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## IN THIS ISSUE

*Focus of the Month:*
**Topics in Neuropsychiatry**





# Treating Headaches in Psychiatry

We know that headaches are common in the general population, but they are particularly common among patients with psychiatric problems. According to one review, (Pompili M et al., *J Headache Pain* 2009; 10(4):283-290) patients with depression have a 46% lifetime prevalence of migraine, while bipolar patients have a 51% prevalence. Patients with migraines have triple the risk of developing depression than patients without migraines.

In this article, we'll review how to handle patients with headaches in a psychiatric practice. How do we distinguish migraine headaches from tension headaches from more dangerous secondary headaches that may be caused by a tumor or by hypertension? When should we refer headache patients to a neurologist? How should we treat these patients, both pharmacologically and psychotherapeutically?

## Diagnosis

Textbooks will tell you that headaches come in essentially two varieties: primary (migraine, tension-type, and cluster) and secondary (caused by an underlying intracranial problem).

While tension-type headaches (46% prevalence) are more common than migraines (25% prevalence), migraines are more likely to drive patients to seek medical attention, because they are usually more severe and debilitating. Migraine headaches are different from tension-type headaches in several ways. They are often unilateral rather than bilateral; they usually cause a throbbing, rather than a static pain; one third are associated with a prodromal aura; they often are accompanied by nausea and vomiting (so-called "sick headaches"); and patients often complain of photophobia, meaning that bright light makes the headache worse.

In one study a simple three question screen was remarkably predictive of migraine. These three questions are:

1. Are you nauseated or sick to your stomach when you have a headache?
2. Have the headaches limited your activities for a day or more in the last three months?
3. Does light bother you when you have a headache?

When two of these questions are answered "yes," the positive predictive value for migraine was 93%; when all three were answered positively, the predictive value was 98% (Lipton RB et al., *Neurology* 2003;61:375-382).

There's a tendency among non-specialists to view tension-type headaches as being a waste-basket category for anything that is not a migraine, but in reality these headaches have fairly specific diagnostic criteria. According to the International Headache Society, tension-type headaches must meet at least two of the following criteria: 1. Pressing or tightening (nonpulsating) quality; 2. Mild to moderate intensity (meaning not so severe as to prohibit any normal activities); 3. Bilateral location; and 4. No aggravation from walking on stairs or similar activities. Furthermore, tension-

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**Learning objectives for this issue: 1.** Describe common causes and treatments of headache. **2.** Describe how different causes of tremor are diagnosed and treated. **3.** Describe elements of the neuropsychiatric assessment. **4.** Understand the most current findings in the literature regarding psychiatric treatment. This CME/CE activity is intended for psychiatrists, psychiatric nurses, psychologists and other health care professionals with an interest in the diagnosis and treatment of psychiatric disorders.

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type headaches must mean both of the following: 1. No nausea or vomiting; and 2. Only one of the following can be present: photophobia or phonophobia. Tension-type headaches typically last from 30 minutes to seven days. Patients will describe a "band of pain" extending from the forehead around both temples to the back of the head.

Cluster headaches are rare and are described as an excruciating "boring" pain, often over one eye, lasting from 15 minutes to several hours. They may occur several times per day or night. I have a patient, a man in his early 30s with depression and OCD, who gets cluster headaches about once a year, and he is rendered completely out of commission for several weeks. The condition has played havoc with his ongoing efforts to complete a technical degree, and to move out of his parent's house problems that, in turn, have complicated his psychiatric treatment.

When should we refer to a neurologist? The short answer: almost always. Anybody who has severe or chronic headaches might have a secondary cause, and neurologists are experts at ruling this out. The workup will include a detailed neurologic exam, with an emphasis on the fundoscopic exam, and may or may not include a brain MRI, depending on the results of the exam and the neurologist's judgment or philosophy.

## Medication Treatment

**Tension-type headaches.** Once a secondary cause of a headache has been ruled out by a neurologist, psychiatrists are generally qualified to treat headache disorders, largely because of the great overlap between psychiatric drugs and headache drugs.

