracing biomarkers could land us in a similar situation.

Pravesh Sharma, M.D.

Dr. Sharma is a second-year resident in the Department of Psychiatry, Texas Tech University Health Sciences Center, Lubbock, Tex.

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Response to Sharma

To the Editor: I would like to thank my colleague, Dr. Sharma, for bringing up very important issues regarding my article on biomarkers. It is critical to have an objective guideline for biomarker use with respect to inclusion and/or exclusion criteria, as well as a clear idea of risks and benefits to the patient. If we take a step back, these guidelines should be mandatory for all diagnostic and therapeutic tools we have in modern medicine, probably not just biomarkers. Since biomarker discovery is in its infancy, it is natural to be apprehensive, not only about its promise but also about its safety in health care. Taking this into account, the Food and Drug Administration has initiated two quality-control measures: 1) the Biomarker Qualification Program, established to set a standard framework for scientific development and regulatory acceptance; and 2) the Critical Path Initiative, in order to assess safety, demonstrate medical utility, and promote eventual industrialization of such products (1, 2).

Another important issue raised by Dr. Sharma concerns biomarkers and accurate diagnosis. The DSM has come a long way since it was introduced in 1952. Each version has made our assessments not only better but also more objective and specific. The use of validated biomarkers, which have gone through the rigors of the above-mentioned testing, hopefully will add one more dimension to DSM. Hence biomarkers are purported to aid psychiatric diagnosis/treatment and provide psychiatrists with more objective tools to better care for their patients.

To quote the great Marcus Aurelius, "Life is neither good or evil, but only a place for good and evil."

Nilesh S. Tannu, M.D., M.S.

Dr. Tannu is a fourth-year Chief Resident in the Department of Psychiatry and Behavioral Sciences, University of Texas Medical School at Houston.

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Residents' Resources

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Ruth Fox Scholarship **Deadline: November 2014 (specific date TBA)**	American Society of Addiction Medicine	The Ruth Fox Memorial Endowment Fund will fund a limited number of scholarships for selected physicians-in-training to participate in the Society's annual conference.	All physicians-in-training	Kerin Miller kmiller@asam.org 301-656-3920	http://www.asam.org/membership/awards-program/ruth-fox-scholarship

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