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Inside

IN THIS ISSUE

This month's issue of the *Residents' Journal* features articles on a variety of topics. Shan H. Slddiqi, M.D., and Nicholas T. Trapp, M.D., present findings on the use of transcranial direct current stimulation for cognitive enhancement and major depressive disorder. Cornel N. Stanciu, M.D., and Oliver M. Glass, M.D., analyze the literature on opioid-induced hyperalgesia, including data on distinguishing features, underlying biological mechanisms, and management. Aarti U. Jerath, M.D., M.A., examines modifiable, preventive, and unique factors that might delay onset of cognitive decline. Awais Aftab, M.D., provides discussion on a case of delusional memory in a 19-year-old patient with first-episode psychosis. Marc Gunderson, M.D., presents the case of erotomania in a middle-aged patient with a history of schizoaffective disorder and polysubstance use. Lastly, Siddarth Puri, M.D., offers a review of the book *Clinical Manual of Cultural Psychiatry*.

ARTICLE

Transcranial Direct Current Stimulation: Theory, Treatment of Major Depressive Disorder, and Other Neuropsychiatric Applications

Shan H. Siddiqi, M.D., Nicholas T. Trapp, M.D.

The use of noninvasive brain stimulation for the treatment of various neuropsychiatric disorders, including major depressive disorder, has rapidly expanded recently. Transcranial direct current stimulation (tDCS), variants of which have been used experimentally for psychiatric (1–5), neurologic (6, 7), and physical rehabilitation (8, 9) applications, has garnered a great deal of attention. While it is not yet approved by the Food and Drug Administration (FDA) for any indication, its promise is related to its low cost and wide range of applications. Although the breadth of its applicability has been questioned due to heterogeneous data (10), this heterogeneity has been attributed to methodological variability (11).

The safety and tolerability of tDCS were outlined by an early study including 567 sessions in 102 patients. The most common adverse effects were mild tingling/itching at the stimulation site and moderate fatigue. Less frequent effects included headaches (11.8%), nausea (2.9%), and insomnia (0.98%), all of which were mild and transient (2).

The underlying theory is that tDCS modulates the excitability of certain cortical regions (13–15) by passage of a small electrical current through conducting pads applied to the scalp in a minimally painful manner. While the precise mechanism is not fully understood, it likely enhances cortical excitability at the anode and depresses it at the cathode (13, 14, 16).

Proposed mechanisms have been based on data demonstrating relationships between tDCS stimulation and neuropharmacologic effects, cortical electrophysiology, and functional neuroimaging changes. Effects of tDCS on neuroplasticity and cortical excitability have been shown to be differentially modulated by agents affecting neurotransmission via serotonin (citalopram), dopamine (L-dopa), *N*-methyl-D-aspartate (dextromethorphan and d-cycloserine), and GABA (lorazepam). Electrophysiologic changes include differential modulation in the presence of agents that modulate sodium channels (carbamazepine) and calcium channels (flunarizine) (15). Active tDCS shows significant increases in prefrontal cortex activity as measured by functional near-infrared spectroscopy, a technique used to measure cortical oxygenation, during and after stimulation. Notably, functional near-infrared spectroscopy measurements may be limited by interference due to extracranial blood flow and inability to assess deeper structures, so they merely approximate the functional MRI (fMRI) signal in superficial structures (13). Stimulation also increases fMRI activation and connectivity of the underlying cortical regions and hippocampi, although the clinical significance of this is uncertain given that the same study found no behavioral changes (14).

USE FOR COGNITION AND MAJOR DEPRESSION

Since the FDA approval of repetitive transcranial magnetic stimulation of the dorsolateral prefrontal cortex for treatment of major depressive disorder, there has been extensive research regarding noninvasive stimulation of the dorsolateral prefrontal cortex. This has led to some literature showing various types of tDCS to have efficacy as standalone treatment for major depressive disorder (1, 2), as an adjunct to antidepressants (5), and as a means to enhance psychotherapy response via its cognitive effects (3). Some conflicting results have called these findings into question, suggesting that further research is needed to establish the most appropriate methodology (16).

The most widely studied use for tDCS has focused on stimulation of the dominant dorsolateral prefrontal cortex for cognitive enhancement. A recent systematic review of 61 studies found variability regarding tDCS effects on attention, executive function, working memory, and learning—some of which showed improvement, while others did not—and attributed this inconsistency to varying experimental methods (16). The most pronounced effect has been for attention and stimulatory effects; anodal tDCS of the dorsolateral prefrontal cortex was found to be more effective than 200 mg of caffeine for improving performance on sustained attention tasks for several hours after stimulation and was comparable to caffeine after 8 hours of sleep deprivation. Furthermore, the effects of the two were comparable when measuring performance on tasks of reaction time and short-term memory in sleep-deprived patients. Subjectively, tDCS outperformed both caffeine and sham in self-ratings of mood, energy, drowsiness, fatigue, and sustained vigilance (4).

An early study on the use of tDCS monotherapy for major depressive disorder was conducted by Boggio et al. (1), who demonstrated significantly better improvement in depressive symptoms

immediately following stimulation of the dominant dorsolateral prefrontal cortex when compared with occipital cortex stimulation and sham. Loo et al. (2) subsequently demonstrated significant improvement in mood after active tDCS compared with sham in patients already taking antidepressants, but there was no difference in overall response rates between groups after 3 weeks.

Comparison with selective serotonin reuptake inhibitors was first attempted recently in a 2x2 factorial study comparing tDCS/sertraline, tDCS/placebo, sham/sertraline, and sham/placebo over 6 weeks. The combined treatment was superior to all other groups, while the difference between tDCS and sertraline was nonsignificant. The difference between sertraline and placebo did not reach statistical significance, although tDCS alone was superior to placebo. Notably, patients correctly guessed which treatment they received, compromising the integrity of the blind. There was no difference between sertraline and tDCS in this regard, leading the authors to suspect that correct guesses were driven by clinical improvement (5).

Based on the potential efficacy of dorsolateral prefrontal cortex stimulation for both attention/cognition and depression, it has been speculated that this electrode placement would be a useful adjuvant to cognitive psychotherapy, which is otherwise limited by the concentration impairments inherent to major depressive disorder. Brunoni et al. (3) investigated this in a study of tDCS in cognitive control therapy, a form of self-directed cognitive therapy for depression. Interestingly, this study found that patients over age 50 had significantly better response to cognitive control therapy when stimulated with tDCS, while improvement in younger patients did not reach statistical significance. The authors' speculative explanation for this finding is that tDCS-induced neuroplasticity may mitigate the effects of subclinical prefrontal atrophy in older patients. Conversely, concurrent cognitive control therapy was also independently shown to enhance response to tDCS (17).

