CHIEF EDITOR DR. SYED MUBIN AKHTAR

KARACHI PSYCHIATRIC HOSPITAL

Regd. No. SS-237 BULLETIN JUNE 2013

(Psychiatric Research Articles)

UNDERSTANDING MENTAL ILLNESS

-program organized by Karachi Psychiatric Hospital, Hyderabad.

Chief Guest Prof. Dr. Moin Ahmed Ansari
-receiving shield from Dr. Syed Mubin Akhtar

Guest of honor Dr. Kamal Parvaiz
-receiving gift from Dr. Syed Mubin Akhtar

Mental Health Awareness Program organized by Karachi Psychiatric Hospital at Govt. College of Education. Dr. Syed Mubin Akhtar MD. KPH speaking on the occasion.

KARACHI PSYCHIATRIC HOSPITAL

Vol.36 Issue No. VI June - 2013 Regd. No. SS -237

MONTHLY BULLETIN

(Psychiatric Research Articles)

جو شخص خدا کے بجائے
رواج کی پیروی کرے وہ منافق ہے

"اگر کوئی شخص یہ کہتا ہے کہ خدا اور رسول ﷺ کا حکم کچھ بھی ہو، مگر فلاں بات تو باپ دادا سے ہوتی چلی آرہی ہے، اس کو کیسے چھوڑا جاسکتا ہے! یا فلاں قاعدہ تو میرے خاندان یا برادری میں مقرر ہے، اسے کیونکر توڑا جاسکتا ہے! تو ایسے شخص کا شمار بھی منافقوں میں ہوگا، خواہ نمازیں پڑھتے پڑھتے اس کی پیشانی پر کتنا ہی بڑا گٹا پڑ گیا ہو اور ظاہر میں اس نے کتنی ہی متشرع صورت بنا رکھی ہو۔" (خطبات..... سید ابوالاعلیٰ مودودیؒ)

Readers are requested to send their articles, comments & suggestions.
Doctors who desire to get the bulletin should send their address, contact number and email to:

Address: B-1/14, Nazimabad # 3, Karachi-74600
Phone:111-760-760
Fax: 36681610
Email: editor@kph.org.pk
Email: support@kph.org.pk

CONTENTS

REFINEMENTS IN ECT TECHNIQUES

Karon Dawkins, MD- Psychiatric Times

Electroconvulsive therapy (ECT) has been practiced since 1938 and is one of the best-studied treatment modalities. Because of its long history, it has been the comparator for a number of subsequent therapies, including pharmacological and other somatic interventions. Even with ECT, however, the response rate for treatment-refractory patients is sobering, and the treatment is not without risks and adverse effects. Still, it can be effective when other interventions have failed.

Treatment-refractory major depression is the primary indication for ECT, although ECT may have particular utility in psychotic depression, catatonia, and suicidality as well. The American Psychiatric Association (APA) guidelines also list an indication for schizophrenia.

The Case Vignette illustrates a fairly typical case for ECT referral, and the patient is a reasonable candidate.

To suppress the electrically induced tonic-clonic motor seizure activity, and thus minimize musculoskeletal complications, paralytic agents are administered. Because conscious paralysis would be very distressful, patients receive general anesthesia. ECT entails a series of treatments, typically 6 to 12, to achieve response or remission. Cognitive adverse effects are the major concern, especially for patients and their families. Even with full remission, relapse rates remain high, particularly for the nonpsychotic medication-resistant population. Generally, once symptom remission is achieved, ECT is frequently discontinued. **However, some patients get continuation (to prevent relapse) or maintenance (recurrence prophylaxis) ECT.**

WHEN TO GIVE ECT

The decision to pursue ECT is based on a number of factors, including "diagnosis, type and severity of symptoms, treatment history, consideration of the anticipated risks and benefits of ECT and alternative treatment options, and patient preference."

The response rate for patients who are not medication-resistant is higher, up to 90% in one multi-site study (log-linear analysis x2= 6.82, df = 1, P = .009).

The FDA efficacy review estimated the overall treatment effect to be 78%. For treatment-refractory patients, the ECT response rate may be about 50% to 60%.

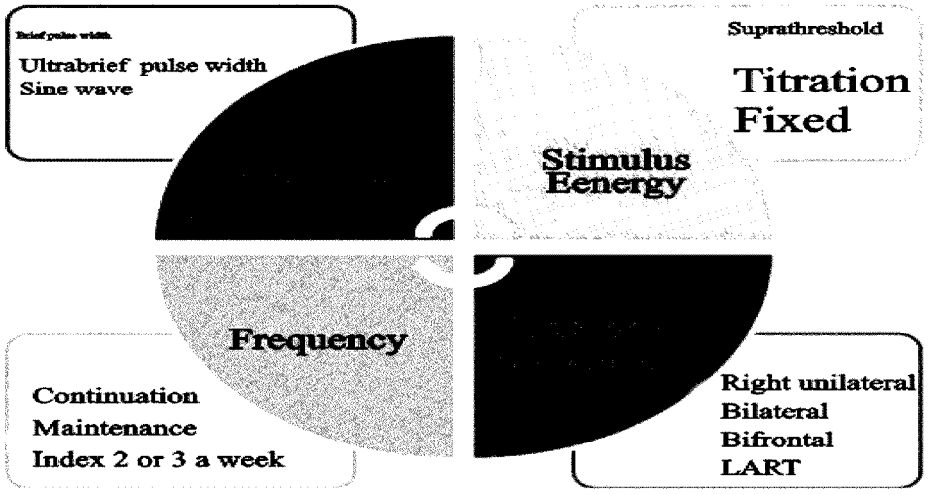
ECT can be rapidly effective in mania and catatonia, and it can be an early consideration for depressions with psychotic features. ECT can be considered in patients with medication-resistant schizophrenia. There are special considerations for children and adolescents and for pregnant women.

CONTRAINDICATIONS

Unfortunately, some practitioners see

Figure 1

ECT technique: electrode placement, stimulus energy, waveform, and frequency of treatment



ECT, electroconvulsive therapy; LART, left anterior, right temporal.

ECT as a treatment of last resort, whatever the presenting diagnosis. This kind of thinking on the part of referring psychiatrists and patients is discouraged. There are relative contraindications for ECT, but few absolute ones.

Electrically induced seizures can be associated with transient cardiovascular changes (asytole, bradycardia, tachycardia, hypertension, increased myocardial oxygen demand), increased intracranial pressure, increased intraocular pressure, release of catecholamines, and other effects that can exacerbate preexisting conditions. These conditions require optimization of their management and/or procedural changes to minimize morbidity and mortality.

Precautions must be taken in patients with unstable or severe cardiovascular conditions (eg, recent myocardial infarction, unstable angina, congestive heart failure, uncontrolled hypertension), aneurysm, increased intra- cranial pressure, recent cerebral infarction, or pulmonary conditions (eg, chronic obstructive pulmonary disease, asthma, pneumonia), and in patients who fall into the American Society of Anesthesiologists rating level 4 or 5.

The mortality rate has fallen over the decades of ECT practice. The number of deaths resulting from ECT is estimated to be 1 per 10,000 patients, or 1 per 80,000 treatments. The Veterans

Affairs (VA) National Center for Patient Safety database estimate is less than 1 death per 73,400 treatments (or less than 1 per 14,000 patients). No associated deaths were reported in any VA hospital between 1999 and 2010. In California from 1984 to 1994 and in Texas from 1993 to 1998, there were less than 2 deaths per 100,000 treatments.

TECHNIQUE

The mainstays for the reduction of adverse effects include electrode placement, stimulus energy, waveform, and frequency of treatment.

ELECTRODE PLACEMENT

The two best-studied placements are right unilateral (RUL) and bilateral (BL, also known as bitemporal and bifrontotemporal) placements. RUL ECT has fewer cognitive adverse effects than BL ECT; however, unless it is administered well above seizure threshold, RUL ECT is less effective (double-blind randomized trial, P = .054). Suprathreshold RUL ECT can approach the efficacy of BL ECT. In one study, the response rates of both were 65%. In another study, in which patients were randomized to titrated moderate supra threshold or fixed high-dose RUL ECT, the response rates were 39% and 67% respectively.

ECT practitioners must often choose between maximizing effectiveness and minimizing adverse effects. This prompts some practitioners to use BL placement, frequently at high stimulus energy, almost exclusively. Other start with RUL and switch to BL placement if there is a lack of response generally after 4-6 treatments. The APA guidelines sugg4est reassuring, the indication for continued ECT and making a change in implementation after 6 to 10 treatments if the patient has slow or minimal clinical improvement. These changes could include switching electrode placement from RUL to BL, increasing stimulus energy, and augmentation strategies.

Semkovska and colleagues performed a meta-analysis of 39 studies (1415 patients) to clarify the advantages of RUL relative to BL, stimulus intensity, and time interval between final assessment and cognitive assessment. They found that cognitive differences associated with electrode placement and stimulus intensity were limited to the first 3 days after unilateral treatment: effect sizes ranged from ?1.10 to ?0.21 (95% confidence interval [CI], ?1.53 to ?0.67, ?0.40 to 0.01, respectively).

Within this time frame, there were fewer cognitive disturbances compared with BL placement and more compared with higher stimulus intensity. After this subacute period, there were no significant differences between RUL and BL and no remaining significant relationship between stimulus intensity and cognitive performance after RUL ECT. The time interval after final treatment was predictive of continuous improvement in antero-grade episodic memory, autobiographical memory, and executive functioning.

Left unilateral ECT is considered an alternative when RUL is contraindicated,

typically when there is a skull defect. It is also a consideration for left-handed patients, in an effort to avoid stimulation of language centers. However, the lateralization of language centers for an estimated 70% of this group of patients is similar to that for right- handed patients; 15% have bilateral representation; and only 15% are right dominant for language. Handedness may also be an unreliable predictor. While most practitioners use RUL placement regardless of handedness, an alternative strategy is to switch laterality over the initial sessions to gauge which is associated with fewer cognitive adverse effects.

Bifrontal ECT has been investigated as a way to maximize response without increasing cognitive adverse effects (by avoiding stimulation over the temporal region) and directing treatment toward circuits in the frontal cortex that may play an important role in depression. As described by Abrams bifrontal placement was initially side- lined because benefits were intermediate; the electrodes were too close together and there were skin burns secondary to shunting. Interest was renewed in the 1990s with a number of published clinical trials and apparent widespread use.

One showed bifrontal ECT had the best antidepressant (P < .01) and cognitive (P < .05) outcomes and required the fewest treatments, compared with BL and RUL ECT. However, the differential cognitive effects were not evident 3 months after the last ECT.

