ASSESSING FRONTAL LOBE FUNCTIONING IN THE CONTEXT OF VIOLENT AND AGGRESSIVE BEHAVIOR: A NEW MULTIMODAL APPROACH

By

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Abstract

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Numerous neuropsychological and neuroimaging studies have suggested that there is a strong relationship between frontal lobe impairment and aggressive behavior. Most of the studies undertaken to date, however, have failed to use valid and reliable tests and techniques to assess this relationship, which calls into question the findings of these results. Furthermore, due to the inherent limitations of all currently available tests of frontal lobe functioning and neuroimaging techniques, such as CT and SPECT, basing a conclusion of frontal lobe dysfunction on a single test or technique, is inappropriate. In addition, *Daubert* requires that scientific evidence proffered in a court of law must have scientific validity and evidentiary reliability. Although *Daubert* does not specify at what point the error rate of a test or technique exceeds the reliability requirement, given the dramatic increase of defendants who assert various defenses due to frontal lobe impairment it is imperative that the diagnoses is based on a sound methodology and valid, and reliable tests, and techniques.

At present, the methodologies employed for the assessment of frontal lobe functioning vary widely; however employing a consistent assessment approach is crucial since doing so will assist in establishing the true strength of the relationship between frontal lobe impairment and aggression. It is hoped that the multimodal approach that

has been developed here will further our understanding of the relationship between aggression and the frontal lobes, as well as provide a methodology that is likely to withstand a *Daubert* challenge, and sophisticated cross-examination.

This dissertation is dedicated to my husband, Shankar, and to the memory of Dr. Krishnamurty Karamcheti.

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“Whatever you think can do, or dream you can, begin it.
Boldness has genius and power and magic in it”
(Johann Wolfgang von Goethe).

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Namaste

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INTRODUCTION

> **"Can brain scans be used to determine whether a person is inclined toward violent behavior or criminality?"** (Snead, 2007, pg. 1266).

This question asked by Senator Biden at the nomination hearing of John G. Roberts as Chief Justice of the United States, clearly illustrates the extent to which cognitive neuroscience, amplified by the growing influence of neuroimaging techniques has captured the imagination of many who make, interpret, and study the law (Snead, 2007).

There is no doubt that understanding violence and aggression[1] has become an increasingly important question for, both, legal and mental health professionals. Traditionally, explanations for aggressive and violent behavior have focused on psychosocial explanations that emphasize factors such as family structure, poverty, and/or racism. In recent years, however, much interest has been directed toward the role of neuropsychological and neurological factors that may influence our understanding of aggression and violence. We now are aware and accept that numerous research studies have established a strong relationship between brain function and aggressive behavior Snead, 2007). More specifically, it has become clear that if we want to understand violent and aggressive behavior we must understand how these behaviors arise from neural structures and, more importantly, how deficits and/or injuries to these structures can lead to aggressive behavior. Although, to date, there is no evidence to suggest that

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[1] For purpose of this discussion, the terms "aggression" and "violence" are used synonoumosly and refer to violent behavior that is punishable by law, and presumed to be as a result of frontal lobe dysfunction or impairment. Although clearly a form of aggression, this discussion will not include sexual assaults. While evidence of brain dysfunction is frequently found in individuals, to date, no study has reliably linked sexual violence to frontal lobe dysfunction.

aggression and violence are the sole result of neurobiology, research studies have suggested that neurobiology, in particular dysfunction of the prefrontal cortex may play a much larger role than previously thought (Seguin, 2004; Brower & Price, 2001; Critchley, Simmons, Daley, et al., 2000).

For centuries, the law has taken an intense interest in the mind of violent offenders. Indeed, in almost all criminal cases, a successful conviction requires the prosecution to establish not only that the defendant engaged in the behavior he/she is charged with, but also that the crime in question was the product of a guilty mind. Thus, many legal issues have turned on the question: what was he/she thinking (Greene and Cohen, 2004).

As policy makers seek a deeper understanding of violent and antisocial behavior, neuroscientists now assume that the key components of the human condition, including free will, empathy, and morality, are the calculable consequences of a vast assembly of firing neurons. From this viewpoint, the discoveries of neuroscience resonate far beyond philosophical banter and have important implications for the way the legal system operates. For example, to the extent that a legal system attempts to fairly adjudicate criminal offenses, the legal system's effectiveness can clearly be improved by deepening our understanding about why people behave as they do. Thus, neuroscience may have important implications for how we understand the multiple influences on violent behavior, and how the legal system may better engage with violent offenders (Greene & Cohen, 2004).

The birth of what has been coined modern "forensic neurology" lies in John Harlow's 19th century observations of Phineas P. Gage. Gage, a railroad worker, suffered

the unfortunate experience of having an iron bar blasted through the front of his brain, which resulted in extensive damage to the prefrontal cortex (PFC). Despite Gage's miraculous physical and intellectual recovery, noticeable changes in his personality were reported. Indeed, the once diligent and courteous man became explicitly antisocial. As Gage's friends famously articulated: Gage is no longer Gage (Mobbs, Lau, Jones, & Frith, (2007). The case of Phineas Gage is compelling to legal scholars and neuroscientists because it provided the first indication that reasoning and regard for others can be compromised by frontal lobe injury. More specifically, Harlow's observations have led many experts to speculate that neurological insult may be a prominent factor in recidivistic and violent criminal transgressions (Mobbs, et al., 2007).

Modern empirical endeavors support the claim that the human PFC is what makes us rational and moral individuals. For example, in one of the largest study of patients with brain damage undertaken to date, Grafman and colleagues (1996) found that increased aggressive and violent scale scores were most strongly associated with localized PFC lesions in a sample of 279 Vietnam War veterans. Along with clinical observations, this study has led many researchers to suggest that damage to the PFC results in "acquired sociopathy" or "pseudopsychopathy." Given the PFC's historical and theoretical relevance to adaptive social behavior, it is not surprising that this region was among the first to be examined in antisocial and violent populations. For example, Raine and colleagues (2000) used noninvasive structural brain imaging to show an 11% reduction in PFC grey matter in individuals diagnosed with antisocial personality disorder (APD). Similar reductions have been observed in a study of pathological liars and aggressive patients with temporal lobe epilepsy (Yang, Raine, Lencz, Bihrle,

LaCasse, et al., 2005; Woermann, van Elst, Keopp, Free, Thompson, et. al., 2000). Using brain imaging to look at function rather than structure has also revealed a relationship between brain and behavior. Indeed, using Positron Emission Tomography (PET), numerous neuroscientists have found attenuated resting regional cerebral blood flow in the frontal lobes of convicted criminals and violent individuals, suggesting frontal lobe dysfunction (Raine, Buchsbaum, Stanley, Lottenberg, Abel, et al., 1994; Volkow & Tancredi, 1987). Collectively, these studies indicate that impulsive violent acts may stem from diminished function of the PFC.

The PFC is not the only area where damage may increase propensity for violent and antisocial behavior. Brain imaging and lesion studies have also suggested a role of the amygdala in theory of mind and aggression (van Elst, Trimble, Ebert, & van Elst, 2001). For example, using functional Magnetic Resonance Imaging (fMRI), Birbaumer and colleagues (2005) found that the limbic structures, such as the amygdala and hippocampus, of psychopaths were functionally abnormal. Research studies have also shown that activity in the amygdala decreases with increased scores on the Psychopathic Personality Inventory (Gordon, Baird, & End, 2004).

It has also been contended that different violent and aggressive behaviors can arise from different etiologies. More specifically, violent behavior can be placed into two distinct, categories: (1) affective aggression (i.e., impulsive, autonomic arousal, and emotional) and (2) predatory aggression (i.e., premeditated, goal-directed, and emotionless). With this dichotomy in mind, Raine and colleagues (1998) analyzed PET data to determine the functional differences between impulsive affective murderers and premeditated psychopaths and found that compared to control subjects, impulsive

murderers had reduced activation in the bilateral PFC. Conversely, predatory psychopaths had relatively normal prefrontal functioning, but increased right subcortical activity, which included the amygdala and hippocampus. These results suggest that predatory psychopaths are able to regulate their impulses, in contrast to impulsive murderers who, because of prefrontal dysfunction, lack the ability to do so (Mobbs, et al., 2007).

Numerous research studies have also suggested that mental illness is higher in incarcerated populations and it has been estimated that as many as 25% of defendants evaluated for competency are legally incompetent to stand trial. However, only 36% of the public perceive recidivistic crime as an organic disorder (Raine, 1993). Consequently, weighing discrepancies between intuitions, expert views, and empirical findings is of fundamental importance to a legal system. While it must be pointed out that any given population of incarcerated offenders is not a representative sample of all criminals, or even of all criminals who pass through the prison system, a systematic review of studies examining mental illness in 23,000 prisoners has suggested that prisoners are more likely to have some form of psychosis or major depression, and are more likely to exhibit antisocial personality disorder (APD), than the general population (Fazel & Danesh, 2002). Further, individuals with APD and violent behavior frequently have a history of childhood maltreatment or trauma and having such a history has been linked to the anomalous development of regions associated with antisocial behavior, such as the PFC (Mobbs, et al., 2007). However, there is at present no reason to believe that all violent criminal behaviors are the result of organically dysfunctional brains, but there is ample evidence to suggest that some kinds of dysfunction are likely to increase the

probability of violent and aggressive behaviors. Clearly, more research designed to elucidate the links between mental illness, neurological disorder, and aggressive and violent behavior is urgently needed. To that end, modern and rapidly improving neuroimaging techniques can provide significant contributions.

Few implications for the legal system are more important than trying to gain a better understanding of important influences on criminal behavior. This very significance, however, brings its own important challenges. Indeed, while a better understanding may lead to more effective deterrence and treatment, and to more morally sound sentencing, determining criminal responsibility is an idiographic legal conclusion, not an empirical factual one, made in the context of a variety of often conflicting goals (Morse, 2006). Therefore, even the best neuroscientific study can only provide factual evidence to be weighed alongside other behavioral evidence and considerations, rather than actually resolving the legal question as to which of the factual evidence is relevant.

In general, in the Anglo-American criminal justice system, an individual can be held criminally responsible if he/she performs a prohibited act intentionally and with a statutorily specified mental state. Yet, even if these criteria are satisfied, a defendant can be excused from liability if legally insane. The possibility of being "not guilty by reason of insanity" can be traced back to the well-known M'Naghten case. In 1843, while attempting to kill the British Prime Minister, Daniel M'Naghten mistakenly killed the Prime Minister's secretary. Experts maintained that M'Naghten exhibited such a vast deterioration in his reasoning abilities that he had no comprehension of the act he committed (Sapolsky, 2004).

The modern standards for determining legal insanity, in the long wake of M'Naghten, markedly vary across jurisdictions, with results that have prompted many calls for reform. For example, mental health professionals have been plagued by the need to answer dichotomously whether a defendant is mad or bad, or to opine that it is not him, it is his disease (Sapolsky, 2004). Furthermore, medical research indicates that patients with selective damage to the PFC can often know right from wrong, but still be unable to act on such knowledge. Consequently, this has led many prosecutors and defense attorneys to pursue more objective ways of determining whether a defendant is competent to stand trial, and if so, whether he can be held legally responsible for his actions. This, in turn, has generated significant interest in brain imaging evidence concerning a defendant's mental functioning. Consider the following:

In 1998, 15-year old Kip Kinkel shot and killed his parents and two high-school students, in Oregon. Brain imaging was used as evidence in court to support Kinkel's not guilty by reason of insanity plea with the defense providing evidence of small cavities in Kinkel's frontal lobe. Since there was no evidence that this abnormality caused his behavior, Kinkel was ultimately convicted as an adult and sentenced to 111 years in prison. This example raises important questions not only about the extent to which neuroimaging evidence may affect trial outcomes, but also about the ways in which the legal system can come to understand the changing views of the brain, assess when those views are relevant, and determine how to integrate that knowledge into the legal decision-making process (Feigenson, 2007).

These research findings have special relevance in countries such as the United States where the death penalty is applied. Indeed, it is possible that the 2005 decision of

the Supreme Court of the United States that made it illegal to use capital punishment for any offender who was under the age of 18 when the crime was committed may have been due in part by evidence presented in amicus briefs, which included neuroscientific evidence (see, *Roper* v. *Simmons*, 2005). While we should not assume that this evidence affected the outcome in this case, it is noteworthy that neuroimaging techniques were, for the first time, tapping on the door of the U.S. Supreme court (Feigenson, 2007).

Given the increasing public interest in brain imaging it is, therefore, crucial for proper legal decision-making that judges and jurors understand the limitations of brain imaging. True understanding, however, requires collaboration among various disciplines such as psychology, biology, and neuroscience. Indeed, an assessment of frontal lobe functioning in the context of violence and aggression requires a multimodal and multidisciplinary approach that employs valid and reliable measures, tests and techniques. In addition, in order to establish the true strength of the relationship between FLD and violent behavior the scientific community must employ a consistent methodology to assess FLD in the context of violent behavior. Finally, the law also requires that the evidence proffered has evidentiary reliability or scientific validity. However, most studies undertaken, to date, have specific methodological limitations which must be considered and addressed in order to establish a reliable and valid methodology for the assessment of frontal lobe dysfunction. Following is a brief outline of the issues to be addressed and to be discussed in greater detail later:

- Aggressive behavior has been associated with various neuropsychiatric, neurological, and neuropsychological disorders, and structural and functional brain abnormalities. More specifically, violent subjects have shown to have decreased glucose

uptake in the areas of the frontal, prefrontal, and temporal cortex. These hypoperfusions, however, may be completely independent of, for example, substance abuse or certain major psychiatric diseases.

- Since the cause of aggression is likely to be multifactorial, a simple correlation between an aggressive act and brain dysfunction is rarely possible. Indeed, aggressive behavior occurs in a social context and, therefore, other variables like drug and alcohol abuse must also be considered. Further, neurobehavioral predisposition to violence does not always lead to antisocial behavior and most individuals with brain dysfunction do not commit aggressive acts. Clearly, association is not causation and, while a brain lesion may change the threshold for violence, it is most likely not the sole or direct cause of an aggressive act.
- Almost all studies undertaken to date are retrospective and anecdotal, with small sample sizes and often inconsistent results. This, clearly, limits our ability to generalize these findings and calls into the question the accuracy of these findings.
- The identification of brain dysfunctions is imperfect given the limitations of diagnostic classifications and certain neuroimaging techniques, neurological examination, and neuropsychological assessments. One striking aspect of studies undertaken to date is that there are dissociations between, for example, CT and MRI findings and neuropsychological measures of brain dysfunction. While this may seem contradictory, it must be remembered that CT and MRI are structural measures while neuropsychological tests are functional measures. These findings, thus, may reflect dissociations between

function and structure, and clearly illustrate the need for the assessment of both, structure and function.[2]

- Most studies frequently involve unusual populations and are often conducted in highly specialized settings under unusual circumstances, such as pretrial evaluations in forensic centers. Research findings are, therefore, skewed toward those whose official records are analyzed, leaving undetected many other violent or aggressive individuals. Consequently, the potential for violence in the general population (the true base rate of violence) remains difficult to assess and predict.
- Most neuroimaging studies undertaken, to date, have failed to make use of specific tasks which challenge or activate those brain areas thought to be dysfunctional in aggression and violence. Doing so, however, is crucial, since activation studies provide stronger findings and help develop a more theory-driven approach to understanding aggression.
- In assessing frontal lobe dysfunction in the context of violent behavior it is crucial to obtain baseline, or resting values, prior to the activation of the area of interest; however many studies have failed to do so. It has been suggested that caution should be used when using the resting state as a control since this state has been associated with its own pattern of regionally decreased or increased activity. However, the resting state is a useful comparison if one is examining changes in regional cerebral blood flow and glucose metabolism (Stern & Silbersweig, 2001).

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[2] It is important to note that structural and functional measures may also not correlate since the brain can be structurally impaired and still function within normal limits, as well as be functionally impaired and yet appear structurally normal.

- Studies measuring glucose metabolism are, by design, based on small sample sizes and, therefore, preclude the use of statistical tests of significance. Consequently, firm conclusions cannot be drawn from these studies.
- A criticism of CT, MRI, fMRI, PET and SPECT studies is that the extent to which deficits are relative are often not assessed. For example, whereas CT scans may reveal abnormalities in temporal areas, a question remains as to whether these abnormalities are greater relative to another brain area. Similarly for PET data, it is important to establish that a certain brain area shows reduced glucose metabolism relative to another brain area if one wants to establish specificity of findings. Data from studies that demonstrate such a relative deficit constitute more powerful evidence of specific brain dysfunction, but are rare.
- In order to reliably assess FLD it is invaluable that only tests that have been established to reliably activate the brain region of interest, such as the frontal lobes, are used in the assessment of aggressive and violent behavior. However, many published studies have failed to employ neuropsychological tests that have adequate sensitivity and specificity.
- Although the evaluation of malingering occurs in many criminal forensic cases, it is not addressed specifically in statute. However, in terms of case law, *United States v. Greer* (1998) emphasizes the importance of malingering assessment.

- It has been well established that the rate of malingering in criminal forensic settings is approximately 30%.[3] Any assessment of violence and aggression in the context of frontal lobe impairment must, therefore, also include reliable tests of malingering of cognitive deficits and mental illness. However, to date, only one study has done so.
- Many layers of signal processing, statistical analysis and interpretation separate imaged brain activity from the psychological traits and states inferred from it. There is a danger that the public, including judges and juries, will ignore these complexities and treat brain images as indisputable truth.
- There is, at present, no standardized methodology for the assessment of FLD in the forensic population, or the population, in general. Therefore, contradictory literature exists on the actual predictive accuracy of various measures of frontal lobe dysfunction. This lack of a unified approach makes it difficult, if not impossible, to conduct meta-analyses. Doing so, however, is important since findings from individual studies are limiting due to small sample size. Clearly, combining multiple studies and, thereby, increasing sample size, provides enhanced power and increases our ability to detect small, but perhaps meaningful, effects.

There is no doubt that the assessment of frontal lobe functioning in the context of violence and aggression is a daunting task. While it is impossible to ascertain whether or not an individual was impaired at the exact time the crime was committed, it is possible to determine whether or not a structural or functional brain abnormality may have been a

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[3] Although psychotic symptoms are most commonly feigned, some individuals also attempt to malinger cognitive impairment.

contributing factor. It is, therefore, not surprising, that with the advent of neuroimaging, defense attorneys have come to increasingly rely on evidence of brain dysfunction, in the form of brain scans, to argue against culpability and/or for mitigation. Consequently, expert opinions regarding frontal lobe functioning, now more than ever before, must be based on a sound methodology, including valid and reliable tests and measures. This requirement set forth in *Daubert*, as well as the development of more sophisticated measures to assess FLD within the forensic population, has forced judges to become more judicious in their evaluations of scientific evidence proffered in a court of law. The courts responsibility as "gatekeepers" under *Daubert* and the increased sophistication needed to make an admissibility determination is clearly illustrated in *People v. Hix* (2009).

At trial, Dr. Perrotti testified that a Single Photon Emission Computed Tomography (SPECT) scan is a generally accepted tool to diagnose brain trauma or injury. Dr. Perrotti also testified that he did not believe appellant suffered from schizophrenia. Dr. Perrotti stated that he was not an expert in the area of SPECT scans or brain imaging and did not know whether the correct procedures for the SPECT scan were used in this case. After Dr. Perrotti testified, the trial court held that he was not qualified as an expert to determine whether the correct procedures were applied and that defense expert, Dr. Amen, would be required to testify regarding the procedures used in appellant's SPECT scan.

Dr. Mayberg, a professor of neurology and psychiatry, who conducted research on the use of brain imaging technology on the study of behavior, including major depression, testified that while SPECT scans are reliable for determining the presence of

stroke, epilepsy and dementia, they are not reliable for other conditions, such as schizophrenia. She also testified that (1) MRI scans have made SPECT scans obsolete for many purposes, and (2) MRI scans rather than SPECT scans are now used routinely in head injury cases. She further testified that the Society of Nuclear Medicine has established guidelines for SPECT scans which state that substantial variability may be noted between normal individuals and a single individual obtained at different times and that controlled studies with SPECT scans have only been conducted for stroke, Alzheimer's disease, and epilepsy. Moreover, substances such as antidepressants, alcohol, drugs, and caffeine can affect the results of the SPECT scans because they affect blood flow and metabolism. Dr. Mayberg also commented that the defense expert's report did not indicate the basis for his determination of what constituted normal or abnormal blood flow, or the basis for his conclusion that appellant's blood flow was not normal. In addition, the defense expert failed to take into consideration the effects of antipsychotic and antidepressant drugs that appellant was taking at the time of the SPECT scan and appellant's chronic drug and alcohol abuse, all of which could have affected the SPECT scan.

Defense expert, Dr. Amen, noted that SPECT scans are not generally accepted within the scientific community to draw inferences regarding behavior, judgment, insight or motive for the commission of a crime. He agreed that there is significant controversy regarding the use of SPECT to diagnose or treat psychiatric disorders. Appellant's SPECT scan showed significant decreased activity in his prefrontal cortex, which manifests in lack of impulse control, forethought and empathy and Dr. Amen opined that the damage to appellant's brain was likely due to a head injury to the prefrontal cortex. But on cross-

examination, Dr. Amen acknowledged that appellant was on anti-depressant and anti-psychotic medications when the SPECT scan was administered, and that variables including poor diet, negative thoughts, and prior alcohol abuse can affect a scan. He did not screen appellant for illicit drugs prior to the scan, because the "odds are he wasn't using drugs in jail."

The trial court, consequently, excluded the results of the SPECT scan, concluding that while SPECT scans are generally accepted within the community of neurologists and radiologists for diagnosing dementia, epilepsy, and seizures, they are not generally accepted within the field of neurology in determining brain damage. Moreover, the routine use of SPECT in brain injury or post concussion syndrome is not supported by the literature. The court noted that it is still unclear as to what constitutes normal versus abnormal rates of blood flow as measured by SPECT scans. The trial court also expressed concern that Dr. Amen did not test appellant for illegal drugs before the SPECT scan on the assumption that while in jail, appellant did not have access to illegal drugs. The trial court noted that drugs are available in jail and that there are many unknown variables, such as drug use and mental disorders that could affect cerebral blood flow.

In 2009, *Hix* appealed contending that he was denied due process and the right to present a meaningful defense because the trial court required a *Kelly* hearing on the admissibility of the SPECT scan and, subsequently, excluded the scan. At the *Kelly* hearing, appellant stated that the SPECT scan would be used during the guilt phase to show that he had a mental defect, did not harbor any intent to kill, and was insane at the time he committed the crimes. On appeal, however, appellant focused on the use of the

SPECT scans to corroborate evidence that he suffered from dysfunction of the frontal and temporal lobes, and to show brain blood flow, and perfusion.[4]

The Appellate court affirmed the judgment and concluded that an independent review of the evidence presented at trial indicates that while SPECT scans are generally accepted in the scientific community of neurology to diagnose Alzheimer's disease, stroke, and epilepsy, they are not generally accepted in the scientific community to diagnose brain injuries or mental disorders such as schizophrenia or depression. Further, appellant did not cite to, nor did the Court find California cases holding that SPECT scans are generally accepted to diagnose schizophrenia or brain damage negating appellant's intent to kill or proving that he was insane at the time he committed the crimes. In addition, the Court argued that studies have shown that wide variations occur among individuals tested at different times as well as among normal subjects, and that drugs and alcohol can affect the scan.

The relationship between frontal lobe dysfunction and violent behavior is the topic around which this dissertation is developed. More specifically, it is hoped that this research will provide the scientific community with an "algorithm" for the assessment of frontal lobe functioning and, consequently, (a) clarify the relationship between frontal lobe impairment and violent behavior, and (b) ensure that expert evidence of frontal lobe impairment proffered in a court of law is based on a sound methodology that is likely to withstand a *Daubert* challenge.

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[4] The Appellate Court held that the SPECT scan is a scientific technique to which a jury might ascribe an inordinately high degree of certainty. Therefore, the trial court did not err in holding a *Kelly* hearing.

CHAPTER I. THE LAW AND FRONTAL LOBE FUNCTION

Law and science, both, seek to ascertain the truth though they embrace disparate objectives. While law aims to resolve dispute between two parties, science seeks to understand and explain phenomena. Indeed, as once noted by the Supreme Court "the balance struck by the Rule of Evidence is designed not for the exhaustive search for cosmic understanding, but for the particularized resolution of legal disputes" (*Daubert v. Merrell Dow*, 1993, pg. 579). Thus, while science seeks a universal truth, law "seeks repose" (Dreyfuss, 1995, pg. 1795). Despite these differing objectives, however, law and science meet each time scientific evidence is sought to be admitted into a court of law.

There is no doubt that neuroimaging technology and tests of neuropsychological functioning have advanced our understanding of how the brain operates by providing, for example, structural and functional images of both healthy and diseased brains. In fact, neuroimaging technology now pervades our society and has particularly affected the legal arena. Indeed, some judges have argued that scientific evidence, which offers insight into an offender's mental state, is crucial because it is the only means of determining whether an offender's punishment is proportional to his crime. Other judges have argued that "objective" evidence does not "wholly determine the controversy," and, instead, have focused on their duty as gatekeepers to independently evaluate scientific evidence (Baskin, Edersheim, & Price, 2007, pg. 239).

With respect to understanding the relationship between the brain and aggressive and violent behavior, the state of scientific knowledge is still relatively limited, but promising. However, while aggression and violence are most likely multifactorial behaviors that are not easily reducible to a specific neuropsychological test score, or

brain function, or region, the frontal cortex and executive dysfunction, in particular, have been linked to aggression and violence. It is, therefore, not surprising that, to date, numerous defendants have sought to admit evidence of FLD, especially in the form of neuroimaging scans and tests designed to assess executive functioning.

Neuroimaging

The foundation for using neuroimaging evidence in criminal trials lies in a massive and growing body of scientific literature on both the neuroanatomical and neurochemical bases of violence. For example, in 1998 and 1999, an interdisciplinary group of experts were convened to create a consensus statement on the relationship between the mind, the brain, and violence. To this end, these experts conducted an exhaustive literature survey of the role of the brain in violent behavior and, in 2001, issued a statement noting that the frontal lobes and the limbic system "are thought to play preeminent roles in [violent] behavior" (Filley, Price, Nell, Antoinett, et al., 2001, pg. 5). More specifically this statement asserted that:

> "Aggressive behavior has been thought to arise from the operations of the limbic system under certain circumstances, and the amygdala is the structure most often implicated.... Prefrontal functions may ... provide an individual with the capacity to exercise judgment in the setting of complex social situations in which actions have significant consequences. In many cases, this capacity for judgment may serve the important function of inhibiting limbic impulses, which, if acted on, could be socially inappropriate or destructive.... Therefore, there exists a balance between the potential for impulsive aggression mediated by temporolimbic structures and the control of this drive by the influence of the orbitofrontal regions" (Filley, et al., 2001, pg. 5).

This view of aggression and violence was informed, and has been reinforced by numerous neuroimaging studies. The first such study, published by Raine (1994), used PET to illustrate diminished activity of the PFC of defendants accused of murder. Other

research studies have affirmed the widespread association of prefrontal dysfunction and violence, and articles and literature reviews written by prominent neuroscientists have reached similar conclusions (Barkataki, Kumari, Das, Hill, et al., 2005; Bufkin & Lutrell, 2005; Frierson & Finkenbine, 2004).

In addition, a significant area of research on the disposition to criminal violence concerns the neurobiological correlates of psychopathy and antisocial personality disorder (APD). As noted in an amicus brief filed by the American Psychological Association (APA) in *Roper v. Simmons* (2006), "psychopathy is presumed to be deep seated, stable over time, and resistant, if not absolutely impervious, to change" (APA Brief, pg. 20-21). APD is a related diagnostic construct of the Diagnostic and Statistical Manual of Mental Disorders (4th ed. 1994) (DSM-IV-TR, 2000) that is based on behavioral characteristics such as "a pervasive pattern of disregard for, and violation of, the rights of others that begins in childhood or early adolescence and continues into adulthood" (DSM-VI-TR, pg. 645). A recent survey, of structural and functional neuroimaging studies relating to APD and psychopathy, concluded that "the functional but not the structural neuroimaging studies strongly suggest dysfunction of the frontal and temporal lobes, and possibly other structures including the angular gyrus and corpus callosum, in psychopathy (Saxby, Chambers, & McArthur, 2005). Similarly, Raine and colleagues (200), showing a structural brain deficit in subjects with APD, tentatively concluded that this structural deficit "may underlie the low arousal, poor fear conditioning, lack of conscience, and decision-making deficits that have been found to characterize antisocial, psychopathic behavior. Many other neuroscientists likewise have undertaken inquiries using neuroimaging techniques to explore the potential connection

between aggression and brain abnormalities. By linking brain abnormalities to specific behaviors and, specifically, to violent and aggressive behaviors, these studies have provided a foundation for the use of neuroimaging evidence in criminal trials.

It has been contended that "the evaluation of research on the neurobiology of violence demands conceptual clarity, along with careful analysis of methods and data to prevent misunderstanding and possible abuse of the results" (Brower & Price, 2000, pg, 145-146). Indeed, while brain imaging may appear to offer great objectivity, current brain imaging techniques, by themselves, may be no more objective than the modalities that came before them, such as EEG, or neuropsychological testing. More specifically, because the state of scientific knowledge continuously changes, there are multiple contradictory scientific views of a given issue, including the causal link between FLD and aggressive behavior, all of which may be potentially credible and reliable to some degree.

As previously noted, developments in neuroimaging techniques have affected the law both indirectly and directly. The indirect developments are visible in the large amount of discussion that has occurred about speculative applications of this new technology. The direct impact has occurred where neuroimaging evidence has been proffered in the courtrooms and has led to the creation of a body of decisional law that has shaped and continues to shape the legal landscape. Challenges to the admissibility of scientific evidence are rare in criminal cases due to the longstanding practice of deferring to experts and the usually limited resources available to many criminal defendants (Mobbs, et al., 2007. However, while the reality of FLD and its effect on judgment is well accepted in the scientific community, the evidentiary reliability of a diagnosis of

FLD and evidence about how it may have contributed to the criminal offense may be open to challenges particularly with respect to neuroimaging results.

Clearly, if judges and juries seek to rely on brain images and neuropsychological test results to assist them in making culpability determinations, neuroimaging techniques and neuropsychological assessment tools must meet pertinent legal standards for the admissibility of scientific evidence. To that end, there are number of legal guidelines that assist courts in their admissibility determinations.

A. Federal Rules of Evidence (FRE)

FRE 104(a) provides that the judge, not the jury, determines preliminary questions concerning the admissibility of evidence and the qualification of a person to be a witness. Since evidence of FLD generally requires expert testimony by expert witnesses, FRE 702 provides the general standard of admissibility in federal courts. That rule permits a witness qualified as an expert by knowledge, skill, experience, training, or education to testify to scientific, technical, or other specialized knowledge if that knowledge can assist the trier of fact to understand the evidence or to determine a fact in issue."

In 2000, an amendment to FRE 702 added the following language: if (l) the testimony is based upon sufficient facts or data, (2) the testimony is the product of reliable principles and methods, and (3) the witness has applied the principles and methods reliably to the facts of the case. The 2000 amendment was enacted explicitly in response to *Daubert v. Merrell Dow Pharmaceuticals, Inc.* (*Daubert*), the 1993 Supreme Court case that set the current standard for judicial application and interpretation of FRE 702 in many states. Expert testimony that survives scrutiny under FRE 702 may still be

excluded under FRE 403. FRE 403 provides that relevant evidence may be excluded if its probative value is substantially outweighed by the danger of unfair prejudice, confusion of the issues, or misleading the jury, or by considerations of waste of time, undue delay, or unnecessary presentation of cumulative evidence. One might expect that FRE 403 would rarely operate to exclude expert testimony because: (1). FRE 702 might itself exclude expert testimony that would be unfairly prejudicial or confusing, since such testimony would not assist the trier of fact to understand the evidence or to determine a fact in issue, and (2) a trial judge may conclude that, although the evidence passes 702 standards, there is a danger that jurors will use the evidence for an impermissible purpose, or that the evidence will serve primarily to confuse the jurors because they are not likely to understand the evidence. In the case of brain imaging techniques, in particular, a trial judge may fear that jurors (a) will give too much deference to the images, or (b) fail to understand the technological limitations inherent in all neuroimaging techniques, and, thus, fail to perform their duty to assess the credibility of the evidence.

B. *Daubert v. Merrell Dow Pharmaceuticals, Inc.*

The plaintiffs in *Daubert* were minors born with "serious birth defects" (pg. 582), who claimed that Bendectin, a prescription drug sold by Merrell Dow and taken by the plaintiffs' mothers during their pregnancy to control nausea, caused their birth defects. Merrell Dow moved for summary judgment and offered an affidavit from a well-credentialed expert who concluded that “maternal use of Bendectin during the first trimester of pregnancy has not been shown to be a risk factor for human birth defects" (pg. 582). The plaintiffs offered opinions from eight experts of their own that Bendectin

could cause human birth defects and the federal district judge granted defendant's motion for summary judgment (*Daubert*, 1989).