KEY POINTS/CLINICAL PEARLS

- Transcranial direct current electrical stimulation (tDCS) of the dorsolateral prefrontal cortex has been found to be beneficial as a standalone treatment and as an adjunct treatment for major depressive disorder.
- Cognitive enhancing effects of tDCS have been difficult to predict, but this is limited by methodological variability.
- Compared to other neuromodulatory treatments, tDCS is safe, well-tolerated, and inexpensive; however, it is likely somewhat less effective.
- Cognitive effects have been useful for sustaining attention/vigilance and improving cognition in Alzheimer's disease.

Notably, results of studies evaluating the cognitive effects of tDCS are heterogeneous (10, 16). This heterogeneity is associated not only with variable experimental methods, but also with intersubject variability in size, shape, and fat tissue content of the patient's scalp. A recent meta-analysis by Horvath et al. (10) challenged the idea that tDCS has any reliable cognitive effects, although a subsequent critique of this review demonstrated that it failed to account for significant differences in effects with different treatment parameters (for example, the pooled data sets included studies with both short treatment durations and long treatment durations, which are known to produce conflicting results due to calcium overflow mechanisms). Furthermore, the review was found to contain several errors, incorrectly/incompletely cited data, and other conceptual flaws in the data pooling methods (11).

As a result of these findings, multisite trials are in progress investigating larger sample sizes (ClinicalTrials.gov identifiers: NCT01562184, NCT01644747, and NCT01346306).

GENERAL NEUROPSYCHIATRIC APPLICATIONS

tDCS has also been implicated as a potential treatment for several other neuropsychiatric disorders. Early studies suggested efficacy of tDCS as an adjunct in stroke rehabilitation (6) and fibromyalgia (8, 9), while more recent studies have suggested possible uses in Alzheimer's dementia (18).

The most prominent evidence in stroke rehabilitation has been found in post-stroke aphasia; anodal excitation of the damaged Broca's area and cathodal inhibition of the contralesional area led to faster overall recovery and improved speech fluency. Furthermore, excitation of the contralesional area may accelerate cortical relocation to the nondominant hemisphere in patients with large lesions (6). While a recent Cochrane review found that changes in poststroke aphasia did not reach statistical significance, its sample size was limited by the fact that it only included studies that reported picture naming as an outcome measure rather than speech fluency and recovery speed (19).

Motor cortex stimulation has also been used for fibromyalgia. Fregni et al. (8) demonstrated at least 21 days of improvement in pain scores for fibromyalgia patients with 5 days of anodal excitation over the primary motor cortex when compared with left dorsolateral prefrontal cortex and sham stimulation. Several other studies have replicated this phenomenon (9). A recent Cochrane review did not find a significant effect for chronic pain syndromes overall, but the review did not make a distinction between fibromyalgia and other causes of chronic pain in its pooled analysis (20).

Promising findings for improving attention, memory, and executive function have led to recent research aimed at evaluating dorsolateral prefrontal cortex stimulation for alleviation of cognitive deficits seen in Alzheimer's dementia. A recent randomized controlled double-blind trial in patients taking memantine found that a series of 10 daily sessions led to statistically significant improvements in Mini-Mental Status

Examination scores, digit span, and performance IQ; these effects were noted immediately and sustained for at least 2 months after treatment (7). While this study was limited by a small sample size, a subsequent meta-analysis found that tDCS-induced cognitive gains in patients with Alzheimer's dementia were significantly better than in age-matched healthy control subjects (9).

CONCLUSIONS

Transcranial direct current stimulation has demonstrated some promise as a treatment for various disorders, including major depressive disorder. However, important questions remain unanswered, including precise mechanisms of action, ideal stimulation parameters for different disorders, optimal dosing schedules, and long-term safety (16). Although further research must answer some of these questions before its clinical utility becomes clear, tDCS appears to be a relatively safe, flexible, durable, inexpensive, and convenient method of treatment that holds potential for treatment of disorders affecting various parts of the cortex.

Drs. Siddiqi and Trapp are third-year residents in the Department of Psychiatry, Washington University School of Medicine, St. Louis.

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ARTICLE

Painkillers That Cause Pain: Review of a Rising, Poorly Recognized Complication

Cornel N. Stanciu, M.D., Oliver M. Glass, M.D.

Chronic, nonmalignant pain is quietly rising in the United States, affecting hundreds of millions and causing significant functional loss and disability, reduced quality of life, increased health care costs, and premature deaths (1). The various types of pain, with exact pathophysiology poorly delineated, continue to pose management challenges. Opioids, despite lack of support for long-term effectiveness in managing chronic no-cancer pain, are often prescribed for extended periods. This is reflected by the 10-fold increase in prescriptions over the past two decades (2, 3). Dosages per prescription are also rising. To emphasize the magnitude, the United States, with 5% of the world's population, accounts for 84% of global oxycodone and 99% of global hydrocodone consumption (3). The increasing prevalence of opioids places a greater number of patients at risk for adverse reactions. With long-term continuous use, tolerance can develop, leading to increased dosing followed by eventual loss of analgesic effect. Accumulating evidence indicates chronic opioid exposure aimed at alleviating pain could also paradoxically render some patients more sensitive to pain or aggravate pre-existing pain, a complication termed opioid-induced hyperalgesia (OIH) (3). In such cases, patients are more sensitive to nociceptive stimuli with increasing doses, and this type of pain may be the same as or different from the underlying pain. This is difficult for most physicians to recognize in the clinical setting. Reported worsening pain sensation is often misattributed to underdosing, leading to uptitration.

OIH is an under-recognized condition of unknown prevalence but with a rapidly expanding body of evidence. The purpose of the present article is to alert clinicians to this phenomenon, review recently published literature on the topic, highlight some distinguishing features in comparison to tolerance, examine underlying biological mechanisms, and discuss key points in management.

METHOD

We searched PubMed and Medline (PubMed/Medline, PsychINFO, PsychARTICLES, CINAHL, EMBASE, Scopus, Academic OneFile) for all literature concerning OIH from 1995 to 2015 using the title search terms "opioid induced hyperalgesia," "allodynia," and "opioid tolerance." Search results were supplemented by references gained from recent reviews and citations of searched returns. Based on pre-established criteria, only peer-reviewed original papers in English were considered. Emphasis was placed on human literature. Because our primary focus was discussion of hyperalgesia associated with exogenous opioids, we did not include any study reporting opioid-mediated inhibition of pain sensation or hyperalgesic effects or hyperalgesia as consequence of abstinence.

OPIOID TOLERANCE AND DIFFERENTIATION FROM OIH

Escalation of opioid dosing without significant benefit may signal tolerance, defined as loss of drug potency through desensitization of inhibitory nociceptive pathways to opioids following repeated or prolonged administration (4, 5). It has been categorized as innate (genetically determined, usually noted during the first opioid administration) and acquired, which is further subdivided into pharmacokinetic (changes in distribution, metabolism reducing drug availability), pharmacodynamic (changes in receptor density or sensitivity altering response), and learned (environmental cues consistently paired with drug decreasing therapeutic effect) (6). Patients may also experience hyperalgesia, a state of increased pain sensitivity involving sensitization of activating nociceptive pathways (7, 8).