Bailine and colleagues randomized 48 patients to either BL or bifrontal ECT (stimulus titration at first treatment, subsequent treatments 1.5 times seizure threshold). All 24 patients in the bifrontal group and 23 of the 24 patients in the BL group met remission criteria by the 12th treatment. The BL group had a significant decrease in the mini-mental state score that was considered to be clinically significant, albeit small (P = .03).

The researchers attributed efficacy and improved cognitive performance to avoidance of stimulation over the temporal areas while retaining bilateral stimulation. They also speculated that bifrontal placement may have the advantage of fewer dental complications, since the masseter muscles may receive less direct stimulation.

Kellner and colleagues report the results of a large, double-blind, controlled trial of 230 patients who were randomized to bifrontal placement at 1.5 times seizure threshold, BL at 1.5 times seizure threshold, or RUL at 6 times seizure threshold. Antidepressant outcomes were comparable, with remission rates of 55% for RUL (95% CI, 43-66), 61% for bifrontal (95% CI, 50-71), and 64% for BL (95% CI, 53-75). BL placement resulted in a faster reduction of symptoms, but there were few cognitive differences among the 3 treatment arms. The researchers note that BL placement could be used preferentially in urgent clinical situations. RUL placement can be effective at high stimulus intensity and could be the initial choice if there is concern about retrograde amnesia. Bifrontal placement did not have a cognitive advantage, nor was RUL

consistently better than BL placement on cognitive measures.

One potential advantage of bifrontal ECT is a lower risk of bradycardia and asystole compared with BL and standard pulse width RUL ECT. Bifrontal ECT could be considered for patients with arrhythmia. Ultrabrief pulse width RUL ECT is associated with less bradycardia (P = .004) and asystole (P = .001) than standard pulse width RUL ECT.

Left anterior, right temporal (LART, or left frontal, right temporal) placement seeks to decrease cognitive adverse effects by minimizing the impact on the temporal/hippocampal areas. This placement is less well studied, although in small open and double-blind pilot trials, LART had comparable efficacy and fewer cognitive ad- verse effects.

STIMULUS ENERGY

It is well established that the stimulus energy must be above seizure threshold (suprathreshold) to maximize treatment efficacy. Seizure threshold- the minimum amount of energy to elicit a generalized seizure-is highly variable, up to 40-fold in some estimates. Men, the elderly, and BL placement tend to have a higher seizure threshold. Seizure threshold is also inconstant because ECT has an anticonvulsant effect that raises seizure thresh- old (paired t, P < .001). Treatment protocols vary from titration procedures to fixed-dose strategies because there are different schools of thought about how much above seizure threshold a stimulus should be.

Some practitioners, during the first treatment session, determine seizure threshold by delivering increasingly larger stimuli until a generalized seizure of adequate length (at least 15 seconds as measured by motor activity or electroencephalogram) is obtained. There are a number of methodologies by which this stimulus titration is done. Electrode placement, sex, age, dosages of anesthetics, and concomitant medications can be used to predict seizure threshold. However, seizure threshold typically increases over the series. Proxies such as seizure length, seizure quality, and clinical response are signs that stimulation is no longer suprathreshold and stimulus intensity should be increased.

Seizure threshold increases with age; thus, a method that sets the stimulus energy based on the patient's age can be used. Because this might cause patients to be stimulated at higher intensity than necessary, some practitioners use the "half age" method. The APA guidelines recommend that unilateral treatments be moderately to markedly suprathreshold (2.5 to 6 times seizure threshold) and that BL treatments be moderately supra- threshold (1.5 to 2.5 times seizure threshold) because the efficacy of BL placement is less sensitive to suprathreshold dosing than is unilateral placement.

There have been calls for higher-output machines in the United States to achieve the dosages required for effective unilateral ECT. However, whatever the placement, higher-energy stimulation is associated with increased cognitive

adverse effects. Manipulation of pulse width may allow for more efficiency in inducing seizures, with fewer cognitive adverse effects.

WAVEFORM

The first ECT apparatus used sine waves, which were inefficient in inducing seizures and were associated with significant cognitive adverse effects. Brief pulse has fewer cognitive adverse effects, without a reduction in efficacy. As a result, the APA guidelines state that the continued use of sine wave stimulation is not justified. However, the ideal pulse width continues to be studied.

The chronaxie, or optimal width for neuronal polarization, is estimated to be 0.1 to 0.2 millisecond. Until recently, most devices had a pulse width range of 0.5 to 2 milliseconds. Ultrabrief pulse width of less than 0.5 millisecond had been considered earlier in ECT history but was not pursue because of concerns about efficacy. Now there is resurgent interest.

Using a double-masked study, Sackeim and colleagues sought to determine whether ultra- brief pulse and RUL placement would minimize cognitive adverse effects but not at the expense of efficacy. Patients were randomized to RUL ECT at 6 times seizure threshold or BL ECT at 2.5 times seizure threshold, using brief pulse (1.5 milliseconds) or ultrabrief pulse (0.3millisecond). **The remission rate was 73% for ultrabrief pulse RUL ECT, 65% for standard brief pulse BL ECT, 59% for standard brief pulse RUL ECT, and 35% for ultrabrief pulse BL ECT (all P < .05 after covariate adjustment). The ultrabrief pulse RUL group had less severe cognitive adverse effects (P < .001).**

Niemantsverdriet and colleagues compared 0.25-millisecond ultrabrief pulse with 0.5-milli- second brief pulse in BL ECT. No significant difference was found between the two groups (P = .947).

Sienaert and colleagues compared ultrabrief (0.3 millisecond) bifrontal ECT at 1.5 times seizure threshold with RUL ECT at 6 times seizure threshold. Of the 64 patients, 78% were responders (n = 50), and 65% (n = 42) were remitters. There was no significant difference in response and remission between the two groups.

The authors caution that ultrabrief ECT may require more sessions (and therefore may produce more cognitive adverse effects), that perceived memory improvement may be a function of improved depression.

Loo and colleagues compared ultrabrief pulse (0.3millisecond) at 6 times seizure thresh- old with brief pulse (1.0 millisecond) at 5 times seizure threshold in RUL placement. If those patients who completed treatment with RUL ECT only are considered, the response/remission rates were 97%/61% for the ultrabrief pulse RUL group and 79%/57% for the brief pulse RUL group.

Patients who received ultrabrief pulse width did require more ECT treatments. However, their cognitive measures were superior, especially on retention (P < .05) and autobiographical memory (P < .01).

Quante and colleagues compared 3 stimulus intensities (4, 7, and 10 times

seizure threshold) of ultrabrief pulse width (0.3 millisecond) in RUL ECT in a randomized, double-blind, pilot study. There was no significant difference in the response rate.

Ultrabrief pulse may be a way to increase the effectiveness of RUL ECT by allowing for suprathreshold stimulation without increasing cognitive adverse effects. It may also allow for lower stimulus intensity in BL ECT, which would decrease cognitive adverse effects while maintaining efficacy.

FREQUENCY OF TREATMENTS

In the United States, index ECT is typically done 3 times a week, usually for 6 to 12 treatments. Some patients have dramatic response to the first treatment and significant improvement after a few treatments, while others require more than 12 treatments to achieve maximum response.

In the United Kingdom and other countries, ECT is done twice a week. Lerer and colleagues found that twice-weekly ECT was just as effective, with fewer cognitive adverse effects (P = .05), although the total number of treatments was higher.

Charlson and associates performed a comprehensive review and meta-analysis of the existing literature to examine the efficacy of 2- and 3-times-a-week treatment schedules. Their findings showed similar efficacy between the two schedules. **With the twice-a-week regimen, the duration of each treatment was longer but the number of actual treatments was lower. Findings suggest that twice-weekly ECT was associated with fewer cognitive adverse effects.**

CONTINUATION ECT

Continuation ECT is done weekly to monthly as prophylaxis against relapse, which can occur as quickly as 1 week after the last index treatment. In a multisite, randomized, parallel, 6-month trial, Kellner and colleagues compared continuation ECT with pharmacotherapy in patients who achieved remission after ECT. In the continuation ECT group, 37.1% of the patients relapsed and 46.1% of the patients maintained symptom remission. In the continuation pharmacotherapy group (nortriptyline plus lithium-a combination shown to be effective in relapse prevention after successful ECT), 31.6% of the patients relapsed and 46.3% of the patients maintained symptom remission. They were statistically equivalent.

Maintenance ECT can be used long-term to prevent new episodes, much as one would continue effective pharmacotherapy.

COGNITIVE ADVERSE EFFECTS

A prominent safety concern about ECT is its cognitive adverse effects, which can include acute/short-term as well as longterm cognitive adverse effects. According to an FDA review, patients can experience postictal confusion that typically quickly resolves, and there has been no evidence of persistent disorientation. Antero-grade memory disturbances typically resolve within 2

weeks. Global cognitive functioning is unchanged or improved within 3 to 6 months compared with baseline-the latter perhaps a function of improved depression.

Retrograde amnesia, both autobiographical and impersonal, is a particular concern. The ability to retrieve historical or factual information seems to return to baseline within 6 months, but evidence is inconclusive about personal memory.

Retrograde amnesia with ECT appears to be more problematic than anterograde amnesia. ECT produces deficits in both autobiographical and impersonal memory, but both improve after an ECT course. However, some patients are left with residual deficits. Retrograde amnesia is worse with BL ECT and sine wave stimuli.

A seminal study by Lisanby and associates attempted to tease apart the differential effects on autobiographical and impersonal memory. For the cognitive assessments, patients with major depression were randomized to RUL or BL ECT (each at either low or high stimulus energy). The researchers created the Personal and Impersonal Memory Test (PIMT) to elicit memories of events 4 years before assessment and to rate each personal event for its salience. The PIMT was administered to affected individuals and controls at baseline, during the week after ECT, and at 2-month follow up.

The authors noted that it had been thought that ECT had its most negative impact on autobiographical memory. In contrast, they found that deficits were greatest and most persistent for impersonal memory (knowledge about the world and public events), recent events compared with distant ones, and less salient events. As expected, BL ECT produced more profound deficits, especially for impersonal events ($P < .001$).

Sackeim and colleagues also found that BL ECT caused more severe and persisting retrograde amnesia 6 months after acute treatment.

OTHER ADVERSE EFFECTS

Common adverse effects can include headache (including precipitation of migraine headaches), musculoskeletal discomfort (including jaw pain and exacerbation of temporomandibular joint problems), and nausea. There is a risk of injury to teeth and tongue laceration if they are not adequately protected by a bite block. Cardiovascular adverse effects include bradycardia, tachycardia, and hypertension. Infrequent but serious complications include postictal agitation or emergence delirium, takotsubo cardiomyopathy (catecholamine release causes myocardial stunning and a reversible cardiomyopathy), prolonged seizures, and status epilepticus. Rarer still is rupture of the bladder.

