THE MMPI-2 RESTRUCTURED CLINICAL (RC) SCALES

AND MEASUREMENT OF DEPRESSION

IN PATIENTS WITH EPILEPSY

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THE MMPI-2 RESTRUCTURED CLINICAL (RC) SCALES AND MEASUREMENT OF DEPRESSION IN PATIENTS WITH EPILEPSY

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This study examined the Restructured Clinical (RC) Scales of the Minnesota Multiphasic Personality Inventory-2 (MMPI-2) in a sample of 137 patients with epilepsy. The purposes of the current study were (1) to provide information regarding the psychometric performance of the RC Scales in an epilepsy population, and (2) to examine interpretive characteristics of the RC Scales in an epilepsy population. Internal consistency, internal discriminant validity, and external discriminant and convergent validities were assessed for select RC Scales. Results indicated that the RC Scales showed a modest improvement in general psychometrics over the Clinical Scales.

Specifically, the RC Scales displayed slightly better internal consistency and lower scale intercorrelations. Using the Inventory of Depressive Symptomatology, Self Report (IDS-SR) as a criterion, RCd ("demoralization") outperformed RC2 ("low positive emotions") and Scale 2 ("Depression") in predicting depressive severity. Throughout this study, results consistently indicated a close relationship between RC Scales measuring depression, anxiety, and health symptoms; this relationship seemed to reflect comorbidity of symptoms rather than substantial construct overlap. Additionally, several subgroups were defined based on Clinical Scale scores (i.e., a "conversion V" group, a "floating profile" group), and IDS-SR scores; RC Scale scores were examined in these groups. Subjects who demonstrated the conversion V profile tended to have large elevations on RC1 in the absence of comparable elevations on RCd and RC2, and a low score on RC3. Those with a floating profile showed only slight elevations on RCd, RC1 ("health complaints"), RC2, and RC8 ("aberrant experiences"). Subjects classified as at least moderately depressed using the IDS-SR, had only mild elevations on RCd and RD8, with highest elevation for RC1. In summary, the RC Scales showed acceptable performance in this sample, though they were differentially elevated in comparison to the Clinical Scales, indicating that even slight elevations on the RC Scales should be carefully considered in interpretation.

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CHAPTER I: INTRODUCTION

Epilepsy is one of the most common conditions seen by neurologists, with an approximate prevalence of 1% (Guberman & Bruni, 1999). In most developed countries, the incidence of epilepsy ranges from 40 to 70 per 100,000 per year (Guberman & Bruni, 1999). In these countries, incidence is highest in children up to one year old and in the elderly (Hauser & Hesdorffer, 2001). For a diagnosis of epilepsy, individuals must have at least two seizures that cannot be accounted for by drug reactions or other medically-induced causes. A seizure may be defined as "an abnormal and excessive discharge of brain neurons involving hypersychrony accompanied by some behavioral change" (Guberman & Bruni, 1999, p. 1).

Seizures are classified in a variety of ways, depending on origin ("focus") in the brain and the spread of discharge (Hopkins & Appleton, 1996). In partial seizures, the discharge remains in one part of the brain and spreads only locally to nearby neurons. Temporal lobe epilepsy (TLE) is the condition of repeated partial seizures in the temporal lobe of the brain. Partial seizures may be either simple, which are generally brief and involve no alteration of consciousness, or complex, which are associated with altered consciousness and often precipitated by "auras" (e.g., sense of déjà vu, lightheadedness, unusual emotions, altered vision/hearing, hallucinations; Guberman & Bruni, 1999). Partial seizures with secondary generalization occur when the discharge spreads not only to local neurons, but also to cells centrally grouped in the brain, that consequently spread the discharge throughout the brain. This leads to a convulsive ("tonic-clonic" or "grand mal") seizure if motor neurons are affected. Abnormalities in central neurons themselves lead to generalized seizures. Discharge in these neurons will spread throughout the brain

and lead to either tonic-clonic or absence seizures. Absence ("petit mal") seizures are characterized by brief interruptions in consciousness, during which the individual will "cease what she is doing, stare, look a little pale, perhaps flutter her eyelids, and drop her head slightly forwards" (Hopkins & Appleton, 1996, p. 13). These seizures are predominantly seen in children.

Seizures may have a wide variety of etiologies. Almost any type of lesion affecting the cortex can lead to epilepsy or seizures (Guberman & Bruni, 1999). Some of the more common causes of seizure include congenital malformation, anoxia, head trauma, tumors, encephalopathy, degenerative disorders, and alcohol abuse (Hopkins & Appleton, 1996). In a relatively small percentage of cases, genetic factors may play a role in the development of epilepsy (Hopkins & Appleton, 1996). Whatever the cause, epilepsy can have serious psychosocial and neurophysiological ramifications. Research has indicated that epilepsy patients are more likely to experience depression, anxiety, and suicidality (among other psychiatric conditions) than the general population (e.g., Jones et al., 2005a; Matthews & Barabas, 1981; Mendez, 1988; Swinkels, van Emde Boas, Kuyk, van Dyck, & Spinhoven, 2006). The comorbidity of these psychiatric conditions may lead to significant complications in the diagnosis and treatment of epilepsy, as well as psychological or social problems (Grabowska-Grzyb, Jedrzejczak, Naganska, & Fiszer, 2006).

A link between epilepsy and psychiatric disturbances has long been assumed. For many years, seizures were viewed as behavioral disorders rather than the result of abnormal neurophysiology (Mendez, 1988). The term "lunatic" was originally synonymous with "epileptic," and the Greek physician Galen (129 – ca. 216 AD)

speculated that the moon regulated seizures (Mendez, 1988). In 1904, Kraepelin formalized the behavioral aspects of epilepsy in his textbook *Psychiatrie* by describing epileptic patients as "'pedantically precise,' 'irritable,' 'awkwardly helpful,' and predisposed to develop dementia praecox" (Mendez, 1988, p. 193). The notion of an epileptic personality is no longer held, though researchers have identified certain behavioral tendencies toward which epilepsy patients may be predisposed, especially in the case of TLE. Lesions in the temporal lobe may often affect the limbic system, a set of structures associated with behavior and emotion (Helmstaedter, Sonntag-Dillender, Hoppe, & Wyler, 2004). By the early 20th century, effective treatments, precipitated in part by the development of antiepileptic drugs (AEDs) and electroencephalography (EEG), led many neurologists to believe that patients with epilepsy were otherwise normal when their seizures were controlled (Mendez, 1988). However, following a report by Gibbs in 1951, the later half of the 20th century saw the re-popularization of the idea that epilepsy can change personality (as cited in Ritaccio & Devinsky, 2001). At that time, Gibbs described the occurrence of behavioral disturbances in up to one-third of his patients with TLE (Ritaccio & Devinsky, 2001). The coincident development of the knowledge that the limbic system plays a large role in emotional regulation led many to believe that patients with TLE might indeed suffer from certain common types of personality disturbances (Ritaccio & Devinsky, 2001).

The notion that epilepsy is somehow linked to psychopathology has been tested repeatedly. For instance, in a study by Swinkels, Kuyk, de Graaf, van Dyck, and Spinhoven (2001), the Composite International Diagnostic Interview (CIDI) was used to interview 209 epilepsy patients, and results were compared to the general population.

The findings indicated that the most common psychiatric complaints were of anxiety and mood disorders. The prevalence of these disorders in the year prior to being interviewed was 25% for anxiety and 19% for mood, a rate that is significantly higher than for the general population.

The measurement of psychopathology is particularly important in patients with epilepsy, as identification of depressive or anxiety symptoms can be critical for effective diagnosis and intervention—whether surgical, pharmacological, or psychiatric (Grabowska-Grzyb et al., 2006). Substantial physical, psychological, and social ramifications are often associated with a diagnosis of epilepsy (Alonso et al., 2006; Griffith et al., 2005). However, as noted by Jones and colleagues (2005a), Axis I morbidity is often unrecognized and untreated in patients with epilepsy. Standardized structured interviews such as the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I) are often too time-consuming for routine clinical use (Jones et al., 2005a). If neuropsychological assessment is performed, some measure of emotional functioning is often included. The Minnesota Multiphasic Personality Inventory – Second Edition (MMPI-2; Hathaway & McKinley, 1943)—a "gold standard" in the assessment of psychopathology—is often used to detect psychiatric comorbidity, but numerous researchers have identified concerns regarding its use, both in terms of general psychometrics (e.g., Rogers, Sewell, Harrison, & Jordan, 2006; Tellegen et al., 2003; Tellegen et al., 2006), and in its applicability to epilepsy patients (e.g., Derry, Harnadek, McLachlan, & Sontrop, 1997; Derry et al., 2002; Dikmen, Hermann, Wilensky, & Rainwater, 1983; Karzmark, Zeifert, & Barry, 2001).

This study will examine several diagnostic tools used as part of a neuropsychological battery in a sample of patients with epilepsy. Two primary sets of scales, the MMPI-2 Clinical Scales, and Restructured Clinical (RC) Scales, will be evaluated in terms of utility for measuring mood symptoms in patients with epilepsy. The Inventory of Depressive Symptomatology – Self Report (IDS-SR; see Appendix A) will be used as an external correlate for measuring convergent validity for measurement of depressive symptoms

CHAPTER II: LITERATURE REVIEW

An extensive literature has indicated that psychiatric comorbidity, particularly of depression and anxiety, is seen relatively frequently in patients with epilepsy (e.g., Altshuler, Devinsky, Post, & Theodore, 1990; Devinsky et al., 2005; Gilliam & Kanner, 2002; Grabowska-Grzyb et al., 2006; Jones et al., 2005a; Kanner & Nieto 1999; Kanner & Palac, 2000; Kobau, Gilliam, & Thurman, 2006; Strauss, Wada, & Moll, 1992). Research in a sample of epilepsy patients has indicated that nearly half of the variance in self-reported health-related quality of life can be accounted for by ratings of mood (Gilliam & Kanner, 2002). Studies have also indicated that the presence of mood symptoms in patients with epilepsy may cause significant diagnostic, therapeutic, or social difficulties (Grabowska-Grzyb et al., 2006). Consequently, the identification of depression and anxiety symptoms is an important task in the psychological testing process with epilepsy patients.

Depression and Epilepsy

Mood disorders are the most commonly presenting difficulties seen in outpatient mental health settings, with major depressive disorder (MDD) being the most frequent of these (Maxmen & Ward, 1995). The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) lists the criteria for a major depressive episode (MDE) as being five or more of the following symptoms, with either of the first two necessarily occurring: (1) depressed mood; (2) loss of interest or pleasure; (3) weight loss or gain; (4) insomnia or hypersomnia; (5) psychomotor agitation or retardation; (6) fatigue; (7) feelings of worthlessness or guilt; (8) diminished concentration; or (9) thoughts of death (American Psychiatric Association, 1994). Of the first two symptoms,

many contemporary theorists believe that anhedonia (i.e., loss of interest or pleasure) is the most distinct core feature of MDD, whereas depressed mood may be more pervasive amongst psychiatric disorders (e.g., Ben-Porath, 2008; Clark & Watson, 1991; Tellegen et al., 2006; Watson, 2005). Consequently, MDEs characterized by anhedonia may be more likely to contribute to a larger pattern of MDD. Patterns of depressive symptomatology can be seen amongst many patients with epilepsy, particularly in those patients with partial epilepsy of the temporal or frontal lobes (Kanner & Nieto, 1999).

The comorbidity of epilepsy and depression has been recognized for thousands of years. Sometime around 400 BCE, Hippocrates wrote that "melancholics ordinarily become epileptics, and epileptics melancholics: what determines the preference is the direction the malady takes; if it bears upon the body, epilepsy, if upon the intelligence, melancholy" (Gilliam & Kanner, 2002). Several hundred years later, in his treatise *Epilepsy and Melancholy*, Galen wrote that both disorders arise in the brain and that they may have similar causes (Gilliam & Kanner, 2002).

Today, epidemiological studies have revealed that depression is indeed one of the most common comorbid conditions with epilepsy, with estimates of the prevalence of comorbidity ranging from 20% to 80%, depending on type and intractability of seizures (Kanner & Nieto, 1999; Kanner & Palac, 2000; Grabowska-Gryzb et al., 2006). According to Mendez (1988), patients with epilepsy are four times as likely to be hospitalized for depression as similar non-epilepsy controls. Kanner and Palac (2000) report that endogenous (i.e., biologically-based, as opposed to reactive) depression has been found in up to 40% of patients in some epilepsy centers, that 20% of TLE patients become depressed, and that up to 62% of patients with complex partial seizures (CPSs)

have a history of depressive episodes. The same authors also note that up to 80% of epilepsy patients report "feelings of depression."

Comorbid depressive symptoms can be classified in terms of the temporal relationship between the onset of depressive symptomatology and the occurrence of seizure. Ictal depression is a direct result of the seizure, peri-ictal depression is comprised of symptoms that directly precede or follow seizure occurrence, and interictal depressive symptoms occur in between and independently of seizures. Ictal depression is predominantly the clinical expression of a simple partial seizure, and may be its only symptom (Kanner & Palac, 2000). Psychiatric symptoms occur in an estimated 25% of seizure auras, and of these, 15% may involve mood disruptions (Gilliam and Kanner, 2002). Peri-ictal depression presents primarily as dysphoric mood either directly before or after a seizure, and symptoms may extend for hours or days (Kanner & Palac, 2000). Affecting up to 60% of patients with CPSs, interictal depression is the most common manifestation of mood disorder among epilepsy patients (Kanner & Nieto, 1999). Although interictal depression often presents as atypical depression, these episodes may also present as MDD, dysthymia, or bipolar disorder (Kanner & Palac, 2000).

The DSM-IV characterizes atypical depression as the following symptoms during a MDE or dysthymic disorder: mood reactivity and two or more of either (1) weight gain or increase in appetite; (2) hypersomnia; (3) leaden paralysis; or (4) sensitivity to interpersonal rejection that impairs social or occupational functioning (American Psychiatric Association, 1994). Atypical presentation of interictal depression is common. In a study by Mendez, Doss, Taylor, and Salguero (1993), 50% of the depressive episodes of epilepsy patients had to be classified as atypical depression, based upon

DSM-III criteria. The German psychiatrist Emil Kraepelin first described atypical interictal depression in 1923, referring to it as interictal dysphoric disorder (IDD; as cited in Kanner & Nieto, 1999). The presentation of IDD closely resembles dysthymic disorder because of a chronic course that is interrupted by short, symptom-free periods (Kanner and Palac, 2000). Blumer, Montouris, and Hermann (1995) predicted that one-third to one-half of all epilepsy patients suffer from atypical symptoms (e.g., chronic dysthymic state, irritability, anxiety, and/or somatoform symptoms) of sufficient severity to warrant pharmacologic treatment.

Watson (2005) proposed a tripartite theory of emotional disorders that may help explain the presentation of atypical depression. In this model, emotional disorders are split into "bipolar disorders," "distress disorders," and "fear disorders." Depression, dysthymia, and generalized anxiety all fall under the umbrella of "distress disorders." Through the lens of this tripartite theory, it is easy to see how, as members of the same class of emotional disorders, anxiety symptoms and depressive symptoms might "bleed" together into an atypical depression. Many rating scales, such as the Hamilton Rating Scale for Depression (HRSD), do not inquire about symptoms of irritability (a major symptom of IDD), and thus should be used with caution when identifying depression in populations with epilepsy (Kanner & Nieto, 1999). With wider and more evenly-weighted symptom coverage, a measure such as the Inventory for Depressive Symptomatology (IDS) may be an ideal measure of Watson's (2005) "distress disorders." Indeed, Ben-Porath (2008), one of the authors of the MMPI-2 Content Scales and Restructured Clinical (RC) Scales, has asserted that inventories such as the HRSD may actually be better measures of distress or demoralization than major depression.

Besides temporal onset, comorbid depression may also be classified in terms of etiology. Kanner and Nieto (1999) categorized the depressive symptoms in epilepsy patients in terms of four different causes. First, depression may be the result of an intrinsic process related to the neurophysiologic changes that seizures can create in the limbic system over time. Research has indicated that while patients with epilepsy tend to have higher prevalence of mood disorders than patients with comparable physical disabilities, the rates are similar to those of patients with other types of neurologic dysfunction (Altschuler et al., 1990). Therefore, mood disorders may be due more to changes in brain function than the psychosocial consequences of disability. Second, antiepileptic drugs (AEDs) might iatrogenically induce depressive symptoms. Third, the symptoms might be the psychosocial result of a reactive process to chronic difficulties that necessitate multiple adjustments. Finally, genetic factors that may be involved in the patient's vulnerability to these symptoms must also be taken into account.

Biologic contributions to depression in patients with epilepsy may come in various forms. According to Robertson (1989, p. 199), "abnormalities of many neurotransmitters have been implicated in both illnesses." Though exact mechanisms are unknown, the levels of certain neurotransmitters in the brain seem to provide a link between depression and epilepsy. Studies examining the levels of a metabolite of norepinephrine (NE) in cerebrospinal fluid have linked lower levels of the metabolite to disruptions in mood (Robertson, 1989). Studies using rats have found that injection of a drug that reduces levels of NE in the brain significantly lowers electroconvulsive threshold and also reduces the efficacy of anticonvulsants (Robertson, 1989). A similar pattern can be seen for serotonin (SE). Decreased levels of a principal product of SE

metabolism have often been reported in patients with depression (Robertson, 1989). Selective serotonin reuptake inhibitors (SSRIs), a typical form of antidepressant medication, work by maintaining increased levels of SE in the brain. Regarding epilepsy, depletion of SE has been linked to a lower convulsive threshold, and increased serotonergic transmission is associated with a raised threshold (Robertson, 1989). The inhibitory neurotransmitter GABA has also been implicated. A variety of studies have found significantly reduced levels of GABA in patients with depression (Robertson, 1989). Like NE and SE, GABA also seems to be important in the neurochemistry of epilepsy. Inhibitory GABA-ergic neurons appear to act preventatively toward seizure buildup, and impairment of GABA functioning has been shown to lower seizure thresholds: reduction of GABA by 40% can induce generalized tonic-clonic seizures (Robertson, 1989). Additionally, some toxins which block GABA receptors are convulsants (Robertson, 1989). In summary, decreased levels of NE, SE, and GABA all appear to be related to depression and also to increased vulnerability to seizures.

The issue of suicide deserves special consideration for patients with epilepsy. According to Kanner and Nieto (1999), the rate of attempted suicide is four times as great in epilepsy patients as in the general population. Mendez (1988) has additionally reported that in a sample of TLE patients, suicide risk was measured as 25 times greater than that of the general population. The unpredictability of seizures, restrictions on normal activities (e.g., driving), reliance on medications, possible transmission of illness to children, and social rejection can all present major stressors for individuals with epilepsy (Matthews & Barabas, 1981). These factors and others could lead to the frustration and feelings of defeat that might often accompany epilepsy. Some

researchers, coining the term “epileptic despair,” cite the “severe social limitations, sharply alternating periods of health and prostrating illness, and oftentimes the belief in evitable mental deterioration” as key factors that may predispose patients toward suicide (Lennox & Lennox, as cited in Matthews & Barabas, 1981, p. 516).

Anxiety and Epilepsy

The DSM-IV defines around a dozen anxiety disorders, a handful of which seem to commonly co-occur with epilepsy (American Psychiatric Association, 1994; Beyenburg, Mitchell, Schmidt, Elger, & Reuber, 2005). In a study examining psychiatric morbidity in epilepsy, Jones and colleagues (2005a) used structured clinical interviews to find the rate of Axis I DSM-IV disorders in an epilepsy patient sample. Of 174 subjects, around 52% reported sufficient symptoms for diagnosis of some form of current anxiety disorder: 3.4% reported panic disorder; 15.5% reported agoraphobia; 10.9% reported social phobia; 3.4% reported obsessive-compulsive disorder (OCD); 5.7% reported post-traumatic stress disorder (PTSD); and 13.2% reported generalized anxiety disorder (GAD). The 2004 HealthStyles survey, a large mail panel survey designed to be representative of the U.S. population, found that adults with self-reported epilepsy were twice as likely to have experienced anxiety in the previous year as adults without epilepsy (Kobau, Gilliam & Thurman, 2006). In an experiment examining levels of psychiatric symptoms in epilepsy patients, Devinski and colleagues (2005) found that moderate to severe levels of anxiety existed in 24.7% of pre-operative epilepsy patients. Cramer, Brandenburg, and Xu (2005) used the Hospital Anxiety and Depression Scale to assess epilepsy patients with partial seizures. Results indicated that 25% of respondents

experienced mild anxiety, 16% experienced moderate anxiety, and 7% experienced severe anxiety.

The DSM-IV lists the criteria for GAD as clinically significant distress including prolonged anxiety and worry, and at least three of the following six symptoms: (1) restlessness; (2) fatigability; (3) difficulty in concentration; (4) irritability; (5) muscle tension; and (6) sleep disturbances (American Psychiatric Association, 1994). Considerable worry can result from any medical investigation into serious ailment, and the common diagnostic delays in disorders such as epilepsy can heighten this anxiety (Beyenburg et al., 2005). Fear of future seizures, apprehension about disease progression, and fear of complications may all contribute to GAD following a diagnosis of epilepsy (Beyenburg et al., 2005).

Phobias are characterized by extreme or unreasonable pervasive fears of a particular object or situation (American Psychiatric Association, 1994). Individuals with epilepsy may experience fears related to seizures occurring in an unfamiliar or uncontrolled environment, which, if taken to an extreme, could lead to agoraphobia (Beyenburg et al., 2005). Fear of public embarrassment may likewise escalate into social phobia (Beyenburg et al., 2005). Agoraphobia may affect over 15% of epilepsy patients, and social phobia may affect nearly 11% of epilepsy patients (Beyenburg et al., 2005).

Obsessive-compulsive disorder (OCD) is characterized by the presence of either (1) pervasive thoughts, impulses, or images that are intrusive and cause distress, (2) repetitive behaviors or mental acts that the person is driven to perform in order to prevent or reduce stress, or both (1) and (2) (American Psychiatric Association, 1994). Beyenburg and colleagues (2005) report that in one study, 22% of epileptic participants

reported obsessive-compulsive symptoms in the clinical range, whereas only 2.5% of controls reported clinical levels of symptoms. Obsessive thoughts may also be part of an aura in seizures of the temporal lobe (Beyenburg et al., 2005).

Panic attacks are distinguished by sudden, discrete periods of fear or discomfort accompanied by somatic complaints which may include palpitations, shortness of breath, dizziness, trembling, and sweating, among others (American Psychiatric Association, 1994). Recurrent panic attacks with significant distress related to the attacks (e.g., concern about additional attacks) is significant for a diagnosis of panic disorder (American Psychiatric Association, 1994). Panic attacks are up to six times as common in epileptic populations as in controls (Beyenburg et al., 2005). Differential diagnosis of panic attacks and seizures may be difficult, and panic disorder is the most likely of all anxiety disorders to be generated by seizures (i.e., "ictal fear"; Beyenburg et al., 2005).

Like depression, anxiety may occur at a variety of times in relation to temporal onset of seizures. Anxiety may occur as an ictal, post-ictal, or interictal phenomenon (Beyenburg et al., 2005). Ictal anxiety (i.e., "ictal fear") is most often experienced by patients with simple partial seizures of temporal lobe origin and manifests as feelings of fear or panic. In fact, experiential auras have been found to be associated with a high prevalence of both depression and anxiety (Mula et al., 2006). This type of anxiety is most easily diagnosed when it occurs repetitively, shortly before the onset of partial seizures (Beyenburg et al., 2005). Post-ictal anxiety occurs in the period directly following a seizure, but is not thought to be the direct result of electric discharge in the brain. This type of anxiety is commonly comorbid with dysphoria or depression. Anxiety sometimes occurs in isolation post-seizure, but an expression with mixed mood

symptoms is much more common. In a study of 100 adults with epilepsy, Kanner, Soto, and Gross-Kanner (2004) found that 45 individuals presented with post-ictal anxiety in a 72-hour period following a seizure. Specifically, 33 reported constant worrying, 29 reported agoraphobic symptoms, 20 reported fear of seizure recurrence, and 26 reported anxiety due to self-consciousness. Interictal anxiety is difficult to differentially diagnose from independent anxiety, especially because symptoms do not closely follow the seizure (Beyenburg et al., 2005). Interictal anxiety may be linked to seizures in a couple ways. Increased stimulation of the amygdala could increase susceptibility towards anxiety. In addition, interictal anxiety may be an expression of accumulated worries or fears about the condition and associated complications. Interictal anxiety might also lead to agoraphobia or social phobia (Beyenburg et al., 2005). People with anxiety tend to overestimate the risk of situations triggering their anxiety and underestimate their ability to cope with their anxiety once triggered (Beyenburg et al., 2005). These distortions can lead to anticipatory anxiety, avoidance, and isolation.