For an episode of tension-type headaches, aspirin (500-1000 mg) and ibuprofen (400 mg) work well, and better than acetaminophen (1000 mg) (see Loder E and Rizzoli P, *Brit Med J* 2008; 336:88-92 for a good review of tension-type headache management). When these over-the-counter meds don't work, you can move on to bigger guns such as Fioricet, which is a combination of acetaminophen (325 mg), butalbital (50 mg), and caffeine (40 mg). Why is this triad of drugs so effective? Well, acetaminophen is a standard analgesic, butalbital is a barbiturate that relaxes the scalp's muscle contractions, and caffeine relaxes blood vessels, improving blood flow to scalp muscles. Fioricet works for both tension and migraine headaches, but it is addictive and can cause intermittent headaches to convert to chronic headaches.

Generally, when headaches become chronic, neurologists try to switch from "abortive" drugs (those that stop the headache in its tracks) to prophylactic drugs. The best prophylactic drug for chronic tension headaches is amitriptyline, usually dosed from 10 mg up to 75 mg one or two hours before bedtime; nortriptyline may be as effective, with fewer side effects. While the newer antidepressants such as the SSRIs and SNRIs are somewhat effective as well, they are not as effective as amitriptyline.

**Migraine headaches.** Migraines come in many varieties, ranging from mild and rare to severe and frequent. I have had two migraines in my life. Each was triggered by looking briefly at the sun, and each began with a strange visual aura like hundreds of translucent overlapping gears spinning frenetically. The aura lasted about 15 minutes, followed by the onset of a pulsing left-sided headache. On each occasion, I took 600 mg of ibuprofen, and felt the need to lie down in a dark room. Within two hours or so, I recovered.

I describe this case to illustrate the spectrum of migraines and their treatment. Anybody who presents themselves to a specialist will have had migraines far more severe and more frequent. While over-the-counter analgesics are effective for milder cases, often you need to ramp up treatment to drugs such as Fioricet, codeine, and various narcotics, all of which are considered acceptable treatments for the occasional migraine. But for more severe and frequent migraines, the preferred abortive treatments are triptans (such as sumitriptan and rizatriptan) which have replaced the ergots as the treatment of choice. There are now seven different triptans to choose from and each company will brandish studies to convince you theirs is best. Because of this complexity, neurologists are usually the better specialists to initiate treatment with a triptan.

More important is for psychiatrists to know something about the controversy surrounding the safety of combining triptans with SSRIs. In 2006, the FDA issued an alert that combining triptans with either SSRIs or SNRIs can cause serotonin syndrome. The agency said this was based on 27 cases gathered over five years. The announcement was met widely with skepticism, because millions of patients had been combining triptans with SSRIs over the years and serotonin syndrome had rarely, if ever, been reported in the literature. Despite requests, the FDA has not made details of these 27 cases of supposed serotonin syndrome public, and a recent review concluded that "withholding these medications due to fears of serotonin syndrome is difficult to justify" (Wenzel R et al., *Ann Pharmacother* 2008;42(11): 1692 -1696).

Most of the drugs effective as prophylactics against migraines (as opposed to abortive drugs) are well-known to psychiatrists, including amitriptyline, nortriptyline, Depakote (divalproex sodium), Neurontin (gabapentin), and Topamax (topiramate). Effective non-psychiatric prophylactic agents include the beta blockers propranolol and atenolol, and the ACE inhibitor verapamil. This is a good list to keep in mind for those "two-fer" opportunities, such as when you are choosing something for a depressed patient with migraines (go with the tricyclics) or for a migraine patient with bipolar disorder (the obvious choice would be Depakote).

## Non-pharmacological Approaches

While few psychiatrists will be investing in a biofeedback machine, this treatment may be the most effective non-pharmacological approach for headaches, especially tension-type headaches. One recent meta-analysis found that biofeed-

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back was more effective than headache monitoring, placebo, and relaxation therapies (Nestoriuc Y et al., *J Consult Clin Psychol* 2008;76:(3):379–396.)

