The Residents' JOURNAL

a publication of
The American Journal of Psychiatry

January 2011 Volume 6 Issue 1

Inside

In This Issue

This month's issue of *The Residents' Journal* includes a section theme on internal medicine skills and psychiatry. The section begins with a review article by J. Chase Findley, M.D., who discusses the psychiatric symptoms associated with systemic lupus erythematosus. Next, Rosalyn Womack, D.O., shares her experience in treating patients with hyperglycemia while serving as psychiatric house officer. Paulette Lassiter, M.D., provides information on hypothyroidism in patients with mental illnes. Last, Toral Parikh, M.S., Kimberly Hamilton, M.D., and Luis M. Baez-Cabrera, M.D., present a case report of insulin sensitivity in a patient requiring antipsychotic medication.

Editorial

Discharge Planning

Joseph M. Cerimele, M.D.
Editor-in-Chief

In most departments, much of residency training takes place on the wards. Psychiatry residents generally spend the first and second years of residency on inpatient units and become proficient in the delivery of hospital-based care by the end of the second year. On the wards, residents learn to diagnose and manage common presentations (e.g., an exacerbation of chronic schizophrenia), how to recognize disease progression, how to document efficiently, and other basic hospital skills. One component of hospital care, discharge planning, may seem foreign or uninteresting to many residents. "Leave it for the social worker," or "I didn't learn about this in medical school" are two common complaints I have heard (and said at one point) while on the wards.

Because much of the first 2 years of training is spent in-house, junior psychiatry residents usually do not have experience working in the outpatient clinics and may be unfamiliar with the different types of outpatient care, levels of intensity of care, supportive housing options, and other variables available for the management of ambulatory patients. I entered the outpatient clinics in July of the current academic year and have seen patients in numerous settings. Now that I am somewhat familiar with how to care for outpatients (and after spending nights on-call in the emergency room), I understand the importance of coordinated ambulatory care and that much of a patient's outpatient treatment plan can be initiated during an acute hospitalization.

Problems can arise during discharge planning. Sukhera (1) described his experience in an article published in the August 2010 issue of *The Residents' Journal*. Furthermore, although some programs have demonstrated improved continuity of care subsequent to hospitalization (2), only 59.3% of Medicaid patients with schizophrenia attended an outpatient psychiatry appointment within 30 days of discharge from an acute hospitalization in 2003 (3). The transition to outpatient care can be challenging.

We plan to publish several articles on the topic of discharge planning in the June and July 2011 issues. Manuscripts to be considered for publication in the June and July issues must be submitted by March 31 and April 30, respectively. We hope authors will consider writing on the following topics: the physician's role in discharge planning, evidenced-based methods of improving adherence with posthospitalization care, ways to reduce the rates of substance relapse, discharge planning for special populations (e.g., veterans, children, the elderly), and (scholarly review article of) when to recommend state hospitalization. We will consider manuscripts outside of these subjects. Finally, we are also searching for a guest editor for this section. The guest editor's responsibilities will be to solicit pieces, correspond with the journal editors and manuscript authors, review manuscripts, and prepare a review article for publication. Residents interested in serving as guest editor should e-mail a CV and letter of intent (describing interests and plans as guest editor in less than 300 words) to me by February 1.

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Psychiatric Elements of Systemic Lupus Erythematosus: A Review

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Systemic lupus erythematosus is a chronic, debilitating, autoimmune disease with a remarkably wide array of both medical and psychiatric symptoms. In addition to experiencing diverse physical symptoms affecting nearly every organ system, a significant number of patients with systemic lupus erythematosus will experience mood, anxiety, cognitive, or psychotic symptoms during their lifetimes. Psychiatric symptoms are often related to the disease process itself but may also be secondary to the medical treatment of the disease (which often includes high-dose corticosteroids). Many patients with this disease will seek out psychiatric care on their own or be referred for psychiatric evaluation by other healthcare providers. Thus, it will benefit the psychiatric resident to have a basic understanding of the central pathophysiology of systemic lupus erythematosus as well as the medical, psychiatric, and social implications of living with the disease. The present article will review the immunological basis of systemic lupus erythematosus, its major associated physical and psychological symptoms, and the rate of psychiatric diagnoses in affected patients.

Epidemiology and Pathophysiology

The prevalence of systemic lupus erythematosus in the United States is 15–50 cases per 100,000 people (1). Of those affected, 90% are women of childbearing years, with the highest prevalence among African Americans. This gender disparity is related to the general nature of many mammalian species females to mount higher immune responses than their male counterparts, which may be the result of the activation and increased survival of T and B lymphocytes when exposed to the female sex hormone estradiol.

Systemic lupus erythematosus is characterized by the immune system's abnormal formation of autoantibodies and immune complexes, with subsequent damage to cells, tissues, and organs throughout the body. The underlying mechanism of the disease is the impairment of normal phagocytosis and removal of apoptotic cells (1). As a result, certain antigens (DNA/protein or RNA/protein complexes) are inappropriately accessible to the immune system. The resulting immune response causes cumulative tissue and organ damage, including cerebral tissue damage, and is responsible for the characteristic symptoms of the disease.

Diagnostic Criteria

The symptoms of systemic lupus erythematosus are numerous and diverse and may involve the nervous, musculoskeletal, cutaneous, hematologic, renal, cardiopulmonary, and gastrointestinal organ symptoms (1). However, the diagnosis of the disease is based on the patient displaying the presence of at least four out of 11 diagnostic criteria, which include various physical symptoms, physical exam findings, and laboratory results. The presence of four or more of these criteria has been shown to have 95% specificity and 75% sensitivity for systemic lupus erythematosus (Table 1).

Table 1
Diagnosis of Systemic Lupus Erythematosus[a]

Criteria
Malar rash: Fixed erythema, flat or raised, over the malar eminences.
Discoid rash: Erythematous circular raised patches with adherent keratotic scaling and follicular plugging; atrophic scarring may occur.
Photosensitivity: Exposure to light causes rash.
Oral ulcers: Includes oral and nasopharyngeal ulcers, observed by physician.
Arthritis: Nonerosive arthritis of two or more peripheral joints, with tenderness, swelling, or effusion.
Serositis: Pleuritis or pericarditis documented by ECG or rub or evidence of friction.
Renal disorder: Proteinuria >0.5 g/d or cellular casts.
Neurologic disorder: Seizures or psychosis without other causes.
Hematologic disorder: Hemolytic anemia or leukopenia (<4000/μl) or lymphopenia (<1500/μl) or thrombocytopenia (<100,000/μl) in the absence of offending drugs.
Immunologic disorder: Anti-double stranded DNA, anti-smooth muscle antibodies, and/or antiphospholipid.
Antinuclear antibodies: An abnormal titer of antinuclear antibodies by immunofluorescence or an equivalent assay at any point in time in the absence of drugs known to induce antinuclear antibodies.