There are a variety of factors that may cause anxiety; explanations for the etiology of anxiety will vary depending on the school of thought. Behaviorists maintain that anxious reactions to certain stimuli are the result of repeated and historically negative associations with those stimuli (Maxmen & Ward, 1995). Anxiety of this type may lead to the formation of social phobia or agoraphobia, as described above. Biological theorists claim that anxiety originates in the physical structures of the brain, which in turn produces symptomatology (Maxmen & Ward, 1995). The agitation of the amygdala and subsequent susceptibility to interictal anxiety (described above) is one example. Additionally, investigation has revealed that the locus coeruleus (LC), production site of

much of the brain's NE, is likely involved (Beyenburg et al., 2005). Stimulation of the LC via the drug yohimbine increases subjective anxiety, and inhibition of the LC suppresses anxiety. In addition, benzodiazepine (BZ) and GABA receptors may be involved. In this case, it appears that BZ somehow augments a GABA-inhibitory system that has anti-anxiety effects; the exact mechanism is not known (Beyenburg et al., 2005). In addition to the NE system and the BZ and GABA system, it seems that SE is also related to anxiety. By increasing the extracellular level of serotonin in the brain, selective serotonin reuptake inhibitors (SSRIs) can be effective in treating panic disorder, OCD, and GAD (Maxmen & Ward, 1995). Biological explanations for anxiety may be especially pertinent for people with epilepsy because insofar as seizures may alter brain physiology, they may also cause anxiety if it is regulated by the affected structures.

Research has indicated that several psychosocial factors may play a mediating role in the development of anxiety in epilepsy populations. In separate studies, Raty, Soderfeldt, and Larsson (2007) and Velissaris, Wilson, Saling, Newton, and Berkovic (2007) have found convergent evidence for the development of differing trajectories for the development of anxiety due to coping style in epileptic patients. Velissaris and her colleagues (2007) found that two different coping trajectories develop in response to perception of control after newly diagnosed seizures. Following a new diagnosis, patients tended to diverge into groups that either experienced limited loss of sense of control or pervasive loss of control. Patients who perceived a higher loss of control displayed increased awareness of susceptibility and mortality, a higher fear of recurrence, and anxiety. Räty and her colleagues (2007) administered a questionnaire asking young adults to describe daily life with epilepsy and performed a content analysis to separate the

emotions described. In this case, two different groups appeared, with some patients regarding themselves as generally healthy and some that felt that they were handicapped. The "healthy" group's members tended to be more active and flexible, focusing on possibilities and planning ahead. The "handicapped" group held members who seemed to be negatively resigned to epilepsy, focusing on obstacles and being critical of themselves. This group also expressed anxiety (e.g., "I worry that my friends or other people would…notice something regarding the epilepsy condition and how that would affect my working situation and relations with others"), despair, and fears. In the case of both of these studies, coping mechanisms seem to shape how epilepsy affects the individual, with anxiety being a common outcome of poor coping. O'Neill (2005) also examined factors that might exacerbate psychological sequelae of epilepsy. After examination of a sample of individuals with epilepsy, psychosocial factors (e.g., mental health, cognitive functioning, physical health) were found to be significantly correlated to both anxiety and depression. Of these psychosocial factors, the domain of mental health (including emotional well-being, emotional role-limitations, social support, fatigue, and overall quality of life) had the highest predictive power for anxiety and depression. Furthermore, research indicating that epileptogenic focus may have little association with anxiety level might lend credence to the idea that psychosocial factors mediate anxiety level (e.g., Altshuler et al., 1990; Swinkels et al., 2006).

Psychological Testing and Epilepsy

Psychological testing may be utilized for patients with epilepsy for a variety of reasons. Psychological testing can be helpful diagnostically, aid in research, and assist mental health providers in a variety of ways. Diagnostically, psychological testing has

been used for the differential diagnosis of epileptic and psychogenic seizures (e.g., Cragar, Berry, Fakhoury, Cibula, & Schmitt, 2002), location of seizure foci (e.g., Paradiso, Hermann, Blumer, Davies, & Robinson, 2001), diagnosis of learning disorders and other neurological impairments comorbid with seizure-related brain dysfunction (e.g., Binnie, Channon, & Marston, 1990), and diagnosis of psychiatric concerns such as depression (e.g., Griffith et al., 2005), suicidality (e.g., Matthews & Barabas, 1981) or anxiety (e.g., Swinkels et al., 2006).

In research, psychological testing is used for evaluation of surgical procedures through measurement of psychiatric symptomatology (e.g., Alonso et al., 2006; Rossitch & Nashold, 1991), for trials of AEDs and measurement of their psychological effects (e.g., Aldenkamp et al., 1993), and evaluations of alternative treatments (e.g., Riklan, Cullinan, Shulman, & Cooper, 1976).

Living with epilepsy is often associated with substantial medical, psychological, and social consequences (Alonso et al., 2006). Health providers often use psychological testing in order to better aid in the treatment of patients with epilepsy. For instance, quality of life (QOL) is often measured as an overall indication of a patient's "perceived well-being in physical, mental, and social domains of life" (Devinski et al., 1995, p. 1090). Though seizures usually are brief and occur infrequently, their social and psychological sequelae can be persistent (Devinski et al., 1995). QOL assessment may be useful in many areas, such as determining ideal AED treatment based on the day-to-day impact of side-effects. The MMPI-2 is frequently used to measure psychiatric symptoms or complaints that influence QOL, (Swinkels et al., 2004). However, because the MMPI-2, and in fact the majority of psychiatric self-report inventories, are not

designed with an epilepsy population in mind or normed for epilepsy patients, they have sometimes been deemed at a disadvantage for use with epilepsy patients (Swinkels et al., 2004). To counter this, a number of epilepsy-specific inventories have been created.

Bear and Fedio (1977) created the Bear-Fedio Inventory in an attempt to measure the "epileptic personality." Substantial criticism has since been aimed at its method of creation and its specificity to epilepsy patients, especially because the study lacked non-epilepsy controls (Mendez, 1988). Several subsequent studies found that the Bear-Fedio Inventory measured non-specific overall psychopathology and not psychopathology specific to epilepsy (Mungus, 1982; Rodin & Schmaltz, 1984). Other more successful inventories, such as the Washington Psychosocial Seizure Inventory (WPSI) have also been specifically developed for epilepsy patients (Dodrill, Batzel, Queisser, & Temkin, 1980). The WPSI has shown good reliability and validity, and measures constructs such as "Emotional Adjustment," "Adjustment to Seizures," and "Medicine and Medical Management," (Swinkels et al., 2004). However, it seems that a large proportion of the published research regarding epilepsy patients and psychiatric disturbance has utilized standard, non-epilepsy-specific inventories such as the MMPI-2 (e.g., Derry et al., 1997; Derry et al., 2002; Dikmen et al., 1983; Karzmark, Zeifert & Barry, 2001; Nelson, Elder, Groot, Tehrani, & Grant, 2004; Swinkels et al., 2003).

Asserting that testing for comorbid psychiatric disorders should be routine for patients with epilepsy, Jones and colleagues (2005a) espoused the use of standardized interview procedures. However, as the most common of these, the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I), is often too time consuming for routine clinical use, the authors evaluated a briefer procedure, the Mini International

Neuropsychiatric Interview (MINI) in terms of its concurrent validity with the SCID-I. Results indicated high concordance between MINI and SCID-I diagnoses for depression, especially for current major depression (r = .86). The authors concluded that current Axis I disorders can accurately be identified by briefer standardized methods. To date, there have been few studies of comorbidity in terms of multiple psychiatric diagnoses in epilepsy samples (i.e., concurrent depression and anxiety, as opposed to depression comorbid with epilepsy or anxiety comorbid with epilepsy). Jones and colleagues found that 73% of their test sample were diagnosed with both depressive and anxiety disorders. In the same sample, around 48% met criteria for more than one anxiety disorder. Perhaps one of Jones and colleagues' most important findings was that of those patients diagnosed with a depressive disorder, 43% were not treated with antidepressants, indicating that mood disorders may often go untreated in patients diagnosed with epilepsy. Furthermore, the authors point out that in the epilepsy literature, intervention papers typically assume that treatment of depression is incorporated into typical care by primary physicians. However, randomized studies in general medical populations have shown that ideal treatment is collaborative and includes biomedical, psychiatric, and other interventions (Jones et al., 2005a).

Though psychological testing has been used to measure more than just depression and anxiety in patients with epilepsy (e.g., Mendez et al., 1993), many studies suggest that mood and anxiety disorders are the most frequently diagnosed psychiatric disturbances in patients with epilepsy (e.g., Jones et al., 2005a; Mendez, 1988; Swinkels et al., 2006). Consequently, disorders of this type are likely to be the primary focus of psychiatric intervention. Depression and anxiety are especially important to assess in

epileptic populations because their treatment may significantly contribute to comprehensive management of patients, especially when issues like quality of life are involved (Griffith et al., 2005).

The MMPI-2 Clinical Scales

The original MMPI, published in 1943 by Hathaway and McKinley, was intended to be a more efficient psychodiagnostic alternative to clinical interviews and mental status examinations (Graham, 2006). The original MMPI Clinical Scales were created to diagnose hypochondriasis, depression, hysteria, psychopathic deviation, paranoia, psychasthenia, schizophrenia, and hypomania. At a later time, two additional scales were added: the Masculinity-Feminity (Mf) scale, which was originally intended as an indicator of homosexuality, and the Social Introversion (Si) scale. The MMPI was groundbreaking, both in terms of its comprehensive approach to personality assessment, and because of its validity scales, which were developed to detect deviant test-taking (Graham, 2006).

Over time, it became apparent that the MMPI was not as good at identifying clinical groups as was originally intended (Ben-Porath, 2008). Because of this, the interpretive approach shifted away from diagnosis, and toward identifying empirical correlates for each scale, as well as the interpretation of profile patterns. For this reason, the original diagnostic labels for each scale were replaced with scale numbers (see Table 1). In the 1980s, a restandardization committee began work on what would become the MMPI-2 (Butcher, Williams, Graham, Tellegen, & Kaemmer, 1989).

Insert Table 1 here

The MMPI-2 featured updated norms and revisions to the item pool, including removal of sexist wording, modernization of idioms, grammatical clarification, and simplification of wording (Graham, 2006). Several sets of subscales were also subsequently developed. The Harris-Lingoes subscales were created to aid in the analysis of item subgroups within the Clinical Scales (Harris & Lingoes, 1955, 1968). The Content Scales were developed to further assess content dimensions of the MMPI-2 item pool (Butcher, Graham, Williams, & Ben-Porath, 1990).

The MMPI/MMPI-2 has been regarded as a "gold standard" for personality assessment. In 1985, the Society for Personality Assessment reported that the MMPI was second only to the Rorschach for personality assessment among its members (Graham, 2006). More recent survey research has indicated that the MMPI-2 is the most frequently used personality measure among clinical psychologists (86%), and second only to Wechsler intelligence tests when all psychological tests are considered (Graham, 2006). Since 1989, roughly 3,000 journal articles have reported studies using the MMPI-2 in a variety of applied settings. For these reasons, the MMPI-2 is widely regarded as a definitive pencil-and-paper tool for personality assessment (Graham, 2006).

However, as previously mentioned, some researchers have been dissatisfied with the use of the MMPI/MMPI-2 with epilepsy patients because of its non-specificity to the epilepsy population (Swinkels et al., 2004). Other researchers have indicated that the Clinical Scales are a less-than-perfect measure of psychiatric adjustment in a more general way. Specifically, researchers have often pointed out problems relating to the Clinical Scales' method of construction, heterogeneity of content within single scales, scale intercorrelations, and the interpretive challenges of certain profiles.

The development of the original MMPI in the 1930s – 1940s was an empirical and not a theoretical or intuitive process (Meyerink, Reitan & Selz, 1988). The inventory's Clinical Scales were composed by compiling questions that had been endorsed by certain previously-diagnosed populations (Graham, 2006). For example, if a particular item reliably differentiated between a person with schizophrenia and a person without, it was added to Sc, the "Schizophrenia" scale (now Scale 8; Graham, 2006). Face validity of the items was irrelevant. This type of methodology is commonly referred to as an "empirical keying method" (Simms, Casillas, Clark, Watson, & Doebbeling, 2005). The rationale behind the empirical keying method was that the test items were presumably sensitive to underlying personality traits that characterized certain groups. Ambiguity in individual item meanings was intended to allow differential interpretations by test-takers, which in turn would give overarching person-specific profiles for analysis by clinicians (Rogers et al., 2006). Because of the emphasis on ambiguity, clinical interpretation of individual item responses was discouraged. Though 15 Content Scales were eventually added to allow content interpretation, subsequent studies found that the incremental validity of the Content Scales is at times very modest (Rogers et al., 2006). The now-common practice of interpreting MMPI profiles with code types stemmed from the unreliability and heterogeneity of empirical correlates (Ben-Porath, 2008). Because the Clinical Scales are not psychometrically optimal in isolation, interpretation shifted from the scale level to the pattern level over time (Ben-Porath, 2008).

The lack of dependable, empirically-determined correlates for individual Clinical Scale elevations is not the only unfortunate result of the empirical keying method.

Clinical Scales often show heterogeneity of content and multidimensionality within scales (i.e., low internal consistency). Using Scale 3 as an example, Simms and colleagues (2005) illustrated the resultant challenges to interpretation. The 60 items on Scale 3 tend to fall into two distinct categories: items that reflect somatic symptoms and items that represent the test-taker's belief that he or she is well-adjusted. Endorsing many items from both of these categories will lead to a large elevation on Scale 3. However, a moderate elevation ($T = 65$) may be reached by endorsing many items from either of the two categories, or by endorsing a few items from each category. Thus, proper interpretation of an elevation on Scale 3 requires examination of the individual item responses. This type of heterogeneity exists for nearly all of the Clinical Scales (Simms et al., 2005).

The empirical keying method also resulted in high inter-scale item redundancy (i.e., low discriminant validity between scales). Because all items were eligible for inclusion in each Clinical Scale, a large number of items overlap between scales. On the MMPI-2, an average of 6.4 items overlap between any given pair of the 10 standard Clinical Scales (Simms et al., 2005). On some scale pairs, the amount of overlapping items is considerably higher. For instance, 20 items overlap between Scales 1 and 3, 17 items overlap between Scales 7 and 8, and 13 items overlap between Scales 2 and 7. As noted by Simms and colleagues (2005), this degree of redundancy may make Clinical Scale interpretation particularly challenging. The distinctiveness of individual scales is reduced, decreasing their discriminant validity and causing high scale intercorrelations. Furthermore, the meanings of elevations on Clinical Scales may be unclear, especially when endorsement of a particular set of items causes scale covariation. Symptoms such

as anxiety and dysphoric mood occur in numerous clinical syndromes (e.g., "IDD"), so the presence of these symptoms is not particularly helpful in discriminating between, for instance, schizoaffective disorder or MDD (Nichols, 2006). Further complicating the problem of item overlap is the fact that items with high face validity (i.e., "obvious" items) are disproportionately represented among the overlapping items (Nichols, 2006). These obvious items represent personality characteristics that are recognizable as abnormal. They are therefore more likely to be endorsed by test-takers who intend to convey their distress, but are not particularly helpful diagnostically. As Nichols (2006) points out, "they function well to detect that 'something is wrong,' [but] relatively poorly to indicate what that specific 'something' is" (p.121).

With complications such as low internal consistency and low discriminant validity, certain Clinical Scale patterns can be particularly difficult to interpret. One type of difficulty is the two-scale elevation between scales with high item overlap, such as the 27/72 code type mentioned above. When a test-taker's scores display this type of elevation, thorough interpretation requires investigation of the constituent items. Investigation is also necessary when a Clinical Scale profile shows elevations that would not be expected, based on what is known about the client's presenting problems. Forbey and Ben-Porath (2007) illustrated this point for a client with a history of substance abuse. Substance abusers typically elevate on Scale 4, a measure of the tendency to act out, but may also elevate on Scales 2, 6, 7, and 8 due to emotional or thought disturbance. Forbey and Ben-Porath mentioned that interpretation of these types of multiple elevations often require utilization of Harris-Lingoes subscales or other supplementary resources, complicating and lengthening the interpretive process.

Two types of profiles that are particularly difficult to interpret are the floating profile and the within-normal-limits (WNL) profile. Because of scale intercorrelations, test-takers occasionally—particularly if they are extremely distressed or characterologically impaired—score above the clinical threshold on most or all of the Clinical Scales. These profiles (similar to that of the substance abuser above) can be an interpretive challenge because of the difficulty of determining the scale on which to focus or which scale represents the origin of the distress (Wallace & Liljequist, 2005). Another major limitation to interpretation may occur when a profile shows no clinical elevations. The inverse of the floating profile, the WNL profile may account for nearly one third (30.1%) of all clinical profiles (Rogers et al., 2006). Because of the multidimensional character of the Clinical Scales, it is possible (though not necessarily probable) that a sub-clinical score may be the result of one dimension obscuring another. Staying with the example of Scale 3 given previously, a test-taker may endorse a moderate amount of specific somatic concerns, but virtually no items relating to a sense of adjustment, resulting in an overall sub-clinical score. From the Clinical Scale profile, and without further examination of Harris-Lingoes, the examiner would have no indication of the test-taker's somatic preoccupation.

Though item overlap and scale multidimensionality may seem helpful for capturing the syndromal character of many psychiatric diagnoses, they exact a significant price in internal consistency, discriminant validity, and interpretation.

The MMPI-2 Clinical Scales and Patients with Epilepsy

Though some researchers have criticized the MMPI's use with epilepsy patients in a generalized way, asserting that a measure not normed for epilepsy patients is inferior

to one specially designed for patients with epilepsy, other researchers have criticized its use for patients with epilepsy in a more specific way (Swinkels et al., 2004).

For instance, perhaps the most widely researched aspect of the MMPI's validity in epilepsy patients involves Clinical Scale 8 (Schizophrenia). The Clinical Scales' tendency toward a high false positive rate of diagnosis for psychosis in epilepsy patients has been well-documented (e.g., Lewis, Lachar, Voelker, & Vidergar, 1984; Modrego, Pina, Galindo, & Minguez, 2002; Nelson et al., 2004). Many items that feed into Scale 8 may actually reflect seizure or seizure aura symptomatology, and not psychosis (e.g., "Peculiar odors come to me at times," "I have little or no trouble with my muscles twitching or jumping," "I have had attacks in which I could not control my movements or speech," "I have had blank spells in which my activities were interrupted and I did not know what was going on around me"; Lewis et al., 1984; Nelson et al., 2004). Lewis and colleagues (1984) compared scores on Scale 8 of patients with epilepsy, patients with comorbid epilepsy and psychosis, and patients with schizophrenia. Scale 8 scores were not able to reliably differentiate between epilepsy patients with and without psychosis (mean Scale 8 T scores for each group were > 70). The only qualitative difference between any of the groups was an emphasis on religiosity in the psychotic epilepsy group. Modrego and colleagues (2002) found significantly higher scores on Scale 8 in a sample of epilepsy patients than in controls. Furthermore, 57% of the epilepsy patients had clinically significant elevations on Scale 8, but only 17% met DSM-IV criteria for schizotypal personality. Nelson and colleagues (2004) developed correction procedures for MMPI-2 profiles of patients with epilepsy. Using a statistical correction procedure, the mean Scale 8 T score of patients with intractable seizures in their sample fell from the

clinical to non-clinical range (66.4 to 61.8). By using combined statistical and rational procedures, the average score fell from 66.4 to 60.8. These findings indicate that MMPI-2 Clinical Scale scores can be vulnerable to spurious inflation due to disease-related items.

According to Gass (1991),

> Interpretive norms are based on a psychiatric sample, despite the fact that the MMPI contains numerous items that reflect bona fide physical and cognitive symptoms of brain lesions, the presence of which may be totally unrelated to psychopathology or personality characteristic. (p. 27)

It should not be surprising, then, that other Clinical Scales, including Scales 2 and 7, may be equally as vulnerable to inflation due to somatic (and not psychiatric) symptoms. A number of researchers have found that MMPI profiles of patients with neurologic injury or disease contain variance on these scales due to neurologic sequelae (e.g., Gass, 1991, 1996; Meyerink, Reitan, & Selz, 1988). Meyerink, Reitan, and Selz (1988) found that items that reflected a neurological disease process may artificially inflate Scale 2 scores up to 4 points for patients with multiple sclerosis. In a study of patients with traumatic brain injuries (TBI), Gass (1991) found that the major source of variance on Scales 2 and 7 between TBI patients and a control group were items that reflected physical and cognitive sequelae of head injury. In a study of patients with stroke, Gass (1996) found that standard profile interpretation can overestimate symptoms relating to depression and anxiety. Studies such as these indicate that it is reasonable to assume that individuals with neurologic disorder will report physical and cognitive symptoms on the MMPI-2, even if these symptoms are unrelated to psychopathology (Gass, 1996).

Nevertheless, few studies have examined potential inflation in Scales 2 and 7 in the profiles of patients with epilepsy. Derry and colleagues (1997) generated MMPI-2 profiles for 100 epilepsy patients and then rescored them with questions reflecting seizure content removed. Results indicated that the decrease in Scale 2 was statistically significant, but the authors concluded that the difference was not clinically significant. Derry and colleagues (2002) also examined the MMPI-2 profiles of patients with epilepsy before and after surgery. The mean decrease in Scales 2 and 7 was around 14 points for each scale. However, the authors concluded that the lower scores were not indicative of lowered seizure content, but rather of actual declines in psychological distress due to the resolution of the patients' seizures.

However, the studies by Derry and his colleagues define the sequelae of epilepsy only in terms of ictal phenomena. This interpretation ignores non-ictal neurologic symptoms, such as cognitive deficits. For example, Meyerink, Reitan, and Selz (1988) identified 6 items with neurologic content that fed into Scale 7, whereas Derry and colleagues (1997) identified none. Using a more comprehensive interpretation of "seizure content," Karzmark, Zeifert, and Barry (2001) evaluated the accuracy of Clinical Scale 2 of the MMPI-2 and the Beck Depression Inventory (BDI) in identifying depression in patients with epilepsy. Though the BDI measures somatic content (as do other specific inventories of depression), these somatic items tend to be limited to depressive symptomatology, and are not likely to overlap with the sequelae of epilepsy (Karzmark, Zeifert, & Barry, 2001). The authors therefore assumed that the BDI would be less impacted by somatic bias, and compared both it and Scale 2 with a criterion measure of depression (a structured clinical interview). Results indicated that the BDI

had more specificity, a lower false positive rate, better positive predictive value, and equivalent negative predictive value, when compared to Scale 2. Scale 2 had higher sensitivity and a lower false negative diagnosis rate than the BDI. Therefore, it seems that the validity of assessment tools for depression varies with epilepsy patients. Overall, it appears that a briefer measure (e.g., BDI) may be as effective in this sample as a time-consuming measure like the MMPI-2. However, the BDI items lack subtlety, and may be prone to underreporting of symptoms, especially as it does not have validity scales like the MMPI-2.

An optimal measure for an epilepsy population would likely be focused strictly on depressive (or anxiety) symptomatology (avoiding somatic content), and be shorter, but also include validity indicators. Consequently, a measure such as the MMPI-2 Restructured Clinical (RC) Scales may be well-suited as a screening tool for depression and anxiety in patients with epilepsy.

The MMPI-2 Restructured Clinical (RC) Scales

In 2003, Tellegen and his colleagues departed from MMPI tradition with the creation of the RC Scales. By creating the RC Scales, Tellegen and his associates sought to "modernize the basic sources of information on the test" (2006, p. 149). The purposes of doing so were many: (1) to deemphasize empirically derived scales and interpretations in favor of theoretically informed, yet empirically tested scales; (2) to create greater distinctiveness between scales; (3) to increase homogeneity within individual scales; and (4) to remove nonspecific variance due to a common "first" or "prime" factor (Simms, 2006; Tellegen et al., 2006). Their attempt to realize these goals was accomplished via

exploratory factor analysis coupled with Jackson's (1970) sequential approach to construct validity of personality measures (Rogers et al., 2006).

> The Jacksonian method of personality test development stresses four principles. These are, first, the overriding *importance of psychological theory*; second, *the necessity for suppressing response style variance*; third, *the importance of scale homogeneity, as well as generalizability*; and fourth, *the importance of fostering convergent and discriminant validity* at the very beginning of a program of test construction. (italics in original; Jackson, 1970, p. 63)

In constructing the RC scales, Tellegen and colleagues (2003) incorporated the first, third, and fourth of these principles. Though the second principle was not explicitly considered during the RC Scales' creation, the RC Scales' vulnerability to response-style variance has since been examined (see discussion below of Sellbom, Ben-Porath, Graham, Arbisi, & Bagby, 2005).