Headache clinics often have psychologists and psychiatrists on staff and they have developed techniques that you can incorporate into your practice for patients with comorbid depression or anxiety and migraine headaches. Called "behavioral medicine," these are typically versions of cognitive behavioral therapy. The usual strategy is to determine how much the patient is allowing headache to limit activities, and to encourage them to "get back out there", on the theory that they are inadvertently reinforcing their depressed mood through passivity. Help the patient make a list of enjoyable activities and come up with a graded schedule for re-engaging in life. Such techniques have been shown helpful in decreasing headache severity in some studies (Smitherman TA et al., *Headache* 2007;48(1):45-50).

Other techniques that have some empirical support for relief of chronic headaches include standard progressive muscle relaxation exercises and acupuncture.

**TCPR VERDICT:** ***Psychiatrists have a major role in headache treatment, ranging from psychopharmacology to psychotherapy.***

# Evaluating and Treating Tremor

While tremor is traditionally thought of as a neurological issue, the symptom pops up often in psychiatric practice, and some basic knowledge of its diagnosis and treatment comes in handy.

Case vignette: A 67-year-old woman whom I'll call "Paula" came to see me for depression and anxiety. She ascribed her depression to her belief that neighbors were coming into her apartment and stealing things—which her daughter assured me was implausible. Her memory and cognitive processing were normal otherwise, and her PCP had already obtained an array of labs and a head CT to rule out a contributing medical illness. I provisionally diagnosed her with depression with psychotic features, and I treated her with a combination of Celexa (citalopram) and low dose Risperdal (risperidone), with Ativan (lorazepam) on an as needed basis. On Paula's next visit, she held out her hands in front of me and said, "Doctor, I've gotten even shakier." I did notice a fine tremor, but further questioning revealed that her depression and paranoia had improved markedly. Nonetheless, she interpreted her "shakiness" to mean that she was worse.

So what was causing Paula's tremor? Anxiety? Celexa? Risperdal? A neurological condition such as Parkinson's disease? Or was this "essential tremor"?

By far, the most common cause of tremor is essential tremor, also called "benign familial tremor." (See Smaga S, *Am Fam Physician* 2003;68:1545-1552, for a helpful review of the differential diagnosis of tremor). It generally begins in the 50s, is familial in up to 60% of cases, and usually presents as a symmetrical fast, fine tremor of the wrist most visible when the patient stretches the arms in front of you. It is gradually progressive, and sometimes affects the head, causing either yes-yes or no-no head movements. Essential tremor is typically a constant tremor, but may wax and wane, and famously improves transiently with alcohol ingestion.

Upon careful questioning, it turned out that Paula's father had a tremor for much of his life, and in fact Paula said she had a tremor for many years. Studies have shown that essential tremor often goes unrecognized because it can be very mild—in fact, about 50% of people with essential tremor are unaware of it (Eible RJ, *Mov Disord* 1998;13:457-464).

Drug-induced tremor is also common and often looks just like essential tremor. Psychiatric drugs that can cause tremor include the SSRIs, the tricyclic antidepressants, lithium, and Depakote (divalproex sodium). (For a recent review of tremor in psychiatric practice, see Arbaizar B et al., *Psych Clin Neurosc* 2008;62:638-645.) Antipsychotics, especially the first generation antipsychotics, also can cause tremor, but this tremor is distinct from other tremors in that it mimics the tremor of Parkinson's disease: it is worse at rest, and it is a slower, "coarse" tremor. Tremors are often technically described in terms of "frequency" (the speed of the tremor) and "amplitude" (the breadth of the tremor). Thus, essential tremor and most drug-induced tremors are of high frequency and low amplitude, while a Parkinsonian tremor is of lower frequency and higher amplitude.

Differentiating essential tremor from drug-induced tremor is not easy. Did it clearly begin after the patient started taking the drug? Did it go away when the patient had a gap in compliance? Is the tremor more prominent an hour or so after ingesting the drug (often seen in lithium-induced tremor)? "Yes" answers support a drug-induced tremor.