Both hyperalgesia and tolerance result in increasing dose requirements (9). Because the same molecular processes govern both, they are likely to coexist, clinically clouding the picture. Since tolerance is faced frequently, clinicians often fail to recognize OIH by classifying all cases of poor response to escalating opioids as tolerance (4). Tolerance should not limit escalating opioid therapy in the setting of chronic pain (5), but at the same time pain worsening during the course of therapy should not automatically be regarded as tolerance unless a diligent clinical evaluation fails to explain the etiology of the pain (4, 7). Differential diagnoses should include OIH but also disease progression, physical dependence or withdrawal, addiction, and abuse (Table 1) (10, 11).

One unique feature of OIH is that the novel pain becomes more widespread, often anatomically distinct from the original pain complaint prompting treatment, and this may differ in quality. Allodynia, a pain response to innocuous stimuli, often accompanies (8). OIH can occur with any route of administration, more frequently with intermittent boluses rather than continuous infusion (4, 7). Despite more evidence of occurrence with long-term high-dose opioids (8), there are reports on opioid-naive patients receiving low-dose short

TABLE 1. Differential Diagnosis of Worsening Pain in Patients Taking Opioids

Consideration	Pain Onset	Clinical Features	Pain Response to Opioids
Opioid-induced hyperalgesia	Abrupt or gradual	Paradoxical increase in pain that is more widespread and often anatomically distinct; may even differ in quality, and is sometimes accompanied by allodynia.	Worsens
Opioid tolerance	Gradual	Persistent, localized pain no longer relieved by current regimen.	Improves
Dissease progression	Abrupt or gradual	Worsening pain; may extend into adjacent anatomical sites.	
Opioid addiction	Gradual	Pain may or may not be present. Characteristic behavior includes impaired control and compulsive use of opiates, despite harm, and cravings.	May improve
Pseudo-addiction	Variable	Pain at original site that is undertreated. Due to patients seeking pain relief, addiction is wrongfully suspected.	Improves
Opioid withdrawal	Abrupt	Autonomic hyperarousal (tachycardia, hypertension, sweating) accompanied by flu-like symptoms and gastrointestinal distress. Pain sensitivity increases, with distribution likely extending as well.	
Physical dependence	Gradual	Pain at original site characterized by a state of adaptation, with chronic opioid use resulting in tolerance and even physical withdrawal symptomatology when undergoing dosage reduction or abrupt discontinuation.	

courses during or after surgeries (4, 7, 10, 11). The degree of tolerance is related to either duration of opioid exposure, total dosage of opioid required, kinetic properties of the receptor, or a combination of these. Here, increased pain can be overcome by increasing the dose, providing a way to differentiate clinically (5). If opioid reduction improves pain control, then OIH is more likely, whereas if pain worsens then tolerance is more likely (4, 5, 7). Quantitative sensory testing, a way of assessing the intensity of stimuli required to elicit specific sensory perceptions, has been used to predict OIH (12). Through a computer testing system, small nerve endings are assessed by measuring response to cold temperatures and large nerve endings by measuring response to vibration, and results compare the patient's unaffected side with a series of "normal" patients. It is performed before opioid initiation and repeated during treatment course, and any change in pain threshold can suggest OIH. The complexity and time consumption limits its use.

UNDERLYING BIOLOGICAL MECHANISMS AND PATHOPHYSIOLOGY

Opioids act by inhibiting spinal cord neuronal transduction and ascending pain signals at the midbrain nuclei, as well as through modulation of limbic system pain perception (5). The mechanism behind tolerance is believed to include desensitization, or internalization, of opioid receptors decreasing opioid binding sites available for pain relief. Sensitization of *N*-methyl-D-aspartate (NMDA) receptors may also play a role, as antagonists such as ketamine attenuate tolerance development and decrease dosage-delaying tolerance development (14). The exact underlying neurobiology of OIH is not completely understood (4, 8). Current literature supports involvement of excitatory glutamanergic systems with pathological activation of NMDA system-enhancing glutamate in the spinal cord (4, 11). Several other postulated mechanisms exist, such as sensitization of peripheral nociceptors, decreased reuptake of excitatory neurotransmitters, increase in spinal dynorphins, genetic mechanisms, and enhanced descending facilitation of nociceptive pathways from rostral ventromedial medulla due to neuroplastic changes (4, 7). Prostaglandins may also contribute by stimulate glutamate release in the spinal cord (8).

Tolerance and OIH can be regarded as lying on a spectrum, with the underlying mechanism linking them being mediated through the NMDA receptor; increase in responsiveness of NMDA receptor contributes to development of tolerance and hyperalgesia (13). Presynaptic NMDA receptors on primary afferent fibers may promote neurotransmitter output with opioid receptors inhibiting the same (14). Stimulating mu receptors increase NMDA-mediated glutamate response, resulting in sensitization of spinal column dorsal horns to noxious stimuli (15). One study showed that administration of the NMDA antagonist ketamine perioperatively for chronic back pain patients undergoing spine surgery was more effective (lower post-operative pain scores and opioid requirement) in those on high opioid doses prior to surgery and thus more tolerant (16).

MANAGEMENT

Escalation of opioid dose will eventually lead to tolerance. Addition of adjuvant nonsteroidal anti-inflammatory drugs, tricyclic antidepressants, and gabapentin has been shown to reduce opioid requirement and delay tolerance development (Table 2) (5). Ketamine, aside from antagonizing NMDA, has analgesic properties at low doses (5, 17).

Management of OIH requires individualized strategies (4). Proper approach involves reducing opioid dosages while replacing with nonopioid analgesics (18). Low-dose naloxone has been shown to aid in this process. Opioid

TABLE 2. Adjuvant Treatment Options for Opioid-Induced Hyperalgesia to Assist With Opioid Reduction

Agent	Mechanism of Action	Cautions
Nonsteroidal anti-inflammatory drugs	Blunt inflammatory response by inhibition of prostaglandin synthesis by inhibition of cyclooxygenase isozymes.	May have gastrointestinal side effects.
Tricyclic antidepressants	Neurotransmitter (especially serotonin and norepinephrine) reuptake inhibitor.	May cause orthostatic hypotension and anticholinergic side effects, especially in elderly patients.
Gabapentin, pregabalin	GABA analogues, interact with descending noradrenergic and serotonergic pathways originating from the brainstem to reduce pain transmission from the spinal cord.	May cause CNS depression and potentiate other sedatives and alcohol when used concomitantly.
Ketamine, dextromethorphan, amantadine	N-methyl-D-aspartate (NMDA) receptor antagonism preventing glutamate release in the spinal cord.	Ketamine may cause respiratory depression, and long-term use poses risk of addiction.
Methadone and buprenorphine	Opioid switching with some NMDA antagonism; buprenorphine preferred due to partial mu agonism and delta, kappa antagonism.	Incomplete cross-tolerance may be seen with methadone. Respiratory depression and sedation also of consideration.
Clonidine	Alpha-2 receptor agonist.	May cause orthostatic hypotension.

