Psychiatry, “Dangerousness” and the President

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It’s hard to keep up with the flurry of books, articles, op-eds and letters addressing the issue of President Trump’s mental state, many written by well-known psychiatrists 1–5. Reaction to these writings has sometimes been extreme, including reported “death threats” in response to one recent book about the President’s alleged “dangerousness.”

In an op-ed piece in the Boston Globe, the editor and one contributor to this book argued that they are not “diagnosing” Mr. Trump—which would violate psychiatry’s now famous “Goldwater Rule”—but rather, focusing “squarely on Trump’s dangerousness.” Furthermore, they argue that “dangerousness” can be “reliably established from public records” and “does not require a face-to-face interview.” They go on to depict their portrayal of Mr. Trump as nothing more than “expert commentary,” provided “… for the purpose of enhancing the public’s understanding, awareness, health and safety.”

We don’t doubt the good intentions of these colleagues, and we recognize that opinion among psychiatrists is often sharply divided, as regards the Goldwater Rule. Nevertheless, we find the above claims regarding “dangerousness” deeply problematic. When psychiatric terms of art are used capriciously to label a public figure, this stigmatizes not only the person labeled but also those with genuine mental illness. This impedes our ability to pro-

Allan Tasman, MD | Editor in Chief

I’d never heard of the Aokigahara Forest near the base of Mt. Fuji, and none of the psychiatrists I’d met on my professional visits to Japan had ever mentioned it to me. I’d guess most of you hadn’t heard of it either. Until maybe recently, that is.

This forest is the place made infamous in a YouTube video (now removed) by a fellow named Logan Paul, one of the most widely watched bloggers on YouTube. The video caused a major international outcry of condemnation, because the video contained a view of a person who committed suicide hanging from a tree in the Aokigahara Forest, which is known in Japan as a frequently used site for suicide. The frequency of suicides there is so high that authorities...
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provide vitally important psychiatric care and diminishes the credibility of our profession.

Accordingly, in this essay, we attempt to (1) update readers’ understanding of the “Goldwater Rule” in its latest incarnation; (2) describe what the term “dangerousness” ordinarily means in clinical psychiatry, and how it is properly ascertained; (3) examine the claim that a psychiatrist can determine a public figure’s “dangerousness” without having evaluated the person clinically; and (4) discuss the psychiatrist’s ethical responsibilities and legitimate options when he or she believes a public figure is “dangerous,” absent a clinical evaluation of that individual.

Goldwater redux
First, a quick update regarding two recent clarifications (or modifications) of the Goldwater Rule (GR) from the American Psychiatric Association. (One of us (RP) discussed and critiqued earlier formulations of the GR in a piece published on this website in October, 2016).6

In March, 2017, then APA President Maria Oquendo MD, PhD, issued this statement:

…APA’s Ethics Committee asserts that while it is perfectly fine for a psychiatrist to share their expertise about psychiatric issues in general, it is unethical to offer a professional opinion about an individual without conducting an examination. The committee clarified that the rule applies to all professional opinions offered by psychiatrists, not just diagnostic.

For example, saying an individual does not have a mental disorder would also constitute a professional opinion.7

The Ethics Committee defined the term “professional opinion” as follows: “…when a psychiatrist renders an opinion about the affect, behavior, speech, or other presentation of an individual that draws on the skills, training, expertise, and/or knowledge inherent in the practice of psychiatry, the opinion is a professional one.”8

These clarifications (or modifications) of the GR stimulated a robust “pro and con” exchange in Psychiatric Times between psychiatrists Leonard Glass, MD, and Rebecca Brendel, MD, JD.9,10

Then, in a statement released Jan. 9, 2018, the APA vigorously upheld the principles articulated in the Goldwater Rule, writing:

We at the APA call for an end to psychiatrists providing professional opinions in the media about public figures whom they have not examined, whether it be on cable news appearances, books, or in social media. Armchair psychiatry or the use of psychiatry as a political tool is the misuse of psychiatry and is unacceptable and unethical. A proper psychiatric evaluation requires more than a review of television appearances, tweets, and public comments. Psychiatrists are medical doctors; evaluating mental illness is no less thorough than diagnosing diabetes or heart disease. The standards in our profession require review of medical and psychiatric history and records and a complete examination of mental status.11

Three features are worth noting in the latest iterations of the GR:

1. The word “opinion” is always preceded by the term “professional” or “professional medical”

2. The rule is always applied to public statements about a particular individual, not about policies.

3. The rule says nothing-and evidently does not apply-to private communications between a psychiatrist and an appropriate official.

Thus, a psychiatrist who writes or states publicly, “In my opinion, the President doesn’t understand climate change” is not expressing a professional opinion as defined by the rule (because the opinion does not draw on the skills, training, expertise, etc., inherent in the practice of psychiatry). By the same token, a psychiatrist who writes or states publicly, “I believe the proposal to end the Affordable Care Act is ill-advised” is not voicing the kind of professional opinion covered by the Goldwater Rule (which applies to public statements dealing with “the affect, behavior, speech, or other presentation of an individual.”). Finally, a private or confidential letter from a psychiatrist to, say, an appropriate member of the US Congress, expressing concerns about a public official’s behavior, does not appear to be prohibited by the Goldwater Rule. We stress these fine points because they directly affect the ways in which psychiatrists can enhance the public’s “health and safety” without violating professional ethics.

“Dangerousness” in clinical and forensic psychiatry
In general, the term “dangerousness” usually refers to legal, rather than psychiatric criteria.12 Most psychiatrists will be familiar with the term in the context of civil commitment, when “dangerousness” is defined by state statute. This usually involves behavioral evidence that an individual has already engaged in a behavior that presents a danger to others—either by threats or acts.13

In forensic psychiatry, the terms “violence risk” or “risk of violence” are preferable to “dangerousness.” Determining violence risk entails a clinical or forensic assessment of an individual at a particular time. In addition to assessing violence risk, the goal is to discover any psychosocial or modifiable risk factors that may targeted in a risk reduction plan. The key point is that mental health professionals cannot successfully “predict” low base rate human behaviors (such as violence) with long-term accuracy. Furthermore, because violence risk (like suicide risk) is dynamic and influenced by many variables, periodic assessments over time are required.

Misuse of psychiatric terminology
Unfortunately, in the popular media, psychiatric terms of art are often used in a casual or reckless manner. Take, for example, the forensic-legal phrase “duty to protect,” stamped into the annals of psychiatric history by the 1976 Tarasoff ruling. This ruling created a legal “duty to protect” which overrode the confidentiality of the therapist-patient relationship in California. The final ruling in Tarasoff emphasized that therapists have a duty to protect individuals who are being threatened with bodily harm by their patient.14 Thus, the forensic/legal phrase “duty to protect” applies only to therapists who have a therapist-patient relationship in the eyes of the law—not broadly to psychiatrists and the public at large.

Consequently, there is no legal sense in which psychiatrists are called upon to assess “dangerousness” in relation to large groups or communities—much less entire nations. Thus, those who use the terms “dangerousness” or “duty to warn” in a general, socio-political sense—as in, “Trump is a danger to the country and we have a duty to warn the public”—are misappropriating these terms from their legitimate medico-legal context.

Indeed, in clinical and forensic psychiatry, the putative “dangerousness” of a patient must be assessed according to well-established norms and procedures, including but not limited to a thorough, face-to-face, clinical evaluation. Elements of the evaluation include such factors as the specific person threatened; presence of a specific plan; past history of vio-
perience of command hallucinations; history of impulsivity; alcohol and other substance misuse, and many other factors that cannot be assessed without an in-person evaluation.12

Dangerousness without a clinical evaluation?

So: can dangerousness be “established from public records” alone? In general, the answer is no. In rare cases, the “public record” may reflect documented instances of assault, domestic abuse, arrests for disorderly conduct, etc., such that one might reasonably consider the individual at “high risk” for future violence. But in general, publicly attributing “dangerousness” to an individual one has not assessed clinically is inconsistent with both good medical practice and the intent of the Goldwater Rule. Reliable risk assessment cannot be based solely on a public figure’s television appearances, tweets, and public comments. Psychiatrists must assess clinical risk as thoroughly as other physicians assess risk in heart disease or cancer.

One important, albeit implicit, goal of the Goldwater Rule is to prevent the arbitrary and capricious stigmatization of public figures. As Dr. Brendel notes, at the ethical core of the rule is “respect for persons”, and “…making professional judgments about an individual’s mental health is intrusive and can have negative consequences. We need not look further than Barry Goldwater to see that these conclusions, even when ‘non-clinical,’ can be harmful.”19

In our view, labeling a (clinically una)examined public figure as “dangerous” can do as much or more harm as promulgating a specific psychiatric diagnosis. Thus, the supposed distinction between “diagnosing” a public figure and labeling that individual as “dangerous” is spurious and misleading.

The psychiatrist’s ethical responsibilities to the public

Some psychiatrists have defended their public allegations of the President’s “dangerousness” on the basis of psychiatry’s professional duty to contribute “…to the improvement of the community and the betterment of public health.” Indeed, this duty is set forth in Section 7, subsections 1 and 2, of the APA Code of Ethics. “The psychiatrist is therein encouraged “…to serve society by advising and consulting with the executive, legislative, and judiciary branches of the government”, and to “…interpret and share with the public their expertise in the various psychosocial issues that may affect mental health and illness.” We strongly agree that these forms of public education and engagement are part of the psychiatrist’s ethical responsibilities. However, this should not serve as a rationalization for breaching the much more specific directives of Section 7, subsection 3 of the Code, which states, “…it is unethical for a psychiatrist to offer a professional opinion unless he or she has conducted an examination and has been granted proper authorization for such a statement.”14

A similar view is expressed in a report from the American Medical Association’s Council on Ethical and Judicial Affairs.13 In its recent Guidance for Ethical Physician Conduct in the Media,10 the AMA notes that “the media industry can have interests and goals that are out of step with physicians’ ethical obligations to patients, the public and the medical profession,” and that “Disseminating a message that is inaccurate, questionable, or false may be perceived as authoritative when it comes from a physician in a position of public trust.”

When unreliable or biased medical information is given to the media cloaked in the mantle of medical authority, this tends to undermine public trust in the medical profession. Accordingly, the AMA policy states that physicians should “refrain from making clinical diagnoses about individuals (eg, public officials, celebrities, persons in the news) they have not had the opportunity to personally examine.”11

The legitimate purview of psychiatry

Psychiatrists are trained primarily to diagnose and treat mental illness according to specific professional standards and methods—not to offer opinions on what constitutes good or bad leadership. Neither are psychiatrists specifically trained to define “fitness” or “unfitness” to hold public office. With respect to the presidency, “fitness” is to be determined through the political process outlined (albeit sketchily) in the 25th amendment to the US Constitution. Of course, as psychiatrists Paul Summergrad and Stephen N. Xenakis point out, medical opinion regarding a president’s physical and mental health may be one component of this determination.15

Unsubstantiated, “armchair” opinions from psychiatrists often appear foolish with the passage of time. Consider the Goldwater Fact Magazineiasco, and one psychiatrist’s

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carefully considered opinion, pre-
served for the ages: "[Goldwater] is a mass-murderer at heart and . . . a dangerous lunatic. Any psychiatrist who does not agree with the above is himself psychologically unfit to be a psychiatrist."

Indeed, the crux of the problem is the misapplication of psychiatric labels in order to vent displeasure at a public figure. This not only trivializes genuine mental illness, it also per- petuates the stigma long associated with mental illness and confuses the general public as to what serious mental illness is and is not.

Ethically responsible input from psychiatrists

To be clear: psychiatrists do not relinquish their first amendment right to freedom of speech by virtue of being psychiatrists. There are several ways psychiatrists can voice concerns about the mental state and fitness of a public official who has not been clinically evaluated.

First, they can express concerns or misgivings as private citizens, so as to avoid “…cloaking their public statements with the authority of the profession.”7 (APA Code, Sec. 7.1). The Goldwater Rule does not “gag” psychiatrists; rather, it defines the conditions under which psychia- trists may render a professional opinion.

Psychiatrists may criticize specific policies of public officials or their administrations. Indeed, the APA itself has criticized a number of poli- cies or legislative initiatives associated with the Trump administration, such as repeal of the individual man- datory component of the Affordable Care Act.18

Psychiatrists can publish educational al pieces and participate in media interviews concerning, for example, the differential diagnosis of impulsivity, cognitive impairment, aberrant behavior, etc.

In rare instances in which a public figure is deemed an immediate or se- rious threat to the public safety, psychia- trists can communicate their concerns privately to appropriate civil authorities, congressional representa- tives, or magistrates.

