

Obsessive-Compulsive Disorder, Comorbid Depression, Substance Abuse, and Suicide Attempts: Clinical Presentations, Assessments, and Treatment

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Persons with obsessive-compulsive disorder (OCD) in singular presentation may experience profound threats to self-esteem; activities of daily living; marital, family, social relationships; and occupational functioning. OCD is present in a number of comorbid conditions, the most risky of which elevate suicide risk. This paper examines the prevalence, assessment methods, clinical features, and treatment approaches to OCD, and depression, substance abuse, or psychotic episodes in comorbid presentation, where threats to the functioning and survival of the individual as well as complications in clinical treatment may arise because of the interplay of symptoms of OCD, substance abuse, brief psychotic episodes, and depression. Case illustrations are used to demonstrate effects of comorbidity and considerations in treatment planning. This article closes by calling for longitudinal research on the causes, consequences, and efficacy of integrated treatment of OCD, depression, substance abuse, and suicidality. [*Brief Treatment and Crisis Intervention* 3:145–167 (2003)]

KEY WORDS: obsessive-compulsive disorder, major depression, substance abuse, suicide ideation, suicide attempts, assessment, cognitive-behavioral therapy, exposure and response prevention, psychotropic medication.

Treating persons with complex comorbid psychiatric disorders is challenging and controversial. The National Institute of Mental Health (NIMH) and the National Comorbidity Survey (NCS) indicate that approximately 2.5% of the population of the United States have a lifetime prevalence of OCD; that almost 14% have a one-year prevalence rate of depression; and that an estimated 7.3% of the U.S. population were binge drinkers, substance dependent, or users of an illicit drug during 2001 (Substance Abuse and Mental Health Services Administration, 2002; Narrow et al., 2000, 2002; NIMH,

1999; Cohen, Kessler, & Gordon, 1995). These three disorders often coexist with overlapping symptoms. The etiology and progression of OCD with comorbid depression, substance abuse, and/or brief psychotic episodes are unknown. This review article documents the limited research and clinical management on OCD and the most common coexisting multiple disorders.

Despite the dearth of research on evidence-based practice with comorbid disorders, North American estimates reveal large percentages of persons with mental disorders suffering from untreated or undertreated comorbid psychiatric

and medical disorders. A specific gap in the professional literature relates to evidence-based assessment and treatment plans for comorbid OCD, major depression, substance abuse, psychotic episodes, and suicide attempts. While drug trials and outcome studies on the effectiveness of a number of the newer antipsychotics, selective serotonin reuptake inhibitors (SSRIs), and anticonvulsant medications have been widely disseminated, hardly any attention has been given to determining the efficacy of combined psychotherapeutic and pharmacological treatment of comorbid disorders. The absence of empirically validated longitudinal data on the psychopharmacological treatment of OCD and comorbid depression, substance abuse, psychotic episodes, and suicide attempts is one of the most crucial missing facets of the clinical research literature.

This article demonstrates that OCD is present in a number of comorbid conditions, the most risky of which elevate suicide risk. We begin with a discussion of OCD and comorbid depression, and we include case illustrations and cognitive-behavioral treatment (CBT) plans. Our second goal is to review the life-threatening nature of the clinician's failure to identify comorbidity and the misdiagnoses related to drug-induced and other types of psychotic episodes or major depression and OCD. We then

examine alcohol, illegal drugs, OCD, and craving vs. obsessional ideations. The next major section focuses on the impact of OCD and comorbid substance abuse, depression, and suicidality. The final section of this article calls for future controlled studies and longitudinal research on OCD and comorbid depression, substance abuse, and suicide attempts.

Mitchell and Pollard (personal communication, February 6, 2003; Anxiety Disorders Clinic at St. Louis University School of Medicine, St. Louis, Missouri) help to put into context the complexity of clinical assessment and management of comorbidity in OCD spectrum, Axis I, and Axis II disorders:

Clinical social workers, psychologists, and psychiatrists treating OCD often encounter comorbidity, especially depression, substance abuse, brief psychotic episodes, impulse control disorders, and personality disorders. Identifying comorbidity under the constraints of managed care requires a delicate balancing act. On the one hand, it is important for dedicated and ethical clinicians to be thorough and appropriate in their selection of diagnostic procedures for providing the most effective diagnostic and assessment methods. Clinicians must weigh the advantages of a diagnostic procedure's ability to detect a disorder against the cost of the procedure. Practitioners must also consider the consequences of misdiagnosis, and the percentage of cases in which a particular diagnosis is likely to be present. It is important that a comprehensive diagnostic evaluation include consideration of medical disorders that can mimic or exacerbate OCD and other psychiatric symptoms e.g. neurological, endocrinologic, and cardiovascular disorders. Each evaluation (inpatient or outpatient) should begin with a comprehensive physical examination, inclusive of medical and neurological tests as indicated. There are sev-

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eral widely accepted instruments for detecting psychiatric syndromes and diagnosing psychiatric disorders that sometimes accompany OCD and other anxiety disorders. But the complexity of assessing and diagnosing these complicated cases requires experienced clinical judgments.

The full range of assessment tools and scales for OCD spectrum disorders are discussed in Gail Steketee and Fugen Neziroglu's assessment article in this special issue.

Careful review of the patient's response to prior treatments and medications should be plotted on a timeline and weighed carefully against symptom management. The reason is that diagnostic procedures continue to provide important sources of data for potential response to future treatment recommendations. Regardless of the caution applied and regardless of the diligence and selection of diagnostic procedures a clinician uses, the possibility of misdiagnosis always exists.

In fact, when a patient's response to therapy is poor or when he has received significantly different diagnoses, the potential contributions of unaddressed comorbidity should also be seriously considered. For example, the signature symptoms of temporal lobe and frontal lobe epilepsy may mimic some of the characteristics of persons diagnosed with poor insight OCD, schizophrenia, and/or personality disorders (Holzer and Bear, 1997; Koran, 2000). Therefore, a neurological workup (e.g., video EEG, MRI brain scan) may be critical in order to rule out neurological abnormalities.

Cases of OCD and Comorbid Depression

Cases of the authors illustrate compromises to quality of life when OCD results in the development of depression and when OCD pre-

sents with depression and OCD spectrum disorders.

Case 1: OCD's Resulting in the Development of Major Depression

Karen L. is a 32-year-old married mother of three, referred by her husband for treatment of OCD. Karen indicated that six months prior to evaluation she had become preoccupied about accidents happening to her children as a result of errors on her part—for example, using spoiled food ingredients that would cause her children to die from food poisoning and permitting her children to play in sports at school that could result in blood exchange with another student who might be HIV-positive. As these worries mounted, she experienced difficulty in falling asleep, early-morning awakening, loss of appetite with a 15-pound weight loss, anhedonia, crying spells, and an inability to concentrate. Karen's husband related that soon after their marriage (10 years earlier), he discovered that his wife would leave the bed to check doors, locks, windows, and stove repetitiously. When she could not disengage from repetitive checking, he volunteered to check for her to lessen her anxiety. Karen's husband related that he "made" his wife pursue psychiatric evaluation because in the week prior to evaluation, she pleaded with him to understand her need to suicide since in her estimation, she was a complete failure as a mother and a wife. Karen was admitted to the psychiatric inpatient shortly after evaluation after she attempted to stab herself with a knitting needle in the stomach while pleading for her husband to suicide with her. After being started on an antidepressant and a neuroleptic (because of mood-congruent psychotic features), she was seen on a longer-term basis in the partial hospitalization program during which time she showed mild to moderate improvement in regard to vegetative symptoms of

depression, obsessional thought, and checking behaviors.