switching, where another opioid may be introduced while tapering the original, may abolish symptoms (8, 11). Opioid reduction might induce withdrawal symptoms, including an increase in pain (4). The long course required for weaning, and for results to be noted, may not be an option for some. In such cases, evidence suggests that NMDA receptor blockers, such as low-dose ketamine (5, 19) and, less often, dextromethorphan (5, 20), may be used to modulate OIH (4, 7, 11, 17). Amantadine, also an NMDA antagonist, may mitigate central sensitization (14). Both methadone and buprenorphine have NMDA antagonist actions and may be considered, although the latter is preferred due to partial mu agonism and delta, kappa antagonism (4, 11). Other approaches include adding cyclooxygenase inhibitors (nonsteroidal anti-inflammatory drugs) such as ketorolac or parecoxib sodium as adjuvant treatment and providing opioid sparing effects, in addition to modulating sensitized receptors by blocking prostaglandins (9, 11). Gabapentin, pregabalin, and alpha-2 agonists such as clonidine can also attenuate the symptoms (4, 7, 11).

The biggest challenge in managing such patients is psychological: developing a consensus between providers, patients', and families to proceed with reduction of pure opioid agonists. With the opioids likely affecting the patient's mood, as well as physiological dependence playing a role, attempts to implement a rational plan might be challenging.

CONCLUSIONS

Although opiates are widely used for treatment of chronic, noncancer pain, they are not free of adverse effects. OIH may contribute to patient discomfort and carry harmful consequences if not recognized and addressed. As part of an informed consent, it seems reasonable to discuss OIH with patients prior to instituting opioid treatment. It is common to misdiagnose OIH as tolerance. Clinicians should suspect OIH in pain patients on chronic opioids when analgesic effects wane despite lack of disease progression. This is particularly seen in the context of unexplained pain reports or diffuse allodynia unassociated with original pain and increasing pain levels with increasing doses. Current guidelines support reducing opioid doses, tapering with aid of adjuvant analgesics, or supplementation with NMDA modulators. The potential short-term increase in pain while opioids are reduced is a tradeoff to a longer-term reduction in suffering and improved overall quality of life.

KEY POINTS/CLINICAL PEARLS

- Despite lack of evidence when used long-term for managing chronic noncancer pain, opioids are being prescribed more frequently and with higher dosing.
- Chronic opioid exposure aimed at alleviating pain can paradoxically render some individuals more sensitive to nociceptive stimuli, aggravate preunderlying pain, and even induce a new pain, a complication termed opioid-induced hyperalgesia (OIH).
- Clinically, OIH and tolerance both present with worsening pain despite dosage escalation. In the context of unexplained pain reports or emergence of diffuse allodynia unassociated with original pain, OIH should be suspected.
- Management of OIH involves reduction in the opioid dose, tapering with the aid of an adjuvant analgesic, or supplementation with *N*-methyl-D-aspartate modulators.

Drs. Stanciu and Glass are third-year residents in the Department of Psychiatry, East Carolina University, Brody School of Medicine, Greenville, N.C.

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ARTICLE

Prevention of Dementia

Aarti U. Jerath, M.D., M.A.

Dementia, a form of cognitive impairment, with the most frequent cause as Alzheimer's disease, is a major health concern in the midst of an aging population in the world. Furthermore, the prevalence of cognitive impairment increases with age (1). Although the DSM-5 uses the term neurocognitive disorder, "dementia" will be used in the present article because it was used in all the research for the article.

Based on estimates from the Aging, Demographics, and Memory Study in 2007, 13.9% of people age 71 and older in the United States have dementia (2). Dementia affects 6.4% of Europeans 65 years and older (3); in particular, dementia affects 16.7% of the Chinese population over age 85 (4) and 43% of the German population over age 85 (5).

Being able to identify ways to prevent dementia are important, especially to help improve activities of daily living in an aging population and because there is no curative treatment for dementia. The purpose of this article is to outline modifiable, preventable, and treatable factors that are not typically thought about and that can help delay the onset of cognitive decline. These unique factors can be subdivided into the following categories: substance use, metabolic syndrome, an active lifestyle, and depression.

Epidemiological research has shown that modifying one's use of substances, modifying factors contributing to metabolic syndrome, keeping one's mind active, and treating depression can help delay the onset of, or possibly prevent, dementia.

MODIFIABLE FACTORS

Substance Use

Epidemiological studies show that the relationship between alcohol use and cognitive decline follows a J-shaped curve (i.e., the risk for dementia is higher in those who are abstinent compared with those with low-moderate alcohol consumption and further increases in those who consume alcohol excessively). Davis et al. (6) showed that there are factors related to low-moderate alcohol consumption that contribute to maintaining better global cognitive function. However, at high levels of alcohol consumption, there might be factors related to reduced brain volume.

Other studies have shown that reduction in smoking increases the number of years lived without cognitive impairment (7) and that smoking is significantly associated with smaller hippocampal (8).

Metabolic Syndrome

Epidemiological studies have shown that obesity, hypertension, high cholesterol, poor diet, diabetes mellitus, and atherosclerosis may be modifiable factors contributing to dementia. Furthermore, those factors leading to metabolic syndrome might contribute to increasing the risk of atherosclerosis, which results in less blood flow to the hippocampus. In one study, high diastolic blood pressure and cholesterol were associated with low hippocampal volume (8).

Atherosclerosis, in particular in the extracranial carotid arteries, is related to higher risk of dementia and cognitive decline (9). Other factors, including high levels of homocysteine, have shown to have a direct adverse effect on the hippocampus (10). Nutritious diets, such as the DASH [Dietary Approaches to Stop Hypertension] diet and the Mediterranean diet, have been shown to contribute to slowing cognitive decline (11), but results across studies have been inconsistent (12). In addition, results have also been inconsistent among studies related to Vitamin D. One study demonstrated that low-serum 25-hydroxy vitamin D was associated with greater cognitive decline and dementia (13); other randomized controlled trials have shown that Vitamin D supplementation did not show an effect on improving cognitive ability or preventing dementia (14, 15) Sobów et al. reported that nutritional and behavioral assessment can help evaluate the risk of dementia (16).

Diabetes has inconsistent results. One study found that diabetes affects glucose metabolism and insulin signaling in the brain, which in turn results in Aβ/tau-dependent pathological changes and subsequent modifying of cognitive function, ultimately leading to dementia; this evidence suggests that controlling diabetes can help reduce metabolic changes that will eventually impair cognitive function (17). Two randomized controlled trials that examined the effect of adequate diabetes control on cognitive function, however, revealed that controlling diabetes does not prevent cognitive decline (18, 19).