While my patient Paula did, indeed, have a history of essential tremor, it was also clear that her tremor had worsened since she began Celexa and Risperdal. In addition to her fine postural tremor (consistent with essential tremor), I noticed that she had a coarse resting tremor of her wrists, typical of a neuroleptic-induced tremor.

My solution was to discontinue the Risperdal, with the intention of replacing it with Seroquel if her paranoia returned. Her tremor improved back to her baseline, and luckily the combination of the Celexa and lorazepam kept her psychiatric symptoms at bay.

Another kind of tremor-related symptom deserves special mention: anti-

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depressant-induced jitteriness. In 1990, an influential case series of six patients who developed intense suicidal ideation after treatment with Prozac (fluoxetine) brought this issue to our attention (Teicher MH, et al., *Am J Psychiatry* 1990;147:207-210). Since then, case studies have been published associating SSRIs and other antidepressants with what has variously been termed: jitteriness, akathisia, shakiness, activation, and agitation.

Recently, a group of psychiatrists conducted a systematic review of all 107 articles they could locate describing antidepressant-induced jitteriness/anxiety (Sinclair LI et al., *Br J Psychiatry* 2009; 194:483-490). Since there is no agreed-upon definition of AD-induced jitteriness, the published incidence rates are all over the map, ranging from 4% to 65%. The two antidepressants most likely to cause jitteriness were Prozac and imipramine (perhaps merely because they were the most studied in relation to jitteriness), although it has been described for other tricyclics and other SSRIs including Zoloft and Paxil. The syndrome is particularly likely in patients with panic disorder, and somewhat less so in depression. It generally begins within the first two weeks of treatment. Trying to distinguish jitteriness from akathisia has proven very difficult, and these reviewers concluded that they may be one and the same entities. Regarding the risk of suicide, an FDA-commissioned analysis of antidepressant clinical trials in children and adolescents found an elevated risk of suicidal behaviors among youth who experienced symptoms of "activation" (Hammed T www.fda.gov/ohrms/dockets /ac/04/slides/2004-4065S1_08_FDA-Hammad.ppt). In Pfizer's clinical trials of sertraline for depression in children, aggression, agitation, and akathisia led to study discontinuation in eight of 189 patients taking sertraline compared to 0 of 184 patients taking placebo, a statistically significant difference (Wagner KD et al., *JAMA* 2003; 290:1033-1041).

## Management of Tremor

The management of tremor and jitteriness depends on the cause. When it is clearly caused by a drug, discontinuing the offending agent is the best solution, but often a drug works so well that the patient prefers to stay on it. Antidotes to tremor include the following (for references, see the review article Arbaizar B et al., *Psych Clin Neurosc* 2008;62:638-645).

**Beta-blockers**. Propranolol (Inderal) is usually used, starting at 10 mg BID or TID; some patients require up to 40 mg TID. You can also try the extended release version, Inderal LA, which has the advantage of once-a-day dosing; the beginning dose is 60 mg QD. Warn patients about the common beta-blocker side effects of fatigue and postural light-headedness.

**Benzodiazepines**. Both alprazolam and clonazepam have been shown to be helpful for tremor, and are also helpful (as are all the other benzos) for AD-induced jitteriness.

**Primidone (Mysoline)**. Primidone is an anti-seizure drug that it also effective for essential tremor. Its mechanism of action is unknown. Start at 12.5-25 mg QHS, and increase gradually as needed. Effective doses range from 50-250 mg QD, usually given at bedtime because of the side effect of sedation.

**Topiramate (Topamax)**. Topiramate has been shown to be more effective than placebo for essential tremor. The starting dose is 25 mg/day, which may need to be increased up to 200-400 mg/day.

**Gabapentin (Neurontin)**.Somewhat less effective than the options already described, gabapentin sometimes eases both tremor and anxiety, but the effective dose varies widely, with a suggested maintenance dose of 1200-3600 mg/day.