The choice of anesthetic agent (eg, methohexital, propofol, ketamine, etomidate, remifentanil) can have a differential impact on adverse effects such as nausea and cardiovascular events without necessarily reducing efficacy. The choice of anesthetic can also influence seizure threshold and seizure length, which becomes an issue when it is difficult to induce an adequate seizure in patients

with high seizure thresholds. If stimulus energy can be lowered, cognitive adverse effects may be minimized.

IMPROVING OUTCOMES

The FDA review estimated an ECT response rate of 78% but found its impact limited to the acute phase of less than 4 weeks. Efforts made to improve ECT outcomes include increasing seizure length with hyperventilation, intravenous caffeine, and switching anesthetic agents. However, seizure length may not correlate with clinical outcome.

The choice of anesthetic agent may have differential effects on cognition. These effects may be a function of impact on seizure threshold, because lowering seizure threshold allows for less stimulus energy to induce a seizure, less energy to stay above seizure threshold, and perhaps fewer treatments as a result of this improved efficiency. Some patients get switched to BL placement, which is associated with more cognitive adverse effects, primarily because of an issue with seizure threshold and seizure length. This is an important consideration if high-dose (either titrated or fixed-dose) RUL ECT can approach the clinical efficacy of BL ECT.

Ketamine has garnered interest as an anesthetic agent, an antidepressant in its own right, and a cognitive enhancer with improved short-term memory after ECT. Although more research is needed, combining ECT with anti- depressants may improve outcomes in some patients.

Sackeim and colleagues undertook a prospective, triple-masked, placebo-controlled study of patients who were randomized to nortriptyline, venlafaxine, or placebo during high-dose RUL or moderate-dose BL ECT. **The patients who received concomitant nortriptyline and venlafaxine had improved remission rates without an increase in adverse effects.**

Nortriptyline improved remission rates by about 15% and had a slight cognitive advantage (except for measures of retrograde amnesia, all other measured cognitive adverse effects were reduced with nortriptyline). Venlafaxine also improved ECT outcomes, although less so than nortriptyline, with a range of no impact to modestly negative impact on cognition.

CONCLUSION

Manipulating stimulus intensity (by lowering seizure threshold), electrode placement (avoiding temporal areas), waveform (ultrabrief pulse width), and frequency of ECT (consider twice a week if there are cognitive adverse effects) and adding concomitant antidepressants may allow improved response as well as decreased adverse effects. However, because ECT is frequently used because of past treatment failure, when conservative implementation fails, providers reasonably become more assertive in all of those parameters. This can increase cognitive adverse effects.

We know ECT is not always effective. The APA guidelines indicate that patients can be considered nonresponders after at least 10 treatments if optimization efforts are not successful. For severe depression, ECT remains a safe and effective intervention.

MIGRAINE AND PSYCHIATRIC COMORBIDITY

Diagnostic and Treatment Issues

By Todd A. Smitherman, PhD and others

Migraine is not a psychiatric disorder, although behavioral factors can critically influence the onset and course of headache episodes. Beginning in the 1950s, the conceptualization of migraine as a psychophysiological disorder by Wolff and others ultimately supplanted the earlier and purely psychogenic or psychopathological view of migraine based on psychoanalytic principles of psychosomatic medicine. Just the same, migraine disproportionately presents comorbidly with a variety of psychiatric illnesses. Identifying and managing comorbid illness is essential and can prove challenging in the treatment of migraineurs.

Epidemiology

Migraine affects approximately 12% of Americans each year. It is 3 times more common among women than men (17.1% vs 5.6%, respectively) and peaks in prevalence between ages 25 and 55. Whereas migraine often manifests as an episodic disorder, migraine is now recognized as a chronic disease that persists or progresses over time as a function of biological and psychosocial risk factors. Depressive and anxiety disorders are recognized as modifiable risk factors for transformation of episodic (fewer than 15 d/mo) into chronic (at least 15 d/mo) migraine and for development of frequent and refractory headache resulting from overuse of acute medications (medication overuse headache [MOH]).

Individuals with migraine are 2 to 4 times more likely to suffer from MDD than are those without migraine. Lifetime rates of MDD range from 22% to 32% among those with episodic migraine and are as high as 57% among those with chronic migraine. Migraineurs also have a 3 to 4 fold increase in risk for bipolar disorder and for suicide attempts, even after controlling for psychiatric history.

Among migraine sufferers, anxiety disorders are twice as common as depression, and migraineurs have a 3 to 5 fold increase in risk for various anxiety disorders compared with controls. Panic disorder, phobias, and generalized anxiety disorder (GAD) are most common among migraineurs, although there is growing interest among researchers and clinicians in comorbidity with PTSD. Rates of depression, suicide attempts, and anxiety are most common among those who have greater headache frequency, those with aura symptoms, and those who seek treatment.

The temporal relationship between

migraine and depression is bidirectional either disorder increases the subsequent risk of developing the other. This bidirectional relationship appears unique to migraine, because depression typically occurs subsequent to onset of other severe headache diagnoses. Some evidence suggests that panic disorder and migraine also occur bidirectionally. However, depression is rarely present without anxiety among migraineurs, and among those with both diagnoses, the anxiety disorder typically predates depression. The patterning of these relationships indicates that depression and anxiety observed among migraineurs are not merely reactions to living with this chronic pain condition.

Impact of psychiatric comorbidities

Psychiatric comorbidities are relevant clinically because they negatively impact the patient and complicate clinical decision making. Com-pared with migraineurs without a comorbid disorder, those with psychiatric comorbidity incur $5000 to $7800 more in yearly medical expenses, experience greater headache-related disability pertaining to daily role functioning, and have poorer quality-of-life and coping skills. In many cases, the comorbid disorder perpetuates a preoccupation with somatic sensations and fear of pain, in turn fostering avoidance of situations and behaviors that are actually unrelated to migraine.

Clinicians often expect the presence of psychiatric comorbidity to portend a poorer migraine prognosis. Clinical studies that have examined psychiatric comorbidities as prognostic factors are few and their findings are somewhat mixed. Nevertheless, most researchers and clinicians continue to maintain that psychiatric comorbidities likely will interfere with headache treatment outcomes under a variety of circumstances.

Particularly compelling evidence is provided by a longitudinal study that found the presence of psychiatric disorders (particularly multiple diagnoses) to be predictive of poor headache outcomes. Results demonstrated that 86% of headache sufferers who had 2 or more comorbid psychiatric disorders either had no improvement or had deterioration in their headache condition. Similarly, 62% with a single comorbid condition remained unchanged or worsened. In contrast, the absence of psychiatric disorders was associated with remission of headaches after 8 years.

Comorbid depression or anxiety is a negative prognostic indicator for patient compliance across a wide variety of medical conditions, including headache. Patients with MOH syndromes are particularly at risk for mood, anxiety, and psychoactive substance use disorders, and certain psychological states may play a key role in headache medication overuse (eg, fear of headache, anticipatory anxiety, obsessional drug-taking behaviors, psychological drug dependence). Headache patients with personality disorders (particularly cluster B disorders-antisocial, borderline,

histrionic, narcissistic) are especially likely to be nonadherent. This negative prognosis associated with psychiatric comorbidity emphasizes the importance of psychological assessment and identification of psychopathology among headache sufferers.

Potential mechanisms of comorbidity

Several mechanistic hypotheses might account for the comorbidity of migraine and psychiatric disorders, including serotonergic dysfunction, ovarian hormone influences (for women), and processes perpetuating central sensitization. None of these is likely to be the sole contributor. Central serotonergic dysfunction is the explanation most commonly proffered, because the pharmacological agents of choice for depression/anxiety (ie, SSRIs and SNRIs) and for acute migraine (ie, triptans) are those that increase central serotonergic availability. A lowered serotonergic disposition predisposes migraineurs to cortical spreading depression (the source of migraine aura) and subsequent sensitization of trigeminovascular pathways.

The female preponderance of migraine and affective comorbidities argues also for a strong role of hormonal influences. Precipitous drops in estrogen levels, such as those that occur during menstruation and perimenopause, are linked both to migraine attacks and affective disturbance. Obviously, large declines in estrogen are not relevant for male migraineurs. Among persons with chronic migraine, central sensitization and dysregulation of the hypothalamic-pituitary-adrenal axis also have been implicated. These 3 hypotheses await further research.

Assessment and diagnostic implications

All patients with headache should be evaluated for depression and anxiety. The recommended self-report screening measure for depression is the 9-item depression module of the Patient Health Questionnaire (PHQ-9) and for anxiety, it is the Generalized Anxiety Disorder 7-item scale (GAD-7). The GAD-7 has adequate sensitivity and specificity for detecting not only GAD but also PTSD, panic disorder, and social phobia. A score of 10 or higher on either measure indicates significant symptoms that merit further assessment. Because symptoms such as sleep disturbance, nausea, irritability, muscle tension, and difficulty in concentrating are common to both migraine and affective disorders, differential diagnosis is facilitated by focusing primarily on the core cognitive and emotional symptoms of the suspected psychiatric condition.

In rare cases, headache may be a symptom of a psychiatric disorder. "Headaches secondary to psychiatric disorders" are usually distinguished from migraine by headache occurrence only during active phases of the psychiatric condition. Most commonly, headache occurs as one of many unexplained symptoms in a somatization disorder or

as a delusion during psychosis, such as that occurring as part of a severe major depressive episode, schizophrenia, or delusional disorder (somatic type). In the case of headache as delusion content, the delusion typically centers on the origin of the head pain (eg, alien insertion, undiagnosed brain tumor despite clear evidence to the contrary). Sufferers are usually women in whom numerous standard therapies have failed and who obtain headache relief only after direct and successful treatment of the psychiatric disorder.

If migrainous headache occurs at times other than during the active phase of the psychiatric illness or fails to remit when the psychiatric symptoms abate, then migraine is the appropriate diagnosis. Assessing the temporal patterning of headache in relation to psychiatric symptoms is most valuable diagnostically. Migraine is also the appropriate differential diagnosis if a psychiatric illness worsens a preexisting headache.

Treatment and management

Pharmacological management of comorbid psychiatric disorders may involve administering a single agent for both migraine and the comorbid disorder or using separate agents for each condition. In clinical practice, monotherapy usually is unrealistic because of differing efficacy and dosing profiles by condition. Preventive migraine medications (eg, propranolol, sodium valproate) are indicated for patients with 4 or more headache days per month or significant functional impairment.

