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Best practices for the treatment and management of Schizophrenia

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Best Practices for the Treatment and Management of Schizophrenia

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A research project submitted to the Graduate Faculty of

JAMES MADISON UNIVERSITY

In

Partial Fulfillment of the Requirements

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Department of Graduate Psychology

May 2010

Dedication

This manuscript is lovingly dedicated to Corliss Ann Davis (1947 – 2004) and Allan Myers Davis (1944 – 2000). Corliss, you were the best of us in all the ways that truly mattered. Dad, I'm sorry you never found your way. Rest easy. And Godspeed.

Acknowledgements

A project of this magnitude is never a solitary effort. For the past two years, I have been blessed to have the support and love of my partner, Tamera Ratchford. Tam, thank you for your unfailing patience, understanding, and encouragement, especially over the past difficult months. I could not have done this without you. I would like to acknowledge the support of my committee chair, Dr. Jack Presbury, as well as committee members, Dr. Renee Staton, and Dr. Lennie Echterling. I offer my thanks and gratitude to each of you for sharing your amazing gifts with me. I would also like to acknowledge Dr. Ed McKee. Ed, thank you for recognizing my potential and giving me a chance. It takes a village to raise a child, and I would not have achieved this milestone without the abiding love and support of my family, especially my parents, Mary T. and Gerry Yeiser. Mother, thank you for teaching me what was really important in life and then being patient and wise enough to let me believe I'd figured it out on my own. You have been my rock and my hero for the last 45 years. Pop, thank you for teaching me that a father's love isn't defined or limited by shared genes. You were as generous and goodhearted a man as ever walked the earth, and I was proud to be your daughter.

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Abstract

Master's-level counseling psychology programs strive to build a solid foundation of core competencies in students but do not typically emphasize treatment strategies for the chronically mentally ill or psychopharmacology. This paper is intended as a resource for counselors who serve clients with schizophrenia spectrum disorders. It discusses some of the most widely-supported evidence-based individual and family psychosocial interventions, including cognitive behavioral family therapy, cognitive behavioral therapy for schizophrenia, social skills training, and cognitive training and remediation. It also contains a chapter on psychopharmacology.

Chapter 1: Introduction

In 1941, when she was 23 years old, Rosemary Kennedy underwent a pre-frontal lobotomy in a horribly misguided attempt by her father to cure her violent mood swings and increasingly unpredictable and volatile behavior. The procedure, performed while she was awake, left the oldest sister of the future president of the United States with the mentality of an infant and unable to speak intelligibly or meet even her most basic needs. For the next 64 years, until her death, Rosemary lived in an institution. Although the Kennedy family has consistently held Rosemary was mentally retarded, it is likely she was instead suffering from mental illness and perhaps some form of learning disability. A former director of the National Institute of Mental Health (NIMH), noting Rosemary's ability to perform multiplication and division, states:

I think it's likely Rosemary was somewhat slower than her siblings. It could be she had an IQ of 90 in a family where everyone was 130, so it looked like retardation, but she did not fall into IQ 75 and below, which is the definition of mental retardation. ... There is no way I can picture her at less than a 90 IQ, but in that family, 90 would be considered retarded (Kessler, 2006, p. 236).

Kessler (2006) speculates it would have been impossible for John Kennedy to be elected president in 1960 if the public had known his oldest sister suffered from mental illness. "The stigma of mental illness in those days was like tuberculosis, cancer, or worse. Mental retardation is more benignly not your fault. ... Even in 1941, performing a lobotomy on someone who was mentally retarded would have been medical malpractice" (p. 237).

Sadly, while the stigma associated with mental illness has certainly diminished over the past 60 years, it nonetheless remains a formidable obstacle to those living with a diagnosis and their families. For many people, mental illness continues to be a shameful secret.

I encounter shame and stigma every day in my work with the 18 to 26-year-old clients I serve at the residential treatment facility where I am doing my internship. Usually my clients and their families have been struggling for years with the symptoms of serious mental disorders. Weekly family therapy sessions frequently reveal a lack of understanding about the nature of the disorders in question and as well as an absence of any previous dialogue within the family system about the mental illness. The unspoken message is “Please just fix my kid so he/she can become a productive member of society.”

I often find myself looking to my own experiences, especially within my family of origin, to help me conceptualize what I encounter with my clients. Usually I do not have to search far to find the parallels in my own life’s experience. Both my father and his sister lived their entire lives with undiagnosed mental illness. Like a lot of families, we just didn’t talk about it.

Just before Christmas 2004, my aunt Corliss died. She died alone, in the psychiatric ward of a local hospital, having recently been hospitalized for yet another in a continuing series of psychotic breaks. The previous summer Corliss had been found roaming her neighborhood naked and nearly catatonic. When her inpatient insurance benefits ran out, the hospital pumped her full of Haldol and discharged her. Over the remaining months of her life, she slowly emerged from the recesses of her own mind, and

her caretaker, my uncle Bill, had begun to make plans to move Corliss into a small mother-in-law's suite in his home. Unfortunately, this would never come to pass. I knew nothing of this last sad chapter of her life until after my aunt died.

Despite the fact she had for most of my life remembered every birthday, sending me a check well into my adulthood, I most often relegated Corliss to the periphery of my consciousness and easily found plausible excuses for not staying in touch. For a time after my father drowned in 2000, I was made it a point to remain in contact with my aunt. However, by the time of her death, I had not spoken to Corliss in perhaps a year.

Corliss's life had not been a happy one. For several years prior to her death, she had lived alone with only her cat for company and solace. My aunt had few, if any, friends or close relationships, including those within her family. She married in her mid 30s to a man 25 years her senior. This marriage was as much of a surprise to me as her death, and it was sadly no happier than her life. It was an open secret in our family that Corliss had been "in the loony bin" before and took medication for "her nerves" and "wasn't right." Though it is unfortunately now impossible for me to piece the real story together, my knowledge of psychopathology leads me to believe my aunt was very likely suffering from a schizophrenia spectrum disorder as well as Asperger's Disorder, learning disabilities, and possibly early onset Alzheimer's Disease.

As I sat at her funeral listening to impersonal platitudes spoken by a minister who didn't know my aunt, I turned to my own memories. I remembered Corliss not as the sad person she had become, but as the kind, loving, if quirky woman she had been when I was a child. Corliss always made time for me. Whenever I visited, she took me to a local

department store, let me buy whatever I wanted, and did not argue if I wanted the tomboy set of toy six-shooters instead of a Barbie doll.

One spring break she taught me how to sign when I stayed with her at the Kentucky School for the Deaf, where she worked. Although I wasn't very good at it, she never let on. I have a vivid image of the way her hands flew and remain amazed to this day she could do that. It is ironic that the family member that struggled so with school and never measured up to the Davis standard of success, is to my knowledge, the only member of our family who was fluent in a second language. While preparing this manuscript, I realized Corliss was probably also the first person in my life to actively instill a sense of multi-culturalism and tolerance within me. Because of her I thought deaf people were cool and learned to embrace people different from myself. This was a gift she never knew she gave, and I am so grateful to her for it.

The bitter cold morning we buried her I remembered how she took me every year to view the best Christmas lights in Somerset, Kentucky. It was fun to be a kid with her around because she loved listening to Alvin and the Chipmunks, watching movies that I liked, and eating popcorn from a big plastic bowl with an icy cold "Cocola" to wash down the kernels.

Sadly, Corliss frequently functioned in our family system as the butt of cruel ridicule, whether it was about her propensity for talking incessantly with her nervous stutter, or her weight, which had grown to morbidly obese levels by the time of her death. To this day I am ashamed of how little I did to stay connected to her. I wish my career in mental health had begun earlier so that I might have helped her advocate for herself and perhaps educate my family about mental illness.

When I began my graduate studies and progressed through my training as a therapist, my focus was certainly not on developing a knowledge base of treatment strategies for the chronically mentally ill. As I immersed myself in the intense experiential learning experience offered by JMU's counseling psychology program, I was quite comfortable staying within the confines of the "worried well" to develop my core counseling competencies. During my two semesters as a counseling practicum student, I routinely encountered cases or clients that taxed my knowledge base. I came to see this was the nature of the beast. Very early on it became clear that while I had had the support of my supervisor, it was my job to find out what I needed to know about treating a client with a particular type of concern. No one was going to spoon feed it to me. Whether it was finding resources that would help me use play therapy as an intervention for an 8-year-old or bringing myself up to speed on best practices for helping a client suffering from PTSD, I bore the lion's share of responsibility in ensuring my own competency. Some days it frankly felt as if I had bitten off far more than I was able to chew.

At my internship site, the first client who added to what would become a challenging caseload, was a 20-year-old young man who had been diagnosed with a myriad of disorders, the most daunting of which was schizophrenia. It became rapidly and painfully apparent that my attempts at insight therapy with this young man were failing and, in fact were often a source of frustration for us both. I had no doubt I could build a relationship with this client; but a working alliance alone wasn't going to help him deal with the profound social and vocational deficits he was living with on a daily basis. Thus began my professional journey to educate myself on the treatment and management of schizophrenia. Much to my own surprise, I have found myself becoming

a resource for the other clinicians with whom I work. As one of my co-workers said recently during case review, “I don’t know much about schizophrenia and it scares me.”