Despite the rancor and confusion surrounding the Goldwater Rule’s in- tegrity, perhaps the silver lining is this: the debate has opened a broader discussion about the importance of recognizing genuine psychiatric illness and the requirements of psychiatric ethics.

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have placed signage geared to direct those considering suicide to seek help. And, the number of park employees at the site have been increased.

My thinking about the Aokigahara Forest and of Japan’s concerns about the effect of social contagion on suicide barely preceded the release of the CDC annual report on suicide rates and trends. Unfortunately, unlike YouTube videos which can be removed, this report and the reality that underlies it are here to stay.

The American Foundation for Suicide Prevention (AFSP) has analyzed and summarized some of the key findings in the CDC report and posted them on its website. Nearly 50,000 people commit suicide in the US every year. In spite of improved understanding of risk factors and increased vigilance for suicide risk among mental health and other medical practitioners and families of those at risk, the suicide rate in the US continues to go up.

Because of the lag in data collection and analysis, the CDC report covers data with a year’s delay. What is seen, though, is that between 2006 and 2016 the overall US suicide rate went up from 10.97 to 13.26 per 100,000 people. Those numbers may seem small, but they reflect an increase of just over 20% in these 10 years. And, the trend lines don’t look much different going back to 2000.

For reasons that are unclear, the suicide rates are much higher among white people, although the rates in those identifying as Native American are close. White males accounted for 70% of suicides in 2016. And, although there has been a small uptick in rates in the last few years for African American or Asian heritage people, those rates are much lower than for white and Native Americans. The AFSP notes that data for those of Hispanic origin are not reported in the same way, since individuals in any of the other groups may be Hispanic.

Another interesting demographic finding is that the states that have the lowest population densities have the 10 highest per capita suicide rates. These states are, in order of suicide rates (highest to lowest): Wyoming, Alaska, Montana, New Mexico, Utah, Idaho, South Dakota, Oklahoma, Colorado, and Arkansas.

The CDC also, as you know, segregates rate data by age groups. The recent data show that the highest suicide rates are in those aged 45 years and older, clustering around 15 per 100,000. And, the rate of increases in the suicide rate in the 45 to 54 and 65 to 74 age brackets are especially steep. The most concerning finding to many, however, is the fact that the suicide rate for those 15 to 24 is now 12.5, the highest since 1995.

In 1970, the suicide rate for 15- to 24-year-olds was 8.8 per 100,000, which rose to 13.8 by 1994. This phenomenon led then US Surgeon General David Satcher, a pediatrician, to declare that adolescent suicide prevention was his top public health priority. While it is unclear whether federal attention was the major factor, the suicide rate in this age group fell to 9.9 by 2002. This nearly 33% drop was by far the greatest change of any age group, although other age groups also dropped by small or moderate amounts. It is, thus, distressing to see that since 2002, not only have suicide rates increased for nearly every age group aged younger than 75 years, but that the adolescent suicide rate is nearly back to a 5-decade high.

There is a cause for concern that suicide rates may actually be higher than those reported by the CDC. In a recent article, Olfson and colleagues' evaluated attempted suicide in the US. They used data from national epidemiologic surveys from 2004-2005 and 2012-2013. Data analysis from nearly 70,000 people between the 2 survey years showed a 24% increase in suicide attempts in less than 10 years. But, the authors warned that data collection in their study sample was subject to inaccuracy to some degree. One source of inaccuracy was the fact that a prior suicide attempt reflects high risk for completed suicide and those who had succeeded, and who may have had prior attempts, could not be included in the surveys. Thus, many attempts were likely missed.

In fact, the AFSP analysis asserts that while the data suggest a nearly 12-fold greater number of attempts—nearly 500,000 a year—the actual number may well be twice as high because of the wide range of reporting inaccuracies. Of course, this means there is a likelihood that the actual suicide rates may be higher than those reported by the CDC as well.

The demographic analyses by Olfson and colleagues found that those groups with particularly increased prevalence of suicide attempts included young adults and those with only a high school education. In addition, those with a variety of psychiatric illnesses had an increased risk, including anxiety and depressive disorders, and those with borderline, antisocial, and schizotypal personality disorders. Not surprisingly, the risk of a suicide attempt was highest in those with a previous attempt.

One bit of good news also came from their data, which was that while the majority of those in the survey data who attempted suicide had a diagnosis of borderline personality, this group also had a significant decrease in risk of suicide attempts over the study period. The authors hypothesized that this important and welcomed decrease might be due to greater access to treatments such as dialectical behavior therapy (DBT). DBT and other parallel approaches were developed specifically to reduce the impulsivity experienced by patients with borderline personality.

Unfortunately, there’s a great deal of bad news in the recent CDC report, and in analyses by other authors, of both suicide attempts and completed suicides. I didn’t see much very surprising in the findings of factors endowing high risk for suicide attempts or completed suicides. And, I do think that the actual rates for both are underestimated. As just one example, the number of not completely unintentional drug overdoses... (CONTINUED ON PAGE 6)
Cultural Perspectives on Migration and Psychopathology

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One of the most powerful social phenomena during the past 2 or 3 decades is, without a doubt, the continuous and seemingly unstoppable growth of the migration of millions of people across practically all countries, regions, and continents of the world. It is said that nowadays about 5% of the world population is made up of migrants. While technological advances provide the physical machinery pushing globalization ahead, migration represents the heterogeneous human component of this process—the dramatic mix of realities, decisions, collisional emotions, expectations, and uncertainties that constitute life on our planet. And as with any other human enterprise, cultural assumptions are challenged and different behaviors and potential psychopathologies emerge among all involved—migrants and hosts.

There are innumerable lines of reflection about this topic. The following paragraphs attempt to outline 10 of them—examining points of conflict or contention, special aspects of the migratory process, mental health and eventual public policy issues—from a cultural psychiatry perspective. The conceptual connections may appear obvious at times, complex or distant at others. They are, in any event, invitations to think further and explore ways to, at least, be capable of asking better questions.

1. The acculturation process, that is, the immigrant’s level of acceptability and adaptability of the host society’s habits and traditions is an important phase of the migratory experience. Social scientists speak of a fluid, mild or moderate, delayed or rejected acculturative phase—each one with potential implications of emotional stability or conflict. Occasionally, a “new” culture or sub-culture evolves out of this process. The strength of the cultural legacy brought in by the immigrant and his or her family is a key factor that results in flexibility or rigidity, adaptation or alienation. Obviously, the host society’s receptivity and attitudes toward the new neighbors are another decisive factor with outcomes of integration or rejection and their respective behavioral or clinical expressions. Acculturative Stress or Acculturation Problem are diagnostic labels included in the current versions of DSM and ICD.

2. There are also internal and external migrations. The former occurs within contiguous geographic zones, usually in the same country. Internal migrants move from untenable to more tolerable situations, generally out of fear or in an almost desperate attempt to prevent worse developments. Typical examples are seen in countries that became true civil war scenarios (ie, guerrilla groups fighting government forces in countries like Colombia, Peru, Congo, or Cameroon). External migrations take place when international borders are crossed. Again, the psychological contexts of these 2 types of migrations are different—the sense of loss, fracture, disruption, frustration, impotence, rage, or hopelessness may reach deeper or broader levels, carrying a variety of psychopathological consequences.

3. The acculturative process, that is, the immigrant’s level of acceptability and adaptability of the host society’s habits and traditions is an important phase of the migratory experience. Social scientists speak of a fluid, mild or moderate, delayed or rejected acculturative phase—each one with potential implications of emotional stability or conflict. Occasionally, a “new” culture or sub-culture evolves out of this process. The strength of the cultural legacy brought in by the immigrant and his or her family is a key factor that results in flexibility or rigidity, adaptation or alienation. Obviously, the host society’s receptivity and attitudes toward the new neighbors are another decisive factor with outcomes of integration or rejection and their respective behavioral or clinical expressions. Acculturative Stress or Acculturation Problem are diagnostic labels included in the current versions of DSM and ICD.

4. Diagnostic issues have strong cultural implications when applied to migrant populations. A crucial topic is the validity of diagnostic criteria used in the host country vis-a-vis the clinical pictures displayed by the newly arrived. Specific behavioral, symptomatic, or syndromic presentations (ie, cultural syndromes or cultural concepts of distress, according to DSM-5) must be carefully explored in order for clinicians not to fall into stereotyping or plainly stigmatizing labeling. The use of instruments aimed at an objective estimation of cultural identity, help-seeking patterns, and explanatory models are mandatory for an objective clinical evaluation.

5. What is the most frequent psychopathology observed among migrants? It may not be PTSD, although it is indisputably the most dramatic and pervasive one. Depressive and anxiety-related disorders, a good number of them in the context of “situational” or “adjustment” conditions, abound. The causative pathogenic chain is understandable: feelings of loss, grief, panic, hopelessness, and helplessness sometimes prevail against culturally based protective factors. A growing prevalence of substance use disorders, particularly in younger members of migrant groups (some of them creating “gangs” that convey, in many cases, a self-protective as well as an aggressive message), is also a result of the loss of individual and family control, a search for unknown “solutions,” or desperate evasive modes. Physical concomitants, frequently seen in these fragile human groups, make the situation worse. A
careful, comprehensive health assessment is vital.

6 Clinical epidemiology studies may show some intriguing results. One of them from the 1970s was the then-called “Hispanic Paradox,” the finding of a lower prevalence of mental disorders among Latino immigrants compared with similar groups of American-born individuals. This went clearly against the usual assumption, documented by earliest studies among Scandinavian immigrants. The initial explanation was a self-selective process: immigrants-to-be, those capable of deciding to leave the country of origin, had to be the physically and psychologically strongest and healthiest to successfully face the new realities in the host country. An indirect confirmation of this was the finding of increased percentages of mental illness among second-generation immigrants’ children, at levels very similar to those of American-born individuals; this led the media to half-jokingly comment that “living in America makes people sick.” Nevertheless, the “paradox” was later questioned on the basis of different nativities and sociodemographic, family, and cultural characteristics that better explained epidemiological findings in Latino sub-populations.

7 Immigrant subgroups particularly vulnerable to the distressing adversities of the experience are those of special subpopulations such as children, adolescents, women, and the elderly. Victims of neglect or abuse (ie, the dramatic cases of violation of women users of “The Beast,” the train that transports migrants from Central America and Mexico to the US border), exploitative financial charges by “Coyotes” (individuals who take the immigrants through the border, only to abandon them after they reach an unknown or unfamiliar town in America), and the case of children who make the journey by themselves are situations of potentially profound emotional damage. This feature of the so-called “culture” of the migratory experience is not different in many refugee camps in the Middle East, Asia, and Eastern European countries.

8 The establishment of public health policies, with strong recognition of culturally based factors (risk promoting and protective) involved in the abusive situations described above, must be a matter of concern for national governments and international organizations, beyond emotional pronouncements of help or refusal. Even more, the translation of policies into definite actions (from helping or Assistance Homes to competent health and mental health personnel in hospital or community-based institutions) and preventive or early intervention measures, communication, and coordination with potential employers are indispensable steps. These arrangements require the will and determination of the highest governmental spheres of the countries involved.

9 The treatment of declared cases of psychopathology must be equally prompt and efficiently implemented. Community psychiatry care seems to be regaining an advantageous position in terms of policy-driven material and human resources. From the clinico-cultural perspective, close emergency medical and psychiatric consultations; availability of essential medications; and individual, family, or group psychotherapies are also critically important. Interpreters, culturally trained psychotherapists, and the use of specific cultural psychotherapies (if available) must be substantial components of a comprehensive management process.