Regarding antidepressants, SSRIs are not efficacious for migraine prevention and SNRIs have not been sufficiently evaluated in large controlled trials. The TCA amitriptyline is the only antidepressant or anxiolytic with strong evidence of effectiveness for migraine prevention, but the dosage required to treat affective disturbance is much higher than that used for migraine and often causes sedation and weight gain. As such, pharmacological management of migraine and psychiatric disorders typically requires separate agents by condition, in which case, the prescribing physician should be attentive to potential drug interactions and consider using a "staggered" initiation.

Acute and abortive medications are indicated for patients with infrequent migraines. Triptans are the abortive agents of choice, and generic sumatriptan remains the most cost-effective option for patients. Other triptans that include additional agents or use alternative delivery systems (ie, sublingual, intranasal, transdermal) may benefit patients who have difficulty with oral or needle-based formulations or experience nausea or stomach upset. Despite the FDA's black box warning, empirical data indicate that risk of serotonin syndrome among patients receiving a triptan and other serotonergic agent (for depression/anxiety) is incredibly low, with most affected patients experiencing mild symptoms that remit on discontinuation of one of the medications.

Other commonly used acute and abortive medications include ergotamine derivatives, opioids, and other analgesics. Continuous opioid therapy for headache should be avoided except as a last resort, particularly for those with severe psychopathology or a history of substance abuse, because opioid use at a frequency of even 2 or 3 days per week can increase headache frequency, render migraine refractory to other treatments, and beget MOH.

Any effective acute or abortive medication (even over-the-counter analgesics) can lead to MOH, but the risk of MOH is highest with opioid analgesics. The most common cause of chronic migraine is opiate overuse, and migraineurs who take opiates 10 or more times per week should be assumed to have MOH until proved otherwise. Patients with MOH often prove highly refractory to headache treatment until they undergo withdrawal from the overused medication. These patients should be referred to a neurologist, preferably a headache specialist, for management of their headache. Because they are also the patients most likely to have psychiatric comorbidity, close collaboration between psychiatrist/mental health practitioner and neurologist is essential for treatment success.

A final treatment option is to supplement pharmacotherapy with behavioral management of migraine or the psychiatric disorder. Over the past 4 decades, behavioral headache treatments (including relaxation training, biofeedback, cognitive-behavioral therapy/stress-management training) have amassed a sizable evidence base that shows improvement rates that are competitive with prophylactic pharmacotherapies for migraine.

The strength of this evidence has led numerous professional practice organizations to recommend use of behavioral headache treatments alongside pharmacological treatments for primary headache. Mild to moderate depression responds equally well to behavioral therapy as to medication. Patients with panic disorder, obsessive-compulsive disorder, and PTSD are best treated with exposure therapy because it is more effective than medication and because benzodiazepine use can function as an avoidance mechanism and lead to addiction.

Mild depression or anxiety among migraineurs often is sufficiently managed nonpharmacologically and improves as headache decreases. Migraineurs with more severe affective distress are likely to require pharmacological management of the psychiatric comorbidity and/or intensive psychotherapy from a mental health provider with expertise in behavioral medicine.

Conclusion

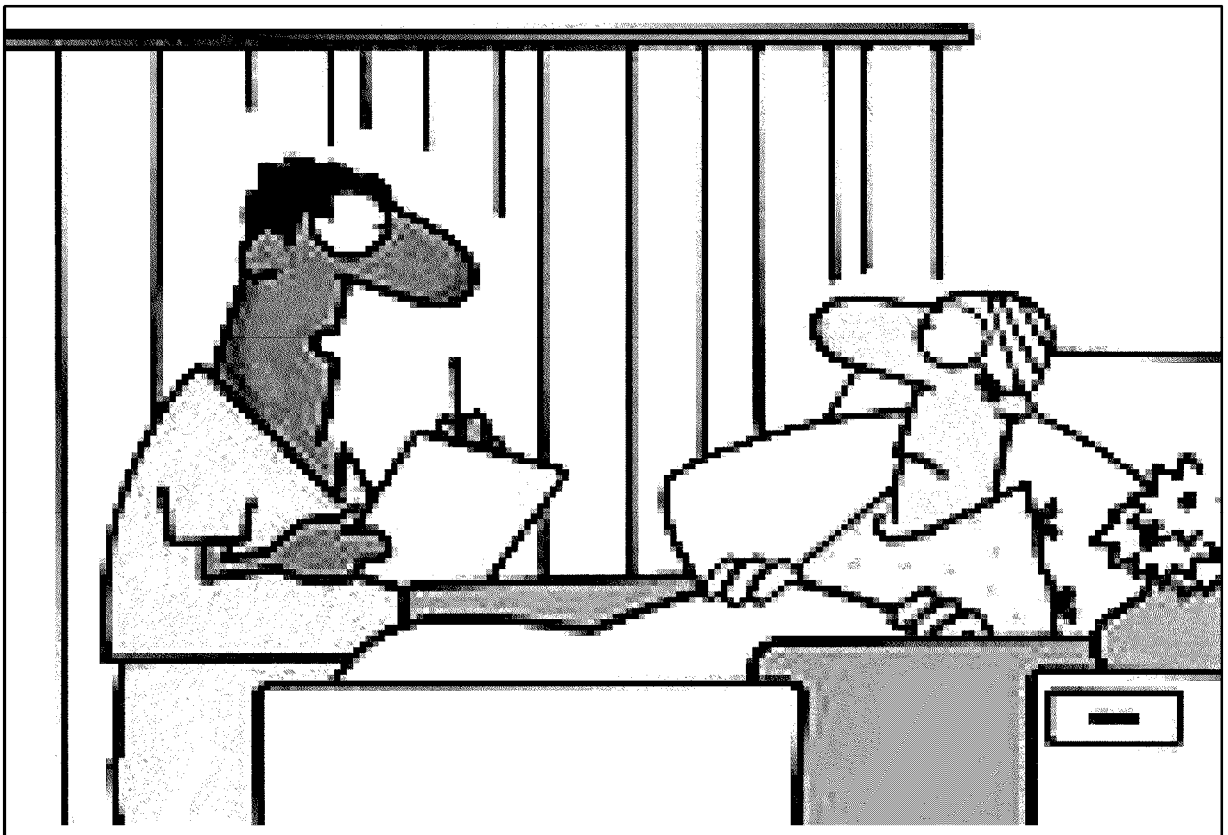
Migraine, particularly chronic migraine, as well as other chronic headaches, have high rates of comorbidity with mood and anxiety disorders. Migraine and psychiatric disorders share underlying pathophysiological mechanisms, with bidirectional, interdependent effects.

Psychiatric comorbidity complicates headache and may portend a poorer prognosis for treatment. Emerging evidence suggests that the psychiatric disorder itself may contribute to transformation of episodic migraine to chronic and daily headaches.

Effective treatment for comorbid mood and anxiety disorders requires screening headache patients and accurately diagnosing specific psychiatric disorders when present. Many well-validated and relatively simple screening tools exist to facilitate recognition of psychiatric comorbidity and quantification of psychiatric symptoms. Pharmacological interventions that target both headaches and comorbid depressive or anxiety disorders, which often require separate agents by condition, can lead to improved headache treatment outcomes.

---

psychiatrictimes.com/display/article/10168/2122210?pageNumber=4



"You caught a virus from your computer and we had to erase your brain. I hope you've got a back-up copy"

EMPTY WORDS IN PSYCHIATRIC RECORDS: WHERE HAS CLINICAL NARRATIVE GONE?

By Thomas G. Gutheil, MD and Robert I. Simon, MD

Careful medical documentation is a primary factor in patient care, risk management, and liability prevention. Such documentation represents a contemporary record of the flow of patient care; thus, it can be extremely helpful to the practicing clinician and can be an important factor in error prevention.

Unfortunately, although a number of forms of documentation may simulate valid recordkeeping, in actuality these are hollow shells of verbiage rather than appropriate chart content. Here we address some of these problems of meaningless phrasing, empty shells, and template-distorted recording in an attempt to improve clinical documentation for both clinical care and risk management.

The seductive lure

It is important to face the grim realities that underlie the issue of documentation: documentation is hard, boring, time-consuming, and unloved by almost all practitioners. In the here and now hurly-burly of clinical activity, it is hard to appreciate the value and long-term benefits of a solid, useful chart. Thus, it is easy to understand the seductive lure of any shortcut (eg, abbreviations) that promises-no matter how speciously-to lighten the load. While some abbreviations may be well understood and quite useful (MMPI-III is much more convenient than the full title written out), the examples below reveal abbreviations that do not accomplish a valid clinical purpose.

The lure is especially seductive given the striking (perhaps appalling) strictures on the time that doctors are currently allocated to spend with patients; under managed care in the modern era, the 5-minute medication check is not uncommon. The temptation also to compress the note taking can become extremely strong.

Modern documentation occurs in a novel electronic context. The electronic health record (EHR) was promised by the Health Information Technology for Economic and Clinical Health (HITECH) Act of 2009 to "save 100,000 lives a year and $77 billion annually." In reality, no savings have been produced and medical errors have not been reduced nor have lives been saved; indeed, errors have been added. Moreover, since the EHR infrastructure is fundamentally insecure, 513 separate data breaches from 2005 to 2011 have affected more than 20 million patient

records-and this number is limited to those actually reported.7

Empty words and letters

One of the most common documentation shortcuts that offers an illusion of appropriate mental health assessment is the famous triad: no SI/HI/CFS. This, of course, stands for "no suicidal ideation, no homicidal ideation, contract for safety." Although these abbreviations suggest that at least some questions were asked, this handful of letters does not represent an adequate suicide or homicide risk assessment of the patient, especially in an evaluation in which these particular concerns arise. These 7 letters are so deeply ingrained in clinicians that in a recent chart review, we found the statement "continuing suicidal ideation" in the history section but noted, in complete contradiction, "no SI/HI" in the mental status segment.

Another shorthand expression is WNL, "within normal limits." While this abbreviation might be adequate for narrow aspects of a physical examination or certain highly specific laboratory values, it is difficult to attribute any meaning to this acronym when applied to mental status issues that require at least some elaboration. What does it mean to say one's fund of information is within normal limits? How could any clinician possibly identify what is "normal" about particular mental status entries without more specific content? If one could identify "normal," what would it mean? Two variants on this latter topic include "NML," meaning normal, and "unremarkable," perhaps the vaguest of them all.

Life's but . . . full of sound and fury,
signifying nothing.
William Shakespeare

"Pt. left AMA" without further content fails to offer subsequent treatment providers a clue as to the reason: disagreement with the treatment plan, rage at treatment providers, inability to pay, lack of insight into illness? Each of these precipitants and others not listed would be quite important for appropriate responses by subsequent clinicians.

Yet another shortcut is NKDA, "no known drug allergies." Although it may be reassuring to indicate that no allergies are known, this acronym does not answer the question of whether the issue was actually raised by the interviewing clinician or whether it is merely a statement of that clinician's ignorance of a potentially important clinical factor.