Case 2: OCD with Major Depression and OCD Spectrum Disorders

Ken D. is a 24-year-old single unemployed college graduate, referred by his parents for follow-up psychiatric treatment following a psychiatric inpatient hospitalization for depression. Ken experienced severe symptoms of social anxiety during his grade school years. In late adolescence, he began skin picking. He developed ordering rituals with valued objects in his bedroom, such as cassette tapes, CDs, and Nintendo cartridges. He began to worry that if he did not order his personal objects in “the correct configuration,” his mother, who had successful surgery for removal of a brain tumor, would develop a malignant and aggressively growing tumor that would end her life. For the past several years, he had become preoccupied about abdominal muscle tone, and he exercised several hours a day to ensure that his stomach muscles had a “washboard” appearance. He began to restrict foods, fearing that a fatty food would cause his abdominals to become misshapen, thus leaving him subject to ridicule by others. When Ken could not leave his bedroom because of exercising and ordering, without prompting and pleading by his parents, he lost 20 pounds over a 2-month period and could only question his parents about the shape of his abdominals and about the worries regarding the return of his mother’s brain tumor. At that point, Ken’s parents questioned him about his being depressed. He responded that he believed that he was “valueless” and that he had had periods of strong suicidal ideation but would not kill himself because by doing so, he could not monitor his mother’s health. In the hospital, Ken responded appropriately to a psychoeducational presentation regarding OCD and depression, and psychopharmacological and cognitive-

behavioral interventions. Ken’s progress continued in the course of outpatient care. Ken was able to work on a half-time basis, perform volunteer work, and begin a dating relationship.

Issues in Clinical Assessment and Treatment Planning: OCD and Comorbid Depression

Koran (2000) outlined approaches related to the evaluation of the patient with OCD and comorbid depression. The standard diagnostic interview should encompass chief complaint, history of present illness, screening for comorbidities, past psychiatric history, personal and family history (including history of mental disorders), medical history, medications taken for medical and psychiatric problems, substance-use history, psychosocial stressors, and mental status exam. Koran noted that practice principles used in the treatment of uncomplicated depression may be extended to the treatment of OCD with comorbid depression—that is, rule out organic causes of depression (such as hypothyroidism, nutritional deficits, renal failure, neurosyphilis, effects of medication, and drug and alcohol abuse). The clinician should evaluate suicide risk and thus intervene. Step-by-step strategies for crisis intervention in patients’ presenting in crisis with acute suicidal ideation are explained and illustrated in the *Crisis Intervention Handbook* (Roberts, 2000). Koran encourages the clinician to explore the patient’s personal or family history of mania, hypomania, and substance abuse. He recommends assessment of the patient’s living situation and supports from family and significant others that could be utilized in treatment. Additionally, he recommends that family and significant others, with consent from the patient, receive education regarding the nature and treatment of depression. Koran indicated that when the clinician treats a patient presenting with OCD and comorbid major depression of mild to moderate intensity, the decision to use psychotherapy alone or in combi-

nation with psychotropic medication includes evaluation of nature and extent of intrapsychic conflicts, severity of psychosocial stressors, meaningful supports (which can be utilized in treatment), and the patient's willingness and motivation to pursue different forms of psychiatric treatment (Koran, 2000). For OCD complicated by depression, the Expert Consensus Guideline Series (2000) recommends CBT plus a serotonin selective reuptake inhibitor (SRI) as a first-line intervention; as second-line interventions, CBT alone, CBT plus a monoamine oxidase inhibitor (MAOI), or CBT plus an SRI plus a benzodiazepine (BZD).

In planning treatment of patients presenting with OCD and severe comorbid depression, the clinician may advance the likelihood of the patient's engaging in and remaining in treatment by establishing therapeutic relationship at the onset and by describing to the patient the methods utilized in treatment. Steketee (1993) reviewed ways in which the clinician may explain to the patient treatment of OCD and respond to the patient's questions about treatment of OCD. The clinician does well to recognize that with OCD alone, the patient may be highly doubtful and indecisive about whether treatment could prove effective; with depression, as noted by Beck (1976), the depressed patient may present with the cognitive triad of negative views of self, negative concept of self, and negative appraisals of the future. Koran (2000) advises appropriately that the clinician should advise the patient of the anticipated time frame of anticipated positive medication response: in general, 2 to 4 weeks to discern substantial therapeutic effect, and 6 to 8 weeks with medication at the optimal level for full medication therapeutic effect to become manifest. With chronic major depression, with depressive symptoms lasting 2 years or longer, Koran noted that full remission may take as long as 12 weeks.

In regard to the effect of depression on exposure and response prevention (ERP), Baer

(1992), stated that severely depressed persons may not have the energy for the rigors of ERP when depressed; and that even if the severely depressed individual attempts ERP, the patient might not habituate to the anxiety evocative stimulus, thus negating the anticipated gains of behavioral practice. A study by Basso, Bornstein, Carona, and Morton (2001), using neuropsychological testing, suggests that executive function abnormalities in OCD are related to severity of comorbid depression whereas sensory-motor deficits seemed more consistently related to basal ganglia and orbital-frontal dysfunction in OCD. Abramowitz, Franklin, Street, Kozak, and Foa (2000) studied the effects of comorbid depression on OCD using CBT with and without medication. Patients with pretreatment scores of 30 or above on the Beck Depression Inventory, even on medication, showed less reduction in Y-BOCS scores following ERP as compared to patients with moderate, mild, or no depression. However, and quite importantly, while the severely depressed patients showed a lower rate of improvement as compared and contrasted with less depressed or nondepressed subjects, the 12-point mean reduction on the BDI was still meaningful clinically, thus resulting in the authors' suggestion that ERP be offered to even severely depressed patients.

A third case of the authors illustrated the use of CBT, psychoeducation, and bolstering of supports to help alleviate depression and permit the patient to proceed productively with both ERP and trials of psychotropic medication.

Case 3: Case Illustration of Treatment of Severe Depression Comorbid with OCD

Tom C. was presented to the author as a 38-year-old single unemployed former bookkeeper, referred by the Obsessive Compulsive Foundation, a national clearinghouse for information on OCD and referral for treatment of OCD. At

the time of initial evaluation, Tom noted core depressive symptoms—sleep disturbance, diminished appetite, anhedonia, decreased libido, passive suicidal ideation, and a desire but inability to cry—as having been present on nearly an everyday basis for two years prior to evaluation. His initial score on the Beck Depression Inventory (BDI) was 28. He reported frequent feelings of irritability and anger. He indicated that at age 13 he had developed extensive concerns about dirt, germs, and contamination, as well as related ritual and avoidance behavior, after he believed that he might have been observed masturbating. He said that he showered three hours per day and washed his hands with any change in physical activity or when he thought he might have accidentally come into contact with a pathogen or the body fluid of another person. Ritual and avoidance behavior resulted in his being dismissed from numerous jobs. His initial score on the Y-BOCS was 28. He lived with his parents, who were rather hermetic given their having immigrated to the United States only recently and their not having achieved fluency in English. Tom related that he had a recent medical evaluation that was entirely negative. He denied substance abuse, history of physical or sexual abuse, familial or personal history of mania or hypomania, or any other psychiatric disorders. He commented that he had been seen episodically over the years for unsuccessful outpatient care, with and without medication, which did not result in any change in his level of depression or in any lessening difficulty with symptoms of OCD. He maintained that about 10 years prior to seeing the author for evaluation, he had been tried on medication specifically designed to treat OCD and was instructed in behavioral therapy. He stopped the antidepressant (Anafranil) because of problems with side effects and said that he had limited involvement with ERP, which he viewed as “an unusual annoyance” since he expected that with “the right medication” his difficulties would resolve quickly.