10 In addition, most if not all of the requirements listed above necessitate the harmonious development of medical and psychiatric education norms at all levels. General and mental health professionals who work with migrant populations would benefit from education that includes topics such as globalization (and its primary components of global health and global mental health), cultural psychiatry (including diagnosis, treatment, and prevention), and the various links between migration and psychopathology. This type of training, in turn, would make possible the work of multidisciplinary teams that better understand the dynamics and expectations of migrants and help them to quickly integrate into the new environment. Preventive concepts could extend to the desirable possibilities of peaceful, mature, and durable political evolutions in the countries and regions where today’s waves of migrants come from.
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ELECTROCONVULSIVE THERAPY
ECT for Self-Injurious Behavior in Autism: A New Indication

Since its introduction 8 decades ago, ECT has been a standard treatment for severe affective and psychotic illness, as well as less common indications including catatonia, Parkinson disease, neuroleptic malignant syndrome, and status epilepticus. A new indication for ECT has emerged for intractable self-injurious behavior (SIB) among individuals with autism and intellectual disabilities.1 Response to ECT is typically swift and robust, with great benefit for patients and their families. SIB is defined as any act toward the self that causes physical injury, and has been recognized for decades to occur among individuals with autism spectrum disorders and intellectual disability. Estimates of SIB prevalence vary widely, with higher rates usually found among those with greater cognitive and communication impairments, and psychiatric and medical comorbidities. Common manifestations of SIB include head banging; hand-to-head, knee-to-head, and hand-to-body hitting; self-pinning; scratching and biting; and body slamming against hard surfaces. The frequency of SIB varies widely, from occasional, to daily, hourly, or even a per-minute basis. Frank bodily injury is common and behavioral improvement of a few minutes in a restricitve institutional setting.5

Jay is a 19-year-old man with autism and intellectual disability. He was initially hospitalized at age 12 after several years of severe head- and body-directed SIB with frequent facial bleeding, as well as aggressive behaviors toward his family, caregivers, and school staff. SIBs were so intense that a caregiver inadvertently broke Jay’s arm while attempting to block hand-to-head striking. Jay also displayed mood instability with sobbing and wild laughter, irritability, physical hyperactivity, agitation, and insomnia. He had exhausted multiple drug trials from all US-available psychotropic classes, as well as years of intensive behavioral services. He demonstrated adequate and behavioral improvement of a few months’ duration on lithium carbonate, aripiprazole, and riluzole, but deteriorated again with such behavioral dyscontrol that his parents feared their only option to maintain his safety was placement in a locked residential facility.6

Standard treatments
Standard treatments of SIB in autism and intellectual disability are behavioral and psychopharmacological interventions. While many individuals respond to these modalities, there has remained a subgroup of treatment-refractory patients who have exhausted dozens of psychotropic medications, years of intensive behavioral therapy, and multiple out-of-home placements and hospitalizations. Such patients may be padded and immobilized head-to-toe in protective equipment, and frequently receive psychotropic medications as chemical restraint—often with limited benefit and high risk of adverse effects. Despite such efforts, these patients often sustain permanent injuries including blindness from retinal detachment and cerebral hemorrhage.7 Recent suggestions that SIB might be one of many symptoms of catatonia in autism spectrum disorders—including mutism, negativism, posturing, rigidity, and repetitive speech and acts—led to treatment trials with ECT, the long-recognized effective treatment for catatonia, with remarkable behavioral improvement and relief.7

CASE VIGNETTE
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A trial of 3 times per week bilateral ECT was initiated. Jay demonstrated dramatic reduction in SIBs after just a few treatments, as well as improved sleep and more stable mood. ECT frequency was gradually reduced while monitoring for recurrence of SIB or mood deterioration; ultimately, a maintenance ECT regimen of every 9 or 10 days was established.

Jay was able to remain at home, where he participated fully and safely in family life with his parents and 2 siblings. He continued in a program emphasizing community-based living and functioning activities, and was able to master skills never thought possible for him before, such as independent grocery shopping from a list, followed by check-out with a debit card and meal preparation.

He tolerates ECT well and is able to go to school without incident after early-morning treatment. He remains on a concomitant regimen of lithium and aripiprazole; SIB is limited to rare incidents of hand-biting with no resultant injuries. Jay has received an estimated 290 sessions of ECT over the past 8 years.

Evidence for ECT
The resolution of SIB with ECT was first documented in a 1982 report of a 25-year-old man with severe mental retardation and head-directed SIB of several years’ duration.8 In 1999, a report presented a 14-year-old boy with mental retardation whose persistent head-banging and self-scratching were alleviated by ECT, which allowed discharge after many months in a restrictive institutional setting.

The relationship between self-injury, catatonia, and ECT was first questioned in the 2008 case report of an autistic girl with posturing, rigidity, mutism, stupor, grimacing, food refusal, and global skill regression, alternating with bursts of extreme self-injury that led to bilateral traumatic cataract formation and retinal detachment.8 ECT resulted in resolution of catatonic symptoms, as well as nearly complete eradication of SIB, which allowed for vision preservation in one eye.

The following year, the case of an 8-year-old autistic boy with ongoing agitation and stereotyped movements (in the form of nearly 110 SIBs per hour) was reported.9 After treatment

References
3. Dr. Wachtel is Associate Professor of Psychiatry, Kennedy-Krieger Institute, Johns Hopkins School of Medicine, Baltimore, MD; Dr. Kellner is Chief of Electroconvulsive Therapy, New York Community Hospital, Brooklyn, NY; Dr. Fink is Professor Emeritus, Departments of Psychiatry and Neurology, Stony Brook University, Stony Brook, NY.
4. Since its introduction 8 decades ago, ECT has been a standard treatment for severe affective and psychotic illness, as well as less common indications including catatonia, Parkinson disease, neuroleptic malignant syndrome, and status epilepticus. A new indication for ECT has emerged for intractable self-injurious behavior (SIB) among individuals with autism and intellectual disabilities.1 Response to ECT is typically swift and robust, with great benefit for patients and their families. SIB is defined as any act toward the self that causes physical injury, and has been recognized for decades to occur among individuals with autism spectrum disorders and intellectual disability. Estimates of SIB prevalence vary widely, with higher rates usually found among those with greater cognitive and communication impairments, and psychiatric and medical comorbidities. Common manifestations of SIB include head banging; hand-to-head, knee-to-head, and hand-to-body hitting; self-pinning; scratching and biting; and body slamming against hard surfaces. The frequency of SIB varies widely, from occasional, to daily, hourly, or even a per-minute basis. Frank bodily injury is common and includes bleeding, bruising, swelling, fracture, infection, disfigurement, traumatic ossification, cataract development, retinal detachment with associated loss of vision, and concussion or intracranial damage. Other deleterious consequences of SIB include reduced social, educational, family, and occupational functioning, with higher risk of placement in more restrictive settings and limited life opportunities.
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6. Dr. Wachtel is Associate Professor of Psychiatry, Kennedy-Krieger Institute, Johns Hopkins School of Medicine, Baltimore, MD; Dr. Kellner is Chief of Electroconvulsive Therapy, New York Community Hospital, Brooklyn, NY; Dr. Fink is Professor Emeritus, Departments of Psychiatry and Neurology, Stony Brook University, Stony Brook, NY.
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Deaths is impossible to estimate. We know, however, that failed attempts at kicking an addiction not uncommonly lead to feelings of hopeless-ness and helplessness—feeling states associated with suicidality. The good news is that decreases in suicide attempts and completed suicides correlate with both federal government actual initiatives (not just making a declaration of a public health emergency), and the avail-ability of treatments for predispos-ing illnesses and traits such as impulsivity. In addition, we should value the moderating influence of all the programs and organizations that are already at work on suicide prevention. I’m sure the suicide and suicide attempt rates would be higher without their hard work.

To me, however, the most impor-tant take-home message from the findings I’ve discussed here is the importance of maintaining the per-spective embodied in the biopsychoso-cial model as we try to unravel the complex etiology of suicide, which can lead to better prevention and treatment. Early work looking at ge-netic variants and suicidality have so far been disappointing. Moreover, the social determinants have been inadequately studied (is it simply random that the 10 states with the highest suicide rates are among the most sparsely populated?) and the current understanding of the psy-chological components of complet-ed suicide have not been helpful in reducing the suicide rates.

We have tremendous opportuni-ties to increase our knowledge and improve the success in preventing suicide. And, of course, making sui-cide prevention a real priority at all levels of government would, I be-lieve, make a significant positive difference. I’ve said a number of times in my column that our role as advocates to government and other decision makers is essential. In this case, lives really do depend on it.

with ECT, he averaged only 20 SIBs per hour. It was postulated that his SIB was a manifestation of catatonia.

In 2010, Wachtel and Dhossche proposed that self-injury in autism represented a variant of catatonia. They reviewed the history of these conditions, exploring self-injury as a stereotypy common to both catatonia and autism, and further connecting catatonia, autism, tic disorders, and the self-injury that occurs in the 3 conditions. Subsequent reports have continued to call for recognition of repetitive SIB as a sign of catatonia, with clear implications for the use of ECT in its treatment.

**ECT treatment protocols**
ECT for adolescents with autism spectrum disorder and SIB includes standard ECT protocols of general anesthesia with propofol or methohexitol, muscle relaxation with succinylcholine, and continuous oxygenation via face mask. Flumazenil, the benzodiazepine reversal agent, may be used at the time of treatment when patients have recently taken these medications. Electrode placement is typically bilateral, and stimulation is with modern brief-pulse devices.

Moderately suprathreshold stimulus dosing takes into account the lower seizure threshold of younger patients. Seizure adequacy is monitored by the duration and quality of the recorded EEG. The ECT course begins with seizures 3 times per week, until SIB frequency has been markedly reduced; ECT frequency is then gradually reduced over the en-
suing weeks, while monitoring for sustained SIB reduction. For these patients, ECT is a procedure analogous to renal dialysis, with frequency of treatment guided by objective symptom evaluation, and the need to optimize the quality of patient and family life.

**Conclusion**

While the mechanism of the relief of catatonia by ECT is unclear, the neuroendocrine studies of melancholia offer potential clues. Studies of cortisol and thyroid functions in these patients find chemical abnormalities that resolve with repeated ECT, suggesting that seizures normalize the hypothalamic-pituitary-adrenal axis and other endocrine functions. While we know little about the specific neuroendocrine disturbances in autism spectrum disorder or in catatonia, there are likely commonalities in the underlying physiology between these disorders and melancholia. At present, the best understanding of the mechanism of the beneficial effect of ECT in adolescents with SIB is the neuroendocrine explanation developed for melancholia. Additional mechanistic theories are enhanced neurogenesis, angiogenesis, and neuronal connectivity.

ECT came into widespread use at a time when psychodynamic thinking dominated child and adolescent psychiatry and the application of the...
somatic therapies was considered anathema. Since the 1990s, this attitude hurdle has been slowly overcome with the realization that some severe childhood and adolescent disorders—notably, melancholia, mania, and catatonia—are safely and effectively relieved by modern ECT procedures. Recognizing SIB in autism spectrum disorder as a form of catatonia has encouraged this application. Indeed, ECT has been documented as nothing short of lifesaving for many children and young adults with autism spectrum disorder who were previously wholly incapacitated by SIB.

References
Greenberg On The Arts | Blade Runner 2049: Tears in Rain

Over his long career, Ridley Scott has directed movies in many genres—historical and science-fiction pictures predominating. At times, he has adhered strictly to genre conventions. He’s also radically subverted them (eg, Thelma and Louise’s [1991] tragi-comic feminist critique of the macho buddy road movie).

Alien (1979) was Scott’s first genre-blender, melding tropes of horror and science-fiction. Audiences came in droves to see it—and to watch it again. The return phenomenon, so dear to Tinseltown’s machers and shakers, was caused...
by *Alien*’s formidable cinematic power and its gruesome chest burster sequence. Viewers eager for an even bloodier *Alien* sequel were disappointed and puzzled by Scott’s next film, *Blade Runner* (1982), based on Philip K. Dick’s short story, *Do Androids Dream of Electric Sheep?* Most critics were also displeased.

The original *Blade Runner* is set in the devastated, yet still functioning Los Angeles of 2018. Dank rain drizzles incessantly from a blemished sky; the opalescent air is breathable, but permanently polluted. The rusted-out mechanisms of this dominion of detritus are not replaced, but “retrofitted,” like the water lines snaking up building exteriors. The entire mise-en-scène has a distinctive “retro” look, conflated with Japanese and—curiously—vaguely Mayan architecture and interiors.

The wealthy live in totemic skyscrapers. Packed grotty streets are reminiscent of an older Tokyo or an Istanbul grand bazaar. Street-people hustle whatever trinkets or grub they can; speak an incomprehensible argot of Japanese, English, whatever. The swirling crowds comprise an omnium-gatherum of races and faces. Clothing comprises a dizzying assortment of high fashionista gear and Salvation Army cast-offs.

Febrile consumerism is ubiquitous in this mercantile dystopia, where global corporations, many Japanese, vie for power (possibly alluding to the escalating economic sway of Japan in the real 1980s). Stupendous fluorescent Coca-Cola
signs blaze across building facades. Gaudy blimps hawk the pleasures of life on the “off-world colonies,” where every whim is gratified by obedient androids.

“Replicants”—servants, soldiers, whores—have been genetically engineered by genius entrepreneur, Dr. Eldon Tyrell. Despite an adult appearance, the early models were immature; potentially unstable. So Tyrell provided them with a 4-year life span.

Tyrell’s latest “Nexus” models match or surpass human capabilities. But their dawning sentience is sowing seeds of revolt. Replicants are sporadically killing their masters. A special LA police “Blade Runner” squad has been formed to “retire” them.