Satisfying the first Jacksonian principle, Tellegen and colleagues posited that a theoretical, nonspecific distress factor—demoralization—was common to clinical populations (Rogers et al., 2006). As a nonspecific distress factor, demoralization would act as a common source of covariance, and confound scores on the Clinical Scales. Identification of the demoralization factor (Dem) relied upon the work of several authors. Jerome Frank (1974) stated that "the chief problem of all patients who come to psychotherapy is demoralization" and that its characteristic features "are feelings of impotence, isolation, and despair" (p. 271). He further indicates that "the most frequent symptoms of patients in therapy—anxiety and depression—are direct expressions of demoralization" (Frank, 1974, p. 271). Watson and Tellegen (1985) used factor analysis

to identify two first-order dimensions of affect: “Positive Affect” and “Negative Affect” (see Figure 1). A second-order dimension, “Pleasantness-Unpleasantness,” lies midway between “Positive Affect” and “Negative Affect.” A combination of “High Positive Affect” and “Low Negative Affect” constitutes “Pleasantness,” a general sense of happiness, contentedness, and satisfaction. “Low Positive Affect” and “High Negative Affect” combine in “Unpleasantness,” an analog of Dem that includes sadness, loneliness, unhappiness, and grouchiness. Around that time, Tellegen (1985) also described demoralization as a general factor that inflates correlations on inventories (such as the MMPI) between scales that would be expected to be more or less independent (e.g., depression and anxiety).

It should not be surprising that Dem was included in each of the MMPI Clinical Scales. According to Ben-Porath (2008), the empirical keying method yielded at least two different groups of items in each Clinical Scale. One of these item groups is comprised of those responses unique and specific to a particular normative patient sample, vis-à-vis being a member of that sample. For instance, hallucinations and delusions were for the most part unique to the schizophrenia patient sample. However, all of the patients in each normative sample had something in common: they were all psychiatric inpatients. Therefore, it follows that there would be some items, common to all psychiatric inpatients, that would get included in each Clinical Scale. Examination of these items, which could be seen as the byproduct of being a psychiatric inpatient, revealed that they seem to describe a state of demoralization (Tellegen et al., 2003).

With a theory-driven rationale for the redesign satisfying Jackson’s first principle, the third and fourth principles were addressed in the actual item-by-item

construction of the RC Scales. The separation of Dem and creation of new scales was accomplished via a four step process. In Step 1, Tellegen and colleagues (2003) judged MMPI-2 Clinical Scales 2 and 7 to be most heavily saturated with demoralization content. This assumption follows from Watson and Tellegen's (1985) two-dimensional map of affect. The "Unpleasantness" construct (an analog of Dem) falls between the "Low Positive Affect" factor (i.e., depression) and the "High Negative Affect" factor (i.e., anxiety), and so items from Scales 2 and 7 should theoretically be ideal to measure it. Principal components analyses (PCAs) were used to identify items from these scales with high (>.49) loadings on both their data set (Clinical Scale) and Dem. This analysis yielded 10 items, which formed the basis of the Dem scale. Additional items from the remainder of the MMPI-2 item pool were added on the basis of their correlations to the Dem factor, resulting in a final demoralization scale (RCd) of 24 items. In Step 2, RCd was appended to each Clinical Scale in turn, and the resultant item sets were factor analyzed. Items which gravitated toward the demoralization scale were removed from their Clinical Scales. In Step 3, items which had not loaded highly on demoralization in Step 2, and which correlated more highly with their parent Clinical Scale than with any other Clinical Scale, were identified as the core components of each RC seed scale. In Step 4, the RC seed scales were augmented by additional items that correlated above scale-specific minimum values. A final series of ad hoc adjustments were made to optimize scale content, increase internal consistency, or maximize relationships with external criteria (in light of the Jacksonian principles). The consequent item groups, alongside the previously formed RCd, were the 9 RC Scales (see Table 2).

Insert Table 2 here

The introduction of the RC Scales, which were published in the form of a supplemental monograph (Tellegen et al., 2003) to the *MMPI-2 Manual for Administration and Scoring* (Butcher et al., 1989), attracted much critical attention. Published concurrently in a special edition of the *Journal of Personality Assessmen*t, reviews by Rogers and colleagues (2006) and Nichols (2006) offered strikingly different commentary on the RC Scales.

Rogers and colleagues (2006) were predominantly supportive in their analysis of the RC Scales. In a cross-validation of the RC Scales using previously gathered data from 7,330 clinical cases, homogeneity was confirmed by large alpha coefficients (consistently >.80 for males and females) as well as optimal inter-item correlations (.15 < *r* <.50). By and large, Rogers and colleagues were also able to cross-validate the selection of items for Dem and the seed scales with their own data by following the construction procedure outlined in the monograph. Despite their largely optimistic tone, Rogers and colleagues raised several concerns, most notably the lack of information regarding clinical interpretation of RC Scale elevations, and the effects of response style variance (Jackson's second principle) on RC Scale outcomes. Both of these concerns have been addressed elsewhere (see below; e.g., Sellbom, Ben-Porath, & Graham, 2005; Sellbom, Ben-Porath, McNulty, Arbisi, & Graham, 2006; Simms et al., 2005).

In contrast to Rogers and colleagues' cautious optimism, Nichols (2006) criticized the RC Scales by raising various conceptual concerns. Among these are the loss of the multivariate structure ("syndromal fidelity") of the Clinical Scales, the

selection of demoralization as the first factor of common variance in the Clinical Scales, the danger of construct drift, and the utility of the RC Scales.

Referring to the "syndromal character" of the Clinical Scales, Nichols stated that they "model the clinical syndromes they were developed to measure by incorporating…diverse aspects of emotion, thinking, and behavior" (2006, p. 122). To illustrate this idea, he pointed out that depression encompasses elements of dysphoric mood, impaired cognition, and somatic symptoms, and Scale 2 therefore contains diverse items to capture all of these domains. This seems to be a reasonable explanation, but as Finn and Kamphuis (2006) have pointed out, classical test theory has shown that conjunctive constructs (e.g., syndromes) that contain several sub-domains are best assessed through the use of disjunctive (separate) measures of the individual domains, and not through use of composite measures. For instance, an elevation on Clinical Scale 8 could mean numerous things, including psychosis, cognitive disorganization, seizure/stroke semiology, or social withdrawal, limiting interpretation without further measures of these sub-traits. Finn and Kamphuis (2006) also mentioned that unifactorial scales are much more likely to be useful over time. For instance, Scales 3 and 7 were designed to measure syndromes that are largely no longer recognized (e.g., hysteria, psychasthenia). In any case, the lack of the RC Scales' syndromal fidelity is likely a moot point, considering that they have better convergent validity with syndromal diagnoses than the Clinical Scales (Ben-Porath, 2008).

Nichols also expressed concern over the theoretical model of affect that Tellegen and colleagues (2003) used to identify demoralization as the cause of Clinical Scale covariation. In sum, Nichols reviewed a series of older correction methods that

attempted to find a first factor of covariance through empirical analysis and remove it, leaving the Clinical Scales otherwise intact. Nichols praised these previously identified first factors as being relatively balanced with respect to depression, anxiety, tension, obsessiveness, and low self-esteem content, and criticized RCd as disproportionately favoring depressive content. Essentially, Nichols (2006, p. 124) took issue with the decision to use a theoretical construct (demoralization) as the source of covariance, saying, "the appropriateness and advantages of the decision to embrace a theoretically rather than an empirically driven strategy for constructing…RCd are doubtful." However, as indicated by Simms (2006), the model of affect used by Tellegen and colleagues was previously validated in many empirical investigations (e.g., Sellbom & Ben-Porath, 2005; Tellegen, 1985). Tellegen and colleagues (2006, p.157) have insisted that their methods "yield[ed] an empirically supported and theoretically informed construct and scale."

Nichols also emphasized concerns over the potential for construct drift due especially to the methods used in the fourth stage of RC Scale construction (i.e., augmenting the seed scales by adding additional items that were highly correlated). He summarizes his fears by saying, "the more items that are added,…the greater the risk that the content…will have drifted away from its core construct, perhaps to the extent of measuring a substantially different construct" (Nichols, 2006, p. 133). He took RC7 as an example. According to Nichols, this scale overemphasizes aggressive content and deemphasizes "desirable" psychasthenia content. However, RC7 is labeled as "dysfunctional negative emotions," and is not purported to be a measure of psychasthenia (Finn & Kamphuis, 2006).

With regard to the utility of the RC Scales, Nichols raised several concerns. First, he questioned the usefulness of RC2 and RC7. Because the RCd items were initially drawn from Clinical Scales 2 and 7, and because they favor depressive symptomatology, he stated that RC2 "is missing substantial core variance for depression" and that RC7 conversely suffers from under-extraction of first-factor variance (Nichols, 2006, p. 131). Put simply, Nichols feared that RC2 does not measure enough, while RC7 measures too much. He supported the claim that RC7 measures too much (i.e., something other than psychasthenia) by showing its moderate correlations with psychoticism and anger-hostility. This contention is again based upon the idea that RC7 is supposed to be measuring psychasthenia. Emphasizing that the RC Scales are not intended as analogs of the Clinical Scales, Tellegen and colleagues (2006) gave evidence that RC7 is a valid measure of anxiety-related symptoms, and further, that RC2 is a valid measure of depression-related symptoms. In regard to RC7, Tellegen and colleagues pointed to recent research (i.e., Watson, 2005) which proposed that some anxiety disorders (e.g., GAD, PTSD) fall under a general category of distress disorders, while others (e.g., panic disorder, agoraphobia, social phobia) are more related to fear (e.g., Clark & Watson, 1991; Watson, 2005). Thus, the distress-related disorders would be expected to correlate more strongly with RCd, while the fear-related disorders would be expected to correlate with RC7. Indeed, research with measures such as the Internal States Scale, the State-Trait Personality Inventory (STPI) Trait Anxiety Scale, and the Fears Questionnaire have supported this idea (see Tellegen et al., 2006). Therefore, RCd and RC7 may actually be useful as potential indicators of different types of anxiety disorders. Incidentally, because Nichols reported concern that RC7 is overly correlated

with anger-hostility and psychoticism, Tellegen and colleagues (2006) correlated RC7 with the STPI Trait Anger Scale, the Magical Ideation Scale, and the Perceptual Aberration Scale. RC7 was more highly correlated with anger than Clinical Scale 7, but this was expected because anger falls into the category of "dysfunctional negative emotions." However, Scale 7 was more correlated with psychoticism than RC7. With regard to RC2, Tellegen and colleagues pointed out that a growing body of evidence has indicated that the distinctive core feature of depression is anhedonia, or the absence of positive emotional experiences (as opposed to subjective dysphoric mood). For example, Joiner and colleagues (2005) make a distinction between clinical depression and depressed mood by saying that anhedonia is unique to major depression, whereas depressed mood is not specific to just that syndrome, but rather more akin to demoralization. As a measure of "low positive emotions," RC2 should therefore be an apt measure of the core of MDD symptomatology.

Nichols also questioned the utility of the RC Scales in terms of their incremental validity over other content scales. The main argument in this case is that the RC Scales are merely a redundant repackaging of extant Content or Supplementary scales. Opposing this stance, Finn and Kamphuis (2006) presented two cases in which the RC Scales were more useful than the Clinical Scales, the Content Scales, the Supplementary Scales, or the Personality Psychopathology Five (PSY-5) Scales for identification of clients' central concerns. In the first of these case studies, a 32-year-old suicidal male presented with elevations on all Clinical Scales except for Scales 1 and 9 (which was depressed). When the RC Scales were analyzed, the only significant elevation was RCd, indicating that the client was severely demoralized and felt overwhelmed. In an aside,

the authors point out that the client's RC2 score was not significantly elevated, and this may be why he was not responding to antidepressant medication. In the second case study, a 27-year-old man was assessed following an event that looked objectively like a suicide attempt, but which he described as an attempt to free evil energy from his body. The client's Clinical Scale profile indicated significant elevations on Scales 6, 8, 0, 7, and 2. Examination of the RC Scales deemphasized depression and anxiety, and illuminated primary difficulties with delusional beliefs and disordered thinking. In both cases, subsequent interview with the clients validated the RC Scale-based interpretation of the client's situation. Therefore, the RC Scales may be seen as having substantial utility in clarifying confusing or complex MMPI-2 profiles with multiple elevations.

Finn and Kamphuis (2006) are not the only researchers to have examined the RC Scales. Several other groups have also attempted to either show the validity or utility of the RC Scales. For instance, Sellbom and colleagues (2005) compared the susceptibility of the Clinical Scales, RC Scales, and Content Scales to over- and underreporting. Noting that the method of selection for RC Scale items resulted in the inclusion of more transparent or obvious items than the Clinical Scales' empirical keying method, the authors sought to find if this transparency represented a vulnerability to response style variance. The analysis included archival data from five samples used in previous investigations; on two samples (both comprised of college students) participants were instructed to underreport symptoms, and on three others (one of college students, one of psychiatric inpatients, and one of medical patients), participants were instructed to overreport symptoms. Results of the analysis suggested that Clinical Scales were slightly less susceptible (the 95% confidence intervals of all samples overlapped) to

underreporting than RC Scales and Content Scales. However, post hoc analyses revealed that this effect was an artifact of the subtle items' effect on the Clinical Scales (underreporting participants actually scored higher on Subtle Scale items than controls). Effect sizes were comparable for each scale in the overreporting condition, indicating that RC Scales are not more susceptible to overreporting than Clinical Scales. The authors concluded that equal caution should be exercised when interpreting the three sets of scales if under- or overreporting are suspected.

Other reports have offered information regarding the interpretation of RC Scales. Following a psychometric evaluation of the RC Scales, Simms and colleagues (2005) offered considerations regarding the interpretation of scales RCd, RC2, and RC7. In their analysis, RCd proved to be the strongest predictor of current and lifetime ratings of syndromal depression. This is not unexpected, given Joiner, Walker, Pettit, Perez, Cukrowicz's (2005) distinction between depressive mood and MDD (which has anhedonia as its prime component). Therefore, in concert, RCd and RC2 can be valuable indicators of the nature of a patient's depression. For instance, Simms and colleagues (2005) make the case that if both scales are elevated, anhedonic MDD would be a reasonable hypothesis. On the other hand, if solely RCd is elevated, sadness and nonspecific symptoms such as concentration difficulties or sleep problems may be present. Simms and colleagues report a large correlation between RCd and RC7. The authors make the case that concurrent elevations of RC7 and RCd may be indicative of GAD, which is characterized by both generalized negative emotionality and persistent emotional distress. As mentioned previously, Tellegen and colleagues (2006) indicate that elevations on RCd would be more indicative of GAD, whereas elevations on RC7

would likely indicate the presence of panic disorder or phobias. Combining these two perspectives, it may be the case that an elevation on RCd alone would indicate generalized demoralization or distress (minimally related to anxiety), whereas concurrent elevations on RCd and RC7 would indicate a distress-related anxiety disorder (e.g., GAD, PTSD), and an elevation on RC7 with less or no elevation on RCd would indicate a fear-related anxiety disorder (panic, phobias).

Sellbom, Ben-Porath, McNulty, Arbisi, and Graham (2006) examined the interpretive implications of differences between Clinical and RC Scale elevations. Comparison to criterion measures indicated that when corresponding Clinical and RC Scales are both elevated, the core descriptors (e.g., depression for Scale 2 and RC2, anxiety for Scale 7 and RC7) should be emphasized. They additionally found that core descriptors should be emphasized in the presence of elevations on RC4, RC6, and RC8, regardless of whether the corresponding Clinical Scale is elevated. Furthermore, in cases where a Clinical Scale is elevated but the corresponding RC Scale is not, RCd should be consulted to determine if demoralization is the cause.

Recently, an increasing number of studies have validated the RC Scales in various settings. Sellbom, Graham, and Schenk (2006) used the RC Scales in a private practice setting and reported acceptable internal consistency, as well as promising discriminant and convergent validity (when compared with scores from the Multiaxial Diagnostic Inventory (MDI)). Additionally, correlations with the MDI indicated that the RC Scales had more incremental validity than the Clinical and Content Scales in predicting self-reported clinical symptoms. Sellbom, Ben-Porath, and Graham (2006) studied the psychometric properties of the RC Scales by comparing them to therapist

ratings of psychiatric symptoms in a college counseling setting. They replicated findings of increased convergent and discriminant validity (compared to Clinical and Content Scales) from previous studies. Further, when compared to Clinical and Content Scales, the RC scales showed stronger correlations with therapist-rated conceptually relevant criteria than with nonrelevant criteria.

Frye (2007) examined the RC Scales as a predictor of rehabilitation outcomes in a medical sample with chronic pain and mild traumatic brain injury. RCd was found to be a more powerful predictor of recovery than any other RC Scale in isolation, and even RC1, RC2, and RC3 in combination. Furthermore, RCd was a stronger predictor of rehabilitation outcome than the "role limitations due to physical health" scale from the Medical Outcomes Study (MOS) Short-Form Health Survey (SF-36). Forbey and Ben-Porath (2007) replicated previous findings by comparing RC Scale scores to Clinical Scale scores in a substance abuse treatment setting at a VA hospital. Results indicated that the RC Scales show improved convergent and discriminant validity with extra-test measures of psychopathology when compared to the Clinical Scales. Wygant and colleagues (2007) evaluated the RC Scales in a sample of bariatric surgery candidates. Again, results indicated that the RC Scales were generally more internally consistent than the Clinical Scales, and that the RC Scales displayed better convergent and discriminant validity for a number of behavioral and psychological variables relevant to preoperative evaluations. The authors also identified a number of empirical correlates for each RC Scale. The correlates for RCd included life dissatisfaction, a history of psychiatric illness, poor insight/judgment with regard to weight, and obesity negatively affecting self-image and quality of life. The correlates for RC2 similarly included life

dissatisfaction, a history of psychiatric illness, and obesity negatively affecting self-image and quality of life. The correlates for RC7 included trauma/stress leading to overeating, obesity negatively affecting self-image, and poor insight/judgment. The authors conclude that RCd, RC2, and RC7 in particular can be used as a gauge for the patient's emotional stability pre-surgery, and to differentiate between depressive and anxiety disorders.

In a sample of patients with epilepsy, analysis via the RC Scales may have a number of benefits. As unifactorial measures, RC2 and RC7 lack much of the somatic bias that is present in various Clinical Scales (Ben-Porath, 2008). In addition, the RC Scales separate demoralization, depressed mood, and anxiety into more meaningful and theoretically relevant constructs than do the Clinical Scales (Tellegen et al., 2006). This parsing out of symptomatology could be particularly helpful in diagnosing and treating comorbid psychopathology in epilepsy patients. The RC Scales may provide valuable information for pre-surgical evaluations, behavioral interventions, and medication management. For instance, elevations on RCd, RC2, or RC7 might indicate the need for interventions focused on emotional stability, whereas elevations on RC4 or RC9 may indicate the need for behavioral interventions for treatment adherence (Wygant et al., 2007). The RC Scales also have the advantage of being a less time-consuming measure for both clinicians (e.g., Forbey & Ben-Porath, 2007) and test-takers (if they are administered as part of the MMPI-2-RF; see Appendix B).

Depression and anxiety can be problematic barriers to medical treatment, and research has indicated that these disorders all too often go undiagnosed (e.g., Jones et al., 2005b). Therefore, RCd, RC2, and RC7 may show excellent utility as part of a psychological test battery, especially given their incremental validity over the standard

Clinical Scales (Sellbom, Ben-Porath, & Graham, 2005; Sellbom, Graham, & Schenk, 2006; Simms et al., 2005). However, despite the potential benefits of use with patients with epilepsy, no studies have examined or validated the RC Scales in this population.

Purpose of Study

Psychological testing is an important component in the treatment of patients with epilepsy. In medical settings, the MMPI-2 is often used to detect psychopathology that may interfere with treatment and recovery. However, researchers have identified potential difficulties with the MMPI-2 Clinical Scales, both in terms of general psychometric properties, and in terms of their use with patients with neurologic disorders. Research has indicated that mood and anxiety disorders are significantly more prevalent in patients with epilepsy than in the general population. However, when used with epilepsy patients, Clinical Scales such as 2 and 7 may show spurious inflation due to scale covariation, the presence of nonspecific variance (demoralization), or the inclusion of somatic content. The Restructured Clinical (RC) Scales were designed to be more precise and psychometrically sound interpretive alternatives to the Clinical Scales. The present study was designed to accomplish several goals: (1) to describe the psychometric properties of the RC Scales in an epilepsy sample; (2) to determine if the RC Scales more adequately measure depression and anxiety in an epilepsy sample; and (3) to examine characteristics of RC Scale profiles in an epilepsy sample.

Hypotheses

The Restructured Clinical (RC) Scales will show better internal and external validity than the Clinical Scales in a sample of patients with epilepsy.

I. Based on previous research, RC Scales are expected to yield high internal consistency coefficients ($.70 < \alpha < .99$) in this sample.

II. Based on previous research, RC Scale intercorrelations are expected to fall within the .4 to .6 range in this sample, whereas Clinical Scale intercorrelations are expected to fall within the .6 to .8 range.

III. In this sample, pertinent RC Scales will demonstrate greater convergent validity with an extra-test measure of mood symptoms than pertinent Clinical Scales.

    a. RC2 will show greater convergent validity with the IDS-SR, an external measure of depressive symptom severity, than will Clinical Scale 2.

    b. The combination of RCd and RC2 will better predict total score on the IDS-SR than will Clinical Scale 2.

IV. In this sample, RC Scales will demonstrate greater discriminant validity with a measure of somatic complaints than the corresponding Clinical Scales.

    a. RC2 will show greater discriminant validity with Hy_4, a scale labeled "Somatic Complaints," than will Scale 2.

    b. RC7 will show greater discriminant validity with Hy_4 than will Scale 7.

Exploratory Analyses

RC and Clinical Scale profiles will be examined in a sample of patients with epilepsy.

I. RC Scale profiles will be examined for subjects whose Clinical Scale profiles display a “conversion V” pattern.

II. RC Scale profiles will be examined for subjects whose Clinical Scale scores display a “floating profile.”

III. RC and Clinical Scale profiles will be compared for subjects whose IDS-SR total score indicates the presence of at least moderate depressive symptoms (total score ≥ 26).

CHAPTER III: METHOD

Participants

Subjects included 137 patients with epilepsy, aged 18 to 67, who underwent neuropsychological evaluation as part of a comprehensive assessment of their seizure disorder. Subjects were evaluated in the University of Texas Southwestern Medical Center Neuropsychology Service or the Parkland Health and Hospital Systems Epilepsy Monitoring Unit from 1995 to 1998. Diagnosis of epilepsy was made by the referring neurologist based on clinical history, electroencephalography (EEG), and neuroimaging studies. Subjects who had undergone neurosurgery for epilepsy previous to their neuropsychological evaluation were not considered for inclusion in the current study.

To be included in this study, subjects must have completed neuropsychological assessment including the MMPI-2 and IDS-SR. Subjects must also have been primarily English speaking, age 18 or older, and have had valid MMPI-2 results. A valid MMPI-2 profile was defined *a priori* using Graham's (2006) guidelines for inpatients: the T score on F must have been less than or equal to 100, the VRIN raw score must have been less than 13 ($T < 80$), and the TRIN raw score must have been less than 13 ($T < 80$ in the direction of true) and greater than 5 ($T < 80$ in the direction of false). Of the original subject pool of 161 patients, 24 had invalid MMPI-2 profiles, and were therefore eliminated from analysis, resulting in a final subject pool of 137 patients.

Materials

Minnesota Multiphasic Personality Inventory-2 (MMPI-2; Butcher et al., 1989). The MMPI-2 is used routinely in diagnostic assessments to evaluate psychological and personality features. The MMPI-2 was normed for English-speaking individuals ages 18

and older. The test consists of 567 self-report true/false questions, which further divide into validity scales, ten basic Clinical Scales, the Restructured Clinical (RC) Scales, and various content scales. The validity scales (L, F, K, VRIN, TRIN) provide information regarding whether the examiner may be answering untruthfully, inconsistently, or attempting to either "fake good" or "fake bad," permitting the scaled scores to either be accepted with some level of confidence, or indicating that caution must be exercised during profile interpretation. The MMPI-2 provides T scores from which meaning can be drawn. Graham (2006) suggests that severity thresholds may be somewhat scale-specific. For example, a T score above 70 is indicative of clinically significant depressive symptoms on Scale 2, but when medical patients score above 60 on Scale 1, "a strong psychological component to the illness should be suspected" (Graham, 2006, p. 67). In most settings, a cutoff of $T \geq 65$ is used to indicate clinical significance.

In assessing convergent and discriminant validity, the present study primarily examined Clinical Scales 2 and 7; RC Scales RCd, RC2, and RC7; and Harris-Lingoes subscale Hy_4 ("Somatic Complaints"). All RC Scales, and Clinical Scales 1 – 4 and 6 – 9, were assessed for intercorrelations. As Clinical Scales 5 (Masculinity-Femininity) and 0 (Social Introversion) were developed somewhat later than the other Clinical Scales, because they do not generally describe pathological traits (Graham, 2006), and since they have no RC Scale counterparts, they were not used in the current study. For the current study, a T score of 65 or greater on all scales was used to indicate clinical significance.

Due to the retrospective nature of the current study, only K-corrected T scores were available for the Clinical Scales. As the RC Scales and Harris-Lingoes do not contain K-correction factors, non-K-corrected Clinical Scale scores would have been

ideal for comparison. However, previous studies (e.g., Wallace & Liljequist, 2005) have made comparisons using K-corrected Clinical Scales, and found comparable findings to studies using non-K-corrected Clinical Scales.