The Ninth Circuit affirmed, citing *Frye v. United States* (*Frye*), 1923 D.C. Circuit case upholding the exclusion of the result of a "systolic blood pressure deception test," commonly known as a "lie detector" test, offered by the defendant in a second-degree murder case. In this short opinion the D.C. Circuit stated that admissibility of "a scientific principle or discovery" depends on whether it is "sufficiently established to have gained general acceptance in the particular field in which it belongs" (*Frye*, 1923, at 1013). This brief statement provided the dominant test for admissibility of scientific evidence for seventy years, including the years after the adoption of the Federal Rules of Evidence in 1975. The *Frye* or "general acceptance" test appears to be based on the notion that judges did not have to make their own judgments about the quality of scientific evidence, but rather were to look to the judgment of those who were knowledgeable in the particular field.

The United States Supreme Court vacated the judgment of the Ninth Circuit that had upheld the district court's granting of defendant's summary judgment motion and remanded the case for further proceedings. All nine justices agreed that FRE 702 provides the standard for judging the admissibility of scientific evidence in federal courts and that 702 supersedes the Frye test. The Court looked at the "permissive backdrop" of the Federal Rules and concluded that the "austere standard" of the *Frye* test was incompatible with the Federal Rules and should not be the standard in federal courts (*Daubert*, 1993, at 589). Justice Blackmun's opinion of the Court, joined by six other justices, attempted to provide some reassurance that the FRE 702 standard does not mean

that trial judges are powerless to exclude any scientific evidence or that all scientific evidence is admissible; to be admissible under FRE 702 the scientific evidence must be "not only relevant, but reliable" (*Daubert*, 1993, at 589). Judges must determine that the offered evidence is "scientifically valid" and that it "properly can be applied to the facts in issue" (*Daubert*, 1993, at. 593). Justice Blackmun then identified the following five factors that judges might consider in ruling on offers of scientific evidence: (1) the "falsifiability, or refutability, or testability" of the expert's "reasoning or methodology"; (2) "peer review and publication" of the expert's "theory or technique"; (3) "the known or potential rate of error" of the particular scientific technique; (4) "the existence and maintenance of standards controlling the technique's operation"; and (5) "general acceptance" in the "relevant scientific community" (*Daubert*, 1993, at 592-594). Justice Blackmun closed by responding to competing underlying concerns. To those who might fear that abandoning the "general acceptance" test will result in pseudoscientific evidence confounding juries, he pointed out the effectiveness of the "conventional devices" of "[v]igorous cross-examination, presentation of contrary evidence, and careful instruction on the burden of proof." To those who might fear that a "gatekeeping" role of judges "will sanction a stifling and repressive scientific orthodoxy and will be inimical to the search for truth," he noted the important differences between the quest for truth in the courtroom and the quest for truth in the laboratory. The fact that judges have to decide legal issues "finally and quickly" requires exclusion of "[c]onjectures that are probably wrong" (*Daubert*, 1993, at 595-597).[5]

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[5] It must be noted that some states still apply the *Frye* general acceptance test.

C. Post-*Daubert* cases

The Supreme Court returned to the topic of admissibility of expert testimony four years later in *General Electric Company v. Joiner* (1997). In *Joiner* the federal district court granted summary judgment to defendants on the grounds that the testimony of the plaintiff's experts failed to establish a link between his lung cancer and exposure to chemicals on his job and was thus inadmissible. The Eleventh Circuit reversed, holding that because the Federal Rules of Evidence governing expert testimony display a preference for admissibility, a particularly stringent standard of review to the trial judge's exclusion of expert testimony must be applied. The Supreme Court reversed, holding unanimously that abuse of discretion is the proper standard of review for rulings on the admissibility of scientific evidence. The Court went on to hold, with all but Justice Stevens joining, that the trial judge did not abuse his discretion in excluding the testimony of plaintiff's experts. Further, in 1999, in deciding *Kumho Tire Company v. Carmichael* (*Kumho*), the Supreme Court concluded that the trial judge's basic gatekeeping obligation does not only apply to scientific testimony but to all expert testimony. The Court emphasized that the *Daubert* "list of factors was meant to be helpful, not definitive" (*Kumho*, 1999, at 151). The Court also stated that the abuse-of-discretion standard approved in *Joiner* applies as much to the trial court's decisions about how to determine reliability as to its ultimate conclusion. Finally, the Court reversed the Eleventh Circuit and upheld the district court's exclusion of the testimony of plaintiff's expert that a defect in the design or manufacture of a tire caused it to blow out.

Research studies have shown that judges apply different *Daubert* criteria according to the kind of evidence under review, and that the decision about which criteria

to use is closely related to perceptions that some kinds of evidence are more scientific than others (Dahir, Richardson, Ginsburg, Gatowski, et al., 2005). In fact, a recent study which performed a content analysis of district court cases to investigate judges' evaluations of expert characteristics and evidence characteristics for damage, toxicology, and psychological/psychiatric, testimony, found significant differences in the application of *Daubert* criteria. More specifically, Merlino and colleagues (2008), found that:

1. Of the factors associated with the *Daubert* guidelines, the most frequently mentioned was general acceptance, followed by falsifiability, peer review and publication. The *Daubert* factors were mentioned most frequently in the toxicology cases. Falsifiability, peer review, and publication were evaluated twice as often in toxicology cases as in psychological/psychiatric, or damages cases. Error rate and general acceptance were the most frequently mentioned *Daubert* criteria in damages cases. *Daubert* factors were mentioned relatively infrequently in psychological/psychiatric cases, where general acceptance was the most frequently mentioned characteristic; and

2. The number of challenges to psychological/psychiatric testimony remained at a fairly constant level across the period of this study (1993-2004), and these challenges were successful in 21 of 64 cases. Although the number of challenges to damages testimony did increase over this period, these challenges were also successful in only 21 of 64 cases. Challenges to toxicology testimony, which also increased during this period, were successful in 49 of 64 cases.

From these findings the authors concluded that the complexity of the evidence, which is related to the number of factors applied to it, may be the key factor in whether

or not the evidence is found to be admissible (Merlino, Murray, & Richardson, 2008). Furthermore, Gatowski and colleagues (2001) reported that judges believe that and admissibility determination should be made ‘‘on a case-by-case basis, depending on the nature of the evidence proffered, the purpose for which the evidence is proffered, the qualifications of the expert, and existing precedents’’ (pg. 449). Finally, it has been established that the mean length of discussion of *Daubert*, FRE 104, and FRE 702 greatly increased following the *Daubert* decision, suggesting that judges have placed significant importance on the *Daubert* guidelines (Groscup, Studebaker, Huss, O'Neil, et al., 2002). The courts' increasing attention to the scientific validity and evidentiary reliability of proffered evidence is also reflected in cases that have sough to admit expert testimony of impairment of cognitive, neurological, and psychological impairments.

D. Case law - neuroimaging techniques

1. General acceptance

Courts who regard neuroimaging evidence as too novel sometimes deny its admission into evidence under the *Frye* standard, on the grounds that this evidence has yet to achieve "general acceptability." This was the case in *People v. Protsman* (2001) in which the defendant sought to admit PET scan evidence and psychiatric testimony to the effect that he was suffering from decreased frontal lobe activity and, therefore, could not formulate the necessary intent for first degree murder. However, since the evidence had not yet achieved general acceptance, it was not admitted. Similarly, in *People v. Yum* (2003), the defendant, after having been convicted of second degree murder argued on appeal that the trial court erroneously refused to admit his proffered evidence of a single photon emission computed tomography (SPECT) brain

scan which showed diminished activity in his left temporal lobe and damage caused by brain trauma, causing him to kill his mother and sister. Because of the novelty of the diagnostic approach (using a SPECT scan to diagnose brain trauma and post traumatic stress disorder), the defendant failed to satisfy the court that the scientific evidence was "generally accepted."

2. The right to present evidence

There are a number of cases in which neuroimaging evidence was found to meet the requisite standard for scientific testimony and, consequently, was admitted. In 1985, the U.S. Supreme Court has ruled that the Constitution requires that a state provide access to an expert's assistance when the question of a criminal defendant's sanity is being litigated (*State v. Oklahoma*). At least one court has also held that this right extends to the provision of neuroimaging tests. Indeed, in *People v. Jones* (1994), the appeals court reversed a murder conviction on the grounds that the defendant was denied neurological testing that supported his defense that he was suffering from brain damage that impaired his ability to think quickly and flexibly, and to perceive risk.

3. Determination of guilt

There are a few cases in which neuroimaging evidence has been introduced at the guilt phase to support claims of lack of requisite culpable mental state or excuse defenses based on insanity. Consider the following:

In *United States v. Erskine* (1978) the defendant, after having been convicted of making false statements to an official of a federally insured bank, argued on appeal that the court erroneously prevented him from introducing testimony and a brain scan that he claimed showed that he lacked the mental capacity to formulate the specific

intent to influence a bank. The U.S. Court of Appeals agreed that defendant was entitled to introduce such evidence on the issue of specific intent and reversed the conviction. Similarly, in 1995, former United Way executive Aramoy, charged with numerous counts of embezzlement, introduced neuroimaging evidence in support of his claim that he was suffering from "brain atrophy" and thus unable to satisfy the requisite intent requirement to commit embezzlement. Shortly after this evidence was introduced, he secured a favorable plea bargain (Chandrasekaran & Miller, 1995).

Some defendants, however, have not been as successful in demonstrating a lack of *mens rea* by appeal to neuroimaging evidence. For example, in *State v. Anderson* (2002), the defendant presented expert testimony, supported by neuroscience evidence, that brain-damage-induced depression and paranoia precluded him from being able to premeditate and deliberate in a manner sufficient to justify the charge of first-degree murder. The jury was not persuaded and found him guilty on all counts. Similarly, in *U.S. v. Mezvinsky* (2002), the court held that the defendant, who had been indicted on 66 counts of fraud and related offenses, was not entitled to introduce PET scan evidence in support of his claim that he was incapable of deception (the requisite *mens rea* for his charges).

Further, one of the first high-profile cases involving the use of neuroimaging to assert insanity occurred during the 1981 trial of President Reagan's would-be assassin, John Hinckley Jr. More specifically, an expert for the defense introduced Hinckley's CT scan, which depicted a "widening" of sulci in Hinckley's brain, in support of a diagnosis of schizophrenia. Further, the neuroradiologist for the defense testified that the degree of atrophy was abnormal and possibly indicated the presence of

organic brain disease, while another witness for the defense testified that the evidence of atrophy increased the statistical likelihood that the defendant was suffering from schizophrenia. The court admitted this evidence in order to give the jury all possibly relevant evidence bearing on cognition, volition, and capacity in considering the defendant's claim of insanity and the defendant was, subsequently found not guilty by reason of insanity. However, it has been argued that the use of a CT scan in the psychiatric diagnosis of schizophrenia lacks any real scientific justification (Taylor, 1982). Further, even if CT scans were valid and reliable tools for psychiatric diagnosis, Hinckley's brain scan did not show the enlarged ventricles that research indicates are the more highly correlated morphological feature of schizophrenia (Stone, 1984).

Likewise, in *People v. Weinstein* (1992), the defendant, accused of strangling his wife, successfully introduced PET scan images, which he asserted showed reduced brain function in and around an arachnoid cyst in his frontal lobe. The evidence was presented in support of the defense's theory that Weinstein was not responsible for his actions due to mental disease or defect. Shortly after the judge ruled the PET scan to be admissible, the prosecution agreed to negotiate a plea bargain for a reduced charge of manslaughter.

States may also provide juries with the option of returning a verdict of Guilty but Mentally Ill (GBMI) when the jury finds that the defendant suffers from a mental illness but does not find that he/she was insane. In fact, there are a number of reported cases in which it appears that the jury's GBMI verdict was based, in large part, on evidence of the defendant's FLD (see, e.g., *Ward v. Sternes* (2003).

4. Imposition of punishment

While there are few reported cases in which defendants have secured acquittals on the strength of neuroimaging evidence, defendants have enjoyed some measure of success in the context of sentencing. For example, such evidence has been introduced as an adjunct to support a plea for leniency or claim of mitigating circumstances. Indeed, in early 2004, MRI and PET scan evidence helped to defeat two separate death sentences for Simon Pirela. In April of 1983, and in a separate murder trial in May of 1983, the defendant received two death sentences. When the second death sentence was vacated (due to reversible error for prosecutorial misconduct) and resentencing ordered, defense attorneys introduced MRI and PET scans as evidence in support of mitigating factors of diminished capacity, brain damage, and mental impairment. The jury recommended unanimously that Pirela be resentenced to life in prison rather than executed (*Commonwealth v. Morales*, 1997). Similarly, in *McNamara v. Borg* (1991), PET scan evidence was introduced in support of the defendant's mitigation claim that he was suffering from schizophrenia. The defendant was sentenced to life imprisonment rather than execution. According to post-sentencing interviews, jurors acknowledged that they were significantly influenced by the neuroimaging evidence in their decision to spare the defendant's life. In addition, in *Hoskins v. State* (1999), the Florida Supreme Court vacated the defendant's death sentence and remanded the case for a new penalty proceeding so that the defendant would have an opportunity to present a PET scan showing a brain abnormality. More specifically, the court held that the failure to allow neuroimaging evidence at the sentencing phase was reversible error. In contrast, in *People v. Kraft* (2000), the defendant, after having been convicted of 16 counts of murder and other crimes, introduced PET scan images during his mitigation

case, which experts testified were consistent with obsessive-compulsive disorder. However, the jury was not moved by this evidence and sentenced the defendant to death. Likewise, in *People v. Holt* (1997), the defendant, convicted of murder, robbery, rape, and other crimes, introduced PET scan images and an EEG showing abnormalities in both temporal lobes and damage to the cingulate gyrus region of the brain. The jury was not persuaded by this evidence and sentenced the defendant to death.

5. Incompetence

When a defendant's FLD is severe or present alongside other mental impairments, it may form the basis for a finding of adjudicative incompetence (*State v. Hall*, 2001). Such individuals may not, for example, fully appreciate their legal situation, maintain motivation and attention when interacting with counsel, or be able to testify effectively or make sound judgments about their options (Redding and Frost, 2001). In addition, evidence of FLD has been used to challenge a defendant's competence to plead guilty or waive constitutional rights. For example, in *State v. Marshall* (2001), the defendant sought to withdraw his guilty plea, arguing that he was incompetent to enter a plea due to FLD and other serious mental disorders. The Supreme Court of Washington remanded the case for a competency hearing to determine whether the plea was entered voluntarily, given evidence of the defendant's significant mental impairment, including an MRI which revealed that "the decision-making area of his brain had shrunk significantly .... and frontal lobe damage that affects his ability to plan ahead, conceptualize the future and make reasoned decisions" (*Marshall*, 2001, pg.196-197. Similarly, in *U.S. v. Ward* (2002), the Illinois U.S. District Court found that the defendant's waiver of his Sixth Amendment right to testify was not a voluntary, knowing,

and intelligent waiver since FLD impaired his understanding of the important right he was waiving.

Evidence of FLD has also been used to argue that a defendant's confession should be suppressed because the Miranda rights waiver was not given knowingly, voluntarily, or intelligently (*People v. Wilson* (2000). However, these claims are seldom successful. Indeed, in *State v. Mears* (2000), the court held that despite FLD and other mental impairments, defendant's understanding was sufficient to establish a knowing, voluntary, and intelligent waiver of his Miranda rights.

6. Mitigation

Under the Supreme Court's decision in *Lockett v. Ohio*, (1978), all mitigating evidence must be heard in a capital case. Statutory mitigating evidence has been defined by the Federal Death Penalty Act to include impaired capacity defined as:

> The defendant's capacity to appreciate the wrongfulness of the defendant's conduct or to conform conduct to the requirements of law was significantly impaired, regardless of whether the capacity was so impaired as to constitute a defense to the charge (18 U.S.C. § 3592(a)(1) (2006).

Frontal-lobe dysfunction has satisfied a similar standard of impaired capacity under the Federal Sentencing Guidelines. Indeed, a district court found, based on the testimony of a psychologist, that defendant's frontal-lobe damage constituted "diminished capacity" (*United States v. Pineyro,* 2005, at 139).

For a jury to accept a mitigating factor, only one juror needs to find that the defendant established the factor by a preponderance of the evidence (*Simmons v. South Carolina*, 1994). This standard differs dramatically from the standard required for aggravating factors, which is a unanimous jury finding beyond a reasonable doubt. The

standard of proof for mitigating evidence is significant because of the high degree of deference that jurors, generally, give to expert witnesses. With only one juror misunderstanding the neuroimaging testimony of an expert, a mitigating factor will be established and thus be weighed against aggravating factors in the sentencing phase. Such a low standard places an increased burden on expert witnesses to be cautious when presenting neuroimaging testimony (Barth, 2007).

Further, a substantive obstacle in the path of a defendant seeking to introduce neuroimaging evidence is demonstrating impaired capacity at the time of the offense. As the definition of "impaired capacity" stands, the defendant must prove that he was unable to conform his actions to the requirements of the law when he committed the offense. Therefore, the effects of the brain dysfunction must be present during the commission of the offense for the mitigating factor to be satisfied. The significance of this obstacle diminishes when an anatomical brain defect is detected before the commission of the crime and after the brain is generally considered fully mature (Barth, 2007). If such a defect is detected during that time-frame, then the defendant has a strong argument establishing that his brain dysfunction existed when he committed the crime (United *States v. Llera Plaza*, 179 F.Supp.2d 464, 487 (E.D. Pa. 2001).

Neuropsychological testing

Although attempts to generate normative and potentially diagnostic brain imaging data are under way, the neuroimaging literature, at present, offers few findings specific enough to inform critical legal questions of volitional or cognitive impairment (Society of Nuclear Medicine Brain Imaging Council, 1996). In fact, complex mental phenomena such as judgment or intent will be especially difficult for researchers to isolate with brain

imaging since the neural effect of such phenomena are widely distributed and may vary greatly from person to person. Therefore, instead of relying, solely, on neuroimaging scans, clinician should obtain additional information about an individual's cognitive and psychological functioning from carefully conducted neuropsychological testing, since there is more normative data in neuropsychological assessment (Keefe, 1995).

Neuropsychologists are frequently called as expert witnesses in criminal cases to provide testimony concerning the cause and extent, if any, of cognitive impairment, psychopathology, and the possibility of malingering. However, the admissibility of such testimony is often challenged as being scientifically unreliable under evidentiary standards established by the United States Supreme Court. Although courts have yet to directly address the admissibility of tests and measures designed to assess malingering and frontal lobe dysfunction, there are a number of legal cases which illustrate the inherent problems associated with basing expert opinions on findings of neuropsychological test results. For example, the question of whether a plaintiff's neuropsychologist's method met the scientific reliability requirement for the purposes of admissibility in evidence was addressed in *Baxter v. Temple* (2008).

In *Baxter* the plaintiff alleged that she sustained permanent cognitive impairment as a result of exposure to lead paint and consequently retained an expert who performed two neuropsychological test batteries to determine whether she was suffering from brain damage as a result of lead paint toxicity. Dr. Bruno-Golden found that the plaintiff demonstrated a 20-point decline in her full scale I.Q. between the first test, administered in 2002, and the second test, given in 2004. Before trial, an evidentiary hearing was held on the defendant's motion to exclude plaintiff’s expert’s conclusions as being

scientifically unreliable. At the hearing, the plaintiff's expert testified that she employed a neuropsychological testing technique called the Boston Process Approach (BPA). A second expert, Dr. Sheehan, testified that the BPA is a flexible battery approach in which the clinician utilizes a collection of standardized neuropsychological tests to assess various brain functions.

The defendant challenged the reliability of the BPA methodology and the specific battery of tests chosen by Dr. Bruno-Golden as being scientifically unreliable. The defendant argued that the specific battery of tests had not been subjected to peer review and publication, had no known rate of error, and was not generally accepted in the scientific literature. *Baxter* contended that the individual subtests in the battery had all been tested individually and were generally accepted in the field of neuropsychology. Dr. Bruno-Golden conceded, however, that the particular battery of tests had never been tested, and the results of the BPA could not be independently verified because the methodology varies between neuropsychologists.

Following a six-day evidentiary hearing, the Superior Court precluded Dr. Bruno-Golden from testifying. The trial court found that the proffered testimony did not meet the requirements for the admission of expert testimony set forth by the United States Supreme Court in *Daubert*. In excluding the evidence, the court made specific findings of fact on each of the relevant *Daubert* considerations. Specifically, it ruled that the absence of peer review testing of the specific battery of subtests, the lack of a known potential error rate in the testing methodology, and the failure to demonstrate a general acceptance of this methodology required exclusion of the evidence. The New Hampshire

Supreme Court subsequently concluded that the exclusion of the neuropsychological testimony was in error (*Baxter*, 2008).

The exclusion of neuropsychological evidence based upon the failure to administer an accepted test battery is consistent with other courts that have reached similar conclusions. For example, in *State of Connecticut v. Griffin* (2005) a clinical psychologist was precluded from testifying as to defendant's competency based on a test battery that had not been peer reviewed, or generally accepted as scientifically valid. However, in *United States v. Eff* (2006), the court held that although the expert's opinion of insanity was not a reliable conclusion, the battery of tests administered to measure the defendant's cognitive abilities, including the WAIS-III and WCST, was reliable because the individual tests had been widely administered, had reasonable confidence levels, and could be repeated.

Jurisdictions are also split as to whether neuropsychologists may testify as to the cause of a defendant's alleged impairment. Although neuropsychologists are generally permitted to testify concerning the tests administered, the test results, and an interpretation of the results used to reach the conclusion, whether the neuropsychologist may testify on the issue of causation often turns on the state's statute concerning the scope of a psychologist's practice (see, e.g., *Minner v. American Mortgage & Guaranty Co.*, 2000). However, some jurisdictions appear to permit neuropsychologists to testify as to the cause of, for example, brain injury. Indeed, in *Rustenhaven v. American Airlines, Inc.* 2003, the expert was allowed to opine that the neuropsychological testing revealed that cognitive deficits had their genesis in a right frontal brain injury. Similarly, in *Bonner v. ISP Technologies, Inc* (2001), a neuropsychologist testified that defendant's

product caused the plaintiff permanent organic brain dysfunction; the defendant did not challenge the neuropsychologists' qualifications and the court of appeals refused to revisit the issue on appeal.

Although the reality of frontal lobe impairment and its effect on behavior and judgment is well accepted in the scientific community, the reliability of a diagnosis of frontal lobe dysfunction and evidence about how it may have contributed to a given offense may be open to *Daubert* or *Frye* challenges, particularly with respect to neuroimaging results. Indeed, neuroimages are not direct visualizations of the brain. Rather, they "simplify complicated data about the brain, but ... are mutable, constructed representations, far more similar to charts and line graphs than to photographs" (Reeves, et al., 2003, pg. 96). There are also, at present, no uniform or well-defined criteria for differentiating normal from abnormal imaging results, or for quantifying the extent of frontal lobe impairment in the context of violent behavior. Furthermore, as will be discussed in detail later, many neuropsychological tests designed to assess frontal lobe functioning, lack scientific validity to be relied upon as a sole measure of FLD. Given the consequences of a finding of FLD or a non-finding of FLD all evidence related to FLD in the context of violent and aggressive behavior must, therefore, have scientific validity and evidentiary reliability. This is especially crucial, in light of the following:

According to a recent study, mock jurors were more likely to find a defendant not guilty by reason of insanity when provided with neuroimaging showing FLD than when presented with clinical testimony alone (Gurley & Marcus, 2005). This singular persuasiveness of neuroimaging evidence is also illustrated in the dissenting opinion of a Florida Supreme Court Justice, who complained that the experts' conclusions, based

largely on the defendant's history and neurological examinations, were not based on "objective" testing "such as brain scans ... as differentiated from the experts' subjective conclusions" (*Crook v. State*, 2002).

Clearly, while neuroscience evidence ultimately cannot answer questions about criminal responsibility, the belief that neuroimages convey truth, significantly distracts from the evaluation of the limitations of the various tests, measures, and techniques currently used to assess FLD in the context of violent and aggressive behavior. Further, it may be argued that even if one suffers from FLD one still has the ability to restrain one's actions, because the mind almost always has veto power over what the brain decides. This phenomenon has been demonstrated through brain imaging studies that show that the body acts after the brain does; the mind has "the power to stop the operation" (Thompson, 2006, pg. 53). Therefore, until a direct and causal link between FLD and aggression has been scientifically established expert witnesses must educate the trier of fact about the gap between brain function/structure and the manifest behaviors, as well the limitations inherent in all currently available measures, tests, and techniques.

CHAPTER 2. THE ISSUE OF EVIDENTIARY RELIABILITY

In science reliability refers to the reproducibility of measurement while validity is defined as the extent to which a test measures what it claims to measure. In contrast, in law evidence is considered to be reliable if it proves what it purports to prove. Under *Daubert*, evidentiary reliability is construed as the "trustworthiness" of the data and "for scientific evidence *evidentiary reliability will be based on scientific validity"* (*Daubert*, 1993, pg. 2795; italics in original).

In the years since *Daubert*, the standards for admissibility at trial of expert testimony in general and scientific evidence in particular have become more demanding. In fact, reviews of recent cases and empirical studies of federal judges' and attorneys' practices indicate that judges are more likely to consider the admissibility of expert evidence prior to trial, to inquire more deeply into the reasoning and methodology that supports the expert opinions, and to limit or exclude such evidence from presentation at trial (Cecil, 2005). Furthermore, courts increasing skepticism concerning expert testimony and scientific evidence is apparent in many recent published cases. Consider the following:

In *U.S. v. Flaherty* (2008), the defendant, after having been convicted by a jury of conspiracy to commit securities fraud, mail fraud, wire fraud, and conspiracy to commit money laundering, filed a motion for a new trial arguing that recently discovered evidence showed the defendant to be mentally incompetent. More specifically, neuropsychological test results purportedly showed that she suffered from a low IQ and significant cognitive impairments. Defendant's new counsel argued that these mental defects are newly discovered and negatively affected defendant's ability to exercise

judgment, participate in her defense, conform her conduct to the law, and form the requisite intent to defraud.

The expert neuropsychologist had administered a comprehensive test battery and concluded that defendant's results were consistently low, falling in the range of borderline intelligence. For example, the tests showed that defendant had a full-scale IQ of 73. The expert also stated that defendant scored extremely low on tests that evaluate achievement, scoring in the borderline range for reading and characterized the neuropsychological profile as grossly abnormal. The expert further concluded that defendant is a naive individual with borderline intelligence, with superimposed cognitive impairments that extend beyond this limited IQ. The expert further opined that defendant is concrete and inflexible in her thinking and that persons with this level of intellectual challenge usually require some direction from others and typically function at lower-level jobs.

The Court concluded that this newly discovered evidence of mental incompetence, clearly produced for purposes of avoiding judgment, is biased and unreliable and, therefore, does not provide valid grounds for a new trial. More specifically, the Court reasoned that aside from the expert's inherent bias, his reports seem to possess several methodological weaknesses. For example, the tests failed to adequately take into account the fact that Ms. Flaherty is a Taiwanese immigrant whose native tongue is Mandarin, not English. Her poor performance on intelligence tests asking questions about American history and European literature and culture can thus be easily explained by her foreign upbringing. Additionally, defendant's low IQ of 73 is somewhat invalid because it is made up of both culturally weighted English language subtests as well as nonverbal subtests. Her verbal comprehension index was no doubt largely

affected by English and cultural factors. Review of the testing materials reveals that all of the tests, both verbal and nonverbal, were administered in the English language and made no accommodation for the fact that defendant was born and schooled in Taiwan. Therefore, these results should be treated with suspicion.

The court also contended that the expert's conclusions do not fit with the other evidence regarding defendant's behavior in this case. More specifically, defendant took an active role in managing the business entities, directed the corporation's promotional activities, and personally made several misrepresentations to investors in both the English and Mandarin languages. These facts do not harmonize with expert's picture of a "naive" individual who requires some direction from others and typically functions at lower level jobs. Defendant's active involvement in her legal defense also is at odds with the expert's conclusions. Ms. Flaherty has actively participated in this case from the outset. The Court observed her involvement during trial and throughout the other stages of the case. Her active role in the case does not support the picture of a borderline mentally incompetent individual. Consequently, defendant's motion for trial is denied.

Daubert offers no guidance as to what point an error rate exceeds what is acceptable for a reliability determination and the scientific community, likewise, has yet to establish an acceptable level of error for tests designed to assess FLD and malingering. However, procedures that have been deemed to have clinical utility in assessing FLD and malingering are those that have high specificity and reasonable sensitivity. Therefore, although a determination of FLD and malingering is, generally, not based on neuropsychological test results or on a single test score, the scientific validity and evidentiary reliability of each test must nevertheless be critically evaluated. In fact, it has

been contended that even if a test, measure, or technique is not being presented to the trier of fact for the purpose of alone influencing a judgment of FLD or malingering, it must meet the standard of the scientific validity. Clearly, a test, measure, or technique that fails to prove what it purports to prove when used in isolation cannot be deemed scientifically valid when used within a battery of tests or as part of a multimodal approach (Vallabhajosula & van Gorp, 2001).

Judging the reliability and validity, however, is difficult, at best. As noted by Saxe and Ben-Shakar (1994):

> Reliability and validity are complex, multidimensional concepts and encompass the methodological requirements for research that can assess scientifically based techniques…The underlying question is as follows: What constitutes a proper evaluation of a technique and the theory from which it is derived? It is not sufficient to demonstrate that a technique has been tested; the key question is whether the test is adequate and what generalizations are appropriate (p. 207).

Scientific validity and predictive accuracy

In general, most classification research has focused on four indices of predictive accuracy or scientific validity: sensitivity, specificity, positive predictive power (PPP), and negative predictive power (NPP). Sensitivity is the probability of a person with a certain condition or trait being picked up by a test while specificity is the probability that a person without a certain condition or trait is being identified by the test as not having that condition or trait. In other words, sensitivity is the proportion of true positives that are correctly identified by the test, while specificity is the proportion of true negatives that are correctly identified by the test. Positive predictive power (PPP) is the likelihood of a person identified by a test as having the condition or trait actually possessing that characteristic and negative predictive power (NPP) indicates the probability of a person

identified by a test as not having a certain condition or trait actually not having that characteristic (Butcher, 2002).[6]

Although the importance of these indices may vary depending on the type of prediction offered, it is arguable that in the case of malingering and/or frontal lobe dysfunction, PPP is the most clinically relevant index to consider. For example, erroneously classifying a genuinely impaired individual as a malingerer (false positive) may result in the individual being subject to prosecution despite neurocognitive impairment. The failure to identify malingering when malingering is present (false negative) is also problematic and may lead to a failure to prosecute a competent defendant. However, the latter risk seems the lesser of the two injustices since in most cases where actual malingering is not detected the consequences are not permanent.

In determining the ability of an actuarial model to predict future dangerousness, one would likely also want to optimize PPP. Similarly, in the case of risk assessment, the danger and damages from failing to detect potentially violent individuals represents a far greater harm than that inflicted by the unnecessary treatment and hospitalization of a nonviolent individual (Rosenfeld, Sands, & van Gorp, 2000). More specifically, while the involuntary hospitalization of nonviolent individuals is not a harmless loss, it is less likely to result in permanent consequences. There may exist other scenarios exist in which different indices may be the most appropriate to optimize; however, many researchers attempt to optimize all of these indices, essentially presuming that each is

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[6] See Table 1, which shows how to calculate the various accuracy indices (Vallabhajosula & van Gorp, 2001).

equally important (Rogers, 1997; Rosenfeld, Sands, & van Gorp, 2000;; Vallabhajosula & van Gorp, 2001).

The importance of considering base rates

The issue of base rates in discussing the efficacy of neuropsychological tests is a crucial one. Indeed, over a half-century ago, Meehl and Rosen (1955) described how base rates can impact the accuracy of clinical judgments in psychology, stating that the diagnostic utility of a test is based not only on the test's sensitivity and specificity but also on the base rate of the diagnosis in the population of interest. More specifically, it is well known that the predictive accuracy varies as the base rate varies but, to date, few studies have considered the influence of base rates on these various accuracy indices. However, doing so is important, especially in light of the existing literature, which suggests that, regardless of the setting, the base rate of, for example, malingering is unlikely to exceed 30% (Rosenfeld, Sands, & van Gorp, 2000; Vallabhajosula & van Gorp, 2001). Consider the following example:

With an original base rate of malingering of 48% the nonverbal subtest of the Validity Indicator Profile (VIP) was found to have 83% PPP, while the verbal subtest demonstrated 79% PPP. However, with a base rate of 30% the PPP of the nonverbal and verbal subtests decreased to 69% and 63%, respectively. Further, if a conclusion of malingering is based on both subtests being invalid, the VIP quite accurately detects malingerers with a 53% base rate (90% PPP). With base rates of 30% and 15%,

however, the PPP decreases to 78% and 60% respectively (Vallabhajosula & van Gorp, 2001).[7]

Since the diagnostic utility of a test is relative to the base rate of the diagnosis in the population of interest, the extensive use of tests in neuropsychological assessment makes knowledge of the base rates of various disorders and dysfunctions, thus, highly relevant. Unfortunately, to date, the base rates of various illnesses, disorders, and dysfunctions, in the population, has yet to be established.