It is from these experiences that this manuscript emerges. It is my hope that this paper will be used as a resource for other counselors who find themselves struggling to help clients with schizophrenia spectrum disorders. While it would be far beyond the scope of this project to include all possible interventions for treating this client population, this guide will attempt to cover some of the most widely supported evidence-based individual and family interventions. Chapter 2 discusses the historical, etiological, and modern definitions of schizophrenia. Chapter 3 provides an overview of psychopharmacology. Chapter 4 discusses available interventions and reviews the literature. Chapter 5 pulls everything together to provide a case conceptualization.

Chapter 2: What is Schizophrenia: History, Etiology, and Current Theories

Schizophrenia is a severe form of mental illness that affects approximately 1% of the population worldwide (Andreasen, 2001). The American Psychiatric Association (*DSM-IV-TR*, 2000) defines it as “a disorder that lasts for at least six months and includes at least one month of an active-phase symptoms (i.e., two [or more] of the following: delusions, hallucinations, disorganized speech, grossly disorganized or catatonic behavior, negative symptoms” (p. 298). Symptoms of schizophrenia are further categorized as either positive or negative. Positive symptoms are hallucinations and delusions. Negative symptoms include social withdrawal, anhedonia (inability to experience pleasure), apathy, and social inappropriateness. While the positive symptoms associated with a psychotic break are usually the presenting concern, the most debilitating symptoms of schizophrenia are the cognitive deficits that remain after the positive symptoms have been treated and have either remitted or decreased to a manageable level. These deficits frequently result in impaired social and vocational functioning that leaves the individual with schizophrenia unable to have meaningful relationships or maintain employment.

Schizophrenia can present as multiple combinations of symptoms and can also follow a number of distinct courses depending on the age of onset, severity, and treatment received (Marley, 2004). It affects men and women equally. However, the average age of onset for men is earlier (15-25) than for women (25-35) (Stone, Faraone, & Tsuang, 2004). Often, poor pre-morbid social functioning is predictive of a poor prognosis. Due to its heterogeneous presentation, schizophrenia may be better conceptualized as a spectrum of disorders rather than a single illness (Marley, 2004).

Although the incidence of schizophrenia in the general population is low, the human and financial costs are staggering. In the United States, schizophrenia accounts for about 40% of mental health hospital beds and about 15% of all hospital beds, which breaks down to \$19 billion annually in direct costs (hospitalization, physician and therapist services, and medications) and \$46 billion in indirect costs (disability, lost productivity, etc.) (Patterson, Alcala, McCahill, & Edwards, 2010). Schizophrenia accounts for 2.5% of all health costs, 75% of all mental health costs, and 20% of the total days of Social Security disability benefits (Patterson et. al, 2010; Marley, 2004).

In addition, mortality rates for individuals with schizophrenia are between 1.6 and 2.6 times higher than the general population and life expectancy is 20% shorter. Several factors account for these statistics. About half of individuals with schizophrenia will attempt suicide, and 10-15% will die as the result of a completed suicide. Persons in this group have significantly higher smoking rates than the general population, which exposes them to cardiovascular and pulmonary risks. They are also more likely to be obese and suffer from diabetes and heart disease (Patterson et al., 2010).

Sadly, funding for and availability of comprehensive and integrated community-based outpatient mental health services has dwindled over the past 20 to 30 years. In 1999, state funding for family-based services for the severely mentally ill was estimated to be \$100,000 annually per state (Dixon, Goldman, & Hirad, 1999). The deinstitutionalization movement that began in the 1960s with the Community Mental Health Centers has increasingly resulted in “transinstitutionalization” of the chronically mentally ill whereby they are removed from the mental health care system and

reinstitutionalized in other systems, such as prisons or nursing homes (Brent & Giuliano, 2007).

History

The evolution of the modern understanding of schizophrenia over the past century cannot be adequately discussed without including the contributions of Emil Kraepelin (1855-1926) and Eugene Bleuler (1857 – 1939). The current classification system of mental disorders contained in the *DSM-IV* can be traced back to Kraepelin's theories (Green, 2003). Kraepelin was the first to describe the symptoms of schizophrenia and attribute them to a single illness he named *dementia praecox*, meaning early onset of deteriorating intellectual functioning (Mueser & Gingerich, 2006). He identified the characteristic symptoms of schizophrenia as hallucinations, delusions, impaired attention span, and social withdrawal. Kraepelin conceptualized the course of the disorder as progressive and irreversible mental deterioration. Bleuler disagreed with Kraepelin's characterization of the early onset and course of the disorder and instead believed the key feature of the illness was a split in the mind between perception and reality. Bleuler believed the term *dementia praecox* was incorrect and would be misunderstood and instead coined *schizo* (split) *phrenia* (mind) (Torrey, 2006). Green (2003) notes that "the disorder had the misfortune of going from a label that was pessimistic and incorrect to one that became confusing and misleading" (p. 10). One of the most commonly held and persistent myths about schizophrenia is that people who have it are suffering from multiple or split personalities (Mueser & Gingerich, 2006).

Green (2003) also highlights another of Bleuler's key contributions to the understanding of schizophrenia – namely the distinction between *fundamental* and *accessory* symptoms.

Fundamental symptoms included affectivity (mood), ambivalence, and alterations in association. Disturbance in association (loose associations in thought and speech) was afforded special status as the abnormality most closely linked to the disease process. These simple fundamental symptoms combined to form compound fundamental symptoms, including disturbances in attention. . .

Accessory symptoms, which were derived from fundamental symptoms, included hallucinations, delusions, and a variety of behavioral and speech abnormalities (pp. 10 -11).

This distinction was downplayed for most of the 20th century and the focus of treatment was on the positive symptoms. However, today many mental health professionals have adopted Bleuler's view of schizophrenia, conceptualizing it “predominately as a disorder of cognition with neurocognitive deficits featuring prominently and strongly influencing functional recovery” (McGorry, 2005, p. 3).

Etiology

It is far easier to describe what schizophrenia is than to discuss its etiology. Despite more than 100 years of research and scientific advances that have resulted in the mapping of the human genome and the ability to neuroimage the human brain, as of this writing, the causes of schizophrenia are not known. By far the most prevalent theory, and one that is extensively supported in the literature, is that schizophrenia is caused by a combination of factors. As Miklowitz (2004) notes:

There is substantial evidence that genetic, biological, psychological, and social variables are ‘moving targets,’ which are mutually influential in the course and onset of psychiatric illness. Notably, psychosocial variables affect the structure and function of the brain and the occurrence of or timing of gene expression (p. 668).

The stress-vulnerability model posits that genetic, biological, and environmental stressors combine to cause the disorder (Mueser & Gingerich, 2006). The interplay of these factors in a given individual accounts for the wide variability in presentation and course of the illness. Anjum, Gait, Cullen, and White (2010) surmise that people with stronger genetic loading may require fewer environmental influences to “pass through the threshold of illness” and that in turn, genetic predisposition may be offset by an optimal environment (p. 364).

Genetic factors. Family studies provide some of the most compelling evidence of a genetic basis for schizophrenia. In a meta-analysis Gottesman (1991) calculated the concordance rates across studies at 48% for monozygotic (MZ) twins and 17% for dizygotic (DZ) twins. Prescott and Gottesman (1982) estimated the rate of concordance for MZ twins raised apart at 58%. It should be noted that this is an extremely rare phenomenon with only 12 cases appearing in the worldwide literature (Green, 2003). An important conclusion can be drawn from these findings: schizophrenia is both an environmental and a genetic disorder. If the disorder were caused by only environmental factors, one might expect to see identical concordance rates between MZ and DZ twins. In contrast, if schizophrenia were solely a genetic disorder, then the concordance rate between MZ twins would be nearly 100%. Comparing a group of MZ twins raised in

similar environments with a group of DZ twins also raised in similar environments, Stone et al. (2004) found the concordance rate for MZ twins to be nearly twice that of DZ twins (Stone et al., 2004).

Clearly, research was needed that could separate and examine both environmental and genetic factors. The most logical way to do this is to compare the prevalence of schizophrenia in adoptees with the prevalence in their biological and adoptive relatives. Many studies doing so originated in Nordic countries because of thorough records regarding adoptions and psychiatric hospitalizations kept by these countries (Green, 2003).

An early study compared the prevalence of schizophrenia in 47 children of schizophrenic mothers adopted at birth by non-biological relatives with that of a control group of 50 adoptees with non-schizophrenic biological mothers (Heston, 1966). In that study, 10% of the children of schizophrenic biological mothers were found to have schizophrenia as compared to none in the control group. In a similar study involving 5,483 children given up for adoption by schizophrenic mothers, 33% were found to develop schizophrenia in contrast to 18% of adoptees in the control group (Kety, Rosenthal, Wender, & Schulsinger, 1971). Table 1 contains a breakdown of heritability probabilities based on family relationship (National Coalition for Health and Professional Education in Genetics, 2010).