Rick Deckard (Harrison Ford), a disillusioned ex-Blade Runner, is unwillingly re-enlisted to execute a particularly vicious replicant hit-squad, led by a super-Nexus warrior, Roy Batty. Deckard is summoned to Tyrell’s corporate flagship to test “a human subject,” his niece Rachel (Sean Young), a ruby-lipped beauty in iconic 1940 Vogue apparel.

Tyrell later reveals to Deckard that, unbeknownst to her, Rachel is actually an “experimental” Nexus, with memory implants to provide “a cushion” against incipient rebellion. Suspecting her true identity, she flees Tyrell’s dubious protection.
Deckard is immediately drawn to her, and dismayed by Tyrell’s latest callous manipulation—“You’re talking about memories!!!” he protests. He goes on to kill Batty’s crew—but only when each attacks him. After Tyrell tells Batty that he cannot give him more life—the hope of that spurred the replicants’ return, Batty crushes his skull like an eggshell, then hunts down the hunter, Deckard.

The chase ends as Batty dangles Deckard over an urban abyss. Suddenly he hauls Deckard to safety, and quietly dies. “I never knew why he saved my life . . . I guess he loved life . . . any life . . . my life,” muses the amazed Deckard. Later, he discovers Rachel has returned to his apartment. The 2 renegades depart, their fate obscure.

The film’s acting is uniformly superb: Ford’s quotidian, ironic woodenness makes him a perfect Deckard. The script—by Hampton Fancher and David Peoples—is terse, mysteriously hieratic, dense with occulted meaning. Thus, Deckard’s furious superior officer: “Stop right there! You know how it is, Dec, you’re either cop or little people!” Batty, pursuing Deckard: “Come on, come on—are you the good . . . MAN????

*Blade Runner* owes much of its power to Vangelis’s magnificent score, mostly electronic, in which Kabuki wails yield to 40’s torch songs. The music, heard alone, imbues the film with a soaring operatic quality.
Blade Runner probes the big questions without flinging them in the viewer’s face. Is memory fixed or plastic, with the potential for manipulation and even effacement by a malign state? Is our humanity—and morality—solely defined by past remembrance? How does memory articulate with one’s sense of origin and purpose—and, above all, of death?

Following a lackluster initial reception, Blade Runner went on first to acquire a cult following, then was lauded by critics and film-makers alike. Not only was it prized for narrative and technical achievements far ahead of its day, but for a prophetic vision of a degraded future.

After several recent years of intense speculation and secrecy, comes before us Blade Runner 2049 (2017). Unlike the first film, it has been universally praised by past viewers, current critics, and favorably received by viewers who never saw the first film.

I’ve said before that the best remake further probes the original film’s concerns. One wonders why the gifted Scott (producer), Denis Villeneuve (director), and Hampton Fancher (co-writer of both films) couldn’t create finer stuff than this empty vessel, ridden with the dumbed-down sound and fury of mainstream, franchise films today.
PICS has become an increasingly important phenomenon in older adults for several reasons. First, the number of older adults with critical illness is rapidly increasing as the population ages and now accounts for about 50% of ICU admissions.1 Second, more than 70% of older adults hospitalized in the ICU develop delirium, which is a major risk factor for ICU-acquired cognitive impairments.2 Third, cognitive and functional impairment before an ICU hospitalization increases the likelihood of cognitive and functional decline afterward.

**CASE VIGNETTE**

Paul, a 76-year-old widower, was found lying on the floor of his home. When the emergency medical technicians arrived, he was having trouble breathing and was not oriented to place or time. In the emergency department, the diagnosis was sepsis secondary to pneumonia. He was intubated and placed on mechanical ventilation. In the ICU he was treated with intravenous fluids, vasopressors, and antibiotics for overwhelming infection and septic shock. After 1 week, he was extubated and transferred to the regular floor. For the first few days on the regular floor, he remained confused and only sometimes recognized his surroundings. Eventually, he improved physically and was fully oriented to person, place, and time upon discharge.

Two months later, Paul’s neighbor brought him to his primary care physician. Paul was having difficulties paying his bills and keeping track of his medication regimen. He was still able to drive a few blocks to the grocery store but became tired if he drove more than 30 minutes. He was also less willing to drive to unfamiliar places because he had difficulty following the GPS. The neighbor noted that Paul was frequently repeating himself and misplacing items. Paul seemed withdrawn and depressed.

Cognitive symptoms

ICU-acquired long-term cognitive impairments (LTCIs) affect 30% to 80% of survivors. LTCIs appear to affect multiple domains, including executive functioning, memory, and attention. For many, LTCIs improve within the first year, although they can persist for many years in some people. Risk factors for LTCIs in older adults include neurological dysfunction, infection or severe sepsis, and acute dialysis. Duration of delirium is also a risk factor for LTCIs.

Current theories suggest that LTCIs from delirium may be distinct from Alzheimer disease (AD). While beta-amyloid deposition appears to be a key factor in the development of AD, long-term cognitive impairment from delirium appears to be caused by hypoxia and pro-inflammatory cytokines. This theory is consistent with the observation that the onset of LTCIs from delirium may depend on the duration of exposure to the pathophysiologic processes of delirium.

(Continued on Page 16)
Post-Intensive Care Syndrome: Recognizing the Critical Need for Psychiatric Care

Evidence also suggests that LTICs from delirium appear to differ from AD in several important ways. First, many patients’ LTICs improve after the acute hospitalization. This trajectory is quite different from the progressive nature of AD. Second, executive functioning in LTICs is associated with more severe depression and subsequently worse mental health quality of life. Finally, LTICs are another important resource. In studies of the acute hospitalization, this trajectory is quite different from the progressive nature of AD. Second, executive functioning in LTICs is associated with more severe depression and subsequently worse mental health quality of life. Finally, LTICs may be associated with white matter damage and smaller superior frontal lobes, thalamus, and cerebellar volumes, whereas AD is classically associated with predominant hippocampal atrophy. However, it is interesting to note that smaller hippocampal volumes are also associated with predominant hippocampal atrophy. However, it is interesting to note that smaller hippocampal volumes are also associated with hippocampal atrophy. However, it is interesting to note that smaller hippocampal volumes are also associated with hippocampal atrophy. However, it is interesting to note that smaller hippocampal volumes are also associated with hippocampal atrophy.

Mental health symptoms
Depression, anxiety, and PTSD are the best-characterized mental health impairments in LTICs. Among adult ICU survivors, the prevalence of depression is 19% to 37%; the prevalence of anxiety is 32% to 40%; and the prevalence of PTSD is 19% to 22%. Psychiatric comorbidity in ICU survivors is 4 to 6 times more common than in the general population (25% to 33% of ICU survivors versus 6% for the general population). Moreover, patients in whom critical illness develops may be more likely to have premorbid psychiatric illness compared with those hospitalized in general wards and the general population.

CASE VIGNETTE (CONT’D)

Paul was referred to an interdisciplinary ICU survivor clinic for further workup and management. He completed the Repeatable Battery for the Assessment of Neuropsychological Status Update (RBANS), a 30-minute cognitive screen. His test results showed that he had mild cognitive impairment, multidomain amnestic type, and comorbid moderate depression. The consulting psychiatrist recommended starting venlafaxine to help with his depression and neuropathic pain. He was also referred for additional outpatient physical therapy and to a psychologist for cognitive behavioral therapy for his depression.

Treatment strategies
There are no multisite randomized, double-blind, placebo-controlled studies that show the efficacy of medications to ameliorate long-term cognitive impairment or mental health symptoms of LTICs. Studies are underway to test the efficacy of treatments for LTICs. The mobile Critical Care Recovery Program is an ongoing clinical trial studying the efficacy of a home-based multidisciplinary intervention for LTICs. The IMPROVE trial is another ongoing clinical trial to test whether combined physical and cognitive training can improve outcomes of long-term cognitive impairment. Physical and occupational therapy should be utilized to ameliorate functional difficulties. Home health services can be invaluable for the patient who remains in the home independently, while reducing caregiver burden. Added medications during the hospitalization can result in polypharmacy, which psychiatrists can help reduce. ICU patients are often discharged on inappropriate medications, and they continue to take these medications for a significant duration. These medications include anticholinergics, opioids, nonbenzodiazepine hypnotics and benzodiazepines, and atypical antipsychotics. Despite the recommendation to minimize use of antipsychotics, close to one-quarter of patients who were given a new antipsychotic during their hospitalization were discharged on an antipsychotic. If clinicians decide to continue psychotropics upon discharge from the ICU, they should carefully weigh the benefits versus risks, such as the FDA black box warning for antipsychotics.

Evidence for the efficacy of psychotropics specific for ICU survivors is limited. It makes sense, however, to deliver evidence-based mental health treatment and for primary care physicians to screen for depression and anxiety using self-report questionnaires such as the Patient Health Questionnaire-9 (PHQ-9) for depression and Generalized Anxiety Disorder-7 (GAD-7) for anxiety. ICU survivors with depression and anxiety should be aggressively treated with antidepressants and depression-focused psychotherapies. Those who have a history of treatment with mental health specialists, have been hospitalized on psychiatric wards, received ECT, or are not responding to first-line treatments should be referred for subspecialty mental health treatment. Dedicated post-ICU survivor clinics are another important resource. In 2016, the Society of Critical Care Medicine (SCCM) sponsored the creation of a national collaborative network of ICU survivor clinics, known as the Thrive Post-ICU Clinic Peer Collaborative. A small longitudinal study of the Critical Care Recovery Center in Indianapolis found beneficial effects on patients’ cognitive and functional symptoms. Future studies will need to further examine the effectiveness of ICU survivor clinic across the US.

Special challenges with older adults
There are a number of special challenges with older adults. Although age is not a risk factor for long-term cognitive impairment, older adults are more likely to develop delirium compared with their younger counterparts. Older adults are also more likely to develop functional difficulties post-ICU, since they may already have functional disability before hospitalization.

Studies have found mixed results on whether age increases the risk of mental health symptoms. Nevertheless, older adults frequently encounter additional barriers as they seek mental health treatment. For example, older adults who need psychotropics may be at increased risk for adverse effects of medications and may have to overcome more physical challenges to attend psychotherapy sessions.

Future directions
The rapid growth in numbers of older ICU survivors presents the field of medicine and surgery with an unprecedented clinical challenge to care for this population. Psychiatric morbidity in ICU survivors is associated with adverse effects on patients’ quality of life and increased acute care service utilization after discharge from the ICU. However, the role of mental health professionals in these ICU survivorship models has not been well-defined.

Psychiatrists can play a key role in building and leading new health delivery models for PICS. Because clinical practice guidelines for the neuropsychiatric sequelae in ICU survivors do not exist, psychiatrists provide invaluable input on the diagnosis and management of post-ICU cognitive and mental health impairments. Next, psychiatrists need to collaborate with the leadership in the hospital to build system-wide interventions for ICU survivors. These interventions should focus on a proactive assessment of psychiatric comorbidity for both patients and caregivers upon admission. These interventions should create a plan for ongoing management throughout patients’ entire hospitalization and in the outpatient setting. A recovery care coordinator needs to be identified during the ICU hospitalization. This ensures that care coordination starts with patients and caregivers during the ICU hospitalization. The recovery care coordinator can collaborate with the psychiatrist to implement the individual care plans and ensure continuity of care in outpatient settings. Most importantly, the recovery care coordinator continues to work with the patient and family in the outpatient setting until the care plan goals are achieved.

All psychiatrists, including geriatric psychiatrists, can be part of interdisciplinary teams by providing clinical services and leading clinical practice and research innovations. They can provide diagnostic and...
Understanding Sleep Disorders in Older Adults

Kate Richards, MD, Jeremy Demartini, MD, and Glen Xiong, MD

Dr. Richards is a Resident Physician in Family Medicine and Psychiatry, UC Davis Health; Dr. Demartini is Chief Resident Physician in Internal Medicine, UC Davis Health, Internal Medicine and Psychiatry; and Dr. Xiong is Associate Professor, UC Davis Health, Internal Medicine and Psychiatry.

Sleep disorders are highly prevalent in the older population and frequently encountered in psychiatry and primary care. More than one-half of the elderly have at least one sleep complaint.1 Given the impact of sleep on quality of life, cognitive functioning, and health outcomes, understanding sleep disorders in older adults is vital to their overall care.

The most common sleep disorder both in the general population and in seniors is insomnia. Women tend to have a higher incidence of insomnia than men. Findings indicate that insomnia affects 31% to 38% of adults aged 18 to 64 years but 45% of those aged 65 to 79 years.2 Many factors can contribute to insomnia, including psychiatric or neurological disorders, medical conditions, polypharmacy, medication adverse effects, substance use, environmental changes (home, hospital, care home), decreased sensory input (blindness, deafness), unremitting pain, sensory input (blindness, deafness), unremitting pain, sleep-onset latency (taking longer to fall asleep), more nighttime awakenings, and decreased total sleep. With aging, progressively more time is spent in light sleep (stages 1 and 2), and less time is spent in deep sleep (stages 3 and 4). In addition, REM sleep is decreased. As sleep becomes more fragmented, there are more frequent shifts between sleep stages and more opportunities for awakenings.