The use of multiple tests

The true positive diagnostic accuracy rate (sensitivity) of psychological tests is generally positively related to the rate of false negative diagnoses (specificity), the prevalence of exaggeration for the claimed disorder, and the complaints of the individual. Consequently, it has been suggested that informed clinical practice requires that multiple sources of information are considered in the assessment of malingering, cognitive and/or psychological deficits, and/or mental disorders (Samuel & Mittenberg, 2005). While concerns that a single test will produce incorrect results have often been countered by assertions that the use of multiple tests will reduce the likelihood of false-positive findings, this assertion, however, may be inaccurate. Indeed, the use of multiple measures is often necessary and appealing, given the high rates of NPP associated with many tests (Samuel & Mittenberg, 2005).

The issue of cutoff scores

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[7] These accuracy values were calculated using data from the VIP validation study.

Classification accuracy numbers are specific to the cutoff scores used in each study. More specifically, widely applicable cutoff scores have been difficult to establish because: (1) optimal cutoff scores vary across studies, and (2) optimal cutoff score are defined as those that best balance specificity and sensitivity. However, as the consequences of Type I and Type II classification errors are likely to vary across settings, optimal cutoff scores also will vary, depending on which type of error is more important to minimize in each setting. Therefore, clinicians who choose to use a different cutoff score in order to minimize a specific type of error may obtain different classification accuracy rates (Baer & Miller, 2002). This point is illustrated by the following:

Horowitz and colleagues (2008) examined the utility of the Halstead-Reitan neuropsychological test battery to predict neuropsychological impairment in adults. Using a cutoff score of 9, as recommended by Reitan and Wolfson, the screening battery had excellent specificity but only fair sensitivity for identifying individuals with neuropsychological impairment. With a cutoff score of 8, however, both the sensitivity and specificity of the screening battery increased. From these findings the authors concluded that optimal cutoff scores for the screening battery may vary with different populations. Similarly, in assessing the ability of the Digit Span (DS) score of the WAIS-III in detecting malingering, the DS recommended cutoff of 7 resulted in a sensitivity of 62% and a high false positive rate (23%). Dropping the cutoff to 6 raised the specificity to 93% but decreased the sensitivity to 45% (Babikian, Boone, Lu, & Arnold, 2006).

As the foregoing indicates, sensitivity, specificity, PPP, and NPP values can be manipulated. Indeed, depending on the cutoff score used by a clinician, tests that lack

sufficient predictive accuracy with one cutoff score may be deemed highly accurate if the cutoff score is changed. This is troublesome for a number of reasons. First, many tests were validated using a specific cutoff score and many test developers have, in fact, provided recommended or "optimal" cutoff scores. Changing these cutoff score to maximize the predictive accuracy may, arguably, invalidate the test results. Second, depending on the sophistication of the cross-examination of an expert witness, opinions based on test results that have employed cutoff scores other than those recommended by the test developers may be open to a *Daubert* challenge. Third, standardized tests gain their power from their standardization. More specifically, reliability, validity, sensitivity, specificity, and other accuracy indices have emerged from an actuarial base: a well-selected sample of individuals providing data in response to a uniform procedure in a uniform condition. When the instructions, test items, administration, or scoring are changed, attempts to draw on the actuarial base become questionable (Pope & Vasquez, 2005). Finally, unless a uniform methodology is used for the assessment of FLD in the context of aggressive and violent behavior, the exact relationship between FLD and aggression will remain uncertain.

CHAPTER 3. ETIOLOGY OF FRONTAL LOBE DYSFUNCTION

Gaining insight into the brain is at the very core of understanding what it is that makes us so uniquely human but, as Watson (1996) has pointed out, "bridging the gap between understanding the brain and understanding the mind and behavior continues to elude us" (p. 544). Violent and aggressive behavior has been, and continues to be a significant, social problem. It is, therefore, not surprising that for centuries a substantial amount of research has been undertaken to determine the likely cause of such behavior. In more recent years, in particular, researchers have sought to determine the effects of functional and structural brain abnormalities on behavior, and findings from these studies have provided compelling evidence for an association between brain dysfunction, and aggression. Some of the most striking findings, in fact, have come from studies of death row inmates. More specifically, of the fifteen death row inmates examined by Otnow-Lewis and colleagues (1986) all were found to have a history of severe head trauma, five displayed major neurologic impairment and seven others demonstrated neurological soft signs (e.g., involuntary movements in the absence of gross neurological damage). Similarly, in a recent study of juveniles condemned to death, every subject exhibited signs of frontal lobe dysfunction on neuropsychological tests, neurological examination, or both (Otnow-Lewis, Yeager, Blake, et al., 2004).

While the tendency to behave violently and aggressively across a variety of situations could be interpreted as evidence of brain dysfunction, it may also simply be attributed to personality pathology. Clearly, evidence of a high level of brain dysfunction, in general, and frontal lobe abnormalities, in particular, among criminal

offenders does not prove a causal connection between brain dysfunction and criminality. There are, however, theoretical reasons for predicting such a connection.

Speculations regarding the relationship between neuroanatomical aspects of aggressive and violent behavior began at the end of the 18th century with Franz Joseph Gall. His observations eventually involved into phrenology, a theory that assigns specific mental functions to specific topographical regions of the skull (Bassarath, 2001). After centuries of research, the exact neuroanatomical region responsible for criminal or violent behavior still remains somewhat elusive. However, with the development of new structural and functional imaging techniques it is now possible to ascertain regional brain dysfunction with much higher accuracy and sensitivity than was previously possible.[8] In fact, converging evidence from numerous studies of structure and function, as well as from studies employing neuropsychological and neurological tests and measures, have suggested that abnormal prefrontal circuitry, in particular, is likely to be involved in antisocial behavior (Filley, Price, Nell, et al., 2001).

As previously noted, brain function and structure are under environmental and genetic control, thus, human behavior is determined by a combination of these influences. More specifically, behavior is variably governed by the interaction of factors as diverse as genetic endowment, early life experiences, acquired brain damage, learned patterns of

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[8] Structural imaging techniques consist of computerized tomography ("CT") and magnetic resonance imaging ("MRI"). These techniques look at brain architecture and are specifically designed to assess the presence or absence of gross abnormalities, such as brain tumors. Functional imaging techniques consist of functional magnetic resonance imaging ("fMRI"), single photon emission tomography ("SPECT"), and positron emission tomography ("PET"). These techniques add the dimension of live neural activity and are used primarily to assess regional blood flow, glucose metabolism, or to study neurotransmitter and neuroreceptor biochemical systems. Functional imaging studies can be done under activation or while the subject/patient is resting. However, only fMRI and PET generate quantitative or semi-quantitative data.

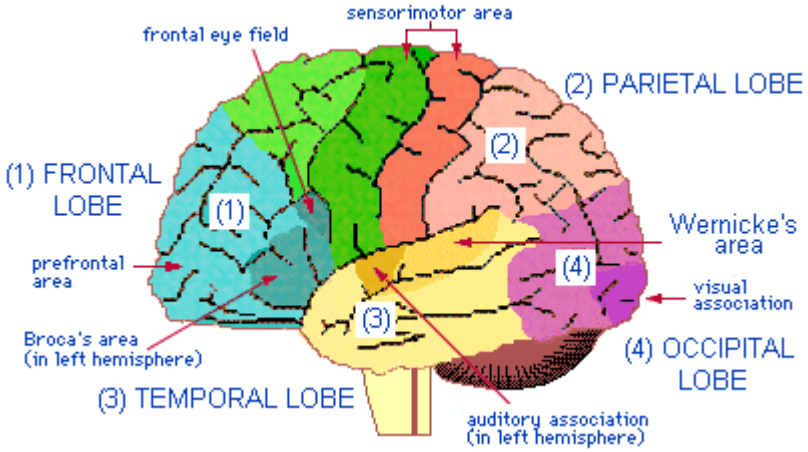
behavior, and situational contingencies. Violence and aggression, like all other behaviors, however, ultimately derive from the normal or abnormal operations of the brain. Indeed, numerous research studies have found high rates of neuropsychiatric abnormalities in individuals with violent and criminal behavior, suggesting a relationship between aggressive dyscontrol and brain injury involving, in particular, the frontal lobes. These findings have led to the assertion that evidence of brain-behavioral impairment may mitigate or excuse criminal conduct (Filley, Price, Nell, et al., 2001; Martell, 1992). Frontal lobe dysfunction in particular, has been invoked to explain the behaviors of defendants charged with, or convicted of, violent crimes (Brower & Price, 2000; Pincus, 1999).

Aggressive and violent behavior has also been associated with decreased glucose metabolism and cerebral blood flow in imaging studies of murderers, violent psychiatric patients, and criminal offenders, abnormalities which may be independent of major mental disorders, current medication, or substance abuse. Therefore, since this dissertation specifically focuses on the relationship between aggression and FLD all variables known to contribute to aggressive behavior and FLD must be considered in the evaluation of FLD.

Anatomy of the Frontal Lobes

Aggression and other emotions are regulated by a complex neural circuit that involves several cortical areas, including the frontal lobes.

Figure 1. Illustration of the Human Brain



(http://webschoolsolutions.com/patts/systems/speech.htm)

The frontal lobes are the area of the brain anterior to the central sulcus. Directly in front of the central sulcus is the premotor and primary motor cortex, and anterior to these areas are the prefrontal cortices, areas of the brain which are larger and most highly developed in the human brain (Petrides & Pandya, 2002). This is the region of most interest for cognitive control processes (executive functions) and violent and aggressive behavior.

It has been well established that the prefrontal cortex has several architectonically distinct regions. These connections from the prefrontal cortex to other cortical and subcortical regions are particularly important since they have been observed to involve two anatomically and functionally distinct systems (Petrides & Pandya, 2002). The first system, which mediates emotional tone, is ventrally located and involves the orbital surface of the frontal lobes as well as paralimbic regions. The second system mediates the sequential processing of sensory, spacially-related, and motivational information

through a dorsolateral stream, which involves the dorsolateral and medial areas of the frontal lobes as well as interconnections with the cingulated gyrus and posterior parietal lobe. In summary, the frontal lobes are the site where these systems, involving external sensory information and internal cognitive and emotional responses, are integrated and processed to modulate motivation and facilitate motor responses (Lichter & Cummings, 2001).

A pattern of anatomical and functional duality has also emerged from studies examining the connections between the prefrontal cortex and subcortical structures, in particular the basal ganglia. The basal ganglia is a subcortical system that is important in regulating and coordinating cortically-originating movement and consist of the striatum, globus pallidus, substantia nigra, and subthalamic nucleus. Efferent projections from functionally related areas of the prefrontal cortex converge upon discrete areas of the striatum, which is known to share functional properties (Lichter & Cummings, 2001). More specifically, the dorsolateral system of the frontal lobes connects to the dorsolateral caudate nucleus, while the ventral system maps onto the ventromedial portion of the caudate and adjacent portions of the nucleus accumbens. Information processed by the cortex, therefore, is received and processed by the striatum in a way that maintains partial separation and specialization of functional domains (Lichter & Cummings, 2001).

Middleton and Strick (2002), extended the neuroanatomy of frontostriatal circuits by including seven categories of circuits based in research findings in primates, but only four of these circuits are of relevance here. The four main circuits of interest here are those that involve projections between the basal ganglia and the following regions of the prefrontal cortex: (1) dorsolateral; (2) lateral orbitofrontal; (3) medial orbitofrontal; (4)

and anterior cingulate. As the foregoing suggests, the prefrontal cortex appears to be a site of convergence for information from distinct yet overlapping circuits that are involved in motivation, cognitive processes, emotion, and motor output, and recent studies have supported the existence of four distinct circuits connecting the prefrontal cortex to subcortical regions (Mesulam, 2002).

Prefrontal behavioral syndromes

Three distinct behavioral neurological syndromes have been observed following injury to certain areas of the prefrontal cortex:

1. Damage to the dorsolateral region may result in a "frontal abulic" syndrome, which is characterized by loss of creativity and initiative, a tendency towards emotional apathy and flat affect, and reduced ability to concentrate (Mesulam, 2002). Fuster (1997) noted that a disruption of "intensive and selective" attention is at the forefront of this syndrome, which impairs the ability to focus or direct general arousal upon a particular internal or sensory experience (p. 172). Cognitively, these symptoms manifest as the traditional "dysexecutive syndrome" which involves problems with working memory, verbal fluency, planning, perseveration, and temporal organization of behavior (Fuster, 1997).

2. Damage to the orbitofrontal region may result in a "frontal disinhibition" syndrome, which is characterized by deficits in the "exclusionary" aspect of attention (Mesulam, 2002; Fuster, 1997, p. 174). More specifically, individuals with this disorder have difficulty suppressing information from internal and external stimuli. The result is too much drive and behavioral excesses characterized by impulsivity with little insight, foresight, or ability to learn from experiences. Indeed, these individuals

show impaired moral judgment and disregard social conventions, and their affect is generally euphoric, with irritability, paranoia, and contentiousness. Cognitively, individuals with frontal disinhibition syndrome have problems with focused attention (Fuster, 1997).

3. The third syndrome, referred to as "akinetic mutism, may result from damage to the medial/cingulate cortex (Fuster, 1997; Pennington and Ozonoff, 1996). This disorder is poorly defined but is presumed to involve apathy and deficits in the ability to initiate speech and other spontaneous behaviors. More specifically, too much anterior cingulate activity may be associated with tic-like and obsessive-compulsive symptoms, while too little activity has been linked to depression and diminished self-awareness. Cognitively, the anterior cingulated cortex is important for motivation, initiation, and response selection (Devinsky, Morrell, & Vogt, 1995).

It has also been suggested that many of the functional changes that are observed following frontal lobe damage parallel the behavioral sequelea of a number of psychiatric disorders, providing a conceptual connection between psychopathology and frontal lobe circuits (Lichter & Cummings, 2001). Indeed, clinical parallels can be drawn for each of the three neurological prefrontal syndromes. The dorsolateral-frontal abulic syndrome resembles the symptomatology of Attention-Deficit-Hyperactivity Disorder (ADHD), as well as depression to some degree. The orbitofrontal-disinhibition syndrome resembles antisocial personality disorder (APD) and mania. Finally, the cingulated-akinetic mutism syndrome resembles the symptoms of major depressive disorder, while excessive cingulated activity is analogous to symptoms of anxiety disorders (Lichter & Cummings, 2001).

The frontal lobes and aggression

Society draws a clear moral and legal distinction between the consequences of actions presumed to be under an individual's volitional control and those presumed to be outside such control. Volitional control, however, implies more than conscious awareness; it implies the ability to anticipate the consequences of one's actions, the ability to decide whether a specific action should be taken, and the ability to choose between inaction and action. At a cognitive level, the capacity for volitional behavior depends on the functional integrity of the frontal lobes and the capacity for restraint depends, in particular, on the OFC (Goldberg, 2001).[9]

Luria viewed the frontal lobes as the controlling and directing source of the brain; the place where awareness is translated into action (Hall, 1993). In fact, the frontal lobes are considered to be a key in regulating the active state of the organism, in deciding what to attend to and in switching or sustaining attention when needed (Golden, Jackson, Peterson-Rohne, & Gontkovsky, 1996). As previously discussed, the frontal cortex is extensively connected to the limbic system as well as to cortical association regions, thereby receiving both emotional and higher-order information. More specifically, material from all sources (e.g., internal and external, conscious and unconscious) is integrated into ongoing activity so that behaviors can be modulated to satisfy drives within the constraints of the internal and external environments (Golden, et al., 1996).

Based upon a constant monitoring of internal needs and external demands and possibilities, the frontal lobes control the essential elements of intentions. Planning and

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[9] It is this region with is most often the subject of study and frequently implicated in aggressive and violent behavior.

programming of complex activity is required to enact these intentions, and to provide continuity and coherence of behavior across time. Further, even the most simple of intentions requires translation into action, including simply starting or stopping specific behaviors, thus, plans must be implemented and progress toward goals monitored so that ongoing adjustments can be made (Golden, et al., 1996).

Damage to the frontal systems results in consequences that are diverse, multifaceted, and often catastrophic. Prefrontal lobe damage frequently produces deficits in motivation or "drive," resulting in inertia and apathy sometimes labeled "pseudodepression" (Kwentus, Hart, Peck, & Kornstein, 1985). Indifference and shallowness are key features, resulting in a reduced capacity to feel or express the normal range of human emotions. A loss of interest in social interactions, when coupled with a lack of concern regarding the consequences of social behavior, can result in lewdness with a loss of social graces, and inattention to appearance and hygiene (Silver & Yudofsky, 1987). Problems in maintaining focus contribute to these difficulties. Marked emotional instability is also common. Irritability may quickly give way to euphoria, and increased extraversion is frequently reported (Kandel & Freed, 1989). Victims of prefrontal injury may behave in a childlike and selfish manner, resulting in features often labeled "pseudosociopathic" (Kwentus et al., 1985).

Individuals with frontal lobe impairment may also be intrusive, boisterous, speak loudly with free use of profanities, and engage in elevated risk-taking, indiscriminate eating, and unrestrained drinking. In short, basic drives and emotions are poorly regulated and the individual's behavior is highly impulsive (Silver & Yudofsky, 1987). Further, since the prefrontal cortex engages in temporally oriented programming to accomplish

tasks, damage to it often results in disorganization, impaired problem-solving, and an inability to anticipate consequences (Kandel & Freed, 1989). These problems can be severe, even when coupled with a normal IQ (Lezak, 1995). Even if the individual has the capacity to plan, deficits in behavioral initiation may result in the commonly observed breakdown between stated intentions and actual behavior. Indeed, many patients display diminished spontaneity and initiative, reduced productivity, and diminished verbal output. Frontally injured individuals also often lack the ability to adequately appraise progress toward intended goals. More specifically, they may fail to recognize their mistakes and their impact on others, and fail to properly evaluate social situations (Lezak, 1995).

Individuals with frontal lobe impairment may also experience difficulty adapting to changing circumstances, even when their ability to monitor is intact and they intend to be flexible in their response. Such individuals often get locked into behavioral or mental patterns, resulting in perseveration (i.e., maladaptive repetition or persistence of a behavior beyond its useful span). Implicated in these difficulties is the inability to use knowledge to regulate or rapidly adjust behavior based on external cues. Furthermore, individuals with frontal lobe impairment often display a concrete attitude in which events are taken at their face value, and the individual is unable to separate him/herself from the surroundings (Lezak, 1995).

As previously noted, the OFC, in particular, is presumed to contribute to aggressive and violent behavior. To be more precise, the OFC receives highly processed sensory information concerning a person's environmental experiences and is hypothesized to play a crucial role in mediating behavior based on social context (Duffy

& Campbell, 1994). The OFC is also plays a role in the perception of social signals, such as facial expressions of anger. Indeed, Blair and colleagues (1999), using PET scans, assessed 13 male volunteers as they viewed static images of human faces expressing varying degrees of anger and found that increasing the intensity of angry facial expressions was associated with enhanced activity in participants' anterior cingulated cortex and the OFC. Individuals with OFC damage also tend to exhibit poor impulse control, aggressive outbursts, verbal lewdness, and a lack of interpersonal sensitivity, which may increase the probability of sporadic so-called crimes of passion and encounters with the legal system (Duffy & Campbell, 1994). While research has led to an association between frontal lobe impairment and aggressive and violent behavior, there is also clear evidence that the problems associated with frontal lobe dysfunction are not solely restricted to damage to the frontal lobes. As Goldberg (2001) observed "...frontal lobe dysfunction does not always signify a frontal lobe lesion. In fact, in most instances it probably does not. Instead, it is a remote effect of a diffuse distributed or distant lesion" (pg. 116).

Perhaps the most notable behavioral effect of frontal lobe dysfunction is personality change. Frontally damaged individuals often display marked apathy, tactlessness, impulsivity, irritability, and the inability to empathize with the feelings of others. Some researchers refer to "emotional blunting" to describe the apathetic attitude associated with frontal lobe dysfunction (Lezak, 1995). On the other end of the spectrum, frontal lobe dysfunction can cause pseudopsychopathic or disinhibited behavior. Because the frontal lobes regulate the ability to reason, to control impulses, and to make socially responsible judgments, damage to the frontal lobes is presumed to

be a significant cause of aggressive and violent behavior. More specifically, if those areas fail to function properly, an individual may act inappropriately and/or impulsively. The associated inability to act in a civilized manner, thus, can result in increased criminality (Miller & Cummings, 1999).

Executive functioning

Executive functions (EF) have historically been associated with activity in the frontal or prefrontal cortex. These historical underpinnings for the relationship between the frontal lobes and EF come from early observations of seemingly different effects following frontal lobe damage (Pennington & Ozonoff, 1996). More specifically, while the disrupted processes appear to be quite different from one another, they can all be understood as involving dysfunction in goal-directed activity that could not be attributed to deficits in more basic cognitive processes. Consequently, it is presumed that the frontal lobes are involved in "supervisory" or "executive" functions (Pennington & Ozonoff, 1996).

Many behavioral deficits resulting from damage to the prefrontal region have been associated with a disruption of executive functions. The term executive function has been used to describe those higher-level cognitive skills that allow an individual to use mental and physical resources effectively in unstructured or novel situations. Although various definitions of EF have been provided, there is significant overlap among them. For example, Shallice (1998) suggested a supervisory system responsible for negotiating advantageous disruptions of the routine selection of routine operations. Other authors have included inhibition of responses as a component of EF, but broadened the definition to include the formation of a strategic plan of action and relevant sequences of steps, and

the formation of mental representations of tasks including relevant information in memory and desired goal-state (Welsh & Pennigton, 1988). In summary, executive functions are comprised of four principal neuropsychological components: (1) goal formulation, (2) planning, (3) carrying out goal-directed behavior, and (4) the ability to monitor effective performance.

As previously noted, a diverse set of functional deficits has been attributed to impairment of EF. Indeed, individuals with executive dysfunction have been shown to have difficulties initiating appropriate actions, setting reasonable goals, regulating attentional resources, planning and organizing behavior, inhibiting inappropriate behavior, monitoring their own behavior, and shifting between activities (Ylvisaker & Feeney, 1998; Fuster, 1997). Furthermore, it has been suggested that aggressive individuals score in the impaired range on tests of executive functioning. There remains an ongoing debate regarding whether executive functions are, in fact, regulated by the frontal lobes, leading to ambiguity of definition (Miyake, Friedman, Emerson, et al., 2000; Welsh, 2002). Nevertheless, it is almost impossible to find a discussion of executive dysfunction that does not make reference to frontal lobe abnormality and in parallel fashion there is rarely a discussion of frontal lobe impairment without a reference to executive dysfunction (Alvarez & Emory, 2006).

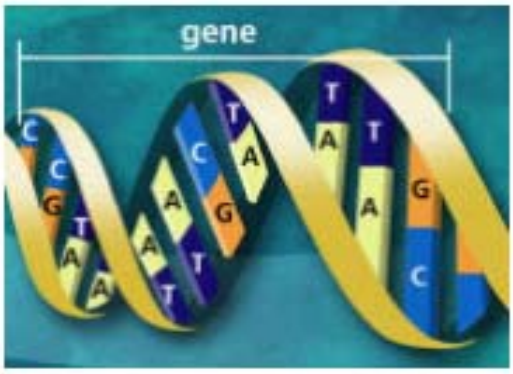
Other variables known to contribute to aggression and violence

1. Genetics

The possibility of a genetic contribution to aggressive and violent behavior has received considerable attention. However, the extent to which genes play a causative role

in aggression and violence is unclear, and the results of genetic research are prone to complexities in interpretation because certain types of findings encourage stereotyping.

Figure 2. Picture of the Double Helix



(Drawing courtesy of the US Department of Energy (http://www.wonderquest.com/gm-genes-insect.htm).

Numerous genetic studies have estimated the heritability for many behaviors to be in the range of 50% (Plomin, 1990). This statistic raises the possibility of heritable influences on aggression and violence; however, no gene for human violence has yet been discovered. Available evidence from molecular genetics instead suggests that multiple genes may interact to predispose individuals to violent behavior. For example, observations in mouse knockout models have suggested that targeted disruptions of single genes can induce aggressiveness in male mice and diminish nurturing in female mice (Brown, Ye, Bronson, et al., 1996; Nelson, Demas, Huang, et al., 1995). Furthermore, there has been evidence relating aggression in animals and human beings to genes regulating central serotonin metabolism (Cadoret, Leve, Devor, et al., 1997). Thus, a variety of genes may interact to alter neurotransmitter function and predispose individuals to violent and aggressive behavior.

Although, genetic studies have shed some light on the genesis of violence, genetics operate at the population level and cannot make predictions about an individual's propensity to violence. Moreover, these studies typically provide only an estimate of the relative influence of genetic factors on a given behavior, which implies that other factor may also contribute to this type of behavior. Further, the precise extent to which genetics contribute to violent and aggressive behavior remains largely unknown. Perhaps most importantly, however, we must recognize that genetic influences on violence and criminality do not necessarily signify a dire fate. Even if the heritability for violence is 50%, factors, such as neuropsychological and/or neuroanatomical deficits, may also likely play an equally important role in the etiology and expression of aggression and violence (Cadoret, Leve, Devor, et al., 1997).

2. Neuropsychiatric variables

Our knowledge of aggression and violence in humans is largely derived from studies of individuals who have committed violent acts. These studies use the lesion method of behavioral neurology to find associations between structural brain damage and a behavioral pattern; as such they do not consider neurochemical aspects. Although there is no violence center in the brain, a number of different brain structures, aside from the frontal lobes, and various neuropsychiatric disorders are thought to play preeminent roles in the etiology of aggressive and violent behavior.

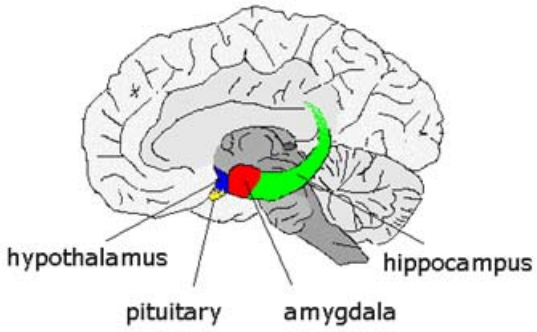
From an evolutionary perspective, aggression is a response to a potential threat or provocation across a variety of species and seems to be an inborn response tendency. Human beings in particular have evolved higher-order cortical centers that serve to suppress the emergence of more primitive forms of aggression when these are deemed

inappropriate. To understand the neurobiological basis of aggression, an understanding of both the cortical inhibitory mechanisms and the more primitive limbic systems involved in the generation and modulation of aggression is required (Siever, 2002).

A. The limbic system

The limbic system consists of a group of structures around the upper brainstem that are widely regarded as the anatomic substrate for many aspects of emotion. The hypothalamus is a critical component of the limbic system because of its role in the autonomic and endocrine systems that participate in the expression of emotion. Because the limbic system is intimately connected to the temporal lobe, the term temporolimbic has often been used to reflect this close neuroanatomic relation. Violent and aggressive behavior is presumed to arise from the operations of the limbic system under certain circumstances, with the amygdala being the structure most often implicated. Indeed, violent behavior has been observed in patients with limited amygdala and hippocampal involvement (Tonkonogy, 1991).

Figure 3. Drawing of the Limbic System

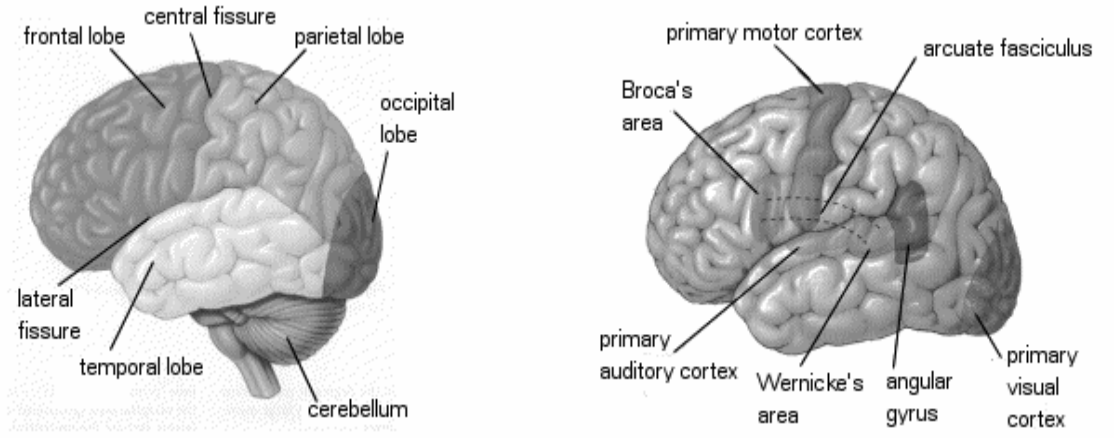


The part of the limbic system shown is that which is along the left side of the thalamus (hippocampus and amygdala) and just under the front of the thalamus (hypothalamus) (webspace.ship.edu/cgboer/limbicsystem.html).

B. The temporal lobes

Temporal lobe dysfunction has been demonstrated by episodes of exaggerated and/or unprovoked anger, intellectual impairment, auditory and/or visual hallucinations, delusions, and receptive language impairment. This type of episodic dyscontrol is most commonly associated with damage to the medial portion of the temporal lobes, which contain limbic system structures important for regulating emotion and behavior (Miller, 1990).

Figure 4. Ilustration of the VariousBrain Regions



(www.thebigview.com/mind/brain.html)

The clinical presentation of episodic dyscontrol varies. In severe cases, aggressive behavior can appear as unprovoked outbursts that are poorly organized in nature and directed at the nearest person or object, while in less severe cases outbursts appear organized and clearly directed against the source of the irritation. In general, however, aggression following temporal lobe damage involves a loss of behavioral control that is not confined to particular situations, times, and individuals, and occurs with no premeditation and little provocation (Barrett, 1993; Miller, 1990). In summary, aggression resulting from temporal lobe impairment, unlike aggression resulting from

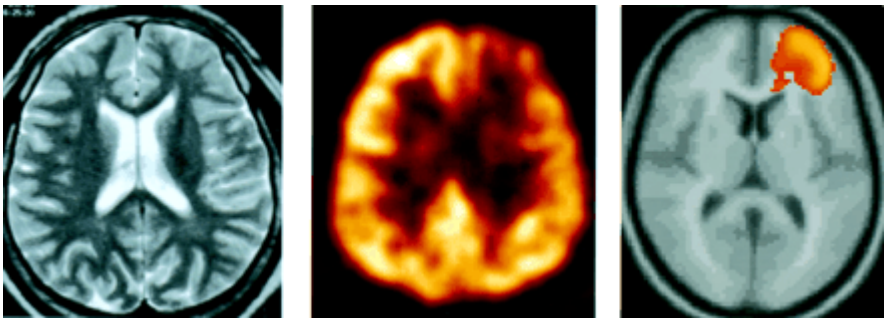
frontal lobe impairment, does not have clear antecedents or goals that frontal lobe aggression appears to demonstrate. It has been suggested that damage to medial temporal lobe structures may reduce aggression (Geshwind, 1973), however, functional imaging studies have reported reduced glucose metabolism and blood flow in the medial temporal lobes of murderers pleading not guilty by reason of insanity and in violent psychiatric inpatients (Amen, et al., 1996; Raine, et al., 1997, Seidenwurm, et al., 1997, Wong, et al., 1997; Volkow, et al., 1995).

C. Epilepsy

Episodic dyscontrol has also been associated with some forms of epilepsy.[10] Epilepsy can be present with or without identifiable neuropsychological correlates and with or without EEG abnormalities during routine testing. The disorder can be associated with a clear etiology (e.g., head trauma or tumor) or may have no clearly identifiable cause. Individuals with epilepsy suffer from a chronic condition characterized by recurrent seizures. The ictal event of a seizure is the actual seizure itself, while the nonictal periods are usually classified as preictal, postictal, and interictal.

Figure 5. Scans showing Frontal Lobe Epilepsy

[10] Epilepsy is a disorder characterized by sudden surges of disorganized electrical impulses in the brain. It is the most common chronic neuropsychological disease affecting the general population.



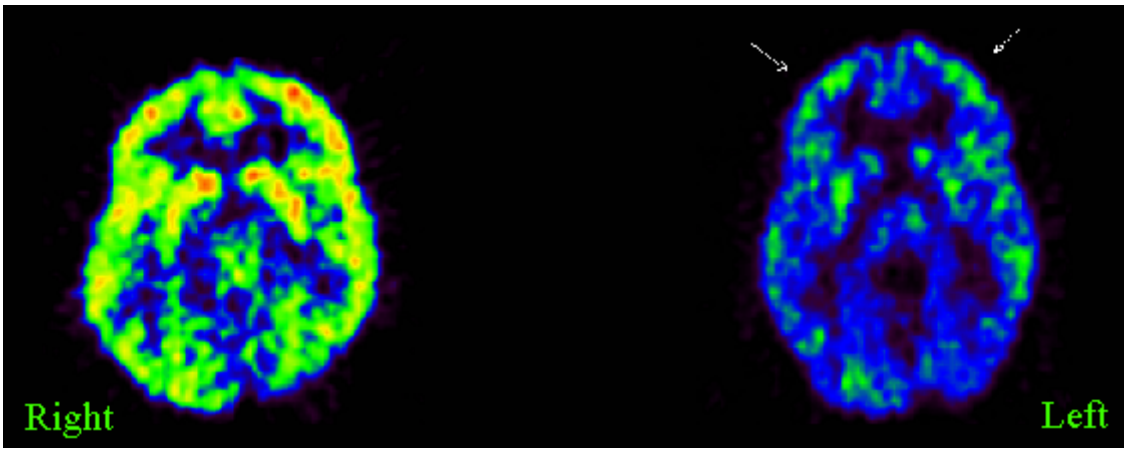
(From left to right, brain MRI findings, of a patient with frontal lobe epilepsy, were normal. An ^{18}F-FDG PET scan, however, shows decreased metabolism in left frontal lobe, as did SPM (statistical parametric mapping) (Kim, Lee, Lee, Chung, et al., 2000).