Another regrettably generic phrase is "by history." This phrase is often used to refer to past significant events of a clinically relevant nature, such as past suicide attempts, incarcerations, and involuntary hospitalizations. Because it lacks any reference to a particular source, it is often unclear whether "by history" refers to the history that the patient is providing by self-report or whether the datum is recorded in some medical documents; if the latter, what

are those documents?

According to DSM, the abbreviation NOS, "not otherwise specified," represents a presumably appropriate inclusive attempt to describe a patient whose complex and idiosyncratic presentation offers a diagnostic pattern that simply fails to fit into the convenient pigeonholes provided by the diagnostic manual. In some charts, the abbreviation suggests that the writer of the entry cannot be bothered to be specific or to commit to a particular diagnosis. We have heard a story of a forensic psychiatrist who when testifying as an expert witness in an insanity context, refers to the diagnosis of all his examinees as "psychosis NOS" in order to forestall being cross-examined on the listing of necessary diagnostic features from the manual.

A puzzling entry often seen in the nursing notes in assessments in psychiatric inpatient charts is the comment: "skin warm and dry." Neither the psychiatric nor mental status significance of this entry is clear, but it may merely suggest that the nurse in question has just transferred from a medical-surgical setting and has internalized this rote phrase from frequent usage in that earlier context.

"Patient pleasant on approach" begins a number of nursing notes that we have seen. "Pleasant" is sufficiently vague to embrace everything from cooperativeness to frank hypomania; but does "approach" mean seeking out the patient for an evaluation or passing within the patient's social orbit in a random encounter? Or does it refer to the patient approaching staff?

"Non-contributory," as in "family history non-contributory," gives the illusion of performing some kind of "rule-out" function but is sufficiently generic not to be useful. Can one seriously maintain that a patient's family history does not contribute in any way to the patient's condition, symptoms, or psychological makeup? Here again, the value judgment inherent in that term should have been replaced by a narrative about what the history actually revealed.

Note that the expression "rule out," which indicates a task to be done in the future, is often misinterpreted by attorneys as a statement about the past, namely "that diagnosis has been ruled out." Some clinicians suggest using "consider" as a preferable alternative

Finally, in addition to empty words, one can find a chart with no ending: even after years of treatment notes, the record simply stops-blank. Did anyone notice that the patient was no longer coming, or was this an agreed-on (although completely undocumented) termination? If the former, were any efforts made to reach the patient? Was the patient transferred? Did the patient die?

Little boxes

Psychiatric records depend on narratives to provide a living and comprehensive picture of the patient. This goal is

thwarted by the increasing use of checklists and simplistic templates that leave record notes stripped of personal meaning and context. Some authors suggest that the very appearance of this template-driven recoding is influenced by the DSM itself, which promotes a checklist approach to diagnosis.

Such devices are a mixed blessing: on the one hand, they may act as prompts to ensure that certain questions will be asked and certain topics considered; on the other hand, in the common form of a chart page teeming with many, many tiny checkboxes, such devices completely obscure any narrative picture of the patient. In addition, a box or two may be unnoticed, and thus a number of important areas of inquiry are overlooked. Furthermore, the template designer may not be a clinician, and therefore a clinically important area may simply have been omitted. If some element is omitted, the computer blindly responds and fills in the blanks, often redundantly, as seen in the following true example: PSYCHIATRIC HISTORY: History of depression, history of anxiety. History of anxiety, history of depression. No psychiatric history.

Another major fault of some computer-generated templates is their tendency toward redundancy, repeating some or all previous entries. For example, a note on the fifth hospital day is preceded by all the notes of the previous 4 days and the sixth day's note has the previous 5. Keeping pace with the evolution of the clinical case becomes increasingly daunting; simultaneously, the redundancy fails to allow the clinician to obtain an evolutionary picture of the patient as a person undergoing changes.

Other forms of redundancy also appear: Social history: Denies abuse, denies tobacco abuse, denies drug abuse: patient smoked tobacco but quit at least 10 years ago. Patient consumes alcohol socially; lives at home with family. Denies alcohol abuse, denies tobacco abuse, denies drug abuse. Patient smoked tobacco but quit at least 10 years ago. Patient consumes alcohol socially; lives at home with family.

Documentation remains very important, but practitioners should avoid deluding themselves into thinking that checking off forms and boxes (although sometimes necessary) constitutes adequate and sufficient documentation. This caution should be heeded by all clinicians and taught to trainees in all the relevant disciplines.

Risk management implications

The core risk management issue is the fact that stock entries usually suggest that the proper assessment or intervention was not done, especially when unaccompanied by narrative that supplies the necessary context. It is too easy for a fact-finder or subsequent decision maker-be it in a liability claim, a board of registration complaint, or an ethics complaint-to see the stock

phrases as indices of a rote mentality that fails to take into account the individual clinical needs of the particular patient in question.

One of the most important risk management functions of documentation is capturing the use of clinical judgment: exercise of clinical judgment is an antithesis of negligence. But judgment cannot be captured through a host of checkboxes, especially when the checklists are designed primarily for recording individual symptoms or signs rather than complex mental processes.

If writing abbreviated notes is such a bad idea, is there really a need for documentation? Lawyers sometimes take the position that "bad documentation is worse than no documentation." While one can understand the lawyers' perspective and their concerns about what to bring to court, clinicians need to remind themselves that documentation is not primarily for lawyers: the main concern is always the direct effect on patient care. If the clinical picture of the whole patient is absent or blurred, care suffers.

Narrative in psychiatry

At least hospital has eschewed the use of an electronic medical record for psychiatric emergency admissions. In part, this move is justified by the fact that the existing data entry system allows only a few computer characters to record historical information. For psychiatric patients arriving at the emergency department, there was simply not enough space for narrative entries. A new version of the entry system allows narrative to be included for these patients.

Historically, psychiatric material has been presented in narrative form. Similarly, patients tell us their histories in the same format-their narratives provide the chronology of their experiences as persons. Moreover, the vocabulary, syntax, grammar, and associative connections in the patients' own stories tell us far more about them as persons than a simple listing of the facts and events that mark their life experiences.

While today the definition of a formulation has taken various forms, an old version popular in Boston was the triad of premorbid personality, external precipitating stress, and type of reaction. Although this model leaned heavily on an event-based theory of the emergence of psychopathology, it contained a narrative vision of what the patient experienced and afforded a holistic view of the patient.

A narrative framework is still needed to capture and convey what a patient is all about. This model should be taught as part of clinical training in all disciplines. Since writing clinically relevant narratives is not an inborn skill, and since all narratives are not uniformly useful in providing a valid picture of a patient, composing clinically meaningful narratives ought to be a seminal component of all training programs.

---

http://www.psychiatrictimes.com/display/article/10168/2020927

WHY PANIC ATTACKS ARE NEARLY ALWAYS PATHOLOGICAL
PANIC ON THE PRECIPICE (PART 1): DOES "CONTEXT" DETERMINE DISORDER IN PSYCHIATRY?

By Ronald W. Pies, MD

When.....you see someone pale with worry.....this man is disordered in his desires and aversions.

-Epictetus, Discourses.

Let's say a patient comes to you with a recent history of a single florid panic attack, in the context of giving a speech before an audience of 2000 colleagues. I don't mean a case of the "jitters"-I mean a 10-minute episode of palpitations, shaking, sweating, choking, dizziness, derealization, and the belief that he is dying. Since you are remarkably empathic, and have had some public speaking anxiety yourself, you think, "I can understand how someone could have an attack like that, under those circumstances.

Let's hold off on suggesting any formal diagnoses (a panic attack is not a formal DSM diagnosis-only a "building block" for one). Was this episode normal and non-disordered anxiety, because it is understandable to you? What about a patient with the identical set of symptoms, in the context of, say, hanging by his fingers over the edge of a cliff? If you can understand the occurrence of a panic attack in this context, was it therefore normal? These may sound like very theoretical questions, but they go to the heart of what we think of as normal or disordered, in both psychiatry and general medicine. How we answer these questions also has important implications for what we mean by the term "false positive" in psychiatry, and what categories we create for DSM-5.

At the last annual meeting of the APA-where I had just spoken on the experiential differences between grief and major depression-a very well-respected senior researcher in the audience rose to comment, evidently quite perturbed. He expressed great surprise at my claim that an explanatory context shouldn't determine our clinical assessment of disorder or abnormality. My critic gave the example of someone who has a full-blown panic attack while hanging by his fingers, over a steep cliff. Surely, he insisted, context is critically important in such a case. After all, the context explains the person's panic

attack, and thus renders the attack non-pathological.

This is a perfectly plausible position, and probably represents the prevailing opinion among the general public. Indeed, many clinicians may be inclined to say, "Hey, I'd have a panic attack, too, if I were hanging by my fingers, over a cliff!" So calling such a panic attack normal is just common sense. Maybe so-but as Einstein once reminded us, "Common sense is the collection of prejudices acquired by age eighteen." Science is the systematic testing of "common sense" assumptions against the range of alternative theories.

In my view, the hypothetical panic attack on the precipice is inherently pathological and disordered. And this "disorderness" -that state in which healthy and adaptive organismic function is disrupted-is not mitigated by any explanatory context. Furthermore, I want to suggest that "explanatory context" is usually a misleading guidepost, in so far as the determination of disorderness is concerned. It leads us to erroneous conclusions in other areas of psychiatry, besides panic attacks, such as whether to regard bereavement-related major depressive syndromes as instances of normal sadness or of bona fide MDD.

And so, I want to suggest that the general concept of disorderness in psychiatry ought to be-with very few exceptions-non -contextual. But before my psychodynamically oriented colleagues recoil in horror, I hasten to add that context is critically important in working psychotherapeutically with patients. After all, psychotherapeutically speaking, there is a world of difference between a severely depressed patient who has just lost a loved one and an equally depressed patient who is being investigated for bank fraud-although, in my view, both are in a disordered state and deserve professional treatment.

The background story: panic on the precipice

Recently, I came across an article that may have been the genesis of my distinguished colleague's "hanging off the edge of a cliff" scenario. In his 2007 review of the book The Loss of Sadness, Dr Kenneth Kendler wrote:

If an individual experience[s] a full-blown panic attack when . . . he looses his grip and falls 40 feet before his rope catches him . . . no psychiatrist I know would consider this to be a psychopathological phenomenon. A panic attack is not-in and of itself-psychopathological. It only becomes pathology when it occurs in certain contexts-at times and in places when it should not. Thus the diagnostic status of panic disorder is inherently contextual. It is not a disorder in and of itself but only in certain contexts. . . .