Table 1

Heritability Percentages for Schizophrenia

Relationship	Incidence
Parent of individual with schizophrenia	5-10%
Sibling of individual with schizophrenia	8-14%
Offspring of individual with schizophrenia	9-16%
Offspring of two parents with schizophrenia	46%
Uncle or aunt of individual with schizophrenia	2%
Nephew or niece of individual with schizophrenia	1-4%
Grandchildren of individual with schizophrenia	2-8%
Half-sibling of individual with schizophrenia	4%
First cousin of individual with schizophrenia	2-6%
Identical twin of individual with schizophrenia	40-60%
Fraternal twin/full sibling of individual with schizophrenia	10-16%

Environmental factors. Numerous research findings support the influence of environmental factors on the development and course of schizophrenia. Prenatal insults that disrupt neural development during the second-trimester of fetal development have been linked to schizophrenia. “An unfavorable intrauterine environment, whether the result of infection or hypoxia, may result in altered development of the fetal brain” (Anjum et al., 2010, p. 364). Since 1988, studies have supported the hypothesis that maternal exposure to the influenza virus during gestation increases the risk the fetus will later develop schizophrenia (Mednick, Machon, Huttunen, & Bonett, 1988; O’Callaghan,

Sham, Takei, Glover, & Murray, 1991; Takei, Van Os, & Murray, 1995). However, in a recent meta-analysis examining whether birth during the 9-month period after the 1957 influenza pandemic was a risk factor for schizophrenia, Selten, Frissen, Lensvelt-Mulders, and Morgan (2010) found the data insufficient to support the maternal influenza hypothesis. The debate will no doubt rage on.

Several studies identify maternal malnutrition during gestation as another risk factor for schizophrenia. Susser et al. (1996) found that exposure to the Dutch Hunger Winter of 1944 and 1945 during the first trimester of prenatal life doubled the risk of schizophrenia. St. Clair et al. (2005) and Xu et al. (2009) replicated these findings in studies examining the effects of the Chinese famine of 1959-1961.

Researchers have also identified obstetrical complications as an early risk factor for schizophrenia (Schiffman, Carter, & Mednick, 2004). Examples of obstetrical complications include: oxygen deprivation during pregnancy, labor, and delivery; preeclampsia; bleeding during pregnancy; and premature delivery. In a 28-year follow-up study of 11,017 subjects, Jones, Rantakallio, Hartikainen, Isohanni, and Sipila (1998) found subjects with schizophrenia were six times more likely to have been born prematurely and seven times more likely to have sustained perinatal brain damage.

Chapter 3: Psychopharmacology and Medication Management

It may strike the reader as odd that a discussion of psychopharmacology appears with such prominence in a paper intended as a resource for counselors. However, it is my belief that the effective treatment of any mental disorder must encompass a knowledge and understanding of psychopharmacology. I encounter this every day with the clients I serve at my internship site, the majority of whom are being treated with an array of medication that must be carefully monitored and managed. I simply could not be effective as a counselor without an understanding of psychopharmacology.

Such an understanding becomes paramount when working with a client who has schizophrenia, since there is little hope of alleviating the suffering wrought by the disorder without the aid of appropriate medication. Patterson et al. (2010) note:

A single intervention is only part of the therapeutic process. In the case of starting medication it is equally, if not more important to understand how to talk with your clients about medication, how to engage in collaborative conversations with the medical professionals who prescribe your client's medication, and how to support your clients and their family members after your client has started medication (p. 207).

Both my professional and personal experience has taught me the value of collaboration between the therapist and physician. Some years ago while suffering from a debilitating clinical depression, I sought therapy with an LPC. Through gentle probing and careful assessment of my symptoms, she broached the subject of medication. Although I was suffering terribly, I did not have a physician I could turn to for help. Because my therapist had established a collaborative relationship with a local physician,

she was able to make a referral for me. From that point forward, I had two trusted professionals who were working together for my common good. Periodically, with my permission, they consulted about my care. Either one working separately could not have provided the quality of care they offered through their collaboration.

At my internship site, the entire clinical staff meets monthly with the psychiatrist who treats all of our clients. During these meetings, each client is discussed and clinicians are given the opportunity to raise any concerns regarding medication side effects or new symptoms. In addition, all therapists are not only encouraged, but expected to consult with the psychiatrist about any new problems that arise between these monthly visits.

The efficacy of all of the interventions discussed in the next chapter are linked directly to the client's willingness to take medication. The goal of treating with medication is to reduce symptoms so that the individual can function better and benefit from other forms of treatment such as individual, group, or family therapy and social and vocational rehabilitation.

Antipsychotics are the first line of defense in the treatment of schizophrenia. Approximately 70% of individuals with schizophrenia who take antipsychotics will dramatically improve on these drugs (Torrey, 2006). Persons who do not take antipsychotics have twice the chance of being re-hospitalized within the first year after a psychotic break as those who do take drugs (Hogarty, 2002).

As with many drugs, antipsychotics can cause unpleasant, irreversible, and even life-threatening side effects. A medical doctor, preferably a psychiatrist, will be prescribing these drugs. However, in most instances, the therapist will have far more frequent and sustained contact with the client than the prescribing physician. The

therapist who is well versed in the side effect profiles of the drugs a client is taking, as well as potential interactions with other substances, can play an invaluable role in keeping the client well. In rare instances, the therapist who knows what to look for can literally save a client's life. The side effects of antipsychotics vary and are discussed in detail later in this chapter.

First-Generation Antipsychotics

In 1952, French psychiatrist Pierre Deniker discovered that the surgical sedative chlorpromazine (Thorazine) could also be used to effectively treat the positive symptoms of schizophrenia (Fuller, 2006). This discovery ushered in the era of first-generation antipsychotics (FGAs) that continued until 1990 when clozapine (Clozaril) became available in the United States. Table 2 contains a list of FGAs.

FGAs are classified in terms of their potency: high and low. The more potent the drug the lower the dosage required to achieve a therapeutic effect. Low-potency typical antipsychotic drugs, such as Thorazine, require higher doses to be effective but typically cause fewer motor side effects. However, higher dosages of these drugs increase sedative effects and can also cause high blood pressure, dry mouth, constipation, urinary retention, and blurred vision (Hogarty, 2002).

Table 2

First- Generation Antipsychotics

Chemical Name	Brand Name	Potency
Chlorpromazine	Thorazine, Largacil	Low
Thioridazine	Mellaril	Low
Mesoridazine	Serentil	Low
Fluphenazine	Prolixin, Permatil	High
Trifluoperazine	Stelazine	High
Perphenazine	Trilafon	High
Prochlorperazine	Compazine	High
Thiothixene	Navane	High
Haloperidol	Haldol	High
Primozide	Orap	High
Loxapine	Loxitane	High
Molindone	Moban	High

FGAs are also commonly referred to as conventional and “typical” based on their common mechanism of action: they block dopamine receptors in the brain. The dopamine hypothesis states that dopaminergic pathways in the brain are dysregulated in individuals with schizophrenia. The mesolimbic tract, which is responsible for arousal, memory, stimulus processing, and locomotor activity is flooded with too much dopamine resulting in positive symptoms. In contrast, the mesocortical tract, which is responsible for

cognition, communication, social activity, learning, and memory, is dopamine starved resulting in negative symptoms (Patterson et al., 2010). FGAs block dopamine receptors, thereby reducing or eliminating positive symptoms. However, the trade off for these drugs is that they can actually worsen negative symptoms and often cause a myriad of unwanted extrapyramidal signs (EPS) such as muscle rigidity, inexpressive facial features, and tremors (Preston, O'Neal, & Talaga, 2010).

Other side effects of FGAs. Probably the most feared adverse effect of FGAs is tardive dyskinesia (TD). Torrey (2006) describes TD as

. . . involuntary movements of the tongue and mouth, such as chewing movements, sucking movements, pushing the cheek out with the tongue, and smacking the lips. Occasionally these are accompanied by jerky, purposeless movements of the arms or legs, or rarely of the whole body (p. 212).

The symptoms of TD typically appear after an individual has been taking a first-generation antipsychotic for a prolonged period of time. There is no known treatment for TD and its effects are often irreversible.

Several long-range follow up studies have shown the incidence of TD to be 5% per year cumulatively, so that eventually 50 - 60% of people taking FGAs will develop TD over the course of their lifetimes (Hogarty, 2002). However, Torrey (2006) cites evidence that TD occurs as part of the disease process as well as being a side effect of conventional antipsychotics. "Most estimates of the incidence of tardive dyskinesia have assumed that all cases are drug-related when in fact a substantial percentage are not" (p. 212). The best treatment for TD is to switch the person to a second-generation

antipsychotic (SGA), especially clozapine (Clozaril). SGAs, also referred to as “atypical” and novel, will be discussed in detail later in this chapter.

Other neurological side effects of conventional antipsychotics include: acute dystonia (spasm of tongue, throat, face, jaw, eyes, neck or back muscles); akathisia (restlessness and an inability to sit still); and pseudoparkinsonism (rigidity in the limbs, tremors in either the limbs or hands, and shuffling gait). Unlike TD, these symptoms typically begin within 24 hours to 1 week of beginning medication (Preston et al., 2010). It is little wonder that 50% of patients who are prescribed typical antipsychotics discontinue their use within the first four to six months (Patterson et al., 2010).