Episodic violence associated with epilepsy can appear during one or more of the previously mentioned epileptic phases. The preictal, or prodromal, phase is a period of minutes, hours, or even days prior to the onset of a seizure. Aggression seems to be a relatively rare occurrence during this stage. More common are nonspecific psychological changes, such as anxiety, irritability, depression, or changes in behavior (Fenwick, 1989). Secondly, although relatively rare, aggression during the ictal phase is characterized by acts that may be highly coordinated, but which occur in a confused mental state, and are inappropriate to the situation (Elliott, 1992; Fenwick, 1989). The proposed link between epilepsy and aggression has been presumed to be resulting from permanent changes in personality functioning produced by the seizure activity itself, but this has not been supported by the research to date, which indicates that such individuals are no more likely to be violent than other groups (Elliott, 1992; Jones, 1992; Mendez, Doss, & Taylor, 1993). It has, however, been shown that frontal lobe metabolic values, assessed with PET, are strong predictors of executive functioning in patients with epilepsy (McDonald, Swartz, Halgren, et al., 2006). Additionally, neuropsychological testing with the Wisconsin Card Sorting Test, which is presumed to be highly sensitive to

frontal-lobe-mediated executive dysfunction, has revealed poor performance in patients with temporal lobe epilepsy (Hermann, Wyler, & Richey, 1988).

D. Major depressive disorder (MDD)

Figure 6. Scan of Clinically Depressed Patient



(A clinically depressed patient (right) compared to a matched control (left). Blue represents less activity and red represents more activity) (http://www.musc.edu/fnrd/petdep.htm).

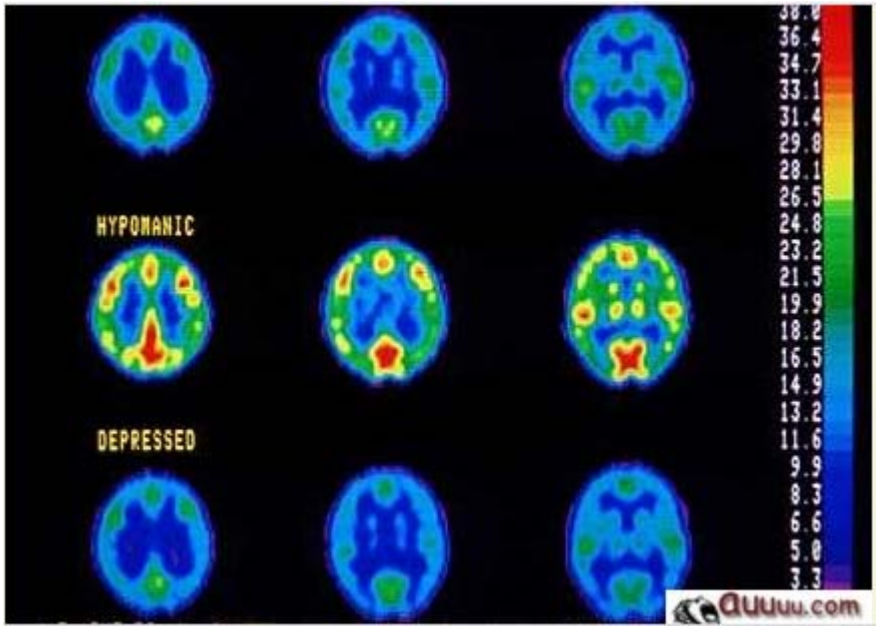
Major depressive disorder (also known as clinical depression, major depression, unipolar depression, or unipolar disorder) is a mental disorder characterized by a pervasive low mood, low self-esteem, and loss of interest or pleasure in normally enjoyable activities (DSM-IV-TR, 2000). Involvement of frontal lobes in primary depression has been demonstrated with functional neuroimaging neuropsychological studies (Baxter, Schwartz, Phelps, Mazziotta, et al., 1989; Starkstein & Robinson, 1998). In fact, executive abnormalities have been consistently found in studies on depressive disorders, with stronger results apparent with more severe pathology and these neuropsychological disturbances have been correlated with reduced blood flow in the mesial prefrontal cortex (Bench & Friston, 1993; Starkstein & Robinson, 1998). Further, in tests demanding executive functioning, the cingulate cortex and striatum could not be

activated in patients with MDD (Elliott and Baker, 1997). Likewise, structural changes have been identified in the cingulate gyrus and white matter of the orbitofrontal and prefrontal cortex, including smaller orbitofrontal cortex volumes in young adults and in geriatric patients with MDD (Bremner, Vithilingham, Vermetten, Nazeer, et al., 2002; Lai, Payne, Byrum, Steffens, et al., 2000; Taylor, Steffens, McQuoid, Payne, et al., 2003). Neuropathological studies have also documented structural cortical changes in frontal lobes of depressed patients, such as decrease in cortical thickness (Rajkowska, et al., 1994).

E. Bipolar Disorder (BD)

Bipolar disorder is a psychiatric diagnosis that describes a category of mood disorders defined by the presence of one or more episodes of abnormally elevated mood, clinically referred to as mania or, if milder, hypomania. Individuals who experience manic episodes also commonly experience depressive episodes or symptoms, or mixed episodes in which features of both mania and depression are present at the same time. These episodes are usually separated by periods of "normal" mood, but in some individuals, depression and mania may rapidly alternate, known as rapid cycling type BD. Extreme manic episodes can sometimes lead to psychotic symptoms such as delusions and hallucinations (DSM-IV-TR, 2000).

Figure 7. PET Scan of Patient with Bipolar Disorder



(PET scans of person with bipolar disorder show the subject shifting from depression (top row), to mania (middle row), and back to depression (bottom row). Blue and green indicate low levels of activity, while red, orange, and yellow indicate high levels of activity (http://www.auuuu.com/health/medicine/).

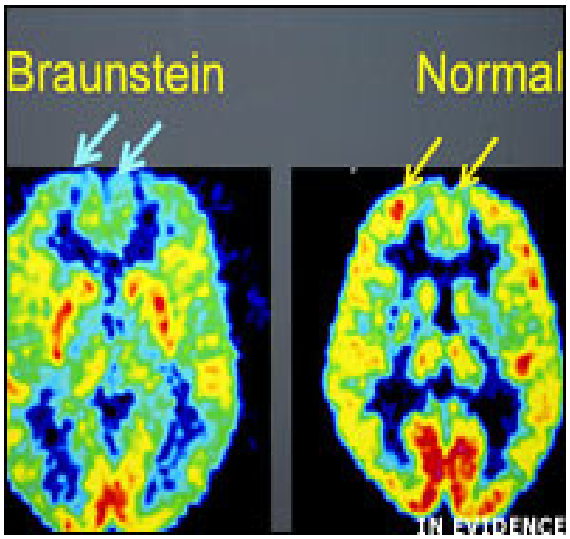
Numerous studies using magnetic resonance imaging (MRI) have identified neuroanatomical abnormalities in patients with bipolar disorder. For example, white matter hyperintensity, particularly in the frontal lobes, has consistently been reported. The frontoparietal junction, the basal ganglia, and deep white matter lesions have also been associated with a poor outcome in bipolar disorder (Moore, Shepherd, Eccleston, et al. 2001). In addition, MRI studies have reported ventricular enlargement, temporal lobe reduction and volumetric abnormalities of the striatum (Bearden, Hoffman, & Cannon, 2001).

It has also been suggested that functional neuroimaging by PET and fMRI with cognitive activation paradigms may provide the best methods of verifying regions associated with neuropsychological task performance. One preliminary study in a small sample of remitted bipolar patients confirmed abnormal prefrontal cortex function during

the performance of a verbal fluency task (Curtis, Dixon, Morris, et al. 2001). However, a PET investigation using a continuous performance task illustrated that abnormalities in bipolar disorder are not restricted to the frontal lobe, with reports of metabolic disturbances of the anterior cingulate cortex (ACC), cerebellum, and posterior cortical areas in such patients, irrespective of mood state (Ketter, Kimbrell, George, 2001; Rubinsztein, Fletcher, et al. 2001). Furthermore, recent studies have suggested that bipolar disordered patients are significantly impaired on different tests of executive function, such as the Wisconsin Card Sort Test, the Trail Making Test and the Stroop Test (Pradhan, Chakrabarti, Nehra, & Mankotia, 2008; Roth, Koven, Randolph, Flashman, et al., 2006).

F. Schizophrenia

Figure 8. PET Scan of Peter Braunstein



(Dr. Buchsbaum, a defense witness testified that the PET scan of Peter Braunstein, a New York writer accused of kidnapping and assaulting a former colleague, showed abnormalities consistent with schizophrenia. He also suggested that this "marked" deficiency would affect Braunstein's frontal lobe, which dictates, among other things, the capacity for making "executive decisions." The alleged deficiency is crucial to the success of Braunstein's defense strategy, which requires that a jury find he was incapable of conscious decision-making the day of the incident and in the months before, while he planned the attack (courttv.com., May 17, 2007).

Schizophrenia is a mental disorder characterized by abnormalities in the expression or perception of reality. It most commonly manifests as auditory hallucinations, bizarre or paranoid delusions, or disorganized speech and thinking with significant social or occupational dysfunction (DSM-IV-TR, 2000). Aggression and violence is sometimes found in schizophrenic patients, with theorists suggesting a pathophysiological role for certain areas of the brain, including the frontal cortex, the limbic system, and the basal ganglia. The thalamus has also been implicated, because it functions as an integrating mechanism, as has the brainstem due to the fact that it is one of the primary locations for the ascending aminergic neurons. The limbic system, because of its role in the control of emotions, has proved to be the most fertile area for neuropathological studies of schizophrenia (Spaletta, Troisi, Alimenti, Pau, et al., 2001).

The basal ganglia has also been of theoretical interest in schizophrenia because many patients with this disorder have odd movements, such as awkward gait, facial grimacing, and stereotypies, even in the absence of medication-induced movement disorders. In addition, of all the neurological disorders that can have psychosis as an associated symptom, the movement disorders involving the basal ganglia are those most commonly associated with psychosis in affected patients. A third factor implicating the basal ganglia in the pathophysiology of schizophrenia is the fact that they are reciprocally connected to the frontal lobes, thereby increasing the possibility that frontal lobe abnormalities seen in some brain imaging studies may be due to pathology within the basal ganglia, rather than the frontal cortex (Kaplan, Sadock, & Grebb, 1994).

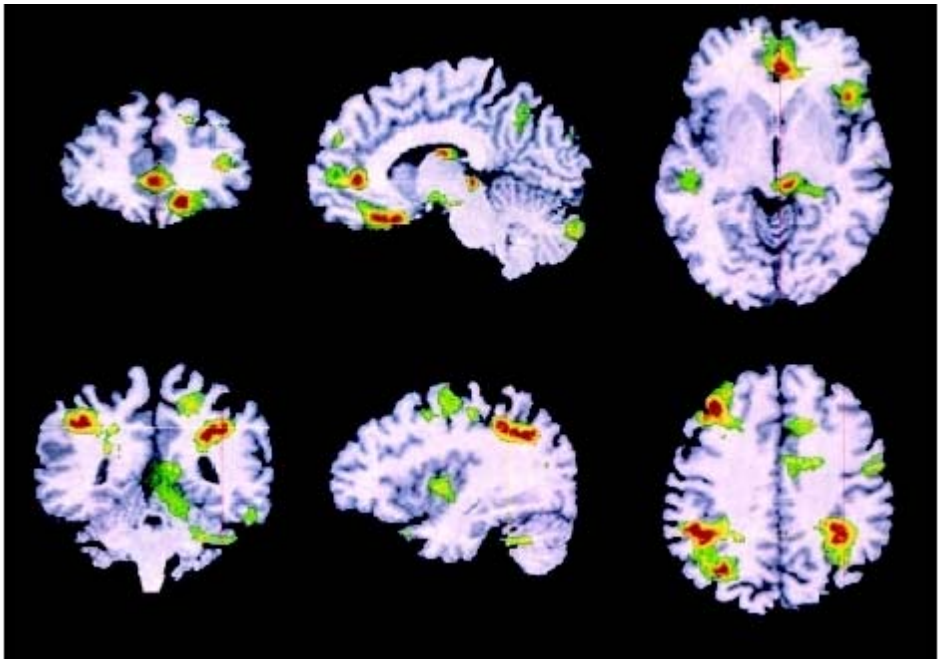
To date, numerous studies examining the association between aggression and psychiatric disorders have, in particular, attempted to identify the clinical parameters

of violence. For example, Roy and colleagues (1987) investigating select clinical and developmental indices of both violent and nonviolent schizophrenic patients found that incidences of verbal and physical violence were highly intercorrelated and could be utilized to meaningfully differentiate patients. More recently, reduced prefrontal cognitive activation, assessed with SPECT, has also been associated with aggression in schizophrenia (Spaletta, Troisi, Alimenti, Pau, et al., 2001). The association between aggression and schizophrenia, however, is questionable. While individuals with schizophrenia may display agitation, as well as poor impulse control when ill, the available data indicates that a schizophrenic patient is no more likely to commit homicide than is a member of the general population (Kaplan et al., 1994). Indeed, while negative symptoms have been associated with frontal lobe dysfunction, as assessed with tests sensitive to the dorsolateral prefrontal cortex, and persecutory delusions, and command hallucinations have been associated with violence in some schizophrenics, serious violent behavior, such as homicide, by such individuals is infrequent (Bender, 2006; Martino, Bucay, Butman, & Allegri, 2007).

G. Obsessive-Compulsive Disorder (OCD)

Obsessive-compulsive disorder is a mental disorder most commonly characterized by intrusive, repetitive thoughts resulting in compulsive behaviors and mental acts that the person feels driven to perform, according to rules that must be applied rigidly, aimed at preventing some imagined dreaded event; however, these behaviors or mental acts are not connected to the imagined dreaded event. Individuals with OCD suffer from recurrent anxiety-provoking thoughts (obsessions), and ritualized behaviors directed at reducing this anxiety (compulsions) (DSM-IV-TR, 2000).

Figure 9. PET Scan showing Obsessive-Compulsive Disorder



(PET scans of a brain showing active areas in obsessive-compulsive disorder; positive correlations (activity increases as symptoms get stronger), top row; negative correlation (activity decreases as symptoms strengthen), bottom (http://www.healthofchildren.com/N-O/Obsessive-Compulsive-Disorder/html).

Most neurobiological studies on OCD point toward an underlying dysfunctional prefrontal-striatal system. For instance, several structural neuroimaging studies have shown reduced orbitofrontal cortex and basal ganglia volumes (Calabrese, Colombo, Bonfanti, Scotti, et al., 1993; Szeszko, Robinson, Alvir, 1999). Functional neuroimaging studies have also shown hyperactivity in these same regions during rest-state, symptom provocation, and cognitive activity (Breiter, Rauch, & Kwong, 1996; Lucey, Burness, & Costa, 1997). Suggestions have also been made that the primary dysfunction is in the OFC but neuroimaging studies have also suggested dysfunction in the anterior cingulate and dorsolateral regions (Aycicegi, et al., 2003; Basso, et al., 2001; van den Heuvel, et al., 2005; van Veen & Carter, 2002).

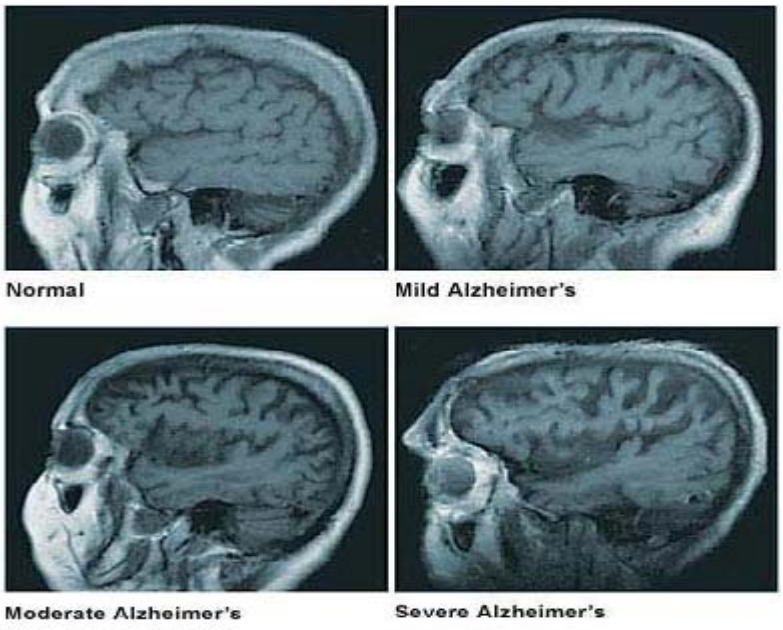
3. Neurological variables

A. Dementias

Dementia of the Alzheimer's type (DAT), the most common form of dementia, is generally diagnosed in people over 65 years of age, although the less-prevalent early-onset DAT can occur much earlier. This disease is characterized by loss of neurons and synapses in the cerebral cortex and certain subcortical regions. More specifically, this loss results in gross atrophy of the affected regions, including degeneration in the temporal lobe, parietal lobe, and parts of the frontal cortex, and cingulate gyrus (Wenk, 2003). Evidence from longitudinal and neuroimaging studies converge to indicate that psychological functions other than episodic memory are affected very early in the course of DAT and may predate or influence the apparent memory deficits. In fact, changes in personality and difficulty in executive function, especially in terms of attentional and inhibitory control, are especially prominent (Storandt, 2008).

Neuropsychological problems commonly associated with DAT, include memory and visual spatial problems, inhibition, confusion, disorientation, delusions, and hallucinations (Kolb & Whishaw, 1990). In addition, violent and aggressive behavior has been linked to DAT. More specifically, individuals with DAT may (1) be misperceiving their environment believing that they are defending themselves, and (2) be disoriented and confused about where they are, while some aggression may be the result of delusions or hallucinations. Indeed, in a descriptive survey of fourteen elderly patients who attempted or committed homicide, Ticehurst and colleagues (1992) found that these patients showed a high level of neuropsychiatric disturbance, with dementia diagnosed in over half of the cases.

Figure 10. MRI Scans of Patients with Alzheimer's Disease

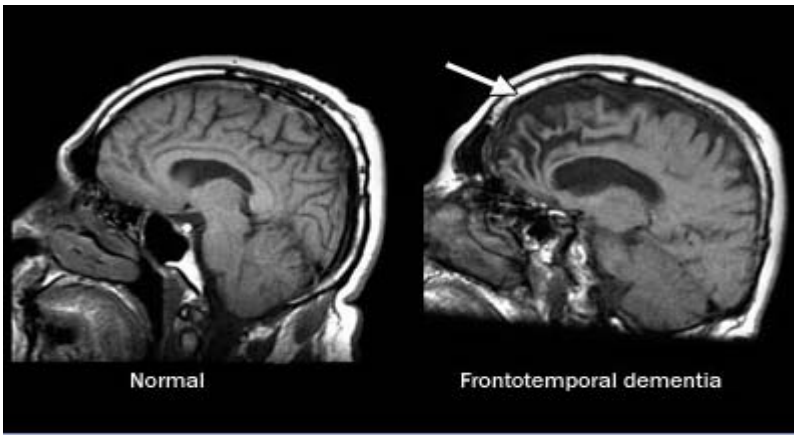


(MRI images of four different individuals with differently sized and shaped brains. The widening grooves and fissures of the cerebral cortex indicate progressively severe brain atrophy and loss of brain mass (MayoClinic.com).

Another form of dementia, frontotemporal dementia (FTD) is a clinical syndrome caused by degeneration of the frontal lobe of the brain, which may extend back to the temporal lobe. More specifically, in FTD portions of the lobes atrophy, or shrink. The signs and symptoms of this disease vary, depending upon the portion of the brain affected, however, some people with FTD undergo dramatic changes in their personality and become socially inappropriate, impulsive or emotionally blunted, while others lose the ability to use and understand language. In fact, the symptoms of FTD can be classified into two groups which underlie the functions of the frontal lobe: (1) behavioral symptoms (and/or personality change) and (2) symptoms related to problems with executive function. Behavioral symptoms include apathy and aspontaneity, while

impairment of executive function consists of the inability to perform skills that require complex planning or sequencing (Neary, Snowden, & Mann, 2000).

Figure 11. MRI Scan showing Frontotemporal Dementia



(MRI image on the right, particular the area near the white arrow shows the brain shrinkage common in FTD (MayoClinic.com).

Structural MRI scans often reveal frontal lobe and/or anterior temporal lobe atrophy, but in early cases the scan may seem normal. FDG-PET scans classically show frontal and/or anterior temporal hypometabolism, which helps differentiate from DAT (PET scan in DAT classically shows biparietal hypometabolism (Rosen, Gorno-Tempini, Goldman, et al., 2002).

B. Delirium

Delirium is a complex neuropsychiatric syndrome that typically involves a plethora of cognitive and non-cognitive symptoms, resulting in a broad differential diagnosis dominated by mental disorders. Delirium represents a generalized state of brain impairment that is acute rather than chronic in nature. There are a number of organic causes for delirium, including alcohol abuse, illegal substance abuse (e.g., LSD, PCP, cocaine, heroin), use of legal medications in improper doses, head trauma, meningitis,

encephalitis, diseases of major organ systems, and metabolic imbalances (Jones, 1992). Delirium results in a confused state that impairs the individual's judgment and perception of reality. More specifically, delirious patients may be hallucinating and delusional, and may show symptoms of a frontal lobe disorder such as disinhibition, irritability, inability to plan or anticipate consequences and to integrate information (Jones, 1992).

Although individual delirium symptoms are non-specific, their pattern is highly characteristic: acute onset (sometimes abruptly, but often over hours or days), fluctuant course (symptoms tend to wax and wane over any 24-hour period and typically worsen at night) and transient nature (in most cases, delirium resolves within days or weeks) are typical. Delirium also frequently involves a prodromal phase over 2-3 days of malaise, restlessness, poor concentration, anxiety, irritability, sleep-disturbance and nightmares. A consequence of this broad symptom profile is that delirium has many guises and, depending on prevailing pattern, is easily mistaken for dementia or functional psychiatric disorders (Meagher, 2001).

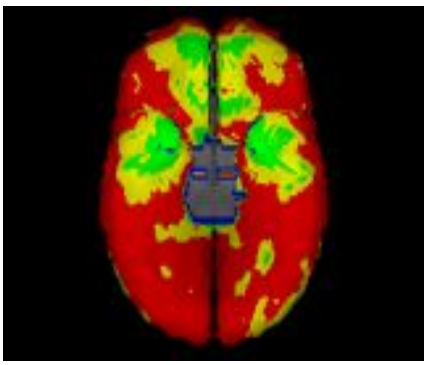
Individuals diagnosed with delirium may engage in aggression for a number of reasons: (1) they may believe that they are defending themselves from external threats, (2) they may react to heightened internal emotions without inhibitory abilities, or (3) they may overreact to real events because of heightened irritability. Unlike individuals with frontal lobe impairment, delirious individuals are generally easy to identify because of a wide range of physical and psychological symptoms that are more pronounced than focal prefrontal injuries. In addition, violence is limited to the acute times they are affected by the cause. In contrast, individuals with frontal lobe impairment may show symptoms of delirium more quickly than normal individuals and appear to be

much more sensitive to the effects of drugs (legal or illegal) and various medical disorders (Jones, 1992).

C. Traumatic brain injury (TBI)

Traumatic brain injury occurs when an outside force traumatically injures the brain. TBI can be classified based on severity (mild, moderate, or severe), mechanism (closed or penetrating head injury), or other features (e.g. occurring in a specific location or over a widespread area).

Figure 12. SPECT Image of Traumatic Brain Injury



SPECT scan showing decreased blood flow (yellow and green colors) to the right and left prefrontal inferior orbital area (top of the picture) and to the right and left temporal lobes (middle of the picture) after motor vehicle accident (http://braininspect.com/spect_lib/).

The scientific exploration of the relationship between violent behavior and neurological impairment has led to systematic examinations of head injury and possible neuropsychological deficits in male domestic violent offenders. For example, investigating the prevalence of TBI in spousal abusers, Rosenbaum and Hodge (1989) found that the 61.3% rate of head injury in this sample of batterers far exceeded that found in the population at large, which is estimated to be 5.9%. Similarly, a 1994 study by Rosenbaum and colleagues that evaluated male spousal abusers found a history of head injury in 53% of the abusers compared to 25% of the nonviolent, unsatisfactorily married men, and 16% of the nonviolent, satisfactorily married men. Based on

examination of the temporal order of the head injury and violence, the authors also found that in 93.1% of the head-injured batterers, the head injury preceded the first instance of marital aggression (Rosenbaum, et al., 1994). More recently it has also been shown that TBI patients score more poorly than non-TBI patients on measures of IQ and executive functioning, such as the Wisconsin Card Sort Test (WCST) (Marsh & Martinovich, 2006).

D. Neurochemistry

The neurochemistry of aggressive and violent behavior has received considerable interest, motivated in part by the hope that neuropharmacologic agents could be used to reduce these behaviors. More specifically, in view of the much higher incidence of violent behavior among male individuals, testosterone, the primary male sex hormone, merits considerable consideration. In nonhuman animals, clear evidence for a causal link between testosterone and aggression exists, but a similar association in human beings has yet to be convincingly demonstrated (Archer, 1991). More specifically, while incarcerated violent criminals have been shown to have higher salivary testosterone levels, elevated testosterone may be an effect rather than the cause of aggression and violence (Dabbs, Frady, Carr, et al., 1987).

Neurotransmitters, including acetylcholine, dopamine, and gamma aminobutyric acid, have also been considered in the origin of violent behavior. However, the neurotransmitters, for which the data are most convincing, are serotonin and norepinephrine. More specifically, in the central nervous system, serotonin is presumed to play an important role in the modulation of anger and aggression. The first suggestion of a link between reduced serotonin function and impulsive aggression was advanced in

1983 by Linnoila and colleagues, who showed that concentration of a primary metabolite of serotonin in the cerebrospinal fluid, 5-hydroxyindoleacetic acid, was lower in impulsive violent offenders than in non-impulsive violent offenders. Subsequently, the idea of a low serotonin syndrome has been substantiated using various measures of serotonin function (Caspi, Sugden, Moffitt, Taylor, et al., 2003; Linnoila and Virkunnen, 1992). For example, Coccaro and colleagues (1998), in studying subjects with personality disorders, found an inverse relation between platelet serotonin binding sites and aggressive acts. A role of reduced serotonin in violence finds additional support from a study of depressed patients with anger attacks showing that fluoxetine, a serotonergic agent, can cause these attacks to remit in over half the patients (Fava, 1998). Although the evidence is not entirely consistent, most studies increasingly support the association of lowered central serotonin levels and impulsive violence. Aggression and violence has been associated with increased activity of norepinephrine. For example, experimentally induced hostile behavior in normal human subjects has been related to increased plasma levels of norepinephrine (Gerra, Zaimovic, Avanzini, et al., 1997). Further aggression has been produced in knockout mice with selective deficiency of monoamine oxidase catechol-O-methyl transferase, the two major enzymes involved in the metabolic degradation of norepinephrine (Gogos, Morgan, Luine, et al., 1998; Cases, Seif, Grimsby, et al., 1995). In clinical studies, low monoamine oxidase activity has also been found in platelets of violent offenders. Further, among patients suffering from schizophrenia and schizoaffective disorder, 64% of those homozygous for the low-activity catechol-O-methyl transferase allele were violent, whereas 80% of patients homozygous for the high-activity allele were nonviolent (Lachman, Nolan, Mohr, 1998).

4. Personality disorders

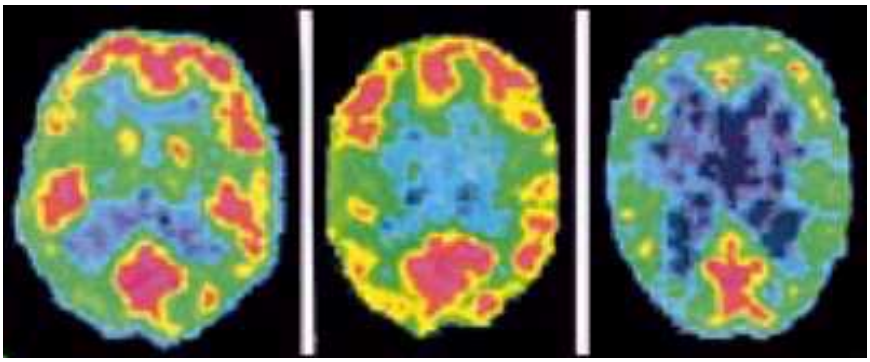
A. Antisocial personality disorder (APD)

The antisocial personality has been described as being impulsive, self-centered, and aggressively opportunistic. Such individuals seem to enjoy taking unnecessary chances, appear easily bored, cannot delay prospects for immediate gratification, and evidence a low tolerance for frustration (Miller, 1987). Compared with other male criminals, individuals with APD commit a disproportionate number of crimes that are more violent and aggressive than those of other criminals (Elliott, 1992). Although the etiology of APD is disputed, much has been made of the relationship between antisocial or psychopathic behavior and brain dysfunction, in particular frontal lobe dysfunction (Elliott, 1987).

As previously discussed, the frontal lobes are generally considered to be responsible for planning, judgment, higher abstraction, sustained motivation, as well as self-regulation. Generalized brain damage impairs the mechanisms that inhibit or regulate emotional response, and the frontal cortex appears to be especially vulnerable. As a result, the patient has little, if any, control over sudden shifts of mood or the rapid change in basic drives that direct behavior; including a lower threshold for aggression (Miller, 1990). Indeed, studies have shown that behavioral changes as a result of bilateral prefrontal lesions strongly resemble features of antisocial personality disorder, including apathy, egocentricity, lack of insight, aggressiveness, lack of conscience, poor judgment, inability to learn from experience, and a poor tolerance for alcohol (Elliott, 1988). Research also indicates that individuals with APD often presents with abnormal EEG, CT, MRI, fMRI, PET, and SPECT results (Finn, Ramsey, & Earleywine, 2000; Goethals,

Audenaert, Jacobs, Van den Eynde, et al., 2005; Intrator, Hare, Stritzke, et al., 1997; Raine, Lencz, Bihrle, et al., 2000; Raine, Buchsbaum, & LaCasse, 1997). Furthermore, APD has been associated with impairment of test designed to assess executive functioning (Morgan & Lilienfield, 2000; Raine, Lenz, Bihrle, et al, 2000). A more recent study, however, has failed to support this association (Crowell, Kieffer, Kugeares, & Vanderploeg, 2003).

Figure 13. PET Scans of Normal Subject and Murderers

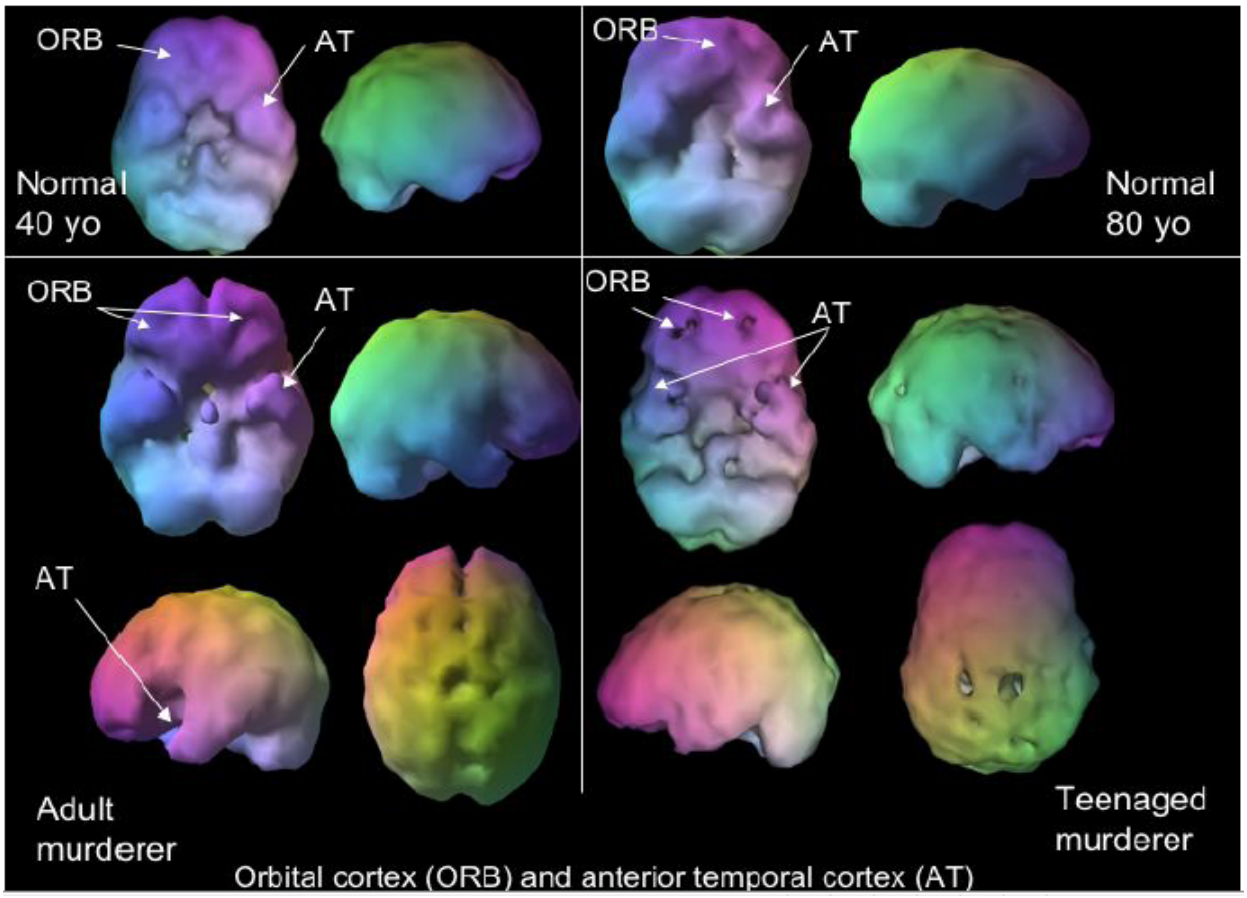


(PET scans of normal person (left), murderer with deprived background (middle) and murderer with non-deprived background (right). Areas in red and yellow show high metabolic activity, while areas in black and blue show low metabolic activity. The brain of a sociopath (right) has a very low activity in many areas, but which is strikingly absent in the frontal area (upper part of the images) (Images by Raine, Phil, & Stoddard, 1998).