**TCPR VERDICT:** *When faced with tremor, think essential vs. drug-induced.*

# Research Updates IN PSYCHIATRY

Section editor, Glen Spielmans, PhD

Glen Speilmans, PhD has disclosed that he has no relevant financial or other interests in any commercial companies pertaining to this article.

## DEPRESSION

### *Role of Depression "Risk Gene" Questioned*

It has long been thought that one's genetic makeup combines with stressors to cause depression, but for many years there was little data to support this hypothesis. This changed in a 2003 paper by Caspi and colleagues, which strongly supported the idea that variation in the serotonin transporter gene, when combined with stressors, drastically influences the rate of depression (Caspi A et al., *Science* 2003;301:386-389). By itself, this variation did not predispose participants to depression. But people with one short allele of "serotonin transporter linked polymorphic region" (5-HTTLPR) were more likely to become depressed in the face of stressful life events, and people with two short alleles were even more so. These results supported the popular serotonin theory of depression, since short alleles decrease the production of the serotonin transporter, leading, presumably, to faulty regulation of serotonin in the synapses. Intrigued by such findings, other scientists attempted to replicate them. A new meta-analysis

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# Research Updates IN PSYCHIATRY

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examined the results of 14 such studies, and found that the short allele result was apparently a chance finding. In fact, only four studies supported the link between the short allele and depressive response to life events, whereas two found opposite results (long allele was linked to depressive response to life events), with others finding no consistent link between 5-HTTLPR and depressive response to stress. When the studies were combined, there was no link between the 5-HTTLPR genotype and responding to stressors with depression. (Risch et al., *JAMA* 2009;301:2462-2471).

***TCPR's Take***: Several studies were excluded from the meta-analysis because their measurements were not directly comparable to the original Caspi et al. paper; this could have biased the findings somewhat. However, such exclusions happen in most meta-analyses and are unlikely to have altered the results significantly. Given the complexity of our genetic makeup and the frequent failure to replicate psychiatric genetics findings, it is likely wise to wait for consistent replication when a "risk gene" is identified.

## CLINICAL PRACTICE

### *Restricting Drug Samples May Reduce Costs*

Drug samples are a staple of pharmaceutical marketing. The drug industry argues, quite logically, that samples are useful to treat patients who lack the funds to purchase medications. Yet some reformers have called for drug samples to be banned, an idea that has received a decidedly mixed reception. A small body of research has investigated the topic of drug samples, including a recent study that examined changes in prescriptions when samples of brand-name statins, levothyroxine, and SSRIs were removed from an internal medicine clinic in Iowa. Over the 180-day period in which samples were removed, generic prescriptions increased by 23% for statins, 10% for SSRIs, and 3% for levothyroxine (Miesner AR et al., *Arch Intern Med* 2009;169:1241-1242). In another study, a university-based internal medicine clinic moved, leaving its sample closet inaccessible for nine months. During this time, the rate of generic prescribing for uninsured patients increased from 12% to 30%, though there was no change in generic prescribing for Medicaid patients (Miller DP et al., *South Med J* 2008;101:888-893).

Surprisingly, drug samples are more often given to insured Americans than to the uninsured or poor (Cutrona SL et al., *Am J Public Health* 2008;98:284-289). In that case, drug samples also tend to increase costs for patients who receive them (Alexander GC et al., *Med Care*; 46:394-402); when patients receive a sample, they often remain on that more expensive drug once the sample expires rather than switching to a less expensive generic drug. Samples probably produce overall cost savings only if they will be given indefinitely to patients who have no coverage for prescriptions and cannot afford to pay out of pocket for generic drugs.

***TCPR's Take***: The combined results of these studies suggest that clinicians should be wary of drug samples and consider carefully whether they are actually helping to reduce costs for patients.