Both FGAs and SGAs can cause a rare and potentially fatal condition known as Neuroleptic Malignant Syndrome (NMS). Although NMS can develop in individuals who have been taking medication for several years, it usually appears within 10 days of the introduction of a new antipsychotic. The symptoms, which develop over three days, consist of muscle rigidity, fever, confusion, coma, pallor, sweating, and rapid heart rate (Torrey, 2006). If NMS is not diagnosed and treated promptly, it can result in irreversible coma or death (Preston et al., 2010).

Most FGAs elevate the hormone prolactin, which can cause women to stop menstruating and prevent conception. Other less common side effects of conventional antipsychotics include sedation, dry mouth, blurred vision, constipation, weight gain, urinary retention, impaired sexual function, breast discharge in both men and women, rapid heartbeat or fainting, photosensitivity, liver damage, and eye damage.

Therapists treating clients on these medications should be especially mindful of the constellation of possible side effects as well as potential drug interactions and be able

to assess which warrant immediate medical attention. Any client experiencing confusion, falls, an inability to urinate, prolonged or severe constipation, rash, high fever, involuntary movements, jaundice, severe sedation, severe restlessness, or muscle spasm should be referred to a doctor for immediate medication reassessment (Preston et al., 2010).

Advantages of FGAs. Despite troubling side effects, FGAs are still frequently prescribed for the treatment of schizophrenia. FGAs are very effective in the treatment of positive symptoms, although (as noted earlier) they have little impact on negative symptoms and cognitive deficits. Also, since FGAs were the only treatment available for well over 30 years, physicians are experienced with their use. Since the patents have long since expired for most FGAs, inexpensive generic forms of the drugs are available unlike their cost-prohibitive second-generation counterparts. Until recently, FGAs were the only drugs available in a long-acting injectible form used to treat patients who are not medication compliant (Patterson et al., 2010). Depending on the drug, the costs of a 30-day supply of FGAs vary between \$82 and \$136 (The Medical Letter, 2003).

Second-Generation Antipsychotics

Clearly, there was a great deal of room for improvement in the treatment of psychoses, and clozapine produced results that raised expectations for the efficacy of antipsychotic therapy (Patterson et al., 2010). Following the release of clozapine, six other SGAs were introduced. SGAs available in the United States are listed in Table 3.

Table 3

Second-Generation Antipsychotics

Chemical Name	Brand Name
Aripiprazole	Abilify
Clozapine	Clozaril
Iloperidone	Fanapt
Olanzapine	Zyprexa
Paliperidone	Invega
Quetiapine	Seroquel
Risperidone	Risperdal
Ziprasidone	Geodon

Clozapine. Clozapine was the first antipsychotic to be effective against negative symptoms and is now the drug of choice for individuals who have been unresponsive to other antipsychotics. Studies have shown that 40% of those who have failed to respond to haloperidol respond to clozapine (Preston et al., 2010). Clozapine also has low incidence of EPS and akathisia and produces almost no TD (Torrey, 2006). The results of a worldwide study showed that clozapine, unlike most other antipsychotics, is effective in reducing suicidal behaviors (Patterson et al., 2010).

Dosage information and routes of administration for clozapine. Clozapine comes only in 25 mg. and 100 mg. tablets. For patients known to “cheek” their medications, it is available in a form that dissolves upon contact with saliva. The usual

dosage is 300-700 mg. per day (*PDR Drug Guide for Mental Health Professionals*, 2007).

Side effects of clozapine. Agranulocytosis, a potentially life threatening drop in white blood cells, requires patients to take weekly blood tests for the first six months and then every month indefinitely (Torrey, 2006). Preston et al. (2006) note the need for weekly blood tests can cost patients as much as \$10,000 per year.

Other side effects of clozapine include weight gain and increased risk for diabetes; myocarditis; sedation; seizures, especially at higher doses; dizziness and vertigo; and hypersalivation (Preston et al., 2010; Torrey, 2006). All persons taking clozapine should have their weight, blood sugar, and lipid levels regularly monitored (Preston et al., 2010). Women switching from a first-generation antipsychotic to clozapine are at an increased risk of pregnancy because clozapine does not elevate prolactin (Torrey, 2006).

Drug interactions of clozapine. Smoking and caffeine use are of particular concern for individuals taking clozapine since nicotine can increase levels of clozapine in the blood and caffeine can decrease it. Clozapine should not be taken with any other drug that reduces white blood cells, such as Tegretol. Clozapine and benzodiazepines can interact to cause respiratory arrest (Torrey, 2006.)

Other negatives. Clozapine may take months to be effective. Kane et al. (2001) reported response in 40% of subjects after 4 weeks of treatment with clozapine and in 60% of subjects by week 17. In 2006, the cost of a 30-day supply of the lowest usual dose of clozapine ranged between \$243 and \$432 depending on the route of administration (The Medical Letter, 2006).

Olanzapine (Zyprexa). Olanzapine was introduced in the United States in 1996. It is associated with a low incidence of EPS and TD. Research has shown olanzapine to cause modest improvement in negative symptoms. It may also have a mood stabilizing effect and has been approved by the FDA for treatment of mania (Torrey, 2006).

Dosage information and routes of administration for olanzapine. Olanzapine comes in 2.5 mg., 5 mg., 7.5 mg., 10 mg., 15 mg., and 20 mg. tablets. It also comes in a dissolvable form as well as short-acting intramuscular injectible preparation. The usual dosage is 10-20 mg. per day with doses up to 40 mg. for treatment resistant patients. (*PDR Drug Guide for Mental Health Professionals*, 2007).

Side effects and drug interactions of olanzapine. Olanzapine can cause sedation and should be taken at bedtime. Olanzapine can also cause significant metabolic effects such as weight gain, increased blood sugar, and diabetes. All persons taking olanzapine should have their weight, blood sugar, and lipid levels regularly monitored (Preston et al., 2010). As with clozapine, nicotine can increase levels of olanzapine in the blood and caffeine can decrease levels (Torrey, 2006). Women switching from a first-generation antipsychotic to olanzapine are at an increased risk of pregnancy because olanzapine does not elevate prolactin (Torrey, 2006). In 2006, a 30-day supply of the lowest usual dose of olanzapine was \$338 for tablets and the orally disintegrating form and \$378 for the liquid form (The Medical Letter, 2006).

Risperidone (Risperdal). Risperidone was introduced in the United States in 1994. It has several advantages over other SGAs. Risperidone increases prolactin levels, which means unwanted pregnancies are less likely to occur. Of all SGAs, it is least likely

to cause sedation. Risperidone may have a mood stabilizing effect and has been approved by the FDA for treatment of mania (Torrey, 2006).

Dosage information and routes of administration for risperidone. Risperidone comes in 0.25 mg., 0.5 mg., 1 mg., 2 mg., 3 mg., and 4 mg. tablets. It also comes in dissolvable and liquid forms as well as a long-acting injectable form. The usual dosage for risperidone is 2-6 mg. per day (Torrey, 2006).

Side effects and drug interactions of risperidone. Compared to other SGAs, risperidone is more likely to cause EPS and TD, especially at higher doses. Risperidone can cause weight gain, increased blood sugar, and diabetes, but less so than with clozapine and olanzapine. (Torrey, 2006). It can also cause decreased libido in both sexes and a cessation of menstrual periods in women. In 2006, the cost of a 30-day supply of the lowest usual dose of risperidone was \$260 (The Medical Letter, 2006).

Quetiapine (Seroquil). Quetiapine was introduced in the United States in 1997. Quetiapine is associated with a low incidence of EPS, akathisia, and TD. It may have a mood stabilizing effect and has been approved by the FDA for the treatment of mania. (Torrey, 2006).

Dosage information and routes of administration of quetiapine. Quetiapine comes in 25 mg., 100 mg., 200 mg., and 300 mg. tablets. The usual dose is 400-800 mg. per day. It may be used in higher doses for treatment-resistant patients. It is usually taken twice a day (Torrey, 2006).

Side effects and other issues with quetiapine. Quetiapine can cause weight gain, increased blood sugar, and diabetes, though not as much as clozapine and olanzapine. Quetiapine can cause sedation, which can be especially troublesome since it is taken

twice a day. Women switching from a first-generation antipsychotic to quetiapine are at an increased risk of pregnancy because quetiapine does not elevate prolactin (Torrey, 2006). An eye exam is recommended for persons starting quetiapine therapy and then periodically thereafter to ensure cataracts are not developing (Torrey, 2006). In 2006, the cost of a 30-day supply of the lowest usual dosage of quetiapine was \$325 (The Medical Letter, 2006).

Ziaprasidone (Geodone). Ziaprasidone was introduced in the United States in 2001. It is associated with a low incidence of EPS, TD, and akathisia. Of all SGAs, it has less of a tendency to cause weight gain. It also causes little sedation. (Torrey, 2006)

Dosage information and routes of administration for ziprasidone.

Ziaprasidone comes in 20 mg., 40 mg., 60 mg., and 80 mg. capsules as well as a short-acting intramuscular preparation. The usual dose is 80-160 mg. per day (Torrey, 2006).