Changes in circadian rhythm can also occur, because of decreased responsiveness of the suprachiasmatic nucleus.

TABLE 1. Medical conditions that can contribute to insomnia

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Key sleep-disrupting symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congestive heart failure</td>
<td>Paroxysmal nocturnal dyspnea, orthopnea</td>
</tr>
<tr>
<td>Chronic lung disease (asthma, COPD, pulmonary hypertension)</td>
<td>Coughing, oxygen desaturation, shortness of breath</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>Nocturnal angina pectoris</td>
</tr>
<tr>
<td>Gastroesophageal reflux disease</td>
<td>Heartburn worse while lying down or eating too close to bedtime; nocturnal cough</td>
</tr>
<tr>
<td>Anemia</td>
<td>Fatigue, poor sleep quality, restless legs</td>
</tr>
<tr>
<td>Dementia (Alzheimer disease, Parkinson disease, vascular disease)</td>
<td>Sleep disturbances with dementia-related sleep behaviors, cognitive decline, hypoxia, confusion, and shifted sleep-wake cycle; increased association of REM sleep behavior disorder with Parkinson disease</td>
</tr>
<tr>
<td>Delirium</td>
<td>Waxy and waning mental status; disrupted sleep-wake cycles</td>
</tr>
<tr>
<td>Post-stroke</td>
<td>Increased risk of obstructive sleep apnea; hypersomnia (increased daytime sleepiness)</td>
</tr>
<tr>
<td>Obstructive sleep apnea</td>
<td>Awakenings or arousals with apneic episodes; daytime fatigue despite adequate time asleep</td>
</tr>
<tr>
<td>Acute or chronic pain (arthritis, cancer, gout, neuropathy, fibromyalgia, musculoskeletal)</td>
<td>Delay in falling asleep; frequent awakenings; difficulty finding a comfortable position</td>
</tr>
<tr>
<td>Benign prostatic hyperplasia</td>
<td>Nocturia, urinary retention</td>
</tr>
<tr>
<td>Renal disease</td>
<td>Excessive daytime sleepiness, restless legs</td>
</tr>
<tr>
<td>Endocrine (menopause, thyroid disease)</td>
<td>Hot flashes, night sweats, nocturnal awakenings, daytime fatigue</td>
</tr>
<tr>
<td>Diabetes</td>
<td>Polyuria, polydipsia, and polyphagia; fluctuating blood glucose levels (hyperglycemic and hypoglycemic episodes)</td>
</tr>
</tbody>
</table>

COPD, chronic obstructive pulmonary disease.
Understanding Sleep Disorder in Adults

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nucleus (which controls our internal clock) to external cues (light). Circadian rhythm dysfunction can be exacerbated by the loss of daily structure (or typical work routines), increased daytime napping, or inconsistent exposure to light, such as in nursing homes where day and night blend together. This often leads to advanced sleep phase syndrome, a circadian shift in the sleep-wake cycle where older adults feel tired earlier and earlier each evening, and then wake up earlier and earlier each morning. These changes in sleep architecture can result in less total sleep, decreased sleep efficacy (the proportion of time asleep compared with time in bed), and early morning awakenings.

Medical disorders, substance use disorders, and medications

Older adults often have one or more medical comorbidities that can contribute to difficulty with sleep. Data from an epidemiologic study of 6800 elderly adults over 3 years showed that only 7% of the cases of insomnia were in isolation of common comorbid conditions. Heart disease, diabetes, and respiratory disease were all associated with long-term persistence of insomnia at the 3-year follow-up (Table 1).

Finding and treating the underlying cause of the sleep disturbance is critical to effective management. For example, awakenings from sleep to urinate can be triggered by nocturia from benign prostatic hyperplasia, from polyuria with diabetes, from drinking water or alcohol too close to bedtime, or from evening doses of diuretics.

Substance use is often underreported in older adults and can adversely affect sleep, especially with metabolic changes with age. For example, caffeine can have a stimulant effect for over 8 to 14 hours in older adults, and alcohol can also take longer to metabolize, leading to disrupted sleep and nocturnal awakenings. Nicotine can also interfere with falling asleep and decrease sleep duration. These changes increase sleep fragmentation and can worsen symptoms from other sleep disorders, such as obstructive sleep apnea.

Careful review of pharmaceutical agents is necessary in geriatric patients with sleep disruptions (Table 2). Older adults, especially those with multiple psychiatric or medical comorbidities, are usually excluded from clinical drug trials. Clinicians also must consider reducing usual doses for older adults, as there is increased accumulation of drug due to a larger volume of distribution (the elderly have a higher relative ratio of fat to muscle) and decreased drug clearance (due to age-related decrease in renal or hepatic function). Furthermore, older adults have a higher risk of adverse events related to polypharmacy, so clinicians should consider stopping or decreasing the dose of a current medication before adding a new medication.

Patient education regarding timing of medications is advised, so that stimulating medications are taken in the morning and sedative medications are taken in the evening. Medications intended to treat insomnia, such as antideprerpessants, benzodiazepines and their agonists, antipsychotics, and antihistamines, can cause excessive daytime sleepiness in older adults, which may lead to delirium, falls, or rebound insomnia, or paradoxically disrupt the sleep-wake cycle.

Psychiatric disorders and risks of antipsychotic use for insomnia

When assessing sleep, screening for psychiatric disorders is vital because many people with depression or anxiety initially present with insomnia or fatigue. In one study, 65% of patients with depression, 61% of patients with panic disorder, and 44% of patients with generalized anxiety disorder reported insomnia. Patients with PTSD can also have disrupted sleep with distressing nightmares. These diagnoses may be unrecognized or undertreated in older adults.

Inadequate sleep can also worsen psychiatric disorder symptoms and can contribute to suicidality. In one 10-year study among older adults with poor quality sleep, the risk of death by suicide was increased 1.4-fold. Similarly, in a 2-year prospective study, risk of depression recurrence was 5 times more likely in older adults with insomnia, independent of other symptoms of depression, antidepressant use, other medical comorbidities, or socio-demographic factors. This model predicted that treating 5 cases of insomnia could prevent one patient from relapsing into depression.

Although second-generation antipsychotics are often used for bipolar spectrum and psychotic disorders in older adults, these medications are frequently prescribed off-label for sleep or sedation. A meta-analysis on the off-label use of second-generation antipsychotics showed that there was a small benefit in using them for psychosis, agitation, and other behavioral symptoms in elderly patients with dementia, but there was not enough evidence to justify their use for insomnia. Furthermore, adverse effects, especially cardiovascular symptoms, fatigue, extrapyramidal symptoms, falls, and urinary symptoms were common, and there was a 1.5-fold increase in risk of death.

Significance for the Practicing Psychiatrist

Sleep disorders, including insomnia, are commonly seen in older adults, and special considerations are needed for safe management and treatment in this population.

- Physiologic changes; environmental triggers; polypharmacy; and comorbid medical, psychiatric, and substance use disorders can all affect sleep in older adults.
- Sleep hygiene and cognitive behavioral therapy for insomnia are safe, first-line tools to effectively manage insomnia with successful short- and long-term outcomes.
- Medications for sleep have significant risks in the geriatric population and are an option of last resort; prudent clinical judgment is needed in cases where judicious prescribing is warranted.

Treatment recommendations

To effectively manage sleep disorders in older adults, physicians must first screen for symptoms of fatigue or poor sleep. Next, a thorough history that includes symptom severity, chronology, exacerbating/ameliorating factors, associated symptoms, sleep-wake patterns, and details about the sleep environment is gathered. A sleep log is a helpful tool for collecting additional data. Medical history, psychiatric history, substance use, and medications should also be reviewed for potential triggers. Then, a physical examination (focusing on the oral cavity, neck, heart, and lungs) and a mental status exam are done. Basic laboratory studies can screen for thyroid disorders, anemia, liver/renal dysfunction, and blood glucose abnormalities. Any signs of untreated medical conditions or sleep disorders (such as obstructive sleep apnea) should be further evaluated. (The Figure illustrates a suggested treatment algorithm.)

The first step in treatment is to optimize management of comorbid medical, psychiatric, or substance use disorders as these often can contribute to insomnia. Without treatment of the underlying etiology, the sleep dysfunction is unlikely to resolve.

Improving sleep hygiene is very effective and low-risk in older adults. Interventions include establishing a consistent bedtime that is late enough to decrease early morning awakenings, finishing meals several hours before going to bed, having a relaxing bedtime routine, minimizing caffeine/alcohol/nicotine intake, managing stress, and increasing morning exercise. Older adults should avoid sleep-promoting activities, such as daytime napping, using the bed for activities other than sleep, and looking at the TV/computer before bed, as the light can be stimulating. Instead of lying in bed awake and watching the clock, getting up and doing a soothing, non-stimulating activity can be helpful.

The bedroom should be comfortable, cool, dark, and quiet (except for white noise or a fan). Progressive muscle relaxation (thoughtfully recognizing and controlling muscle tension), biofeedback, listening to music, or meditation can also be helpful.

Cognitive behavioral therapy for insomnia (CBT-I) is a recommended first-line treatment, as it “produces reliable, durable benefits in 70% to 80% of patients, may reduce the use of sedatives, and improves time to fall asleep, continuity, restfulness, and duration of sleep.” CBT-I was found to be superior to medication use in short- and long-term manage-
Strategies of CBT-I include correcting cognitive distortions about insomnia, improving sleep hygiene, addressing maladaptive behaviors, reducing stimuli that promote wakefulness, and incorporating relaxation training/biofeedback. Patients can receive CBT-I through trained therapists or self-guided modules. Brief behavioral treatment for insomnia (BBTI) is derived from CBT-I but delivered in one session with 2 to 3 brief follow-ups, focusing on sleep consolidation. When medications are considered

<table>
<thead>
<tr>
<th>Medication</th>
<th>Benefits and recommendations</th>
<th>Selected risks</th>
<th>Warnings in older adults (Beers list)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>FDA-approved</strong></td>
<td></td>
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<tr>
<td>Benzodiazepines</td>
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<tr>
<td>Estazolam, flurazepam, quazepam, temazepam, triazolam</td>
<td>For frequent awakenings; short-term use only</td>
<td>Rebound insomnia, complex sleep-related behaviors, daytime withdrawal; long-term effects unknown; not recommended for patients with respiratory disorders (risk of respiratory depression) or substance use disorders (potential tolerance or addiction)</td>
<td>Cognitive impairment, delirium, falls, unsteady gait, syncope, accidents, fractures</td>
</tr>
<tr>
<td>Benzodiazepine receptor agonists</td>
<td></td>
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<tr>
<td>Zolpidem, zolpidem CR, zaleplon, eszopiclone</td>
<td>Increased sleep duration; rapid onset to decrease sleep latency; not shown to decrease number of awakenings</td>
<td>Rebound insomnia and adverse effects similar to benzodiazepines; long-term effects unknown; not recommended for patients with respiratory disorders (risk of respiratory depression) or substance use disorders (potential for tolerance or addiction); residual daytime sedation and next-day impairment</td>
<td>Cognitive impairment, delirium, falls, syncope, motor vehicle accidents, fractures, minimal benefit</td>
</tr>
<tr>
<td>Melatonin receptor agonist</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Ramelteon</td>
<td>For sleep onset and maintenance; less risk of tolerance or addiction</td>
<td>Do not take with melatonin</td>
<td>N/A</td>
</tr>
<tr>
<td>Tricyclic antidepressant</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Doxepin</td>
<td>Sleep maintenance; less risk of tolerance or addiction</td>
<td>Dizziness, nausea/vomiting, ringing in ears, weight gain, breast swelling, decreased libido</td>
<td>Anticholinergic: confusion, orthostatic hypotension, sedation, urinary retention, constipation, dry mouth, cognitive impairment, delirium; SIADH</td>
</tr>
<tr>
<td>Orexin receptor antagonist</td>
<td></td>
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<tr>
<td>Suvorexant</td>
<td>Sleep maintenance</td>
<td>Next-day impairment, headache, dizziness, abnormal dreams, diarrhea</td>
<td>N/A</td>
</tr>
<tr>
<td><strong>Not FDA-approved</strong></td>
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<tr>
<td><strong>Antihistamines</strong></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Diphenhydramine, hydroxyzine, doxylamine</td>
<td>Inexpensive; over-the-counter</td>
<td>Reduced clearance in the elderly; avoid in patients &gt; 60 or in any patients with closed-angle glaucoma, prostatic hypertrophy, severe asthma, or COPD; limited data regarding efficacy and safety; residual daytime sedation and weight gain</td>
<td>Anticholinergic: confusion, orthostatic hypotension, sedation, urinary retention, constipation, dry mouth, cognitive impairment, delirium</td>
</tr>
<tr>
<td>Antidepressants</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Trazodone</td>
<td>Consider in patients with comorbid depression</td>
<td>Residual daytime sedation, headache, dizziness, orthostatic hypotension, priapism, cardiac arrhythmias, psychomotor impairment in the elderly, some evidence of tolerance</td>
<td>N/A</td>
</tr>
<tr>
<td>Mirtazapine</td>
<td>Consider in patients with comorbid depression</td>
<td>Sedation is inversely dose dependent (higher doses are less effective for sleep); increased appetite/weight gain</td>
<td>SIADH</td>
</tr>
<tr>
<td>Tricyclic antidepressants</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amisulpride, imipramine, nortriptyline, desipramine</td>
<td>Consider in patients with comorbid depression</td>
<td>High risk in the elderly due to anticholinergic, antihistamine, and cardiovascular adverse effects; can reduce seizure threshold</td>
<td>Anticholinergic: confusion, orthostatic hypotension, sedation, urinary retention, constipation, dry mouth, cognitive impairment, delirium; SIADH</td>
</tr>
<tr>
<td>Antipsychotics</td>
<td></td>
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<tr>
<td>Quetiapine</td>
<td>Consider in patients with comorbid psychotic or bipolar disorder</td>
<td>Residual daytime sedation, orthostatic hypotension, unsteady gait, cognitive impairment, constipation, syncope, falls, and metabolic changes (weight gain, dyslipidemia, glucose dysregulation)</td>
<td>Increased risk of stroke and death in patients with dementia; SIADH</td>
</tr>
<tr>
<td>Other</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Melatonin</td>
<td>Physiological; inexpensive; over-the-counter</td>
<td>Delayed onset of action</td>
<td>Ongoing studies, but limited data</td>
</tr>
</tbody>
</table>