In terms of treatment design, the author explained to Tom the significance of the severity of his depression, which might need to be treated initially with medication, supportive therapy, and CBT, before he might have the motivation and energy to participate in ERP. The author suggested that Tom read *Getting Control* (Baer, 1992) and *Stop Obsessing!* (Foa & Wilson, 1991) and that he attend a local OCD support group. The author advised Tom of the potential value of exercise 3 to 5 days per week at a local health club to help obviate his ruminating in isolation, which appeared to aggravate the symptoms of both OCD and depression, and Tom agreed to do so. The author explained the use of his being seen for reevaluation of medication, despite an initial unsuccessful trial on Anafranil. The author explained to the patient the methods of cognitive therapy for depression as noted by Aaron Beck (1976) and Judith Beck (1995). In CBT sessions for depression, the patient was able to come to recognize how his absolute, black-or-white thinking; his catastrophic, projective thought; his selective abstraction (disqualifying the positive); and his other cognitive errors as noted by Burns (2000) contributed to the creation and aggravation of depressive states. Most important, he was able to see how core perfectionist traits resulted in his having unrealistic expectations for himself and others, in his eventuating in anger at others, and in his view of himself as a failure. Tom was able to develop cognitive corrections and behavioral strategies based on the cognitive corrections that proved more adaptive. As his mood improved, he was able to begin volunteer work, which increased his self-esteem. Despite his having to undergo a washout on the first SRI because of no noted improvement with obsessional thought, Tom was able to remain in treatment and begin a trial on a second SRI.

When Tom noted some improvement in sleep, overall energy level, and appetite, and when his score on the BDI lowered to 16, the author discussed the usefulness of Tom’s beginning ERP,

now that his depression was in better remission. In terms of framing goals for ERP, the author reviewed with Tom his perfectionist expectations, which might exacerbate anxiety in the course of actual physical exposures and might thus result in post hoc negative evaluations following each ERP session. In a focused manner, Tom proceeded with ERP and showed a significant decrease in ritual behavior and ruminative thought, with his score on the Y-BOCS decreasing to 15 at the last point of measurement. Tom began dating and was evaluating his search for competitive employment. He indicated that with the combination of careful titration of psychotropic medication, modifications in depressogenic thought with cognitive therapy, exercise, continuation of ERP, and structure afforded by volunteer work and support afforded by the local obsessive-compulsive disorder support group, he rated his overall improvement "in the 90% range."

Issues in Clinical Assessment and Treatment Planning: OCD, Substance Abuse, and Psychotic Episodes

One of the most overlooked categories is long-term OCD patients (particularly OCD poor-insight group) who have suicide ideation and who are also substance abusers or experience a first-order or street-drug induced psychosis. This group of OCD sufferers usually experiences a high degree of hopelessness about their future; they have tormenting or delusional thoughts of suicide; they are preoccupied with pessimistic thoughts about death; and they encounter heavy stressors. According to Shea (2002, p. 21), an international expert on psychiatric interviewing, suicidal ideation is often triggered by:

- External stress (e.g., serious illness, public humiliation, violent threat by a gang member);

- Internal conflict (e.g., cognitive distortions, psychological impasses); or
- Neurobiological dysfunction (e.g., exogenous toxins such as alcoholism and/or cocaine abuse).

A psychotic episode is defined as a biologically based condition that affects the brain and manifests itself in what appears to be a loss of reality content. It occurs among young adults with a prevalence of approximately 3% of the general population. First-time psychoses are frequently puzzling as to the source of the psychotic episode and therefore difficult to accurately diagnosis. Common types of first episode psychoses are as follows.

Drug-Induced Psychosis The essential features of a drug-induced psychotic disorder are prominent hallucinations or delusions that are due to the direct physical effects of a drug of abuse, a medication, or a toxin exposure. The presence of a drug-induced psychotic disorder versus a primary psychotic disorder can be determined by consideration of the onset of symptoms, course, and other related factors. Specifically for drugs of abuse, sufficient evidence must be gathered from history of use, physical examination, and laboratory findings to document substance dependence, abuse, or intoxication. Differentiation between dependence, abuse, or intoxication is defined within the *DSM-IV-TR* diagnostic criteria for each drug of abuse. It is not necessary for substance dependency to be present for the diagnosis of drug-induced psychosis. Clinicians must also consider whether the current psychotic episode is not better accounted for by the presence of another mental disorder and whether the disturbance does not occur specifically within the course of delirium (American Psychiatric Association, 2000).

Substance-induced psychotic disorders occur within the context of two specifiers: onset during intoxication and onset during withdrawal.

While psychosis occurs within one of the two specifiers, symptoms may persist for weeks following onset. The key differentiating diagnostic factor is that primary psychotic disorders either precede the onset of substance use or may occur throughout periods of sustained abstinence.

Specific drugs that have been associated with the onset of psychotic symptoms include cocaine, amphetamine compounds (including methamphetamine), "club" drugs (including ecstasy), and cannabis. Persons at particular risk are those with a preexisting mental disorder, complicated by an emergent comorbid substance-use disorder. Specifically, mentally ill chemical abusers are at particular risk for onset during intoxication, as in the example of an individual with the diagnosis of OCD, who takes a club drug such as ecstasy to "fit in" with his or her high school crowd. In this case, the amphetamine compounds may establish a negative interaction with any psychotropic medication on board (e.g., SSRIs such as Prozac or Luvox); in addition, amphetamine compounds can trigger an intoxication-based psychotic disorder in persons predisposed to mental illness. On the other hand, those using cannabis over extended periods of time may mask the extent and severity of their OCD. Once cannabis use is ceased over an extended period of time, the full impact of cannabis on emergent psychotic symptoms may become evident and complicated as an individual withdraws from cannabis.

Drug-induced psychosis frequently takes place when a person with a concurrent mental or neurological disorder abuses substances such as amphetamine-based or amphetamine-like substances. Experimental use of mood-altering substances can produce a drug-induced psychotic episode within minutes, and the individual may experience hallucinations or delusions that they believe are a part of the drug experience; however, the delusions and hallucinations may progress to produce extremely frightening paranoid responses. In this case, the per-

son may become overwhelmed and may attempt to self-harm in an effort to control the emergent psychotic episode. At times, well-intended attempts of individuals to intervene may exacerbate the hallucinations by overstimulating the individual experiencing an emergent psychotic disorder. (For more information, see www.crisisinterventionnetwork.com.)

The Neurophysiological Mechanism. Methylendioxyamphetamine (MDMA), commonly referred to as "ecstasy," is an amphetamine compound combined with lysergic acid diethylamide (LSD) that has become increasingly popular with the young adult population within the United States. The psychostimulants (both licit and illicit) are sympathomimetic amines with both peripheral and central nervous system stimulant actions. These actions are controlled primarily by noradrenergic and dopaminergic neurons: increased neurotransmitter release, reuptake reduction, and monoamine oxidase inhibition are all considered to be contributing factors to the effects of these drugs (Seiden, Sabol, & Ricaurte, 1993; Zickler 2001a).

Exact contributing mechanisms of the stimulant drugs are not fully understood. Current understanding indicates that stimulants including methamphetamine stimulate the release of dopamine into the ventral tegmental area (VTA) of the brain (Zickler, 2001a). Drugs such as cocaine block the reuptake of dopamine resulting in a mass accumulation of dopamine at the synaptic cleft, which results in increased sensations of pleasure for some and possibly the emergence of psychotic symptoms for those predisposed to mental illness (Zickler 2001b; Buffenstein, Heaster, & Ko, 1999). Psychotic symptoms are well-known side effects of all stimulant medications. Caplan and Tanguay (1991) reported that medications such as pseudoephedrine and methylphenidate (MPH) utilized for the treatment of attention-deficit hyperactivity disorders (ADHD) have caused psychotic symptoms

in children who received therapeutic dose ranges. Both tactile and visual hallucinations have been present among children receiving therapeutic doses of amphetamine compounds for the treatment of ADHD (Lucas & Weiss 1971; Spensley & Rockwell 1972; Bloom, Russell, Weisskopf, & Blackerby, 1988).

More recently in Japan, characteristics of methamphetamine abusers were tracked to determine differences in progressiveness of symptoms in various route distributions of the drug—for example, smoking versus intravenous drug use. In all cases, differences in overall occurrence of psychotic symptoms were noted as smoking methamphetamine led to more rapid loss of control and early onset of psychotic symptoms. In this, the third emergence of methamphetamine epidemic use within Japan, researchers are linking the impact of amphetamine compounds on emergent psychotic symptoms in an increasingly younger, more vulnerable population of drug users (Matsumoto et al., 2002).