Side effects and other issues with ziprasidone. Patients taking ziprasidone are at increased risk of developing potentially fatal cardiac arrhythmias because of prolonged cardiac electrical impulses known as QTc intervals. Ziprasidone should not be taken with any drug that increases the QTc interval as the combined effects could be life-threatening. A partial list of drugs that should not be taken with ziprasidone includes Anzemet, Avelox, chlorpromazine, halofantrin hydrochloride, Inapsine, Lariam, thioridazine, Nebupent, Orap, levomethadyl acetate hydrochloride, Pentam, probucol, Prograf, gatifloxacin, Trisenox, and sparfloxacin (*PDR Drug Guide for Mental Health Professionals*, 2007). Women switching from a first-generation antipsychotic to ziprasidone are at an increased risk of pregnancy because ziprasidone does not elevate

prolactin (Torrey, 2006). In 2006, a 30-day supply of the lowest usual dosage of ziprasidone was \$334 (The Medical Letter, 2006).

Aripiprazol (Abilify). Aripiprazol was approved for use in the United States in 2003. It is associated with a low incidence of EPS and akathisia. Like ziprasidone, it is less likely to cause weight gain than other SGAs (Torrey, 2006).

Dosage information and routes of administration for aripiprazol. It comes in 5 mg., 10 mg., 20 mg., and 30 mg. tablets. The usual dose is 15-30 mg. per day.

Side effects of aripiprazol. Aripiprazol can cause sedation. It may also cause an increase in blood sugar and diabetes. Women switching from a first-generation antipsychotic to aripiprazol are at an increased risk of pregnancy because aripiprazol does not elevate prolactin (Torrey, 2006). In 2006, a 30-day supply of the lowest usual dose of aripiprazol was \$338 (The Medical Letter, 2006).

Controversy About Prescribing Patterns

This chapter would be incomplete without addressing the controversy about prescribing patterns relating to first- and second-generation antipsychotics. Although numerous double-blind, industry-sponsored studies found better efficacy and tolerability for SGAs, many questioned the results citing a need for studies that were free of a connection to the pharmaceutical industry. In response to this, the NIMH sponsored two drug trials that compared FGAs with SGAs: Clinical Antipsychotic Trials of Intervention Effectiveness (CATIE) and Cost Utility of the Latest Antipsychotic Drugs in Schizophrenia Study (CUtLASS). Both trials had broad inclusion criteria and long follow-up, and tried to mimic routine clinical practice (Naber & Lambert, 2009).

The findings of both trials were surprising and have spurred much debate among mental health practitioners.

CATIE confirmed clozapine's superiority in efficacy to all other antipsychotics but found few differences between the treatment with other SGAs and a low dose of the FGA perphenazine. CUtLASS failed to show any difference between FGAs and SGAs (Gelenberg, 2009, p. 21).

Despite these findings, both physicians and the public have accepted that SGAs are superior to FGAs. Current standards of care both in the United States and abroad also show a clear preference for the use of SGAs (Naber & Lambert, 2009).

Length of Drug Therapy

It is impossible to reliably predict what drugs are going to work for any given individual or how long they will take to work. The process of finding the right drug and the right dosage to achieve the maximum clinical improvement is typically one of trial and error. For first-episode patients, the mean interval between beginning medication and achieving the maximum clinical improvement was 35 weeks (Torrey, 2006). Contrast these findings with those that show a substantial number of patients will discontinue medication within three to four months of beginning it, and the problem with medication compliance becomes readily apparent.

Treatment guidelines regarding the length of drug therapy vary depending on whether the person is suffering from a first or subsequent episode of psychosis. First episode sufferers should continue medication for one year after the symptoms of the initial episode have subsided. Individuals who have suffered two or more episodes should continue a maintenance dose of medication indefinitely. Weeks of trial and error can be

saved if persons with schizophrenia or their care givers keep a list of drugs tried, dosage levels, side effects, and response.

Chapter 4: Interventions and Literature Review

Nothing can really prepare a therapist for the first encounter with an actively psychotic client. The intensity of this experience and the feelings of incompetence and fear evoked may threaten to overwhelm the therapist. The Rogerian concept of “being with” the client takes on an entirely new meaning when the client’s world is a jumbled maze.

While my experience working with clients is limited, I can say unequivocally that never was the importance of the therapeutic alliance so poignantly demonstrated to me as it has been in my work with clients suffering from psychosis. Such clients are frequently terrified of the delusions or hallucinations they are experiencing. In addition, clients for whom the diagnosis is not new are often weary and suspicious of the efforts of well-meaning doctors, therapists, and family members who are unintentionally invalidating in their attempts to keep the client well.

As a therapist working with psychotic clients, I have found it necessary to balance my need to be genuine with my client’s need to be heard and validated. This is a tricky business because what a therapist cannot do is collude with the client by validating the existence of the hallucination or agreeing with the delusional thought process. Such collusion may help to build a relationship, but the shaky foundation will inevitably crumble as soon as the therapist must be honest with the client. However, the therapist who pushes the client to face reality by arguing against the client’s delusional thinking will often close down the lines of communication.

The delusions or hallucinations may not be real to the therapist, but they are very real indeed to the client, as are the intense feelings associated with them. A common

symptom experienced by individuals with schizophrenia is anosognosia, which is a failure to recognize one is ill. This lack of insight on the part of the client often serves as a major source of frustration to caregivers. Family members, and even therapists, often mistakenly interpret the lack of insight as nothing more than stubbornness on the part of the client. The power struggles that can ensue because of this interpretation only serve to further alienate the client from potential sources of help.

Amador (2007) offers an analogy that illustrates how very important it is for helpers to enter the world of the person experiencing delusional thinking or hallucinations.

Imagine I told you that you did not live where you live. You might laugh and tell me to stop joking around. But what if I produced a restraining order from a court that ordered you to stay away from what you told me was your home address? Now let's say you live with other people, perhaps members of your family, and you saw that they had signed off on this court order. What would you think? And imagine that you then called them to ask why they signed off and they said something like, 'You seem like a nice person, but if you keep coming around here we are going to call the police. You don't live here and we don't want to press charges, but we will if you put us in that position. Please stop calling here; you need help!' (p. 42).

Certainly I know where I live, where I work, what I do for a living, and who my family members and friends are. If I found myself in a situation described in the previous quote, I would likely think the people around me had gone crazy or that I was the subject of

some new reality TV show. Sadly, this is often precisely what a client who is experiencing delusions or hallucinations must endure.

What I was taught as part of my graduate training is no less true for this population: there is no single intervention or approach that is going to work for every person with this diagnosis. The treatment plan and interventions should be tailored to fit the client. However, the reality of working in a managed health care environment has a huge bearing on the type of treatment offered. Through narrow reimbursement policies, insurance companies create a “treat ‘em and street ‘em” approach that puts profits before the needs of the client. For instance, I was recently submitting billing at my internship site and noticed the “Cadillac” coverage carried by most of our clients reimburses for a maximum of 30 psychotherapy sessions per year! While it is beyond the scope of this project to provide critique of the insurance industry’s lack of mental health parity, the limitations mental health providers face still needs to be noted, especially when dealing with clients who are chronically ill. Numerous studies have shown that most people with schizophrenia must live with severe and ongoing social and vocational impairment even when taking medication (Coreyell et al., 1993; Racenstein et. al 2003).

The primary focus of this chapter is evidence-based psychosocial interventions for the treatment of schizophrenia. I am not, however, suggesting a cookbook style approach to treating clients with schizophrenia. The therapist should always be tailoring these interventions to meet the unique needs of each client. Patterson and Leeuwenkamp (2007) state:

Apart from the obvious need to select a form of psychosocial treatment that best addresses the needs of the individual patient, the usefulness of any psychosocial

therapy may be influenced by such things as the presence and severity of cognitive or affective disturbances, pharmacotherapeutic control of psychotic symptoms, and the extent of family support and participation in the patient's treatment (p. 109).

Family-Based Interventions

No mental illness can be conceptualized as having a singular impact on the person bearing the diagnosis. This is especially true for schizophrenia because it typically strikes early (affecting men in their late teens and women in their twenties) and often takes a chronic course. Families are frequently left with the emotional and financial burden of supporting the chronically mentally ill family member. Brent and Giuliano (2007) note that 70-80% of individuals with schizophrenia have on-going contact with their families. In a study of middle-aged and older adults in a 9-county region in Kentucky, Meeks and Murrell (1997) found 17% were living with family members and 88% lived in family-owned housing either alone or with others.

Contemporary family systems theory and expressed emotion. The application of traditional family systems theory and approaches in families with a schizophrenic member has unfortunately often served to alienate and demoralize caregivers (Miklowitz, 2004). Wynn, Shields, and Sirkin (as cited in Miklowitz, 2004) articulate the contemporary systems view.

Modern approaches . . . do not view the family as the primary causal agent in the patient's illness, nor do they view the patient as a 'scapegoat' for all the family's hidden dysfunctions. Instead, modern approaches focus on the family's way of reacting to and organizing itself around episodes of a member's psychiatric

disorder, and how these reactions protect against or contribute to the risk of occurrences. Illness is seen as a developmental event . . . in the family's life cycle that marks the beginning of an era in which relationships, family interactions, and future planning must change . . . to meet the new demands imposed by the illness (p. 669).