B. Psychopathy

Psychopathy, which is characterized by a shallow, callous, and manipulative interpersonal style combined with antisocial and reckless behavior, has frequently been associated with violent behavior. More specifically, psychopaths not only show little concern about the effects of their actions on others, but also appear to show little regard for the impact of actions on themselves. Thus, they often commit impulsive, poorly planned crimes for which the likelihood of being caught is high, and fail to avoid behaviors for which they have previously been punished (Hare, 1991).

Figure 14. SPECT Scans of Normal subjects and Murderers



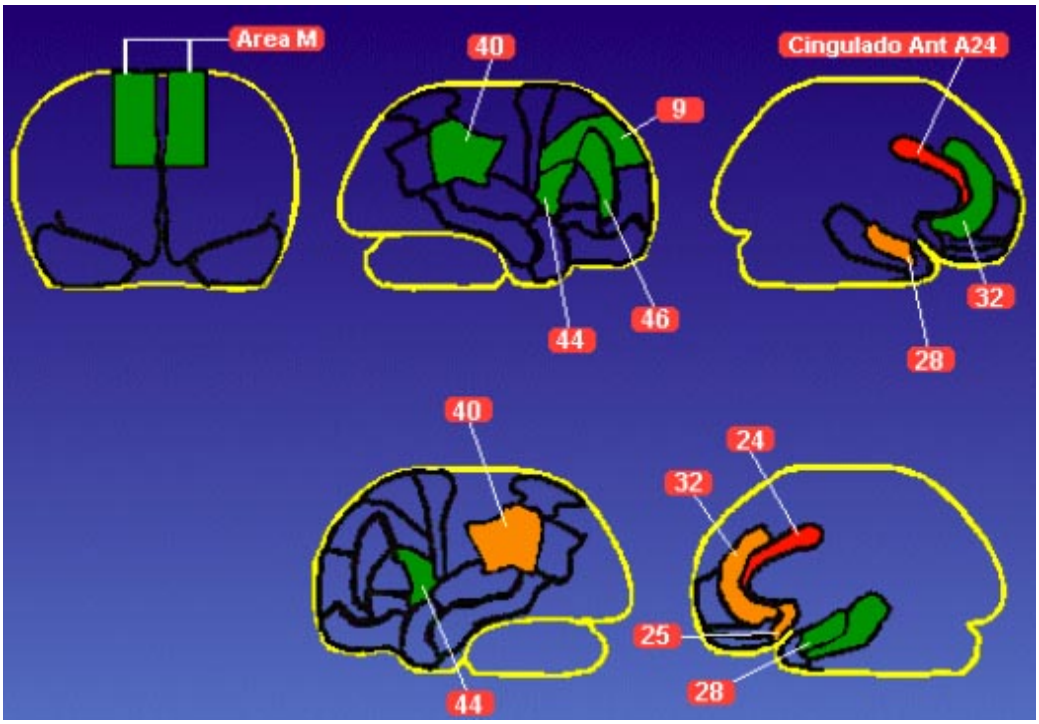
(SPECT scans of a normal forty year old (top left) and an eighty year old (top right). Note that the brain surfaces are smooth, including over the orbital cortex and anterior temporal lobe. In a young psychopathic murderer (bottom right) and adult psychopathic murderer (bottom left) the orbital cortex and anterior temporal lobes show pitted surfaces, indicating reduction or loss of function. In the teenaged murderer, there is also pitting in the posterior parietal cortex (Courtesy, Amen Clinics).

Unlike individuals with a diagnosis of APD, psychopathic individuals show no indications of impairment on measures of executive function associated with the dorsolateral prefrontal cortex (DLPFC), such as the Wisconsin Card Sort Test (WCST) or the Controlled Oral Word Association Test **(**COWAT) (LaPierre, Braun, & Hodgins, 1995; Mitchell, Colledge, Leonard, & Blair, 2002; Roussy & Toupin, 2000). However, individuals with psychopathy do present with frontal lobe dysfunction albeit a

dysfunction that is selective to those executive functions that are mediated by the OFC rather than the DLPFC (Mitchell et al., 2002; Roussy & Toupin, 2000).

C. Borderline Personality Disorder (BPD)

Figure 15. Picture of Brodmann's Areas



(Statistical significant areas of Brodmann after activation: 24, 28, 32, 40, 44 in the left and right hemisphere and the area defines as M (left and right) which corresponds to the anterior projection of the DLPFC (http://www.alasbimnjournal.cl/revistas/).

Borderline Personality Disorder is characterized by severe deficiencies in impulse control and emotion regulation, which can result in self-destructive and aggressive behaviors. Accordingly structural imaging studies have focused on the temporal lobe and frontal cortex, areas known to be associated with affect, emotion regulation, cognition, and impulsivity (McCloskey, Phan, & Coccaro, 2005). To date, evidence for morphological (structural) abnormalities in the frontal cortex of BPD patients appears to be non-definitive. For example and MRI study comparing 25 patients

with BPD with a matched healthy control ground found a 6.2% decrease in frontal lobe volume (Lyoo, Han, & Cho (1998). Further, Van Elst and colleagues (2003) initially found significant volume loss associated with BPD in the orbitofrontal cortex, but later failed to replicate these findings using different MRI methodology.

The evidence for altered frontal activity and/or function in BPD has also received support from PET studies which have found differences in frontal lobe metabolism between control subjects and patients with BPD (Soloff, Meltzer, Becker, et al., 2003; Soloff, Meltzer, Greer, et al., 2000). Finally, patients with BPD who showed impulsive behavior were also found to have diminished regional cerebral blood flow (rCBF) in areas of the temporal and right prefrontal cortex (Goethals, Audenaert, Jacobs, Van den Eynde, et al., 2005).

D. Intermittent Explosive Disorder (IED)

Intermittent Explosive Disorder is characterized by recurrent acts of impulsive, affectively-driven aggression that are disproportionate to any actual provocation (Coccaro, 2003). Of note, these aggressive acts are not attributable to another neurological or psychiatric condition. Individuals with IED also have elevated levels of trait anger and hostility and typically have frequent (e.g. twice a week) acts of verbal and physical aggression (McCloskey, Berman, Noblett, & Coccaro, 2006). Recent epidemiological studies suggest that IED is highly prevalent (5% lifetime prevalence) in the United States population. Furthermore, IED confers functional impairment equal to or greater than most other Axis I and Axis II disorders (Kessler, Berglund, Demler, Jin, et al., 2005). Despite the public health impact of IED, relatively little is known about the neurobiology of the disorder. At the neurochemical level, IED appears to be associated

with dysregulation of the serotonergic system, however, to date only once imaging study has attempted to identify the specific brain regions affected by IED. More specifically, Coccaro and colleagues (2007) have demonstrate a link between amygdala-OFC dysfunction and impulsive aggression in IED on three levels of evidence: 1) exaggerated amygdala and diminished OFC reactivity to faces conveying direct threat (anger) in IED subjects relative to controls; 2) lack of amygdala-OFC functional connectivity during the face processing task in IED subjects, but a significant reciprocal (inverse) interaction between amygdala and OFC in controls; and 3) direct, positive correlation between amygdala reactivity to angry faces and extent of prior aggressive behavior.

5. Cognitive impairment

While neurobiological factors are known to play a role in human aggression, relatively few studies have examined neuropsychological contributions to propensity for violence. Neuropsychological deficits just as neurological deficits can rarely be implicated as the direct or sole cause of an aggressive or violent act; however, cognitive dysfunction is one factor that has been shown to play a role. Indeed, neuropsychological deficits can reduce the number of options an individual perceives or has available with which to respond to a given situation. As a component of the human flight or fight response, aggression, thus, may serve as an adaptive mechanism. When cognitive dysfunction is present, however, aggression may dominate the behavioral response and override avoidance or withdrawal. Deficits, including impulsivity, inability to draw on past learning to recognize a dangerous course of action, impaired capacity to anticipate future consequences of present behavior, and insufficient self-monitoring, may all conspire to conceal more socially desirable courses of action. Specifically, it has been

hypothesized that cognitive deficits, especially impulsivity, poor planning ability, mental inflexibility, low verbal intelligence, and impaired attention, limit an individual's ability to cope with other biological and environmental vulnerabilities; these limitations, in turn, lead to feelings of frustration and anxiety and, ultimately, to difficulty with regulation of emotion and increased aggressive behavior. Although social and environmental factors have major effects on the expression of aggression and violence, neuropsychological integrity also helps to determine the ability to behave in a socially acceptable manner (Krakowski, 1997).

An important point is that the contribution of brain dysfunction to aggression may stem not only from structural damage in a given brain region but also from neurochemical or neurophysiological disturbances not detectable by conventional neurodiagnostic methods. Therefore, brain correlates of aggression and violence must also be studied by examining the neuropsychological profile of aggressive and violent individuals, irrespective of demonstrable brain lesions. Indeed, research into the neurocognitive functioning of aggressive individuals has consistently shown that violent adults perform in the impaired range on tasks of executive functioning that may be linked to structural and functional brain abnormalities, particularly in the prefrontal regions (Easton, Sacco, Neavins, Wupperman, et al., 2008; Kuruoglu, Arikan, Vural, et al., 1996; Raine, Lenz, Bihrle, et al., 2000).[11] Reliable neuropsychological tests and measures may, in fact, be more sensitive to brain dysfunction at a microscopic level than other

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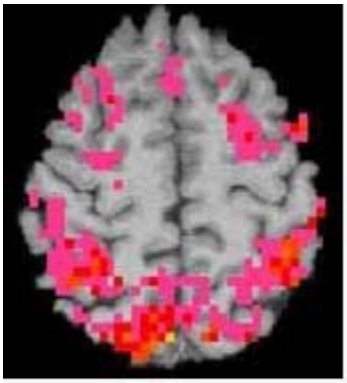
[11] However, violent and aggressive individuals generally score within the normal range on test of intellectual functioning, such as the Wechsler Adult Intelligence Scale-III.

techniques and may provide unique insights regarding the cerebral mediation of violent behavior (Filley, et al., 2001).

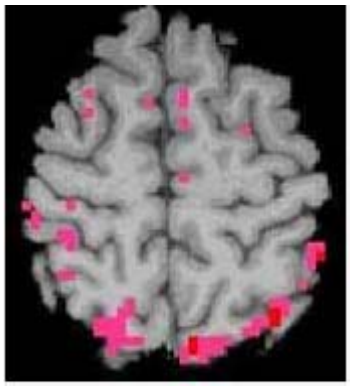
6. Alcohol abuse

Numerous researchers have found a relationship between aggression, brain damage, and alcohol abuse (Rosenbaum & Hoge, 1989; Ticehurst, Ryan, & Hughes 1992). Alcohol is the most commonly investigated drug and the main effect of alcohol intoxication is the depression of inhibitors that predispose individuals toward aggressive behavior (Elliot, 1992). More specifically, alcohol abuse and brain damage may have a synergistic effect on the disinhibition of behavior, together predisposing, to a greater extent, individuals with developmental or acquired brain defects toward aggression (Elliot, 1992; Miller, 1990). Furthermore, Manning and colleagues (2008) found significant increases in performance scores post detoxification in working memory, verbal fluency and verbal inhibition but not in non-verbal executive function tasks (mental flexibility and planning ability). However, despite increased scores on tests of verbal and memory skills complex executive abilities showed little change, after three weeks of abstinence (Manning, Best, Hill, Reed, et al., 2008). Finally, a study assessing the long-term effects of Korsakoffs syndrome found that although general knowledge, visual long-term memory, verbal fluency, and executive functions improved slightly after two years, they still remained within pathological range (Fujiwara, Brand, Borsutzky, Steingass, et al., 2008).

Figure 16. Scans of a Nondrinker and an Alcohol-dependent subject



20-year old female nondrinker's response to a spatial working memory task. Brain activation is shown in bright colors.



Alcohol-dependent 20-year old female's response to the spatial working memory task. Brain activation is shown in bright colors (http://www.narconon.ca/Alcohol.htm.)

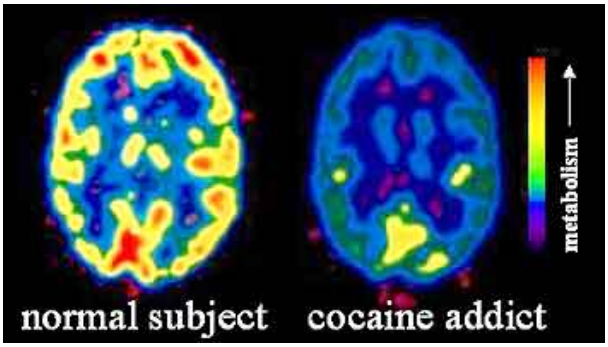
7. Drug abuse

Addiction is a complex disease process of the brain that results from recurring drug intoxication and is modulated by genetic, developmental, experiential, and environmental factors. The neurobiological changes that accompany drug addiction are not, yet, well understood, however, until recently it was believed that addiction predominantly involves reward processes mediated by limbic circuits. Results from neuroimaging studies have implicated additional brain areas, especially the frontal cortex. For example, frontal lobe volume losses have been identified in cocaine-dependent and heroin-dependent subjects (Franklin, Acton, Maldjian, Gray, et al., 2002; Goldstein & Volkow, 2002). The latter study also noted negative correlations between normalized

prefrontal volumes and years of either cocaine or heroin use, implying a cumulative effect of substance abuse on frontal volumes.

Individuals with a history of long-term opiate abuse and dependence may also suffer cognitive impairments, primarily within the domain of executive functioning (Ersche & Sahakian, 2007; Ersche, Clark, London, Robbins, et al., 2006; Verdejo-García, Perales, & Pérez-García, 2007; Verdejo-García and Pérez-García, 2007). More specifically, executive dysfunction has been linked to functional abnormalities of the dorsolateral prefrontal cortex (Pezawas, Fischer, Podreka, Schindler, et al., 2002). It has also been suggested that long-term opiate addicts may have emotional disturbances associated with dysfunctions of limbic structures and the orbitofrontal cortex (Botelho, Relvas, Abrantes, Cunha, et al., 2006; Ersche, Fletcher, Roiser, Fryer, et al., 2006; Pezawas, Fischer, Podreka, Schindler, et al., 2002).

Figure 17. PET Scans of Normal Subject and Cocaine Addict



PET images showing brain function in a normal subject and in a cocaine addict. The cocaine addict has depressed metabolism in frontal regions when compared to the normal subject. Low frontal metabolism may underlie the loss of control in cocaine addiction (http://www.bnl.gov/pet/FDG.htm).

Brain-imaging studies of methamphetamine (MA) abusers have also reported various kinds of frontal brain abnormalities, including impairment of neuropsychological function (Ernst, Chang, Leonido-Yee, & Speck, 2000; Kalechstein, Newton, & Green, 2003; Paulus, Hozack, Zauscher, Frank, et al., 2002; Sekine, Minabe, Ouchi, Takei, et al., 2003). The cognitive impairment in MA abusers may be related to abnormalities of

frontal lobes of the brain, as shown in functional magnetic resonance imaging studies which have reported failure of normal prefrontal activation during a decision-making task (Paulus, Hozack, Zauscher, Frank, et al., 2002). Indeed, Kim and colleagues (2006) have shown that decreased grey-matter densities and glucose metabolism in the frontal region of the brain correlated with the impairment of frontal executive functions in MA abusers.

It has also been established that the acute effects of cannabis include interference with both visuo-spatial working memory and tasks loading heavily on executive function (Grant, Grant, Contoreggi, & London, 2000). More specifically, Pope and Yurgulun-Todd (1996) have suggested that heavy cannabis use is associated with reduced attentional executive function which manifests as decreased mental flexibility, increased perseveration and reduced learning. These cognitive deficits were detectable up to seven days following cannabis use and appear reversible. Indeed, evidence to support permanent persistent cognitive effects in heavy cannabis users is equivocal. Some studies indicate long-term effects (after 24 h to 28 days) on short-term memory and attention (Bolla, Brown, Eldreth, Tate, et al., 2002; Eldreth, Matochik, Cadet, & Bolla, 2004; Pope, Gruber, Hudson, Huestis, et al., 2001). In addition, chronic recreational cannabis use has been associated with an indication of diminished neuronal and axonal integrity in the DLPFC (Hermann, Sartorius, Welzel, Walter, et al., 2007).

Empirically validating the unique impact of alcohol and substance abuse on aggression is difficult due to the complex nature of the phenomenon. Indeed, the effects cannot be easily separated from other influencing variables, such as emotional disorders, cognitive deficits, and environmental factors. However, the link between violence, drug abuse, and brain abnormality may be mediated by altered cognitive capacities, in

particular those involving executive functions, such as attention, abstract reasoning, and planning goal-oriented behaviors (Fishbein, 2000). Furthermore, it has been suggested that, at the biological level, alcohol may induce violent and aggressive behavior in susceptible individuals, in part by inducing a depletion of brain serotonin levels (Badawy, 2003).

8. Childhood antecedents

Research findings have suggested that low educational achievement and intelligence, child abuse, antisocial parents and peers, low family income, impulsiveness, and disrupted families, in particular, are risk factors for APD (see, e.g., Goodman, Simonoff, & Stevenson, 1995; Juby & Farrington, 2001; Lispsey & Derzon, 1998; Maxfield & Widom, 1998). Whether a child's exposure to violence leads to withdrawal or to increased aggression and violence is likely to depend on a variety of factors, including the age at which the trauma occurred, the supports in the environment, and the characteristics of the child. It has also been contended that conduct disorder in childhood and adolescence and juvenile delinquency predicts APD and adult crime (Farrington, 2005; Lahey, Loeber, Burke, & Applegate, 2005; Piquero, Farrington, & Blumstein, (2003).

Several studies have also found that male children and adolescents with high ratings on both Attention Deficit Hyperactivity Disorder (ADHD)[12] behaviors and conduct problems have higher levels of early adult criminality than youth with either elevated ADHD ratings or conduct problems alone ((Fischer, Barkley, Smallish, &

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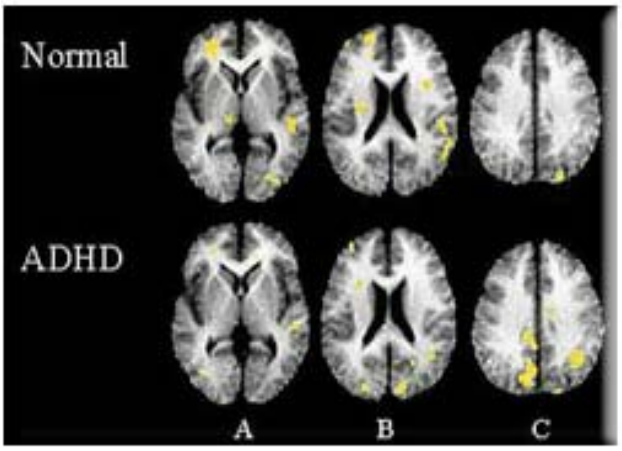
[12] ADHD is considered to be a neurobehavioral developmental disorder that is characterized by a persistent pattern of impulsiveness and inattention, with or without a component of hyperactivity.

Fletcher, 2002; Pulkkinen & Pitkaenen, 1993). More recent studies, however, have failed to find an association between a diagnosis of ADHD in childhood and adult criminality (Lahey, Loeber, Burke, & Applegate, 2005; Loeber, Burke, & Lahey, 2002). In addition, studies examining the relation between APD in parents and their offspring have concluded that APD is both heritable and familial (Langbehn & Cadoret, 2001; Rhee & Waldman, 2003).

Figure 18. PET Scan of ADHD



PET scan with and without ADHD. Normal subject shows more activity in the frontal part of the brain that is associated with attention (http://www.edweek.org/ew/).

CHAPTER 4. MALINGERING AND ITS ASSESSMENT

That mental illnesses and cognitive deficits can be feigned has been known since antiquity. Indeed, according to Homer, Ulysses feigned madness to avoid participating in the Trojan War. Two Shakespeare plays also contain accounts of feigned madness: Edgar in King Lear, who feigned madness in order to escape the persecutions of his brother, and Hamlet, when he was attempting to escape the machinations of his uncle (Chesterman, Terbeck, Vaughan, 2008).

While malingering of deficits has a long history, determining exactly how to detect an individual who feigns cognitive or other impairments has proven to be difficult. Indeed, Rosenhan's (1973) study clearly demonstrated the inability of mental health professionals to distinguish mental illness from normal behavior. In this classic study eight volunteer pseudo-patients, a psychology student, a pediatrician, three psychologists, a psychiatrist, a housewife, and a painter, all were admitted to psychiatric hospitals after claiming to hear voices. Although, all claims of hearing voices stopped immediately following admission, all were diagnosed as suffering from schizophrenia and remained in the hospital from 9 to 52 days. From these findings Rosenhan (1973) concluded: "It is clear we cannot distinguish the sane from the insane in psychiatric hospitals" (p. 250).

Forensic mental health experts are frequently asked to determine the nature and severity of cognitive and/or neurological, and/or psychological deficits of individuals. Consequently, forensic clinicians "bear a heavy responsibility to society in differentiating true disease from malingered madness" (Resnick, 1994, p. 552). It is, thus, not surprising that professional interest in methods of detecting suboptimal effort or malingering has burgeoned over the past decade and as the literature has matured, three primary methods

of detecting suboptimal effort or malingering have emerged: (1) unusual poor performance on tests designed specifically to detect poor effort or malingering, such as the Test of Memory Malingering (TOMM) and the Portland Digit Recognition Test (PDRT); (2) unusual patterns of errors on tests originally designed to assess some aspect of neurocognitive ability, such as the California Verbal Learning Test (CVLT), or the Wechsler Adult Intelligence Scale-Revised; and (3) unusual patterns of variability within the same tests across time (Williamson, Green, Allen, & Rohling, 2003).

Given the abundance of available tests and techniques currently available, deciding exactly which test or technique to employ to obtain the most reliable information is not an easy task. To make this decision, a careful examination of each test's predictive accuracy is, therefore, necessary. A number of issues bear consideration in this regard. Many of the currently available tests were developed using normal samples simulating impairment rather than clinical samples. While this is a helpful starting point, normal samples simulating impairment and clinical samples, however, may not behave in identical ways (Rogers & Cruise, 1998). A clinician's confidence in the generalizability of these test or techniques is, therefore, limited. Many of the clinical samples used to validate tests have limitations as well. For example, some are limited to patients without severe neurocognitive impairment; consequently, the extent to which one may encounter "false positives" in the face of severe impairment is unknown (Curtiss & Vanderploeg, 2000). In addition, the contexts in which many clinical validation studies are performed bear little similarity to the context often encountered by the practicing clinician: that is, when the results of the evaluation are explicitly linked to the possibility of a subject receiving secondary gains (Binder & Rohling, 1996).

There is no doubt that the assessment of malingering of cognitive deficits and/or psychiatric disorders must be an integral part of criminal forensic evaluations since failure to detect malingering of disorders and dysfunctions can have serious adverse consequences for the administration of justice. Indeed, defendants who successfully feign impairments may avoid criminal prosecution if acquitted by reason of insanity. In addition, if a defendant successfully feigns a mental illness or cognitive dysfunction and is found not to present a substantial risk by reason of mental illness or mental defect, the final result can be outright release and the avoidance of all criminal and civil commitment sanctions. Successful malingering of a psychiatric disorder in sentencing proceedings can also result in the imposition of a shorter sentence due to the mistaken finding that the feigned symptoms mitigated the offense. Falsely concluding that a defendant is malingering, when they do present with a genuine psychiatric disorder, can also have adverse consequences, given the pejorative nature of labeling someone a malingerer. More specifically, wrongly inferring that mentally ill defendants are malingering can result in denial or delay in the provision of needed psychiatric treatment. Further, a defendant could be denied a mental health defense that would have otherwise been available had he/she not been believed to be malingering (Kucharski, Duncan, Egan, & Falkenbach, 2006).

Because of the secondary gains of avoiding criminal sanctions and the negative consequences associated with wrongly opining that a defendant is malingering, forensic mental health evaluators, therefore, must employ validated methods for the identification of malingering (Kucharski, Duncan, Egan, & Falkenbach, 2006). Given that many currently available tests of malingering lack adequate predictive accuracy when used in

isolation, this is best accomplished by a employing a multimodal approach to the assessment of malingering.

Definition of malingering

The American Psychiatric Association (2000) has defined malingering[13] as "the intentional production of false or grossly exaggerated physical or psychological symptoms motivated by external incentives…" (p.739). The Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR-2000) does not view malingering as a specific disorder, thus, guidelines rather than diagnostic criteria are provided for the assessment of malingering. These guidelines include: (1) medicolegal context of presentation, (2) marked discrepancy between objective findings and claimed disability, (3) lack of cooperation in assessment and treatment, and (4) the presence of Antisocial Personality Disorder (APD) (American Psychiatric Association, 2000).

Malingering is not an all-or-nothing phenomenon; it exists on several levels. Indeed, an individual who is exaggerating genuine symptoms in an attempt to create the appearance of a more severe form of psychopathology or cognitive impairment represents one level, while a person who uses deceit to extend legitimate symptoms back to the time of the criminal activity in order to reduce culpability involves a different level of malingering. Further, there are those individuals who completely fabricate symptoms for the sole purpose of receiving external incentives. These incentives, include, but are not limited to: (1) a finding of (a) not guilty by reason of insanity, (b) diminished capacity, (c) incompetency to stand trial, (d) incompetency to be executed, and (2) mitigating

[13] For purpose of this discussion the terms "malingering" and "feigning" are used synonymously. These terms refer to disorders and illnesses that have functional but no structural abnormalities, such as schizophrenia.

factors. Finally, criminal charges may be dismissed entirely (Frederick, Crosby, & Wynkoop, 2000).

The difference between simple unreliable reporting and malingering cognitive dysfunction or mental illness is a matter of the individual's intent. More specifically, feigning, by definition, is deliberate. In contrast, where intentionality is in doubt, the individual may be classified as simply being unreliable. In other words, the information provided may be inaccurate, in that it does not present a valid portrait of the individual's impairment or illness, but this is not due to purposeful distortion (Rogers, 1997). Further mental illness or cognitive impairment, and malingering are not mutually exclusive phenomena. In fact, research has shown that some of the more effective malingerers are those individuals who have experienced or are experiencing actual symptoms of mental illness and/or cognitive impairment. Thus, malingering and neurological, psychiatric, and psychological disorders may co-exist with some malingerers simply exaggerating the symptoms of genuine disorders (Rogers & Bender, 2003).

The concept of malingering also should not be confused with Somatoform and Factitious disorders. The major difference between malingering and Somatoform disorders is motivation. Where the feigning of a mental illness or a cognitive deficit is due to a conscious effort, it can be referred to as malingering, whereas Somatoform disorders are motivated by unconscious or involuntary processes. Factitious disorders can be distinguished from malingering in that there are no external incentives present (Rogers, 1997).

In response to the DSM-IV-TR's (2000)broad categorization, a number of criteria have been proposed to more precisely define malingering, but the most thoroughly

outlined proposal has been provided by Slick, Sherman, & Iverson (1999). Slick et al. (1999) define malingering as "the volitional exaggeration or fabrication of cognitive dysfunction for the purpose of obtaining substantial material gain, or avoiding or escaping formal duty or responsibility." (p. 552). The authors describe three categories of malingering: (1) possible, (2) probable, and (3) definite. For a patient to classify into one of these categories a combination of four criteria is to be met. These four criteria are:

• *Criterion A: Presence of a substantial external incentive* - at least one clearly identifiable and substantial external incentive is present at the time of testing.

• *Criterion B: Evidence from neuropsychological testing* - evidence of exaggeration or fabrication on neuropsychological tests as evidenced from at least one of the following: (1) definite response bias - below chance performance ($p<.05$) on one or more forced-choice measures; (2) probable response bias - performance on a well-validated test is consistent with fabrication or exaggeration; (3) discrepancy between test data and known patterns of brain functioning; (4) discrepancy between test data and observed behavior; (5) discrepancy between test data and reliable collateral reports; and (6) discrepancy between test data and documented background history.

• *Criterion C: Evidence from self-report* - significant inconsistencies or discrepancies in a patient's self-reported symptoms that suggest exaggeration or fabrications as evidenced by one of the following: (1) self-reported history is discrepant with documented history; (2) self-reported symptoms are discrepant with known patterns of brain functioning; (3) self-reported symptoms are discrepant with behavioral observations; (4) self-reported symptoms are discrepant with information obtained from collateral informants; and

(5) evidence of exaggerated or fabricated psychological dysfunction - performance on well-validated validity scales or indices on self-report measures of psychological adjustment are strongly suggestive of exaggeration or fabrication.

• *Criterion D: Behaviors meeting necessary criteria from groups B or C are not fully accounted for by Psychiatric, Neurological, or Developmental Factors* - behaviors are the product of an informed, rational, and volitional effort aimed at least in part toward achieving or acquiring external incentives.

To qualify as definite malingerer, a subject must meet criteria A, B1, and D (there must be substantial external incentive, the presence of a definite negative response bias on neuropsychological test(s), and no psychiatric, neurological, or developmental factor that would significantly diminish one's capacity to appreciate laws or mores against malingering). To qualify as a probable malingerer, a subject must meet criterion A, two or more from B1-B6, and D, or criterion A, one from B1-B6, one from C1-C5, and D (someone can be classified as probable malingerer in two ways: (1) by having the presence of external incentive, two pieces of evidence from neuropsychological testing, and no psychiatric, neurological, or developmental disorder, or (2) by having external incentive, one piece of evidence from neuropsychological testing, one piece of evidence from self-report, and no psychiatric, neurological, or developmental disorder). There are also two ways in which an individual can qualify as a possible malingerer: (1) the person must either meet criterion A, one from C1-C5, and D, (external incentive, evidence from self report, and no psychiatric, neurological, or developmental disorder) or (2) must meet

criteria that would classify him/her as definite or probable malingerer with the exception of criterion D (Slick, et al., 1999).[14]

Although relatively new, these proposed definitions and criteria have gained support in the research community and several recent studies have classified subjects according to these criteria (Greve & Bianchini, 2007; Greve, Bianchini, Doane, & Mathias, 2006; Greve, Bianchini, Houston, & Crouch, 2002; Greve, Bianchini, Mathias, Houston, & Crouch, 2002; Larrabee, 2003; O'Bryant, Engel, Kleiner, Vasterling, & Black, 2007).

Malingering and the law

There is no doubt that the assessment of malingering in the forensic context must be comprehensive and should not rely on a single test, measure, or technique because of the consequences associated with a misclassification. Therefore, in addition to the standard clinical interview, acquiring collateral information to verify the veracity of a claim of cognitive impairment or mental illness is crucial. Although the evaluation of malingering occurs in many criminal cases, to date, it has not been specifically addressed in statute. However, in terms of case law, *United States v. Greer* (1998), a federal case decided by the Fifth Circuit Court of Appeals, clearly emphasizes the importance of malingering assessment. Consider the following:

In 1994, Greer was arraigned on federal charges of kidnapping and firearms violations. State charges had initially been dismissed after Greer was determined to be incompetent as the result of a mental disorder. The federal prosecutor pursued the case,

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[14] See also Table 2.

and Greer was hospitalized at a federal medical center for mental health evaluation. The evaluating psychologist testified that Greer was competent and malingering psychopathology and cognitive impairment. The judge ruled that he was competent. However, Greer was so disruptive in jail over the next year while awaiting trial that he was reevaluated on an outpatient basis, ruled to be incompetent after another hearing, and committed to a different federal medical center for restoration of competence. After a period of hospitalization Greer was again evaluated by a psychologist, who concluded that he was malingering and competent. A third competency hearing was held, and the court agreed that Greer was malingering and competent. He was eventually convicted on all counts.