## TRICHOTILLOMANIA

### *N-Acetylcysteine Effective For Trichotillomania*

Trichotillomania (TTM) is an impulse control disorder in which patients feel that they can relieve tension by pulling out hair from different parts of their bodies. While sharing some features with obsessive compulsive disorder, DSM-IV-TR does not officially classify it as a type of OCD. The pharmacological treatment of trichotillomania has often frustrated patients and clinicians alike. Clomipramine has outperformed placebo, but its benefits appear to diminish substantially after discontinuation and SSRIs have failed to beat placebo across four trials (Bloch MH et al., *Biol Psychiatry* 2007;62:839-846). A recent study compared an amino acid (N-acetylcysteine, or NAC) to placebo. Why NAC? Because NAC is converted to cysteine, which reduces synaptic release of glutamate. Some research has implied that repetitive behaviors like TTM my be caused by excessive glutamate, providing a biochemical rationale for a trial of NAC for TTM. Of the 50 enrolled patients, most had at least one comorbid condition, including depression (28%), an anxiety disorder (28%), or another impulse control disorder such as compulsive skin picking (36%). About half were taking other psychotropic medications, and maintained their dose of these drugs during the study. Participants took 1200 mg/day of NAC for six weeks (or matching placebo), then increased dosage to 2400 mg daily for the remaining six weeks. Patients on NAC outperformed placebo patients on all trichotillomania measures by a quite large effect size. 44% of NAC patients experienced a 50% or greater drop in trichotillomania symptom rating scores compared to zero patients on placebo. NAC did not improve depression or anxiety scores more than placebo. Remarkably, no patients on NAC reported any side effects (Grant JE et al., *Arch Gen Psychiatry* 2009;66:756-763), though NAC may worsen asthma.

***TCPR's Take***: NAC can be found at many health food stores and is cheaper than most prescription medications. The very promising safety and efficacy profile demonstrated in this study provides much needed hope to patients and clinicians alike, though replication is needed. Also keep in mind that habit reversal therapy (a form of behavior therapy), has demonstrated efficacy for trichotillomania.

Q&A With the Expert

## *This Month's Expert:*
## Thinking like a Neurologist
## Bruce Price, MD

*Chief, Department of Neurology, Mclean Hospital*
*Assistant Professor of Neurology, Harvard Medical School*

Dr. Price has disclosed that he has no relevant relationships or financial interests in any commercial company pertaining to this educational activity.

**TCPR: Dr. Price, for most of your career, you've worked closely with psychiatrists in treating patients with neuropsychiatric disease. I thought we might start by discussing how psychiatrists might bring more neurological thinking into their evaluations. When should psychiatrists start thinking neurologically? Which patients should raise the red flag that something "organic" is going on?**

**Dr. Price:** When a patient has an atypical history that doesn't quite fit the usual DSM definitions or that doesn't fit your clinical experience. For example, when there is an unusual age of onset of a psychiatric symptom, like a late onset of mania or major depression, especially when it is out of the blue, with no psychosocial explanation. Also, if there is an unusually acute onset of a sudden change in behavior, or the absence of a family history of a disorder that is usually familial, such as bipolar disorder.

**TCPR: So, basically, any patient whose history and symptom pattern don't quite jibe with what we normally see.**

**Dr. Price:** Right. I also look at treatment history. If a patient has not responded to multiple psychiatric medications or therapies that are normally effective, you should wonder about an underlying neuropsychiatric problem. And then, of course, there are certain symptoms that are red flags. Patients who, in addition to typical psychiatric symptoms, also report things like new or worsening headache, somnolence, incontinence, focal weakness, sudden incoordination or gait difficulties, may have neurological illness.

**TCPR: Are there any other things that we should be focusing on in the history?**

**Dr. Price:** Yes, it is crucial to ask about a history of head injury. Traumatic brain injury can lead to syndromes that can appear "psychiatric," like impulsivity, poor memory, anger, and the like. And when you ask about head injury, try to get the full story of what actually happened. Was there a loss of consciousness, and if so, how long did it last? And after the patient regained consciousness, how many hours or days did it take for him or her to regain normal memory functioning? Answers to these questions will give you a sense of how severe the head injury was, and how likely it is that current symptoms might be due to the event. On the other hand, getting a detailed history might reveal that the head injury was less severe than you originally thought. Sometimes families will exaggerate the extent of the injury because of an understandable desire to find some explanation for the patient's psychiatric symptoms.