The Inventory of Depressive Symptomatology – Self-Report (IDS-SR; Rush et al., 1986, Rush, Gullion, Basco, Jarrett, & Trivedi, 1996). The IDS-SR is a self-report scale of depressive severity. The test consists of 30 Likert-scaled items; answer choices range from 0 to 3, with higher scores indicating more severe symptoms of depression. Possible total scores on the test range from 0 to 84, and severity thresholds are as follows: no depression (≤ 13); mild (14 – 25); moderate (26 – 38); moderate to severe (39 – 48); and severe (≥ 49; Rush et al., 1996). The IDS-SR has highly acceptable psychometric properties, including internal consistency (Cronbach's alpha = .92), concurrent validity (r = .54 for DSM-IV fifth digit diagnoses, r = .91 with IDS-C, r = .95 with the Hamilton Rating Scale for Depression [HRS-D], r = .86 with the Beck Depression Inventory [BDI]), and item-total correlations (22 of 28 scored items have rs ≥ .50, 11 of 28 scored items have rs ≥ .65; Rush et al., 1996; Rush et al., 2004; Trivedi et al., 2004). For further information regarding the IDS, see Appendix A.

Procedure

The MMPI-2 and the IDS-SR were administered to subjects as part of an extensive battery of neuropsychological tests. The neuropsychological evaluations were conducted in the Epilepsy Monitoring Unit of Parkland Health and Hospital Systems or the Neuropsychology Service at the University of Texas Southwestern Medical Center. All tests were administered and scored by an experienced psychometrician or psychology intern who had no knowledge of the current study's aims or hypotheses. All patients

gave informed consent to have the measures administered as part of their clinical evaluation, but no patients had knowledge of the hypotheses of the current study. All data was de-identified prior to analysis. Clinical Scales, Content Scales, and Harris-Lingoes Scales were scored at the time of evaluation. Restructured Clinical (RC) Scales were later scored by hand using scoring templates from Pearson Assessments. This study was approved by the Institutional Review Board at the University of Texas Southwestern Medical Center.

Data Analysis

Data were analyzed with the Statistical Package for the Social Sciences for Windows, version 15.0 (SPSS; 2006). Additional analyses were conducted with FZT, a program that computes correlation and regression comparison tests (Garbin, n.d.). A random subset of subjects was chosen for analysis for Hypothesis I. To determine inclusion into this subset, a random integer generator was used (Haahr, 2008). The following statistical analyses were conducted for the hypotheses and exploratory analyses.

Hypothesis I: Based on previous research, RC Scales are expected to yield high internal consistency coefficients ($.70 < \alpha < .99$) in this sample.

Hypothesis I was evaluated by computing Cronbach's (1951) alpha coefficient of internal consistency for RC and Clinical Scales. It was expected that alphas for the RC Scales in this sample would fall within the .70 to .99 range. A subset of 20 subjects for whom item responses were entered were used for analysis. These 20 subjects were chosen randomly using a true random integer generator (Haahr, 2008). Wygant and colleagues (2007) have previously used a subset of subjects from a medical patient

sample to examine alpha coefficients for the RC and Clinical Scales. In their subset, mean alpha coefficients approximated values found in previous psychometric research of the RC Scales (alpha = .74 for RC Scales, alpha = .66 for Clinical Scales; Tellegen et al., 2003; Sellbom et al., 2006; Simms et al., 2005). Additional exploratory analyses were conducted by examining mean item-total correlations for each Clinical and RC Scale.

Hypothesis II: Based on previous research, RC Scale intercorrelations are expected to fall within the .4 to .6 range in this sample, whereas Clinical Scale intercorrelations are expected to fall within the .6 to .8 range.

Hypothesis II was evaluated using Pearson product-moment correlations among the RC and Clinical Scales. Mean correlations between each RC Scale and the other RC Scales were computed, and likewise for the Clinical Scales.

Hypothesis IIIa: RC2 will show greater convergent validity with the IDS-SR, an external measure of depressive symptom severity, than will Clinical Scale 2.

Hypothesis IIIa was evaluated by computing Pearson product-moment correlations between RC2 and IDS-SR total score, and Clinical Scale 2 and IDS-SR total score. Steiger's (1980) Z, a statistic that tests the equality of correlation coefficient means for dependent samples, was used to determine if a significant difference existed between the two correlations. Findings were considered significant at the $p < .05$ level. Additional exploratory correlational analyses were performed to find the relationship between RCd and IDS-SR total score.

Hypothesis IIIb: The combination of RCd and RC2 will better predict total score on the IDS-SR than will Clinical Scale 2.

Hypothesis IIIb was evaluated using a multiple regression analysis. Initially, two separate regression analyses were conducted. In the first, RCd and RC2 were used as predictor variables and IDS-SR total score was used as a criterion variable. In the second, Clinical Scale 2 was used as a predictor variable, and IDS-SR total score was used as a criterion variable. Findings were considered significant at the $p < .05$ level. Additional exploratory regression analyses were conducted to identify whether other combinations of RCd, RC2, and Scale 2 would better account for the variance in IDS-SR total score.

Hypothesis IVa: RC2 will show greater discriminant validity with Hy_4, a scale labeled "Somatic Complaints," than will Scale 2.

Hypothesis IVa was evaluated by computing Pearson product-moment correlations between RC2 and Hy_4, and Clinical Scale 2 and Hy_4. Steiger's Z was used to determine if a significant difference existed between the two correlations. Findings were considered significant at the $p < .05$ level.

Hypothesis IVb: RC7 will show greater discriminant validity with Hy_4 than will Scale 7.

Hypothesis IVb was evaluated in the same manner as Hypothesis IIIc. Pearson product-moment correlations were computed between RC7 and Hy_4, and Clinical Scale 7 and Hy_4. Steiger's Z was used to determine if a significant difference existed between the two correlations. Findings were considered significant at the $p < .05$ level.

Exploratory Analysis I: RC Scale profiles will be examined for subjects whose Clinical Scale profiles display a "conversion V" pattern.

Exploratory Analysis I was evaluated by creating a subset of subjects whose Clinical Scale profiles displayed a conversion V pattern. Graham's (2006) definition of a conversion V was used: a 132/312 code type pattern in which Clinical Scales 1 and 3 are often significantly higher than Scale 2. Using this criterion, 35 subjects were identified as having a conversion V. Descriptive statistics were generated to examine RC and Clinical Scale scores in this subset of subjects. Regression analyses were conducted in order to determine whether individual RC Scales or combinations of RC Scales would account for significant proportions of the variance in whether or not a subject displayed a conversion V pattern.

Exploratory Analysis II: RC Scale profiles will be examined for subjects whose Clinical Scale scores display a "floating profile."

Exploratory Analysis II was evaluated in the same manner as Exploratory Hypothesis I. A subset of subjects whose Clinical Scale profiles displayed a floating profile was created. Clinical Scale profiles were considered floating if 5 or more Clinical Scales (not including Scales 0 and 5) were elevated ($T \geq 65$). Using this criterion, 38 subjects were identified as having a floating profile. Descriptive statistics were generated to examine RC and Clinical Scale scores in this subset of subjects. Regression analyses were conducted to determine whether any RC Scales or combinations of RC Scales could account for a significant amount of the variance in whether or not a subject displayed a floating profile. An independent samples *t*-test was used to determine whether scores on RC and Clinical Scales were significantly different for subjects with and without floating profiles.

Exploratory Analysis III: RC and Clinical Scale profiles will be compared for subjects whose IDS-SR total score indicates the presence of at least moderate depressive symptoms (total score ≥ 26).

Exploratory Analysis III was evaluated by creating a subset of subjects whose IDS-SR total score fell above the cutoff for moderate depressive symptoms (total score ≥ 26). Using this criterion, 57 subjects were identified as having moderate severity of depressive symptoms. Descriptive statistics were generated to examine RC and Clinical Scale scores in this subset of subjects. Regression analyses were conducted to determine whether any RC Scales or combinations of RC Scales could account for a significant amount of the variance in whether or not a subject fell within the moderate depressive severity range. An independent samples *t*-test was used to determine whether scores on RC and Clinical Scales were significantly different for subjects with and without IDS-SR scores in the moderate range of severity.

CHAPTER IV: RESULTS

Descriptive Data

Table 3 provides demographic information for the sample. The sample consisted of 60 males (44%) and 77 females (56%), ranging in age from 18 to 67 years, with a mean age at the time of assessment of 37.82 years (SD = 10.98). The majority of the sample was Caucasian (*n* = 116; 84.7%). Epilepsy diagnoses were available for the majority of the subjects and predominantly consisted of partial epilepsy, though the type of seizure was either unknown or uncharacterized in 25.5% (*n* = 35). Table 4 provides descriptive information regarding scores on the RC and Clinical Scales for the sample (see descriptive analyses of RC and Clinical Scales in "Exploratory Analyses" section).

Insert Tables 3 and 4 here

Analysis of Hypotheses

Hypothesis I. Based on previous research (e.g., Rogers et al., 2006; Sellbom et al., 2006; Simms et al., 2005), the first hypothesis predicted that the RC Scales would yield high internal consistency coefficients, with Cronbach's alpha falling in the .70 to .99 range. This hypothesis was tested by computing Cronbach's alpha for 20 subjects, randomly chosen, for whom item-by-item responses for RC and Clinical Scales were input. Though the Clinical Scales' internal consistency values were not included in the first hypothesis, they were examined in order to provide a point of comparison for the RC Scales. Internal consistency coefficients for each of the RC Scales and Clinical Scales 1 – 4 and 6 – 9 (henceforth referred to as the "Clinical Scales") are presented in Table 5. The data largely support Hypothesis I. Across the RC Scales, the average value for

Cronbach's alpha was .71. Six of the nine RC Scales had alpha coefficients that fell above the .70 threshold; for RC2 alpha equaled .62, for RC6 alpha equaled .33, and for RC8 alpha equaled .55. Of the RC Scales, RCd had the highest internal consistency coefficient (alpha = .90). Across the Clinical Scales, the average value of Cronbach's alpha was .68. Thus, the internal consistency coefficients for RC and Clinical Scales were roughly similar, with alpha for the Clinical Scales falling marginally lower, and slightly below the threshold for a "good" coefficient (i.e., alpha = .70).

Insert Table 5 here

Further exploratory analysis of internal consistency was completed by assessing item-total correlations for the RC and Clinical Scales (using the same data from the 20-subject subset). Mean item-total correlations for each of the RC Scales and Clinical Scales are presented in Table 5. Rogers and colleagues (2006) reported that optimal item-total correlations fall in the .15 to .50 range. Mean item-total correlations across the RC Scales ($M\ r$ = .311) and the Clinical Scales ($M\ r$ = .27) both fall within this range. Of the RC Scales, only RC6 fell outside the expected range, due primarily to low overall item endorsement in the subset. Of the Clinical Scales, only Scales 1, 4, 7, and 8 fell *within* the expected range. It appears that while internal consistency coefficients are roughly similar between the two sets of scales, the RC Scales may have slightly better internal consistency, as indicated by mostly optimal item-total correlations. All of the RC Scales used to test the remaining hypotheses in the current study displayed acceptable internal consistency. Of these, only RC2 displayed internal consistency outside the

optimal range: Cronbach's alpha for RC2 equaled .62, which is marginally below the threshold for an ideal score (alpha ≥ .70).

Hypothesis II. Based on previous research (e.g., Simms et al., 2005), the second hypothesis predicted that the RC Scales would display intercorrelations in the .4 to .6 range, whereas Clinical Scales would display intercorrelations in the .6 to .8 range. This hypothesis was evaluated by calculating Pearson product-moment correlations among the RC Scales and among the Clinical Scales. RC and Clinical Scale intercorrelations are presented in Table 6 and 7, respectively. Though the mean of intercorrelations across all RC Scale comparisons (*M r* = .41) fell within the expected range, the mean of intercorrelations across Clinical scales (*M r* = .46) did not. Of RC Scale intercorrelations, 34 of 36 (94.4%) fell within or below the expected range (.4 < *r* < .6) and only two of 36 (5.6%) were higher than predicted (RC2 and RC1 with RCd). Of Clinical Scale intercorrelations, seven of 28 (25.0%) fell within the predicted range (.6 < *r* < .8), whereas 20 of 28 (71.4%) fell below this range (0 < *r* < .59), and one of 28 (3.6%) surpassed the expected range.

Insert Tables 6 and 7 here

RC Scale intercorrelations tended to be lower than Clinical Scale intercorrelations, and fell within the expected range. However, Clinical Scale intercorrelation values were not as high as expected, falling largely below the predicted range. Therefore, the results only partially support the hypothesis.

Hypothesis IIIa. Part "a" of the third hypothesis predicted that RC2 would be more closely correlated with total score on the IDS-SR than would Clinical Scale 2. This

hypothesis was evaluated by calculating Pearson product-moment correlations between RC2 and IDS-SR total score, and Scale 2 and IDS-SR total score. Results are presented in Table 8. Correlations were significant for both comparisons. However, results indicated that the IDS-SR's correlation with Clinical Scale 2 ($r = .67$, $p < .01$) was significantly higher than its correlation with RC2 ($r = .54$, $p < .01$; Steiger's $Z = 2.52$, $p < .05$). Because RC2 was not more highly correlated with IDS-SR than Clinical Scale 2, the results do not support the hypothesis.

Insert Table 8 here

Further exploratory analysis indicated that RCd was more highly correlated with IDS-SR total score than either RC2 or Clinical Scale 2 ($r = .74$, $p < .01$). The correlation between RCd and IDS-SR was significantly greater than the correlation between RC2 and IDS-SR (Steiger's $Z = -4.10$, $p < .001$). However, the correlation between RCd and IDS-SR was not significantly greater than the correlation between Clinical Scale 2 and IDS-SR (Steiger's $Z = 1.69$, $p > .05$).

Hypothesis IIIb. Part "b" of the third hypothesis predicted that the combination of RCd and RC2 would better predict IDS-SR total score than would Clinical Scale 2. This hypothesis was tested with multiple linear regression (MLR) analyses. Results of regression analyses are presented in Tables 9 and 10. As a combined model, RCd and RC2 accounted for a significant amount (55.3%) of the variance in IDS-SR total score, $F_{(2, 134)} = 82.90$, $p < .001$. Individual regression coefficients were examined to determine the extent of each scale's contribution to the model. The regression coefficient associated with RCd was significantly different from zero, $t = 8.89$, $p < .001$. However, the

regression coefficient associated with RC2 was not significantly different from zero, t = .93, p = .36. Therefore, in this model, while RCd significantly contributes to the prediction of IDS-SR total score, RC2 does not. As an individual predictor, Scale 2 also accounted for a significant, but smaller, amount (45.1%) of the variance in IDS-SR total score, $F_{(1, 135)} = 110.94$, $p < .001$. The regression coefficient associated with Scale 2 was significantly different from zero, $t = 10.53$, $p < .001$. Though RC2 was not a significant contributor, the RCd/RC2 model nevertheless accounted for more variance in IDS-SR total score (55.3% vs. 45.1%). Therefore, the results support the hypothesis.

Insert Tables 9 and 10 here

Exploratory analysis indicated that as an individual predictor, RCd accounted for 55.0% of the variance in IDS-SR total score, $F_{(1, 135)} = 165.12$, $p < .001$. When RC2 was used as an individual predictor, it accounted for only 28.9% of the variance in IDS-SR total score, $F_{(1, 135)} = 54.98$, $p < .001$.

Because as individual models RCd and Clinical Scale 2 seemingly accounted for much more IDS-SR variance than RC2, post hoc regression analyses were used to determine if a model combining these two scales would better predict IDS-SR total score than any of the other models. Together, RCd and Clinical Scale 2 accounted for 58.5% of the variance in IDS-SR total score, $F_{(2, 134)} = 94.51$, $p < .001$. Both regression coefficients were significantly different from zero, $t = 6.58$, $p < .001$ (RCd); $t = 3.36$, p = .001 (Scale 2). This model was marginally better at predicting variance in IDS-SR score than RCd alone, and the slight increase in predictive power may have been due to item overlap between RCd and Scale 2. Therefore, while results supported the hypothesis that

as a combined model, RCd and RC2 better predicted IDS-SR total score than Scale 2, RCd was the simplest and most powerful predictor of IDS-SR total score.

Hypothesis IVa. Part "a" of the fourth hypothesis predicted that Clinical Scale 2 would be more highly correlated with the Harris-Lingoes scale Hy_4 ("Somatic Complaints"), than would RC2. This hypothesis was evaluated by calculating Pearson product-moment correlations between Clinical Scale 2 and Hy_4, and RC2 and Hy_4. Results are presented in Table 11. Correlations were significant for both comparisons, with the correlation between RC2 and Hy_4 ($r = .36$, $p < .01$) being significantly lower than the correlation between Clinical Scale 2 and Hy_4 ($r = .55$, $p < .01$; Steiger's $Z = -3.05$, $p < .01$). Therefore, the results supported the hypothesis that RC2 is less-correlated with a scale of somatic complaints than Clinical Scale 2. Examination of the correlation between RCd and Hy_4 in a post hoc analysis revealed a significant relationship ($r = .54$, $p < .01$) that was similar to that of Scale 2 and Hy_4 (Steiger's $Z = -.21$, $p > .05$).

Insert Table 11 here

Hypothesis IVb. Part "b" of the fourth hypothesis predicted that Clinical Scale 7 would be more highly correlated with the Harris-Lingoes scale Hy_4 ("Somatic Complaints"), than would RC7. This hypothesis was evaluated using a similar procedure as Hypothesis IVa: Pearson product-moment correlations between Clinical Scale 7 and Hy_4 ($r = .50$, $p < .01$), and RC7 and Hy_4 ($r = .521$, $p < .01$) were both significant (see Table 12), and not statistically different (Steiger's $Z = .37$, $p > .05$). Consequently, the results did not support the hypothesis that RC7 is less-correlated with a scale of somatic complaints than Clinical Scale 7.

Insert Table 12 here

Exploratory Analyses

As mentioned previously, in order to examine the characteristics of Clinical and RC Scales in the epilepsy sample as a whole, descriptive statistics were computed for both scale sets across all subjects (see Table 4). Figure 2 presents mean RC and Clinical Scale profiles across all subjects in the current study. Additionally, the mean score on Hy4 was 65.55 (SD = 17.04), slightly above the cutoff for clinical significance. The mean score on the IDS-SR was 23.50 (SD = 12.64), slightly below the cutoff for moderate severity of depression (total score ≥ 26).

In this sample, all RC Scales were below a mean T score of 65. Only RC1 had a mean T score above 60 (T = 64.21 [SD = 13.54]). Of the Clinical Scales, Scales 1 and 2 were above the T = 65 level, though only barely so (T = 65.03 [SD = 13.11] and T = 65.77 [SD = 13.57], respectively). The mean T score for Scale 3 approached clinical significance (T = 64.50 [SD = 15.09]), and its median value was T = 65. All mean RC Scale T scores were lower than those of the corresponding Clinical Scales, except for RC1 and Scale 1, which had similar mean T scores (T = 64.21 [SD = 13.54] and T = 65.03 [SD = 13.11], respectively. Median T scores closely resembled mean T scores for all Clinical and RC Scales (see Table 4).

Insert Figure 2 here

Analysis of the frequency of significant elevations provided information regarding the prevalence of psychiatric symptoms in this sample. Examination of

depression scales revealed that 51.1% (*n* = 70) of subjects had significant elevations on Scale 2 (T ≥ 65) at the time of their evaluation, in contrast to only 24.1% (*n* = 33) for RCd, and 21.9% (*n* = 30) for RC2. With respect to anxiety, a greater percentage of subjects elevated on Scale 7 (32.1%; *n* = 44) compared to RC7 (14.6%; *n* = 20). Scale 1 and RC1 were in closer agreement, with 48.9% (*n* = 67) subjects scoring at or above T = 65 on Scale 1, and 48.2% (*n* = 66) of subjects doing so on RC1.

Following Graham's (2006) suggestions for defining three-point code types (i.e., the three highest T scores, all of which surpass T = 60 and fall within five points of each other), the mean Clinical Scale code type across all subjects would be a 2-1-3 code type. The mean T scores for Scales 8 and 7 also fell within five points of Scales 2, 1, and 3, and were both above 60. Profile interpretation is not recommended for RC Scales, and in any case, only RC1 had a mean elevation above T = 60, the threshold for interpretability as part of a code type (Ben-Porath, 2008; Graham, 2006).

Exploratory Analysis I. The first exploratory analysis sought to examine RC Scale characteristics for subjects whose Clinical Scale profiles displayed a "conversion V" pattern. Of the entire sample of epilepsy patients (*n* = 137), 35 subjects (25.6%) displayed a conversion V pattern. The mean T scores for Clinical Scales 1, 2, and 3 were 74.74 (SD = 10.28), 65.20 (SD = 10.43), and 75.60 (SD = 9.86), respectively. Table 13 presents descriptive statistics for Clinical and RC Scales in this subset. Figure 3 presents the mean Clinical and RC Scale profile for subjects within this subset and outside this subset.

Insert Table 13 here

In the conversion V subset, no other Clinical Scales had mean T scores above 65. The only RC Scale to average a T score above 65 was RC1 (T = 70.40 [SD = 11.78]). The mean T scores for RCd and RC2 were 52.83 (SD = 8.35) and 55.71 (SD = 10.98), respectively. Of the 35 subjects, only four (11.4%) had T scores on RCd above 65, and only six (17.1%) had T scores on RC2 above 65. The average score on the IDS-SR was slightly above the threshold for moderate depression (total score = 26.66 [SD = 11.03]). The median score on the IDS-SR was 25, indicating that outliers did not affect the mean in a pronounced way.

Insert Figure 3 here

The mean RC and Clinical Scale profile for the conversion V subset differed substantially from the mean RC and Clinical Scale profiles for the remainder of the sample. By virtue of its inclusion criteria (i.e., Scales 1 and 3 significantly higher than Scale 2), the conversion V subset failed to show the near-equivalence between Clinical and RC Scale measures of depression and health concerns that characterized both the remainder of the sample, and the sample as a whole.

Regression analyses were used to determine if any RC Scales were able to significantly predict whether or not a subject would display a conversion V pattern. Results are presented in Tables 14 and 15. No RC Scale or combination of two RC Scales was able to account for more than 25% of the variance in whether or not any given patient would display a conversion V. Of the regression models using individual scales as predictors, RC3 (“Cynicism”) accounted for the largest amount of variance (8.9%), $F_{(1, 135)} = 13.21$, $p < .001$. In this model, the regression coefficient associated with RC3 was

significantly different from zero, $t = -3.63$, $p < .001$. Of the regression models with two RC Scales, the combination of RC3 and RC1 accounted for the largest amount of variance (24.1%), $F_{(2, 134)} = 21.27$, $p < .001$. In this model, the regression coefficient associated with RC3 was significantly different from zero, $t = -5.46$, $p < .001$. The regression coefficient associated with RC1 was also significantly different from zero, $t = 5.18$, $p < .001$. A model using RCd and RC1 was able to account for only slightly less of the variance in whether or not a patient displayed a conversion V (21.3%), $F_{(2, 134)} = 18.12$, $p < .001$. In this model, the regression coefficient associated with RCd was significantly different from zero, $t = -4.89$, $p < .001$. The regression coefficient associated with RC1 was also significantly different from zero, $t = 5.65$, $p < .001$.

Insert Tables 14 and 15 here

Exploratory Analysis II. The second exploratory analysis sought to examine RC Scale characteristics for subjects whose Clinical Scale profiles displayed a "floating profile." Of the entire sample of epilepsy patients ($n = 137$), 38 subjects (27.7%) displayed a floating profile. Table 16 presents descriptive statistics for Clinical and RC Scales in this subset. Figure 4 presents mean Clinical and RC Scale profiles for subjects within this subset and outside this subset.

Insert Table 16 here

In the subset, the mean T scores for almost all the Clinical Scales were above 65, with Scale 9 being the exception (T = 58.71 [SD = 10.19]). The mean score on the Infrequency (F) scale was 68.87 (SD = 12.88). Of the RC Scales, only RCd, RC1, RC2,

and RC8 had T scores above 65 (68.58 [SD = 11.09], 74.55 [SD = 13.16], 68.47 [SD = 11.90], and 67.26 [SD = 11.36], respectively). The mean total score on the IDS-SR for subjects with floating profiles was solidly within the moderate severity range (34.37 [SD = 12.00]).

Insert Figure 4 here

Independent samples *t*-tests were used to determine whether significant differences existed between the Clinical and RC Scale T scores of subjects with and without floating profiles. Results are reported in Table 17. For a more conservative comparison of the two groups, equal variances were not assumed. With two exceptions (RC9 and Clinical Scale 9), mean T scores on all Clinical and RC Scales were significantly lower for subjects without floating profiles than for subjects with floating profiles at $p \leq .001$. For subjects with floating profiles, the T score on Clinical Scale 9 was not significantly higher than for those subjects without floating profiles, t (69.92) = -1.76, p = .08. RC9 T scores were significantly higher, but less so than for the other scales, t (50.94) = -2.88, $p < .01$.