The Tasmanian Healthy Brain Project is under way as the world's first prospective study to determine the capacity of university-level education to enhance cognitive reserve and to help reduce cognitive decline. Although the study began in 2011 and is expected to continue for 10–20 years, its hypothesis is that education and mental activity help prevent cognitive decline (20). A recent meta-analysis has shown that lower education attainment was a strong predictor of Alzheimer's dementia (21).

Active Lifestyle

An active lifestyle has also been shown to help prevent dementia in epidemiological studies. In addition to reduced smoking, reduction of obesity and an active lifestyle increase years lived with-

TABLE 1. Modifiable Risk Factors for Dementia Per Epidemiological Studies

Factor
Alcohol use
Tobacco use
Metabolic syndrome
Diet
Diabetes mellitus
Lifestyle–activity level
Depressed mood

out cognitive impairment (7). Therefore, being physically, as well as socially, active may be important in preventing memory loss in the long run.

Another meta-analysis showed that decreased physical activity was a strong predictor of Alzheimer's dementia (21). Ongoing trials such as the AIBL Active trial, FABS II [Fitness for the Ageing Brain Study II], and PROMoTE [Promotion of the mind through exercise] are examining the efficacy of physical activity on prevention of dementia in a randomized-controlled clinical trial design.

Depression

Among rural memory clinic patients with major cognitive impairment or dementia, there was a high prevalence and severity of depression warranting more studies (22). Those with greater education and reading ability showed a greater decrease in memory and in executive and language performances as depressive symptoms increased than those with lower years of education and reading ability (23), thus making depression and dementia more complicated to research.

On the other hand, a recent study reported that compared with nondepressed participants with mild cognitive impairment (defined as a Clinical Dementia Rating scale score of 0.5), depressed participants with mild cognitive impairment showed reduced cortical thickness in the anterior medial temporal lobe and gyrus adjacent to the amygdala bilaterally and greater white matter hyperintensity volume as a percentage of the total intracranial volume. This was associated with cortical thinning in the frontal, temporal, and parietal regions in patients with mild cognitive impairment in the study (24). Whether treatment of depression will prevent the onset of dementia remains an open question.

CONCLUSIONS

The literature findings on the prevalence and causes of dementia discussed in this article could help guide future research on identified modifiable factors—such as treating substance use, modifying metabolic syndrome, treating depression, and keeping an active mind—that can help in slowing and preventing dementia, which in turn might help in the discovery of other novel, modifiable factors. Furthermore, it is important that clinicians become familiar with the latest research and educate patients about the preventable causes of dementia, as well as encourage patients to read and inquire about the topic.

Dr. Jerath is a third-year resident in the Department of Psychiatry, University of Miami Hospital, Miami.

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KEY POINTS/CLINICAL PEARLS

- Epidemiological studies suggest that reducing substance use, dietary modification, control of diabetes and metabolic syndrome, treatment of depression, and maintaining an active lifestyle may help reduce the prevalence of dementia.
- Randomized controlled studies examining these factors have been few in number and have yielded inconclusive results.

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CASE REPORT

Delusional Memory in First-Episode Psychosis

Awais Aftab, M.D.

The present case report discusses the psychopathology of psychosis with a focus on delusional memory. This case also demonstrates how an understanding of phenomenology can enrich psychiatrists' diagnostic appreciation.

CASE

"Mr. A" is a 19-year-old Caucasian man with a past psychiatric history of attention deficit hyperactivity disorder. He was referred by his outpatient psychiatrist to the hospital with concern for first-episode psychosis. He claimed that he had been held at gunpoint and that his girlfriend had been raped in an incident at his college dormitory, an event confirmed by police not to have occurred. Additionally, he was not in a relationship with the girl in question, and she was someone he had been infatuated with since childhood. He claimed that he had "repressed" these memories earlier, but they came back to him a few days prior. He maintained that the girl had not verified the story because her memories were still repressed. When asked why the police were unable to find any evidence and why no one else had come forth, the patient could not offer any explanation and vaguely speculated that it may be a cover-up in which other residents of the dorm were also involved.

He reported recalling other previously repressed memories as well, in particular memories of having sex with his girlfriend when they were both 3–4 years of age. He stated that he and his girlfriend have had this complicated entangled love relationship ever since. He also described hearing voices of his girlfriend and other girls, talking among themselves about him, and of his thoughts being communicated to others.

At the time of admission, the patient also reported experiencing intense déjà vu and reported feeling as though this hospitalization had already happened before in the past. At some moments, it appeared as though he actually believed that he had this hospitalization before, but at most other moments he described it as a feeling rather than a firm belief. No prior history of experiencing déjà vu was obtained. There was no history of seizure disorder, and no history of loss of consciousness or seizure-like movements was elicited.

Per the patient's family and outpatient psychiatrist, the patient's psychotic symptoms were first noticed 4–5 weeks prior, with no past history of observed psychotic behavior. Of note, his father had been diagnosed with schizophrenia and had been on clozapine treatment at the time of his death (due to pulmonary embolism) when the patient was just 5 years old.

The patient had no prior history of substance abuse. His urine toxicology was negative except for amphetamines, which was consistent with him being on prescription psychostimulants. Stimulant abuse and substance-induced psychotic disorder were of low probability given collateral from the family and outpatient psychiatrist. A provisional diagnosis of schizophreniform disorder was made. A CT head scan was conducted and was unrevelatory, with no intracranial abnormality noted. EEG was considered but deferred to the outpatient setting.

DISCUSSION

In descriptive psychopathology, a distinction is often made between primary and secondary delusions. Primary delusions are "primary" in the sense that they do not occur in response to another psychopathological phenomenon, such as a mood disorder or hallucinations. Secondary delusions occur in response to another psychopathology, and their emergence is understandable given the patient's internal and external environment. Secondary delusions can also arise from pre-existing primary delusions. Primary delusions, in contrast, are "ultimately un-understandable" and psychologically irreducible (1–2).

Primary delusions are more common in new-onset and acute schizophrenia and are less common in chronic schizophrenia, in which they are obscured by a morass of secondary delusions, thought disorganization, and other psychotic phenomena (2).

A normal idea or belief can emerge in four different ways: spontaneously (as an intuition), from perception, from memory, and from mood. Analogously, a primary delusion can emerge in these four aforementioned ways, leading to four types of primary delusions: autochthonous idea (delusional intuition), delusional perception, delusional memory, and delusional mood/atmosphere (3).

Autochthonous idea (delusional intuition, also called "sudden delusional idea") is a delusion that arises spontaneously, fully-formed, "out of the blue," without premeditation, and without external cause. Delusional perception is when a person attributes a delusional (usually bizarre) meaning to a normal percept. It is included in Schneider's first-rank symptoms.