How then is the manifestation of psychosis in persons predisposed to mental illness acted out within the community? The following case example will provide insight into the impact.

Danny is a 17-year-old living with his adult parents in a suburban neighborhood. As a pre-adolescent, Danny spent most of his time playing in isolation, finding that ordering prized possessions such as POGS and game cards seemed to create for him a “right” feeling. He resisted many early social events in junior high school, fearing that he might cause harm by making others ill (e.g., give someone a paper cut which would then become infected, causing the person to become ill or die). As a consequence of mounting anxiety associated with such obsessional thought, he used alcohol to “drink and not think.” Over the past four months, Danny has been experimenting with MDMA, mostly with friends during overnights, when several young males and females are able to gather at each others houses or at a local campground.

One evening, Danny was ordered to return home by his parents shortly after taking two “hits” of ecstasy. Danny’s parents were concerned with his presentation as he returned home. His mother entered the room to speak with him; however, when she attempted to hug him, Danny hit her and began to strangle her. Danny’s father entered the room in time to stop this event. Shortly after doing so, Danny became increasingly paranoid and ran to the kitchen grabbing a 10-inch butcher knife. When Danny’s father attempted to intervene, Danny stabbed him in the lower right quadrant of his back. Realizing what he had done, Danny turned the knife on himself, stabbing himself in the stomach three times, before losing consciousness.

Following surgery, Danny was admitted to a local psychiatric facility. He tested positive for amphetamine compound but denied use. In gathering history, the social worker indicated that Danny had been diagnosed as ADHD as a child and had been prescribed amphetamines for a brief period of time; however, the prescription stopped as a result of their leading to the presence of tactile hallucinations. A second trial on amphetamine-based ADHD medications resulted in a brief psychosis four years earlier.

Examination of Danny’s mental status revealed prominent paranoid delusions. Routine neurological workup documented no brain abnormalities, and a serologic test for hepatitis virus and HIV yielded normal results. Initial treatment included Haldol (5 mg) and Atavin (2 mg). However, as time progressed, Danny transitioned to an SSRI to which he responded positively. Cognitive therapy focused on enabling Danny to understand that his ego dystonic thoughts of causing harm experienced in childhood and adolescence were symptoms of OCD qua neurobiological disorder and the risk posed by any further drug experimentation. After approximately 30 days in the psychiatric facility, Danny was released to face criminal charges.

In the case of Danny, recovery is slow to come. For many, in general, the process of returning from such a traumatic event is slow and difficult. In this case, as in many, ongoing management is one of ensuring medication compliance and engagement within treatment processes that facilitate personal growth and development. The same is true for Danny's family. Danny's father indicates that this experience shook him to the core of his existence. Family therapy has continued since the family has been able to be reunited. However, this did not occur immediately. Danny served 6 months of a 15-year sentence for felonious assault. Danny's family was shocked to find that the district attorney chose to proceed with charges to facilitate Danny's recovery within a safe environment. Danny spent the 6-month period within a minimum-security psychiatric facility. During this time, he began prevocational training and has progressed nicely through the process. He is now in preparation to enter into a 2-year computer programming certificate program. Danny continues in a weekly support group and sees his social worker weekly and his psychiatrist one time per month for medication management. Danny's greatest complaint is the ongoing lack of socialization skills, and he continues to work in group and individual sessions to become comfortable enough to "fit in."

Within the context of cocaine dependence, tactile hallucinations are not unusual nor is the presence of cocaine paranoia. However, these symptoms of acute cocaine intoxication do not meet the criteria for a cocaine-induced psychotic disorder because symptoms occur within the context of the direct effects of cocaine (Yeager & Gregoire, 2000). Therefore, the paranoia of cocaine addicts' peering out of windows, searching for the police, crawling on the floor, searching for "chips" of "rock," or scratching at the tactile hallucinations referred to as "crack bugs" do not account for the cocaine-induced psychosis, as demonstrated within the next case example.

Zach is a 22-year-old married Caucasian male who has been using crack cocaine in episodic patterns for the past 3 years. Zach is self-employed and has worked as a motorcycle mechanic since age 16. Zach displays traits of obsessive-compulsive disorder: excessive preoccupation with detail, perfectionism, conscientiousness, working long hours to maximize productivity, hoarding money to hedge against future disasters (to the exclusion of involvement in leisure pursuits). Before opening his business, Zach had developed in mind elaborate specifications of his role as breadwinner as well as complementary functional supportive behaviors to which his wife would acquiesce. Zach's attention to detail, scrupulous morality, and willingness to work additional hours to complete assigned work projects had been valued by former supervisors and customers. Zach has just recently opened his own motorcycle custom shop and is doing quite well financially. One weekend in the slow season, Zach decided to binge on crack cocaine. The binge was prompted by anxiety related to worries that with the recession in the economy, his slow season would extend indefinitely, and his efforts to prepare safely against future disasters by saving money would prove inadequate. He came to fear that he might have made a wrong decision by starting his own business, and he used crack cocaine in the attempt to neutralize his anxiety. That night and the following early morning hours, Zach became increasingly suspicious that his wife had been cheating on him. Zach began to test his concerns of his wife's suspected unfaithfulness by trailing her and spying on her over the next three days.

Eventually, Zach saw his wife with another man. He approached her and the man with a loaded .357 Magnum and interrogated them at gunpoint. The man was a counselor who was meeting with Zach's wife to plan an intervention in hope of bringing Zach to a local treatment facility. While experiencing a cocaine-induced psychosis, Zach was able to understand the implications of his behaviors. He surrendered the

weapon without incident and was admitted to a local treatment facility where he remained an inpatient for a period of 10 days. Zach's psychosis responded quickly to a combination of a low dose of second-generation antipsychotics and SSRIs. Zach became an active member of the local Cocaine Anonymous (CA) community but swears he will never participate in an intervention.

Case Autopsy. Zach, like many cocaine-dependent individuals, never dreamed that he would lose control over his use. Unfortunately, Zach not only lost control of his use; he lost control of his mind for a brief period of time as well. Zach reports little recollection of the events associated with his psychotic episode, and he has experienced no long-term ill effects of this event. Zach continued on the SSRIs for a 9-month period, then discontinued the medication. Initially, Zach was informed that simply stopping the medication would not be a problem; however, when he did so, he experienced remarkable agitation, irritability, and mood swings. He then consulted with a psychiatrist who agreed to slowly taper the medication.

Zach's largest complaint regarding the medication was the sexual side effects, indicating that "it's not what you would think. I experienced what the physician called prolonged ejaculation. My doc laughed and said none of his patients had ever complained about this side effect. Well I did, it ruined everything." Zach has been off medication for nearly a year. He has not experienced any remarkable setbacks, other than falling off his Harley Davidson and breaking his arm. Zach was careful to inform his physicians of his dependence and the possibility of negative reactions to medication. His physicians utilized few mood-altering substances and transitioned Zach to non-mood-altering pain management as soon as possible. Zach remains active within the Cocaine Anonymous community. He is the chairperson of the Saturday night group. He sponsors two newcomers and vows, as long as he is alive, that he

will not plan or participate in an intervention, even though he believes this intervention saved his life.

Alcohol, Drugs, OCD, and Craving For decades, there have been arguments and speculation regarding the origins of and appropriate sequence for the treatment of concurrent alcoholism and mental illness. Many have debated this topic with great zeal and enthusiasm; treatment programming is frequently split with psychiatrists on one side of the room and addictionologists on the other. At the root of this debate is an interesting question. Is alcohol, or drug craving for that matter, an obsession? Thus, is effective treatment for addiction similar to that for obsessive-compulsive disorder? Early within the history of Alcoholics Anonymous (AA), Dr. Silkworth characterized alcoholism in a 1930s letter to Bill Wilson, cofounder of AA. He spoke of alcoholism as an "allergy of the body and an obsession of the mind."