**TCPR: What are some of the typical psychiatric symptoms that can be caused by traumatic brain injury (TBI)?**

**Dr. Price:** There are many. The most common are cognitive impairment which gradually improves over the course of time, and a disinhibited personality, which might present as being atypically quick to anger, or, conversely, as being unusually or inappropriately jovial. Two syndromes that many psychiatrists are not aware of as possibly being worsened by TBI are borderline personality disorder and perpetration of domestic violence. TBI can contribute to both of these by causing impulsivity and disinhibition.

**TCPR: We've heard a lot recently about post-concussive syndrome in athletes leading to depression. What is a concussion, exactly?**

**Dr. Price:** In the old days we used to think that it had to include loss of consciousness, but we now consider it to be any sudden impact of the head that even temporarily and briefly alters your mental state.

**TCPR: Aside from a careful neuropsychiatric history, should psychiatrists be doing more of the actual neurological exam?**

**Dr. Price:** In my dream world, yes, psychiatrists would conduct an elementary neurologic exam on intake, especially for patients whose history hints at a neuropsychiatric problem.

**TCPR: Before going into exactly what that exam ought to be, how should we introduce this to our patients? After all, a neurologic exam involves touching the patient, which is something that most patients are not expecting from a psychiatrist.**

**Dr. Price:** I would say something like, "The brain is so complex and we need to understand different angles to understand it. The neurologic exam offers one angle and we want to be sure we get the correct diagnosis."

**TCPR: That sounds reasonable. So what should we include in our neurological exam?**

**Dr. Price:** Start by examining the visual field to make sure there is not a field cut. You have them look at your nose and you bring your hand out into the four quadrants of their visual field and wiggle your fingers, asking if they can see it. Then examine their gait for stability and for a focal weakness on one side or the other.

**TCPR: My office is pretty small—how do we evaluate gait in a small space?**

**Dr. Price:** I have some small offices too, and I watch them walk for five steps towards the door and then come back five steps to the chair. And I would ask them to do a tandem gait, the heel-to-toe kind of test that cops use to check sobriety. You should tap the basic reflexes, including the biceps, the patellar (just below the knees), and the ankle. And I would stroke the bottom of the foot to make sure the toe goes down. Finally, test strength of the arms and legs, focusing on symmetry of strength.

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# CME Post-Test

To earn CME or CE credit, you must read the articles and complete the quiz below, answering at least four of the questions correctly. Visit www.TheCarlatReport.com to take the test online and print your certificate or mail a photocopy or fax the completed page (no cover sheet required) to **Clearview CME Institute, P.O. Box 626, Newburyport, MA 01950; fax (978) 499-2278**. For customer service, please call (978) 499-0583. Only the first entry will be considered for credit and must be received by Clearview CME Institute by August 31, 2010. Acknowledgment will be sent to you within six to eight weeks of participation. This activity has been planned and implemented in accordance with the Essential Areas and Policies of the Accreditation Council for Continuing Medical Education (ACCME) through the sponsorship of the Clearview CME Institute. Clearview CME Institute is accredited by the ACCME to provide continuing medical education for physicians. Clearview CME Institute is also approved by the American Psychological Association to sponsor continuing education for psychologists. Clearview CME Institute maintains responsibility for this program and its content. Clearview CME Institute designates this educational activity for a maximum of one (1) AMA PRA Category 1 Credit™ or 1 CE for psychologists. Physicians or psychologists should claim credit commensurate only with the extent of their participation in the activity.

*Please identify your answer by placing a check mark or an X in the box accompanying the appropriate letter. Note: learning objectives are listed on page 1.*

1. The most effective prophylactic drugs for tension-type headaches are: (Learning Objective #1)
   - [ ] a. Tricyclic antidepressants.
   - [ ] b. Serotonin reuptake inhibitors.
   - [ ] c. Divalproex sodium (Depakote).
   - [ ] d. Ibuprofen and aspirin.