Insert Table 17 here

As with the first exploratory analysis, regression analyses were completed in order to determine whether certain of the RC Scales were particularly good at accounting for the variance in whether or not a subject displayed a floating profile. Results are presented in Tables 18 and 19. Almost all of the regression models that accounted for noteworthy amounts of variance accounted for around 30% to 45%, whether they

included one, two, or three predictors. These models were all comprised of various combinations of RCd, RC1, RC2, and RC7. Of the regression models using individual scales as predictors, RCd accounted for the largest amount of variance (39.0%), $F_{(1, 135)} = 86.37$, $p < .001$. As an individual predictor, RCd's regression coefficient was significantly different from zero, $t = 9.29$, $p < .001$. Of the two-scale models, the combination of RCd and RC2 accounted for the most variance (42.2%), $F_{(2, 134)} = 48.93$, $p < .001$. In this model, the regression coefficient associated with RCd was significantly different from zero, $t = 5.22$, $p < .001$. The regression coefficient associated with RC2 was also significantly different from zero, $t = 2.72$, $p < .01$. Of the three-scale models, RCd, RC1, and RC2 together accounted for 44.2% of the variance in whether a patient displayed a floating profile, $F_{(3, 133)} = 35.15$, $p < .001$. On an individual basis, RCd contributed most heavily in this model, $t = 3.85$, $p < .001$. The regression coefficient for RC2 was the next-strongest, $t = 2.65$, $p < .01$. The regression coefficient for RC1 was also significantly different from zero, $t = 2.19$, $p < .05$. Incorporating more than three scales into a model, or including scales other than RCd, RC1, RC2, or RC7, tended not to contribute in terms of accounting for variance, and in fact often diminished the power of the model.

Insert Tables 18 and 19 here

Exploratory Analysis III. The third exploratory analysis sought to examine RC and Clinical Scale characteristics for subjects whose IDS-SR total score fell at or above the threshold for a designation of "moderate" depressive severity (total score ≥ 26). Of the entire sample of epilepsy patients ($n = 137$), 57 subjects (41.6%) were designated

with moderate depressive severity via the IDS-SR. Table 20 presents descriptive statistics for Clinical and RC Scales in this subset. Figure 5 presents mean Clinical and RC Scale profiles for subjects within this subset and outside this subset. In this subset, the mean T score for Clinical Scales 1, 2, 3, 7, and 8 was above T = 65 (see Table 20), whereas only three RC Scales had mean T scores above 65: RCd, RC1, and RC8.

Insert Table 20 and Figure 5 here

Independent samples *t*-tests were used to determine whether significant differences existed between the Clinical and RC Scale T scores of subjects with and without at least moderate depression as classified by the IDS-SR. Results are reported in Table 21. For a conservative comparison of the two groups, equal variances were not assumed. Hypothetically, if all scales within a set were largely independent of each other, only a scale of depressive symptoms should differ between subjects within and outside this subset (if patients with depression do not have other comorbid symptoms). Due to item overlap and higher intercorrelations, it was expected that this would not be the case for the Clinical Scales. Results indicated that Scales 1 – 4 and 6 – 8 were significantly higher for subjects within the subset than for the remainder of subjects at $p <$ .001. Scale 9 was significantly higher for subjects within the subset at $p < .05$. Based on the theoretical goals of the construction of the RC Scales (e.g., reduced covariation, increased discriminant validity), it was expected that only RC Scales relating to depression would be more elevated in this subset than in the remainder of the sample. However, this was not the case: mean T scores for all RC Scales were significantly higher in this subset than for the rest of the subjects at $p < .001$. Because the RC Scales were

fairly independent in this sample (see Hypothesis II), these results seem to indicate that the members of the moderate depressive severity subset were more than "just" depressed; their RC Scale profiles indicated that they tended to experience higher levels of psychopathology in general.

Insert Table 21 here

As with the other exploratory analyses, regression analyses were carried out in order to determine whether any RC Scales could account for significant amounts of the variance in whether a subject scored in the moderate range of depressive severity. Consistent with findings from the hypotheses, scales relating to depression and health were most closely related to the extra-test measure of depressive severity. Of the individual predictors, RCd accounted for 45.8% of the variance in whether a patient scored at or above the moderate range of depressive severity, $F_{(1, 135)} = 114.27$, $p < .001$. In this model, the regression coefficient associated with RCd was significantly different from zero, $t = 10.69$, $p < .001$. RC1 was able to account for 44.6% of the variance, $F_{(1, 135)} = 108.71$, $p < .001$. The regression coefficient associated with RC1 was significant, $t = 10.43$, $p < .001$. RC2 was again less-related to the IDS-SR than RCd, accounting for 22.1% of the variance in whether a subject fell at or above the moderate range of depressive severity, $F_{(1, 135)} = 38.40$, $p < .001$. The regression coefficient associated with RC2 was significant, $t = 6.197$, $p < .001$. As was the case in Hypothesis IIb, in models combining RC2 with RCd or RC1, RC2 was not as contributive to the predictive ability of the model. A model combining RC2 and RC1 accounted for 49.4% of the variance, $F_{(2, 134)} = 65.35$, $p < .001$. The coefficient associated with RC1 was significant, $t = 8.49$, p

< .001. The coefficient associated with RC2 also significant, but smaller, $t = 3.55$, $p < .01$. A model combining RCd and RC2 accounted for 45.9% of the variance, $F_{(2, 134)} = 56.81$, $p < .001$. In this model, the coefficient associated with RCd was significant, $t = 7.67$, $p < .001$. However, consistent with the results of Hypothesis IIIb, the coefficient associated with RC2 was non-significant, $t = .33$, $p = .74$. The best predictor of whether or not a subject fell within the moderate depressive severity subset was the combination of RCd and RC1, which accounted for 57.9% of the variance, $F_{(2, 134)} = 92.26$, $p < .001$. RCd and RC1 made comparable contributions to this model. The regression coefficient associated with RCd was significant, $t = 6.51$, $p < .001$. The coefficient associated with RC1 was similarly significant, $t = 6.20$, $p < .001$. These results further substantiate the notion that health complaints and subjective depression seem to be closely related in this sample.

Insert Tables 22 and 23 here

CHAPTER V: DISCUSSION

Psychological testing is an important component in the treatment of patients with epilepsy. Research has indicated that mood and anxiety disorders are significantly more prevalent in patients with epilepsy than in the general population, with estimates of comorbidity reaching 30% for anxiety disorders, and anywhere from 20% to 80% for depressive disorders (Jones et al., 2005a; Kanner & Nieto, 1999; Kanner & Palac, 2000; Grabowska-Gryzb et al., 2006). In medical settings, the MMPI-2 is often used to detect psychopathology that may interfere with treatment and recovery. However, researchers have identified potential difficulties with the MMPI-2's Clinical Scales, both in terms of general psychometric properties, and in terms of their usefulness with patients with neurologic disorders. While the Clinical Scales' method of construction led to heterogeneity within scales, high intercorrelations, and covariation, it may also have resulted in the inclusion of extraneous content within the scales.

Previous research has indicated that the Clinical Scales, including Scales 2 and 7, are vulnerable to inflation due to the inclusion of items reflecting somatic complaints and neurologic symptoms. These studies have focused on patients with neurologic disorders such as multiple sclerosis (Meyerink, Reitan, & Selz, 1988), TBI (Gass, 1991), and stroke (Gass, 1996), as well as patients with epilepsy (Derry et al., 1997; Derry et al., 2002; Karzmark, Zeifert, & Barry, 2001). Correction procedures are often proposed in order to eliminate some of the spurious inflation in the Clinical Scales. However, no studies have examined whether using alternates such as the RC Scales, which research has indicated are more homogeneous and less susceptible to nonspecific variance, might improve discriminant validity in screening for mood and anxiety symptoms in patients with

neurologic disease. Additionally, no studies have examined the psychometric properties or profile characteristics of the RC Scales in an epilepsy sample. The goals of the current study were to (1) examine the general psychometric properties of the RC Scales (including internal consistency and discriminant validity between scales) in a sample of patients with epilepsy; (2) to compare convergent and discriminant validities of the Clinical and RC Scales in an epilepsy sample; and (3) to determine characteristics of RC Scale profiles in a sample of patients with epilepsy. To these ends, psychological testing data from 137 patients with epilepsy were compiled, and scores on the MMPI-2 and IDS-SR were examined. The findings and limitations of the current study will be discussed in the following sections.

Discussion of Hypotheses

Hypothesis I: Internal Consistency

In the original publication of the RC Scales, Tellegen and colleagues (2003) reported alpha coefficients that routinely exceeded .80. This high degree of internal consistency was not unexpected, given that the scales were constructed via factor analysis, and despite that they contain, on average, roughly 60% fewer items than the Clinical Scales (Simms et al., 2005).

Results of the current study supported the hypothesis that the mean value of Cronbach's alpha for the RC Scales would fall within an optimal range (.70 < alpha < .99). In a subset of 20 subjects, the mean alpha coefficient for the RC Scales was .71. Of the nine RC Scales, 6 had alphas above .70 (see Table 5). The exceptions were RC2 (alpha = .62), RC8 (alpha = .55), and RC6 (alpha = .33). For RC6, the low alpha coefficient appears to be the result of low overall endorsement of items within the scale:

of the 17 items on RC6, 11 were not endorsed by any of the 20 subjects in the subset. To provide a point of comparison, alpha coefficients were computed for the Clinical Scales; for the 20 subjects in the subset, the mean alpha coefficient was .68. While mean alpha values for the RC and Clinical Scale sets were roughly equivalent, the mean alpha coefficient for the Clinical Scales was slightly below the "good" range.

Data for this analysis were limited (n = 20). Increasing the number of subjects would likely have increased values for Cronbach's alpha for both the RC and Clinical Scales. Cronbach (1951, p. 328) stated that hypothetically, "as the number of items increases, [alpha] rises toward 1.00." That the RC Scales achieved the .70 threshold for alpha with such a small sample size can be interpreted as an indicator of good internal consistency, and the mean value of alpha would likely only rise, were more subjects to be added to the analysis.

Item-total correlations were also examined to assess internal consistency (see Table 5). Across the RC and Clinical Scales, mean item-total correlations were .31 and .27, respectively. Both of these values fall within the optimal range ($.15 < r < .50$) outlined by Rogers and colleagues (2006). However, while eight of the nine RC Scales fell within this optimal range, only three Clinical Scales did so. Therefore, when item-total correlations are considered, it appears that the RC Scales have slightly better internal consistency, despite the fact that the mean alpha coefficients for each set of scales were roughly similar. These results indicate that previous findings regarding internal consistency of the RC Scales (e.g., Simms et al., 2005; Wallace & Liljequist, 2005) appear to generalize to an epilepsy sample.

Hypothesis II: Internal Structure

The empirical-keying method used to compose the Clinical Scales resulted in significant item overlap between scales, with an average of 6.4 shared items between each pair of the 10 Clinical Scales (Simms et al., 2005). This item overlap can potentially lead to increased intercorrelations, which reduce discriminant validity among the scales. In the current study, scale intercorrelations were calculated in order to assess the discriminant validity of the RC and Clinical Scales in an epilepsy sample.

Results partially supported the hypothesis that RC Scales would display intercorrelations in the .4 to .6 range, whereas Clinical Scales would display intercorrelations in the .6 to .8 range. The mean intercorrelation for RC Scales fell within the predicted range (*M r* = .41), though not far from the value for the Clinical Scales (*M r* = .46). These results are similar to those of previous research (e.g., Forbey & Ben-Porath, 2007; Sellbom, Ben-Porath, & Graham, 2006), insofar as RC Scale intercorrelations were moderately, but not drastically, lower than Clinical Scale intercorrelations.

Among the Clinical Scales, 20 of 28 intercorrelations (71.4%) fell below the hypothesized range (i.e., $r > .60$). Scale 8 displayed the highest intercorrelations with other scales, and accounted for five of the eight correlations above .60. Scale 9 had the lowest intercorrelations among the Clinical Scales, with four of its seven intercorrelations failing to achieve significance. Overall, intercorrelations among the Clinical Scales were not as high as expected; however, of the 13 intercorrelations that involved either Scale 2 or 7, 10 were above .50. Therefore, in samples known to have a high prevalence of

depression or anxiety symptoms, such as epilepsy, interpretation may be significantly complicated by Clinical Scale intercorrelations.

Among the RC Scales, 34 of 36 intercorrelations (94.4%) fell within or below the expected range, indicating fairly good discriminant validity between the majority of scales. The only intercorrelations to exceed the hypothesized range were between RCd and RC2 (r = .67), and RCd and RC7 (r = .80). Unexpectedly high correlations between these pairs of scales, especially between RCd and RC7, have been identified in previous research (Simms et al., 2005). Simms and colleagues (2005) have proposed that in general clinical samples, correspondence between RCd and RC7 may indicate that RC7 is measuring generalized anxiety features. As GAD is characterized primarily by general distress and negative emotionality, RC7 may have in part "recreated" a general distress factor in a general clinical sample.

In instances where two scales are highly intercorrelated, such as these, it seems reasonable to conclude that either (1) the scales possess substantial construct overlap, or (2) that the constructs the scales measure are very closely related in the particular sample. The former explanation may be tempting, especially given that RCd was seeded with items from Clinical Scales 2 and 7. The latter explanation may also be plausible, especially with samples such as epilepsy patients, where the constructs in question (depression and anxiety) are known to have high comorbidity and at times, similar etiologies. A combination of these two explanations may account for the large correlations between RCd and RC2, and RCd and RC7 in this sample: a degree of construct overlap, combined with comorbidity of symptoms, could generate unexpected

intercorrelations. The apparent covariation of several RC Scales is further examined in the following sections.

Hypothesis III: Convergent Validity

As a whole, the third hypothesis predicted that RC2 would be more closely related to an extra-test measure of depressive severity than Clinical Scale 2. The rationale for this expectation rested on the theoretical goals that guided the construction of RC2 and the IDS. Recently, theorists have emphasized the predominance of anhedonic symptoms in MDD, as opposed to dysphoric mood, which may be a feature of many different psychiatric diagnoses (Clark & Watson, 1991; Watson, 2005). Based in part on this rationale, RC2, the "depression" scale of the RC Scales, focuses solely on the absence of positive emotions.

The IDS-SR was used as a criterion measure for the presence of depression in the current study. Part of the theoretical rationale in developing the IDS was to create an inventory that, first and foremost, would cover the nine criterion symptoms for a MDE (Rush et al., 1986, 1996). Because the IDS-SR was developed from the DSM-IV diagnostic criteria for major depression, and because RC2 focuses on the "core" component of major depression, part "a" of hypothesis three predicted that RC2 would better correlate with IDS-SR total score than Clinical Scale 2. It was expected that as a heterogeneous measure of diffuse symptoms, Scale 2 would not correspond as well with the IDS-SR total score. However, the results did not support this hypothesis, and in fact, the opposite was true; Scale 2 was significantly more correlated with IDS-SR total score than RC2. There are a couple potential explanations for why the results did not support the hypothesis. It seems that either (1) the IDS-SR did not adequately measure

depression in this sample, or (2) RC2 did not adequately measure depression in this sample.

Part "a" of the third hypothesis assumed that high scores on the IDS-SR would closely correspond with the presence of a MDE or MDD. However, it may be that this was not the case. Though the IDS-SR was developed based upon symptom coverage for DSM-IV MDE criteria, it also includes items tapping symptoms such as mood reactivity, leaden paralysis, interpersonal sensitivity, irritability, and panic attacks, among others. Other questionnaires, such as the Beck Depression Inventory (BDI), have been identified as measures of demoralization, and not major depression (Ben-Porath, 2008; Sellbom et al., 2006). Though the IDS was developed as an alternative to the BDI, and was intended as a more accurate measure of depression (see Appendix A), it may be that it too actually measures demoralization as a core construct. This idea is supported by the fact that in this sample, RCd was very highly correlated with total score on the IDS-SR (r = .74).

On the other hand, part "a" of the third hypothesis also assumed that clinically significant scores ($T \geq 65$) on RC2 would closely correspond with the presence of a MDE or MDD. It may be that the IDS-SR adequately measured major depression in this sample, but that RC2 did not. This may have been especially true, given that interictal depression often presents as atypical depression (Blumer, Montouris, and Hermann, 1995; Kanner & Nieto 1999; Kanner & Palac, 2000; Mendez et al., 1993). Besides the nine DSM-IV criteria for a MDE, the IDS was also designed to cover symptoms of atypical depression (e.g., mood reactivity, hypersomnia, increased appetite, leaden paralysis, interpersonal rejection sensitivity), among other subtypes. If many of the

depressive symptoms of epilepsy patients present atypically, it could be that the IDS-SR sufficiently measured them, whereas RC2 could not.

Additionally, because the DSM-IV criteria for a MDE—the basis for the IDS—give equal weight to dysphoric mood and anhedonia, it may have been that many high scorers on the IDS-SR were experiencing dysphoric mood at the time of their evaluation, but not anhedonia (especially if they were experiencing atypical depression, which usually contains elements of dysphoric mood, but not loss of pleasure). Expecting these circumstances, part "b" of the third hypothesis predicted that the combination of RCd and RC2 would better account for the variance in IDS-SR total score than Clinical Scale 2. Watson and Tellegen's (1985) map of affect described "Unpleasantness," the factor that was eventually re-termed "demoralization," with the descriptors "blue," "sad," "sorry," and, "unhappy." Therefore, it was expected that as a measure of this factor, RCd would account for dysphoric mood, whereas RC2 would account for anhedonic symptoms in predicting major depression.

The results supported the hypothesis that as a combined model, RCd/RC2 accounted for more of the variance in IDS-SR total score than Clinical Scale 2 (55% versus 45%). However, further analysis revealed that in the RCd/RC2 model, RC2 did not significantly contribute to the prediction of IDS-SR total score. As an individual predictor, RCd accounted for around 55% of the variance in IDS-SR total score. Because RCd and Clinical Scale 2 were the most powerful predictors of the IDS-SR, they were evaluated as a combined model. Together, RCd and Scale 2 were able to account for 58.5% of the variance in IDS-SR total score, marginally more than RCd alone. Because some items overlap between RCd and Scale 2, the slight increase in predictive power

might be due to redundant weighting of these items (i.e., items getting "counted twice"). It seems that in this sample, RCd was the simplest and most efficient individual predictor of IDS-SR total score.

These results are consistent with the findings of part "a" of the third hypothesis. Again, RC2 was less related to total score on the IDS-SR than hypothesized. In fact, RCd was actually most closely related to IDS-SR total score. This additional evidence further demonstrates that one of two things may be true for this sample: (1) the IDS-SR did not adequately measure depression; or (2) RC2 did not adequately measure depression. The evidence seems to provide the most support for the latter of these statements. While previous research has shown that RC2 is a good predictor of major depression (e.g., Sellbom et al., 2006), its utility may be hampered in an epilepsy sample because of the prevalence of atypical symptoms. As a measure that includes atypical symptom coverage, the IDS-SR seems more likely to be sufficiently measuring depressive symptoms in this sample. Though atypical depression is typically characterized by mood reactivity, and not anhedonia, it does include the pervasive sense of sadness common to many depressive disorders. Therefore, of the RC Scales, it seems that RCd, and not RC2, is likely the best indicator of depressive symptoms in patients with epilepsy.

Hypothesis IV: Discriminant Validity

On the whole, the fourth hypothesis predicted RC2 and RC7 would be more distinct and less prone to spurious inflation from genuine somatic or neurologic symptoms than their Clinical Scale counterparts (Scales 2 and 7). In the absence of an extra-test measure of health problems, an MMPI-2 scale of health concerns was used.

The Hy_4 scale was used as a criterion for somatic symptoms because among all the health-related subscales on the MMPI-2, Hy_4 has the smallest item overlap with Scale 2 (only item 18 contributes to both scales), and no overlap with Scale 7, RC2, or RC7. With only one shared item, Hy_4 and Scale 2 are not likely to co-vary because of item overlap.

Part "a" of the fourth hypothesis hypothesized that RC2 would have better discriminant validity from a measure of somatic complaints (Hy_4) than Clinical Scale 2. Somatic symptoms are often experienced as sequelae of depression (Maxmen & Ward, 1995). However, a proportion of the items that are included in Clinical Scale 2 may reflect genuine cognitive or physical symptoms of neurologic dysfunction, and not psychopathology (Gass, 1991). For instance, item 142, which contributes to Scale 2, states, "I have never had a fit or convulsion." On average, studies of neurologic disease samples have found nine items on Scale 2 that reflect authentic neurologic symptoms (Karzmark, Zeifert, & Barry, 2001). By contrast, RC2 is devoid of somatic symptoms, as its items focus only on the absence of positive emotions.

Part "a" of the fourth hypothesis assumed first that on average, those subjects who had experienced many of the health concerns that often accompany epilepsy would likely elevate on Hy_4. Second, it was thought that subjects with high scores on Hy_4 would be more likely to endorse the somatic items within Clinical Scale 2, thus artificially inflating estimates of psychopathology. As hypothesized, the correlation between RC2 and Hy_4 was significantly smaller than the correlation between Scale 2 and Hy_4. It appears that in this sample, RC2 is generally a "purer," less somatically-biased measure of depression than Scale 2.

However, results from the third hypothesis seemed to indicate that RC2 is not an ideal measure of depression in this sample. Rather, RCd may be a better indicator for the atypical presentation of depression that frequently accompanies epilepsy. Therefore, a post hoc analysis was conducted to determine if RCd also displayed greater discriminant validity with Hy_4 than Scale 2. Results indicated that this was not the case; the correlations between RCd and Hy_4, and Scale 2 and Hy_4, were almost equivalent (r = .54 and r = .55, respectively). This finding may invalidate the notion that covariance between scores on Scale 2 and Hy_4 would signify spurious inflation on Scale 2 due to somatic content. Were this assumption to be correct, it follows that every measure without somatic items (e.g., RCd) should be less correlated with Hy_4 than a measure with somatic items (e.g., Scale 2).

A more likely second explanation may be that unlike RC2, RCd is somehow related to health concerns in this sample. In this sample, both Scale 2 and RCd displayed large correlations (i.e., $r > .5$) with Hy_4. While Scale 2 contains a large number of somatically-based items, RCd does not, so it is very unlikely that RCd was somehow spuriously inflated by genuine health concerns in this sample. It seems more plausible that as epilepsy-related health concerns increase, the potential for a sense of demoralization or dysphoric mood also increases.

RCd and Scale 2 were also nearly equivalently correlated with RC1 (r = .56 and r = .58, respectively). These results replicate findings of previous research (e.g., Sellbom, Ben-Porath, & Graham, 2006; Wallace & Liljequist, 2005). Due to the absence of item overlap, the correlation between RCd and RC1 seems to be indicative of an authentic relationship between health concerns and demoralization. Because Scale 2 contains some

demoralization variance, as well as health-related questions, it is likely that construct overlap and the relationship between health concerns and demoralization generally work in conjunction to elevate Scale 2's correlations with somatic measures.

Somatic symptoms also frequently accompany anxiety disorders. In fact, many patients with anxiety disorders initially present to physicians with physical complaints such as palpitations, upset stomach, dizziness, or chest tightness (Maxmen & Ward, 1995). However, as with Clinical Scale 2, a number of researchers have identified items on Scale 7 that may reflect genuine neurologic symptoms. Across various neurologic patient samples, six items from Scale 7 have been consistently identified as reflective of authentic neurologic symptoms. As with RC2, RC7 is devoid of somatic (or neurologic) content.

Part "b" of the fourth hypothesis predicted that RC7 would have better discriminant validity from Hy_4 than Scale 7. Results did not support this hypothesis: RC7 and Scale 7 were nearly equivalently correlated with Hy_4. (r = .50 and r = .52, respectively).

There are several potential interpretations of why the results did not indicate the expected relationship. Part "b" of the fourth hypothesis assumed that subjects who experienced many seizure sequelae would score highly on Hy_4 and be more prone to endorse the neurologic symptoms on Scale 7. However, because the six neurologic items tend to reflect cognitive deficits, and are not precisely related to somatic symptoms, their endorsement may not have been related to endorsement of somatic symptoms (and therefore Scale 7 would not be as closely related to Hy_4 as hypothesized).

Alternatively, the six neurologic items may not be representative of symptoms that frequently result from epilepsy or seizures. Gass (1991, 1996) has removed neurologic items as part of an MMPI-2 correction procedure for use with TBI patients, cerebrovascular disease patients, and stroke patients. However, Meyerink, Reitan, and Selz (1988) found that with multiple sclerosis patients, the six neurologic items had a negligible impact on overall T scores for Scale 7. In the current study, this may also be true, and this negligible impact may account for the marginal difference between the two correlations (r = .50 versus r = .52).