[a]The presence of four or more criteria at any point in a patient's history is required for diagnosis of systemic lupus erythematosus. Data are from Tan et al. (9).

At the time of symptom onset, systemic lupus erythematosus may affect one or multiple organ systems. However, the disease is progressive and tends to affect an increasing number of organ systems over time (1). While the symptoms may go through alternating periods of flare and quiescence, the characteristic symptoms of fatigue, myalgias, and arthralgias are present for a majority of the time.

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Neuropsychiatric Lupus

Of particular interest to psychiatrists are the symptoms of neuropsychiatric lupus. In the past, neurological and psychiatric symptoms of systemic lupus erythematosus have been referred to by a variety of terms, including "CNS vasculitis," "lupus cerebritis," "CNS lupus," and "neurolupus" (2). However, in 1999 the American College of Rheumatology published the results of an ad hoc committee, formed to reach an agreement regarding the nomenclature of these symptoms, with the consensus to use the term "neuropsychiatric lupus" to describe all CNS-related symptoms of systemic lupus erythematosus. Included in these symptoms are mood disorders, psychosis, anxiety disorders, and cognitive dysfunction. While the symptoms of neuropsychiatric lupus are just a subset of the many symptoms of systemic lupus erythematosus, a majority of patients have been shown to experience cognitive dysfunction and mood disorders during the course of the disease (1). Although these symptoms are not criteria for the diagnosis of systemic lupus erythematosus, they may greatly contribute to a patient's physical suffering and disability. Less common but more severe symptoms include psychosis and seizures, the presence of either of which constitutes a criteria item for systemic lupus erythematosus diagnosis.

Systemic Lupus Erythematosus and Mood and Anxiety Disorders

In order to establish the prevalence of mood and anxiety disorders in patients with systemic lupus erythematosus, Bachen et al. (3) published a cohort study in 2009 on 326 Caucasian women with the disease. Forty-seven percent of the participants reported the lifetime presence of symptoms meeting diagnostic criteria for major depressive disorder, which is a rate 25% greater than that found in the U.S. population of adult Caucasian women. Other diagnoses found at an increased rate were specific phobia ([24%] 16% greater), panic disorder ([16%] 6% greater), obsessive-compulsive disorder ([9%] 1% greater), and bipolar I disorder ([6%] 1% greater). One-third of participants reported criteria for two or more diagnoses.

Interestingly, in the group of patients meeting criteria for any of the diagnoses, a majority reported experiencing psychiatric symptoms prior to being diagnosed formally with systemic lupus erythematosus (3). However, the majority of patients reported experiencing physical symptoms prior to psychiatric symptoms. In summary, patients tended to experience physical symptoms first, followed by psychiatric symptoms, and were subsequently diagnosed with the disease. This finding is significant for psychiatrists because it indicates that patients may present for psychiatric evaluation while already manifesting both physical and psychiatric symptoms but without having received formal diagnosis. The importance of addressing mood disorders as early as possible in the course of systemic lupus erythematosus is highlighted in a study conducted by Julian et al. (4), which found depression to have a significant effect on decreasing medication compliance and increasing emergency room visits in patients with systemic lupus erythematosus.

Systemic Lupus Erythematosus and Psychosis

To study the incidence rate of psychosis in patients with systemic lupus erythematosus, Appenzeller et al. (5) followed a cohort of 537 patients with the disease for a mean duration of 5.3 years (SD=1.1). During the period of observation, acute psychosis was diagnosed at some point among 17% of patients. Of the cases of psychosis identified and determined as a primary symptom of systemic lupus erythematosus, 21% occurred at disease onset (psychosis was often the first symptom identified in these patients), and 45% were identified during the course of the disease. In those with psychosis onset during the course of the disease, the mean time from diagnosis with the disease to identification of acute psychosis was 14 months (SD=5.2). The remaining 32% of cases were determined to be secondary to the effects of systemic corticosteroids used in the treatment of the disease.

In a similar cohort study focusing on psychosis in systemic lupus erythematosus, Pego-Reigosa and Isenberg (6) found that patients were more likely to experience psychotic symptoms during periods of florid clinical activity, especially when there was extensive cutaneous and hematological involvement and elevated levels of autoantibodies. Interestingly, Pego-Reigosa and Isenberg describe the use of corticosteroids (intravenous methylprednisolone), immunosuppressive (cyclophosphamide or azathioprine), or plasma exchange as successful treatment interventions for psychosis in these patients.

Appenzeller et al. (5) also demonstrated the presence of antiphospholipid antibodies to be an independent risk factor for the occurrence of psychosis in patients with systemic lupus erythematosus. Other studies have also found associations with depression, stroke, seizures, and cognitive dysfunctions, with a proposed underlying mechanism of antiphospholipid antibodies promoting cerebral ischemia.

Notably, Pego-Reigosas and Isenberg (6) also found that complete long-term remission of psychotic symptoms was the most common outcome for patients experiencing psychosis secondary to systemic lupus erythematosus ([70%] up to 20 years), without the need for long-term psychotropic medication.

Systemic Lupus Erythematosus and Delirium

The pathophysiology of systemic lupus erythematosus also places patients with the disease at risk for acute delirium, which consultation-liaison psychiatrists may be requested to evaluate. The severity varies from mild confusion to more severe agitation and hallucinations (7). Delirium in systemic lupus erythematosus may be associated with the primary disease process (cerebral ischemia or hemorrhage, metabolic derangements) or secondary to treatment of the disease (corticosteroids, immunosuppressives). Careful review of

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a patient's history, physical exam, laboratory findings, and medications should be performed in order to diagnosis and treat the medical cause of delirium.