2. The most common cause of tremor is: (L.O. #2)
   - [ ] a. Parkinsonian tremor.
   - [ ] b. Essential tremor.
   - [ ] c. Drug-induced tremor.
   - [ ] d. Anxiety-induced tremor.



3. A recent meta-analysis found that the short allele of a serononin transporter gene is correlated with high risk of depression. (L.O. #4)
   - [ ] a. True [ ] b. False

4. A recent study of N-Acetylcysteine (NAC) in Trichotillomania found: (L.O. #4)
   - [ ] a. NAC is effective only at low doses of 25-50 mg/day.
   - [ ] b. NAC was effective, but no more effective than placebo.
   - [ ] c. NAC worsened skin-picking symptoms.
   - [ ] d. NAC was more effective than placebo at 1200-2400 mg/day.

5. According to Dr. Price, traumatic brain injury can lead to syndromes that can appear "psychiatric," like impulsivity, poor memory, and anger. (L.O. #3)
   - [ ] a. True [ ] b. False

***PLEASE NOTE: WE CAN AWARD CME CREDIT ONLY TO PAID SUBSCRIBERS***

First Name _____ Last Name _____ Degree (MD, PhD, NP, etc.) _____

Street Address _____

City _____ State _____ Zip _____

Phone _____ E-mail _____

**Your evaluation of this CME/CE activity (i.e., this issue) will help guide future planning. Please respond to the following questions:**

1. Did the content of this activity meet the stated learning objectives? L.O.#1: [ ] Yes [ ] No L.O.#2: [ ] Yes [ ] No L.O.#3: [ ] Yes [ ] No L.O.#4: [ ] Yes [ ] No
2. On a scale of 1 to 5, with 5 being the highest, how do you rank the overall quality of this educational activity? [ ] 5 [ ] 4 [ ] 3 [ ] 2 [ ] 1
3. As a result of meeting the learning objectives of this educational activity, will you be changing your practice performance in a manner that improves your patient care? Please explain. [ ] Yes [ ] No

4. Did you perceive any evidence of bias for or against any commercial products? Please explain. [ ] Yes [ ] No

5. How long did it take you to complete this CME/CE activity? ___ hour(s) ___ minutes
6. **Important for our planning:** Please state one or two topics that you would like to see addressed in future issues.

## Q & A With the Expert

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**TCPR: How long should this exam typically take?**

**Dr. Price:** This is a five-minute neurologic exam. If it's normal, document that so you have that as a baseline, and any abnormalities would prompt a referral to a neurologist.

**TCPR: I know that lately you have been interested in the comorbidity of depression and major neurological diseases. Is there anything in particular psychiatrists should know about?**

**Dr. Price:** Yes, depression is highly comorbid in some very common neurological diseases, such as Alzheimer's disease, traumatic brain injury, multiple sclerosis, Parkinson's disease, stroke, and epilepsy. Between 30%-50% of patients with these diseases will develop depression at some point, and there is increasing evidence that the pathogenesis of neurologic diseases and depression may have common mechanisms.

**TCPR: How might this information be particularly relevant in a psychiatric practice?**

**Dr. Price:** For example, patients who present with late-onset depression (defined as 65 years old or after) have a 2 to 4 times higher incidence of Alzheimer's disease and Parkinson's disease than people with no depression. Thus, late-onset depression may be an independent risk factor for the development of Parkinson's and Alzheimer's.

**TCPR: If I have a late-onset depression patient, should I automatically refer them to a neurologist saying, "Look Ms. Smith, there is data that you are 2 to 4 times more likely to develop a neurologic condition because of what is going on with you; I would like you to see Dr. Price."**

**Dr. Price:** Let's put it this way. It would certainly heighten my awareness as a psychiatrist that there may be an accompanying neurodegenerative disease, and it would lower my threshold for neurologic referral.

**TCPR: Thank you very much Dr. Price.**




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**Next Month in *The Carlat Psychiatry Report*:** The Latest on Antidepressants, including reviews of antidepressant augmentation, an update on Pristiq, and the status of antidepressant psychotherapy research.