Both of these explanations may account for the absence of a significant difference between Hy_4's correlations with Scale 7 and RC7. However, neither explains why both Scale 7 and RC7 are similarly correlated with Hy_4—or why they are correlated with Hy_4 at all. It could be that in this sample, both Scale 7 and RC7 nearly equivalently captured the presence of anxiety symptoms. Their large correlations with Hy_4 may indicate a genuine relationship between health complaints and anxiety symptoms in epilepsy patients. Patients with many seizure sequelae may be particularly prone to developing fears related to seizures occurring in an unfamiliar or uncontrolled environment, or fear of public embarrassment (Beyenburg et al., 2005).

Beyenburg and colleagues (2005, p. 163) have stated that "as the burden of epilepsy increases, so does the anxiety." This relationship may be part of a larger trend of health problems leading to or exacerbating existing psychiatric problems. Part "a" of the fourth hypothesis indicated that mood symptoms also seem to be positively correlated with health concerns in this sample. Without additional extra-test correlates, further interpretation becomes very difficult, as it is unclear whether close relationships between

RC Scale measures of health complaints, depression, and anxiety are due to construct overlap, or authentic comorbidities.

As they are devoid of health-related items, the relationship between RCd or RC7 and health-related measures (i.e., Hy_4, RC1) would seem to indicate a genuine correlation between dysphoric mood and somatic symptoms in this sample. However, similar correlations have been reported in non-medical samples (e.g., Forbey & Ben-Porath, 2007; Wallace & Liljequist, 2005). Therefore, the apparent close relationship between health concerns and mood/anxiety symptoms may generalize to more than just medical populations. The occurrence of bodily symptoms in depression and anxiety is well known, and to a degree, slightly elevated intercorrelations ($.5 < r < .6$) among the RC Scales likely reflect these comorbidities.

Discussion of Exploratory Analyses

In the current study, the Exploratory Analyses focused on identifying characteristics of particular RC Scale profiles associated with conversion V and floating Clinical Scale profiles, and the profiles of subjects who endorsed at least moderate depressive severity on the IDS-SR. RC Scale characteristics were also examined for the sample of epilepsy patients as a whole. The T score means for the RC Scales were all lower than those of the corresponding Clinical Scales, with the exception of RC1 and Scale 1, which were nearly equivalent. This equivalency has been previously reported, but not accounted for, in outpatient and inpatient clinical samples (Sellbom et al., 2006; Wallace & Liljequist, 2005). The correspondence of Scale 1 and RC1 mean scores could indicate that while RC1 may be a more discreet and efficient measure, both scales are ultimately adequate gauges of health complaints. Of corresponding pairs of RC and

Clinical Scales, RC1 and Scale 1 have the highest degree of item overlap, with 74% (n = 20) of RC1's items contributing to Scale 1 (Simms et al., 2005).

The mean T scores of the RC Scales were all below 65, whereas Clinical Scales 1 and 2 displayed mean T scores above 65. Additionally, the median T scores for each RC and Clinical Scale closely resembled the mean T scores, indicating that none of the scales used for subsequent analyses were overly influenced by outliers.

In the current study, the frequency of significant ($T \geq 65$) elevations on RC and Clinical Scales were examined to compare the prevalence of significant mood and anxiety symptoms to previous findings. Jones and colleagues (2005) used the Structured Clinical Interview for DSM-IV Disorders (SCID), a "gold standard" in identifying diagnosable psychiatric disorders, to find the prevalence of mood and anxiety disorders in an epilepsy sample. Their results indicated that anxiety disorders were indicated in 30.4% of their sample, and that mood disorders were indicated in 21.8% of their sample. In the current sample, rates of significant T scores on depressive symptom RC Scales were comparable to the findings of Jones and colleagues (2005). RCd indicated that 24.1% of subjects were experiencing significant demoralization at the time of their assessment, and RC2 indicated that 21.9% were experiencing low positive emotions. By contrast, Scale 2 identified almost half the sample (48.9%) as experiencing significant depressive symptoms, far more than the SCID in Jones and colleagues' (2005) study. For scales of anxiety, the opposite trend was observed. In this sample, the rate of significant T scores on Scale 7 (32.1%) was comparable to rates of anxiety diagnoses in Jones and colleagues' (2005) study. By contrast, RC7 only identified 14.6% of subjects as experiencing dysfunctional negative emotions at the time of their assessment.

Elevations may also be examined in terms of code types. Graham (2006) has suggested that a three-point code type be defined as the three highest T scores, all of which surpass T = 60 and fall within five points of each other. When all subjects were considered with these criteria, the mean Clinical Scale code type was a 2-1-3 code type. According to Graham (2006), persons with this code type are often diagnosed as having depressive or anxiety disorders, and frequently express somatic complaints. In a general clinical setting, these somatic complaints may indicate somatoform disorder, but in a medical sample, it is likely that they represent more authentic complaints. The mean T scores for Scales 8 and 7 fell only slightly below those of Scales 2, 1, and 3, and are both above 60. In an epilepsy sample, small elevations on Scale 8 may be indicative of genuine neurologic symptoms (e.g., seizure auras) which mimic hallucinatory or otherwise aberrant experiences (Lewis, Lachar, Voelker, & Vidergar, 1984; Modrego, Pina, Galindo, & Minguez, 2002; Nelson et al., 2004). Ben-Porath (2008) has recommended against profile interpretation for RC Scales; configural issues such as code types have not been studied for the scale set, and empirical correlates for this type of interpretation are not available. In any case, only RC1 had a mean elevation above T = 60, the threshold for interpretability as part of a code type (Graham, 2006).

Exploratory Analysis I: Conversion V Profiles

In most settings, a conversion V profile is considered to be indicative of somatoform tendencies (Graham, 2006). Broadly, this means that test-takers who display a conversion V may lack psychological insight, prefer medical explanations for symptoms, or tend to express emotional distress through somatic symptoms. In medical settings, it is often difficult to determine whether conversion V patterns represent the

genuine expression of somatic symptoms or health concerns, or whether they represent an exaggeration of health-related symptoms.

The majority of the existing research concerning conversion V patterns in epilepsy patients has been focused on the differential identification of epileptic and psychogenic seizures (e.g., Vossler et al., 2004). Owczarek and Jedrzejczak (2001) compared the aggregate T scores on Clinical Scales 1, 2, and 3 across a group of epilepsy patients and a group of patients with comorbid epileptic and psychogenic seizures. Results of their study indicated that the epilepsy group had relatively low scores on Scales 1 and 3, while the comorbidity group displayed a conversion V pattern. In the current study, all subjects had diagnoses of epilepsy, and patients who had been diagnosed with psychogenic seizures were not considered for inclusion. In this sample, it seems that a conversion V could either denote the exaggeration of genuine symptoms or be an accurate representation of significant health concerns in the absence of emotional distress. Several analyses were carried out to find which of these cases was more likely.

Comparisons between mean RC and Clinical Scale profiles for subjects with and without a conversion V pattern are displayed in Figure 3. Of all the subsets created in the exploratory analyses of the current study, only the group of subjects with conversion V patterns looks substantially dissimilar from the others. The profile configurations of the entire sample, the floating profile subset, and the moderate depressive severity subset all look roughly the same, albeit with different levels of elevations across subjects. For these groups, the Clinical Scales show the aforementioned 2-1-3 code type, and slight elevations on Scales 7 and 8. When other subsets are considered, the RC Scales display small elevations on RCd and RC2, with a more substantial elevation on RC1, and another

slight elevation on RC8 (and sometimes on RC7). In the conversion V subset, the mean Clinical Scale profile shows the same slight elevations on Scales 8 and 7, but Scales 1 and 3 are elevated to near T = 75, and Scale 2 plunges between them to around T = 65. In fact, the mean Scale 2 score is roughly similar for the conversion V subset and the remainder of the sample. The mean RC Scale profile for the conversion V group shows an inverse V pattern, with RC1 spiking near T = 70, and RCd and RC2 in the subclinical range, falling even below the mean scores for the remainder of the sample. From these profiles, it is apparent that the conversion V group scored very differently on measures of health concerns and depression than the remainder of the sample, but it is unclear whether those scores represent exaggerations of true symptoms, or if the conversion V group might simply reduce to those subjects with very serious health concerns.

Regression analyses indicated that several RC Scales were significantly related to whether or not a subject would display a conversion V pattern. When individual RC Scales were considered, RC3 ("Cynicism") accounted for the most variance (8.9%). The combination of RC3 and RC1 accounted for 24.1% of the variance in whether a subject would display a conversion V—slightly more than the combination of RCd and RC1, which accounted for 21.3% of the variance. Regression coefficients associated with RC3 were consistently negative, indicating a potential relationship between naiveté (the inverse of the cynicism measured by RC3) and the tendency to display a conversion V pattern. The presence of naiveté may indicate an absence of psychological insight, a lack of coping skills or resources, or an inability to form good associations between cause and effect, all of which could potentially contribute to conversion or somatization tendencies.

Given that depressive symptoms and health concerns seem to be so related in this sample, one might expect that a very high level of health concerns would be accompanied by a high level of depressive symptoms. Obviously, this was not the case for the conversion V subset. Examination of mean RC and Clinical Scale profiles indicated that members of this subset scored approximately the same as non-members on Scale 2, and lower than non-members on RCd and RC2. In all other groups considered in this study, scores on Scales 1 and 2, and on RCd, RC2, and RC1 were very similar. The absence of equivalence between somatic and depressive symptom scales is unique to this subset, and likely indicative of exaggeration of or preoccupation with somatic symptoms.

In an epilepsy population, it appears that using Clinical or RC Scales to differentiate between conversion and genuine health complaints is not an easy task. With both scale sets, a clinician may look for a failure to display the expected relationship between measures of health concerns and depression: it seems that as health concerns increase, so too should depressive symptoms. The predictive power of RC3 is a potential benefit of analysis via the RC Scales. If conversion is suspected, and the RC profile does not show the expected relationship between RCd and RC1, a low score on RC3, indicative of naiveté, would likely reinforce an interpretation of somatoform tendencies.

Exploratory Analysis II: Floating Profiles

In clinical settings, floating profiles are often interpreted as an indication of either characterological impairment or extreme distress. In the creation of the RC Scales, Tellegen and colleagues (2003) extracted a demoralization or distress component from each of the Clinical Scales and created a unique and discreet measure of demoralization. This process sought to simplify interpretation by lessening the chances that many scales

would elevate due to extreme distress, and thereby confound interpretation (Wallace & Liljequist, 2005). Finn and Kamphuis (2006) presented a model case study of a client with a floating profile. Examination of this client's RC Scale profile indicated an elevation on RCd, and decreased scores on all other scales. The second exploratory analysis of the current study sought to find if, in an epilepsy sample, floating profiles would be attributable to the demoralization factor that is measured by RCd.

A subset of 38 subjects were identified as having five or more Clinical Scales (not including Scales 5 and 0) with T scores at or above 65. In this subset, all Clinical Scales except for Scale 9 had mean T scores above 65. Four RC Scales had mean T scores above 65: RCd, RC1, RC2, and RC8. Besides Scale 1 and RC1, which had comparable T scores, all RC Scales were lower than the corresponding Clinical Scales. When the current study's *entire* sample is considered, no RC Scales had mean values above T = 65. Given that four RC Scales from the floating profile subset did achieve T scores at or above 65, the RC Scales may still be somewhat vulnerable to inflation in this sample, providing that test-takers respond in a style that produces a floating Clinical Scale profile. On the other hand, the RC Scales with mean elevations at or above 65 *are* those which reflect qualities that might be expected in epilepsy patients with many symptoms. These RC Scale elevations may indicate that subjects who produced floating profiles were those with authentic elevations in depressive symptoms, health concerns, and aberrant experiences (often a reflection of seizure occurrences; see Modrego et al., 2002). In other words, whereas floating profiles may reduce to a single elevation on RCd in general clinical samples, they may reduce to elevations on RCd, RC1, RC2, or RC8 in an epilepsy sample.

Regression analyses confirmed that these symptoms were best able to account for the variance in whether or not a subject displayed a floating profile. Consistent with theory, RCd was the best single-scale predictor, accounting for 39.0% of the variance. The best two-scale predictors were the combination of RCd and RC2 (accounting for 42.2% of the variance) and a model combining RCd and RC1 (accounting for 41.3% of the variance). A few three-scale models were slightly more powerful: a model using RCd, RC2, and RC7 accounted for 42.4% of the variance, and a model using RCd, RC1, and RC2 accounted for 44.2% of the variance. Further research that incorporates measures of seizure severity (such as data regarding the frequency and duration of seizures) is likely needed to better understand which factors contribute to floating profiles in epilepsy patients.

Looking at the Clinical Scale profile of a subject in this subset, a clinician would likely come to very different interpretive conclusions—even knowing that floating profiles are often indicative of exaggerated symptoms—than if he or she looked only at the RC Scale profile. As Figure 4 displays, of the seven Clinical Scales with mean T scores above 65, five approach or exceed T = 75. By contrast, of the four RC scales with T scores above 65, only RC1 is elevated above 70. Though the utilization of RC Scales in the interpretation of MMPI-2 profiles will likely decrease over-interpretation of the Clinical Scales, these results seem to indicate a danger in under-interpreting RC Scales. Insofar as it indicates characterological impairment or great distress, a floating profile has some interpretive value. Using only the RC Scales, a clinician may not get a sense of that impairment or distress, especially since the mean value for RCd in this subset was only 68.58 (SD = 11.09). At a minimum, this data seems to signify that even mild elevations

above T = 65 in RC Scales should be interpreted as indicative of significant psychopathology.

Exploratory Analysis III: Depression of Moderate Severity

The third exploratory analysis sought to examine the relationship between a separate measure of depression and RC Scales in this sample by creating a subset of subjects (n = 57) who scored at or above the threshold for moderate depression on the IDS-SR (total score ≥ 26).

While the mean T score for Scale 2 was higher than the mean T score for RCd, the differences between mean scores for subjects within and outside the subset were approximately equal for both scales (16.38 T score points on Scale 2 versus 16.86 T score points on RCd). Therefore, Scale 2 and RCd seem to differentiate between the subjects with and without significant depression in a similar way, though Scale 2 was higher than RCd within both groups. Furthermore, mean RCd scores in the depressed group were only slightly elevated (T = 65.95; SD = 10.34), again illustrating the importance of taking even mild elevations very seriously for RC Scores.

The fourth hypothesis of the current study demonstrated the significant relationship between depression and health concerns in this sample. Of the RC Scales, RC1 had the highest mean value in the moderate depression subset (T = 74.72; SD = 11.03). Additionally, regression analyses identified RCd and RC1 as the two best individual predictors among the RC Scales of variance in whether or not a subject was moderately depressed on the IDS-SR. RCd was able to account for 45.8% of the variance, while RC1 accounted for 44.6% of the variance. As a combined model, RCd

and RC1 were able to account for 57.9% of the variance in whether or not a subject had moderate depressive severity.

Comparison of mean RC Scale profiles for subjects within and outside the subset indicated that subjects with at least moderate self-reported depressive severity scored significantly higher on all nine RC Scales than subjects in the remainder of the sample. This was not expected, given that the RC Scales were constructed to increase discriminant validity and decrease covariation. Assuming that no other symptoms co-occur or co-vary with depression in this sample, only RC Scales related to depressive symptoms should differ between subjects within and outside this subset. These unexpected differences between all nine RC Scales may be indicative of a few conditions. First, the RC Scales may not be as generally free of covariation as had previously been assumed. Results of the second hypothesis generally indicated good discriminant validity between the RC Scales, though RC2 and RC7 were more highly correlated with RCd than expected. Therefore, it seems more likely that in this sample, a mediator variable may be responsible for causing covariation among the RC Scales. Throughout the current study, the apparent close relationship between health concerns and psychiatric symptoms has repeatedly been demonstrated. In the moderate depressive severity subset, RC1 demonstrated the highest mean T score of all the RC Scales (T = 74.72 [SD = 11.03]). It is likely that the covariation of RC Scales in the subset could be another example of how increased health concerns may be associated with generally higher levels of self-reported psychiatric symptoms. In any case, these results indicate that even if depression is identified as a primary problem in the psychological testing process, more than just a depression screening may be required, as symptoms appear to commonly co-vary.

Limitations

The current study may have several limitations. First, a larger subset of subjects for the evaluation of internal consistency would have been ideal. As discussed previously, the addition of data for analysis of Cronbach's alpha tends to increase the value of the coefficient. Since the mean alpha value for the Clinical Scales was slightly below the optimal range, increasing the number of subjects would likely have improved the mean alpha coefficient to the optimal range.

As a byproduct of the manner in which data was collected for this study (i.e., retrospective analysis of an existing database), only K-corrected scores were available for the Clinical Scales. In the original publication of the RC Scales, and in many subsequent studies, non-K-corrected T scores have been used (Ben-Porath, 2008; Tellegen et al., 2003). Non-K-corrected T scores would have been more appropriate for many comparisons made in the current study, as the RC Scales contain no K-correction factor. The K-correction factor was intended to correct for defensive test-taking by adding a portion of the K scale raw score to the raw score of several Clinical Scales. However, research has indicated that the K-correction may not increase validity, and in some cases, may even attenuate validity (see Sellbom et al., 2006). Because of these findings, a K-correction is not included in the RC Scales. Therefore, the findings of the current study are not directly comparable to those of previous research. Nevertheless, the current study yielded findings that are similar to those of previous research.

Additionally, because the current study was a retrospective analysis of extant data, limited types of data were available. For instance, though IDS-SR total scores were available, item-by-item responses were not. Had item-by-item responses been available

for analysis, the relationship between RCd and the presence of atypical depressive symptoms could have been more thoroughly explored. The IDS-SR was the only available extra-test measure of psychopathologic symptoms. An extra-test measure of anxiety would have beneficial insofar as the convergent validity of RC7 could have been examined in this sample. Data from a measure such as the WPSI or health-related QOL ratings would also have been helpful to clarify the relationship between health complaints and psychiatric comorbidity as measured by the RC Scales.

As a measure of depressive severity, the IDS-SR was not an optimal criterion measure of anhedonic major depression. In the current study, the IDS-SR appears to have acted well to identify subjective dysphoric mood and atypical depression, and validated the use of RCd to screen for depression in an epilepsy sample. However, had an additional extra-test measure of strictly anhedonic symptoms been available, the convergent validity of RC2 could have been more thoroughly examined. Without further inspection of how RC2 functions in this sample, its potential for use as a screening tool in an epilepsy population remains largely undefined.

Finally, elevated intercorrelations among RCd, RC1, RC2, and RC7 in this sample hamper interpretation of co-occurring elevations on those scales. As has been previously suggested (Simms et al., 2005), further research is need to determine whether these high intercorrelations (which belie the theoretical grounds for creating the RC Scales) are a byproduct of construct overlap, or an indication of genuine comorbidity in certain samples. However, it should not be entirely surprising that these scales seem to co-vary. To perfectly measure discriminant validity among these scales, one would have to completely control for the comorbidity of psychiatric symptoms. This task would be

very difficult, given that up to 73% of epilepsy patients may experience co-occurring depression and anxiety (Jones et al., 2005a). Furthermore, the genuine relationship between bodily symptoms and mood and anxiety disorders has been well-documented (Maxmen & Ward, 2006). Therefore, it makes sense that RCd, RC1, RC2, and RC7 should co-vary to a degree in this sample.

Conclusions

The primary purposes of the current study were to examine the psychometric properties of the RC Scales in an epilepsy sample, and to determine whether certain Clinical Scale patterns had interpretive implications for the RC Scales in this sample. These goals were pursued with the intent of providing information regarding the refinement of psychological assessment procedures in epilepsy, and to add to the growing body of literature regarding use of the RC Scales in applied settings.

In this sample, the RC Scales seem to represent a modest improvement in general psychometrics over the Clinical Scales. The alpha coefficient of internal consistency was approximately equivalent for both sets of scales, and the RC Scales demonstrated slightly better item-total correlations. The RC Scales additionally showed slightly lower inter-scale discriminant validity, as evidenced by lowered intercorrelations among the scales. Though RC2 was less-related to a scale of somatic complaints than Scale 2, it is unclear how useful RC2 is as a predictor of depression in epilepsy patients, given that much of the depressive symptoms in epilepsy patients may present atypically (Kanner & Palac, 2000). RCd, designed to capture the type of dysphoric mood that may accompany atypical depression, seems to be the best predictor of depression in this sample, and have the highest convergence with an extra-test measure of depressive severity. It was

expected that as unifactorial, homogeneous measures, the RC Scales would be relatively free of the associations that have been found between Clinical Scales 2 and 7 and somatic complaints. This was the case for RC2, but RCd (the best predictor of depressive severity in this sample) and RC7 displayed correlations with Hy_4 ("Somatic Complaints") that were statistically similar to those of their Clinical Scale counterparts. These correlations, along with unexpectedly high intercorrelations between RCd, RC2, and RC7, raise questions regarding the distinctiveness of these scales in this sample. Results of the current study indicate that these relationships seem to either represent some construct overlap or high comorbidity of self-reported symptoms in this sample. However, Simms and colleagues (2005, p. 350) found high intercorrelations among RCd, RC2, and RC7 in two non-medical samples, leading them to conclude that these scales "tap highly overlapping constructs." It seems likely, though, that some of the co-variation of RCd, RC1, RC2, and RC7 may reflect a genuine comorbidity of symptoms.

Current results provide some interpretive implications for use of the RC Scales with epilepsy patients. First, as mentioned above, RCd performed as the best predictor of depressive severity in this sample. Therefore, in an epilepsy sample, when atypical depression or general depressed mood is suspected, RCd is likely to be the most useful indicator of depressive severity. Though its convergent validity was not substantiated in this study, RC2 displayed good discriminant validity from somatic complaints in this sample, and may therefore be a more ideal measure in instances where anhedonic depression is suspected. As originally indicated by Simms and colleagues (2005), the high correlation between RCd and RC7 has several interpretive implications. If both RCd and RC7 are elevated, and some form of anxiety is otherwise suspected, an anxiety

disorder involving nonspecific distress (e.g., GAD) may be responsible. If RC7 is elevated in the absence of an elevation on RCd, then more specific types of anxiety (e.g., phobias) might be indicated.

Results of the conversion V analyses indicated that if RC1 is highly elevated in the absence of an elevation on RCd, somatoform tendencies may be suspected, especially if the score on RC3 is particularly low. Future research may be directed toward developing a formula for the comparison of RCd, RC1, and RC3 that accurately predicts naive exaggeration of somatic symptoms. The primary interpretive contribution of the floating profile analysis was the notion that even modest RC Scale elevations should be taken very seriously in the interpretive process. For subjects with floating Clinical Scale profiles, the mean T scores on RC Scales were substantially lower, even in the absence of a marked elevation on RCd. In the floating profile group, RCd, RC1, RC2, and RC8 were all modestly elevated. In an epilepsy population, concurrent elevations on these four RC Scales may be indicative of the same types of interpretations normally associated with floating profiles (e.g., characterological impairment, psychological distress). Overall, it seems that in this sample, the RC Scales are *capable* of identifying some of the constructs that lead to well-known Clinical Scale configurations, though detection of these constructs may not be as obvious if only RC Scales are used.

APPENDIX A

The Inventory of Depressive Symptomatology (IDS)

The IDS, first published in 1986 as a 28-item inventory, and revised in 1996 as a 30-item inventory, is a free, non-copyrighted measure of depressive symptom severity (Rush et al., 1986, 1996). The IDS also comes in a 16-item version, the Quick Inventory of Depressive Symptomatology (QIDS; Rush et al., 2003). Both the IDS and the QIDS are available in a clinician-rated (IDS-C and QIDS-C) and self-report (IDS-SR and QIDS-SR) version.

The most commonly used clinician-rated measure of depression is the Hamilton Rating Scale for Depression (HRSD) and the most common self-report measure is the Beck Depression Inventory (BDI; Rush et al., 1996). The rationale for developing the IDS, and later the QIDS, rested on evaluation of these and other existing measures. The IDS and QIDS were designed to improve upon existing measures by (1) providing matched patient and clinician ratings; (2) providing equivalent weightings for each item; and (3) providing coverage of all nine DSM-IV criterion symptoms for a MDE (Rush et al., 1996).

In an analysis of its psychometric properties by Trivedi and colleagues (2004), the IDS displayed high internal consistency (alpha = .90 for IDS-C, alpha = .92 for IDS-SR). In a factor analysis study, Gullion and Rush (1998) compiled items from the IDS, BDI, and HRSD, and identified ten factors: hedonic capacity, self-blame, suicide/hopelessness, lack of energy, sleep disturbance, decreased libido, somatic anxiety, anxious/irritable, sleep onset insomnia, and appetite disturbance/weight change. The IDS had more complete factor coverage than either the HRSD or the BDI. Along those lines,

a variety of researchers have concluded that the IDS is of equal or higher value in determining a classification of depression than the HRSD, BDI, Patient Global Impression Improvement (PGI-I) questionnaire, Physician Global Rating Scale (PhGRS), Patient Global Rating Scale (PaGRS), Composite International Diagnostic Interview (CIDI), and Montgomery and Asberg Depression Rating Scale (MADRS; Biggs et al., 2000; Corruble et al., 1999a; Corruble et al., 1999b; Gullion & Rush, 1998; Kessler et al., 2003; Rush et al., 2005).