In clinical work with cocaine addicts, one quickly learns that the overwhelming craving for cocaine begins with a thought. This thought is triggered by environmental cues, such as (as the joke goes) a sunny day, a rainy day, a Monday, a Tuesday, a Wednesday, and so on. Regardless of the addicts' overly simplistic understanding of craving, it is nevertheless a remarkably effective approach to understanding craving.

A growing body of knowledge implicates clearly defined pathways among prefrontal lobes, cingulate gyri, basal ganglia, and thalamus as being directly involved with obsessive-compulsive disorder. Brain imaging in persons suffering with obsessive disorder indicate reductions in prefrontal lobe activities and concurrent enhanced basal ganglia activity (Saxen, Brody, Schwartz, & Baxter, 1998). Research in the field of addictions indicate similar neuroanatomical findings (George, Teneback, Bloomer, Horner & Anton, 1999; Volkow et al., 1994). Specifically, Modell and Mountz (1995) documented increased activity in the head of

the basal ganglia-caudate in alcoholics after a drink of alcohol. This consumption increased basal ganglia activity and correlated positively to reported craving for additional drinks, though correlation does not necessarily indicate causality. Within the cocaine-dependent population, dorsal-lateral-prefrontal cortex (DLPC) activity was correlated to self-reports of cocaine craving. Pierce, Reeder, Hicks, Morgan and Kalivas (1998) concluded that direct inputs for cortical areas to the nucleus accumbens are important factors within the "memory of drug effects" as related to neuroadaptation.

This phenomenon is most simply demonstrated by the idea of mood following memory. If one remembers pleasant times from their past, their mood will likely be positive; however, if one recalls a not-so-pleasant life event (e.g., being punished, perhaps severely for something they did not do), their mood will likely become one of depressed or angry feelings. In a similar manner, the effect of neuroadaptation in some may lead to highly reactive responses to stimuli that might induce a craving for substance use. This neuroadaptation and subsequent sensitization may also be linked directly to memory and learning abilities. The same is true with neuroadaptation within certain neuropathways' leading to repetitive thoughts and behaviors in OCD that are very similar to cravings and that may be a major contributing factor to both alcohol/drug relapse and relapse into less adaptive behaviors related to obsessive-compulsive disorder.

If in fact there is the ability to utilize this sensitization in combination with reinforcement of adaptive behaviors, such as the processes associated with cognitive therapy, then effective treatments can be developed that capitalize on neuroadaptation (e.g., basal ganglia and the nucleus accumbens) for therapeutic treatment approaches for both OCD and addiction cravings. Effective interventions may be based in the subcortical/neuronal effects of sensitization and craving; thus, one only need to translate

these neurological-based impulses to terms of thoughts and behavior patterns driven by neurophysiological processes (Robinson & Berridge, 1993).

Let's consider an example for the cocaine addict in treatment. The example must be simple yet effective. For the purpose of this demonstration, we will divide the brain into two sections. First is the brain stem, which is described as the part of the brain that houses the survival instincts as described by Arsenault et al. (2002) as "fight or flight." The "old" brain houses the most basic desires for food, water, and sex, and it is within the old brain that cravings exist. The second part of the brain is the "new" brain, the cerebral cortex. This portion of the brain houses the ability for logical-thought processes, reasoning, and the functions of the modern human being. Within this section of the brain lies the ability to assess and effect thought processes and behavioral responses. When cravings occur in the old brain, the process of stopping cravings is to move thought processes from the old brain into the new brain. Again, we will turn to the conventional wisdom of AA "old timers" who suggested "don't drink, read the Big Book, and go to meetings" as an effective approach to craving. It is actually the process of reading that shifts thought processes to the new brain (cerebral cortex), thus offering the opportunity for cognitive reframing. To summarize, it appears plausible within this example to postulate that cravings are really correlates of cognitive breakthrough phenomena of subcortical sensitized states. Taken to the next logical step, one can make the assumption that thoughts and associations can be triggered by environmental cues that have been classically conditioned throughout the process of addictive behaviors and can thus be cognitively reframed.

Brief Psychotic Disorder The essential feature of brief psychotic disorder is a disturbance involving sudden onset of psychotic symptoms,

such as delusions, hallucinations, disorganized speech, or grossly disorganized or catatonic behaviors. Time frames for this disorder are at least one day but less than one month. It is important to rule out the presence of a mood disorder, schizoaffective disorder, or diagnosis that better accounts for observed behaviors. In addition, assessment of substance use is important to rule out the contribution of mood-altering substances to the disturbance.

This diagnostic category comprises three notable specifiers. The first category is “with marked stressor(s),” which is indicative of what was referred to in the *DSM-III-R* as the brief reactive psychosis. Within this subcategory, psychotic symptoms develop shortly after and presumably in response to a single or cluster of events that are considered overwhelming to individuals, regardless of race, age, or culture. Within the OCD population, precipitating factors stem from the presence of stressors within treatment approaches—for example, forcing extremely difficult exposures each and every day upon the OCD client, as is done by two Philadelphia psychologists.

The second notable specifier within the diagnosis of brief psychotic disorder is “without marked stressors.” This specifier is indicated if the patient’s symptoms are not in response to stressful events that would be experienced similarly by almost anyone in the described circumstance. The third specifier not closely associated with this article is “with postpartum onset,” which is to be noted if psychotic symptoms emerge within 4 weeks of postpartum status.

Psychotic Disorder Due to General Medical Condition Within these diagnostic categories, the psychosis is characterized by hallucinations or delusions that are believed or known to be the result of a general medical condition. Any number of medical conditions can be associated with or cause psychotic symptoms. These include neurologic disorders or conditions such

as epilepsy (e.g., temporal or frontal lobe), multiple sclerosis, cerebrovascular disease, migraine, neoplasms, and potential visual and auditory nerve damage or central nervous system infections. Psychotic symptoms can also be the result of endocrine conditions, with the most common being hyper- and hypothyroidism and hypoparathyroidism. Metabolic conditions that can lead to psychosis include electrolyte imbalances, hepatic or renal diseases, and autoimmune disorders. Psychotic disorders can also be related to focal brain injury.

It is difficult to predict the course of psychotic disorders when related to a general medical condition. Psychotic symptoms may be limited to a single episode, or they may progress through a series of recurrent psychotic episodes, seemingly consistent with regard to time frame. Treatment of the medical condition associated with a psychotic disorder is certainly necessary to resolve the psychosis; however, it is not necessarily sufficient, as psychotic symptoms may persist for weeks or even months after resolution of the initiating medical condition.

Integrated Treatment Approaches for Obsessive-Compulsive and Comorbid Disorders

Given the cases presented here, there is a need to address OCD and the various comorbid mental illness and substance-abuse disorders associated with the primary diagnosis. For treatment to be effective, several aspects of care require re-vamping. First efforts to integrate care are paramount. The identification of comorbid diagnosis has the potential to provide remarkable impact in the treatment of mental illness. For treatment of persons with comorbid illness to be effective, three concepts must be embraced: parallel, sequential, and integrated care.

Parallel Care Parallel care simply refers to offering equal consideration to emergent co-

morbid psychiatric to psychiatric disorder, substance-use disorder, to substance use disorder and psychiatric to substance comorbid disorders. Growing evidence is documenting that the day of the single-diagnosis patient is gone. Moreover, effective treatment will be based on the clinical staff's ability to detect and effectively address comorbid illness.

Sequential Care Sequential care refers to prioritization of care issues. For example, traditional practice of waiting to see whether depression or obsession clears following detoxification are no longer effective within the time-limited managed-care environments that provide psychiatric and substance-use disorder treatment. Emphasis should be placed on a multidisciplinary team's gathering of data and the placement of data onto a concise timeline depicting the progressive nature of concurrently emerging symptom groups. Application of diagnostic criteria and time frames can then be effectively applied to the information gathered, and sequential care approaches based on history can effectively prioritize and maximize care plans, thus utilizing the most current evidence-based approaches to care.