Social and Life Stressors

In addition to physical symptoms and psychiatric illness, patients with systemic lupus erythematosus are likely to experience increased life stressors as a result of the chronic, debilitating nature of the disease. Patients are subject to a myriad of painful symptoms as well as the side effects of medications used to treat them. Frequent physician visits, expensive medications, and the possibility of the need for hospital admissions may place significant time and financial burdens on these patients. They may also experience aesthetically disfiguring skin changes, which may result in unfortunate social stigmatization. Since systemic lupus erythematosus typically results in a shortened life span (63%–75% 20-year survival rate from the time of diagnosis [1]), a patient may be psychologically impacted by a sense of foreshortened future and awareness of the nearly inevitable increase in painful and debilitating symptoms over time. Furthermore, the experience of chronic stress has been described as worsening the disease process (8).

Navarrete-Navarrete et al. (8) reported the results of a randomized control trial investigating the value of cognitive-behavioral therapy (CBT) in treating patients with systemic lupus erythematosus. The study showed that patients treated with 10 weekly 2-hour sessions of CBT focusing on stress management showed significantly reduced stress, anxiety, and depression. The group receiving CBT also reported improved quality of life and reduced somatic symptoms relative to a comparison group. While there was no difference in disease activity between the groups, it was apparent that CBT was useful in improving the life experience of patients with systemic lupus erythematosus.

Medical Treatment of Systemic Lupus Erythematosus

Since there is no cure for systemic lupus erythematosus, medical treatment of the disease is targeted at the reduction of symptoms, organ damage, and disability. Milder disease, predominated by symptoms of fatigue and pain, is treated with analgesics (nonsteroidal antiinflammatory drugs) and antimalarials (hydroxychloroquine) (1). More severe disease, with organ and life-threatening consequences, is treated with varying combinations of systemic corticosteroids and antineoplastics (including azathioprine, mycophenalate mofetil, and chlorambucil) (1). These latter treatments, especially corticosteroids, may independently cause symptoms of depression, anxiety, psychosis, mania, delirium, and cognitive dysfunction in some patients. Thus, it is often a diagnostic challenge for a psychiatrist to separate the primary symptoms of systemic lupus erythematosus from those secondary to medication side effects.

Psychiatric Treatment of Systemic Lupus Erythematosus

Unfortunately, no controlled trials on the pharmacological treatment of psychiatric symptoms in systemic lupus erythematosus have been performed (7). As in patients without systemic lupus erythematosus, depressive symptoms are often treated with selective serotonin reuptake inhibitors, bipolarity symptoms with mood stabilizers, and psychotic symptoms with atypical antipsychotics (7). Corticosteroids, immunosuppressive agents, or plasma exchange is often used in aggressive treatment of the disease (6). Psychosis secondary to steroid use may resolve by tapering the steroid dose but may also require an antipsychotic medication, such as haloperidol (4, 7). Since patients with systemic lupus erythematosus may also have renal and/or hepatic dysfunction as a result of the disease process, the choice of psychotropic medication used in the treatment of neuropsychiatric lupus symptoms should be guided by careful consideration of the metabolism and side effect profile of the individual medications. For example, lithium should be avoided in patients with renal dysfunction, which is a common consequence of systemic lupus erythematosus.

Conclusion

Systemic lupus erythematosus is a complex general medical condition that is likely to be encountered by the psychiatrist in the course of his or her career. The primary disease process, the treatment of the disease, and the stress of living with the disease may all have significant psychiatric consequences requiring specialized treatment by a psychiatrist. Systemic lupus erythematosus should be considered when forming a differential diagnosis of the general medical etiologies of mood, anxiety, and psychotic disorders, especially in women of childbearing years. A high degree of suspicion for the disease may result in psychiatrists assisting in the early diagnosis and treatment of patients. In partnership with primary care physicians, rheumatologists, and neurologists, the psychiatrist is well equipped to aid in the improvement of the quality of life for these patients.

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Hyperglycemia: A Brief Review for the Psychiatric House Officer

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Residency is the midpoint in medical education, between pupil and teacher (1). A rite of passage of this process is taking in-house calls as the psychiatric house officer. As part of the on-call duties, the psychiatric house officer is expected to provide psychiatric and basic medical coverage to the inpatient psychiatric unit.

A detailed review of the literature revealed no pertinent information regarding the most common medical problems encountered by psychiatric house officers. However, in the present author's experience, hyperglycemia in the diabetic patient is an acute medical concern commonly addressed while on psychiatric house officer duty. Hyperglycemia is defined as a disorder of glucose metabolism, with diabetes being the most severe form (2, 3). In 2007, it was reported that 23.5 million Americans aged ≥20 years had diabetes (4). Numerous studies have noted the prevalence of both diabetes and mental illness, and a detailed analysis of these studies is beyond the scope of the present article. However, through retrospective analysis of 243 psychiatric inpatients, aged 50–74 years, Regenold et al. (5) observed that the prevalence of endogenous diabetes mellitus type II in patients diagnosed with schizoaffective disorder was 50%. The prevalence was 26% in bipolar I disorder patients, 18% in both dementia and major depressive disorder, and 13% in schizophrenia (5). Moreover, many studies have implicated the use of atypical antipsychotics as causing increased risk for development of diabetes type II as well as worsening of pre-existing diabetes (6, 7).

Hyperglycemia in the noncritical patient has been defined as a blood glucose level >130 mg/dl preprandial (8), or >180 mg/dl according to the Society of Hospital Medicine (8). The American Association of Clinical Endocrinologists and the American Diabetic Association define the normal preprandial blood glucose level as 140 mg/dl (8). The insulin sliding scale is commonly used to treat acute increases in blood sugar, which is typically obtained via fingerstick (8).

The use of the insulin sliding scale solely for the management of hyperglycemia is not recommended because it is considered to be a reactionary approach to treatment and does not approximate the body's physiological response to insulin (8, 9). However, when the psychiatric house officer is providing cross coverage, it may be appropriate to use the insulin sliding scale in conjunction with the patient's scheduled diabetic medication regimen until the primary team assumes charge of the patient's care (9). In this instance, good documentation of the amount of additional insulin provided is warranted, since it allows for a sufficient transition of care to the primary team, who can then adjust the patient's treatment accordingly. Having the nurse notify the psychiatric house officer every time the insulin sliding scale is administered, rather than when blood sugar levels are elevated over a set point, would allow for better coverage. Laboratory examinations for newly admitted patients should include a complete blood count, comprehensive metabolic panel, and hemoglobin A1c blood test as well as a beta-human chorionic gonadotropin test for female patients. If the patient is a known diabetic, has at least two readings for a random blood glucose level >180 mg/dl or fasting blood glucose level >126 mg/dl, subcutaneous insulin is the recommended treatment. Table 1 summarizes the amount of insulin that is recommended (8). One-half of the dose should be administered with a long-acting insulin and the other half in three divided doses with meals (8). Recent recommendations also include the use of insulin for patients receiving oral hypoglycemic as outpatients, since these agents may cause complications in the inpatient setting if the patient's medical status were to suddenly change (2,8).