At sentencing, the government asked the court to increase the offense level for purposes of sentencing pursuant to the U.S. Sentencing Guidelines based on the premise that Greer had obstructed justice by pretending to be incompetent. The court granted an enhancement and increased the sentence from 185 to 210 months. Greer appealed to the U.S. Fifth Circuit Court of Appeals, claiming that the sentence enhancement undercut his right to be tried only if competent. The appellate court affirmed the finding of obstruction of justice by malingering, reasoning that malingering constitutes obstruction of justice because it involves egregiously wrong behavior that requires a significant amount of planning and inherent high risk that justice will indeed be obstructed. The court ruled that feigning incompetency is similar to altering evidence and creating a false record: "A defendant who playacts . . . essentially tries to create a record that includes inaccurate testimony and factual conclusions" (*Greer*, 1998, p. 235). Further, as part of its decision

the appellate court recommended that attorneys advise their clients to cooperate during assessment of abilities.

The publication of a National Academy of Neuropsychology position paper on symptom validity assessment also recognizes the importance of considering the appraisal of effort in neuropsychological practice, stating: ‘‘clinicians should be prepared to justify a decision not to assess symptom validity as part of a neuropsychological evaluation’’ (p. 421) (Bush, Ruff, Troster, Barth, et al., 2005). Furthermore, as Iverson (2003) stated in the context of forensic practice, "any neuropsychological evaluation that does not include careful consideration of the patient's motivation to give their best effort should be considered incomplete" (p. 138). Yet, according to a recent survey malingering tests failed to appear among the top 40 most frequently administered assessment instruments by practicing neuropsychologists (Rabin, Barr, & Burton, 2005). In addition, studies have shown that clinicians lack efficacy in detecting malingering solely on the basis of unstructured interviews. For example, psychiatrists are able to detect only approximately 50% of lies in interviews, which is no better than that which would be discovered by chance (Rosen, Mulsant, Bruce, Mittal, et al., 2004).

The need to assess malingering using a multi-modal approach that employs valid and reliable tests, measures, and techniques, and the inherent difficulties associated with making a determination of malingering are perhaps, no better illustrated that in the case of *United States v. Gigante* (1996). Indeed, carefully consider the following:

In 1996, Vincente Gigante was charged with murder and labor racketeering and subsequently found guilty of racketeering, racketeering conspiracy, extortion conspiracy, labor payoff conspiracy, and two counts of conspiring to murder in aid of racketeering.

Following his conviction, he moved for a new trial based on new evidence as to his competency to stand trial, arguing that district court judge Weinstein erred in finding that he was competent to stand trial. However, the appellate court refused to disturb the trial court's determination that defendant was competent to stand trial, stating that it was not an abuse of discretion where the decision was based on credible evidence (*U.S. v. Gigante*, 1999).

Weinstein was not the first judge to make a finding regarding Gigante's competency to stand trial. Gigante's trial had been previously assigned to Judge Nickerson, who conducted the first competency to stand trial hearings. At that time, four psychiatrists testified that Gigante was incompetent, although reservations were expressed that he might be malingering. Judge Nickerson then received testimony from former members of the Mafia, and made the factual findings that "Gigante was a forceful and active leader of the Genovese family from at least 1970 on" and that Gigante had put on a "crazy act" for many years in order "to avoid apprehension by law enforcement" (*United States v. Gigante*, 1996, at 976).[15] After being presented with these findings, two of the examining psychiatrists changed their opinion, indicating that they now thought Gigante was malingering; one said Gigante was competent to stand trial, and the other said it was quite possible that Gigante was competent.[16] The remaining experts held to

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[15] The court described in the findings Gigante's extensive efforts to hide his criminal activities and to evade prosecution by presenting himself as crazy. The record revealed that in addition to these measures Gigante also sought to mislead his doctors by inventing a false medical history and by concealing the true nature of his daily existence (*Gigante*, 1997).

[16] Dr. Rappeport testified that these findings made him think that it is quite possible that Gigante is competent to stand trial and that much or all of his mental illness may have been malingered. Dr. Schwartz stated that the findings convinced him that Gigante is fit to proceed. Likewise, after reading the findings, Dr. Portnow concluded that in 1991 Gigante was, in fact, competent to stand trial. However, he believes to a reasonable degree of medical certainty that Gigante has been

their earlier findings of incompetence. Judge Nickerson found "the weight of medical opinion to show that Gigante is mentally competent to stand trial" (*Unites States v. Gigante*, 1996, at 147).

In 1997, when Gigante renewed his claim of incompetence he submitted additional information regarding his purported mental deficiency, including testimony of expert defense witnesses who had performed a series of experimental tests, including PET scans. Dr. Buchsbaum, a defense expert, interpreted the PET scans and concluded that Gigante was suffering from organic brain dysfunction, possibly due to AD or multi-infarct dementia. However, Dr. Buchsbaum was not able to pinpoint the exact cause of the abnormality, nor could he quantify its level. Whatever the specific problem and etiology, he claimed it rendered the Gigante incompetent to stand trial (*U.S. v. Gigante*, 1997).

A second defense expert, basing his opinion on neuropsychological tests designed to assess malingering, concluded that Gigante was not feigning incompetency but, in fact, suffered from severe cognitive impairment, However, neither one of these experts had ever analyzed the defendant's blood to determine the amount of medication in his system

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incompetent to stand trial since 1995. Before reading the findings, Dr. Portnow had attributed Gigante's incompetency to the combined effects of schizo-affective disorder and organic brain disease, but he now believes that Gigante suffers only from organic brain disease. In contrast, Dr. Halpern testified that these findings had not changed his opinion that Gigante was incompetent to stand trial. He also stated that he could not accept the finding that Gigante was competent and malingering in 1991. He felt that accepting this finding would require him to accept that Gigante is presently competent and malingering, a point that he was unwilling to concede (*Gigante*, 1997).

at the time the tests were administered. Gigante, in fact, was taking Thorazine, Restoril, Lanoxin, Teneormin, Pamelor, Dalmane and other drugs at the time of the examinations and had been taking potent psychotropic medications for an extended period of time. In fact, government witness, Dr. Brodie, testified that (1) the defense produced no convincing testimony about the specific effects the various drugs taken by defendant may have had on the PET scans and neuropsychological tests, and (2) from the limited information presented, there was no way to tell if, or how much, the results of the tests were skewed by these medication (*U.S. v. Gigante*, 1997).

The court, subsequently, concluded that the defense experts' findings were (1) not consistent with other evidence in this case, (2) unreliable, and (3) based upon speculative scientific theories lacking full development, research, and support. More specifically the court argued that:

- Dr. Buchsbaum offered his opinion without having reviewed or compared any earlier PET scans of the defendant's brain; the alleged dysfunction as seen in the PET scans may have benignly existed in Mr. Gigante when he was an active criminal. Further, the PET scans were compared to those of a small group of people in order to ascertain if his brain was functioning normally. This control group apparently was not selected at random and most of its members grossly differed from defendant in age and background;
- Dr. van Gorp's findings were based upon speculative assumptions. For example, he argued that since defendant seemed to be trying on the cognitive tests and scored slightly above the score of "chance" guessing, the conclusion was valid that malingering was not probable.

- Defendant's experts failed to take into account the extensive testimony received by Judge Nickerson proving that defendant's mental difficulties had been feigned for many years (*United States v. Gigante*, 1997, at 147-148).

Vincente Gigante, subsequently, signed a statement admitting that he had been faking mental illness in order to avoid conviction (Newman, 2003).

The detection of malingering

It has been suggested that approximately 75% of attorneys prepare their clients for forensic neuropsychological evaluations by discussing the purpose and content of various tests and measures (Essig, et al., 2001). There is also evidence that attorneys brief their clients on the inclusion of measures designed to detect malingering. The most frequently reviewed test is the Minnesota Multiphasic Personality Inventory-2 (MMPI-2) (29%), followed by the Portland Digit Recognition Test (PDRT) (6%), and the Memory for 15-items Test (Rey-15). In addition to direct warnings of neuropsychological and effort measures, approximately 10% of attorneys also inform their clients of what types of information to disclose concerning their injury and 12% tell their clients what information not to disclose (Essig, et al., 2001). Few studies have examined the susceptibility of effort measures to attorney coaching. For example, Suhr and Gunstad (2000) reported that providing simulated malingerers with brain-injury information had no effect on their performance on the Auditory Verbal Learning Test. Similarly, a computerized version of the PDRT was found to be resilient to coaching (Rose, Hall, Szalda-Petreem, & Bach, 1998). In contrast, Lamb and colleagues (1994) demonstrated the susceptibility of the MMPI-2 to both coaching and brain-injury information. In fact, simulated malingerers, who were provided with information regarding brain-injury and/or

information regarding the ability of the MMPI-2 to detect a "fake-bad" profile, produced valid profiles with significantly elevated clinical scales similar to those obtained by individuals with true head injuries. More believable profiles were also observed on the Nonverbal Forced Choice Test, 21-Item Test, PDRT, and Recognition Memory Test, after participants were provided with information on how to beat these measures (Cato, Brewster, Ryan, & Guiliano, 2002; Dunn, Shear, Howe, & Ris, 2003; Gunstad & Suhr, 2001; Martin, Hayes, & Gouvier, 1996; Rose, et al., 1998).

Another issue to be considered is the fact that the vast majority of malingering research has relied on simulation design which utilizes non-clinical subjects, typically university undergraduates, asked to feign or malinger dysfunctions. Although it has been suggested that simulated malingerers are comparable to actual malingerers, studies utilizing the simulation design have historically been criticized for their lack of generalizability to actual malingerers (Brennan & Gouvier, 2006). A particular concern of this design is that the subjects usually employed in these studies have little or no experience with head-injury. This is considerably different from individuals involved in actual criminal cases; a large number of criminal defendants who are malingering have had experience with and recovered from, for example, TBI.[17] Clearly, the influence of attorney coaching is likely to effect and possibly invalidate the standard neuropsychological assessment (Brennan & Gouvier, 2006).

Further, according to Rogers and Correa (2008), tests designed to assess malingering are different for individuals malingering cognitive impairment and those

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[17] Previous literature has, in fact, suggested that the inclusion of individuals with a history of head-injury would be more generalizable to real-world malingerers (Cato, et al., 2002).

malingering mental disorder or illness. More specifically, in the former case, malingerers must put forth a convincing effort and make believable errors on cognitive measures while in the latter case, malingerers must create a convincing story about the onset and course of their disorder, deciding on the order, frequency, and effects of symptoms. Consequently, different detection strategies are needed for distinct domains of malingering (Rogers & Bender, 2003). Any assessment of FLD in the context of violent and aggressive behavior, therefore, must include tests of cognitive malingering as well as tests designed to assess malingering mental illness or psychological disorders. The importance of doing so is underscored by the fact that a well designed neuroimaging study, that seeks to assess FLD, requires the use of a valid and reliable psychological test known to activate the brain area of interest. For example, in assessing FLD, a finding of below chance performance on tests of frontal lobe dysfunction, and/or on tests designed to assess cognitive impairment can results in a diagnosis of FLD in the absence of any structural or functional abnormality. In addition, a finding of functional abnormality, without collateral information, is not sufficient to diagnose an individual with FLD since a link between any cognitive impairment and functional abnormality can only be inferred.

Detection strategies

There is no doubt that the assessment of malingering is not an easy task. There are, however, a number of strategies available to assist the clinician in making an informed decision. One technique frequently used in the assessment of malingering is the performance curve analysis. The performance curve compares the probability of correctly answering easy items versus more difficult items (Rogers, 1997). In individuals who are not malingering impairment or dysfunction, the clinician should expect to see a decrease

in correct responding as task difficulty increases. Evidence has shown that simulated malingerers do not generate the typical performance curve. In other words, malingerers fail a more-than-expected proportion of easy items compared to their performance on more difficult items (Frederick & Foster, 1991). One malingering measure that relies on the performance curve is the Dot Counting Test. This measure presents stimuli of varying (and mixed up) difficulty levels to determine the consistency of an individual's response time and error-rate (Lezak, 1995). In non-malingering subjects, typically no errors are committed and a positive correlation is observed between difficulty level and time to respond. If there is more than one pronounced discrepancy between the expected and observed patterns of response time and/or if more than two errors are made, malingering should be suspected. Overall, empirical evidence supports error-rate (performance curve) as a strong indicator of malingering (Binks, Gouvier, & Waters, 1996; Frederick, 2002).

Another strategy frequently employed is the floor effect. More specifically, there are many tasks that are easily accomplished by most individuals, including those with brain damage, and malingering detection utilizes this knowledge by examining floor effects. Floor effects are extremely low performances observed when malingerers misjudge the difficulty of easy tasks and perform more poorly than brain-damaged patients (Millis & Kler, 1995). One of the most frequently used floor effect measures is the Rey-15. A major drawback of this test is that it is, in fact, sensitive to true memory impairment and correlates considerably with measures of cognitive competence (Lezak, 1995; Vallabhajosula & van Gorp, 2001).

A third strategy, atypical test performance, suggests that test performance that is markedly different from accepted models of normal and abnormal brain functioning

should alert the clinician to the possibility of malingering. For example, it is well known that implicit memory is preserved following even the most severe brain-injury (Kuzis, Sabe, Tiberti, Merello, Leiguarda, & Starkstein, 1999). The theory behind this method is that the automatic and intentional uses of memory can be separated, and that "conscious control can be measured as the difference between performance when a person is trying *to* as compared with trying *not to* use information from some particular source" (Jacoby, 1991, p. 527). Therefore, any impairment on measures of implicit learning may be indicative of malingering. One measure that utilizes this concept is the Word Completion Memory Test (Hilsabeck, LeCompte, Marks, & Grafman, 2001).

Researchers have also looked at the predictive accuracy of validity indices designed to assess malingering. More specifically, many self-report measures of psychological functioning contain validity scales designed to detect if a subject is answering in a manner that invalidates the overall results. For example, the Minnesota Multiphasic Personality Inventory-2 (MMPI-2) has at least two indices that can be used to assist in the detection of malingering. The F or "infrequency" scale measures the extent to which a person answers in an atypical and deviant manner. A score of 70 or above is suggestive of possible malingering. The Dissimulation or F-K index determines the likelihood and direction of exaggeration. A score of 12 or greater indicates a fake bad profile, while a score of -12 or less indicates a fake good profile (Groth-Marnat, 1997). Similarly, the Personality Assessment Inventory (PAI) contains scales appropriate for use in malingering detection, such as the Negative Impression Management scale (NIM) which measures the degree to which a subject has presented an exaggerated, unfavorable impression of distress (Morey, 2003).

Finally, most of the literature aimed at defining assessment techniques that reliably assess malingering has been devoted to the development and validation of symptom validity tests (SVTs) [18] which were created on the tenet that, with adequate effort, all patients with the exception of the most severely neurologically impaired will score within the normal range (Weinborn, Orr, Woods, Conover, & Feix, 2003). To be more precise, symptom validity testing is a simple strategy based in binomial distribution theory; an individual with a legitimate impairment who cannot discriminate between the two stimuli presented should perform at chance levels over many trials. In contrast, malingerers are likely to select the wrong response deliberately and, thus, perform significantly below chance (Rogers, Harrell, & Liff, 1993). Perhaps most commonly known for their use in forensic settings SVTs can be crucial when making diagnostic decisions in situations involving criminal defendants. In addition, SVTs are more commonly being added to research protocols to screen out possibly invalid data. Without the support of passing SVT scores, lowered scores on other cognitive measures remain suspect because the possibility of malingering makes valid conclusions difficult, if not impossible, to ascertain (Gierok, Dickson, & Cole, 2005).

Recent literature has suggested that symptom validity testing is the best-validated strategy to assess for malingered cognitive impairments (Bush, et al., 2007; Duncan, 2005).[19] However, according to a survey of members of the National Academy of

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[18] In order to place maximum confidence in the ability to accurately interpret results from cognitive measures and/or tests of personality, a determination must be made that the examinee puts forth appropriate effort on tasks and responds honestly to questions. Symptom validity assessment is the process through which such determinations are made.

[19] Binder (2002) also argued that the strongest psychometric evidence of faking impairment occurs when a forced choice test result is significantly below chance performance.

Neuropsychology, the International Neuropsychological Society, and the American Psychological Association Division 40, SVTs are not among the top 40 most frequently administered assessment instruments. This may be due to the lack of knowledge that some psychologists have about the existence of SVTs, differential emphasis in training programs, philosophical opposition to utilization of these instruments, as well as limitations in survey methodology utilized (Rabin, Barr, & Burton, 2005).

A major criticism of this method is that of its low sensitivity. This method is extremely conservative and only the most blatant malingerers are caught. In response to this low sensitivity came the derivation of cut-off scores (Haines & Norris, 1995). A cut-off score typically represents the lowest score achieved by subjects with documented brain damage. Therefore, if a patient with minor, or no, documented brain-injury performs significantly worse than the cut-off, malingering is to be suspected (Haines & Norris, 1995). Utilizing cut-off scores improves the sensitivity of the forced-choice method, but at the cost of reduced confidence in the interpretation (specificity is lowered because of the increase in false positives (Rogers, 1997). A major advantage of symptom validity measures it that demographic variables do not affect test scores. For example, research findings have shown that age and intelligence do not impact performance on the Word Memory Test (WMT) (Brockhaus & Merten, 2004; Green & Flaro, 2003). Similarly, Delain and colleagues (2003) found no significant demographic differences between individuals passing or failing the Test of Memory Malingering (TOMM) with respect to age, gender, ethnicity, or years of education in a sample of criminal defendants,

while Tombaugh (1997) found the TOMM to be insensitive to age and years of education in a sample of community dwelling adults.

There is, at present, no single test that acts as the ‘‘gold standard’’ in the detection of malingering or symptom exaggeration and this lack of a “gold standard” is clearly illustrated by the following:

A recent study investigating neuropsychologists' beliefs and practices with respect to assessing malingering found that the five most frequently used measures were the TOMM, MMPI-2 F-K ratio and FBS scale, Rey-15, and the California Verbal Learning Test (CVLT). However, the TOMM, Validity Indicator Profile (VIP), WMT, and Victoria Symptom Validity Test (VSVT) were rated as most accurate for detecting suboptimal effort.[20] In addition, only approximately 79% of the respondents reported using at least one specialized technique for detecting malingering (Sharland & Gfeller, 2007).

It must be noted that numerous studies have shown that some of these tests lack sufficient specificity, sensitivity, PPP, and NPP to be relied upon as a sole measure of malingering. Therefore, since a single test of malingering may yield incorrect results (misclassifies an honest respondent as malingerer, or the reverse), the use of multiple measures to provide converging evidence of malingering has been highly recommended (Berry, Baer, Rinaldo, & Wetter, 2002; Farkas, Rosenfeld, Robbins, & van Gorp, 2006; Vallabhajosula & van Gorp, 2001). In fact, it may be argued that testifying to a jury that a lack of malingering was found on one test, is unduly prejudicial. Indeed, Federal Rules

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[20] According to a 2004 survey the Rey-15 and TOMM were the most frequently reported measures (Slick, Tan, Strauss, & Hultsch).

of Evidence requires the potential prejudicial effect of scientific testimony to be carefully balanced against its probative value. Thus, the use of multiple malingering measures might provide this balance in a manner that is more consistent with the legal standards set for the admissibility of scientific evidence (Saxe & Ben-Shakhar, 1999). Further, by employing multiple tests the likelihood for a subject, who has been coached, to convincingly feign impairment across multiple measures, is decreased. Also, by using more than one test it is more likely that a particular malingering test will ". . . be relevant to a specific patient's functional or genuine complaints, or both. Thus the best use of a single test will be in combination with other tests – always within the context of a patient's history and clinical presentation – which together will reduce the likelihood of prediction error" (Lezak, 1995, p. 792). Indeed, expert testimony indicating that a defendant passed multiple malingering measures would provide the court with information it needs to decide with a high degree of certainty that a claimed cognitive impairment or mental illness is valid and would more likely withstand a *Daubert* challenge (Thompson, 2002).

Frequently used symptom validity tests

1. Portland Digit Recognition Test (PDRT)

The PDRT is a 72 item (two sets of 36 items) test employing visual recognition of auditorily-presented five-digit number strings. The first 36 trials are referred to as the ''easy'' items and the second 36 are the 'hard'' items based on their apparent difficulty. The published cutoff scores for the easy, hard, and total items sets have been associated with a 0% false positive error rate (100% specificity) in non-compensation-seeking moderate to severe TBI patients (Bianchini, Mathias, Greve, Houston, & Crouch, 2001).

In terms of the test's sensitivity, Bianchini and colleagues (2001), found the PDRT to be positive in about 30% of compensation-seeking head trauma patients. Further, in a study using data from 262 TBI patients who were classified as not malingering, possibly malingering, and malingering based on the Slick, Sherman, and Iverson (1999) criteria found that the original PDRT cutoff scores detected between 20% and 50% of malingering TBI patients with a false positive error rate of 5% or less. When the false positive error rate was held at 5%, across all item sets, sensitivity was found to be as high as 70% (Greve & Bianchini, 2006). However, while it is highly likely that someone whose profile indicates malingering is actually feigning, the PDRT has also shown to yield a large number of false negatives (Rogers & Bender, 2003).

2. Test of Memory Malingering (TOMM)

The TOMM was developed to help detect feigned cognitive impairment. The TOMM requires examinees to memorize 50 simple pictures and then identify correct pictures from 50 two-picture items after two learning trials and a retention trial (15-minute delay). Examinees are given feedback on the correctness of their answers to facilitate learning. The probability of feigned memory impairment is said to be increased if subjects score significantly lower than 45 on Trial 2 or the retention trial on the TOMM, particularly if they score below chance levels. People showing sustained effort on the TOMM generally do not score below 45 on Trial 2 or the retention trial, including those with genuine neurological impairment, dementia, TBI, depression, anxiety, and mild retardation (Ashendorf, Constantinou, & McCaffrey, 2002; Iverson, Page, Koehler, Shojania, & Badii, 2007; Merten, Bossink, & Schmand, 2007; Rees, Tombaugh, & Boulay, 2001; Simon, 2007; Tombaugh, 1996).

Studies evaluating the reliability of the TOMM indicate that this test is highly sensitive to malingering but insensitive to true neurological and cognitive impairments. Indeed, sensitivity estimates range from 60% to 84%, and specificity estimates range from 90% to 100% (Bounds, 2005; Heinze & Purish, 2001; Teichner & Wagner, 2004; Weinborn, Orr, Woods, Conover, et al., 2003; Vallabhajosula & van Gorp, 2001). Furthermore, Powell and colleagues (2004) explored the effects of coaching on TOMM scores. Using the recommended cutoff score of 45 out of 50 correct to classify suboptimal performance the TOMM accurately classified 92.6% of the symptom-coached simulators and 96% of the test-coached simulators. In addition, a review of four studies found the TOMM to have 100% PPP, irrespective of the base rates of malingering in these studies (Vallabhajosula & van Gorp, 2001). Most recently, an evaluation of the TOMM also suggested that Trial 1 alone can be used in predicting overall performance on the TOMM. However, it may be argued that use of any non-standard administration is inappropriate and many no satisfy *Daubert* criteria (O'Bryant, Gavett, McCaffrey, O'Jile, 2008).[21]

3. Validity Indicator Profile (VIP)

The VIP was designed to be administered with tests that assess cognitive capacity. Results of the VIP test indicate whether the individual's performance on other tests of cognitive capacity should be considered a valid representation of his or her abilities. The VIP consists of two subtests each of which can be administered and scored separately: (1)

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[21] Ideally, all tests should have high sensitivity, specificity, NPP, and PPP; however, to date, few such tests exist. Consequently, Vallabhajosula and van Gorp (2001) have suggested that, to meet the *Daubert* standard of admissibility of scientific evidence, a test must have, at a minimum, a PPP of $\geq$80% . It has also been argued that, in clinical practice, malingering tests should have a specificity of at least 90% (Rüsseler, Brett, Klaue, Sailer, & Münte, 2008).

the nonverbal subtest (VIP-NV) presents 100 picture-matrix problems that require simple matching, complex matching, analogous decision making, progression, addition, subtraction, and abstraction and, (2) the verbal subtest (VIP-V) consists of 78 word definition problems. Test-takers are presented with a stimulus word and are asked to choose which of two possible answers is most similar in meaning to the stimulus. For both subtests, the items have a hierarchy of difficulty but are presented randomly with respect to item difficulty. Once administered, the items are re-ordered in terms of difficulty level and then scored (Frederick & Crosby, 2000).

The overall effectiveness of the VIP in correctly classifying the initial validation sample resulted in 73.5% sensitivity and 85.7% specificity rates. The sensitivity of the nonverbal subtest was 66% with a specificity of 90% while the sensitivity and specificity for the verbal subtest were 59% and 94% (Frederick, 1997). In general, this test has received favorable reviews and comments although some concerns, criticisms, and cautions have been voiced (Gebart-Eaglemont, 2001; Ivens, 2001; Ross & Adams, 1999; Vallabhajosula & van Gorp, 2001). More specifically, depending on the base rate of malingering, the VIP may or may not have sufficient sensitivity, specificity, PPP, and NPP (Vallabhajosula & van Gorp, 2001).

4. Victoria Symptom Validity Test (VSVT)

The VSVT is a computer-administered two-alternative forced-choice SVT designed to assess cognitive functioning. This test consists of a total of 48 items, which are presented in three blocks of 16 items. For each item administered, a five-digit study number is first presented on a computer screen during the study trial and then is immediately followed by the retention interval during which a blank computer screen is

shown. At the recognition trial two five-digit numbers are presented on the computer screen, one of which was shown initially in the study trial. The subject is then required to identify which of the two five-digit numbers was shown during the study trial. Subjects are also instructed to make their selection as quickly as they can without making mistakes (Slick, Hopp, Strauss, & Thompson, 1997).

The VSVT is reported to have almost 100% specificity, which is paramount in any test assessing symptom validity. Additionally, because the VSVT cut-off scores are based on binomial probability theory and are not norm-referenced, it is very unlikely that a patient who scores in the invalid range on the VSVT will do so for any reason other than response bias or malingering (Slick, et al., 1997). Further, a study examining the ability of the VSVT to discriminate between a control group and a group of simulated malingerers found that the VSVT exhibited excellent sensitivity and specificity as it correctly classified 88% of the controls and 89% of the simulated malingerers (Strauss, Hultsch, Hunter, Slick, et al. (2000).

5. Word Memory Test (WMT)

The WMT is a computer-based test that is designed to measure both verbal memory and biased responding (malingering). It measures memory on a number of dimensions and contains hidden scales, which serve to check the validity of the person's test scores. More specifically, this test was designed to assess a person's ability to learn a list of 20 word pairs and to evaluate a person's effort to perform well during testing. The WMT has been shown to have good validity (Williamson, Green, Allen, & Rohling, 2003).

The claims for validity advanced by the authors of this test are novel and scientifically provocative. More specifically, it has been suggested that effort, as measured by the WMT, (1) explains a substantial portion of variance in cognitive outcome after TBI and (2) interacts with injury severity to produce patterns of scores after TBI that cannot be explained by injury severity alone (Green, Lees-Haley, & Allen, 2002). Research findings on the diagnostic efficacy of the WMT, however, have been mixed. For example, a recent functional magnetic resonance image study employing the WMT has suggested that this test activates numerous cortical regions that are critical for cognitive effort. More specifically, given the extensive neural network necessary to perform the WMT, this study raised questions about what a WMT failure truly means in patients with TBI, who have an increased likelihood of disruption within this neural network of vision, language, attention, effort and working memory (Allen, Bigler, Larsen, Goodrich-Hunsaker, et al., 2007). In contrast, a study assessing the individual and joint malingering detection accuracy of the PDRT, TOMM, and WMT has shown that at published cut-offs, the PDRT and TOMM are very specific. In addition, joint classification accuracy was superior to that of the individual tests (Greve, Ord, Curtis, Bianchini, et al., 2008). It has also been suggested that this test is fairly resistant to attempts at coaching (Dunn, Shear, Howe, & Ris, 2003).

Other frequently used tests and indices

1. Minnesota Multiphasic Personality Inventory-2 (MMPI-2)

The MMPI-2 is one of the most widely used personality tests designed to assist mental health professionals in identifying psychopathology and personality structure. Further, validity indicators, in particular the F scales (F, Fb, Fp) and the F-K ratio can be

used to generate hypotheses regarding the potential for malingering. Although the MMPI-2 and its predecessor the MMPI were not constructed to identify the presence of malingering specifically, a substantial body of research has focused on the utility of these indicators to detect malingering. For example, a recent meta-analysis has found the F scale to be an effective scale but questioned the routine use of Fb (Rogers, Sewell, Martin, & Vitacco, 2003). Similarly, the F scale has proven to be the best at distinguishing psychiatric patients from research participants instructed to malinger (Bagby, Nicholson, Bacchiochi, Ryder, & Bury, 2002). Further, a study by Kucharski and colleagues (2004) revealed that neither the Fp scale, nor the revised Fp scale added to the F scale in predicting group membership. More specifically, the F scale alone correctly classified 80.8% of cases with no incremental accuracy added by either Fp, or the revised Fp.

Investigations of detection of malingering in neuropsychological settings have also demonstrated superior sensitivity of the Fake Bad Scale (FBS) in comparison to F and related scales (Greiffenstein, et al., 2002; Larrabee, 2003; Nelson, Sweet, & Demakis, 2006; Ross, Millis, Krukowski, Putnam, & Adams, 2004; Tsushima & Tsushima, 2001). However, other authors have expressed concern over the scale's specificity. More specifically, Iverson and colleagues (2002), in examining the specificity of FBS in a sample of prison inmates, inpatients, and medical outpatients found that, while the original FBS cut score of 20 was able to correctly classify most of the malingering inmates, this same cut score resulted in "unacceptably high rates of presumed false positive classifications" (p. 135). Butcher and (2003) similarly concluded that the scale "is likely to classify an unacceptably large number of individuals who are

experiencing genuine psychological distress as malingerers" (p. 473).[22] Furthermore, the F scale is not intended to assess cognitive effort and is generally thought to be insensitive to feigning of neurocognitive impairment (Greene, 2000; Larrabee, 2003). In addition, it has been suggested that the MMPI-2 validity scales are measuring a different construct than tests of malingered memory deficits, and therefore should be interpreted with caution (McCaffrey, O'Bryant, Ashendorf, & Fisher, 2003).

Although numerous studies have found that the F and F-K ratio indices demonstrate large effect sizes for discriminating between honest respondents and those who are feigning, cut scores for detecting malingering have greatly varied across these studies (Greene, 2000; Rogers et al., 2003). Regardless of the cut scores being used, however, the F-K ratio was found to lack sufficient scientific validity to be relied upon as a measure of malingering (see, e.g., Boccaccini, Murrie, & Duncan, 2006).

A new version of the MMPI-2, the MMPI-2-RF, which was released in early 2008, also includes validity scales (L-r and K-r) designed to assess underreporting of symptoms. Data from a recent study has indicated L–r and K–r are able to differentiate between individuals instructed to underreport from those who responded honestly. More specifically, the effect sizes derived from this study were mean *d*s = 1.19 and 1.13 for L and K, respectively (Sellbom & Bagby, 2008).

2. Millon Clinical Multiaxial Inventory - Third Edition (MCMI-III)

This inventory includes 175 true/false items scored on 24 content scales intended to correspond to major DSM-IV Axis I and II disorders. The two scales most often used

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[22] Butcher and colleagues (2003) also concluded that FBS is more likely to measure general maladjustment and somatic complaints than malingering.

to detect malingering are the Disclosure Index (X), which assesses willingness to admit to difficulties, and the Debasement Index (Z), which identifies a tendency to overstate emotional and personal problems. Although the test's manual proposes that raw scores above 178 on Scale X denote excessive symptom exaggeration, no cutoff scores are recommended for use with Scale Z beyond the suggestion that base rate scores above 85 tend to be associated with malingering (Millon, Davis, & Millon., 1997). Few studies have examined the ability of the MCMI-III to detect malingering and those that have, have reported poor classification accuracy for this measure (Daubert & Metzler, 2000; Schoenberg, Dorr, & Morgan, 2003).

3. Structured Interview of Reported Symptoms (SIRS)

The SIRS was developed to assist in the evaluation of feigning of psychosis. It is administered orally, in less than an hour, and includes interpretation instructions. This 172-item generalized measure of feigning mental illness includes eight primary scales and five supplementary scales. Scores from the primary scales are classified into one of four categories: honest responding, indeterminate, probable feigning, and definite feigning (Rogers, Bagby, & Dickens, 1992). Rogers and colleagues' (1992) validation studies yielded a PPP of 99% and a NPP of 64.9% with a base rate of 51%. With a base rate of 20–25%, PPP remained in the range of 96–97%, while the NPP increased to 86%–89% (Rogers, Bagby, & Dickens, 1992). Further, the SIRS manual, summarizing the studies available at the time of its publication, states that the SIRS sensitivity is 48.5% and its specificity is 99.5%. Therefore, if the recommended cut-off scores are used, the scale tends to under-identify malingering but is unlikely to falsely identify an individual as malingering (Rogers, Bagby, & Dickens, 1992). The relatively low sensitivity

supports the developers' recommendation that the SIRS be used in conjunction with other indicators of malingering.