Integrated Care Integrated care refers to the participation of all team members in the care of the patient across the continuum of care. Progressive care approaches rely heavily on the input of all team members to balance the multiple important aspects of care. Efforts to provide integrated team supports as patients progress through step-down levels of care will support individualized treatment needs as they emerge across the continuum of care. Particular attention is to be given to the application of any intervention within each level of care to ensure that stressors associated with interventions do not overwhelm patients. It is important to remember that all change, even positive change, can create stress.

An example of application of these concepts is provided in Figure 1.

Growing evidence supports that biological factors contribute to the emergence and progression of obsessive-compulsive disorder; to that end, the classic psychoanalytic theory has all but disappeared. As obsessive-compulsive symptoms are largely refractory pharmacological, behavioral treatments have become most common. Obsessive-compulsive patients are extremely treatment-resistant by nature. Frequently, this population demonstrates medication noncompliance, and they may refuse to attend sessions or to complete homework assignments that are the hallmark of cognitive theory. Effective pharmacotherapy in obsessive-compulsive disorder has been demonstrated through clinical trials. Initial approaches have traditionally begun with a serotonin specific reuptake inhibitor (SSRI). Utilization of SSRIs have demonstrated increases in patient responsiveness to treatment to a range of 57 to 70% (Jenike, 2001).

When examining comorbid OCD and substance dependence, behavioral therapy in conjunction with utilization of SSRIs is an effective approach. For persons with addictive disorders, treatment may begin within inpatient facilities' providing parallel treatment approaches to OCD and substance abuse. This process begins with initiation of SSRIs and utilization of detoxification protocols to address the specific drug of choice. This treatment is enhanced through daily behavioral therapy approaches to assist in the development of coping mechanisms. Generally, this objective is accomplished through a variety of group settings, including educational, psychoeducational, family, and experiential. After the initial period of stabilization, patients can transition into less restrictive levels of care (Yeager, 2000).

The utilization of day treatment or partial hospitalization provides the individual to test their newly acquired skills within the treatment

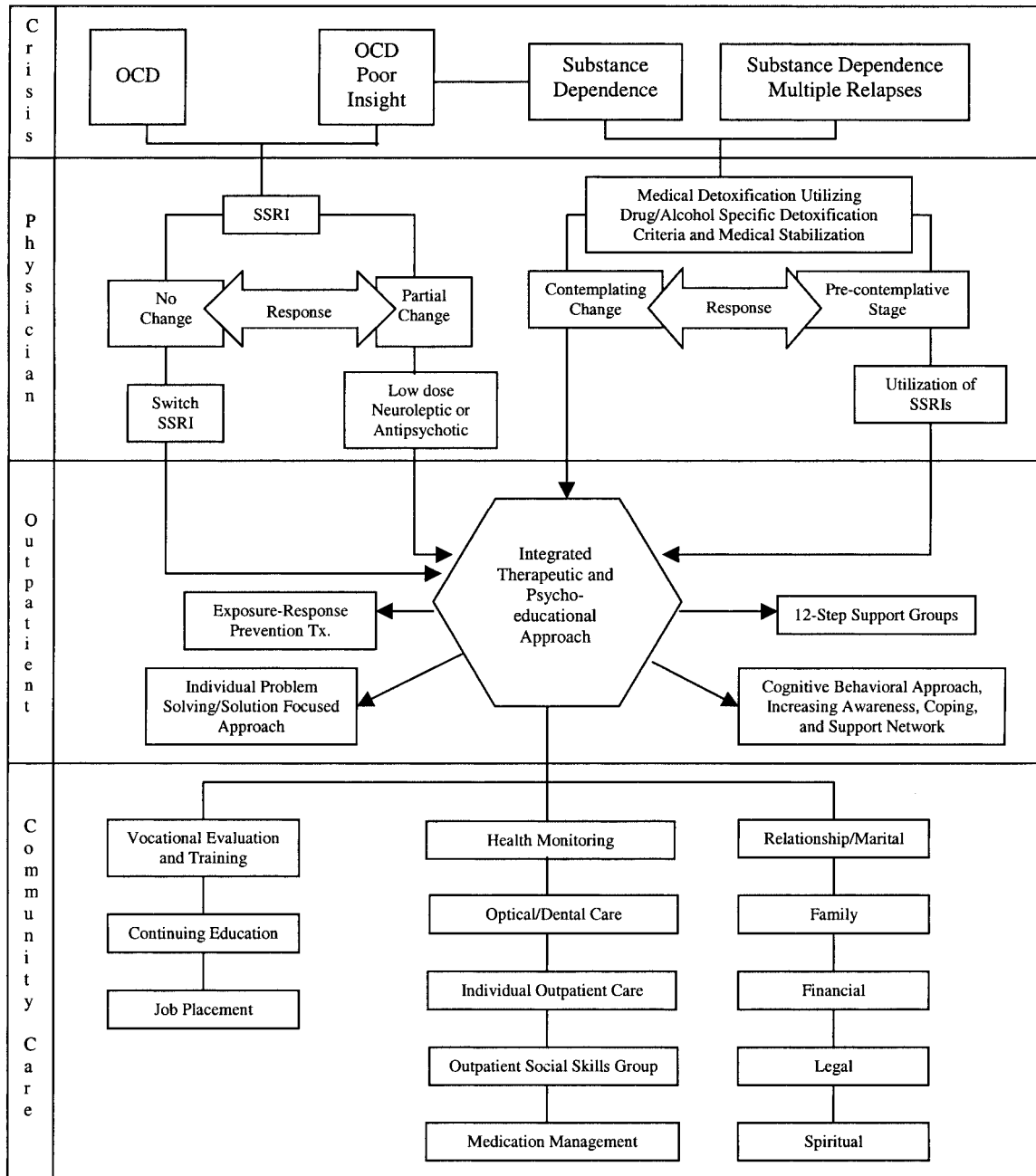


Figure 1
Comorbid Care Management: Parallel, Sequential, Integrated Approach to Treatment

environment. Once therapeutic alliances are established, the task becomes one of development of sufficient coping skills to sustain long-term abstinence. This process is clearly outlined through the application of Roberts seven-stage crisis intervention model (Yeager, 2000; Yeager & Roberts, 2003).

To sustain the patient as they address the challenges presented within their recovery environment, utilization of group therapy and education has proven to be extremely effective in the treatment of persons with multiple comorbid diagnosis. Again, approaches will vary given the individualized treatment need; however, utilizing individual or group therapy assists the individual in addressing the issues that present within their recovery environment. As the individual experiences greater success in addressing the environmental cues previously discussed, they will progress to a less restrictive level of care. For example, individuals with OCD and cocaine dependence with comorbid depressive disorder face similar physiologically based relapse triggers, but they will require different yet similar therapy treatments. While the OCD patient may require systematic exposure treatments, the cocaine addict may benefit from utilization of solution-focused care to address their individualized relapse triggers. Both may be utilizing SSRIs in the form of pharmacotherapy's working to address depressive symptoms, and both the OCD and cocaine-dependence diagnosis are benefiting from the action of the SSRI. If the OCD is identified to be resistant based in poor insight, then consideration should be given to addition of an atypical antipsychotic medication. Should the cocaine-dependent person be experiencing vivid dreams or anxiousness, then it may be appropriate to supplement treatment with Busporine. Both pharmacotherapy and therapeutic approaches are tailored to meet the individualized patient's needs; that is, they are both based in enhancing the development of recovery skills within the recovery environment.

Why is it imperative to conduct a thorough and careful differential diagnosis with persons who seem to be suffering for many years with schizophrenia, hallucinations, psychotic episodes, obsessive-compulsive disorder, ritualism, obsessional thoughts, anger outbursts and temper tantrums, violent rages, aggravated assault, hypersexual episodes, hypergraphia, religiosity, and/or paranoia? First and foremost, symptoms of schizophrenia mimic those of temporal lobe epilepsy, and pharmacological treatment of partial-seizure disorders with anticonvulsants often result in a cessation of the symptoms of schizophrenia and psychosis (Bear & Fedio, 1977; Holzer & Bear, 1997). Finally, treatment with a high regimen of SSRIs and antipsychotics often can exacerbate the schizophrenic via psychosis, hallucinations, and paranoid delusions. As an illustration, consider the following case.