The modern systems view uses a biopsychosocial model to conceptualize schizophrenia. Schizophrenia in one family member will affect the psychological health and relationships of everyone in the family, and the response of family members to the mental illness will in turn impact the functioning and health of the ill member (Wynn et al., 1992)

High levels of expressed emotion (EE) among caregivers have been shown to be an important predictor of functional outcomes for individuals with schizophrenia. The communication style of high-EE relatives can be either overly critical/hostile or overly involved. In a meta-analysis of EE literature, Butzlaff and Hooley (1998) found individuals with schizophrenia who were in close association with high-EE relatives were twice as likely to relapse within 9-12 months following hospitalization than were those who with low-EE relatives.

Therapists working with families are cautioned not to interpret the data on EE in a way that suggests high levels of EE cause schizophrenia. As discussed in Chapter 2, much about the etiology of schizophrenia remains a mystery. If high EE were causal, one would expect to see all individuals from high-EE families become ill and relapse and conversely all individuals from low-EE families remain well. The available research does not support this view.

In some families, high levels of EE may well be a function of the longevity of the schizophrenic member's disorder (Miklowitz, 2004). Using the Camberwell Family Interview to assess EE, Hooley and Richters (1995) found that parents of offspring who had been diagnosed with schizophrenia for 3-5 years expressed an average of 15 critical comments (e.g., I dislike his self-serving attitude about things) as compared to an average of 4.2 critical comments of parents for parents who had coped with the diagnosis for 1 year. "After repeated exposures to episodes of illness, medical nonadherence, and low functioning, relatives may begin to blame the patient for his or her deviant behavior" (Miklowitz, 2004, p. 673).

As patients improve, EE levels change. Between 25-50% of parents of schizophrenic patients rated high EE/critical when their child was acutely ill are reclassified as low EE 1 year later when the patient is in remission. Although the levels of EE criticism drop over the time the most critical relatives are still the most critical even after the patient has remitted (Hooley & Richters, 1995).

The goals of family-based interventions are to decrease stress, reduce relapse rates, and improve social functioning among individuals with schizophrenia (Brent & Guiliano, 2007). Not surprisingly, stress management is the most critical component in both family and individual interventions.

Cognitive behavioral family therapy. Cognitive behavioral family interventions, also referred to as behavioral family therapy (BFT), have been found to be effective in reducing clinical, social, and family morbidity as well as enhancing the social recovery of persons with schizophrenia (Falloon & Coverdale, 1994). Meta-analyses found that BFT reduces relapse rates and the number and duration of re-hospitalizations (Pfammatter,

Junghan, & Brenner, 2006; Pilling et al., 2002; Pitschel-Walz, Leucht, Bauml, Kissling, & Engel 2002). BFT has also been shown to improve treatment adherence (Pharoah, Mari, Rathbone, & Wong, 2006; Pilling et al., 2002) and social functioning (Barrowclough & TARRIER, 1990; Chien, Chan, Morrissey, & Thompson, 2005; Falloon, McGill, Boyd, & Pederson, 1987; Li & Arthur, 2005; Magliano, Fiorillo, Malangone, De Rosa, & Maj., 2006).

As with other types of cognitive behavioral therapy, assessment plays a vital role in BFT. Baseline assessment of family functioning and continual review are the framework on which intervention strategies are constructed (Falloon & Coverdale, 1994).

During the assessment process the therapist works to build a therapeutic alliance with all family members. The alliance building stage allows the therapist to use the presenting problem as a starting point for the analysis of the functioning of the family. Each family member is interviewed individually. The therapist solicits specific information about each family member's observations, thoughts, and feelings about the presenting problem. The therapist also gathers information about each member's interaction within the family system as well as information about each family member's function in settings outside the family. (Falloon, Boyd, & McGill, 1984).

A key component of the assessment process is a determination by the therapist of the strengths and weaknesses of the family as a problem-solving unit. The therapist must also assess the quality of every day life of the family. "At the completion of the assessment, the therapist will be able to specify short-term personal goals of each family member and the conflicts and problems that may need to be resolved to achieve these goals" (Falloon & Coverdale, 1994, p. 215). Once the assessment is complete, the

therapist will use three types of interventions to address the goals of the family: psychoeducation, communication skills training, and problem-solving training.

With psychoeducation, the therapist presents straightforward information about the nature of schizophrenia and its treatment. The vulnerability-stress theory is outlined as a framework for integrating the benefits of combining drug therapy and psychosocial interventions to reduce symptoms and improve quality of life. The family member with schizophrenia is asked to describe his or her experiences with the disorder. Early warning signs of relapse are also discussed (Falloon & Coverdale, 1994).

There is considerable overlap between communication skills training and problem-solving training in BFT. The object of communication skills training is to help family members effectively communicate their feelings (both positive and negative) and needs. Skills are taught through instruction and coaching by the therapist and repeated practice by family members. Reinforcement of progress is identical to that used with social skills training, which is discussed later in this chapter. Homework is a key component that allows the skills to be generalized to every day interactions. Sometimes, when emotions are flaring, the therapist will need to block the negative expression and move the family into problem solving to address the underlying issue (Falloon & Coverdale, 1994).

Enhancing the efficiency of problem solving of the family unit is a key goal of BFT (D'Zurilla & Goldfried, 1971). The therapy sessions resemble training workshops where the family members are taught six steps of problem solving: defining the problem, listing alternative solutions, evaluating pros and cons of proposed solutions, choosing the best solution, planning the implementation of the solution, and reviewing the

implementation. The therapist does not involve him/herself in the actual problem solving but merely teaches the family the steps. Families are asked to meet once per week to engage in problem solving sessions. Only when stresses threaten to overwhelm the family problem solving capacity or when signs of relapse are detected does the therapist become an active participant in the problem-solving effort (Falloon & Coverdale, 1994).

Cognitive Behavioral Therapy for Schizophrenia

Cognitive behavioral therapy (CBT) was originally conceived as a treatment for depression and anxiety (Beck, 1976). Beck believed depression and anxiety were directly linked to faulty core beliefs and automatic thoughts. CBT helps the client identify beliefs and thoughts and then teaches the client to challenge their validity. The underlying premise of CBT is if we can change the way we think then we will change the way we feel.

CBT has since been modified for the treatment of schizophrenia as well as a host of other disorders.

CBT for the treatment of schizophrenia exists in several forms but all of them focus on developing a strong therapeutic alliance and psychoeducation. CBT attempts to achieve reduction of symptoms, reduction of relapse, and enhanced functional capacity by providing rational perspectives on the patient's experience of disease symptoms and response to them. (Patterson & Leeuwenkamp, 2008, p. 109).

Just as with many types of insight-oriented therapy, the cognitive behavioral therapist takes a non-threatening, non-judgmental, empathic stance with the client. This is the foundation on which the rest of the therapeutic dialog is built. The client is invited to

share his/her experiences with schizophrenia and to identify problematic symptoms. The therapist uses guided questions to help the client identify core beliefs about the symptoms as well as coping mechanisms used by the client to deal with those symptoms but does not challenge the symptoms as irrational (Kingdon & Turkington, 2008). Focus of therapy can include belief modification (where the therapist gently challenges the client's delusional beliefs); focusing/retribution (where the therapist encourages the client to focus on auditory hallucinations and reattribute them to an internal source); and normalizing psychotic experiences (where the therapist helps the client see the symptoms as a result of normal life stresses) (Kingdon & Turkington, 2008).

CBT has been found to offer a consistent improvement of positive and negative symptoms (Bechdorf, Kohn, Knost, Pukrof, & Kosterkotter, 2005; Bechdorf & Veith et al., 2005; Drury, Birchwood, Cochrane, & Macmillan, 1996; Gumley et al., 2006; Kuipers et al., 1997; Startup, Jackson, & Bendix, 2005; TARRIER et al., 1999; Temple & Ho, 2005) but only equivocal improvement of global and social functioning (Bechdorf & Kohn et al., 2005; Kuipers et al., 1997; Startup et al., 2005; Temple & Ho., 2005). Gumley et al. (2006) found the effects of CBT to be long lasting ranging from six months to two years after the cessation of treatment.

Social Skills Training (SST)

Building skills in individuals with schizophrenia is based on the assumption that coping and competence can override stress and vulnerability in reducing relapses and improving psychosocial functioning (Lieberman et al., 1986). For nearly 40 years a variety of SST approaches have been developed to address impairment in social skills.

Although skills training programs vary widely in content, duration, and the setting where they are implemented, they share a common set of strategies for teaching skills based on social learning theory (Bandura, 1969), including goal setting, role modeling, behavioral rehearsal, positive reinforcement, corrective feedback, and homework assignments to help improve generalization to the community (Kurtz & Mueser, 2008, p. 491).

An important area of focus often contained in SST interventions is social problem-solving skills training. Social deficits in individuals with schizophrenia include: difficulty initiating and sustaining conversations and an inability to achieve goals or have needs met in situations requiring social interactions. Ultimately, these impairments manifest themselves in profound difficulties in role functioning. For many individuals with schizophrenia, poor social functioning, odd interpersonal behavior, and stigmatizing experiences, in combination with social anxiety, contribute to isolation, inadequate social support, and vocational impairment, which, in an unfortunate cycle, diminish their opportunities to develop and improve their social skills (Morris, Bellack, & Tenhula, 2004, p. 84).