Later in his review, Kendler alludes to what he takes to be a unanimous consensus among psychiatrists, ". . . our all agreeing that the climber dangling from the rope has a clearly 'understandable' and hence non-pathological panic attack." He then contrasts panic attacks with, for example,

a bizarre delusion, such as, "A hard drive has been installed in my head by aliens. . . ." He regards the latter as inherently pathological. But, regarding the panic attack, is Kendler correct?

On a purely pragmatic view of psychopathology, I believe he is correct. Undoubtedly, no psychiatrist would say to our mountain climber, after his cliff-hanger panic attack, "You need psychiatric treatment. Please set up an appointment with me right away!" Nor would many competent psychiatrists say, "You are likely to need psychotherapy and perhaps medication, given that you experienced this panic attack."

So, in terms of clinical praxis, Kendler is right to claim that the panic on the precipice scenario is not an instantiation of psychopathology-at least in the sense that the term "psychopathology" is typically used in the psychoanalytic literature; ie, as a disturbance of internalized objects, unresolved unconscious conflicts, use of primitive ego defenses, etc. Kendler is also technically correct in noting that panic disorder (like its building block, the panic attack) is "inherently contextual," in the limited sense that DSM-IV and DSM-5 criteria require that panic disorder be characterized by recurrent and unexpected, ie, spontaneous, panic attacks. Unexpected attacks are, in essence, contextless attacks-ones that come on "out of the blue." The clear implication is that there is such a thing as expected panic attacks. In DSM-IV, "expected" implies that the attack is associated with a situational trigger, such as a cue or reminder of a previous trauma-ie, the attack occurs in an understandable context. It's not clear how the framers of DSM-IV or DSM-5 would classify the panic attack in our cliff-hanger scenario. But there are no compelling clinical reasons for viewing a context-based panic attack as non-pathological. Thus, I believe Kendler erred in suggesting that the mountain climber's panic attack was ". . . 'understandable' and hence [a] non-pathological panic attack." The problem is with the use of the word "hence." That an event is understandable does not, by itself, render the event non-pathological. (Kendler, of course, is well aware of this with respect to major depressive symptoms in the context of recent bereavement, and has so argued on the DSM-5 Web site.) Similarly, I believe Jerome Wakefield errs when he comments on the Kendler scenario, arguing that the mountain climber's panic attack "was normal because that is precisely the context in which such intense anxiety experiences were biologically designed to occur."

I know of no empirical evidence that human beings are biologically "designed" to experience panic attacks in any circumstance or context-precipice or no precipice. Nor am I aware of any evidence that such intense anxiety in objectively dangerous situations is somehow advantageous to the human organism. In my view, panic attacks do not demonstrate biological design, but

biology gone awry. We should not confuse anxiety with fear, which is a realistic and adaptive emotion in the face of some objective, external threat-such as a Mack truck heading straight for your car. Unlike ordinary fear, panic attacks do not prepare the endangered person for appropriate defensive action-rather, they usually incapacitate him.

Richard Maddock has pointed out that it is theoretically possible for a patient to meet DSM-IV panic attack criteria with only 4 of 13 possible symptoms (eg, tachycardia, sweating, sensations associated with increased respirations, and a fear of dying). In theory, under some threatening circumstances, these particular fear-related symptoms might be adaptive-but patients with such limited panic symptoms are almost never seen in clinical practice. Maddock notes: "Although the simple DSM-IV definition of a panic attack can capture some adaptive fear or stress responses, I believe this has no bearing on clinical practice" (personal communication, November 25, 2012).

It is erroneous to claim that labeling a panic attack as "pathological" or "disordered" represents a "false positive," if the attack occurs in an understandable context. Indeed, the entire notion of a false positive in psychiatry rests on an unproved ontological assumption-ie, that there exist natural types of disease entities (taxons) defined by necessary and sufficient criteria, against which diagnostic claims may be deemed false. Lilienfeld notes, "Such terms as 'false positives' and 'overdiagnosis' carry no ontological meaning in the absence of a taxon [a genuine category that exists in nature], as they presume the existence of at least some true breaking point in nature."

One possible reason for confusion among contextualists is the overlap in DSM criteria for panic attack with what traditionally has been called the fight or flight response, or the general adaptation syndrome (GAS), first characterized by Selye. Most physiologists would indeed regard the GAS as an evolution-based adaptation to acute stress. But despite some overlapping features with panic attacks (eg, adrenergic activation, tachycardia, increased respiratory rate, sweating), the GAS is a fundamentally different process. For example, the GAS usually lacks such panic-specific features as a feeling of choking, chest pain, nausea, dizziness, fear of going crazy, derealization, or paresthesias-none of which appears adaptive.

Although research is still incomplete, there is reason to believe that the physiology of a panic attack differs from that of the prototypical fight or flight response. Maddock, one of the foremost researchers in the area of panic disorder, notes:

.....panic attacks are dysfunctional, while ordinary fight or flight responses are generally adaptive. From the perspective of physiological data supporting this distinction, one difference immediately comes to mind. In the GAS response, elevated [serum] cortisol is the norm.

However, elevated cortisol is distinctly the exception during panic attacks (personal communication, November 16, 2012).

Indeed, panic attacks appear to share more features with acute coronary syndrome-basically, myocardial ischemia-than with the GAS. Moreover-unlike the GAS-panic attacks predict onset and severity of psychopathology beyond anxiety disorders. Whereas the GAS is adaptive-at least, in its earliest stages-it is far from clear that any panic attack, under any circumstances, is ever normal or adaptive. Insofar as it is experienced as terrifying, crippling, death-dealing, or debilitating, a panic attack is always pathological (from pathos, meaning "suffering") and disordered. That said, a single panic attack does not qualify as a discrete disorder, nor does it merit diagnosis of a specific disease entity. Thus, to diagnose panic disorder, DSM-5 requires additional features, such as recurrent attacks and maladaptive changes in behavior.

The second part of this article further explores the role of context in psychiatric diagnosis.

Acknowledgments-My sincere appreciation to Dr Richard Maddock and Dr Sidney Zisook for their helpful comments and suggestions on early drafts or portions of this essay.

---

psychiatrictimes.com/blog/pies/content/article/10168/2122285

UNDERSTANDING DRUG ABUSE AND ADDICTION

Many people do not understand why or how other people become addicted to drugs. It is often mistakenly assumed that drug abusers lack moral principles or willpower and that they could stop using drugs simply by choosing to change their behavior. In reality, drug addiction is a complex disease, and quitting takes more than good intentions or a strong will. In fact, because drugs change the brain in ways that foster compulsive drug abuse, quitting is difficult, even for those who are ready to do so. Through scientific advances, we know more about how drugs work in the brain than ever, and we also know that drug addiction can be successfully treated to help people stop abusing drugs and lead productive lives.

Drug abuse and addiction have negative consequences for individuals and for society. Estimates of the total overall costs of substance abuse in the United States, including productivity and health- and crime-related cost, exceed $600 billion annually. This includes approximately $193 billion for illicit drugs, $193 billion for tobacco, and $235 billion for alcohol. As staggering as these numbers are, they do not fully describe the breadth of destructive public health and safety implications of drug abuse and addiction, such as family disintegration, loss of employment, failure in school, domestic violence, and child abuse.

What Is Drug Addiction?

Addiction is a chronic, often relapsing brain disease that causes compulsive drug seeking and use, despite harmful consequences to the addicted individual and to those around him or her. Although the initial decision to take drugs is voluntary for most people, the brain changes that occur over time challenge an addicted person's self control and hamper his or her ability to resist intense impulses to take drugs.

Fortunately, treatments are available to help people counter addiction's powerful disruptive effects. Research shows that combining addiction treatment medications with behavioral therapy is the best way to ensure success for most patients. Treatment approaches that are tailored to each patient's drug abuse patterns and any co-occurring medical, psychiatric, and social problems can lead to sustained recovery and a life without drug abuse.

Similar to other chronic, relapsing diseases, such as diabetes, asthma, or heart disease, drug addiction can be managed successfully. And as with other chronic diseases, it is not uncommon for a person to relapse and begin abusing drugs again. Relapse, however, does not signal treatment failure-rather, it indicates that treatment should be reinstated or adjusted or that an alternative treatment is needed to help the individual regain control and recover.

What Happens to Your Brain When You Take Drugs?

Drugs contain chemicals that tap into the brain's communication system and disrupt the way nerve cells normally send, receive, and process information. There are at least two ways that drugs cause this disruption: (1) by imitating the brain's natural chemical messengers and (2) by over stimulating the "reward circuit" of the brain.

Some drugs (e.g., marijuana and heroin) have a similar structure to chemical messengers called neurotransmitters, which are naturally produced by the brain. This similarity allows the drugs to "fool" the brain's receptors and activate nerve cells to send abnormal messages.

Other drugs, such as cocaine or methamphetamine, can cause the nerve cells to release abnormally large amounts of natural neurotransmitters (mainly dopamine) or to prevent the normal recycling of these brain chemicals, which is needed to shut off the signaling between neurons. The result is a brain awash in dopamine, a neurotransmitter present in brain regions that control movement, emotion, motivation, and feelings of pleasure. The overstimulation of this reward system, which normally responds to natural behaviors linked to survival (eating, spending time with loved ones, etc.), produces euphoric effects in response to psychoactive drugs. This reaction sets in motion a reinforcing pattern that "teaches" people to repeat the rewarding behavior of abusing drugs.

As a person continues to abuse drugs, the brain adapts to the overwhelming surges in dopamine by producing less dopamine or by reducing the number of dopamine receptors in the reward circuit. The result is a lessening of dopamine's impact on the reward circuit, which reduces the abuser's ability to enjoy not only the drugs but also other events in life that previously brought pleasure. This decrease compels the addicted person to keep abusing drugs in an attempt to bring the dopamine function back to normal, but now larger amounts of the drug are required to achieve the same dopamine high-an effect known as tolerance.

Long-term abuse causes changes in other brain chemical systems and circuits as well. Glutamate is a neurotransmitter that influences the reward circuit and the ability to learn. When the optimal concentration of glutamate is altered by drug abuse, the brain attempts to compensate, which can impair cognitive function. Brain imaging studies of drug-addicted individuals show changes in areas of the brain that are critical to judgment, decision making, learning and memory, and behavior control. Together, these changes can drive an abuser to seek out and take drugs compulsively despite adverse, even devastating consequences-that is the nature of addiction.

Why Do Some People Become Addicted While Others Do Not?