There are two senses in which the term delusional memory is employed: 1) as delusional interpretations of real memories and 2) as delusions that are experienced as memories (i.e., false memories believed to be real and held with the conviction of delusions).

Schneider considered delusional interpretations of real memories as the memory analogue of delusional perception and therefore a first-rank symptom, since the difference between delusional memory and delusional perception is merely temporal (3). He used the term

"mnestic delusional perception" to describe it (4). False memories in the second sense can be described as delusions that are "retrojected in time" and are "retrospective delusions." Oyebode (1) classifies these as primary delusions, although this is not necessarily the case because any delusion (whether primary or secondary) can be retrojected and experienced as a memory (3). When it is a primary delusion, it is the memory analogue of autochthonous idea, arising spontaneously and fully-formed (4). In many cases, it is often difficult to distinguish between the two senses of delusional memory, since it can be difficult to determine how much of the reported event is factual. Delusional memories are usually intensely preoccupying and often bizarre, and there is some evidence that the perceptual characteristics of patients' delusional memories are stronger than those of their real memories (5).

In the patient in the above case, we can determine that his memories of being held at gunpoint and his girlfriend being raped are false delusional memories. Because they arose spontaneously and fully-formed, they are primary delusions and memory analogues of autochthonous idea. Similarly, his memories of having sex with his girlfriend are delusions experienced as memories.

The patient's belief that the girl he referred to was indeed his girlfriend was held consistently. This is likely a secondary delusion, understandable in the context of his life-long infatuation. Déjà vu by itself is a fairly common phenomenon in healthy individuals. However, it has been suggested that there is a continuum of déjà vu, ranging from brief and fleeting in healthy individuals to chronic and prolonged in those with schizophrenia (6). Déjà vu is particularly associated with temporal lobe epilepsy (6), and in the absence of an EEG on the above patient, the possibility of simple partial seizures remains, which may present solely with psychiatric symptoms.

Research on false memories in schizophrenia has yielded significant insight using the Deese-Roediger-McDermott paradigm. In this paradigm, subjects are presented with a list of related words, and they are required to remember as many words from the list as possible. Each list converges on a related word that is not itself part of the list, called the critical lure. Typically, a high percentage of subjects recall the lure as being on the list, representing a false memory. Studies utilizing the Deese-Roediger-McDermott paradigm have shown that schizophrenia patients are overconfident in errors and under-confident in correct responses (decreased confidence gap) (7–8). This leads to increased percentage of inaccurate but confidently held memories (increased knowledge corruption index) (7–8). Furthermore, schizophrenia patients experiencing delusions recall twice as many false-positive memories compared with patients not experiencing delusions and control subjects (9). This has been thought to be associated with a jumping-to-conclusions or liberal acceptance bias in schizophrenia spectrum disorders (10).

KEY POINTS/CLINICAL PEARLS

- Primary delusions, by definition, do not occur in response to another psychopathological phenomenon such as a mood disorder or hallucinations; they are psychologically irreducible. Primary delusions are more common in new-onset and acute schizophrenia and are less common in chronic schizophrenia.
- Four types of primary delusions have been described: autochthonous idea (delusional intuition), delusional perception, delusional memory, and delusional mood/atmosphere.
- Delusional memory can be a delusional interpretation of real memory or a delusion that is experienced as a memory. Delusional interpretation of real memory is the memory analogue of delusional perception and therefore a Schneiderian first-rank symptom.
- Studies utilizing the Deese-Roediger-McDermott paradigm have shown that in individuals with schizophrenia, especially in those with active psychosis, there is an increased percentage of inaccurate but confidently held memories (increased knowledge corruption index).

CONCLUSIONS

Primary delusions and delusional memories are common psychopathological features of acute schizophrenia. Understanding the phenomenology of psychosis can enrich a psychiatrist's diagnostic appreciation. Studies utilizing the Deese-Roediger-McDermott paradigm reveal a decreased confidence gap and increased knowledge corruption index in patients with schizophrenia, suggesting a metacognitive liberal acceptance bias.

Dr. Aftab is a second-year resident in the Department of Psychiatry, University Hospitals Case Medical Center/Case Western Reserve University, Cleveland.

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CASE REPORT

She Loves Me Not? A Case of Erotomania

Marc Gunderson, M.D.

Erotomania is a delusional belief that another person, usually of higher status, is in love with an individual (1). The present case report is of a middle-aged patient with such a delusion, as well as a history of schizoaffective disorder and polysubstance use.

CASE

"Mr. P" is a 44-year-old single, unemployed, Caucasian man with a prior history of schizoaffective disorder, bipolar type, and polysubstance use (alcohol, cocaine, cannabis, methamphetamine, and LSD) who presented in an acute manic episode. At admission, the patient displayed pressured speech, irritable mood, formal thought disorder, paranoid and grandiose delusions, suicidal ideation, and auditory hallucinations. In addition, he was religiously preoccupied. He reported that he had not taken medications for about 6 months.

The patient's history was significant for two prior suicide attempts with significant prescription overdoses. Family history was noteworthy for a completed suicide by the patient's mother.

Collateral information was obtained from the patient's aunt, with whom he had been living. She revealed that he had recently become infatuated with a famous musician and had spent considerable resources travelling to meet her at various concerts. More recently, the patient had given up his apartment and sold his belongings in preparation of marrying and then moving in with the musician. His aunt confirmed that this was very likely not a mutually amorous relationship.

Standard admission laboratory values were within normal limits. After a medication petition was obtained by court order, the patient was initially treated with divalproex sodium extended-release (1,000 mg nightly), olanzapine (10 mg twice daily), and clonazepam (1 mg twice daily). Because the patient's psychotic symptoms showed little response to olanzapine, oral haloperidol (5 mg twice daily) was substituted.

With continued pharmacotherapy, the patient's affective symptoms began to remit. He started to talk more openly about the musician, which further revealed how well systematized the delusion had become. He revealed how the musician had singled him out during a concert with subtle looks and gestures that signaled he was the object of her affection. When irregularities in the relationship were pointed out, such as he had never had a significant conversation with the woman yet they were to be married, he was quick with a rationalization or vague excuse.

There was considerable improvement in his affective lability and thought disorder with complete resolution of suicidal ideation. The paranoid and grandiose delusions were reduced and nonintrusive; however, there remained no reduction in the level of the amorous delusion. The patient eventually agreed to a haloperidol decanoate injection and was given 100 mg intramuscularly prior to discharge. Given the patient's affective stability and resolution of suicidal ideation, as well as the likely chronic nature of his delusions, he was discharged on a court-ordered commitment. Discharge medications included extended-release divalproex sodium (1,750 mg nightly) and haloperidol decanoate (150 mg intramuscularly each month).