Untreated asymptomatic hyperglycemic episodes may progress to severe hyperglycemic episodes, which can result in diabetic ketoacidosis in diabetes type I and a hyperglycemic osmolar state in diabetes type II. Symptoms of diabetic ketoacidosis include polyuria, nausea, vomiting, shortness of breath, and abdominal pain, and physical examination may reveal dehydration, hypotension, abdominal tenderness, shortness of breath, lethargy, and possible coma (2). Signs and symptoms of hyperglycemic osmolar state are similar to those of diabetic ketoacidosis, with the exception of gastrointestinal disturbances and shortness of breath (2).

Table 1

Subcutaneous Insulin Administration[a]

Dose	Criteria
0.3 units/kg	Elderly, receiving dialysis, body mass index <18
0.4 units/kg	body mass index >18 but <25
0.5 units/kg	body mass index >25 but <29
0.6 mg/kg	insulin resistance, receiving glucocorticoids, body mass index >29

[a]*Data from Nau et al. (8).*

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For these patients, an immediate referral to medicine is necessary, since they require a level of care that exceeds the capability of the inpatient psychiatric unit. A review of the literature yielded no relevant information about the frequency of these occurrences with the psychiatric house officer, but given that mentally ill patients are less likely to comply with medical treatment (6, 7) and have higher rates of mortality at younger ages as a result of medical illnesses (including diabetes), versus the general population (6, 7, 10), it is prudent for the psychiatric house officer to have a high index of suspicion for the symptom manifestations of severe hyperglycemia.

Hyperglycemia has been a common medical problem encountered by the present author while covering as psychiatric house officer. To the author's knowledge, no pertinent research has been dedicated to identifying common medical conditions encountered by psychiatric house officers, including the frequency of symptomatic hyperglycemia. However, this is an area for future study. Given the prevalence of hyperglycemia in the inpatient psychiatric population, it is important to know the appropriate use of the insulin sliding scale, since it is likely that psychiatric house officers will have to treat a hyperglycemic patient.

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Hypothyroidism in Psychiatric Patients

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Thyroid hormone plays a vital role in the growth and development of the CNS and is also important in the adult brain (1). Thyroid hormone regulates metabolism in all tissues of the body. Deficiencies of the hormone can cause both metabolic and cardiovascular problems, including increased low-density lipoprotein, decreased myocardial contractility, arteriosclerotic cardiovascular disease, and atrial fibrillation (2–4). In addition, hypothyroidism can also negatively affect mood and cognitive functioning. Furthermore, there is significant overlap between the symptoms of depression and hypothyroidism. The purpose of the present article is to provide a review of the three types of hypothyroidism and discuss etiology and incidence as well as common symptoms and indications for treatment.

Table 1
Causes of Hypothyroidism

Medication	Autoimmune	Illness
Amiodarone	Hashimoto's thyroiditis	Diabetes
Carbamazepine	Atrophic thyroiditis	Chronic renal failure
Iodine	Postpartum thyroiditis	Hypopituitarism
Lithium		Primary adrenal failure
Phenylbutazone		
Phenytoin		
Sulfonamides		

Definition

Grade I hypothyroidism, or overt hypothyroidism, is defined as elevated thyroid stimulating hormone levels and decreased concentrations of triiodothyronine (T3) and tetraiodothyronine (free thyroxine [FT4]) in the serum (1, 2, 4). There is a clear association between overt hypothyroidism and psychiatric symptoms, especially in the elderly. Its presentation can range from mild cognitive dysfunction to reversible dementia. Onset in its most severe form can present as delirium or psychosis (1, 2, 5). Grade II hypothyroidism, also known as mild or subclinical hypothyroidism, features elevated thyroid stimulating hormone concentrations with normal T3 and FT4 serum levels. Patients with this type of hypothyroidism are more commonly asymptomatic but may present with symptoms of the condition, such as hair loss, cold intolerance, constipation, fatigue, menstrual irregularities, and dry skin, or they may have cognitive or mood symptoms. Specifically, decreased attention and memory as well as slowed processing speed and reaction time are cognitive complaints, while mood symptoms are most commonly depression. However, there are case reports of mania and psychosis (6). Up to 30% of patients with subclinical hypothyroidism will be symptomatic. Grade III hypothyroidism, which is the mildest form, is characterized by normal thyroid stimulating hormone and thyroid hormone levels. When challenged with intravenous administration of thyroid-releasing hormone, patients with this type of hypothyroidism will have an exaggerated response (2, 3). Other than diagnosing this mild form of the condition, the thyroid-releasing hormone challenge is of little clinical utility given the sensitivity and specificity of the thyroid stimulating hormone assay (4, 7).

Etiology

The etiology of hypothyroidism is varied. In industrialized nations, autoimmune diseases, such as Hashimoto's thyroiditis and atrophic thyroiditis, are the most common causes. Worldwide, the most common cause is iodine deficiency. Other causes include postpartum thyroiditis, neck irradiation, and medications. Lithium may cause hypothyroidism as well as other drug treatment, including amiodarone, anticonvulsants, iodine, and immune response modulators (2, 3, 6, 8). Chronic illness, such as diabetes type I, chronic renal failure, hypopituitarism, and primary adrenal failure, are also possible etiologies (Table 1). While the prevalence of subclinical hypothyroidism ranges from 1% to 10%, the highest rates are among women and the elderly. Women aged >60 years may have a rate as high as 20%. Some studies indicate that men aged >74 years have a rate of 16%, while other studies have reported this same rate in men aged >64 years. In the elderly, while it is normal to have decreased secretion of T3 and T4, the serum level of FT4 remains relatively the same secondary to metabolic changes associated with aging (2).