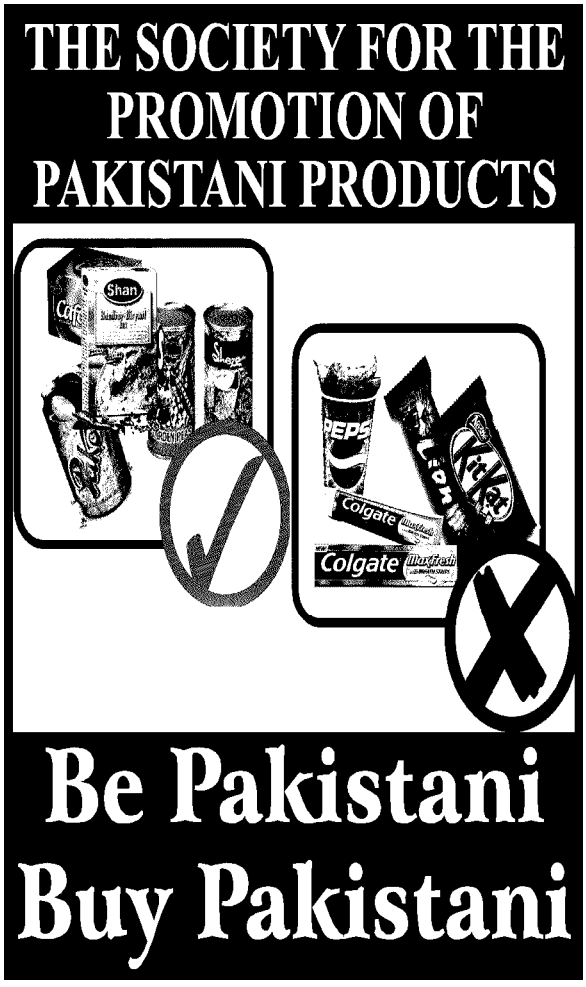
No single factor can predict whether a person will become addicted to drugs. Risk for addiction is influenced by a combination of factors that include individual biology, social environment, and age or stage of development. The more

risk factors an individual has, the greater the chance that taking drugs can lead to addiction. For example:

- Biology. The genes that people are born with-in combination with environmental influences-account for about half of their addiction vulnerability. Additionally, gender, ethnicity, and the presence of other mental disorders may influence risk for drug abuse and addiction.
- Environment. A person's environment includes many different influences, from family and friends to socioeconomic status and quality of life in general. Factors such as peer pressure, physical and sexual abuse, stress, and quality of parenting can greatly influence the occurrence of drug abuse and the escalation to addiction in a person's life.
- Development. Genetic and environmental factors interact with critical developmental stages in a person's life to affect addiction vulnerability. Although taking drugs at any age can lead to addiction, the earlier that drug use begins, the more likely it will progress to more serious abuse, which poses a special challenge to adolescents. Because areas in their brains that govern decision making, judgment, and self-control are still developing, adolescents may be especially prone to risk-taking behaviors, including trying drugs of abuse.

Prevention Is the Key

Drug addiction is a preventable disease. Results from NIDA-funded research have shown that prevention programs involving families, schools, communities, and the media are effective in reducing drug abuse. Although many events and cultural factors affect drug abuse trends, when youths perceive drug abuse as harmful, they reduce their drug taking. Thus, education and outreach are, key in helping youth and the general public understand the risks of drug abuse. Teachers, parents, medical and public health professionals must keep sending the message that drug addiction can be prevented if one never abuses drugs.



PATIENTS' AND DOCTORS' VIEWS ON DEPRESSION SEVERITY QUESTIONNAIRES INCENTIVISED IN UK QUALITY AND OUTCOMES FRAMEWORK: QUALITATIVE STUDY

Dowrick C, Leydon GM, McBride A, et al - BMJ

Brief screening scales should be a clinician's best friend, according to Kurt Kroenke, MD, Professor of Medicine at Indiana University School of Medicine. Medical colleagues routinely monitor patients with sphygmomanometers, peak flow meters, and glucometers, so should psychiatrists incorporate the use of screening tools in their office to help with diagnosis and treatment management, Kroenke told attendees of the 2012 US Psychiatric & Mental Health Congress.

The benefits do not end there, Kroenke added. In addition to their clinical utility for physicians, patients appreciate scales in psychiatric settings.1 For instance, patients in one study said they felt screenings served as "efficient and structured supplement to medical judgment." Patients also said the screenings were evidence that their physicians were taking their problems seriously through a full assessment.

Kroenke shared several basic screening scales for psychiatric disorders that have proven to be effective. The Patient Health Questionnaire (PHQ-9) and a variety of other mobile-ready clinical scales are available on Psychiatric Times' website at http://www.psychiatrictimes.com/clinical-scales.

PHQ-9

Validated in about 10,000 patients, the PHQ-9 screens against 9 DSM criteria for major depressive disorder. It is a self-administered and brief scale. The PHQ-9 can help with probable diagnosis and can help rule out bipolar disorder, acute grief, and organic issues. It is available in more than 80 different languages, and it has been incorporated into professional guidelines. Similarly, all 16,000 nursing homes that participate in Medicare utilize the PHQ-9.

Based on the 9 brief questions, patients can receive scores ranging from 0 to 27. Patients who score between 5 and 10 are considered to have mild depression. Those who score more than 10 have moderate depression. A score of 15 to 20 is indicative of moderate to severe depression, and a score of more than 20 indicates severe depression.

To avoid medicating when it is not necessary, Kroenke uses the score value as guide in determining when to initiate pharmacological treatment. He said he treats patients who score 15 or higher. If the patient's score is between 5 and 9, he monitors the patient, but he does not provide medication. If the patient's score

sits between 10 and 14, he will consider initiating pharmacological treatment on a case-by-case basis.

The PHQ-9 can also help clinicians monitor patient improvement on medications. If a patient's score drops 5 points, Kroenke considers the patient to have significant improvement. For response, he is looking for a reduction in score by about 10% or a score of less than 10. If the score drops to 5 or less, he considers the patient to be in remission.

The PHQ-9 has been studied in special populations, including adolescents, postpartum depression, and geriatric depression, and was found to be comparable or better than its population-specific competitors, Kroenke said. For geriatric populations, for example, it performed better than the Geriatric Depression Scale, he noted. Information on the PHQ-9 can be found at http://www.psychiatrictimes.com/clinical-scales

GAD-7

The GAD-7 brief screening instrument was developed to optimize accuracy and divergent validity, Kroenke told attendees. It can help detect generalized anxiety disorder, social anxiety disorder, posttraumatic stress disorder, and panic. Probably as good as any other general anxiety screener out there, Kroenke said the GAD-7 is better when used for screening than for monitoring treatment.

PHQ-15

To account for the "triangulation" of depressive, anxiety, and somatic symptoms, Kroenke and colleagues developed the PHQ-15 Somatic Symptom Severity Scale. Patients receive a score ranging from 0 to 30, with 0 to 2 points scored for items such as pain (back, head, stomach, chest, arm or leg), fatigue, sexual problems, and bowel problems. In all, the 15 symptoms account for 90% of non-upper respiratory tract symptoms. The somatic scale captures the majority of items on the other 4 major somatic screeners, Kroenke added.

Other Brief Scales

To help clinicians detect problems with pain or risk for suicide, Kroenke suggested attendees use the PEG-3 and P4 screeners, respectively. The PEG-4 screener asks patients 3 questions that rate pain, the impact pain has on their enjoyment of life, and how the pain affected their general activity. Kroenke noted the PEG-3 performs as well as longer scales.

The P-4 is used as a follow-up to patients who answer positively to the question regarding thoughts of hurting yourself or that you would be better off dead on the PHQ-9. Responses are graded into minimal, lower, or higher risk categories.

Concluding Clinical Tips

Screening tools should be integrated in routine psychiatric care, Kroenke told attendees. He noted it can be used a basis for discussions with patients in addition to indicating when patients need further screening and treatment. He cautioned that while the tools are very useful, they are not the gold standard, and clinicians still need to use their best judgment when working with patients.

---

http://www.psychiatrictimes.com/conference-reports/uspc2012/content/article/10168/2116685

EFFECTS OF EARLY PARENTAL DEPRESSION

By Karen Dineen Wagner, MD, PhD

The adverse effects of maternal depression during the prenatal and postpartum periods have received much attention. Maternal depression has been associated with a wide range of problems in offspring that include deficits in social, emotional, temperamental, and cognitive functioning in childhood that may extend into adolescence. Recent studies have focused on critical periods for maternal depression and have also examined prenatal and postpartum depression in fathers.

Timing of maternal depression

Bagner and colleagues1 conducted a study to determine the period of maternal depression that has the greatest negative impact on a child's behavior. The researchers assessed whether the critical period for adverse effects was during the first year of a child's life (ie, from birth to the first birthday). They were also interested in determining how timing of maternal depression and behavior problems of the child differed by sex.

The study was made up of 175 mothers who had lifetime criteria for depressive disorder. Most of the women had at least 1 major depressive episode before pregnancy and after the first postpartum year. These mothers had completed the Child Behavior Checklist (CBCL) for their first child at some time during the first 12 years of the child's life to assess internalizing and behavior problems in their offspring.

Maternal postpartum depression during the child's first year of life significantly predicted internalizing behavior problems. This association was not found if maternal depression occurred before pregnancy or during the prenatal period. The sex of the child did not influence the outcome. The researchers conclude that maternal postpartum depression during the first year of a child's life is a sensitive period that increases the probability of adverse outcomes for the child.

Paternal depression

The majority of the literature focuses on prenatal and postnatal depression in mothers, and little attention has been given to the incidence of prenatal and postpartum depression in fathers. Davé and colleagues2 examined the incidence of paternal depression and maternal depression in primary care practices. The researchers used a database that included 86,957 mother, father, and child triads. The rates of depression were highest in the first year postpartum for both fathers and mothers. The incidence of depression

(per 100-person years) was 3.6 for fathers and 13.9 for mothers. Younger parents (aged 15 to 24 years), parents with a history of depression, and parents from deprived areas were found to be at the highest risk for depression.

This study demonstrates that there is a high risk of depression for both mothers and fathers following the birth of their children. The researchers suggest that fathers, as well as mothers, be screened for depression.

The rates of prenatal and postpartum depression in fathers was also assessed by Paulson and Bazemore. The researchers conducted a meta-analysis of 43 studies that identified depression in fathers between the first trimester and the first year postpartum. The overall rate of paternal depression was 10.4% during the first trimester and 1 year postpartum. It is important to note that the highest rates of depression (25.6%) were found during the 3- to 6-month postpartum period. A moderate positive correlation (r = 0.308) was found between maternal and paternal depression. The findings draw attention to the high incidence of paternal depression in the prenatal period and in the 1-year period following the birth of the child.

Furthermore, an increased risk of suicide has been found in fathers with mood disorders during the postpartum period. Quevedo and colleagues4 assessed 650 men for suicide risk in the antenatal period and within 30 to 60 days postpartum. The prevalence of suicide risk in the postpartum period for fathers was 4.8%. Compared with fathers without a mood disorder, fathers with postpartum depression and those with mixed episodes were 20 and 46 times, respectively, more likely to be at risk for suicide. The researchers recommend that men who have mixed episodes in the postpartum period be specifically evaluated for suicide risk.