4. Personality Assessment Inventory (PAI)

This 344-item self-administered inventory is organized into 11 clinical scales (somatic complaints, anxiety related disorders, depression, mania, paranoia, schizophrenia, borderline features, anti-social features, alcohol problems, drug problems), five treatment scales (aggression, suicidal ideation, stress, non-support, treatment rejection), and two interpersonal style scales (dominance and warmth). It also contains four validity scales (inconsistency, infrequency, negative impression (NIM), positive impression (PIM) and four additional validity indexes (MAL, RDF, DEF, CDF that have been developed since the original introduction of the PAI (Morey, 1996). In addition, the PAI has norms based on correctional populations, which is something the MMPI-2 lacks (Morey, 1996).

Bagby and (2002), found the (RDF) to be clearly superior to other PAI validity indicators for use with a non-forensic population. More specifically, neither the NIM scale nor the Malingering Index (MAL) effectively detected malingered profiles. In contrast, a more recent regression analysis using malingering vs. non-malingering as criterion found that NIM scale but not the RDF or the MAL significantly differentiated the malingering from the non-malingering group (Kucharski, Toomey, Fila, & Duncan, 2007). Further, it has been suggested that NIM has the greatest utility as a faking detection scale as the level of pathology increases, while RDF is better suited to identify subtler and/or more sophisticated attempts at feigning that go overlooked by NIM (Morey & Lanier, 1998; Rogers, Sewell, Morey, & Ustad, 1996). Indeed, NIM and RDF were

found to be most sensitive to unsophisticated attempts to dissimulate by inpatient psychiatric patients (Baity, Siefert, Chambers, & Blais, 2007).

To date, few studies have examined the ability of the PAI to detect malingering in a forensic sample (Boccaccini, Murrie, & Duncan, 2006; Rogers, Ustad, & Salekin, 1998; Wang, Rogers, Giles, Diamond, et al., 1997). However, one study, using the SIRS to classify forensic patients as honest or malingering, found that with the recommended cut score of ≥77 the NIM correctly classified 84% of malingerers and 74% of honest respondents. MAL was clearly less effective as a screening measure, with a cut score of ≥3 correctly identifying only 47% of malingerers and 86% of honest respondents. The performance of RDF was also poor, with the recommended cut score correctly identifying only 51% of malingers and 72% of honest respondents (Rogers, Ustad, & Salekin, 1998; Wang, Rogers, Giles, Diamond, et al., 1997). More recently, with the recommend cut score of ≥77, the NIM was found to have 91% sensitivity, 65% specificity, PPP of 53%, and NPP of 95%, while the MAL, with the recommended cut score of ≥5, demonstrated 13% sensitivity, 97% specificity, PPP of 67%, and NPP of 72% (Boccaccini, Murrie, & Duncan, 2006).

5. Rey-15 Item Test (Rey-15)

This test consists of five rows of three characters each on a card (e.g., A B C, 1 2 3, a b c). Subjects are shown the card for 10 seconds and told to study it carefully in order to later recall as many of the items as possible. The Rey-15 is presented to subjects as a very difficult memory test but it is, in fact, very simple because of the redundancy among items. Therefore, even individuals with significant impairments can perform the test without difficulty. However, it is assumed that malingerers will not know this. Instead,

they will reason that, in order to register a result of being memory impaired, they should only recall only a few items (Reznek, 2005).

Research findings have established some variability in performance among individuals with genuine memory impairment. Consequently, different cutoff scores have been proposed. For example, Lezak (1995) argued that only in cases of severe brain damage should an individual be unable to recall less than nine items, while Lee and colleagues (1992) concluded that a cutoff score of seven should arouse suspicion of malingering of mental deficits. Research studies have also indicated that adjusting the cutoff higher increases the Rey-15's sensitivity but decreases its specificity; therefore, a cutoff score of seven has been recommend to increase diagnostic efficiency (Lee, Lohring, & Martin, 1992).

A meta-analysis of available studies using the Rey-15 has shown that this test has high specificity but low sensitivity (with a cut-off score of seven the specificity is 95% and the sensitivity is 10%; with a cut-off score of eight the specificity decreases to 92% and the sensitivity decreases to 9%; when employing the usual cut-off score of nine the specificity drops to 85% and the sensitivity increases to 36% (Rezneck, 2005). Indeed, Rüsseler and colleagues (2008) contend that the sensitivity of the Rey-15 is too low for clinical use in the assessment of malingering, while Vallabhajosula and van Gorp (2001) have argued that if used as a single procedure, this test appears to be insufficiently sensitive to detect malingering of cognitive impairment, irrespective of the cutoff score applied. Ideally, a test designed to detect malingering of cognitive deficits should be sensitive to malingering but insensitive to genuine cognitive dysfunction. The Rey-15

falls short of this ideal precisely because of its sensitivity to genuine impairment (Vallabhajosula & van Gorp, 2001).

6. California Verbal Learning Test (CVLT)

The CVLT is a list-learning task that requires the subject to learn a list of 16 words throughout five trials and evaluates recall and recognition after a delay. Unlike some neuropsychological tests, the CVLT quantifies numerous cognitive components of verbal learning memory within a single test. More specifically, it examines retroactive and proactive interference in a number of ways as well as the strategy that the individual employs to remember information presented to them verbally (Lezak, Howieson, & Loring, 2004). Although, the CVLT is among the most frequently used methods for evaluating learning, its utility in the detection of cognitive malingering has also been investigated. In fact, this test is presumed to be effective in detecting malingering because malingerers often overestimate the amount of memory impairment that is associated with head injuries and disorders characterized by brain pathology (Coleman, Rapport, Millis, Ricker, & Farchione, 1998). Trueblood (1994) examined the classification accuracy of Total Correct from Trials 1 through 5 (Total 1-5) and Recognition Hits using a known-groups design of mild TBI patients and found the CVLT's sensitivity to be approximately 70%, with a false positive error rate of 5% to 10%. Millis and colleagues (1995) also using a known-groups design, studied the same two variables along with Long Delay Cued Recall (LDCR) and Recognition Discriminability and correctly identified about 85% of TBI patients, with a false positive error rate of only about 8%. In contrast, the CVLT has demonstrated an average sensitivity of 51% in clinical TBI patients and 64% in simulators, with an average false

positive error rate in moderate-severe TBI of about 15% (Sweet, Wolfe, Sattlberger, Numan, Rosenfeld, & Clingerman, 2000). In another study the CVLT was shown to have 80% sensitivity and 97% specificity in correctly identifying patients attempting to feign memory deficits (Connor, et al. as cited in Delis, Kramer, Kaplan, & Ober, 2000). Most recently, however, this test was found to have sensitivity of only 50%, but excellent specificity (> 95%) (Curtis, Greve, Bianchini, & Brennan, 2006). The Forced Choice Recognition and the Critical Item Analysis indices of the CVLT-II have also been identified as being potentially useful, brief screening indicators of effort or malingering in neuropsychological assessment. Indeed, a recent study has suggested that, while a negative finding should not be relied upon as evidence of adequate effort, a positive finding, in the absence of frank dementia, is strongly suggestive of inadequate effort and indicates the need for further testing (Root, Robbins, Chang, & van Gorp, 2006).

7. Wechsler Adult Intelligence Scale-Third Edition (WAIS-III)

Numerous investigators have sought to determine if the Digit Span (DS) subtest of the WAIS-III can accurately detect feigned cognitive impairment. For example, Mathias and colleagues (2002), using the DS subtest as an indictor of effort in patients with TBI, obtained a 67% sensitivity rate and a 93% specificity rate. In contrast, using a cutoff score of $\leq$7 on this subtest yielded an overall correct classification rate of 62.5% with a false positive rate of 0% (Schwarz, Gfeller, & Olivieri, 2006), while Strauss, et al. (2002) obtained a 47% true positive rate and a false positive rate of 5%. Iverson and Tulsky (2003) examined the WAIS-III Vocabulary and DS indicators of six clinical groups with chronic alcohol abuse, Korsakoff's syndrome, left temporal lobectomy, right temporal lobectomy, TBI and AD. Their findings provide further support that it is

clinically rare for individuals with neurological impairment to score below the established DS cutoff scores that are indicative of low effort.

Researchers have also utilized discrepancy scores between the DS and Vocabulary subtests on the WAIS-R and WAIS-III to detect malingering. For example, using the WAIS-III Vocabulary-DS subtests discrepancy cutoff of ≥2 (established by Mittenberg et al., (1995), has shown to yield an overall correct classification rate of 78.1%. However, while the classification rate was relatively high, the Vocabulary-DS discrepancy score also produced a false positive rate of 36.6%. Thus, an unacceptably high number of individuals who were not malingering impairment were labeled as malingerers.

8. Diagnosis of APD and psychopathy

As previously noted, the DSM-IV-TR (2000) advises that malingering should be considered whenever there is a lack of cooperation, the distress reported exceeds observed disability, or APD is present. Indeed, studies have shown that criminal defendants diagnosed with APD score significantly higher than defendants diagnosed with a personality disorder other than APD or no personality disorder on validated measures of malingering (Kucharski, Falkenbach, & Duncan, 2004). However, Kucharski and colleagues (2004) also found that a high percentage (>40%) of those diagnosed with APD did not score above accepted cut-offs for suspecting malingering and that a diagnosis of APD was, in fact, a poor discriminator of malingerers from those believed to be responding honestly. It has also been suggested that a diagnosis of psychopathy may be a good indicator of malingering. There is much intuitive appeal in assuming that psychopathy and malingering are associated, given that deception is an important clinical

component of psychopathy. However, there is little research that supports this association. For example, Gacono and colleagues (1995) studied defendants acquitted of criminal offenses by reason of insanity and found that those acquitted by reason of insanity, who subsequently admitted feigning psychiatric disorder during their trial, were more likely to present with APD and scored higher on the Psychopathy Checklist Revised (PCL-R; Hare, 1991), than those who continued to claim they were mentally ill at the time of the offense. In contrast, Poythress, Edens, and Watkins (2001) found that psychopathic prison inmates were no better at avoiding detection of malingering than nonpsychopathic subjects.

More recently, subjects with severe psychopathy were again found to score higher than those with low psychopathy on measures of malingering. However, these findings are significantly diminished by the observation that while psychopaths score higher on validated measures of malingering, a high proportion of those with severe psychopathy did not show evidence of exaggeration (Kucharski, Duncan, Egan, & Falkenbach, 2006). Based on these findings the authors concluded that although psychopathy does appear to discriminate malingerers from non-malingerers, the relatively poor sensitivity and specificity of psychopathy calls into question its clinical and forensic utility in the detection of malingering. More specifically, "there is a strong likelihood that the reliance on psychopathy as a basis for opining that a defendant is malingering would not clear any evidentiary standard for admissibility if a test of its scientific merit were conducted (Kucharski, Duncan, Egan, & Falkenbach, 2006, p. 642).

The foregoing, clearly, underscores the importance of employing tests that have high specificity, sensitivity, PPP and NPP, in assessing neuropsychological functioning,

especially within the forensic setting. Indeed, since all currently available neuropsychological tests and indices lack adequate predictive accuracy to be relied upon as a sole measure of malingering, the use of multiple measures or tests is not only justified, but necessary. Further, regardless of the classification accuracy of any single indicator of malingering, malingering detection techniques are not perfect and should not be used in isolation for the clinical diagnosis of malingering. As has been noted in many articles, manuals, and book chapters, and systematized by Slick, et al. (1999) criteria, a formal diagnosis of malingering should be based on the integration of diverse clinical information. Consequently, in establishing a valid and reliable methodology for the assessment of frontal lobe functioning, in the context of violent and aggressive behavior, malingering will be determined with the validity scales of the MMPI-2 and the TOMM.

CHAPTER 5. ASSESSING FRONTAL LOBE FUNCTIONING - NEUROPSYCHOLOGY

"Your joys and your sorrows, your memories and your ambitions, your sense of personal identity and free will, are in fact no more than the behavior of a vast assembly of nerve cells and their associated molecules" (Crick, 1994).

The use of neuropsychologists as expert witnesses in criminal cases has been a standard practice for some time and the general purpose of an evaluation in this context is to determine the presence or absence of any psychiatric, cognitive, or psychological disorders. More specifically, the expert witness is typically required to answer a variety of questions such as (1) is impairment present, (2) what is the cause of the impairment, and (3) what functional deficits are related to genuine impairments. Neuropsychologists attempt to answer these questions through a variety of tests and methods, such as a careful review of medical and other records, the clinical interview, and the administration of established neuropsychological tests. However, it is the results from formal tests that usually provide the most weight in establishing cognitive and/or psychological deficits within the forensic setting.

It is, therefore, not surprising that, over the years, courts have become increasingly skeptical about measures and techniques used to form expert opinions and, consequently, have imposed more rigorous guidelines for the admissibility of neuropsychological testimony at trial. Indeed, as previously discussed, under *Daubert* courts are asked to examine the validity and reliability of the instruments used to collect data and to analyze the validity and reliability of the inferential methods used by clinicians to generate opinions and diagnoses. *State v. Cavalieri* (1995) clearly illustrates these points.

In *Cavalieri*, the New Hampshire Supreme Court, focusing directly on the admissibility of the Millon Clinical Multiaxial Inventory-II and the MMPI-2 in addressing sex offender profiles, held that the heterogeneity of test data for sex offenders precluded its admissibility. More specifically, the Court questioned whether studies of sex offenders admitting to their offenses also applied to those denying them, requiring that accurate classifications be rendered based on scientifically acceptable and reliable methodology. Thus, under the *Daubert* standard, a conclusion will not be admissible simply because a part of the methodology is scientifically valid; "the entire reasoning process must be valid" (*Daubert*, 1993, p. 2796).

Various other standards for using psychological assessment methods within forensic settings have also been outlined in the professional literature. For example, Marlowe (1995) provided a hybrid model for operationalizing the admissibility of psychometric evidence using a blend of scientific and legal principles. This hybrid model is based on many of the standards found in the Federal Rules of Evidence, such as relevance; scientific, technical, or specialized knowledge; as well as psychometric principles, such as reliability and validity. Similarly, Heilbrun (1992) suggested the following guidelines for selection of psychological tests in forensic evaluations: (1) commercial availability with a documented manual and peer review; (2) reliability established at a level of 0.80 or explicit justification for lower coefficients; (3) relevance of the test to some legal issue with appropriate validation research; (4) standardized method of administration; (5) applicability to the population and purpose for which the test is used; (6) objective tests with actuarial data applications; and (7) availability of a means for assessing response style/malingering.

Initial Assessment

Although clinicians rely heavily on neuropsychological test results and more recently on imaging results to make a determination of FLD, most clinical or forensic assessments, usually begin with a clinical interview. The first structured and semi-structured interviews[23] were created in the late 1980s and, since then, have been widely used to maximize the reliability and validity of DSM-IV-TR (2000) diagnoses. The exact format of these inventories and interviews may vary; however, they all are based on questions that are keyed into the diagnostic criteria of the DSM-IV-TR (2000).

Since, under *Daubert,* the entire methodology must be valid, any test, measure, and/or technique employed to reach a conclusion must be valid, including the interview process. Furthermore, since, as previously discussed, many Axis I and Axis II disorders have frontal lobe dysfunction and/or aggressive and violent behavior as part of their symptomatology, any assessment of FLD in the forensic context must also include a valid assessment of the presence or absence of any of these disorders.[24]

There are a number of structured, semi-structured, and self-report measures available designed to assess Axis I and Axis II disorders; however the most frequently used diagnostic tools used for assessing these disorders are the Structured Clinical Interview for the DSM-IV-TR (SCID), the Millon Clinical Multiaxial Inventory-III

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[23] A true structured interview asks questions in a specific way and leaves little or no room for interviewers to use their own clinical judgment. Semi-structured interviews also have specified questions, they allow interviewers to use their own skills in eliciting more information, and include more open-ended questions (Lesser, 1997).

[24] Axis I disorders relevant to this discussion include delirium, dementia, bipolar and major depressive disorder, and schizophrenia. Axis II disorders relevant to this discussion include antisocial, borderline, and obsessive-compulsive personality disorders.

(MCMI-III), the Minnesota Multiphasic Personality Inventory-2 (MMPI-2), and the Personality Assessment Inventory (PAI).

1. Structured Clinical Interview for the DSM-IV-TR (SCID)

The SCID is a semi-structured interview for making Axis I and Axis II diagnoses using DSM-IV-TR classifications. The clinical syndromes found on Axis I of the DSM-IV-TR represent acutely disturbing, often fluctuating, and transient patterns of symptoms that are typically experienced by an individual as ego dystonic or foreign to the self. In contrast, the personality disorders diagnosed on Axis II represent pervasive and stable patterns of attitudes, thoughts, and behaviors that are commonly experienced by a person as ego syntonic or not foreign to the self (Haddy, Strack, & Coca, 2005). The first scale, SCID-I, consists of a present mental state interview that provides differential diagnosis for Axis I disorders (primarily mood and substance abuse disorders). The second scale, SCID-II, closely follows the language of the DSM-IV-TR Axis II personality disorders criteria (see Appendix A for examples of questions). Thus, there are 12 groups of questions corresponding to the 12 personality disorders. The scoring is equally simple: either the trait is absent, sub-threshold, true, or there is insufficient information to code (First, Gibbon, Spitzer, Williams, & Benjamin, 1997). The SCID has undergone numerous revisions and has gained widespread use as a criterion measure in several studies to cross validate a number of other diagnostic instruments and clinical diagnoses (Messina, Wish, Hoffman, & Nemes, 2001).

To date, only a few studies have sought to assess the reliability of the SCID, but those that have, have reported good-to-excellent test-retest reliability of the diagnosis of APD generated by the SCID-II in psychiatric and substance-abusing populations with

kappas ranging from 0.75 to 0.95 (Kranzler, Tennen, Babor, Kadden, 1997; Kranzler, Kadden, Babor, Tennen, et al., 1996).[25] More recently, Farmer and Chapman (2002) found that inter-rater and test-retest reliabilities of Axis I disorders assessed by SCID-I were excellent for any Axis I disorder and all investigated Axis I diagnoses, such as substance abuse and major depression. These results were consistent with findings of previous studies using the Structured Clinical Interview for DSM-III, DSM-III-R, and DSM-IV-TR criteria (Skre, Onstad, Torgersen, & Kringlen, 1991; Zanarini, Skodol, Bender, Dolan, et al., 2000). Further, the inter-rater reliability of personality disorders assessed by the SCID-II was found to be good to excellent for the presence of any personality disorder, for cluster B and cluster C personality disorders, for the combination of personality disorders from more than one cluster, and for dimensional rating of all personality disorders (Farmer & Chapman, 2002).[26] Finally, the validity of a diagnostic assessment technique is generally measured by determining the agreement between the diagnoses made by the assessment technique and some hypothetical "gold standard." Unfortunately, to date, a gold standard for psychiatric diagnoses remains elusive. However, a number of studies have used the SCID as the "gold standard" in determining the accuracy of clinical diagnoses (Shear, Greeno, Kang, et al., 2000; Steiner, Tebes, Sledge, et al. 1995).

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[25] Kappa ranges between 0 and 1, with 0 indicating agreement no better than chance, and 1 indicating perfect agreement. It has been suggested that coefficients above 0.75 indicate excellent agreement, those between 0.60 and 0.74 indicate good agreement, those between 0.40 and 0.59 indicate fair agreement, and those below 0.40 indicate poor agreement (Messina, Wish, Hoffman, & Nemes, 2001).

[26] Cluster B disorders include antisocial, borderline, histrionic, and narcissistic personality disorder. Cluster C disorders include avoidant, dependent, and obsessive-compulsive personality disorder.

2. Personality Inventories

Any neuropsychological evaluation would be incomplete without the assessment of emotional status and personality. It is, therefore, not surprising that neuropsychologists also frequently employ personality inventories as part of the assessment of an individual's personality, affect, interpersonal function, and response style. The assessment of psychopathology is especially crucial because remarkable personality profiles frequently impact the neuropsychological findings. Consequently, many neuropsychologists administer at least one personality inventory as part of a standard assessment battery and, generally, rely on the MCMI-III, MMPI-2, or PAI (Camara, Nathan, & Puente, 2003).

a. Millon Multiaxial Clinical Inventory-III (MCMI-III)

The MCMI-III consists of 175 true/false items designed to assess basic personality styles, severe personality disorders, and clinical syndromes. The MCMI was developed to make the pathologies of personality operational and has undergone several revisions to maximize the similarity between DSM criteria and the MCMI scales. The first 10 scales of the MCMI-III are designed to detect basic personality patterns, namely, schizoid, avoidant, dependent, histrionic, narcissistic, antisocial, compulsive, passive-aggressive, aggressive/sadistic, and self-defeating personality. The MCMI-III also includes three pathological personality patterns (schizotypal, borderline, and paranoid). In addition, there are nine clinical syndrome scales (anxiety, somatoform, hypomania, dysthymia, alcohol abuse, drug abuse, psychotic thinking, psychotic depression, and psychotic delusions) as well as a scale that measures subjects' response tendencies (the internal validity scale) (Millon, 1997). To date, very few studies have assessed the

reliability and validity of this MCMI-III. For example, a meta-analysis of the MCMI-III and Axis II disorders concluded that the MCMI-III was insufficiently validated (Rogers, Salekin, & Sewell, 1999). Specifically, these authors contended that (1) the MCMI-III scales lack sufficient "construct validity" to be used in forensic settings and (2) the MCMI-III scales cannot be used to diagnose DSM-IV personality disorders since the test may generate errors in about 80% of diagnosed cases. More recently, it has been suggested that the MCMI-III can be used as a screening inventory for trait prevalence, but diagnosing personality disorders should be done by aggregation of as many different methods as possible to gain information from different sources and reduce inherent method variability (Rossi, Haube, van den Branden, & Sloore, 2003). Finally, it has been argued that any forensic expert who uses the MCMI-III is likely to encounter vigorous opposition to the use of this instrument in their forensic assessment (McCann, 2002).

b. Minnesota Multiphasic Personality Inventory-2 (MMPI-2)

The MMPI-2 is one of the most widely used personality tests and was originally designed for clinical diagnosis. The MMPI-2 is now commonly used in the forensic setting, and it is well accepted as a valuable tool for assessing a variety of factors in this context. More specifically, while the purpose of the MMPI-2 may vary, it is generally used to (a) evaluate the nature and extent of emotional distress, (b) evaluate acute as well as chronic psychopathology, (c) document symptom presentation, (d) evaluate the potential effects of psychological variables (e.g., depression and anxiety) on cognitive test performance, and (e) provide evidence of exaggeration or malingering. The MMPI-2 is the most widely used self-report measure designed to assess psychopathology. It consists of 567 true/false questions and is composed of (a) 10 clinical scales (CS)

addressing patterns of psychopathology, (b) 15 content scales describing common clinical issues that the subject has endorsed, (c) numerous validity scales for evaluating whether or not a subject is malingering, and (d) hundreds of specialized research scales that often have either focused or limited clinical applications (Lees-Haley, Iverson, Lange, Fox, et al., 2002). Although the MMPI-2 is frequently used within the clinical and forensic setting, most research on the MMPI-2 has focused on its utility as a measure of malingering. Indeed, few studies have investigated the predictive accuracy of the various CS. However, it has been shown that in a sample of adult inpatients with a primary psychotic disorder (PPD) or a primary mood disorder without psychotic features (PMD) the MMPI-2 was able to correctly classify PPD and PMD patients 70% of the time (Dao, Prevatt, & Horne, 2008). In addition, a meta-analysis assessing the predictive accuracy of the MMPI-2 Depression scale found that this scale has a PPP of .68 and a NPP of .58, with a sensitivity of .64 and a specificity of .62 (Gross, Keyes, & Greene, 2000). Further, it has been suggested that the Addiction Potential Scale (APS) and the Addiction Acknowledgement Scale (AAS) discriminate well between substance abuse, non-clinical, and psychiatric samples. More specifically, the AAS was found to discriminate between the substance-abuse and non-clinical samples better than the APS, whereas the APS appeared to distinguish between substance-abuse and psychiatric samples better than the AAS (Weed, Butcher, McKenna, & Ben-Porath, 1992).

The MMPI-2 CS were subsequently restructured to address problems in the original CS, including inclusion of questionable subtle items, and the lack of theoretical grounding (Tellegen, Ben-Porath, Sellbom, Arbisi, et al., 2003). Research findings on the validity of the restructured clinical scales (RCS) have been mixed. For example, Nichols

(2006) asserted that the original CS are better suited for the prediction of psychiatric diagnoses than the RCS because of the original scales' "syndromal fidelity"; that is, the multidimensional makeup of the CS is consistent with multifaceted diagnostic syndromes. However, this assertion has been contradicted by empirical findings showing that the RCS outperform the CS in predicting psychiatric diagnoses (see, e.g., Sellbom, Graham, & Schenk, 2006; Simms, Casillas, Clark, Watson et al., 2005; Tellegen, Ben-Porath, Sellbom, Arbisi, et al., 2006). Further, in a study of substance abusers, the RCS demonstrated equivalent or improved convergent and discriminant validity compared to their CS counterparts (Forbey & Ben-Porath, 2007). It has also been contended that the CS are better than the RCS for assessing complex mental disorder, such as psychopathy (Caldwell, 2006).

Although the MMPI-2 is considered to have high clinical utility, clinicians and attorneys often have erroneous beliefs about what the MMPI-2 can and cannot do. First, the MMPI-2 is not likely to withstand a *Daubert* challenge if offered as evidence of brain injury since this it does not measure neuropsychological impairment. Second, assertions that the MMPI-2 indicates specific causation are indefensible under *Daubert*. In fact, a valid MMPI-2 provides evidence related to the condition of the subject, but not the cause of that condition. The test, however, is relevant to causation arguments in an indirect fashion. For example, a normal MMPI-2 might serve as evidence that event X has not caused the subject to be mentally ill. Similarly, an MMPI-2 profile indicative of, for example, schizophrenia may suggest that some problems were pre-existing and not simply produced *de novo* by a recent causal agent (Lees-Haley, Iverson, Lange, Fox, et al., 2002).

c. Personality Assessment Inventory (PAI)

The PAI, authored by Morey (1996), is a 344-item multi-scale self-report measure of psychological functioning that assesses constructs relevant to personality and psychopathology evaluation, such as depression or aggression, in various contexts including forensic assessment. The PAI has 22 non-overlapping scales, providing a comprehensive overview of psychopathology in adults. The PAI contains four types of scales: 1) validity scales, which measure the respondent's approach to the test, including faking good or bad, exaggeration, or defensiveness; 2) clinical scales, which correspond to psychiatric diagnostic categories; 3) treatment consideration scales, which assess factors that may relate to treatment of clinical disorders or other risk factors but which are not captured in psychiatric diagnoses (e.g., suicidal ideation); and 4) interpersonal scales, which provide indicators of interpersonal dimensions of personality functioning (Morey, 1996).

To date, few studies have systematically assessed the validity of the PAI. However, research has supported the validity of the Schizophrenia Scale for identifying psychotic spectrum disorders and their associated features (Morey, 2007). Further, PPP, NPP, sensitivity and specificity for the Depression Scale were found to be .62, .62, .70, and .52, respectively, while the Drug Problem scale was found to have .65 sensitivity and specificity and a PPP and NPP of .61 and .69, respectively (Edens & Ruiz, 2008). It has also been argued that that despite its psychometric superiority over the MMPI-2, the PAI should not be considered a diagnostic measure. While the PAI may assess useful patterns of psychopathology that are related to DSM-IV-TR diagnoses, it does not formally evaluate the DSM-IV-TR inclusion and exclusion criteria (Rogers, 2003).

At present, no "gold standard" exists for the determination of DSM-IV-TR diagnoses; however, it may be argued, that the *Daubert* requirement that expert opinions are based on reliable measures also requires that clinical interviews conducted as part of a forensic evaluation, have a scientific basis and are methodologically sound. Using only structured or semi-structured interviews is crucial especially within the forensic setting, since doing so (1) decreases the information variance (clinicians ask the same questions), and (2) opinions based on these techniques are more likely to withstand a *Daubert* challenge. Therefore, this evaluation of FLD in the context of aggressive behavior will begin with the SCID and followed by the MMPI-2.

Neuropsychological testing

Neuropsychologists frequently use scientifically validated tests to evaluate brain functions. While neurological examination and CT or MRI scans assess the physical, structural, and metabolic condition of the brain, the neuropsychological examination and PET, SPECT, and fMRI scans are the only way to formally assess brain function. Neuropsychological tests cover a wide range of mental processes from simple motor performance to complex reasoning and problem solving. In almost all tests, quantitative results are compared with some normative standard, including data from groups of non-brain injured persons and groups of persons with various kinds of brain injury. If the norms are based on age and educational achievement, valid comparison can be made between an individual's performance and that of persons in known diagnostic categories as well as persons who do not have a diagnosis of brain injury (Spreen & Strauss, 1997).

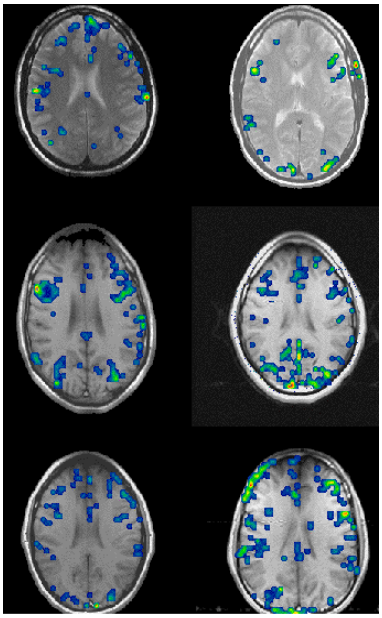
Qualitative assessment of neuropsychological tests provides a look at the processes an individual may use in producing the quantitative scores. Since the analysis

of the pattern of performance among a large number of tests is a key factor in the neuropsychological assessment, the selection of tests used should sample a wide range of functional domains. Indeed, the combination of objective scores, behavioral process observations, and consistency in emerging pattern of results, along with a comprehensive clinical history constitute the art and science of neuropsychological assessment. To that end, most neuropsychologists select a unique combination of tests that focus on the diagnostic and examination questions of interest for an individual (Spreen & Strauss, 1997).

Recall that executive function refers to the ability to plan and execute behavior, while constantly updating representations and goals in an always-changing environment. Central to these control functions is the ability to appropriately select actions that are behaviorally advantageous, and conversely to withhold or suppress actions that are either inappropriate or because they interfere with completion of motor and/or cognitive goals. Numerous studies have suggested a relationship between violent and aggressive behavior, and frontal lobe/executive dysfunction, and most of the evidence for the neural structures involved in executive functions has come from laboratory tasks, such as the Stroop or WCST. More specifically, individuals who have problems with aggression are known to have reduced prefrontal cortical inhibition, which is exhibited as below norm performances on tests of executive functioning, such as the Stroop Color and Word Test (Stroop) and the WCST (Demakis, 2004; Leung, Skudlarski, Gatenby, Peterson, et al., 2000; MacLeod & MacDonald, 2000;; Morgan & Lilienfield, 2000 Ravnkilde, Videbech, Rosenberg, Gjedde, & Gade, 2002).

Much emphasis has also been placed on the ability to suppress inappropriate and unwanted actions, often referred to as response inhibition, not only because of its importance for control of human behavior, but also because deficient response inhibition has been hypothesized to contribute to, for example, frontal lobe lesions, or ADHD (Picton, Stuss, Alexander, Shallice, et al., 2006). Indeed, numerous fMRI studies of response inhibition, using tests such as the WCST, Stroop, or Go/No-go Task, have consistently revealed frontal lobe activation; however, localization within the frontal cortex has varied across studies (Liddle, Kiehl, & Smith, 2001; Mostofsky, Schafer, Abrams, Goldberg, et al., 2003; Wager, Sylvester, Lacey, Nee, et al., 2005). Recent functional neuroimaging studies have also suggested that two areas of the prefrontal cortex, the DLPFC and the ACC may be particularly important for performing executive functioning task (Fellows & Farah, 2005).

Figure 19. fMRI Scans of Activity with the Stroop



fMRI scans of regional activation with the Stroop. While commonalities in activation can be seen across the subjects (e.g., dorsolateral and medial frontal lobe, inferior parietal lobe, occipital cortex), variability between the six subjects is apparent (http://www.uwm.edu/~neuropsy/fmri.html).

Furthermore, as previously noted, executive dysfunction has been linked to psychopathy. In fact, there have been suggestions that specific regions of the frontal cortex, such as the ACC, rather than the entire frontal cortex, may be implicated in psychopathy (Blair, Newman, Mitchell, Richell, et al., 2006). The literature on the effects of damage to the ACC, or other regions of the frontal cortex, however, is inconsistent at best. For example, reduced ACC activation was found in individuals with psychopathy during an emotional memory and an aversive conditioning task (Kiehl, Smith, Hare, Mendrek, et al., 2001). Based on these results, Kiehl and colleagues (2001) concluded that some aspects of psychopathy may be related to abnormal function in the ACC. In contrast, other studies have failed to find a systematic effect of ACC damage on the size of the Stroop effect (Fellows & Farah, 2005; Stuss, Floden, Alexander, Levine, et al., 2001).