SIADH, syndrome of inappropriate antidiuretic hormone secretion; COPD, chronic obstructive pulmonary disease.
for insomnia, shared decision-making with the patient is necessary given that the risks of medications can be severe and include delirium, agitation, falls, and drug-drug interactions (Table 3). Zolpidem has been implicated in more than one-fifth of all emergency department visits for psychotropic adverse drug events in older adults. Moreover, data studies that show drug efficacy are limited and often weakened by biases from corporate sponsorship, short follow-up, comparison with placebo only rather than standard treatment, and small sample sizes. Ideally, medications should not be used alone as the only treatment, and should only be used as short-term adjuncts to behavioral management in cases where benefits of medication outweigh risks. Practical recommendations are to choose a medication with a shorter half-life to minimize daytime drowsiness, and to use the smallest effective dose for the shortest duration of time (3 to 4 weeks only) with intermittent dosing (as needed rather than daily). For older adults, use lower starting doses (half of normal dose), given the increased risk of adverse effects.

Because pharmacotherapy is not intended for long-term use, non-pharmacologic and/or behavioral interventions should always be emphasized concurrently. This can ease rebound insomnia and anxiety associated with discontinuing medications once the short course has ended. Medications are an option of last resort in limited circumstances, and prudent clinical judgment is necessary for cases in which judicious prescribing is warranted.

The authors report no conflicts of interest concerning the subject matter of this article.

References
8. Greenberg OnTheArts | Blade Runner 2049

The new film posits that during a 3-year period of “darkness,” records of all human and replicant identities were expunged. The bankrupt Tyrell empire was succeeded by the Wallace Corporation, its blind CEO, Neander Wallace, designed a new Nexus line, stronger, smarter than the earlier models—and absolutely loyal. This Tiresias in a Zegna suit spouts sententious Zen twaddle about creating an army of replicant “angels” that will spread his beneficence across the galaxy—could he but grind his twins out more quickly.

The protagonist, K (for Kafka?), one of the unfaithfully obedient new breed, is charged with retiring the few remaining Nexus 8’s who went to ground after the “darkness.” K is played by Ryan Gosling, who usually projects an intriguing, distanced quality. Here, he merely runs the gamut of emotions between A and B. While “retiring” a Nexus 8 in a desert shack, K discovers a trunk containing a female skeleton and a tattered baby’s sock. The bones are Rachel’s, who died giving birth to human/replicant twins fathered by Deckard. K is forced to choose between exterminating the twins so that society’s fragile status quo can be maintained; and Wallace’s lunatic plan to capture the female twin in aid of making her the fertile progenitor of his “angel” army.

But K becomes convinced that he himself is one of the twins, and therefore—at least partly—an autonomous human, rather than a thing of compliant biomorphic cog- and-wheels. He turns rebel, takes Deckard from his mancave in a radioactive, gaudy Vegas hotel, and unites him with his grown daughter.

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Functional Connectivity: Probing the Brain’s Astounding Complexity

Barbara Schildkrout, MD

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One of the greatest scientific challenges for 21st century medicine is to illuminate the relationship between the brain and what we call “mind.” Psychiatrists want to know how we get from neurons and synapses to mental suffering. How do learning, development, and cognitive flexibility arise? What accounts for the uniqueness of every human self? What goes wrong in psychiatric disease? Within the psychiatric community, brain science has generally been regarded as being still too elementary to explain such complex phenomena.

In this article I describe how analysis of the brain’s intrinsic functional connectivity has become an important approach for expanding our understanding of the astounding complexity of the human brain. Utilizing this new paradigm, it is possible to explore questions that earlier seemed virtually unfathomable; many of these are relevant, even pivotal, to psychiatry.

In news parlance, functional connectivity is a “rapidly developing scientific story.” And for psychiatrists, it is a story worth following.

What is functional connectivity?

Functional “imaging” measures physiological factors that are considered to be a gauge of neuronal functioning (such as changes in regional oxygen utilization); the accumulated data are then transformed into “human readable” images. Functional “connectivity” utilizes functional imaging data and analyzes the statistical associations between measurements of neurophysiological activity in 2 or more spatially remote areas of the brain. Functional connectivity studies are a mathematical, non-theoretical look at activity over the whole brain, in an attempt to discern in which areas the activity is either correlated or anti-correlated. For example, do areas A and X display increased metabolic activity when area D exhibits decreased metabolic activity?

Traditional functional connectivity methods do not tell us about the direction of connectivity—which region is influencing which. Functional connectivity also does not tell us whether 2 regions are simultaneously being influenced by a third. Nor does functional connectivity say anything about the way in which various brain areas might be structurally connected. Functional connectivity takes a purely statistical look at larger patterns of neurophysiologic activity that emerge from the brain’s hundreds of billions of neurons, reciprocally interacting over both short and long distances at microsecond speed.

Early functional connectivity research was designed to study the brain while an individual was performing a task or interacting with the environment—termed “psychophysiological interactions.” More recent work has shifted to a focus on the brain’s intrinsic neural activity while the subject is at “rest” or engaged in undirected thought.

The historical context

It was not until the early 1800s that modern scientific notions about the brain began to take precedence.2 Franz Joseph Gall promoted the idea that various mental capacities were localized to different brain regions; he also believed that the strength of

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these qualities could be measured by looking at protrusions in the skull overlying these areas. This notion was at the heart of phrenology, extremely popular in the 19th century.

Gall assumed that the brain was symmetrically organized. His categorization of what constituted fundamental, functional capacities of the brain drew on cultural values of his day, including vanity, guile, kindness, and pride. Although scientific advances have discredited many of these claims, Gall’s fundamental insight about localization was profound.

Localization was a powerful new idea at the time; it exhibited the 2 fundamental attributes of a paradigm as first defined by the science historian Thomas S. Kuhn as “... sufficiently unprecedented to attract an enduring group of adherents away from competing modes of scientific activity. Simultaneously, it was sufficiently open-ended to leave all sorts of problems for the redefined group of practitioners to resolve.”

The localization paradigm opened new questions for legitimate scientific inquiry; neurological investigators became the Lewis and Clark of neuro-anatomic territories. More than 200 years of explorations in neurology have been spent mapping the localization of various functional elements within the brain and simultaneously parsing brain activity into its most fundamental capabilities.

For example, until the mid-1950s, memory was considered to be a widely distributed, unitary function of the brain. Then, studies of patient H. M. elucidated a central role for the hippocampus in memory formation. In addition, episodic and procedural memory processes were differentiated.

While the concept of localization drove much of the work of scientific exploration during this time, there also were global, overarching theories of brain function. The eminent British neurologist John Hughlings Jackson posited that the more evolutionarily developed regions of the brain exerted control over primitive brain areas; he articulated how disturbances in this organization were evident in disease states. Indeed, many scientists who are well known for their work on localization (Wernicke, Penfield) also were aware that the brain was extraordinarily complex and that the parts had to work together, like an astoundingly accomplished orchestra.

Structural connectivity

An important step in expanding the localization concept and focusing on how various parts of the brain communicated was the recognition of disconnection syndromes in the 1960s. Disconnection syndromes did not result from lesions in the cortical gray matter itself but rather from interruptions in the white matter fibers and tracts running to and from cell bodies in gray matter—the lines of communication within the brain. If a lesion in white matter isolates or “disconnects” a crucial cortical region, this could lead to a clinical picture similar to one seen with a lesion in the cortical area itself. In other words, now neurologists were turning their attention to structural connectivity, the so-called wiring diagram of the brain.

Astoundingly, all of this work was accomplished by utilizing only clinical observation, scientific reasoning, and post-mortem findings. The only imaging available was pneumoencephalography, a painful technique that allowed clinicians to visualize the shape of the ventricles in the brain of a living patient by using x-rays after injecting air into the spinal column.

Structural brain imaging

Until computed tomography (CT) came into clinical use in 1973, it was not possible to visualize brain parenchyma during life. Not until 1994 was there a technique that spared radiation exposure—magnetic resonance imaging (MRI), CT, MRI, and other brain imaging approaches have allowed us to make remarkable advances in mapping human brain structures and in diagnosing human disease, but these modalities also have inherent limitations. An MRI scan is not a “picture” in the usual sense but rather a computer-generated readout of summary information into a digital image. One voxel (essentially a 3-dimensional pixel) in brain imaging contains information from approximately 1 cubic millimeter. Within 1 cubic millimeter of gray matter are perhaps a million neurons plus glia cells, blood vessels, and extracellular space. In other words, none of these structural imaging techniques approaches the level of the individual neuron. Also, a structure-alone perspective of the human brain has other inherent limitations. For example, even if we take into account ongoing remodeling of neuronal connections (neuroplasticity), how can we account for the brain’s astounding moment-to-moment flexibility?

Functional brain imaging

Functional imaging techniques measure fluctuations over time in factors that gauge neuronal activity such as regional glucose utilization, blood flow, or oxygen consumption. The most widely used functional imaging techniques are positron emission tomography (PET), which utilizes radioactively tagged molecules, and functional MRI (fMRI), which differentiates molecules by their behavioral responses within a magnetic field.

One important functional imaging approach is based on the observation that, in a magnetic field, oxygenated blood behaves differently than deoxygenated blood. This allows researchers to track fluctuations in blood-oxygen levels in human subjects in the scanner without the use of radiation. This blood-oxygen level dependent (BOLD) signal has become a major investigative tool.

Functional imaging techniques also allow clinicians to evaluate patients who may have deficits in neuronal functioning even when structural deficits are not apparent. For example, early in the disease course of frontal dementias, patients may have decreased functioning in frontal regions before any changes are seen on...
Functional imaging has made it possible for investigators to pursue a myriad of fundamental questions. One line of such inquiry examines which parts of the brain are active as someone performs a carefully designed task. For instance, how is a familiar face different from viewing an unfamiliar one? Which parts of the brain are involved in moral decision-making? Also, for a given task, are normal individuals functionally different from people who have specific psychiatric conditions?

Limitations of functional imaging

A serious obstacle for functional imaging research has been distinguishing a “task signal” of significance from background “noise.” The brain is metabolically very active, utilizing approximately 20% of body energy resources even though it represents only 2% of body mass. Most of this energy utilization is from ongoing neuronal metabolism. When performing a demanding cognitive task, the brain’s energy utilization increases by less than 5%. Furthermore, differences that might exist between the normal and the study populations are even smaller. These factors make the task signal difficult to detect.