Prolonged treatment with lithium is known to predispose patients to both clinical and subclinical hypothyroidism. It is estimated that 8%–19% of patients receiving treatment with lithium will develop overt hypothyroidism, and 20%–23% will develop subclinical hypothyroidism. This is more likely to be seen in patients with the presence of antithyroid antibodies at baseline or in those with a thyroid disease history. Lithium may decrease the uptake of iodine into the thyroid gland, inhibit concentration ability, impede conversion of T4 to T3, and interfere with the release of thyroid hormone. In addition to baseline laboratory examination, patients receiving lithium should have their thyroid stimulating hormone level evaluated 3 months after starting the medication and then again every 6–12 months (5, 6, 9).

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Symptoms

In moderate to severe forms of the disease, there is a clear association between hypothyroidism and cognitive deficits. These patients may present with decreased concentration, appetite, and energy as well as sleep disturbance and apathy. Patients become symptomatic once serum concentrations of thyroid stimulating hormone reach a range of 8 μU/ml–12 μU/ml. In mild forms of the disease, mood, somatic, and cognitive symptoms are more likely to be subtle. These patients tend to score higher on depression and anxiety rating scales and lower on memory testing (6). Osterweil et al. (10) conducted a study in older adults that examined neuropsychological functioning in 54 nondemented men and women with both overt (N=38) and subclinical (N=14) hypothyroidism. They found global cognitive deficits with poor performance on tests measuring visuospatial abilities, visual scanning and attention, motor speed, fluency, and learning in patients with hypothyroidism relative to euthyroid comparison subjects (1, 2). Studies in younger adults have examined cognitive deficits and found impaired memory, verbal fluency, selective attention, and slowed reaction time (1).

Incidence

The overall incidence of hypothyroidism in patients with depression is low. However, Haggerty et al. (8) found the lifetime prevalence of depression in patients with subclinical hypothyroidism to be 56% relative to 20% in euthyroid individuals (6, 8). In a study of patients resistant to treatment with antidepressants (11), it was found that the incidence rate was 22%; while in patients that were not treatment resistant, the incidence rate was only 2%.

Treatment

In depressed patients with normal thyroid stimulating hormone concentrations, the recommendation is not to treat for hypothyroidism unless the patient is high risk, such as those with postpartum depression, diabetes mellitus, or a history of autoimmune disease (12). Patients with elevated thyroid stimulating hormone concentrations of ≥10 μU/ml should be given replacement therapy, but a more conservative approach recommends treating levels ≥8 μU/ml. The advantage of treating at levels of ≥8 μU/ml rather than 10 μU/ml would be to capture cases of depression secondary to subclinical hypothyroidism. For patients with mildly elevated thyroid stimulating hormone levels (range: 4.6 μU/ml–8 μU/ml), the recommendation is to treat those that also have decreased T3 or T4, positive antibody titers, or demonstrate cognitive impairment (6, 12).

Conclusion

In summary, since hypothyroidism presents in psychiatric patients as a primary cause of neuropsychiatric symptoms, as a comorbid condition, and as an adverse treatment effect, appropriate screening is important when evaluating this population.

Dr. Lassiter is a fourth-year resident in the Department of Psychiatry, Wilford Hall Medical Center, Lackland Air Force Base, Tex.

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Case Report

Insulin Resistance in Mental Illness: A Case Report and Reassessment

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A link between mental illness and peripheral insulin resistance was well described by the year 1900. Walter Cannon, who coined the term homeostasis, presented important original research on what was called emotional glycosuria (1). Today, emotional glycosuria is referred to as the metabolic syndrome. An important break from the past is the current emphasis on the role of drug treatment for mental illness, such as atypical antipsychotics, in the development of insulin resistance and subsequent cardiovascular disease (2). We present a case in which clozapine-induced insulin resistance in a schizophrenia patient influenced drug selection and subsequent psychiatric course without benefit to the patient.

Table 1

Comparison of Clinical Symptoms With Serum Glucose Levels, Serum Insulin Levels, and Insulin Insensitivity[a]

Date	Glucose (mg/dl)	Insulin level (μU/ml)	Insulin Resistance Index (HOMA-IR)	Psychotropic Medication[b]	Clinical Symptoms[c]
01/23/07	93	9.1	2.09	D, E, G, I	a, b, c, d, e, g, h
10/21/07	84	4.4	0.913	D, F, H, I	b, d, e, g, h
12/05/07	102	6	1.51	D, E, F, I	a, d, g, h
12/12/07	95	4.8	1.13	D, E, F, I	a, d, g, h
05/10/08	91	7.7	1.73	B, F, I	a, g, h
12/02/09	98	2.8	0.678	A, B, C	f, g, h
07/09/10	94	3.6	0.836	B, C	a, b, c, g, h
07/22/10	97	5.1	1.22	B, C	e, h

[a]The serum glucose normal range is 70–120 mg/dl; the serum insulin normal range is 3.3–17.5 μU/ml.

[b]Symbols for psychotropic medication are as follows: A=Clozapine; B=Risperidone; C=Depakote; D=Haloperidol; E=Lorazepam; F=Loxapine; G=Olanzapine; H=Paliperidone; I=Quetiapine.

[c]Symbols for clinical symptoms are as follows: a=Hallucinations; b=Disorganized speech; c=Increased psychomotor activity; d=Dysphoria or irritability; e=Impaired affect; f=Impaired sensorium; g=Impaired thought process; h=Delusions.

Case

"Mr. M" was a 59-year-old Caucasian man with chronic paranoid schizophrenia, which was well controlled with clozapine. The patient was a chain smoker with severe, chronic, obstructive pulmonary disease. He was 66 inches tall and weighed 171 pounds. His body mass index was 27 kg/m^2. In May 2010, he had a myocardial infarction. His outpatient psychiatry team worried that clozapine might be causing metabolic syndrome and thus contributing to the risk of myocardial infarction. Treatment with clozapine was discontinued, and the patient was started on risperidone. After switching to risperidone, the patient experienced repeated exacerbations of psychotic symptoms, with worsening suspiciousness and behavioral responsiveness to his persistent delusions (Table 1). Because of his repeated hospitalizations, the inpatient psychiatry team was able to obtain laboratory results for his fasting insulin and glucose levels.