Henry had a normal childhood, except for difficulty in reading comprehension in second grade. Psychological and intelligence testing in second grade indicated that Henry had a high IQ and no learning disabilities. By the seventh grade, Henry was labeled by teachers as disruptive and as the class clown. He was suspended for refusing to pick up food under the table in the school cafeteria, and he seemed to have great difficulty remembering what page the class was on in the textbook. At the end of seventh grade, the Child Study Team diagnosed Henry with oppositional defiance disorder (ODD) and attention deficit disorder (ADD) or OCD, including contamination fears as well as horrific and tormenting thoughts. Clinical psychologists who treated Henry for the next three years gave him a similar diagnosis of ODD and ADD (without hyperactivity). By the age of 16, he would sometimes receive a secondary diagnosis of either schizophrenia, schizoaffective, or schizotypal disorder. At age 18, two different psychiatrists mistakenly diagnosed Henry with pervasive developmental disorder and Asperger's form of autism. By age 22, Henry had received a primary

diagnosis of schizophrenia and a secondary diagnosis of OCD from four different psychiatrists. Once the Paxil prescription was raised to 50 mg, Henry started to have hallucinations that he could communicate with the basketball players on the television screen in his bedroom. Unfortunately, none of the nine different psychiatrists Henry went to over a 6-year period asked him or his parents about closed head injuries. Had any of the psychiatrists done a psychiatric and neurological assessment, they would have learned that Henry had over four closed head injuries, starting during birth, again at age 6 (when he fell off his bicycle), and during his high school varsity wrestling years, which in all likelihood resulted in temporal lobe epilepsy. The strong positive association of temporal lobe epilepsy with symptoms of schizophrenia and psychosis is well documented. In addition, there is significant overlap in the age of onset of the two disorders. Thus, a neurological workup is imperative, particularly when the young adult receives many different diagnoses over the years. To do otherwise can result in harming the patient through misdiagnosis and continued inappropriate treatment.

Persons suffering from a drug-induced psychosis can experience seizures, severe depression, mood disorders, and schizophrenia. Clinicians who have worked in a large hospital emergency room, inpatient psychiatric unit, or residential addiction treatment program repeatedly observe the thought disorders of patients after the heavy use of cocaine, LSD, or ecstasy. Repeated use of amphetamines can result in delusions that are very similar to those demonstrated within the mental illness diagnostic group of paranoid schizophrenia. Symmetrel (Amantadine), a medication that is useful in reducing some of the pain of Parkinson's disease, on rare occasions has created a depressive state and suicidal ideation resulting in both nonfatal and fatal suicide attempts (Shea, 2002, p. 61). As aptly stated by Shea (2002, p. 58–60): "abnormal biochemistry of the brain can directly cause the

odd thoughts and damaging mood states of diseases such as schizophrenia and bipolar disorder. A malfunctioning in the brain created by an amphetamine induced psychosis and external stressors or internal conflicts can increase and intensify the frequency of suicidal ideations." Henry was first diagnosed with ADD and ODD at age 16. The psychiatrist prescribed long-lasting Dexedrine (an amphetamine). After taking Dexedrine daily for over two weeks, Henry appeared to have a psychotic-like experience, and he expressed recurring fears that he was responsible for most of the students' having the flu in his high school. Although Henry had never missed a day of school up until 11th grade, he refused to return to school because of his obsessional and tormenting thoughts. He also had been exhibiting recurring instances of hypergraphia for several years. Henry's parents immediately took him to a psychiatrist who within one hour diagnosed Henry with schizophrenia. Within the next 4 weeks, after seeing another psychiatrist and a clinical psychologist, Henry had a new misdiagnosis of OCD and schizoaffective disorder and was hence prescribed prozac. Henry was administered the MMPI, which indicated that he had no thought or personality disorders. The clinical psychologist's response was that Henry was so highly intelligent that he fooled the MMPI. Even though Henry experienced several head injuries in wrestling tournaments at this time, unfortunately no one referred him for neuropsychological testing.

The Impact of Comorbid Diagnosis

Another neglected area of research and treatment is the comorbidity of OCD, depression, and suicide attempts. As noted by Clark (2002), clinicians and researchers have written about the relationship among obsessions, compulsions, and depression since the development of modern psychiatry. According to *DSM-IV-TR*

(American Psychiatric Association, 2000), obsessions in OCD are defined as persistent and recurrent images, thoughts, and impulses that are experienced by the individual as intrusive and inappropriate; that cause marked distress; that prompt the individual to ignore or suppress such thoughts, images, or impulses; and that are recognized by the person, at least at some point in the course of the disorder, as excessive and unreasonable. In a depressive episode, the individual may perseverate and brood about negative circumstances and courses of action. Such thought patterns occurring within a depressive episode are considered to be ego-syntonic, mood-congruent aspects of depression. Thus, the significantly depressed individual who ruminates about lack of worth and potential loss is not considered to have obsessions per se given the ego-syntonic nature of such thoughts occurring within the depressive episode. In referencing a study by Black and Noyes (1990), Overbeek, Schruers, Vermetten, and Griez (2002) noted that symptoms of OCD antedate the development of depression in 38% of cases whereas persons with depression develop OCD in 11% of cases. Major depression is the most common disorder comorbid with OCD (Piggott, L'Heureux, Bubbett, Bernstein, & Murphy, 1994). Studies documenting the prevalence of OCD comorbid with major depression have shown a wide range. Rasmussen and Eisen (1998) found that one third of patients with OCD suffer concurrent depression at time of evaluation and that two thirds of persons with a history of OCD experience major depression over the course of their lifetime. In their overview of conditions comorbid with OCD, Piggott et al. (1994) referenced a study that in a sample of 76 patients who met the *DSM-III-R* criteria for OCD, major depression was present in 61% of cases, and the lifetime prevalence rate for major depression was 85%. Overbeek, Schruers, Vermetten, and Griez (2002) cited a report by Milanfranchi et al. (1995) in which comorbidity rates between depression and OCD

range from 19 to 90%. OCD may also present with a significant number of comorbid disorders, such as bipolar disorder (Chen & Dilsaver, 1995), social phobia, panic disorder, generalized anxiety disorder, and dysthymic disorder (Crino & Andrews, 1996)—disorders that may each carry a risk for development of depression. Clinicians treating persons with OCD comorbid with body dysmorphic disorder, trichotillomania, tic disorder, and substance-use disorders are well aware of the depression experienced by individuals with comorbid disorders. Hollander et al. (1996/1997) found that OCD subjects with comorbid psychiatric disorders had a 15% rate of suicide attempts versus a 3.6% rate of suicide attempts in uncomplicated OCD.

Areas of Needed Research in the Treatment of OCD Comorbid with Depression, Tourettes, or Neurological Disorders

Although recent research by Abramowitz et al. (2000) and Overbeek et al. (2002) examined the efficacy of treatment of OCD comorbid with depression, the studies also documented how severe depression treated with existing psychopharmacological and CBT-based approaches resulted in lower gains in treatment as compared to gains in treatment of persons with comorbid depression of lesser severity or with OCD alone. Neurobiological research by Brody and colleagues (1998) showed that subjects with differing patterns of brain metabolism responded preferentially to behavior therapy or medication. Enhancement of such diagnostics might allow for better triage decisions in determining choice of medication or behavior therapy in select cases. Ward, Shapira, and Goodman (2002) reviewed nonpharmacological somatic treatments for anxiety disorders: deep-brain stimulation (DBS), vagus-nerve stimulation (VNS), and repetitive transcranial magnetic stimulation

(rTMS). The authors noted that although advances have been made in pharmacological and CBT-based treatment, many patients remain refractory despite aggressive and long-lived trials of medication and CBT. Perhaps as further research documents the efficacy of these nonpharmacological methods in cases of complex comorbidity, the methods might offer relief for persons with comorbid major depression and OCD with difficulties heretofore considered intractable. Friedman, Hatch, and Paradis (1998) reviewed considerations in assessing and treating OCD in minority populations and concluded that training clinicians in cultural sensitivity in clinical practice and more research related to OCD in minority groups are certainly warranted.