Also, individuals vary in their level of effort, degree of anxiety associations related to the task that come to consciousness, movement during the study, and so on. Although these brain-based activities are not the focus of the study, the metabolic activity they produce shows up in the scanner. Therefore, to amplify the task signal and also draw broad conclusions, a widely used approach to studying task-related questions has been to pool the findings from numerous individuals and average the results onto a standard anatomical brain atlas.

Along with these technical challenges, functional imaging studies also have a theoretical limitation built into their fundamental design. Task-based functional connectivity studies focus on the brain correlates of the task and assume the brain’s ever-present background neuronal metabolic activity is simply “noise.”

Resting state connectivity and its importance

In 2001, an important observation changed the field of functional imaging. Marcus Raichle compared PET and, later, IMRI BOLD signal findings from research subjects who had been engaged in task-based studies with those who were in control groups. Dr. Raichle’s laboratory routinely used the “rest condition” as a control rather than using, for example, a neutral task as a control for an emotional one.

At some point in our work, and I do not recall the motivation, I began to look at the resting state scans minus the task scans. What immediately caught my attention was the fact that regardless of the task under investigation, activity decreases were clearly present and almost always included the posterior cingulate and the adjacent precuneus. . . . Initially puzzled by the meaning of this observation, I began collecting examples from our work and placed them in a folder which I labelled [sic] MMPA for mystery medial parietal area.

Further analysis of data by Marcus Raichle and his colleagues led to the identification of a network of specific brain regions in which activity was anti-correlated with task-based activity no matter what the task was. This network was named the Default Mode Network (DMN) by Michael D. Greicius. The work of Raichle and others was consistent with the first published report of intrinsic resting state functional connectivity by Biswal and colleagues in 1995 that “functionally related brain regions exhibited correlation of low frequency fluctuations in the resting state.”

The importance of these discoveries has been far-reaching

Consider that previously, in task-based functional imaging studies, the challenge had been to find the signal within the experimental background “noise.” Now it had become clear that this “noise” was data. The fluctuating BOLD signal could be mathematically mined as a source of information about the intrinsic functional organization of the brain. Moreover, the data could be obtained relatively easily by placing a person in a scanner and instructing him or her to “rest” or to visually fixate on 1 spot: this made it possible to study some patients who had difficulty cooperating with other protocols.

Moreover, using functional connectivity did not require patient aging. Subjects could be studied individually, making it possible to compare different individuals or individual at different times. These advantages made the prospect of utilizing functional connectivity as a clinical tool more viable.

For researchers, “. . . functional brain connectivity . . . [had] become one of the most influential concepts in modern cognitive neuroscience, especially given the current shift in emphasis from studies of functional segregation to studies of functional integration.” We had long appreciated that detailing the synapse-to-synapse, structural organization of the brain would not capture the brain’s vast neuronal networks at work, operating as a dynamic system at speeds that would support the myriad manifestations of complex human behavior. Now “. . . task-free analysis of intrinsic connectivity networks may help elucidate the neural architectures that support fundamental aspects of human behavior.”

What neuroscience has learned from studies of functional connectivity

The DMN is only one of numerous large-scale, intrinsically synchronized, dynamic and interacting, functionally organized networks in the brain. These intrinsic functional networks and important nodes or hubs in those networks are consistent with synaptic maps of the brain. In other words, these networks do not violate our previous understanding of the anatomical organization of the brain into systems for motor behavior, perception, cognition, and so on.

The intrinsic functional networks can be found during cognitive tasks and in the rest condition, even when the systems for motor behavior, perception of various kinds, cognition, etc., are not being consciously engaged. Indeed, these networks persist during sedation, sleep, and under anesthesia.

While the intrinsic functional networks agree with earlier understanding of anatomical organization, the networks are not restricted to neuroanatomical regions with single synapse connections. The astounding degree of structural neural-network complexity in the brain likely explains how regions of the brain might be “functionally connected” even when their “structural connections” are not clear.

There is rapid coordination and interaction among the intrinsic brain networks and their hub regions. The brain is constantly switching connectivity patterns and reorganizing according to demands of the moment. Although the brain is a massively complex dynamic system, it can be studied by utilizing advanced imaging techniques and innovative mathematical and computational approaches. The importance of collaboration between experts in different fields cannot be overstated.

The most studied networks that relate to cognition are the Central Executive Network (CEN), a Salience Network (SN), and the DMN. The rapid interplay of these and perhaps other networks underpins behavioral changes that are based on the individual’s homeostatic needs, given that conditions (both internal and external) shift rapidly. The CEN is most active during cognitive tasks. The SN is activated in response to salient stimuli and plays a role in emotional processing and in switching from the DMN to the CEN. These networks are found in everyone; however, there is individual variation in features such as the strength of connectivity within each network.

The most far-reaching question we posed was whether regional functional connectivity within the salience and executive-con-
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This idea has been used to look at a large variety of traits and conditions. Indeed, individual variations in the coherence of these intrinsic networks appear to correlate with patient capabilities (eg, fluid intelligence, stressor-associated anticipatory anxiety, executive task performance).1,15 Studies confirm that variations in functional connectivity are reliable indicators of specific behavioral variations outside the scanner, the potential clinical uses of this technology are staggering.

Theoretically, the mechanisms by which genetic variation, maturation, and experience affect an individual may be understood within this paradigm of intrinsic functional network organization in the brain. Early variations in neural development as well as experiences over time have an impact on synaptic strength and network functioning; these individual variations, in turn, have far-reaching effects on information processing which corresponds to the clinical symptoms of depression.16

Some neuropsychiatric “mysteries” are being solved through functional connectivity studies. For example, how do single lesions (as from a stroke) in different parts of the brain produce similar, complex neuropsychiatric syndromes in different individuals? Darby and colleagues17 were interested in this question as applied to the emergence of delusional misidentification syndromes after single lesions. Capgras syndrome is perhaps the best known of the delusional misidentification syndromes, exemplified by: “You look like my wife, but I know you are not my real wife; you are an impostor; my real wife is somewhere else.”

Darby found that the disparate lesions that led to delusional misidentification were functionally connected to brain regions involved in familiarity assessment and in belief evaluation. This finding supports earlier theories about what goes wrong in delusional misidentification: first, the patient recognizes the individual or place but fails to experience that person or place as familiar; and second, there is a failure in “belief evaluation,” namely a failure to realize that it defies logic to believe that this is anything but the real person or place.

Extremely promising work by Emily S. Finn has shown that functional connectivity profiles are specific enough to distinguish individuals, including across different sessions in the scanner and across task and resting states.18 This identification of individuals is referred to as functional connectivity fingerprinting. The ability to capture an individual’s uniqueness from data in the scanner is a truly remarkable achievement.

Conclusion

We have come a long way. It took centuries for the scientific world to understand the fundamentals of brain anatomy and neuro-cognitive architecture. Even as progress was being made in mapping the brain’s neural circuitry, the goal of truly probing the complexity of human behavior felt like a distant star we would likely never reach. Yet, in the mere 25 years since MRI was first introduced into medicine, we have made astounding progress in probing the brain’s complexity. As psychiatrists, we feel a pressing need for new light to be shed on what goes wrong in mental disease. Powerful new scientific paradigms, advancing technology, and cross-discipline collaborations give us reason to be hopeful.

References


Greenberg On The Arts | Blade Runner 2049

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The new film’s overall look is not unimpressive, but this mise-en-scène exists for its own sake, conveying no impression that the sequel has evolved from Blade Runner’s ravaged world. LA’s evocative noir smokiness is replaced by an unperturbing pall, particularly in the Las Vegas sequences: which seem to have been shot through an orange rind.

Other visual citations of Blade Runner are scattered throughout the film willy-nilly, only constituting an empty gesture of familiarity with the original. A prostitute has the same frizzed blond hair and tatty dress as Pris, the much ill-used hooker from the first film. But Pris’s sensitive antic wit is utterly lacking.

Vangelis’s original score was supplemented by sibilant ambient sound often rise to intolerable and meaningless tohubohu.

Dunkirk

As Roy Batty’s life ebbs away before Deckard’s helpless gaze, he’s become more human than his maker in the best attack ships on fire off the shoulder of Orion . . .

I’ve seen things you people wouldn’t believe . . .

I’m saddened—and not a little angry—that Blade Runner’s numinous, lyric beauty, its evocation of grace on the brink of inevitable dissolution, have been all washed away—like tears in rain—by this incoherent hollowed-out replicant.
Adjustment Disorders

Adjustment disorders (ADs) are one of the most frequently diagnosed psychiatric disorders and are a common diagnosis in the military, in children and youth, and in the consultation-liaison psychiatric setting. Their historical evolution, the conundrums of establishing their diagnosis, their relationship with other psychiatric disorders, their treatment, and the potential for a more biological understanding are presented below.

History of adjustment disorder
In 1952 the criteria for transient situational personality disturbance were developed for DSM-I. This emanated from a view developed during World War II that psychological symptoms related to combat experiences were normal responses to abnormal situations and transient unless treated in ways that increased secondary gain. A separate diagnostic category was created for these situationally specific phenomena, and combat-related psychopathology: gross stress reaction.

In DSM-II the category was altered to transient situational disorders—adjustment reactions of life: infancy, childhood, adolescence, late life. It was felt that the symptoms are the means by which individuals struggle to adjust to an overwhelming situation. In the presence of good adaptive capacity, symptom remission occurs when the situational stress diminishes. Persistent failure to resolve the stressors or the effects of the stressor indicates a more severe underlying disturbance. It is important to note that the stressor could be regarded as traumatic or non-traumatic.

The nomenclature was again changed in DSM-III to read ADs, with 10 sub-types: depression, anxiety, depression and anxiety, mixed emotional features, disturbance of conduct, mixed disturbance of emotions and conduct, work or academic inhibition, withdrawal, physical complaints, and not otherwise specified. In addition, other criteria were added: an AD was diagnosed when the individual experienced a stressor (traumatic or not) that was significant (undefined) and that the reaction to the stressor was excessive (undefined) considering the culture of the individual. By intention the stressor, the dysfunction, and the distress were not given measurable values so having these 3 essential entities for the diagnosis was based on clinical assessment alone.

Spitzer, one of the chief architects of DSM-III and DSM-III-TR, regarded the ADs diagnosis as a “wild card” in the psychiatric lexicon by design, in order that there would be a placement for patients who had historical issues that warranted treatment, but who did not reach the threshold for a primary psychiatric diagnosis (eg, MDD, general anxiety disorder). In fact, the ADs diagnosis was not part of the seminal Epidemiological Catchment Area study because it depended on clinical judgment and did not have measurable variables across clinicians or institutions. It was acknowledged that the diagnosis had neither reliability nor validity.

In DSM-IV and DSM-IV-TR, ADs remained an independent diagnosis—an orphan entity—in its own “chapter,” but some of the subtypes were eliminated: work or academic inhibition, withdrawal, physical complaints, not

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otherwise specified. It also remained poorly defined and had no guidelines for the measurement of the diagnostic criteria. The clinician remained the “gold standard” of diagnosis. The term psychosocial stressor was changed to the broader concept of stressor (eg, the Chernobyl reactor incident, cardiac surgery, 9-11 terror attack).

In DSM-5, ADs were combined with a cadre of traumatic disorders in the “Trauma and Stressor-Related Disorders” chapter, which also included PTSD and acute stress disorder (ASD). ADs were no longer an orphan category, and it was anticipated this would not only promote research but upgrade the diagnosis from its historical placement as a sub-threshold entity to a primary psychiatric diagnosis.

The DSM-5 Stress Related Work Group proposed adding 2 additional subtypes, but they were not accepted: AD with PTSD-like symptoms and ASD-like symptoms. This would have allowed a diagnosis for symptom profiles that did not reach threshold for all the required symptoms (ie, not experiencing a traumatic stressor for PTSD and ASD).

Maaeker and colleagues have provisionally made an important difference in ICD-11 diagnostic features for ADs. The criteria require positive symptoms of preoccupation and failure to adapt (ie, inability to recuperate), and dysfunction and distress are required to make the diagnosis. This changes the identifiable criteria for diagnosis of ADs between the 2 major taxonomies for mental disorders and remains a concern for research initiatives.

ADs could appear to be either the prodromal stage of MDD or the state of coming out of major depression as the patient responds to treatment. A refractory AD should make the clinician aware that a more serious depression may be developing. Similarly, AD with anxiety may appear to be a predecessor or successor of an anxiety disorder.

The conundrums of diagnosis
The diagnosis of ADs is subjective and phenomenologically driven, and has no universal biological or genetic markers. In fact, DSM-5 has no measurement standards or check lists for the 4 components essential for diagnosis: the stressor, degree of dysfunction, degree of distress or mood, or alteration of behavior. There is no consensus on reliable and valid measures, algorithms, guidelines, or diagnostic instruments. This makes it difficult to distinguish normal behavior from AD pathology. Phenomenological definitions may be reliable but are not necessarily valid, and this makes the accuracy of the diagnosis difficult.