Table 1 shows the patient's HOMA (homeostasis model assessment) of insulin sensitivity and pancreatic beta cell function, which was first described in 1985. The HOMA of insulin resistance (HOMA-IR) index is now a widely tested and accepted means of expressing the simple relationship between insulin and glucose in insulin resistance. For this particular measure, the fasting insulin level is multiplied by a simultaneously collected fasting glucose level and then divided by a correction factor. When used to assess insulin resistance, a correction factor of 405 is divided into the product of insulin and glucose. The HOMA-IR value tends to rise as insulin resistance increases. There are other ways to measure insulin sensitivity, but we chose HOMA-IR because of its wide acceptance among endocrinologists. An upper limit of normal for the HOMA-IR may be approximately 2.6 (3) Table 1 shows that, by this criterion as well as by the raw fasting insulin and glucose values alone, "Mr. M" was never insulin resistant, even while receiving treatment with clozapine.

The impression of those caring for the

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patient, including his psychiatrists and caretaker, was that he was the least psychotic and the most cooperative and compliant with treatment plans while receiving clozapine. After clozapine was discontinued, he became hostile and suspicious of his caretaker, with whom he had lived for many years. As a result of persistent concerns about the possibility of metabolic syndrome with clozapine treatment, it was not restarted. "Mr. M" developed further myocardial infarctions and underwent multiple hospitalizations for his worsening cardiac condition, which was in part due to his suspiciousness of the cardiac medications, but he did not develop diabetes.

Discussion

At our institution, acute mental illness has been shown to impair insulin sensitivity, while any effective treatment for the mental illness, including atypical antipsychotics, tends to restore insulin sensitivity (4). This is not a new finding. Insulin resistance, measured by HOMA-IR, has been directly correlated with psychotic stress, reaching its peak on admission and decreasing afterward. Treatments ranging from insulin coma therapy to modern psychotropics have been shown to improve insulin sensitivity among mentally ill patients (5).

In the case of the present patient, it appears that he was insulin sensitive during his treatment with clozapine. Furthermore, the consensus of those who worked with the patient is that his mental status improved with clozapine treatment and worsened after it was discontinued. This raises the question of how to manage concerns about insulin sensitivity among patients requiring antipsychotic medication. In the present case, worsening psychosis was an obstacle to effective treatment of cardiovascular disease, but insulin resistance due to clozapine treatment was not.

Toral Parikh is a third-year medical student, Dr. Hamilton is a second-year resident, and Dr. Baez-Cabrera is a fourth-year resident in the Department of Psychiatry, University of Texas Health Science Center at San Antonio, San Antonio, Tex.

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Psychiatrists' Use of Social Networking and Patient-Psychiatrist Interactions

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The use of social networking among physicians and medical students has generated significant interest. However, the use of social networking specifically among psychiatrists has not been reported, and this use is of particular interest to our profession, given the importance of values such as neutrality, privacy, and boundary crossings.

A questionnaire was developed that assessed what social networking tools psychiatrists use, including Facebook, MySpace, Twitter, and LinkedIn. Practitioners were also asked about any patient interactions that occurred via social networking. The Northwestern Memorial Hospital Institutional Review Board granted approval of the survey, which was sent via e-mail to Illinois Psychiatric Society members as well as psychiatry residents in Illinois.

Fifty-four psychiatry responses were returned, but an actual response rate cannot be tabulated because of the large number of e-mail addresses that were sent back by the server as erroneous (>200). A total of 56.6% of psychiatrists were participants in at least one social networking site. Higher participation was reported among more recent psychiatric trainees, with 73.1% of current residents and psychiatrists who graduated <5 years ago (N=26) connected to at least one social networking site, compared with 37.5% of psychiatrists who completed residency >10 years ago (N=24). Given the high degree of participation by psychiatrists on social networking sites, it was not surprising that patient-psychiatrist interactions occurred frequently. A total of 16.6% of respondents who indicated that they were members of a social networking site (N=30) had either sent or received messages from patients via these sites. In addition, 16.6% of the total respondents (N=54) reported that they have had patients ask them questions about their personal lives based on information obtained via an Internet search. Conversely, 14.8% of all psychiatrists reported bringing into their sessions information about a patient they obtained through an Internet search.

To my knowledge, this is the first survey to assess the use of new media by psychiatrists and how this use may blur the lines between our professional offices and personal time. The inability to calculate response rates and the recruitment method via e-mail necessitate caution in concluding from the survey what percentage of all psychiatrists are using social networking sites. However, the utility of this survey is consistent with recognition of more recent graduates having a rate of social networking connections nearly double that of prior graduates. This should serve to emphasize the urgency in which new professional guidelines should be developed as new, more socially connected psychiatrists come into the field every graduation year. In fact, 78.8% of respondents indicated that they would welcome new professional guidelines. As the percentage of psychiatrists using these sites increases, patient interactions will also increase, without proper guidelines. There have already been cases of boundary and privacy violations necessitating disciplinary action by residency and hospital administrators. Another basis for the need to establish professional guidelines is that the more experienced supervisors that train residents may not be able to provide sufficient guidance to their supervisees, since many of them do not use social networking sites.

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An Efflorescence of Autism: From a Child to a Man

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Introduction

"Mr. C" was a 28-year-old Caucasian man with autistic disorder who was brought to my office by his group home manager, "Ms. T," for management. The supervising physician suggested that I see him weekly as a psychotherapy patient. I was unsure how to manage him.

Assessment

Ms. T had reported that the patient frequently displayed physical aggression and had been hospitalized several times. A number of group homes had evicted him due to ongoing physical aggression. The patient had been living in his current group home for the past year. Ms. T was in the process of changing his guardianship because his guardian at the time, a maternal uncle, was very difficult to contact. Ms. T empathetically coordinated the patient's care. In the group home, he communicated only with her. She reported that early treatments were unsuccessful. His social interactions were punctuated by his unpredictable physical and verbal aggression.

In the past 10 years, Mr. C had occasional phone contact with his father but none with his mother. He knew few family members. His group home symbolized a place for children rather than adults. Food, sleep, and activities were usually controlled by staff. Even the initial sessions with me were filled with the speech of his case worker. The words of the "other" took up all the time in our sessions. The patient was not a subject in the room.

After an initial phase of twice-weekly sessions, I began seeing the patient once monthly. His treatment formulation involved a phenomenological perspective.

Beginning of Treatment

At appointments, Mr. C demonstrated little eye contact. He looked at the floor and away from me. He spoke without emotion and spoke to himself without acknowledging me. During the first two appointments, I had to endure silence. Ms. T stayed with us throughout the interviews as an informant, and the patient sometimes acknowledged her with a nod of his head. He never responded to my simple questions. I was curious about how to establish a path of communication with him.