DISCUSSION

Although descriptions of erotomania date back to Hippocrates, it was first made famous by the Frenchman de Clérambault, to whom the syndrome is sometimes still referred. Erotomania is a rare phenomenon. Indeed much of the literature has focused on whether it constitutes a distinct clinical entity, as de Clérambault suggested, or rather a symptom of other more recognizable disorders (2, 3). DSM-5 continues to categorize erotomania as a subtype of delusional disorder, a diagnosis first described in DSM-III-R. In an article published in 1975, Hollender and Callahan (4) described two major subtypes. In primary (pure) erotomania, the amorous delusion is the single presenting symptom, while in secondary (associated) erotomania the delusion is a symptom of a larger presenting condition (4). In a later article, Seeman (5) suggested classifying patients with erotomania in one of two categories. The first is a fixed group in which patients fixate on an object that tends to be more ordinary. In the second group, the delusions are often shorter in duration, more intense, focused on more powerful objects, and recurrent over time. In the Seeman study, subjects in the first group tended to be more psychiatrically ill and timid, while subjects in the second group tended to have better functioning and were more aggressive and impulsive. In a study published in 1985, Ellis and Mellsop (3) attempted to more specifically diagnose erotomania by suggesting nine operational criteria that need to be met (Table 1) (3).

More recently, Kennedy et al. (6) attempted to use the criteria proposed by Ellis and Mellsop, as well as Seeman's two categories, to classify a group of 15 erotomanic patients. In their study, they found the Ellis and Mellsop criteria to be fairly useful. Yet in roughly half the cases, the sudden onset, chronic course, or absent hallucinations were not met. After modifying the criteria slightly, Kennedy et al. noted that all of the sub-

TABLE 1. The Ellis and Mellsop Model for Diagnosing Erotomania[a]

Criteria
A delusional conviction of being in amorous communication with another person.
This person is of much higher rank.
The other person had been the first to fall in love.
The other person had been the first to make advances.
The onset is sudden.
The object of the amorous delusions remains unchanged.
The patient provides an explanation for the paradoxical behavior of the loved one.
The course is chronic.
Hallucinations are absent.

[a] For further details, see Ellis and Mellsop (3).

TABLE 2. The Kennedy et al. Model for Diagnosing Erotomania[a]

Criteria
A delusional conviction of being in amorous communication from on object of generally higher social class.
The object of erotomania should be the first to fall in love and/or make the first advances.
At least two of the following must be present:
• Sudden onset (<7 days)
• The object remains unchanged
• The subject rationalizes the paradoxical behavior of the object
• Chronic course of the delusion
• Hallucinations should be absent or, if present, of a tactile nature clearly related to erotomania

[a] For further details, see Kennedy et al. (6).

jects met full criteria. They found little validity in the descriptive categories suggested by Seeman.

It is interesting to consider the patient in the above case using these different proposed diagnostic systems. It is clear given the patient's history and current presentation that he would fit into the category of secondary erotomania. His current manic symptoms and his long history of paranoid delusions suggest that he is not suffering a single delusion, nor would it be most appropriate to diagnose him as having delusional disorder, erotomanic type. The patient clearly fulfills seven of the original criteria suggested in the model by Ellis and Mellsop. Using the modified criteria by Kennedy et al., the patient would then meet full criteria (Table 2). It is noteworthy that this patient lacked the same three criteria that were most commonly missed in the study participants, perhaps strengthening the argument that the original nine criteria are either too strict or not relevant in a significant number of these patients.

Perhaps more useful is to consider the relationship between erotomania and the DSM-5 psychotic and affective disorders. Much of the older literature on the topic considered erotomania as either a distinct delusional entity or as part of a larger psychotic state without giving much regard to its association with affective disorders (2–4). In a review of de Clérambault's original cases, Signer (7) noted that in both primary and secondary erotomania there were many indicators of mood disorder, predominately elated and grandiose states. Another study reported that 25% and 7% of participants had considerable affective features consistent with schizoaffective disorder and bipolar disorder, respectively (8). The study also noted that as a whole, the erotomanic group displayed more manic symptoms than the general delusional comparison group. In his review, Signer also discussed several studies that reported positive treatment results using mood stabilizers and neuroleptics, further suggesting a link between affective states and erotomania.

Grandiosity and hypersexuality are common manic symptoms that fit well within the context of an erotic delusion focused around an object of higher status. The above patient also presented with religious preoccupation. It was not clear whether this was a result of the delusional object having prominent religious ties or whether the delusion was focused on the object because of previous religious preoccupation. While it is fascinating to think about the underlying psychodynamics, it seems clear that in this patient's case there was a definite congruency between the delusional state and his affective state. Given the close relationship between erotomania and affective states, it seems prudent to recommend that if a patient presents with an erotomanic delusion the clinician should evaluate thoroughly for concurrent mood symptoms.

KEY POINTS/CLINICAL PEARLS

- True erotomania is a rare phenomenon.
- Although it is relatively straightforward to identify the delusion, the paucity of reported cases makes it difficult to identify whether erotomania is a distinct clinical entity or simply a symptom of another disorder.
- Erotomania appears to be associated with congruent affective states. Therefore, it is prudent to screen for these states when the delusion is encountered.

CONCLUSIONS

The debate regarding whether erotomania is a distinct clinical syndrome will likely continue, owing in large part to the rarity of cases with true erotomanic delusions. What is clear is that it is possible to diagnose the delusions with a fair degree of certainty. There also appears to be a significant relationship

between this delusional state and affective disorders, including schizoaffective and bipolar disorders. This association allows a framework in which to begin the diagnostic process when encountering future patients with this delusion. Further study will be required to truly understand the origins of this delusion from a psychodynamic and psychopathologic point of view.

Dr. Gunderson is a third-year resident in the Department of Psychiatry and Behavioral Medicine, Medical College of Wisconsin, Milwaukee.

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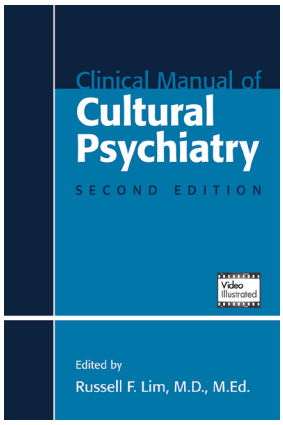
BOOK FORUM

Clinical Manual of Cultural Psychiatry, Second Edition

Reviewed by Siddarth Puri, M.D.

Considering the complex influence of culture adds another necessary layer to the delicate process of a psychiatric interview. Dr. Russell Lim's second edition of the Clinical Manual of Cultural Psychiatry highlights changes in the DSM-5 and establishes a new framework for understanding cultural identity for psychiatrists. This manual incorporates new chapters expanding upon the definition of cultural identity to include women's health, LGB issues, gender nonconforming and transgender patients, and religion and spirituality.