Normality versus pathology
As Casey has described, there is a need to distinguish normal adaptive responses from recognized disorders. The chasm that separates various diagnostic categories from each other and from normality is called the “zone of rarity.” Using this concept there should be a clear demarcation between those who are ill and those who are not, with few, if any, individuals in the intermediate zone. This is the approach used in general medicine where various diseases are recognized and distinguished from others by their symptoms, etiology, pathophysiology (including biological markers), course, prognosis, and response to treatment.

In psychopathology “zones of rarity” do not exist between various psychiatric conditions, especially the common mental disorders such as MDD, ADs, PTSD, and generalized anxiety. ‘The symptoms of one condition are also often found in others, calling into question the validity of many of the syndromes conventionally regarded as discrete psychiatric disorders. The impact of the failure to find “zones of rarity” is that definitive diagnosis is difficult for the clinician. Thus, diagnostic categories are heterogeneous, and symptoms of one condition are present in others. Casey also emphasizes the difficulties with not only the issue of symptom number but the duration criterion. The tendency to falsely diagnose a psychiatric disorder when there is none is referred to as the false-positive dilemma.

The distress-impairment criterion
According to Casey, this heterogeneity stems from the fact that not all the specified symptoms are required to make the diagnosis and all are regarded as equally important. This is known as the polythetic approach and is the one used by DSM. Individuals with the same diagnosis can present with diverse clinical pictures. One way to address this is to require that certain symptoms be essential to making a diagnosis (the monothetic approach). For example, for PTSD, flashbacks or re-experiencing the trauma would be essential symptoms.

The dimensional approach
In light of the absence of zones of rarity between different psychiatric disorders and between normality and mental illness, dimensional measures instead of categorical labels have been proposed. The dimensions might range from biological measures, to symptom severity scores or measures of social functioning, although little work has been done on these for ADs. Yet for all the problems with the categorical approach, there is no agreement on what dimensions should be measured or to which diagnostic criteria should apply. A dimensional approach would not assist in deciding on the boundary between what a normal reaction to stress is and what is pathological unless the cut-off point is defined by research evidence.

In an attempt to capture the continuum from normality to pathology that is the hallmark of mental illness, DSM-5 incorporates a dimensional as well as categorical approach.

Phenomenology, DSM, and reliability
Science depends on measurement and independent validation, and phenomenology of mental illness constitutes a formal description of the subjective experiences of psychiatric patients without any independent confirmation that they are true. ‘Psychiatric classifications rely on a mix of hard objective data (eg, an MRI scan), softer objective data (eg, neuropsychological testing), and careful clinical observation (observed behavior, symptoms, and phenomenology).

What has been realized is that good reliability is not enough to make a good diagnosis. Trained assessors can always achieve good reliability, but a highly reliable diagnosis can still be completely invalid. As a consequence, the emphasis on operational criteria as the gold standard of diagnosis is questioned. And this is the situation we have with ADs.

Research Diagnosis Criteria (RDoC)
The intent of the RDoC concept is to accelerate new discoveries by fostering research that translates findings from basic science into new treatments addressing fundamental mechanisms that cut across current diagnostic categories. It is important to note that the conceptual framework for RDoC is “explicitly agnostic.” Can ADs be better understood and better diagnosed in the context of the RDoC schema?

As a clinical syndrome, depression is related to abnormal activity in the prefrontal cortex, amygdala, anterior cingulate cortex, nucleus accumbens, and multiple monoamine systems and undoubtedly other central nervous system components. Does this have applicability to AD with depressed mood? Are the physiological correlates of depression (eg, relationship to glucose metabolism, platelet activation, cytokine activity, alterations in cortisone) present in AD with depressive features?

Associated features
Findings indicate that ADs are associated with suicide attempts, completed suicide, substance abuse, somatic complaints, other mental disorders, and being a general medical or surgical patient. ‘A study of the neurochemical variables of AD of all patients who attempted suicide revealed biological correlates consistent with the more major psychiatric disorders. Suicide attempters had lower platelet monoamine oxidase activity, higher MHPG (3-methoxy-4-hydroxyphenylglycol oxidase) activity, and higher cortisol levels than controls. An AD may complicate the course of illness by impairing adherence with the medical regimen or increasing the duration of hospital stay. The behaviors of some patients with AD are seriously pernicious and under-

Science depends on measurement and independent validation, and phenomenology of mental illness constitutes a formal description of the subjective experiences of psychiatric patients without any independent confirmation that they are true.
Regardless of whether psychotherapy or pharmacotherapy is employed singularly or in combination, a significant aspect of treatment of the AD is to remain alert that this initial diagnosis may indicate a patient who is in the early phase of a major mental disorder. Therefore, if a patient continues to worsen, becomes more symptomatic, and does not respond to treatment, it is critical to review the patient’s symptom profile and confirm that the patient does not have an evolving major mental disorder.11

Looking toward the future
Stress-induced alterations in the hypothalamic-pituitary-adrenal (HPA) axis function have been demonstrated in depression, PTSD, and other anxiety disorders.12 Research to determine how the HPA system operates in AD and whether each AD subtype exhibits similar psychobiological alterations needs to be undertaken. Given the great body of research demonstrating stress-related HPA reactions, it would be surprising if at least some of the AD subtypes were not associated with HPA mechanisms.

Wang and colleagues13 reported that telomere length is shortened in patients with depression, anxiety, stress, and ADs. They also observed that the mitochondrial DNA copy number is associated with symptoms of depression, anxiety, stress, and AD.13 Further investigation might reveal that a specific AD subtype has commonality with the primary diagnoses (eg, MDD, general anxiety disorder) or that the subtypes have discrete biological functioning that might separate them from each other.

It is hoped that HPA research and the concept of “allostatic load” (McEwen and Rasgon15) will provide a useful psychobiologic conceptual framework within which to conduct both basic research and clinical trials to enhance our understanding of the relationship among stressor response syndromes: AD, PTSD, and anxiety. This would also provide a theoretical context within which to investigate different therapeutic approaches for the different AD subtypes.

Prevalence
ADs occur in children, adolescents, and the elderly (2% to 8%) in community samples; in acute care general hospital inpatients (12%); in mental health outpatient settings (10% to 30%); and, in special settings (eg, following cardiac surgery, up to 50%).16 In children and adolescents, diagnosed AD may progress to more serious mental disorders over time and into adulthood. (The AD may be an earlier form of a more serious illness that appears later.) The diagnosis of AD may not be used if the symptoms are secondary to the physiologic effects of a general medical illness or its treatment (eg, drugs), nor should it be employed for demoralization.

Treatment
Psychotherapy is the mainstay of treatment and should be the first line of therapeutic intervention. Often, as the stressor or its effects remit, the illness resolves without intervention. Some stressors are self-imposed; other stressors may have accentuated value that is not warranted. Reality orientation by counseling, psychotherapy, crisis intervention, family therapy, or group treatment may be utilized to encourage the verbalization of fears, anxiety, rage, helplessness, and hopelessness related to the stressors. Enhancement of coping skills and resilience and group support are therapeutic approaches that can be used to reduce the concerns and conflicts that the patient is experiencing. Cognitive behavioral therapy has been used successfully in young military recruits who experience adjustment problems.

Randomized controlled trials rarely focus on ADs. Pharmacotherapy decisions are based on the subtype present: depression or anxiety. First-generation antidepressants have been prescribed for depression and benzodiazepines for anxiety. Tianeptine, alprazolam, and mianserin were found to be equally effective in those with anxiety AD. Pharmacotherapy may be combined with psychotherapy if there is no or minimal improvement in functioning and/or distress.
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Jacobi Medical Center

Psychiatrist Opportunities in NYC!
Jacobi Medical Center (JMC) is a modern, state-of-the-art, Level 1 Trauma Center located in an attractive and safe residential Bronx neighborhood just 20 minutes north of Manhattan. It is a North Bronx Healthcare Network hospital affiliated with North Central Bronx Hospital and a teaching site and academic affiliate of the Albert Einstein College of Medicine. It offers a full continuum of acute care inpatient and outpatient services in diverse Medical and Surgical Specialties, including Psychiatry. The Department of Psychiatry has 86 Adult Acute Inpatient beds, a Comprehensive Psychiatric Emergency Program (CPPE), a Consultation-Liaison Service, an Adult Ambulatory Practice, and a Community-Based Assertive Community Treatment Program. The department employs evidenced-based best practices in providing the highest quality care to its patients, in a patient-centered approach that is respectful of their individuality, culture, and community.

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Jacobi Medical Center & North Central Bronx Hospital are currently accepting applications and referrals for the following opportunities:

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American Academy of Child & Adolescent Psychiatry

CME Events

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AACAP's 65th Annual Meeting
October 22-27, 2018  ●  ●  Seattle, WA
Washington State Convention Center
Submissions to the Call for Papers for AACAP’s Annual Meeting are due Thursday, February 15, 2018, or Friday, June 15, 2018, for (date) New Research Posters. The online Call for Papers submission form is available at www.aacap.org/AnnualMeeting-2018; all submissions must be made online. Abstract proposals are prerequisites for acceptance of any presentation. Topics may include any aspect of child and adolescent psychiatry.

For information about all of AACAP’s meetings, visit www.aacap.org, email meetings@aacap.org, or call 202.966.7300, ext. 2006.
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Florida Psych Times
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NEW YORK
OSWEGO, NY – Great Work/Life Balance - College Town on Lake Ontario – 20 Minutes from the Northern New York State Parks
- Syracuse – Outdoor enthusiasts’ paradise: numerous lakes; skiing options close by; 40 minutes from the Thousand Islands; festivals and concerts every weekend throughout the summer. Seeking an additional Psychiatrist to work on a 28-bed adult inpatient psychiatric unit in the Oswego Hospital. Work with a great group of people in a very supportive hospital. Offering salaried position with benefits. Please contact Terry B. Good, Horizon Health, at 804-684-5661; terry.good@horizonhealth.com

Penn State Health Milton S. Hershey Medical Center Department of Psychiatry is currently recruiting board eligible/certified psychiatrists for inpatient and outpatient positions in both adult and child psychiatry.

We are a growing, vibrant department in a strong academic medical center. We host specialty clinical and research programs, including research that crosses the translational spectrum. Our educational programs include adult psychiatry residency, child fellowship, psychology internship, externship and post-doctoral fellows. We have a strong collaboration with basic and clinical science in other neuroscience disciplines across several Pennsylvania campuses.

With our clinical partner, the Pennsylvania Psychiatric Institute, the Department staffs several outpatient and partial hospital programs for children and adults, 89 inpatient beds, ECT and other neuromodulation services, specialty sleep and eating-disorders programs, and expanding psychiatric consultation and integrated care programs for Hershey Medical Center.

Successful candidates should have strong teaching as well as clinical skills and, potentially, potential for scientific and scholarly achievement. We offer an attractive compensation package commensurate with qualifications. Tenure-track positions are possible.

For consideration, send your CV to: Jenna Spangler Physician Recruiter Phone: 717-531-4271 Email: jsapplnger2@pennstatehealth.psu.edu
The Penn State Milton S. Hershey Medical Center is committed to affirmative action, equal opportunity and the diversity of its workforce. Equal Opportunity Employer – MOWN/D

NEW JERSEY
South Jersey Outpatient Private Practice
Adult or Child and Adolescent Psychiatrist - BC/BE licensed in New Jersey to join a multi-disciplinary group of expert clinicians in an outpatient fee for service mental health private practice, near Philadelphia.
Fax CV 856-985-8148 or email Kathy at centracp@snip.net

CENTRA
Comprehensive Psychotherapy & Psychiatric Associates
South Jersey Outpatient Private Practice
Adult or Child and Adolescent Psychiatrist - BC/BE licensed in New Jersey to join a multi-disciplinary group of expert clinicians in an outpatient fee for service mental health private practice, near Philadelphia.
Fax CV 856-985-8148 or email Kathy at centracp@snip.net

PENNSYLVANIA
EASTERN PA – SCHUYLKILL COUNTY – Independent Contractor Position, or Full-time Staff Psychiatrist position if preferred, on Adult inpatient psychiatric unit and C/A psych unit in the Lehigh Valley, Schuylkill Hospital in Pottsville, PA. Independent Contractor arrangement available for those in practice who want part-time work; or if employment is preferred, can offer salary with benefits.

Please contact Terry B. Good, 804-684-5661; terry.good@horizonhealth.com; Fax: 1-804-684-5663. EOE

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