The QIDS was constructed by selecting from the IDS only those items that reflected DSM-IV criterion symptoms for major depression (Rush et al., 2003). Rush and colleagues (2003) assessed the internal consistency of the QIDS-SR and its concurrent validity with other measures: the IDS-SR, PGI-I, and HRSD in outpatients with chronic, nonpsychotic MDD. The study found highly acceptable psychometric properties and high internal consistency. Concurrent validity between the 17-item version of the HRSD and the QIDS-SR and IDS-SR was high (r = .81 and r = .84, respectively); relatively simple conversions for these scores were suggested ($HRSD_{17}$ total score × 2.0 = IDS-SR total score, $HRSD_{17}$ total score × 0.8 = QIDS-SR total score, and QIDS-SR total score × 2.5 = IDS-SR total score). Rush and colleagues (2003) also studied the IDS-SR, QIDS-SR, and the HRSD with respect to sensitivity to change over time in several treatment outcome groups (classified via PGI-I). Results indicated that the $HRSD_{24}$, IDS-SR, and QIDS-SR had similar effect sizes (e.g., -80.5 ± 15.8, -77.8 ± 17.8, -75.4 ± 21.4, respectively, for "Very Much Improved" group). In addition, the IDS-SR and QIDS-SR were as sensitive to change over time as the $HRSD_{24}$; changes in IDS-SR and QIDS-SR scores at each measurement point closely paralleled changes in $HRSD_{24}$ scores.

Experimentally, versions of the IDS have been used in a variety of other settings. The Sequenced Treatment Alternatives to Relieve Depression (STAR*D) trial is a large multisite, randomized, prospective, clinical trial of outpatients with nonpsychotic MDD (Rush et al., 2004). Versions of the IDS are an integral part of the STAR*D intake procedure and treatment paradigm. Clinical research coordinators complete the IDS-C and QIDS-C at baseline in order to make decisions about study inclusion and exclusion (Perlis et al., 2005). Patients complete the QIDS-SR at baseline, while the QIDS-C is used to track symptomatology and make decisions about whether or not patients should proceed to more advanced levels of treatment (Rush et al., 2004; Yates et al., 2004). The IDS-SR has also been used as an outcome measure, gauging degree of treatment remission (Rush et al., 2004). Several studies using data from STAR*D have been published. Versions of the IDS have been used to examine the prevalence of significant levels of irritability (Perlis et al., 2005), to compare characteristics of outpatients from primary and specialty care settings (Gaynes et al., 2005), and to examine the relationship between age of onset and severity of MDD (Zisook et al., 2004).

The Texas Medication Algorithm Project, a large trial that uses versions of the IDS, is an evaluation of algorithm-based treatment procedures for the self-declared persistently mentally ill in the public mental health sector (Trivedi et al., 2004). This project, using both the IDS-C and the IDS-SR as treatment outcomes, has found that algorithm-guided interventions show significantly greater improvements in MDD symptom reduction than a treatment-as-usual group following a year of treatment.

Several studies have used versions of the IDS as outcome indicators for the validation of antidepressant medication. The IDS has been used to show improvement in

anxiety, depression, and anhedonia with the SSRI sertraline (Boyer et al., 2000); to document response to the atypical antidepressant nefazodone (Ninan et al., 2002; Trivedi et al., 2001); to evaluate the anticonvulsant tiagabine as a treatment for bipolar disorder (Suppes et al., 2002); to monitor rate of switch in patients with bipolar disorder treated via second-generation antidepressants (Post et al., 2001); and to measure the effects of the SSRI fluoxetine for treatment of minor depression (Judd et al., 2004). Research such as this suggests that self-report measures like the IDS-SR and QIDS-SR could be cost- and time-efficient substitutes to clinician rating scales.

The IDS has also been used to study depression in a variety of applied settings. Yonkers and colleagues (2001) used the IDS to track onset and persistence of postpartum depression in an inner-city health clinic system. Rapaport and colleagues (2002) used the IDS-C to track symptomatology in people with minor depression in order to provide a descriptive analysis of the syndrome. Jenkins, Carmody, and Rush (1998) used the IDS-SR to examine depressive symptoms among radiation oncology patients to find risk factors associated with depression in cancer patients. Denicoff and colleagues (2000) used the IDS-C as a benchmark to aid the validation of the National Institute of Mental Health prospective Life Chart Methodology for longitudinal assessment of bipolar disorder. Yates and colleagues (2004) used the IDS-C to examine clinical features of outpatients with and without comorbid general medical conditions.

In summary, the IDS has displayed its utility among a variety of populations, for a variety of purposes. It has been used to define inclusion and exclusion criteria and to track progress in large trials, to show treatment outcome in trials of antidepressants, and to highlight characteristics of depression in certain populations. It has been used

successfully in studies of patients with MDD, minor depression disorder, bipolar disorder, anxiety, general medical conditions, and cancer.

APPENDIX B

The MMPI-2 Restructured Format (MMPI-2-RF)

Due to be published in July 2008, the MMPI-2-RF is a new, 338-item version of the MMPI-2 (Ben-Porath, 2008). The MMPI-2-RF will be published as an alternative to the MMPI-2, not a replacement; the MMPI-2 will remain available and fully supported. Since the 338 items of the MMPI-2-RF are drawn from the MMPI-2 item pool, MMPI-2-RF profiles will be able to be generated from existing MMPI-2 data.

The objective of MMPI-2-RF development was to represent the good clinical substance of the MMPI-2 item pool with measures that are more psychometrically adequate. Ideally, the built-in MMPI-2-RF scales will eliminate the need for additional content scales (e.g., Harris-Lingoes Scales, Content Scales) and code type interpretation. The MMPI-2-RF scales also have improved construct validity, as they were developed in a process similar to the RC Scales (i.e., using factor analysis and eliminating item overlap).

Development of the RC Scales yielded multiple factors within each Clinical Scale. Besides Dem and the core component for each RC Scale, other factors sometimes emerged (e.g., a shyness component in Scale 3). This necessitated the eventual development of scales measuring these components. Additional scales were also needed to measure clinically significant attributes not measured by the RC Scales (e.g., suicidal ideation).

The MMPI-2-RF is composed of 50 scales, divided into 3 tiers. In the first tier, 3 Higher-Order Scales (i.e., Emotional/Internalizing Dysfunction, Thought Dysfunction, Behavioral/Externalizing Dysfunction) measure dimensional psychopathology similar to

that previously measured by the 27/72, 68/86, and 19/94 Clinical Scale code types. In the second tier, the 9 MMPI-2-RF RC Scales are identical to the MMPI-2 RC Scales. In the third tier, 23 Specific Problems Scales measure somatic/cognitive, internalizing, externalizing, and interpersonal problems. Additionally, 2 Interest Scales measure aesthetic-literary and mechanical-physical interests, and the 5 PSY-5-r Scales are analogs of the MMPI-2 PSY-5 Scales used to measure Axis II psychopathology.

The MMPI-2-RF will be published with 3 documents: a *Manual for Administration, Scoring, and Interpretation*, a *Technical Manual* (which will contain extratest correlates for each scale, score conversions, and MMPI-2/ MMPI-2-RF comparability), and a *User's Guide for Reports*.

APPENDIX C**:** Tables and Figures

Table 1

MMPI/MMPI-2 Clinical Scale Numbers and Corresponding Original Clinical Scale Names

Present Scale Number	Original Scale Name
1	Hypochondriasis (Hs)
2	Depression (D)
3	Hysteria (Hy)
4	Psychopathic Deviation (Pd)
5	Masculinity-Femininity (Mf)
6	Paranoia (Pa)
7	Psychasthenia (Pt)
8	Schizophrenia (Sc)
9	Hypomania (Ma)
0	Social Introversion (Si)

Note. Adapted from Graham, J. (2006). *MMPI-2: Assessing personality and psychopathology* (4th ed.). New York: Oxford University Press.

Table 2

MMPI-2 Clinical Scales and Corresponding Restructured Clinical (RC) Scales

Clinical Scales	Restructured Clinical (RC) Scales
-----[a]	RCd – Demoralization (dem)
1 - Hypochondriasis (Hs)	RC1 – Somatic Complaints (som)
2 - Depression (D)	RC2 – Low Positive Emotions (lpe)
3 - Hysteria (Hy)	RC3 – Cynicism (cyn)
4 - Psychopathic Deviation (Pd)	RC4 – Antisocial Behavior (asb)
6 - Paranoia (Pa)	RC6 – Ideas of Persecution (per)
7 - Psychasthenia (Pt)	RC7 – Dysfunctional Negative Emotions (dne)
8 - Schizophrenia (Sc)	RC8 – Aberrant Experiences (abx)
9 - Hypomania (Ma)	RC9 – Hypomanic Activation (hpm)

Note. Clinical Scales 5 (Masculinity-Femininity) and 0 (Social Introversion) have been omitted here for several reasons: (1) their creation occurred somewhat after that of the other Clinical Scales; (2) they do not focus on psychopathology per se; (3) they have no corresponding RC Scales; and (4) they are not examined in the current study.

[a]No corresponding scale.

Table 3

Demographic Information for Sample of Patients with Epilepsy Used in Current Study

Group	N	Percent	Group	N	Percent
Gender			Age[a]		
Male	60	43.8	18 – 29	32	23.4
Female	77	56.2	30 – 39	43	31.4
Race			40 – 49	45	32.8
Caucasian	116	84.7	50 – 59	12	8.8
African American	6	4.4	60 – 67	5	3.6
Hispanic	11	8.0	Seizure Type		
Asian	1	0.7	TLE[b]	61	44.5
Native American	1	0.7	CPS[c]	22	16.1
Eastern Indian	1	0.7	GTC[d]	4	2.9
Other	1	0.7	Other	15	10.9
			Unknown	35	25.5

Note. Total *n* for each group = 137.

[a]Age of subjects ranges from 18 – 67, mean age = 37.82. [b]TLE = temporal lobe epilepsy. [c]CPS = complex partial seizures.

[d]GTC = generalized tonic-clonic seizures.

Table 4

Descriptive Statistics for RC and Clinical Scales in Sample of Patients with Epilepsy

RC Scales					Clinical Scales				
Scale	Mean (SD[a])	Median	Range[b]	% > 65[c]	Scale	Mean (SD[a])	Median	Range[b]	% > 65[c]
RCd	56.18 (12.35)	54	36 – 86	24.1					
RC1	64.21 (13.54)	64	37 – 100	48.2	1	65.03 (13.11)	64	35 – 99	48.9
RC2	57.44 (12.43)	56	34 – 93	21.9	2	65.77 (13.57)	64	40 – 96	48.9
RC3	54.63 (11.42)	50	34 – 80	19.0	3	64.50 (15.09)	65	35 – 113	51.1
RC4	49.28 (9.94)	48	33 – 77	8.8	4	55.92 (12.40)	53	34 – 100	21.2
RC6	54.42 (11.56)	56	41 – 82	19.7	6	55.80 (13.30)	53	32 – 97	20.4
RC7	52.01 (11.17)	52	32 – 82	14.6	7	60.64 (13.16)	59	35 – 102	32.1
RC8	59.56 (10.92)	59	38 – 85	29.9	8	62.62 (13.06)	60	37 – 101	40.9
RC9	48.32 (9.15)	48	33 – 85	5.1	9	56.21 (10.61)	56	35 – 88	21.9

Note. $N = 137$.

[a]SD = standard deviation. [b]Range values displayed as follows: lowest T score – highest T score. [c]Percent of subjects with T score on scale above T = 65.

Table 5

Internal Consistency of RC and Clinical Scales as Indicated by Internal Consistency Coefficients (Cronbach's Alpha) and Item-Total Correlations

	RC Scales				Clinical Scales		
Scale	*N* of Items	Cronbach's Alpha	Mean Item-Total Correlation	Scale	*N* of Items	Cronbach's Alpha	Mean Item-Total Correlation
RCd	20	.896	.486				
RC1	27	.818	.349	1	32	.862	.939
RC2	17	.624	.239	2	57	.649	.146
RC3	15	.815	.424	3	60	.655	.142
RC4	22	.709	.246	4	50	.659	.169
RC6	17	.327	.065	6	40	.429	.079
RC7	24	.826	.372	7	48	.912	.389
RC8	18	.545	.188	8	78	.858	.228
RC9	28	.810	.434	9	46	.432	.083
Mean Value	21[a]	.708	.311		51[a]	.682	.272

Note. Item-total correlations represent Pearson product-moment correlations (*r*s) between scale items and total score on scale.

[a]Approximate value.

Table 6

Intercorrelations of MMPI-2 RC Scales

	Scale								
Scale	RCd	RC1	RC2	RC3	RC4	RC6	RC7	RC8	RC9
RCd	-----	.562	.672	.475	.455	.385	.796	.553	.408
RC1		-----	.406	.331	.302	.315	.514	.375	.264
RC2			-----	.287	.369	.228	.491	.235	.027*
RC3				-----	.278	.545	.533	.440	.355
RC4					-----	.295	.437	.314	.359
RC6						-----	.404	.394	.405
RC7							-----	.583	.475
RC8								-----	.363
RC9									-----

Note. All correlations significant at $p < .01$, unless otherwise noted.

*Non-significant.

Table 7

Intercorrelations of MMPI-2 Clinical Scales

	Scale							
Scale	1	2	3	4	6	7	8	9
1	-----	.592	.788	.289	.273	.514	.566	.129**
2		-----	.639	.504	.483	.750	.694	-.092**
3			-----	.473	.333	.599	.609	.054**
4				-----	.547	.585	.657	.198*
6					-----	.576	.618	.222
7						-----	.810	.127**
8							-----	.306
9								-----

Note. All correlations significant at $p < .01$, unless otherwise noted.

*$p < .05$

**Non-significant.

Table 8

Correlations between MMPI-2 RC and Clinical Scales and IDS-SR Total Score

	Measure			
Measure	IDS-SR	Clinical Scale 2	RC2	RCd
IDS-SR	-----	.672	.538	.742
Clinical Scale 2		-----	.665	.734
RC2			-----	.672
RCd				-----

Note. All correlations significant at $p < .01$.

Table 9

Amount of Variance Accounted for by Selected MMPI-2 Scales When Predicting IDS-SR Total Score

Model	R^{2a}	F^{b}
RCd/RC2	.553	82.90
Clinical Scale 2	.451	110.94
RCd	.550	165.12
RC2	.289	54.98
RCd/Clinical Scale 2	.585	94.51

Note. All F-ratios are significant at $p < .001$.

[a]R^2 statistics were computed via regression with the scale(s) in each model as the predictor variable(s) and IDS-SR total score as the dependent variable. The R^2 statistic is interpretable as follows: $R^2 \times 100$ = % of variance in dependent variable that can be accounted for by predictor variable(s). [b] The F-ratio evaluates the significance of each R^2 statistic.

Table 10

Regression Coefficients for Selected MMPI-2 Scales When Predicting IDS-SR Total Score

Model	Predictor Variables	Regression Coefficient[a]	t[b]
RCd/RC2	RCd	.710	8.890
	RC2	.073	0.925*
Clinical Scale 2	Clinical Scale 2	.626	10.533
RCd	RCd	.759	12.850
RC2	RC2	.547	7.415
RCd/Clinical Scale 2	RCd	.552	6.581
	Clinical Scale 2	.257	3.362**

Note. All t-ratios are significant at $p < .001$, unless otherwise noted.

[a]Regression coefficients were computed with the scale(s) in each model as the predictor variable(s) and IDS-SR total score as the dependent variable. The regression coefficient is a measure of the linear relationship between the predictor variable and the dependent variable, given that the influences of other predictors are held constant. [b]The t-ratio evaluates the significance of each regression coefficient.

*$p = .356$

**$p = .001$

Table 11

Correlations between MMPI-2 Clinical Scale 2, RC2, and Harris-Lingoes Scale Hy_4

	Measure		
Measure	Clinical Scale 2	RC2	Hy_4
Clinical Scale 2	-----	.665	.550
RC2		-----	.363
Hy_4			-----

Note. All correlations significant at $p < .01$.

Table 12

Correlations between MMPI-2 Clinical Scale 7, RC7, and Harris-Lingoes Scale Hy_4

	Measure		
Measure	Clinical Scale 7	RC7	Hy_4
Clinical Scale 7	-----	.635	.521
RC7		-----	.498
Hy_4			-----

Note. All correlations significant at $p < .01$.

Table 13

Descriptive Statistics for RC and Clinical Scales in a Subset of Subjects Whose Clinical Scale Profiles Displayed a Conversion V Pattern

RC Scales				Clinical Scales			
Scale	Mean (SD[a])	Median	Range[b]	Scale	Mean (SD[a])	Median	Range[b]
RCd	52.83 (8.35)	53	36 – 69				
RC1	70.40 (11.78)	68	52 - 100	1	74.74 (10.28)	74	59 – 99
RC2	55.71 (10.98)	53	43 – 93	2	65.20 (10.43)	64	49 – 96
RC3	48.83 (8.03)	48	34 – 68	3	75.60 (9.86)	74	61 – 108
RC4	48.23 (10.19)	47	33 – 66	4	56.29 (10.70)	55	37 – 84
RC6	49.83 (10.17)	43	41 – 73	6	54.51 (11.88)	52	32 – 81
RC7	48.34 (9.10)	47	34 – 74	7	60.71 (9.23)	57	47 – 83
RC8	58.71 (9.56)	56	38 – 80	8	64.91 (9.40)	65	50 – 89
RC9	46.49 (7.11)	49	33 – 61	9	57.89 (8.33)	56	37 – 79

Note. N = 35.

[a]SD = standard deviation. [b]Range values displayed as follows: lowest T score – highest T score.

Table 14

Amount of Variance Accounted for by Selected MMPI-2 Scales When Predicting Inclusion in a Subset of Subjects with Conversion V Clinical Scale Patterns

Model	R^{2a}	F^{b}
RCd	.025	3.52*
RC1	.072	10.51
RC3	.089	13.21
RCd/RC1	.213	18.12
RC3/RC1	.241	21.27

Note. All F-ratios are significant at $p \leq .001$, unless otherwise noted.

[a]R^2 statistics were computed via regression with the scale(s) in each model as the predictor variable(s) and inclusion in the conversion V subset as the dependent variable. The R^2 statistic is interpretable as follows: $R^2 \times 100$ = % of variance in dependent variable that can be accounted for by predictor variable(s). [b] The F-ratio evaluates the significance of each R^2 statistic.

*$p = .06$

Table 15

Regression Coefficients for Selected MMPI-2 Scales When Predicting Inclusion in a Subset of Subjects with Conversion V Clinical Scale Patterns

Model	Predictor Variables	Regression Coefficient[a]	t[b]
RCd	RCd	-.006	-1.88*
RC1	RC1	.009	3.24
RC3	RC3	-.011	-3.63
RCd/RC1	RCd	-.016	-4.89
	RC1	.017	5.65
RC3/RC1	RC3	-.017	-5.46
	RC1	.013	5.18

Note. All t-ratios are significant at $p < .001$, unless otherwise noted.

[a]Regression coefficients were computed with the scale(s) in each model as the predictor variable(s) and inclusion in the conversion V subset as the dependent variable. The regression coefficient is a measure of the linear relationship between the predictor variable and the dependent variable, given that the influences of other predictors are held constant. [b]The t-ratio evaluates the significance of each regression coefficient.

*$p = .06$

Table 16

Descriptive Statistics for RC and Clinical Scales in a Subset of Subjects Whose Clinical Scale Profiles Displayed a Floating Profile Pattern

RC Scales				Clinical Scales			
Scale	Mean (SD[a])	Median	Range[b]	Scale	Mean (SD[a])	Median	Range[b]
RCd	68.58 (11.09)	54	42 – 86				
RC1	74.55 (13.16)	64	43 – 100	1	74.63 (11.48)	64	51 – 99
RC2	68.47 (11.90)	56	34 – 93	2	79.58 (9.81)	64	57 – 96
RC3	60.45 (11.36)	50	43 – 80	3	77.45 (14.08)	65	47 – 113
RC4	55.61 (8.93)	48	35 – 74	4	67.68 (13.73)	53	44 – 100
RC6	59.95 (11.40)	56	41 – 82	6	67.68 (12.85)	53	37 – 97
RC7	61.24 (11.52)	52	38 – 82	7	75.68 (10.83)	59	47 – 102
RC8	67.26 (11.36)	59	47 – 85	8	77.45 (11.15)	60	55 – 101
RC9	52.45 (11.23)	48	38 – 85	9	58.71 (10.19)	56	37 – 82

Note. $N = 38$.

[a]SD = standard deviation. [b]Range values displayed as follows: lowest T score – highest T score.

Table 17

Results of Independent Samples t-Test of RC and Clinical Scales between Subjects within Floating Profile Subset and outside Floating Profile Subset

	RC Scales				Clinical Scales		
Scale	Floating[a]	Mean (SD[b])	t	Scale	Floating[a]	Mean (SD[b])	t
RCd	Yes	68.58 (11.09)	8.51				
	No	51.41 (9.09)					
RC1	Yes	74.55 (13.16)	5.90	1	Yes	74.63 (11.48)	6.02
	No	60.24 (11.47)			No	61.34 (11.81)	
RC2	Yes	68.47 (11.90)	7.05	2	Yes	79.58 (9.81)	9.91
	No	53.20 (9.78)			No	60.47 (10.81)	
RC3	Yes	60.45 (11.36)	3.78	3	Yes	77.45 (14.08)	6.90
	No	52.39 (10.69)			No	59.54 (12.30)	
RC4	Yes	55.61 (8.93)	5.08	4	Yes	67.68 (12.73)	6.85
	No	46.86 (9.25)			No	51.40 (8.25)	
RC6	Yes	59.95 (11.40)	3.56	6	Yes	67.68 (12.85)	7.06
	No	52.29 (10.96)			No	51.23 (10.35)	
RC7	Yes	61.24 (11.52)	6.18	7	Yes	75.68 (10.83)	10.63
	No	48.46 (8.79)			No	54.86 (8.64)	
RC8	Yes	67.26 (11.36)	5.17	8	Yes	77.45 (11.15)	10.26
	No	56.61 (9.22)			No	56.93 (8.48)	
RC9	Yes	52.45 (11.23)	2.88*	9	Yes	58.71 (10.19)	1.76**
	No	46.74 (7.70)			No	55.25 (10.66)	

Note. Floating profile, $n = 38$; outside subset, $n = 99$. T-ratios are significant at $p \leq .001$, unless otherwise noted.

[a]Whether subjects belong to floating profile subset. [b]SD = standard deviation.

*$p < .01$

**$p = .084$

Table 18

Amount of Variance Accounted for by Selected MMPI-2 Scales When Predicting Inclusion in a Subset of Subjects with Floating Clinical Scale Profiles

Model	R^{2a}	F^{b}
RCd	.390	86.37
RC1	.226	39.34
RC2	.305	59.14
RC7	.264	48.56
RC2/RCd	.422	48.93
RC1/RC2/RCd	.442	35.15

Note. All F-ratios are significant at $p < .001$.

[a] R^2 statistics were computed via regression with the scale(s) in each model as the predictor variable(s) and inclusion in the floating profile subset as the dependent variable. The R^2 statistic is interpretable as follows: $R^2 \times 100$ = % of variance in dependent variable that can be accounted for by predictor variable(s). [b] The F-ratio evaluates the significance of each R^2 statistic.

Table 19

Regression Coefficients for Selected MMPI-2 Scales When Predicting Inclusion in a Subset of Subjects with Floating Clinical Scale Profiles

Model	Predictor Variables	Regression Coefficient[a]	t[b]
RCd	RCd	.023	9.29
RC1	RC1	.016	6.27
RC2	RC2	.020	7.69
RC7	RC7	.021	6.96
RC2/RCd	RC2	.009	2.72*
	RCd	.017	5.22
RC1/RC2/RCd	RC1	.006	2.19**
	RC2	.008	2.65*
	RCd	.014	3.85

Note. All t-ratios are significant at $p < .001$, unless otherwise noted.

[a]Regression coefficients were computed with the scale(s) in each model as the predictor variable(s) and inclusion in the floating profile subset as the dependent variable. The regression coefficient is a measure of the linear relationship between the predictor variable and the dependent variable, given that the influences of other predictors are held constant. [b]The t-ratio evaluates the significance of each regression coefficient.

*$p < .01$

**$p < .05$

Table 20

Descriptive Statistics for RC and Clinical Scales in a Subset of Subjects with At Least Moderate Depression, as Determined by IDS-SR Score (Total Score ≥ 26)

RC Scales				Clinical Scales			
Scale	Mean (SD[a])	Median	Range[b]	Scale	Mean (SD[a])	Median	Range[b]
RCd	65.90 (10.25)	65	47 – 86				
RC1	74.72 (11.03)	73	52 – 100	1	73.72 (10.78)	74	43 – 92
RC2	64.24 (13.18)	64	43 – 93	2	75.22 (12.31)	75	46 – 94
RC3	60.76 (11.09)	64	43 – 80	3	72.40 (15.11)	73	38 – 113
RC4	53.48 (9.15)	52	33 – 77	4	60.41 (12.53)	55	42 – 92
RC6	59.43 (11.16)	64	41 – 82	6	61.52 (14.99)	63	37 – 97
RC7	59.91 (10.08)	60	38 – 82	7	69.47 (13.19)	70	41 – 102
RC8	66.03 (10.54)	66	47 – 85	8	71.86 (12.53)	72	50 – 101
RC9	51.79 (10.83)	50	33 – 85	9	58.83 (10.97)	59	38 – 88

Note. $N = 57$.