Interpretation

1) After the first two sessions, I remained interested in Mr. C's unique behavior. During session four, I told Ms. T she would have several minutes for updates or questions in subsequent visits, since "I would like to spend the sessions with Mr. C." As I said this, the patient gave me momentary eye contact for the first time. He was given a place to speak, and his response was eye contact. This belied an unconscious desire to be seen in the room.

He sat quietly in the corner of my office in the appointments that followed, as we made no verbal communication. My silence created a space for him to find his desire to speak beyond relying on his body to give voice to his unconscious. I comforted him with an enjoyable book.

2) After several sessions with Mr. C, I decided to catch his attention by talking with Ms. T. In the next two sessions, I asked Ms. T for an update on the patient's behavior at the group home and in his vocational rehabilitative program. She reported that he had been non-compliant and was, at times, aggressive with staff. I then let her wait in the reception area. I did not ask the patient anything. He was restless and then walked out of my office.

The group home called after he left my office. He had hit Ms. T without provocation. Was this acting out the result of an unsaid interpretation? He was enraged at losing his place in the room as the intrusive "other" came back. I wondered what other repetitive reactions might ensue, and I asked him to come to my office the next morning.

3) In this appointment, Ms. T reported details of the incident from the previous day. I asked her, "Is there anything you appreciate in Mr. C?" She did not answer. The "other's" expectations and attention had previously focused on his violence and not his potential as a human being. My words left the "other" speechless.

After Ms. T left, I did not look at the patient, nor did I ask him anything. Then, for the first time, he asked me a question. He said, "Do you want to ask me what happened?" I replied calmly, "If you insist," shifting the tables as I communicated that his words were what I wanted to hear. I was certain the desire to speak must now be owned by him. However, he chose to stay quiet. When I told him he could leave, he asked me, "Am I going to go to the hospital?" I told him that I believed in him. I knew this intervention brought me closer to him and that a relationship was being created between us. The transference was built on listening without being intrusive.

4) Even though Mr. C did not talk with me during his sessions, he did draw a Christmas tree during one visit, which I hung in my office. I noted that at each visit, he would look at his picture and then quickly look at the floor. When I rearranged my office, he searched for his drawing and made himself comfortable after spotting it on the wall. Perhaps the drawing was a much needed symbol that he had a place in my office or it was an object that spoke of his desire to have a home where he was wanted. His gaze searched for that object of belonging, just as his gaze looked at me with the same desire. In any case, it was with this that he started speaking occasionally about staff and Ms. T.

5) Mr. C. had reacted belligerently after receiving a phone call from his father. A

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phone call was a direct trigger for his violence in the group home. The man who called was not really a "father" but rather a reminding voice of his father's absence. His body acted out another statement of rage.

Following this outburst, I told him that "a man would never hit a woman." His response was a sustained stare at me. I finally felt that I now existed in our dyad. This session marked the beginning of a new stage in treatment. Perhaps I made an interpretation on the symbolic axis with an instruction about how to live as a man, aimed directly at his unconscious desire.

Emergence of a Man

Mr. C realized that there was an expectation of him as a man. He mumbled, "I am a man." His behavior began to change. He became helpful at the group home as well as the vocational rehabilitation, often repeatedly saying that he was a man who should be helpful to others. He evolved from an aggressive, demanding child into an adult. He now talks in each session. His questions are usually directed toward what is socially appropriate or inappropriate. He explores laws governing behavior. He still sees me for supportive therapy once monthly. In a recent session, Ms. T noted that the patient had changed into the most likable person in the group home. His communicative and social skills continue to improve. This past year was the first in the prior 18 in which he had not been hospitalized. He appreciates the value of human relations, since he "is a man now."

At the time this article was accepted for publication, Dr. Selvaraj was a fourth-year resident in the Department of Psychiatry, Creighton University, Omaha, Nebraska. The author thanks Pam Jesperson, M.A., C.P.C., L.M.H.P., Thomas Svolos M.D., and Daniel R. Wilson, M.D., Ph.D., for their assistance.

Book Review

Book Review of *ADHD Diagnosis and Management: A Practical Guide for the Clinic and the Classroom*

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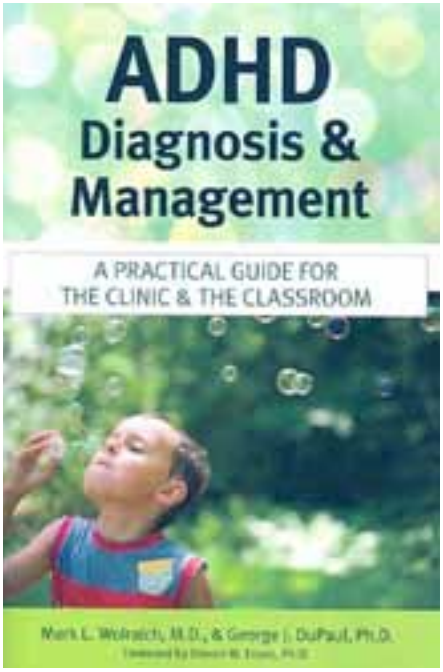
These are exciting times for the psychiatric profession. Just when questions were being raised about the validity of psychiatric conditions, such as attention deficit hyperactivity disorder (ADHD), a recent study (1) unequivocally established genetic evidence for ADHD. Nevertheless, we are yet to witness a key scientific breakthrough that would help diagnose ADHD with valid neurobiological investigations. Until then, the optimal diagnosis and management of ADHD will require effective information sharing with multiple stakeholders. This is where *ADHD Diagnosis and Management* proves to be a welcome addition to the existing literature on the disorder.

The book is reader friendly and delivers evidence-based content, which readers from diverse backgrounds will find useful. The authors discuss the history, screening, diagnosis, and management of ADHD.

Parents and teachers often struggle to differentiate between appropriate and inappropriate behaviors associated with ADHD. The authors point out that most of the scales used in diagnosing the disorder are based on identifying negative behaviors, resulting in non-normative distributions. While acknowledging this as a limitation, the authors recommend several scales that have attempted to incorporate normative distributions. Consequences associated with ADHD can be mitigated if appropriate screening procedures are implemented. The authors make a strong case for universal ADHD screening in elementary school children and screening for comorbid disorders in children diagnosed with the disorder.