Since the development of social skills training procedures in the 1960s and 1970s, the effects of skills training on individuals with schizophrenia and other severe mental illnesses have been extensively studied (Bellack, Mueser, Gingerich, & Agresta, 2004). In a study of 84 men with schizophrenia, Liberman et al. (1998) found the group receiving 12 hours weekly of skills training focused on basic conversational skills, leisure and recreational skills, and medication and symptom management skills had better independent living skills and lower levels of distress than the control group receiving

occupational therapy. This study is of particular note because it showed the “very positive effects of social skills training on long-term outcomes of clients, with treatment effects still present 18 months after the end of the program” (Bellack et al., 2004, p. 24).

Tsang and Pearson (2001) evaluated the effects of a 10-week social skills training program on work outcomes in 97 people with schizophrenia. Clients were randomly assigned to one of three groups: skills training with monthly follow-up contacts, skills training without follow-up contacts, and no skills training.

Work outcomes were tracked for three months after the end of the intervention.

The results indicated that clients who received skills training plus follow-ups had the best outcomes, followed by those who received skills training alone, followed by those who received usual care. This study showed that skills training can be effective at helping clients achieve their vocational goals and that relatively modest follow-up efforts to help clients maintain their skills conferred an additional benefit in improving outcomes (Bellack et al., 2004, p. 25).

Cognitive Training

Researchers have used various terms to describe efforts to address cognitive deficits associated with schizophrenia including cognitive remediation, cognitive rehabilitation, and cognitive training (Twamley, Jeste, & Bellack, 2003). Cognitive deficits are thought to play a central role in the social disability and other problems of daily living as experienced by patients with schizophrenia (Bellack, Gold, & Buchanan 1999). Individuals with schizophrenia have been found to have impairments of pre-attentive abilities, attention, memory, learning, conceptualization, organization, planning,

self-monitoring, and flexibility of thinking (Gur, Ragland, & Gur, 1997; Heaton et al., 2001; Spaulding, Storms, Goodrich, & Sullivan, 1986; Storzbach & Corrigan, 1996).

The goal of cognitive training interventions is to improve neurocognitive abilities such as memory, attention, and overall executive functioning (EF). Maples and Velligan (2008) define executive functioning as “the ability to initiate, plan, and carry out goal-directed activities” (p. 166). Cognitive training interventions can be broadly divided into two categories: restorative and compensatory. Restorative interventions attempt to improve or restore cognitive abilities via repeated practice, and “compensatory strategies attempt to bypass cognitive deficits by establishing supports in the person’s environment that cue and sequence adaptive behavior” (Maples & Velligan, 2008, p. 165). Restorative interventions can be either paper-and-pencil exercises or computer-based.

Compensatory interventions have been used for years for individuals with traumatic brain injuries or mental retardation and include such things as the use of dose-specific pill containers to prevent the individual from inadvertently taking extra doses of medications or the use of alarms to prompt specific tasks necessary for daily functioning (Hart & Jacobs, 1993; Benedict, 1989). Maples and Velligan (2008) rightly observe “environmental supports are utilized every day by individuals who have no mental illness (e.g., hand-held computer to prompt appointments, buzzer to alert drivers that keys have been left in the ignition of a car” (p. 165).

In a meta-analysis of 17 studies, Twamley et al. (2004) found that “different types of approaches, whether computer-assisted or not, all have effective components that hold promise for improving cognitive performance” (p. 359). Olbrich and Mussgay (1990) found automated task practice yielded improvements in the cognitive domains of

vigilance, arithmetic, and verbal abstraction. Wykes, Reeder, Corner, Williams, and Everitt (1999) developed an individualized neurocognitive remediation program that incorporated errorless learning, immediate feedback, and non-didactic training which yielded improvements in tests of attention and working memory, cognitive flexibility, and planning. Computer-assisted automated task interventions were found to yield improvements in the areas of reaction time (Benedict & Harris, 1989), problem-solving ability and working memory (Bell, Bryson, Greig, Corcoran, & Wexler, 2001), vigilance and overall psychiatric symptoms (Medalia, Aluma, Tryon, & Merriam, 1998), and problem-solving ability and self-reported cognitive symptoms (Burda, Starkey, Domenguez, & Vera (1994).

Chapter 5: Case Conceptualization

This chapter discusses the case of an actual client. In order to protect the confidentiality of the both client and his family I have altered any details that might compromise their anonymity.

Presenting Concerns

Adam is a 20-year-old, single Caucasian male referred to my internship site for treatment of issues related to a previous diagnosis of Attention Deficit/Hyperactivity Disorder (ADHD), Obsessive-Compulsive Disorder (OCD), and learning disabilities. He presented with numerous problems related to anxiety, learning and attention impairment, impaired reality testing, and poor independent living skills. Adam has been a resident of this residential treatment program for over two years.

Significant History

Adam is the older of two boys and has an 18-year-old brother currently living at home completing his last year of high school. According to his mother, Adam was the product of an at-risk pregnancy characterized by first trimester bleeding and a planned Cesarean section. Post-delivery complications include an omphalocele, a type of abdominal wall defect in which the intestines, liver, and occasionally other organs remain outside of the body. Immediately after his birth, Adam underwent surgery to correct the defect. As a result of the surgery, certain developmental milestones were delayed including sitting without support, crawling, standing without support, and walking without assistance. The majority of other major developmental milestones were met without delay or complication.

Adam's temperament as a toddler was reportedly characterized by intense expression of emotion and reaction to change in routine. He was otherwise within normal limits for activity level, distractibility, mood, regularity and approach/withdrawal behaviors related to attachment. Adam's mother reports a long-standing history of academic difficulties dating back to kindergarten, which escalated during Adam's fourth-grade year. As a result of these difficulties, he was evaluated and was subsequently diagnosed with ADHD, Developmental Language Disorder, Coordination Disorder, and specific delays in development, including temporo-sequential organization and visual processing.

When Adam was 14, his family relocated from a Midwestern city where he had spent his entire life to a new home. After this move, he began to experience considerable adjustment issues as well as anxiety and depression. This event was pivotal in Adam's life and he recounts significant trauma around the move and loss of the "world he created" for himself there. When asked to name his best friends, Adam talks about people he knew from this period of his life with whom he has not spoken in more than six years. Adam's mother describes Adam as having always been "quirky." For instance, she recounted an odd behavior in which Adam would go into the bathroom and have lengthy conversations with Robin Hood.

Adam's academic difficulties continued throughout late elementary school, middle school, and high school, despite his receiving special education services. When he was 16, Adam was removed from high school in November of his junior year because OCD-related issues consumed him and he was fixated on the idea his school was full of germs. He also engaged in obsessive hand washing and showering to the point his hands

would bleed. It should be noted that his contamination fears seemed only to center around school. For a time he held a job at a local grocery where one of his duties was to empty trash bins, and he reported no problems performing this task.

Adam was subsequently diagnosed with Pediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcal infections (PANDAS). Swedo et al. (1998) coined the term PANDAS to describe a subset of childhood obsessive-compulsive and tic disorders triggered by a Streptococcus bacteria. A review of the literature regarding PANDAS indicates that Adam did not meet at least one of the necessary criteria for the diagnosis, prepubescent onset of symptoms, since the onset of his symptoms occurred when he was 16. Prior to admission at our facility, Adam was treated with penicillin for over two years. The hand washing and showering behaviors remitted within several months of his admission.

His parents report being happily married. However, it is noteworthy that the parents recently disclosed they were separated for a time when Adam was 10 years old while the father sought treatment for his alcoholism. Both parents express a great deal of shame around the issue of the father's alcoholism. Interestingly, Adam holds very rigid beliefs about alcoholism and drug addiction and frequently states that such people are bad and that his family does not want him to be associated with anyone who struggles with addiction.

Adam's mother describes the months he spent at home after leaving high school as a "nightmare" for the entire family. She reports Adam became increasingly hostile, oppositional, and withdrawn to the point that the family could not interact without arguments erupting. Adam recalls this period of time quite differently, denying there

were any problems and describing his family as “very happy and free of conflict.” On the recommendation of the family doctor and a psychotherapist, Adam was admitted for two months to an in-patient facility specializing in the treatment of OCD. His progress while there was minimal due in large part to his lack of motivation and engagement in the program as well as unwillingness to take medication. These factors are readily explained by Adam’s lack of acceptance of both his OCD and ADHD diagnoses. He did not believe there was anything wrong with him and as a consequence felt angry, betrayed, frightened, and abandoned by his parents. Immediately after discharge from that facility, Adam was admitted to this residential treatment program where he has remains.

Not long after his admission to this facility, Adam began to display a number of odd and alarming behaviors that could not be accounted for by OCD or ADHD. For instance, he was frequently observed walking throughout town talking and at times yelling to unseen persons. Because he initially spent many hours per day walking about in the community, he began to attract considerable negative attention with these behaviors. Within the first month of arriving at the program, Adam was escorted from the local public library by the police and permanently banned from re-entry. Library staff reported he had become belligerent over the use of library’s computers and began to threaten and curse at both staff and library patrons. In addition, he was repeatedly found roaming in clearly marked restricted areas of the library, which caused some library staff members to be frightened of him. The loss of library privileges was particularly upsetting to Adam because of his love of reading.