[a]SD = standard deviation. [b]Range values displayed as follows: lowest T score – highest T score.

Table 21

Results of Independent Samples t-Test of RC and Clinical Scales between Subjects within Moderate Depressive Severity Subset and outside Moderate Depressive Severity Subset

RC Scales				Clinical Scales			
Scale	Moderate[a]	Mean (SD[b])	t	Scale	Moderate[a]	Mean (SD[b])	t
RCd	Yes	65.90 (10.25)	10.33				
	No	49.04 (8.19)					
RC1	Yes	74.72 (11.03)	10.17	1	Yes	73.72 (10.78)	7.87
	No	56.49 (9.39)			No	58.65 (10.06)	
RC2	Yes	64.24 (13.18)	5.87	2	Yes	75.22 (12.31)	8.39
	No	52.44 (9.10)			No	58.84 (9.75)	
RC3	Yes	60.76 (11.09)	5.90	3	Yes	72.40 (15.11)	5.67
	No	50.13 (9.44)			No	58.71 (12.24)	
RC4	Yes	53.48 (9.15)	4.55	4	Yes	60.41 (12.53)	3.75
	No	46.20 (9.40)			No	52.62 (11.29)	
RC6	Yes	59.43 (11.16)	4.63	6	Yes	61.52 (14.99)	4.37
	No	50.73 (10.47)			No	51.59 (10.09)	
RC7	Yes	59.91 (10.08)	8.60	7	Yes	69.47 (13.19)	7.71
	No	46.20 (7.91)			No	54.15 (8.62)	
RC8	Yes	66.03 (10.54)	6.67	8	Yes	71.86 (12.53)	8.42
	No	54.81 (8.53)			No	55.84 (8.52)	
RC9	Yes	51.79 (10.83)	3.74	9	Yes	58.83 (10.97)	2.49*
	No	45.77 (6.68)			No	54.29 (9.97)	

Note. Moderate severity subset, $n = 57$; outside subset, $n = 80$. T-ratios significant at $p < .001$, unless noted.

[a]Whether subjects scored above threshold for moderate severity on IDS-SR. [b]SD = standard deviation.

*$p < .05$

Table 22

Amount of Variance Accounted for by Selected MMPI-2 Scales When Predicting Inclusion in a Subset of Subjects with Moderate Depressive Severity, as Classified by the IDS-SR

Model	R^{2a}	F^{b}
RCd	.458	114.27
RC1	.446	108.71
RC2	.221	38.40
RC1/RCd	.579	92.26
RC2/RC1	.494	65.35
RCd/RC2	.459	56.81

Note. All F-ratios are significant at $p < .001$.

[a]R^2 statistics were computed via regression with the scale(s) in each model as the predictor variable(s) and inclusion in the moderate depressive severity subset as the dependent variable. The R^2 statistic is interpretable as follows: $R^2 \times 100$ = % of variance in dependent variable that can be accounted for by predictor variable(s). [b] The F-ratio evaluates the significance of each R^2 statistic.

Table 23

Regression Coefficients for Selected MMPI-2 Scales When Predicting Inclusion in a Subset of Subjects with Moderate Depressive Severity, as Classified by the IDS-SR

Model	Predictor Variables	Regression Coefficient[a]	t[b]
RCd	RCd	.027	10.69
RC1	RC1	.024	10.43
RC2	RC2	.019	6.20
RC1/RCd	RC1	.015	6.21
	RCd	.018	6.51
RC2/RC1	RC2	.010	3.55
	RC1	.021	8.49
RCd/RC2	RCd	.026	7.67
	RC2	.001	0.33*

Note. All t-ratios are significant at $p \leq .001$, unless otherwise noted.

[a]Regression coefficients were computed with the scale(s) in each model as the predictor variable(s) and inclusion in the moderate depressive severity subset as the dependent variable. The regression coefficient is a measure of the linear relationship between the predictor variable and the dependent variable, given that the influences of other predictors are held constant. [b]The t-ratio evaluates the significance of each regression coefficient.

*$p = .74$

Figure Captions

Figure 1. Adaptation of Watson and Tellegen's (1985) two-dimensional map of affect showing factors of High Positive Affect – Low Positive Affect and High Negative Affect – Low Negative Affect, as well as the second-order Pleasantness – Unpleasantness factor.

Figure 2. Mean RC and Clinical Scale profiles for all subjects used in the current study.

Figure 3. Mean RC and Clinical Scale profiles for subjects within (a) and outside (b) conversion V subset.

Figure 4. Mean RC and Clinical Scale profiles for subjects within (a) and outside (b) floating profile subset.

Figure 5. Mean RC and Clinical Scale profiles for subjects within (a) and outside (b) subset of moderate or greater depressive severity, as classified by the IDS-SR (total score ≥ 26).

Figure 1

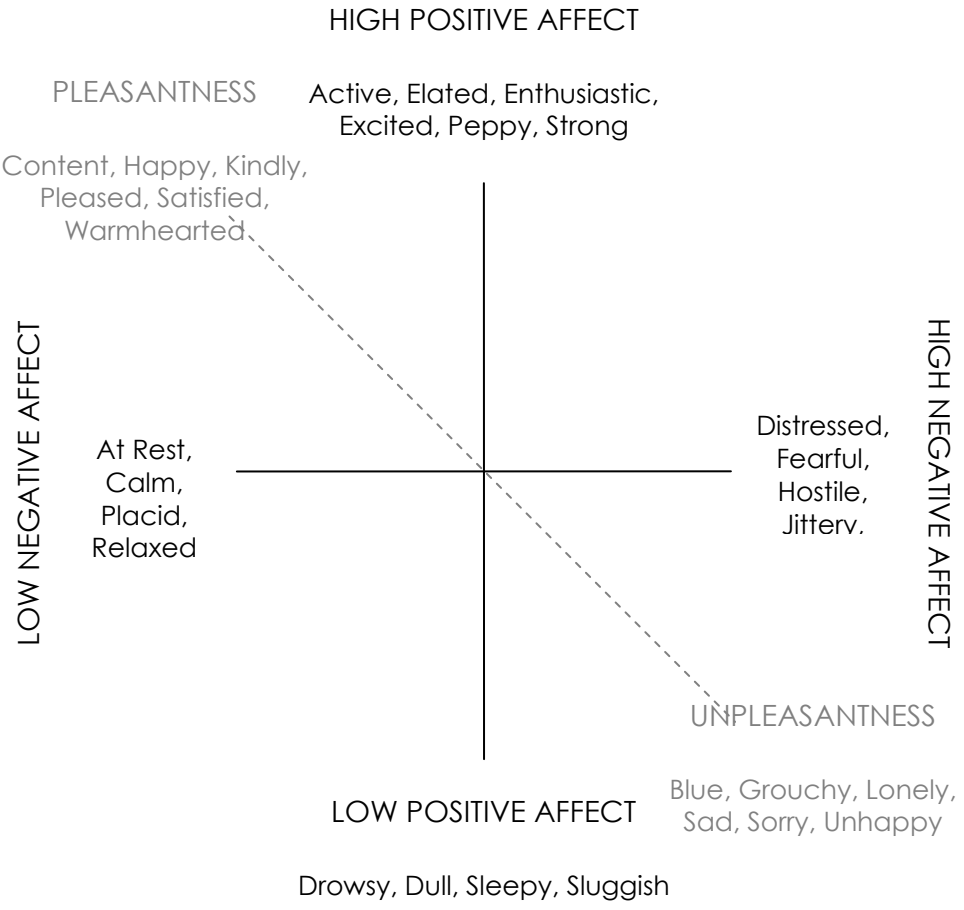
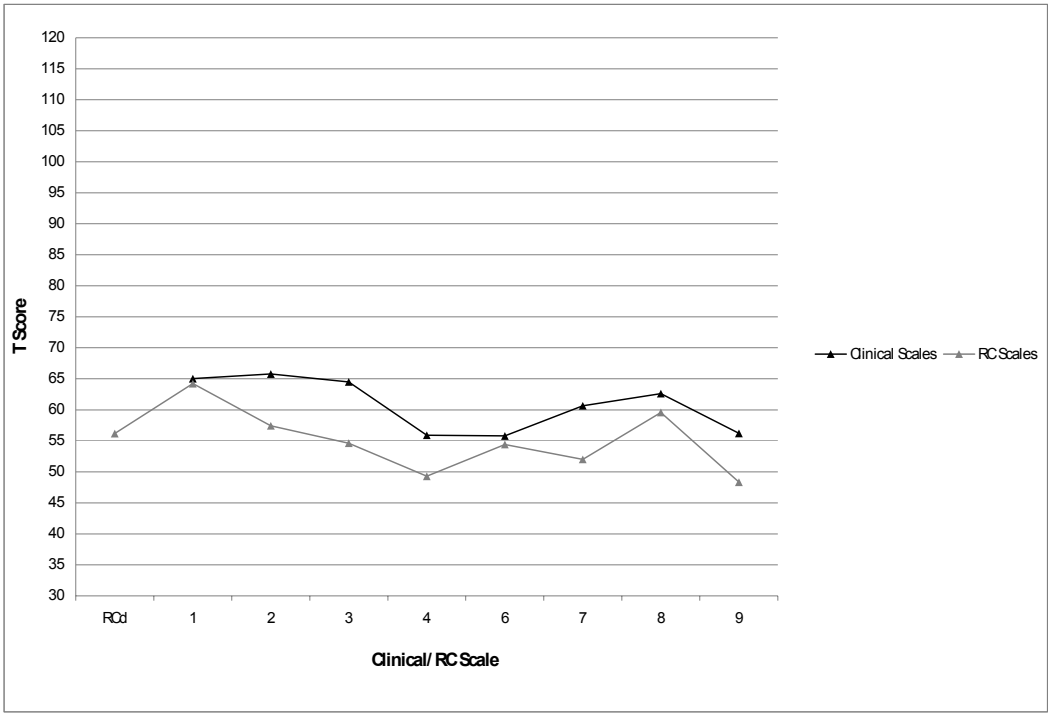

HIGH POSITIVE AFFECT
PLEASANTNESS
Active, Elated, Enthusiastic, Excited, Peppy, Strong
Content, Happy, Kindly, Pleased, Satisfied, Warmhearted
LOW NEGATIVE AFFECT
At Rest, Calm, Placid, Relaxed
Distressed, Fearful, Hostile, Jitterv.
HIGH NEGATIVE AFFECT
UNPLEASANTNESS
Blue, Grouchy, Lonely, Sad, Sorry, Unhappy
LOW POSITIVE AFFECT
Drowsy, Dull, Sleepy, Sluggish

Figure 2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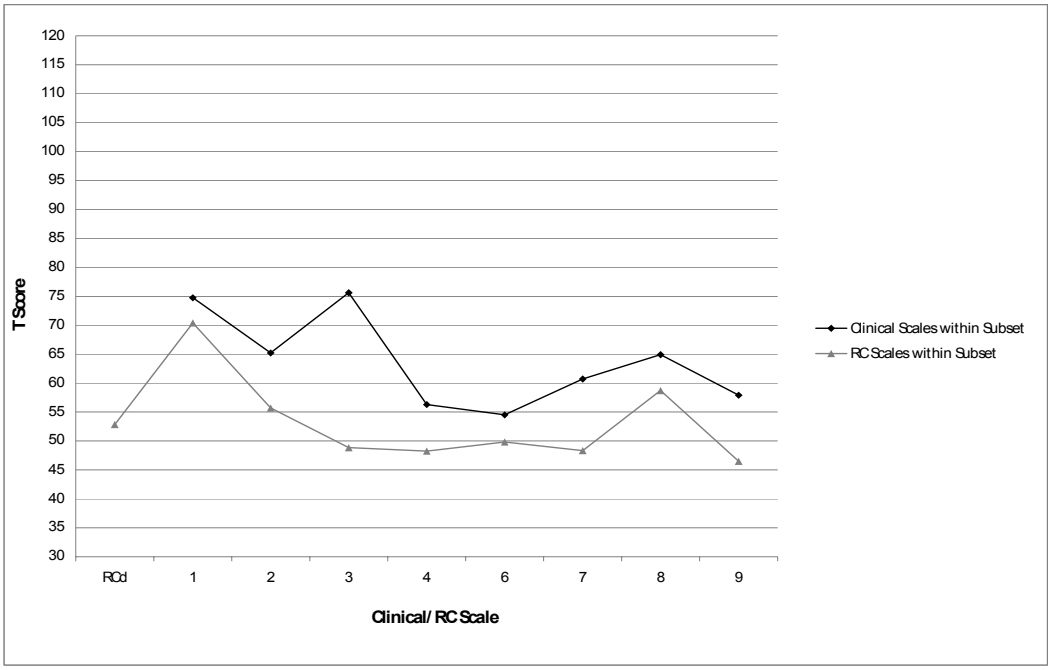
Clinical/ RC Scale
Clinical Scales
RC Scales

Figure 3

a.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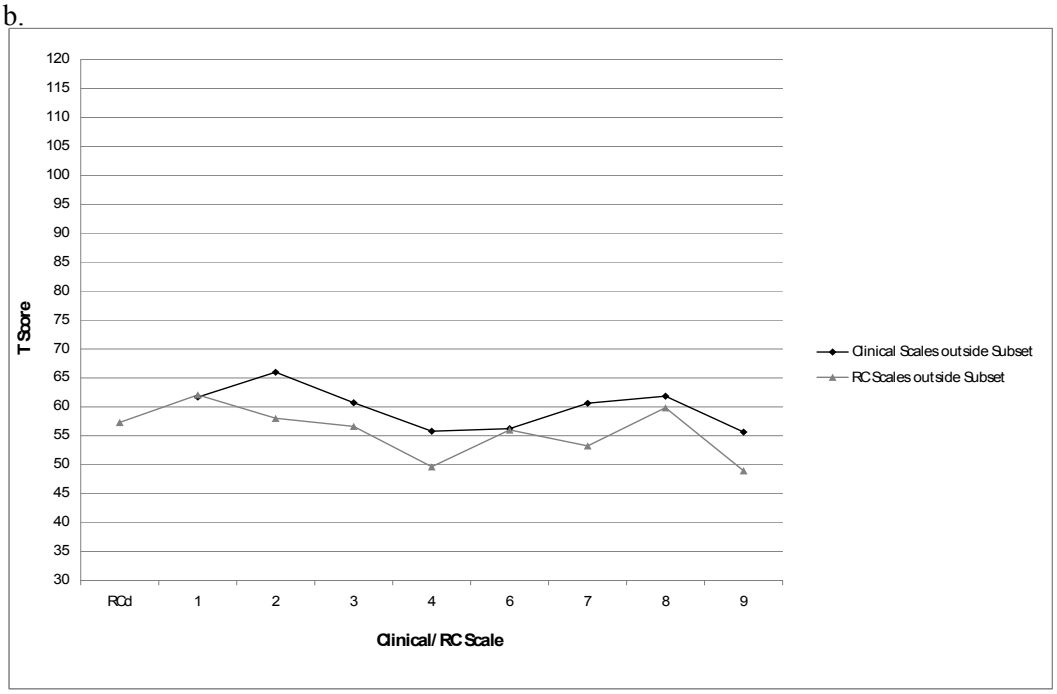
Clinical/ RC Scale
Clinical Scales within Subset
RC Scales within Subset

b.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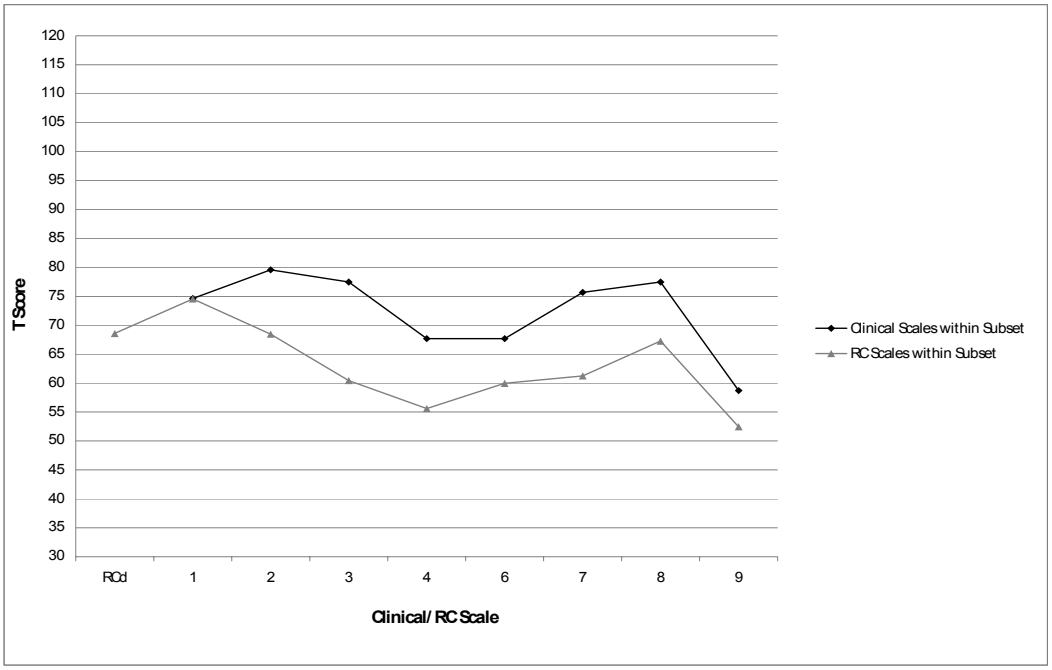
Clinical/ RC Scale
Clinical Scales outside Subset
RC Scales outside Subset

Figure 4

a.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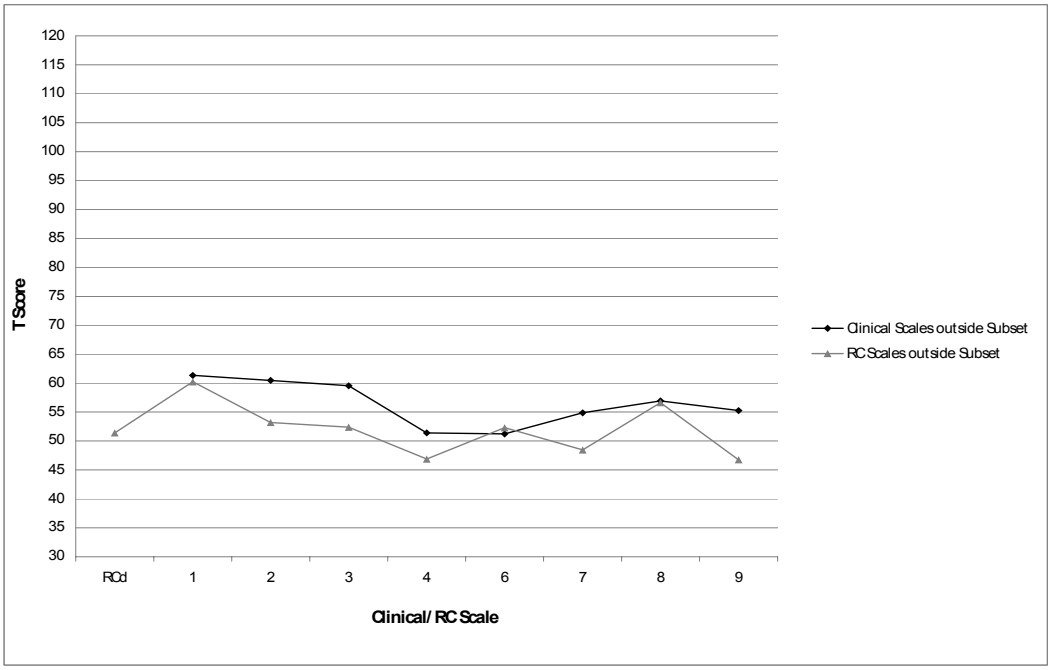
Clinical/ RC Scale
Clinical Scales within Subset
RC Scales within Subset

b.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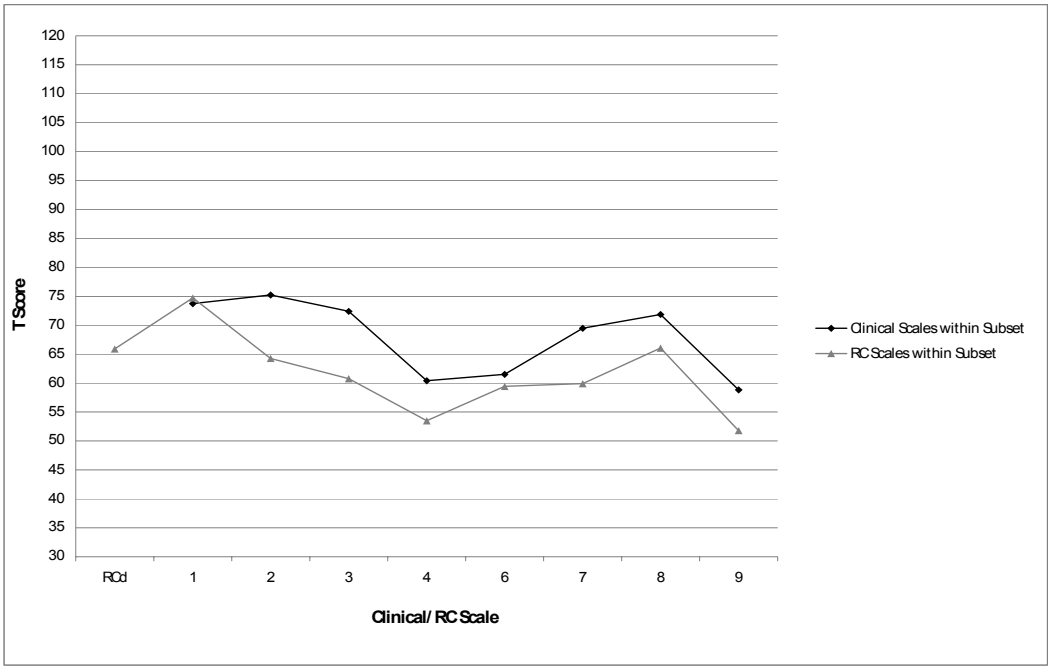
Clinical/ RC Scale
Clinical Scales outside Subset
RC Scales outside Subset

Figure 5

a.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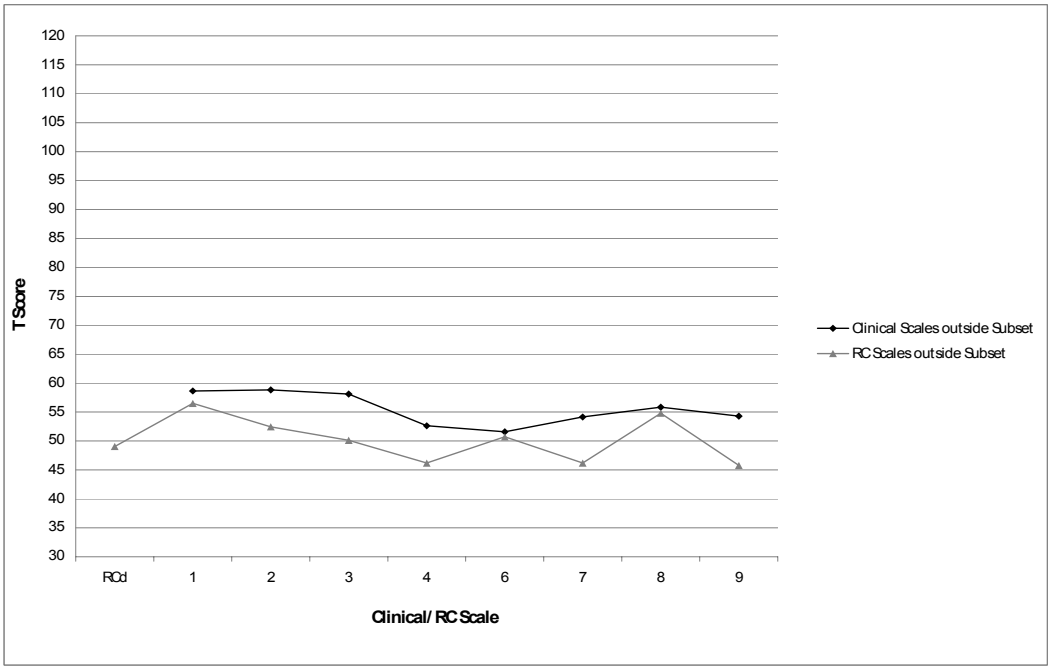
Clinical/ RC Scale
Clinical Scales within Subset
RC Scales within Subset

b.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Clinical/ RC Scale
Clinical Scales outside Subset
RC Scales outside Subset

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