Since children spend time in a variety of settings, information from schools, teachers, and healthcare providers is extremely important in making a valid diagnosis. The authors also encourage readers to develop sound management strategies tailored toward academic and social functioning of children, in addition to symptom management.



By Mark L. Wolraich, M.D., and George J. DuPaul, Ph.D. Baltimore, Brookes Publishing, 2010, 216 pp, $34.95 (paper).

The chapter on treatment strategies includes various evidence-based psychopharmacologic and behavioral strategies. Moreover, the authors' included a short description of strategies that are not supported with sound empirical data, such as use of St. John's wort and exclusion of artificial colorings from the diet. This, along with suggestions for assessing the veracity of resources, helps empower readers in making informed decisions. Readers are encouraged to treat parents as fully informed partners, underscoring the importance of the parental role in successful management of ADHD in children.

The chapter on school procedures is very informative. Several teacher- (e.g., providing task choices), parent- (e.g., daily report card), peer- (e.g., behavior monitors), computer- (e.g., computer-assisted instruction), and self- (e.g., recognizing and recording behaviors) mediated interventions are discussed in sufficient detail. The authors kept the discussion simple, which would enable swift implementation of these strategies in a variety of school settings. The discussion on the consultation model and eligibility for special education and Section 504 services is enlightening and will help readers to procure necessary services for children.

In summary, this book is a succinct and informative text on ADHD. I highly recommend this book to my resident colleagues. Readers will be able to employ management skills discussed in a wide variety of clinical situations not limited to ADHD.

Dr. Prabhakar is Chief Resident (Outpatient Department) and a third-year resident in the Department of Psychiatry and Behavioral Neurosciences, Wayne State University, Detroit.

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TEST YOUR KNOWLEDGE

In preparation for the PRITE and ABPN Board examinations, test your knowledge with the following questions.
(answers will appear in the next issue)

Question #1.
A 28-year-old woman presents with complaints of irritability, fatigue, feelings of inner tension, and crying spells that occur during the week immediately preceding menstruation and end shortly after menstruation begins. The patient states that the symptoms are severe and affect her relationship with her husband. She has also been increasingly irritable at work. She requests a medication to help relieve these symptoms. Which of the following medications is not approved by the Food and Drug Administration for treatment of this patient's condition?

A. Fluoxetine
B. Paroxetine CR
C. Citalopram
D. Sertraline

Question #2.
A 54-year-old man with treatment refractory schizophrenia and poorly controlled epilepsy is initiated on clozapine following failure with two typical and two atypical antipsychotics. It is known that clozapine can lower seizure threshold at a dose-dependent manner. At what dose of clozapine is the seizure threshold noted to be lowered?

A. 100 mg daily
B. 300 mg daily
C. 500 mg daily
D. 600 mg daily

ANSWERS

Answers to December Questions. To view the December Test Your Knowledge questions, go to ajp.psychiatryonline.org/cgi/data/167/12/A30/DC2/1.

Question #1.
Answer: C. Desipramine
Desipramine is the only secondary amine listed among the options. Antidepressant medications with antimuscarinic effects can cause urinary retention. Tricyclic antidepressants have prominent antimuscarinic activity. However if a tricyclic has to be chosen, secondary amines, such as desipramine, are least likely to cause urinary retention or hesitancy.

Reference

1. American Psychiatric Association: Practice Guideline for the Treatment of Patients With Major Depressive Disorder. Am J Psychiatry 2010; 167(October suppl):75

Question #2
Answer: A. Erythema ab igne
Erythema ab igne is the only correct answer. Patients with anorexia nervosa suffer from starvation-induced hypothermia. Erythema ab igne is an irregular, fixed, reticulated hyperpigmented patch that develops secondary to chronic application of a source of heat, such as hot water bottles. In rare cases, there may be malignant transformation associated with Erythema ab igne.

Reference

1. Birmingham CL, Treasure J: Complications by system, in Medical Management of Eating Disorders, 2nd ed. Edited by Birmingham CL, Treasure J. New York, Cambridge University Press, 2010, pp 29–58

We are currently seeking residents who are interested in submitting Board-style questions to appear in the Test Your Knowledge feature. Selected residents will receive acknowledgment in the issue in which their questions are featured.
Submissions should include the following:
1. Two to three Board review-style questions with four to five answer choices.
2. Answers should be complete and include detailed explanations with references from pertinent peer-reviewed journals, textbooks, or reference manuals.
***Please direct all inquiries and submissions to Dr. Fayad; fayad@ufl.edu.**

Author Information for *Residents' Journal* Submissions

The Residents' Journal accepts manuscripts authored by medical students, resident physicians, and fellows; manuscripts authored by members of faculty cannot be accepted.

1. **Commentary:** Generally includes descriptions of recent events, opinion pieces, or narratives. Limited to 500 words and five references.

2. **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 1,000 words and 10 references.

3. **Clinical Case Conference:** A presentation and discussion of an unusual clinical event. Limited to 750 words and five references.

4. **Original Research:** Reports of novel observations and research. Limited to 1,000 words, 10 references, and two figures.

5. **Review Article:** A clinically relevant review focused on educating the resident physician. Limited to 1,000 words, 10 references, and one figure.

6. **Letters to the Editor:** Limited to 250 words (including 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.

7. **Book Review:** Limited to 500 words.

Abstracts: Articles should not include an abstract.

References: Use reference format of *The American Journal of Psychiatry* (http://ajp.psychiatryonline.org/misc/Authors_Reviewers.dtl).

Upcoming Issue Themes

Please note that we will consider articles outside of the theme.

March 2011
Section Theme: The On-Call Experience
Guest Section Editor: Monifa Seawell, M.D.;
mseawell@med.wayne.edu

April 2011
Section Theme: Psychosomatic Medicine
Guest Section Editor: Amit Pradhan, M.D.;
dramitpradhan@hotmail.com

May 2011
Section Theme: Exercise and Psychiatric Disorders
Guest Section Editor: Corey Meyer, M.D.;
cmeyer@challiance.org

June 2011
Section Theme: No specific theme
Guest Section Editor: Deepak Prabhakar, M.D.;
dprabhakar@med.wayne.edu

We invite residents who are interested in participating as Guest Section Editors to e-mail Dr. Cerimele at joseph.cerimele@mssm.edu. If you are interested in contributing a manuscript on any of the themes outlined, please contact the Section Editor for the specified month.