Davis and colleagues5 interviewed 1746 fathers of 1-year-old children in pediatric clinics about their parenting behaviors: 7% of these fathers reported an episode of major depression within the prior year. Depressed fathers were more likely to spank their children in the previous month compared with fathers who were not depressed (41% vs 13%, respectively). The investigators concluded that postpartum depression in fathers may lead to negative parenting behaviors.

Clinical implications

These recent studies highlight the importance of identifying depression in the prenatal and postpartum period for parents. Following the birth of a child, it is critical for clinicians to screen for depression in both mothers and fathers. Identification and treatment of early parental depression may prevent adverse outcomes for their children.

---

http://www.psychiatrictimes.com/mdd/content/article/10168/1890601?GUID=E21FA3C4-1588-4190-85DC-7408FE0917C6&rememberme=1&ts=08012013

DOMESTIC VIOLENCE - ACOG TAKES ON SEXUAL COERCION

By Kathleen Struck - MedPage

Clinicians need to offer "discreet and confidential contraception" to patients who have been coerced into pregnancy by intimate partners because of a "known link" between such coercive behavior and violence. The committee defined reproductive and sexual coercion as "behavior that interferes with contraception use" and said the most common coercive practices included "hiding, withholding, or destroying" contraceptives or threatening a partner in order to force her to become pregnant. These practices are not uncommon, the committee wrote, noting that as many as 25% of teenage girls report that partners "were trying to get them pregnant through interference with planned contraception."

Moreover, the most common cause of pregnancy-related death in the U.S. is homicide and in at least one study, most of those killings were committed by an intimate partner. Although reproductive and sexual coercion are not unusual, interventions do appear to work. For example, the committee said that increased education about intimate partner violence can reduce coerced pregnancies by 71%. And, "women in the intervention group were more likely to report ending a relationship because the relationship was unhealthy or because they felt unsafe."

"Based on this information, healthcare providers should include reproductive and sexual coercion and [intimate partner violence] as part of the differential diagnosis when patients are seen for pregnancy testing or STD testing, emergency contraception, or with unplanned pregnancies because intervention is critical," the committee wrote.The committee offered these five recommendations for ob/gyns:

☆Education ☆Screening ☆Counseling Use of long-acting contraceptives such as IUDs or implants. Inclusion of sexual coercion or intimate partner violence as part of differential diagnosis.

To avoid a controlling partner's insistence at attending exam-room visits, offices and clinics should consider posting reception-room notices explaining that patients are always seen alone, the committee recommended.

The committee cited other health issues related to female reproductive health and violence, such as sexual intercourse starting at an earlier age, substance abuse, sexually transmitted diseases such as HIV, miscarriage and risky behaviors like having multiple sexual partners.

"Integrating assessment and intervention for women who experience reproductive and sexual coercion into standard reproductive healthcare practices can enhance the quality of care and improve reproductive health outcomes," the committee wrote.

---

https://bay002.mail.live.com/mail/#n=663291002&fid=1&

EVALUATION OF THE CLINICAL USE OF MAGNESIUM SULFATE FOR CEREBRAL PALSY PREVENTION

Gibbins KJ et al. - Obstet Gynecol

Intravenous magnesium sulfate given to mothers before preterm delivery greatly lowers risk for cerebral palsy in their offspring, but evaluation of its use outside clinical trials has been limited. In February 2008, Women & Infants Hospital of Rhode Island implemented guidelines for use of magnesium sulfate in women anticipated to give birth at <32 weeks' gestation. Investigators sought to determine how the use of magnesium sulfate for cerebral palsy prevention changed between 4 months before and 3 years after guideline implementation.

During the study period, 373 pregnancies met inclusion criteria. During the 4 months preceding guideline implementation, 20% of eligible women received magnesium sulfate before delivery. In the final 2 months of the study, 94% of such women received magnesium sulfate. Of the women who delivered before 32 weeks' gestation and received magnesium sulfate, 16% in 2007 versus 86% in 2011 were receiving infusions at the time of delivery (a stated goal of the guidelines). No cases of neonatal hypotonia occurred, and no maternal or perinatal harms were reported.

LEVONORGESTREL INTRAUTERINE SYSTEM VERSUS MEDICAL THERAPY FOR MENORRHAGIA

Gupta J et al. -N Engl J Med

Heavy menstrual bleeding is a common problem that often interferes with quality of life. British investigators conducted a 2-year multicenter randomized trial to compare the levonorgestrel intrauterine system (LNG-IUS) with usual medical therapy (tranexamic acid, mefenamic acid, combined oral contraceptives, high-dose progestin, depot medroxyprogesterone acetate, or combinations of these agents) in 571 women (age range, 25-50) who presented with at least three consecutive menstrual cycles having self-perceived heavy menstrual bleeding and no evidence of fibroids or other disorders.

Fully 75% of women in the usual-treatment group received mefenamic acid, tranexamic acid, or both. By 6 months in both groups, scores on a validated quality-of-life measure (the Menorrhagia Multi-Attribute Scale, which measures the effect of heavy bleeding on social life, work routine, and family life) had improved, but significantly more so in the LNG-IUS group (mean increase, 33 vs. 21 points). Moreover, women assigned to LNG-IUS were almost twice as likely (64% vs. 38%) to continue their assigned therapy for the full 2 years. Rates of surgical intervention or of serious adverse events were low and did not differ between groups.

THE ANTISOCIAL BRAIN: PSYCHOPATHY MATTERS: A STRUCTURAL MRI INVESTIGATION OF ANTISOCIAL MALE VIOLENT OFFENDERS

Gregory S et al. Arch Gen Psychiatry

Many people equate the terms psychopathy and antisocial personality disorder (ASPD), but research suggests that psychopathy - lack of empathy, emotional responsivity, and remorse plus continued reactive (impulsive) and instrumental (premeditated) violence - constitutes an additional dimension of ASPD. Presence of psychopathy predicts an earlier onset and greater severity of offending and a poorer response to treatment. Researchers conducted a voxel-based morphometry study of 44 violent offenders with ASPD and 22 nonoffenders without ASPD or psychopathy to identify possible gray-matter differences between these ASPD phenotypes. Seventeen offenders had psychopathy (ASPD+P), and 27 did not (ASPD-P); similar numbers in the offender groups had substance use disorders.

In analyses controlling for age and IQ, the ASPD+P group had smaller gray-matter volumes in the anterior rostral prefrontal cortex and anterior temporal poles than the other two groups, which had equivalent gray-matter volumes. In analyses of the offender groups that excluded individuals with active drug use at scanning or with comorbid personality disorders, the temporal cortex findings remained significant.

http://psychiatry.jwatch.org/cgi/content/full/2012/1001/4?q=etoc_jwpsych&eaf

EFFECTIVENESS OF INTERNET-BASED COGNITIVE BEHAVIOUR THERAPY FOR PANIC DISORDER IN ROUTINE PSYCHIATRIC CARE

Hedman E et al.. Acta Psychiatr Scand

In randomized, clinical trials, Internet-based cognitive-behavioral therapy (ICBT) has shown efficacy for panic disorder. To ascertain whether ICBT might be effective in routine clinical practice, investigators studied 570 consecutively recruited adult patients with panic disorder to ICBT at a Stockholm psychiatric clinic.

Patients taking medication were on stable doses for at least 4 weeks (selective serotonin or serotonin-norepinephrine reuptake inhibitors, 32%; benzodiazepines, 15%). After in-person and online assessments, patients entered a 10-module program of reading plus homework. An online psychologist provided supervision and deemed that a module was completed, allowing the patient to advance to the next module. Modules focused on identifying interoceptive and agoraphobic thoughts and emotions, associated stimuli, and exposure practices. In addition to online access to the therapist, patients could participate in an anonymous discussion group; if necessary, therapists could contact patients by telephone. The closely followed protocol routinely lasted 10 to 12 weeks, occasionally longer.

Treatment was completed by 71% (completion, <5 modules; mean, 7.1). Of patients with post-treatment assessments, 54% reported clinically significant improvement in panic-related anxiety; depressive symptoms also improved, and patient satisfaction was high. At 6-month follow-up, panic was significantly improved in 60% of the 156 patients who were assessed. As the program staff acquired greater experience, rates of patient improvement increased. The pre-post treatment effect size of 1.01 among patients contrasts with the 0.1 to 0.2 values seen in untreated historical control panic-disorder patients evaluated in previous ICBT studies.

http://psychiatry.jwatch.org/cgi/content/full/2013/325/4?

PERINATAL EPISODES ACROSS THE MOOD DISORDER SPECTRUM

Di Florio A et al.. JAMA Psychiatry

All mood disorders tend to recur during pregnancy or the postpartum period. But does the frequency and timing of recurrences vary by type of disorder? To find out, U.K. investigators examined reports of affective episodes during and after pregnancy in 980 women with bipolar I disorder (BDI), 232 with bipolar II disorder (BDII), and 573 with recurrent major depression (RMD).

A similar proportion of women in each diagnostic group (71%-74%) reported an affective episode in pregnancy or within 6 months of delivery sometime during their lives. The risk for a mood episode within 6 weeks of delivery was significantly higher with BDI than with BDII or RMD; more than 20% of pregnancies or postpartum periods in the BDI group were accompanied by mania or psychosis, and 25% were accompanied by depression. Within 4 weeks of delivery, women in the BDI and RMD groups were significantly more likely to experience an episode than those in the BDII group; mania and psychotic depression were most common in the BDI group. In all diagnostic groups, mood episodes were significantly more common during the first month postpartum than during pregnancy. Depression during pregnancy was more common in the RMD group than in either bipolar group.

http://dx.doi.org/10.1001/jamapsyc

Question on addiction, sex, psychiatry or the possession syndromes

First fold here

Second fold here

Remarks about the bulletin

Third fold here

Remarks about the bulletin

_____

_____

_____

_____

* **READERS are requested to send their articles, comments & suggestions.**
* **DOCTORS who desire to get the bulletin or want their colleagues, should send the address, contact number and email address.**
  KPH email: support@kph.org.pk

_____

_____

_____

_____

From

Stamp not necessary if mailed in Pakistan Postage will be paid by addressee

THE EDITOR

KARACHI PSYCHIATRIC HOSPITAL

B-1/14, NAZIMABAD # 3,

KARACHI, PAKISTAN-74600

Staple or stick with tape

Dr. Syed Mubin Akhtar MD. KPH speaking on the occasion of an International Seminar organized by Pakistan Society of Psychiatrists.