Adam has demonstrated delusional thinking, such as a belief his therapists and other staff are engaging in thought control. All of these factors coupled with his clear social and vocational impairment led clinical staff to suspect an additional diagnosis of undifferentiated schizophrenia. Adam's therapist immediately consulted with his psychiatrist about these symptoms, and then suggested to Adam he begin taking sub-therapeutic dose of Risperdal. Although Adam was very reluctant to take medication, stating fears he would become an addict, he agreed. As the dosage was slowly titrated up, many of his bizarre behaviors began to subside.

Over the past two years, Adam's personal grooming and hygiene have radically improved, but he must still be frequently prompted to shower and wear clean and appropriate clothing. Foot fungus has at times caused his feet to smell so badly his parents could not ride in the car with him. Without prompting, Adam appears oblivious to these issues. This is quite ironic, considering his past phobias about germs and obsessive washing rituals.

Social History

Adam has few, if any friends, and prefers to isolate himself from others. In the past he has walked several miles to go to a local bookstore where he can drink coffee and read science fiction geared for adolescents. He identifies as heterosexual and some time ago was discovered to be viewing adult pornographic sites. His parents report Adam has never had a girlfriend, yet Adam has recounted to me detailed stories of girls he has dated as well as his sexual experiences. His social isolation makes it doubtful he has ever engaged in sexual contact with another person and it is hard to know whether his accounts represent wishful or delusional thought processes.

Adam's fondest desire and only real goal is to complete his GED so that he can go back home and live with his parents. His parents remain ambivalent and, in fact, do not agree on whether Adam should live at home again once he leaves this program. His mother has stated that she does not want this, but Adam's father believes Adam should be offered the opportunity to return home. The couple is currently trying to work out what parameters and behaviors would be necessary in order for Adam to live at home again.

Behavioral Issues

Adam appears to be a young man who is at war with himself. He is emotionally immature. At times, he can present as being a very capable, articulate 20-year-old man. However, IQ testing has shown a large discrepancy between his verbal and performance IQs (121/87), a typical schizophrenic pattern on testing. Because of this Adam frequently presents as being far more capable than he actually is, which is usually to his detriment. However, Adam can shift rapidly from being a capable looking and sounding man, to behaving as a child who cries for his mommy and daddy. The "5-year-old" Adam behaves in ways that are totally incompatible with his adult counterpart. He has been known to throw chairs, to spit on the floor in front of staff, fart in session as an expression of displeasure with his therapist, scream at anyone who might happen to be near, and say rude and inappropriate things to people he does not like without provocation.

Adam believes that people who have been born with "mental defects" are not human and has frequently stated such people should be euthanized. This creates an obvious dilemma in light of his own diagnoses. Within the past 8 months he has slowly

gone from being unable to accept any of his diagnoses, to acknowledging them and seeking information about them, which has been a major therapeutic breakthrough.

Diagnosis

Axis I: 300.3 Obsessive-Compulsive Disorder
 314.01 Attention Deficit/Hyperactivity Disorder Combined Type
 315.1 Mathematics Disorder
 295.60 Schizophrenia, Residual Type
 Axis II: Deferred
 Axis III: Omophacele, repaired at birth
 Axis IV: Problems related to primary support group and education
 Axis V: GAF 45 (current)

Medications:

Risperdal	6 milligrams
Vyvance	30 milligrams
Prozac	40 milligrams

Discussion

Working with a client who is living with these kinds of impairments and has very little insight into them is incredibly challenging, difficult, and often frustrating. The work with all clients should begin with setting goals and structuring a treatment contract. This is not easily done when working with a client who does not want to be in treatment.

Many of my clients are initially resistant to treatment, but few take it to the level Adam has. He spent well over a year in treatment before he began to grudgingly acknowledge he might actually need to be here. Adam's apparent goal is to remain a child and return home to be cared for by his parents; Adam's parents' goal is for their son to assume his adult identity. As a therapist working with this family, it is often incredibly difficult to find the common ground between these conflicting positions.

The therapeutic gains for Adam and his family have been slow and hard won. Once Adam and his parents could accept the schizophrenia diagnosis the process of educating them about the disorder could begin. Slowly, the focus of family therapy sessions has shifted to psychoeducation. These parents continue to grieve the loss of their hoped-for son. Communication skills training with the family has netted a reduction in the high level of expressed emotion exhibited by Adams's parents and fewer arguments.

Adam's cognitive deficits make it highly unlikely that he will be able to attend college. The supports and accommodations necessary for him to take and pass the GED have been monumental. Although he has passed portions of the test, it is by no means certain he will be able to succeed on the math and science segments. Like many individuals with schizophrenia, one of Adam's core beliefs is that he is incompetent. Adam's impairments in working memory, attention, and problem solving are profound and only serve to reinforce this belief. Whenever supports and prompts are scaled back in attempt to encourage him to assume responsibility for this schedule and tasks, Adam appears unable to function. If he is to live independently, he may well require the type of prompts and memory aids used in cognitive remediation.

Helping the family and Adam set realistic goals is a major task of therapy. Recently Adam's father stated he didn't care if Adam mowed lawns for a living as long he was happy and productive. This shift in thinking may eventually serve to take the pressure off Adam to rise to the unattainable standards of his highly educated and successful parents. Based on his level of disability, Adam would most likely be eligible to receive disability benefits through the Social Security administration that would provide him with a monthly income as well as insurance coverage through Medicaid. It is only

recently that the family has been willing to discuss pursuing these benefits as well as investigating group living situations.

Adam's prognosis appears mixed. The early onset of his schizophrenia and his poor pre-morbid functioning may suggest a poor outcome. However, because he began drug therapy before he became floridly psychotic and has been engaged in CBT and SST, he might be expected to attain and maintain gains in his overall functioning.

All clients at this facility receive intensive psychotherapy (which includes individual and group sessions) as well life-skills training. Adam's treatment plan is individually tailored to address his specific needs. Because of the nature of Adam's deficits, the interventions selected for his treatment plan are primarily behavioral.

I have worked with this young man for 8 months and have expended considerable energy wondering how to best support him. I dare to hope his future will be brighter than any of us dream. He is by no means without strengths. Adam's quirky nature can be quite endearing. If properly harnessed, this could serve to draw helpers into his sphere. In addition, his interest in the internet could potentially be channeled into a vocational opportunity. If he were to gain employment where he was performing rote tasks using a computer, he might just find his niche. The work the family has been doing gives me reason to be optimistic that the love and support of Adam's family can be leveraged in positive ways that encourage Adam's self efficacy and success.

I remain grateful to Adam and his family for being open to a therapeutic relationship with me. I am likewise humbled by the sacred trust they continue to place in me. I have grown as a therapist and a person through my work with them.

Conclusion

The education and training I have received while attending JMU has laid a solid foundation for me as I begin the next phase of my counseling career. In truth, however, my education has only begun. Just as a house without a foundation will surely fall, a foundation without a house never fulfills its potential. I happen to be starting my counseling career later in life than many of my peers, and every day I am struck by how much I do not know. I am hungry for that knowledge as well as the experience to develop the tools of our trade.

This project has taught me several lessons. First, we are all responsible for our own professional competency and development. This means more than just attending conferences and workshops. It also means we should be active and eager students of the literature. During the countless hours spent reviewing the literature for this project, I discovered I actually enjoyed the process. For some of us, attaining this graduate degree will be the final chapter of our formal education. However, if we are to become masters of our craft, we must never cease to be students.

Second, good counselors view collaboration as an essential component of professional competency. Some collaborative relationships will be easy to form and maintain; others will not. My experiences collaborating with my more seasoned peers have been very positive. Before asking for their time and input, I make it a practice to do my own homework and be prepared. The professional respect that comes with successful collaboration is invaluable, especially to a beginning counselor.

Third, as beginning counselors, we must be mindful and intentional about identifying and assessing our own anxiety. This becomes particularly important when

dealing with clients who are chronically mentally ill. One way I manage my anxiety is by asking for feedback from my peers. When I become anxious about a client, I always ask myself how much of what I am feeling is actually about me and my worries about my professional competence. Because my relationships with my peers and supervisor are so strong, I feel comfortable asking them for feedback and, in turn, use that feedback to become a better clinician. For therapists working in an environment where peer collaboration is not possible, I recommend joining or starting an outside process group. None of us can do our best work if we are focused on our own anxiety rather than the process of attending to our client.

The final lesson can be summed up as follows: Always try the eggplant. Most of us remember a time from childhood when we rejected a new food without ever tasting it. The conversation in our house went something like this. Mom: "Just taste it." Me, "I don't like it." Mom: "How do you know you don't like eggplant if you haven't even tried it!?!?!?" Me: "It's purple. I don't like purple food." As I progressed through my graduate studies, I, like all counselors-in-training, developed a preference for certain therapeutic modalities and interventions. In my own case, prior to working with Adam, I whole-heartedly embraced interpersonal process but rejected behaviorism. While I still prefer insight-oriented therapy, both Adam and I have been well served by my willingness to abandon my negative biases and embrace behavioral interventions. This is a lesson I will carry with me as I continue my career.

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