The first chapter describes the recent DSM-5 changes around cultural identity and expands upon the "outline for cultural formulation" (OCF), which now includes a "cultural formulation interview" (CFI). Written by early-career psychiatrists, this chapter explains how the DSM-5 sharpens the explanation of cultural impact on mental health while also providing examples of how to incorporate the CFI and its sample questions into everyday interviewing. Each subsequent chapter builds on this foundation by focusing on a specific ethnic or sexual minority community or another aspect of culture and includes examples of how to utilize the OCF and CFI in these communities. Weaving together the historical struggles of communities and salient research highlighting psychiatric disparities, Lim justifies why cultural psychiatry remains relevant. At the end of each chapter, Lim recommends specific questions for the populations discussed in the chapter and an overall assessment that highlights potential barriers to care resulting from their cultural identity. He also created a corresponding website for the book, with video vignettes using psychiatrists and actors illustrating the concepts outlined in the book: transference/countertransference with psychiatrists and patients who share similar backgrounds; examples of how psychiatrists and patients with different backgrounds discuss cultural identity and its importance within mental health; and different models of understanding psychiatric illness. While these vignettes appear staged at times, they model how these conversations can be done successfully.



Edited by Russell F. Lim, M.D., M.Ed. Washington, DC, American Psychiatric Association Publishing, 2015, 630 pp., $85.00.

Lim and his contributors have created a manual that builds on the model by Arthur Kleinman et al. (1) focusing on the eight questions that help illicit patients' perspective into their illness as a foundation for diving deeper into how culture impacts mental illness. Through focused questions, psychiatrists can explore topics as varied as gender identity for the LGBT community, immigration history for Asian populations, and religion in the African American community. At the same time, Lim and the contributing authors carefully try to remind readers of "the fallacy of assuming a homogenous explanatory model of illness with large ethnic blocks" (p. 325). This becomes the crux of the criticism that cultural psychiatry often faces given the fact that while describing attributes and perceptions of mental health, it runs the risk of reinforcing stereotypes. Good and Hannah (2) offer general criticism of cultural psychiatry, since it can perpetuate the pigeonholing of patients, and they specifically "recommend that mental health professionals consider the local contexts, with greater appreciation for the diversity of lived experience found among individual patients" (2, p. 219). This should be further emphasized when working with populations that straddle multiple cultural identities, such as the LGB immigrant or the transgender individual with a spiritual crisis. These hyphenated populations will continue to grow, and it will be important to contextualize their experiences holistically versus forcing their psychiatric concerns to fit into a rubric of sample questions.

While Lim succeeds in detailing the revisions to concepts and models surrounding cultural identity in the DSM-5, he also (unintentionally) highlights the age-old tension between understanding culture and reinforcing stereotypes. This book is a valuable purchase for early-career psychiatrists who are curious about understanding the impact culture has on mental health, as well as for those who want to better understand and thereby serve their patients.

Dr. Puri is a second-year resident in the Department of Psychiatry, University of California Los Angeles.

REFERENCES

1. Kleinman A, Eisenberg L, Good B: Culture, illness and care: clinical lessons from anthropological and cross-cultural research. Ann Int Med 1978; 88:251–258
2. Good MJ, Hannah SD: Shattering culture: perspectives on cultural competence and evidence-based practice in mental health services. Transcult Psychiatry 2015; 52:198–221

Residents' Resources

Here we highlight upcoming national opportunities for medical students and trainees to be recognized for their hard work, dedication, and scholarship.

**To contribute to the Residents' Resources feature, contact Hun Millard, M.D., M.A., Deputy Editor (hun.millard@yale.edu).*

OCTOBER DEADLINES

Fellowship/Award and Deadline	Organization	Brief Description	Eligibility	Contact	Website
American Association of Directors of Psychiatric Residency Training (AADPRT) awards **Deadline: October 30, 2015**	AADPRT	• Peter Henderson, M.D., Memorial Award *Best unpublished scholarly paper contributing to the field of child and adolescent psychiatry* • AADPRT/George Ginsberg Fellowship *Acknowledges the excellence and the accomplishments of outstanding residents interested in education and teaching* • International Medical Graduate (IMG) Fellowship *Designed to promote the professional growth and leadership of exceptional IMG residents and fellows* • Anne Alonso, Ph.D., Memorial Award *Best unpublished paper on psychotherapy written by a resident* • AADPRT/AAPDP Victor J. Teichner Award *Designed to promote and improve the teaching of psychodynamic principles of trainees in psychiatry*	See AADPRT's website for details	NA	http://www.aadprt.org/
Opportunity to publish IPS Workshop proceedings in the American Journal of Psychiatry Residents' Journal **Deadline: October 31, 2015**	AJP-Residents' Journal	Residents and fellows are invited to publish a synopsis of the IPS meeting (Oct 8–11, New York) in the AJP-Residents' Journal. The synopsis should cover a topic discussed at the plenary or workshops at the meeting. Format: 500 words (5 references), highlight: 1) what was known about the topic, 2) what is new, and 3) future directions.	Residents and fellows	Editor-in-Chief: Rajiv Radhakrishnan, M.B.B.S., M.D.; rajiv.radhakrishnan@yale.edu	

NOVEMBER DEADLINES

Fellowship/Award and Deadline	Organization	Brief Description	Eligibility	Contact	Website
American Psychosomatic Society (APS) Scholar Awards **Deadline: November 1, 2015**	APS	Between 10 and 24 APS Scholar Awards are presented to outstanding abstract submissions where the first author of an accepted abstract is either a student, resident, or fellow. Each award provides monetary assistance for the APS conference fees, travel, and hotel accommodations.	• APS member or in the process of applying for membership; • First author on an abstract accepted for presentation at the APS Annual Meeting; • Student or trainee enrolled in medical, graduate, or undergraduate school or in residency, internship, or postdoctoral fellow	NA	http://www.psychosomatic.org/awards/
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1. **Commentary:** Generally includes descriptions of recent events, opinion pieces, or narratives. Limited to 500 words and five references.
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3. **Treatment in Psychiatry:** This article type begins with a brief, common clinical vignette and involves a description of the evaluation and management of a clinical scenario that house officers frequently encounter. This article type should also include 2-4 multiple choice questions based on the article's content. Limited to 1,500 words, 15 references, and one figure. This article type should also include a table of Key Points/Clinical Pearls with 3–4 teaching points.
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7. **Drug Review:** A review of a pharmacological agent that highlights mechanism of action, efficacy, side-effects and drug-interactions. Limited to 1,500 words, 20 references, and one figure. This article type should also include a table of Key Points/Clinical Pearls with 3–4 teaching points.
8. **Letters to the Editor:** Limited to 250 words (including 3 references) and three authors. Comments on articles published in *The Residents' Journal* will be considered for publication if received within 1 month of publication of the original article.
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