Anxiety and OCD may arise from a malfunction of the brain or internal biochemical substances—specifically, serotonin, norepinephrine, and dopamine—that control mood, movement, blood pressure, neural impulse conduction. These responses may very well be a part of the human survival instinct that served to preserve the species at one point in the evolution of humankind. However, today norepinephrine may trigger anxiety, nervousness, and elevated vital signs, within individuals who have an overly sensitive response system. Current research indicates that serotonergic agents relieve obsessive-compulsive symptoms and that they appear to be superior to selective norepinephrine reuptake inhibitors. It is believed by many that the serotonin reuptake inhibitors function to stabilize and balance neurotransmitter and neuromodulator activities, thus resulting in cooperative social behavior and stabilization of impulsivity (Swartz, 1996; Soomro, 2001).

In addition, research indicates a neurological disorder link with OCD, as approximately 55 to 70% of persons with head trauma and Tourette's syndrome meet the diagnostic criteria for OCD (Cofey, 1998; Rasmussen & Eisen, 1998; Steketee & Frost 1998). A pattern of neuroanatomical deviation appears to be an emergent

theme among OCD, and convincing studies regarding twins suggest a genetic susceptibility to OCD. Monozygotic twin pairs were 63% concordant for OCD symptoms, and one in four to five family members had OCD when a family history of the disorder existed (Carey & Gettesman, 1981).

It should be noted that to date no controlled study has examined the diagnostic precursors, comorbid affective and anxiety disorders, neurological disorders, acute stress disorders, and crisis episodes that affect the etiology of suicidality and first-time potentially lethal suicide attempts. The sequence of events prior to a first-time suicide attempt are complex and difficult to document. Acute and multiple stressors are also often ignored during inpatient or outpatient psychiatric intake assessments. The misdiagnosis and failure to detect suicidal ideation and suicide plans are compounded when an individual receives five or more different diagnoses and when the sixth clinician continues to fail to order neuropsychological testing and a neurological workup. All treatment refractory patients admitted to inpatient psychiatric units should be thoroughly assessed for both psychiatric and neurological disorders.

Most studies neglect to examine comorbidity, suicidality, and the life-threatening mistakes of misdiagnosis. Dr. Edwin S. Shneidman, the founder of modern suicidology, eloquently stated that the major bottleneck in suicide prevention is not in remediation, since there are well-known and effective treatment procedures for many types of suicidal states; rather, it is in the diagnosis and identification. Thus, if the primary objective of mental health professionals is to save lives and facilitate rapid recovery, then differential diagnosis and suicide assessment is critically important. Dr. Peter Mueller has effectively treated hundreds of individuals with temporal lobe epilepsy. Soon after prescribing Tegretal or Depakote, most of Dr. Mueller's patients become symptom-free. Dr. Mueller indicates that:

Doctors are cutting corners due to managed care constraints, and the dearth of training in neurology they receive during medical school and psychiatric residency programs. The major failure in misdiagnosis of primary psychiatric disorders continually takes place because medical disorders are not ruled out. First and foremost, with any new patient presenting for assessment accurate history of physical and mental health should include questions designed to rule out any neurological disorders, general medical disorders, and comorbid psychiatric and substance use disorders. Millions of dollars have been lost each year because of the misdiagnosis of schizophrenia among persons experiencing neuropsychological problems associated with complex partial seizures. (P. Mueller, personal communication, January 14, 2003; psychiatrist in private practice, Princeton, New Jersey)

The poor prognosis for falsely labeled persons with paranoid schizophrenia is compounded by their desire to recover through self-medication with illegal drugs and the recurring stress of rigid, restrictive, and at times punishment-oriented and locked inpatient psychiatric facilities. It is hard to fathom that in the medically and technologically advanced 21st century, many psychiatrists fail to refer their patients for video EEGs, MRI scans, PET scans, neuropsychological testing, or brain spect imaging in order to confirm a particular psychiatric diagnosis. This trend is particularly true given the high prevalence of parallel symptomatology of neurological and psychiatric disorders. Holzer and Bear (1997) have documented the high prevalence of epilepsy as the second most common neurological disorder after headache in the United States.

Conclusion

Comorbidity of psychiatric disorders, hopelessness, brief psychotic episodes, depression, and suicide ideation clearly represent high-risk factors for suicide and suicide attempts. However, it is important to note that a patient's risk of suicide should never be based on the gut-level feelings of a psychiatrist or social worker, or on the score on a single psychological test or assessment scale. *A comprehensive biopsychosocial assessment of suicide risk factors should include previous psychiatric diagnoses and treatment history, current primary and secondary diagnosis, findings from neurological workup, prior history of suicide attempts, history of alcohol or substance abuse dependence, family history of suicide and mental illness, marital status, and employment history.* Future longitudinal research needs to be planned and carried out on large samples of OCD sufferers with comorbid depression, substance abuse, personality disorders, and psychotic episodes. Evaluation research on the utility of different suicidality measures and scales in predicting suicide attempts needs to be implemented with different age groups and comorbid disorders. Quasi-experimental studies need to be planned that compare a sample of the majority of OCD sufferers with comorbid depression and substance abuse who never attempt suicide, with the small group who do experience delusions, psychotic episodes, and attempt suicide. Future research should also include univariate and multivariate statistical techniques. Because of the life-threatening nature of suicide ideation, suicide plans, and suicide attempts, it is critically important for future research to examine the effectiveness of suicide prevention, crisis intervention, and step-down outpatient treatment programs.

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Obsessive Compulsive Disorder: Theory, Research, and Treatment By Ross G. Menzies; Padmal De Silva Wiley, 2003. Read preview Overview. Anxiety Disorders Comorbid with Depression: Social Anxiety Disorder, Post-Traumatic Stress Disorder, Generalized Anxiety Disorder, and Obsessive-Compulsive Disorder By Dan J. Stein; Eric Hollander Martin Dunitz, 2002. Read preview Overview. Heredity in Comorbid Bipolar Disorder and Obsessive-Compulsive Disorder Patients By Amerio, Andrea Tonna, Matteo Odone, Anna Stubbs, Brendon Ghaemi, S. Nassir Shanghai Archives of Psychiatry, Vol. 27, No. 5, October 2015. PRPEER-REVIEWED PERIODICAL. Comorbidity and suicidal behavior. Comorbid Axis I and personality disorders psychopathology is reported in 14% to 62% of all suicide decedents.^{3,4} In clinical samples of patients who had attempted suicide, as many as 84% had comorbid psychopathologies. More patients with comorbid Axis I and personality disorders psychopathologies had made multiple suicide attempts than those with other comorbid psychopathologies (65% vs 24%).⁵ Predictors of suicidal behavior in persons with personality disorders were examined to gauge the impact of comorbid psychopathology on the risk of suicide behavior. Axis I and axis II disorders as predictors of prospective suicide attempts: findings from the collaborative longitudinal personality disorders study. Obsessive-compulsive disorder (OCD) is characterized by persistent and recurring thoughts, urges, or images (obsessions) that lead to repetitive behaviors or actions. Onset usually in puberty following a stressful event. Incidence of comorbid major depressive disorder, OCD, and excoriation disorder. Clinical features: hair loss, most commonly involving the scalp, eyebrows, or eyelashes. Diagnostic criteria (according to the DSM-5). Hair loss due to recurrent pulling out of one's hair. Repeated attempts to stop this behavior. Results in significant distress/impairment (school, work). Presentation and treatment of complicated obsessive-compulsive disorder. J Clin Psychiatry. 2014; 75(3): p. e07. doi: 10.4088/JCP.13023tx2c.