Clearly, functional neuroimaging techniques alone cannot prove that a given brain region is critical for a specific task performance or cognitive process; neuropsychology is also required. More specifically, evidence from loss-of-function studies, which employ neuropsychological tests that are known to activate the prefrontal cortex, is necessary. Furthermore, while results from neuroimaging techniques can provide another perspective of the relationship between FLD and aggressive behavior, all currently available neuroimaging modalities suffer from inherent limitations. Therefore, any evaluation of FLD in the context of violent and aggressive behavior must also include the administration of a number of valid and reliable tests specifically designed to assess frontal lobe/executive functioning.

Neuropsychological tests of frontal lobe/executive functioning

The most important question to be asked in assessing the validity of the Stroop, the WCST, or any other test presumed to be sensitive to frontal lobe dysfunction is: Are scores on tests of frontal lobe functioning true indicators of prefrontal function? The solution to this question requires that the cognitive operations behind these test scores can be equated to specific brain processes or brain regions (Demakis, 2004).

Both neuroimaging and lesion studies with healthy subjects agree that an intact DLPFC is required for correct WCST performance. In fact, all neuroimaging studies undertaken to date have reported an increase in the metabolism of prefrontal regions during WCST execution and active areas have mostly corresponded with the DLPFC (see, e.g., Barcelo, 2001; Ravnkilde, Videbech, Rosenberg, Gjedde, & Gade, 2002). However, activation has also been reported in the ventro-medial prefrontal cortex and the OFC (see, e.g., Blair, Newman, Mitchell, Richell, et al., 2006). Further, fMRI and EEG studies of the Stroop effect have also revealed selective activation of the ACC, a prefrontal structure in the brain which is hypothesized to be responsible for conflict monitoring (Demakis, 2004; Swick & Jovanovic, 2002). These findings are compatible with our present understanding of higher brain functions in terms of distributed neural networks and with evidence of interconnecting pathways between prefrontal and posterior association cortices as well as with subcortical structures such as the basal ganglia.[27] Indeed, current models of central executive function recognize that executive processes involve a network of brain structures that are not exclusively localized to the frontal lobes (Demakis, 2004). In addition, it has been shown that patients with frontal

[27] The frontal lobes are vast and constitute approximately 30% of the total cortical surface; aggregation of these various areas into a ‘‘lobe’’ neglects known functional subdivisions between frontal regions (Goldman-Rakic, 1984).

damage often do not have executive deficits, while patients without frontal damage often score in the impaired range of measure of executive functioning (Andres, 2003). Thus, to accurately identify FLD researchers need to identify the specific region damaged, not simply the entire lobe as is typically reported (Stuss & Alexander, 2000). To date, only one research team has performed such studies and found that subjects with damage to the inferior medial frontal cortex demonstrate less impairment on the WCST than patients with damage to DLPFC (Stuss, Levine, Alexander, Hong, et al., 2000).

The majority of research studies undertaken to date have failed to make such clear neuroanatomical distinctions and, consequently, subregions with varying functions are often collapsed together, obscuring potential differences (Demakis, 2004). However, making such a distinction is crucial, since, aggressive and violent behavior has been predominantly linked to the OFC. Indeed, as has been previously discussed, psychopathic individuals show no indications of impairment on measures of executive function linked to the DLPFC, such as the WCST, but do score in the impaired range on measures linked to the OFC, such as the Porteus Maze Test (LaPierre, Braun, & Hodgins, 1995; Mitchell, Colledge, Leonard, Blair, 2002; Roussy & Toupin, 2000).

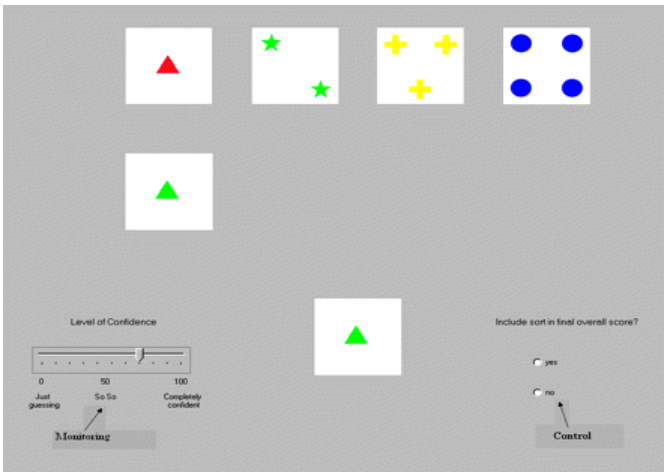
The foregoing clearly underscores the (1) importance of employing only those tests, measures, and techniques that have been proven to be accurate and valid indicators of frontal lobe dysfunction, and (2) need for a multimodal and multidisciplinary approach to the assessment of frontal lobe functioning in the context of violent and aggressive behavior. Let us now look more closely at the predictive accuracy of the most frequently used tests designed to assess frontal lobe/executive functioning.

1. Wisconsin Card Sort Test (WCST)

In clinical practice, this test is widely used by neuropsychologists, clinical psychologists, neurologists and psychiatrists with patients who have acquired brain injury, neurodegenerative disease, or mental illness, such as schizophrenia. The WCST is considered a measure of executive function because of its reported sensitivity to frontal lobe functioning in general, and DLPFC dysfunction, in particular. As such, this test allows the clinician to assess the following "frontal" lobe functions: strategic planning, organized searching, utilizing environmental feedback to shift cognitive sets, directing behavior toward achieving a goal, and modulating impulsive responding (Heaton, Chelune, Talley, Kay, & Curtiss, 1993).

Initially, a number of stimulus cards are presented to the subject. The subject is not told how to match the cards; however, he/she is told whether a particular match is right or wrong. The mistakes made during this learning process, are analyzed to arrive at a score. The original WCST used paper cards and was carried out with the experimenter on one side of the desk facing the participant on the other. However, since the early 1990s, computerized versions of the task have been available. The latter has the advantage of automatically scoring the test, which was quite complex in the manual version. The test takes approximately 12-20 minutes to complete and generates a number of psychometric scores, including numbers, percentages, and percentiles of categories achieved, trials, errors, and perseverative errors (Heaton, Chelune, Talley, Kay, & Curtiss, 1993).

Figure 20. Wisconsin Card Sort Test



(A representative screen from the computerized version of the Wisconsin Card Sorting Test (Koren, Seidman, Goldsmith, & Harvey, 2006).

Many clinical studies on WCST performance have suggested that left frontal damage affects WCST performance more than right frontal damage; however others have found no difference in laterality of damage in the frontal cortex (Demakis, 2003; Goldstein, Obrzut, John, Ledakis, & Armstrong, 2004). It has also been argued that the WCST has limitations and numerous studies have questioned to what extent this test adequately assesses frontal lobe functioning. Indeed, many studies have shown that damage in non-frontal or diffuse (frontal and nonfrontal) brain regions affects WCST performance. More specifically it has been reported that damage to temporal, subcortical, hippocampal, and cerebellar regions result in impaired performance on this test (Giovagnoli, 2001; Igarashi, Oguni, Osawa, Awaya, et al., 2002; Mukhopadhyay, et al., 2008).

Various neuroimaging studies on WCST performance have also reported a significant increase in metabolic or neural activity and in a majority of the cases the increased activation was found in the DLPFC and ventrolateral prefrontal cortex (Gonzalez-Hernandez, Cedeno, Pita-Alcorta, Galan, et al., 2002; Lie, Specht, Marshall, &

Fink, 2006; Monchi, Petrides, Petre, Worsley, et al., 2001). However, many functional neuroimaging studies show a greater network of activation during the WCST. For example, efficient performance on the WCST has been shown to increase neural activity in a widespread network of anatomical regions including areas of the inferior parietal lobes, and temporo-parietal association and primary and association visual cortices (Gonzalez-Hernandez, et al., 2002; Konishi, Hayashi, Uchida, Kikyo, et al., 2002; Lie, Specht, Marshall, & Fink, 2006). It has also been established that the frontal lobes are, functionally, heterogeneous, suggesting that the WCST can only reveal specific aspects of frontal lobe processing, such as the shift from using an old rule to a new rule. (Maes, Vich, J. & Eling et al., 2006; Maes, Maes, Damen, & Eling, 2004; Miller & Cummings, 2007).

Although the WCST is the most widely used test for the assessment of frontal lobe/executive function, surprisingly, to date not a single study has attempted to systematically assess the sensitivity, specificity, PPP, and NPP of this test. However, a recent meta-analysis has concluded that the WCST is, indeed, sensitive to frontal lobe damage, though the effect size between frontally and non-frontally damaged participants was in the small to medium range (0.33).[28] The effect size increased to larger than 1.0 (i.e., greater than a standard deviation), when certain moderator variables, such as administration format or area damaged within the frontal lobes, were assessed (Demakis, 2003).

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[28] In statistics, effect size is a measure of the strength of the relationship between two variables.

As discussed above, the WCST is not specific to frontal lobe function; it does not engage one specific brain area, nor is it a global feature of the whole brain, but involves a widespread neural network of both cortical and subcortical brain structures each of which carries out distinct and specific operations (Fernandez-Duque & Posner, 2001). While an intact DLPFC is necessary to perform attentional set-shifting, it is not enough for the execution of other cognitive operations utilized in the task. Therefore, in its original form the WCST cannot offer a valid description about the type and severity of cognitive deficits, or the anatomical regions in which these deficits occur (Bowden, Fowler, Bell, Whelan, et al., 1998). Based on these findings, the WCST should not be taken as a sole measure of prefrontal functioning. Nevertheless, one may still want to use the WCST scores as an index of the general status of a subject's executive functioning, regardless of its anatomical implications (Lezak, 1995).

2. Stroop Color and Word Test (Stroop)

This test consists of a word page with color words printed in black ink, a color page with 'X's printed in color, and a color-word page with words from the first page printed in colors from the second page (the color and the word do not match). The test-taker looks at each sheet and moves down the columns, reading words or naming the ink colors as quickly as possible, within a time limit. The Stroop yields three scores based on the number of items completed on each of the three stimulus sheets. In addition, an interference score, which is useful in determining an individual's creativity, cognitive flexibility, and reaction to cognitive pressures can also be calculated (Golden, 1978).

Figure 21. Stroop Color and Word Test

Green Red Blue

Purple Blue Purple

Blue Purple Red

Green Purple Green

The Stroop effect refers to the fact that naming the color of the first group of words is easier and quicker than the second.

A number of different test versions have been developed with variations in the color and number of the test items, the number of subtests, and the administration procedure. Despite these variations, the basic paradigm of the Stroop test has remained the same: an individual's performance on a basic task (e.g., reading names of colors) is compared with his or her performance on an analogous task in which a habitual response needs to be suppressed in support of an unusual one (i.e., naming the ink color that incongruously named color words are printed in). The increase in time taken to perform the latter task compared with the basic task is referred to as "the Stroop interference effect" and is considered to be a general measure of control and cognitive flexibility, or executive functioning (Davidson, Zacks, & Williams, 2003; Moering, Schinka, Mortimer, & Graves, 2003; Golden, 1978).[29] Since its development, the Stroop has been utilized to investigate aspects of various psychological disorders, such as ADHD and schizophrenia. The task was further developed by separating the task into four different stages: naming

[29] The Stroop is presumed to be a general measure of executive functioning and dorsolateral prefrontal functioning, in particular.

color fields, congruent color words, incongruent color words, and combined. The additional strain put on the executive function of the brain are presumed to allow for a more precise diagnosis (Davidson, Zacks, & Williams, 2003; Moering, Schinka, Mortimer, & Graves, 2003).

To date, few studies have examined the predictive accuracy of the Stroop. However, a recent meta-analysis assessing the validity of this test has suggested that the Stroop alone would not be sufficient to discriminate between frontal and non-frontal subjects. More specifically, the amount of overlap between the distributions of these two groups at effect sizes ranges from approximately 70% to 89%, indicated little separation of groups and thus relatively poor sensitivity (true positives) and specificity (true negatives) (Demakis, 2004). Similarly, Wildgruber and colleagues (2000) concluded that the Stroop is very poor at discriminating control subjects from individuals with frontal lesions (false negatives 69.2%).

3. Trail Making Test (TMT)

The TMT has been widely used as a measure of scanning, visuomotor tracking, cognitive flexibility, and divided attention, and is usually administered in two parts, A and B. Part A requires the subject to link in ascending order a series of 25 numbers (1-2-3 . . .) randomly distributed in space. The subject instructed to start their "trial" at the circle marked *Begin* and continue linking numbers until they reach the endpoint (circle marked *End*). Part B is similar, although instead of just linking numbers the subject must alternately switch between a set of numbers (1-13) and a set of letters (A-L), again linking in ascending order (1-A-2-B . . .) (Lezak, Howieson, & Loring, 2004).

The value of the TMT as a measure of frontal lobe functioning is questionable, at best. For example, a study involving TBI patients found no correlation between lesion volume and location, and scores on the WCST and the TMT. From these findings the authors concluded that (1) none of these measures can distinguish specific frontal lobe dysfunction and (2) the TMT, if used in isolation, does not add anything unique about frontal lobe integrity and neuropsychological functioning in TBI patients (Anderson, Bigler, & Blatter, 1995). Further, Stuss and colleagues (2001) have suggested that subjects with dorsolateral frontal damage are most impaired on Part B of the TMT, while Demakis (2004) found that Part A was more sensitive to frontal lobe damage than Part B. Other studies have also found considerable brain activity outside the frontal lobes. For example, Nickel and colleagues (2003) performing functional imaging with 18-fluoro-deoxyglucose PET on patients with mesial temporal lobe epilepsy have shown that patients with hypometabolism in the left-lateralized temporal regions had significantly impaired TMT performance in comparison to control subjects (Zakzanis, Mraz, & Graham, 2005).

4. Porteus-Maze Test (PMT)

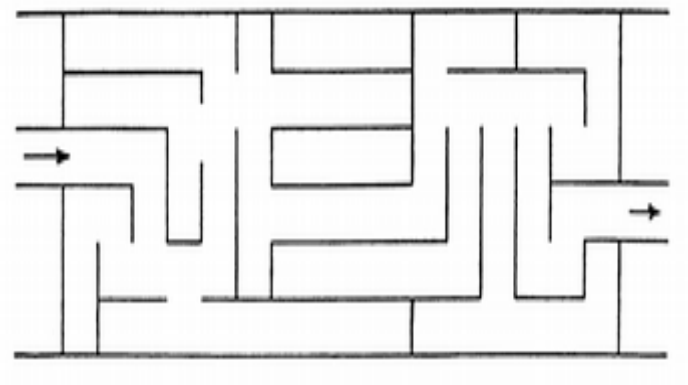
This test was developed as a technique for measuring planning ability. Porteus (1965) believed that planning was fundamental to intelligent behavior and initially devised the procedure as a culture-free means of screening for mental deficiency. Studies of the effects of psychosurgery provided opportunities to evaluate the usefulness of the PMT as a neuropsychological measure and have generally indicated its sensitivity to frontal lobe resection and disconnection (Riddle & Roberts, 1978). The PMT requires the subject to solve a series of mazes of increasing difficulty by drawing a continuous pencil

line from a given start point to a goal point. There are currently three forms of the PMT: the Vineland Revision form, the more difficult Extension series, and the most difficult Supplemental series. The three series of mazes are progressively more difficult in order to compensate for practice effects that have been observed on repeated administrations. Both quantitative and qualitative scoring can be derived. Qualitative aspects of performance on the PMT are evaluated by noting instances of careless, uncritical responding (e.g., entering blind alleys that occur very early or very late in the maze) (Riddle & Roberts, 1978).

In the context of cognitive research in psychiatry, this test is often used to investigate cognitive deficits or to evaluate treatment effects in schizophrenia, dementia, and alcoholism. Mazes have also been used to investigate personality traits such as impulsivity. Further, below norm performance on maze tasks has been related to disturbed executive functions and, therefore, a dysfunction of the PFC (Krikorian & Bartok, 1998). However, although mazes used in the context of spatial learning tasks have been applied in brain imaging studies, the literature lacks functional brain imaging studies on Porteus Maze-like tasks. Indirect evidence for the critical role of the prefrontal cortex comes from lesion studies, which have found impaired performance in patients with lesions of the prefrontal cortex (Riddle & Robert, 1978). The only functional neuroimaging study undertaken to date has suggested that the PMT activates a large network from visual to parietal regions, as well as the bilateral areas of the PFC, and subcortical, and cortical motor areas. From these findings, Kirsch and colleagues (2006) concluded that the PMT is a suitable method to quantify prefrontal cortex dysfunction in

patients suffering from schizophrenia and other neuropsychiatric disorders, using fMRI methodology (Kirsch, Lis, Esslinger, Gruppe, et al., 2006).

Figure 22. Porteus Maze Test-Vineland Revision



It has been suggested that the usefulness of the PMT as a measure of brain dysfunction is well established, but the predictive accuracy of this test has yet to be systematically assessed. However, in a recent study that assessed executive functioning of violent and nonviolent adult offenders, no significant difference was found between the two groups (Greenfield &Valliant, 2007).

5. Controlled Oral Word Association Test (COWAT)

The purpose of this test is to evaluate the spontaneous production of words within a limited amount of time. More specifically, the subject is asked to produce orally as many words as possible, beginning with a given letter of the alphabet. There are three trials administered, each employing a different letter (e.g., FAS, CFL) and subjects are allowed 60 seconds for each trial. During this task, subjects are prohibited from saying proper nouns (e.g., Carl, California) or saying the same word using a different ending (e.g., cancel, canceled) (Loonstra, Tarlow, & Sellers, 2001). Test performance is measured by calculating the total number of acceptable words produced for all three

letters. Errors and perseverations (word repetitions) are not included in this score. Errors include words that begin with the wrong letter, are proper nouns, or words that differ from a previous response by tense, plurality, or grammar usage. Changing a word ending to produce a new word that refers to a noun (e.g., "teach" and "teacher") is considered acceptable and such instances are scored as two separate words (Loonstra, Tarlow, & Sellers, 2001).

The COWAT is believed to be sensitive to executive functions such as cognitive flexibility, strategy utilization, suppression of interference, and response inhibition, and has been used as an activation task in neuroimaging studies designed to assess frontal lobe functioning (Abwender, Swan, Bowerman, & Connolly, 2001). From these findings it has been concluded that left anterior prefrontal cortex and ACC dysfunction is associated with below norm performance on this test (Audenaert, Brans, Van Laere, Lahorte, et al., 2000; Poldrak, Wagner, Prull, Desmond, et al., 1998). Further, according to Strauss and colleagues (2006), the COWAT is a sensitive indicator of brain dysfunction; however, to date, not a single study has systematically assessed the scientific validity and evidentiary reliability of this test.

6. California Verbal Learning Test-II (CVLT-II)

The CVLT-II tests levels of recall and recognition, semantic and serial learning strategies, serial position effects, learning rates, recall consistency, degree of vulnerability to proactive and retroactive interference, short term and long term retention of information, perseverations and intrusions in recall, and false positives in recognition. The test currently consists of two lists of 4 words from four categories (furniture, vegetables, ways of traveling, and animals) presented to the patient. The CVLT-II

measures both recall and recognition of the word lists over 5 different trials. It also measures delayed and cued recall. CVLT-II administration requires that a list of 16 words be presented to the patient for memorization. The patient is then instructed to recall those words over the course of five trials, again after a distracter list and with cues to facilitate memory (Delis, Kramer, Kaplan, & Ober, 2000).

The CVLT-II was developed to enhance diagnosticians' accuracy in identifying and characterizing different memory disorders by evaluating the magnitude of learning and memory impairments, and by evaluating the cognitive processes leading to impaired performance. The CVLT-II is well suited to measure subtle changes in verbal learning and memory ability and is presumed to be sensitive to temporo-hippocampal dysfunction (Delis, Massman, Butters, Salmon, et al., 1991; Osuji & Collum, 2005).

Little is known about the predictive accuracy of this test; however, findings from a recent study suggest that the Recall Discriminability index of the CVLT-II may be useful in improving the diagnostic accuracy of memory disorders across dementia populations (Delis, Spencer, Wetter, Jacobson, et al., 2005). It has also been argued that, if a subject does well on this test, a clinician may reasonably conclude that the subject's verbal memory is intact. On the other hand, if a subject's performance on the CVLT is impaired, further testing may be warranted to more clearly determine the precise nature of the underlying cognitive deficit (McDowell, Bayless, Moser, Meyers, et al., 2004).

7. Category Test (CT)

The Category subtest of the Halstead-Reitan Neuropsychological Test Battery is comprised of a total of 208 pictures consisting of geometric figures. For each picture, individuals are asked to decide whether they are reminded of the number 1, 2, 3, or 4 and

then they press a key that corresponds to their number of choice. If they chose correctly a chime sounds. If they chose incorrectly a buzzer sounds. The key to this test is that one principle, or common characteristic, underlies each subtest. The numbers 1, 2, 3, and 4 represent the possible principles. If individuals are able to recognize the correct principle in one picture, they will respond correctly for the remaining pictures in that subtest. The next subtest may have the same or a different underlying principle, and individuals must again try to determine that principle using the feedback of the chime and buzzer. The last subtest contains two underlying principles (Spreen & Strauss, 1997).

The CT is considered the battery's most effective test for detecting brain damage, but does not help determine where in the brain the problem is occurring. The test evaluates abstraction ability, or the ability to draw specific conclusions from general information. Related abilities are solving complex and unique problems, and learning from experience (Spreen & Strauss, 1997. Surveys also indicate that the CT, whether selectively administered or within the Halstead-Reitan Neuropsychological Test Battery continues to be among the most widely utilized measures by neuropsychologists. More specifically, the CT has been ranked 9th in frequency of use by neuropsychologists (Camara & Puente, 2000).

Although the CT has been used for decades, little is known about the diagnostic efficiency of this test. In fact, to date not a single study has sought to assess the validity and reliability of this test. Further, while this test may be sensitive to cerebral dysfunction a false-positive rate of as high as 18% has been reported (Anderson, Bigler, & Blatter, 1995; Choca, Laatsch, Wetzel, & Agresti, 1997).

8. Go/No-go Task (GNG)

The traditional GNG design involves only two stimuli: a Go stimulus and a No-go stimulus. Subjects are instructed to respond rapidly, generally with a button-press, to presentation of Go stimuli only, and response inhibition is measured by the ability to appropriately withhold responding to No-go stimuli. While some studies employ a more traditional GNG task design, with a single Go stimulus and single No-go stimulus other researchers use more complex designs involving multiple Go cues (Fassbender, Murphy, Foxe, Wylie, et al. 2004; Wager, Sylvester, Lacey, Nee, et al., 2005). For example, in one frequently used version of the task X's and Y's are alternately presented on the screen, and infrequently there is a two-letter repeat, which is the No-go signal; after presentation of an X, Y becomes the Go signal and X the No-go signal, and vice versa (Hester, Murphy, Foxe, Foxe, et al., 2004; Kelly, Hester, Murphy, Javitt, et al., 2004). It has also been shown that the DLPFC is important for representing task set and instructions, both of which are critical to complex GNG tasks (Courtney, 2004). Further, it has been suggested that increased response time variability, or inefficient performance, during a simple GNG is associated with activation of the right prefrontal cortex (Simmonds, Fotedar, Suskauer, Pekar, et al., 2007). Since there is no standardized version of the GNG, no reliability and validity data exist. However, fMRI studies of response inhibition have found frontal lobe activation, although localization within the frontal cortex have varied across studies (Fassbender, Murphy, Foxe, Wylie, et al., 2004; Mostofsky, Schafer, Abrams, Goldberg, et al., 2003; Wager, Sylvester, Lacey, Nee, et al., 2005).

As the foregoing indicates, all currently available tests designed to assess frontal lobe functioning lack sufficient predictive accuracy to be relied on as a sole measure of

frontal lobe dysfunction. However, for purpose of this discussion, the WCST will be used to assess frontal lobe functioning.

CHAPTER 6. ASSESSING FRONTAL LOBE FUNCTIONING – NEUROIMAGING

> "While nothing is easier than to denounce the evil doer, nothing is more difficult than to understand him" (Simon, 2003).

For centuries, criminologists and philosophers have speculated about the possible biological causes of violence and aggression. For example, in 360 B.C., Plato wrote: "For no man is voluntarily bad; but the bad becomes bad by reason of an ill disposition of the body ... [which] happens to him against his will" (Kirchmeier, 2004, pg. 631). Centuries later, Victor Hugo predicted that crime eventually would be seen as a disease, because it is due to factors, outside the offender's control, including biology (Kirchmeier, 2004). Although aggressive and violent behavior is seldom due to a single sociological, biological, or psychological variable, we are closer than ever before in identifying the biological roots of aggression and violence. Indeed, many neuroscientists and mental health professionals now refer to "crime as a disease," the "psychopathology of crime," and "the neurobiology of violence," and a biological brain-proneness toward violence is now widely accepted by neuroscientists (Kirchmeier, 2004; Raine, 1993; Volavka, 1995).

As previously noted, many neuroimaging and neuropsychological studies have suggested that the prevalence rate of brain dysfunction among criminal populations and the prevalence of mental disorders among individuals with criminal justice system involvement are extremely high. It is, therefore, not surprising that the number of defendants seeking to admit evidence of brain dysfunction or mental illness, especially in the form of neuroimaging scans, has increased dramatically over the past few years and will likely continue to increase. However, while our increasing knowledge of structural and functional impairments in violent defendants has helped to clarify, at least to some

extent, the neurobiological underpinnings of aggressive behavior, optimal integration of these findings requires cooperation among many disciplines, such as medicine, criminology, sociology, psychology, and neuroscience (Kirchmeier, 2004).

The important role neuroimaging evidence can play in criminal cases is perhaps no better illustrated than in the case of *Roper v. Simmons* (2005). Consider the following:

In *Roper*, the U.S. Supreme Court entertained a challenge, under the Eighth Amendment's injunction against cruel and unusual punishment to a state law permitting the execution of juveniles who were under the age of eighteen at the time they committed a capital offense. Among the numerous amicus briefs submitted, the briefs by the American Psychological Association (APA) and the American Medical Association (AMA), in particular, captured the public's imagination. Indeed, both briefs made novel use of neuroimaging based evidence to argue that adolescents were categorically less morally blameworthy than adults and, as a result, not deserving of the ultimate criminal sanction of death. More specifically, these briefs cited structural and functional neuroimaging studies showing that the neocortical regions of the brain, which are believed to be responsible for risk assessment, impulse control, and high-level cognition, are not yet fully developed in adolescents. The APA and AMA briefs seemed to have had an impact on the Court's consideration of *Roper*. Indeed, in the opinion, which affirmed the Missouri Supreme Court's conclusion that applying the death penalty to juveniles runs afoul of the Eighth Amendment, the Court agreed with the briefs' arguments, reasoning that juveniles are less blameworthy principally because their disposition to criminal violence is due to "transient immaturity" rather than "irreparable corruption" (*Roper*, 573).

The neurobiological theory of violence set forth by the *Roper* amici, with its focus on FLD, represents one of the main mitigation arguments used by neuroscientists. Indeed, those who represent criminal defendants often "are looking for that one pixel in their client's brain scan that shows ... a malfunction in the normal inhibitory networks," which would allow them to demand leniency on the grounds that their client could not control his actions (Thompson, 2005, pg. 52). Other experts have argued that PET and SPECT scans "have no scientifically supportable exculpatory role in assessing or predicting an individual's responsibility in the commission of a crime" and may mislead a judge and jury (Mayberg, 1996, pg. 200; Reeves, Mills, Billick, & Brodie, 2003).

Although the extent to which neuroimaging findings can be used as mitigating or exculpatory evidence remains the subject of much debate, many defendants, encouraged by some successes, now regularly present evidence of FLD and a growing body of scholarly literature encourages the use of such evidence (Redding, 2004). The foundation for using neuroimaging evidence in criminal trials lies in a massive and ever growing body of scientific literature on both the neuroanatomical and neurochemical basis for aggression and violence. However, while there are many exciting possibilities for how neuroscience and the law may partner, it is important to keep in mind the following major issues and limitations associated with neuroimaging evidence:

- Neuroimages can not tell us what or how person was thinking at the time the crime was committed, nor can they tell, with reliable accuracy, what a person is thinking while being scanned. An image can only provide a post hoc explanation (Raine, 1993). "No pixel in a brain will ever be able to show culpability or nonculpability" (Gazzaniga, 2005, p. 100).

- Neuroimages provide only one window of many into the multiple influences on behavior that may be relevant to understanding why someone acted in an aggressive or violent manner.
- Brain imaging is not yet a pure science. The interpretation of brain scans, just as the interpretation of test data, is, in fact, somewhat subjective.
- Even if brain abnormalities are found, individual differences in the extent and location of the injury, and in recovery, and plasticity, present major problems for the interpretation of brain images in the legal setting (Mobbs, Lau, Jones, & Frith, 2007). While these problems can be reduced in research through averaging across many individuals, these are critical issues when examining a single individual.
- Neuroimaging studies conducted on violent populations have examined group effects and most studies have examined adult males. Thus, the results cannot be generalized to other populations. If brain imaging is to be applied to the forensic evaluation of a single patient, a standardized set of tests, procedures, and imaging parameters are needed to achieve more valid conclusions (Mobbs, Lau, Jones, & Frith, 2007).
- Correlations between violent behavior and brain function are, at best, imperfect, calling into question both the diagnostic and predictive accuracy of brain imaging evidence.
- The generation of an image involves many assumptions, corrections, and various levels of analysis. These steps are not standardized from one technology to the next, or from one machine, or laboratory to the next. Thus, the image being read varies,

depending on the signal threshold, color, contrast, or the brand of machine used (Reeves, Mills, Billick, & Brodie, 2003).

- The conditions under which a scan was obtained may be idiosyncratic and impossible to compare in a meaningful way with data obtained at other centers (Reeves, Mills, Billick, & Brodie, 2003).

The basics of neuroimaging

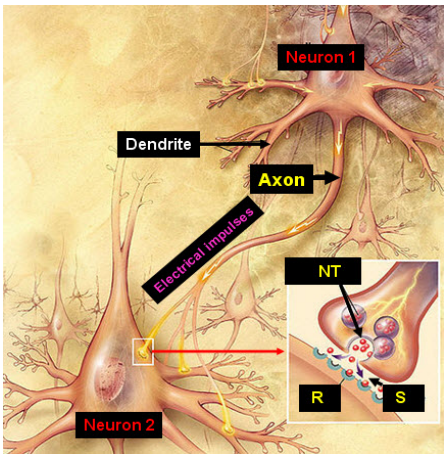
The scientific study of the nervous systems has undergone a significant increase in the second half of the twentieth century, primarily due to revolutions in molecular biology, electrophysiology and computational neuroscience. However, while it is now possible to understand, in much greater detail, the complex processes occurring within a single neuron, how exactly networks of neurons produce intellectual behavior, cognition, emotion, and physiological responses, is still poorly understood. As Kandel and colleagues (2000) noted:

> "The task of neural science is to explain behavior in terms of the activities of the brain. How does the brain marshal its millions of individual nerve cells to produce behavior, and how are these cells influenced by the environment...? The last frontier of the biological sciences--their ultimate challenge--is to understand the biological basis of consciousness and the mental processes by which we perceive, act, learn, and remember" (p. 5).

The emergence of powerful new techniques such as neuroimaging, combined with sophisticated experimental techniques from cognitive psychology, allows neuroscientists and psychologists to address abstract questions such as how human cognition is mapped to specific neural circuitries, and how dysfunctions may affect cognition and behavior. Thus, at the cognitive level, neuroscience addresses the questions of how cognitive functions are produced by the neural circuitry.

As illustrated below, brain function is directly related to neural signaling and transmission of electrical activity via the chemical and electrical synapses.

Figure 23. Picture of Neurotransmission of Electrical Activity



Electrical impulses travel from Neuron 1 through the axon, which forms a connection with Neuron 2, via a synapse (S). Electrical impulse facilitates the release of neurotransmitters (NT) at the end of the axon, which bind to specific receptors (R) on Neuron 2, and transmit the signal (http:/wikipedia.org/wiki/neuron).

In addition, neuronal activity depends on the continuity of the supply of oxygen and glucose[30], which are provided by cerebral blood flow (CBF) (Miller & Bell, 1987). This dependence makes the brain the most sensitive and vulnerable organ to CBF variations. In fact, a lack of blood supply for only seconds leads to metabolic impairment while a lack of blood supply for more than five minutes can lead to irreversible neuronal damage. Changes in electrical activity of neurons, which can be in the form of excitation or inhibition, have been indirectly attributed to the alterations in regional cerebral blood flow (rCBF) or regional cerebral metabolic rate for glucose (rCMRglc), are referred to as activations (Goldberg, 2001).

Neuroimaging includes the use of various techniques to either directly or indirectly image the structure, function/pharmacology of the brain and can be classified into the following two broad categories: structural (anatomic neuroimaging) and

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[30] Glucose is the only sugar used by the brain as a fuel to produce energy.

functional neuroimaging. Structural neuroimaging deals with the structure and abnormal pathology of the brain and can be used for the diagnosis of gross (large scale) intracranial diseases, such as stroke, and tumor, and injury. The most common structural imaging techniques are: CT and MRI. CT scanning generates grayscale images that represent the degree to which different types of brain tissue absorb and deflect X-ray beams. In MRI, grayscale images are constructed from the electromagnetic signals that are emitted by the proton nuclei of hydrogen atoms, which are found predominantly in tissue water (Brenner & Hall, 2007).

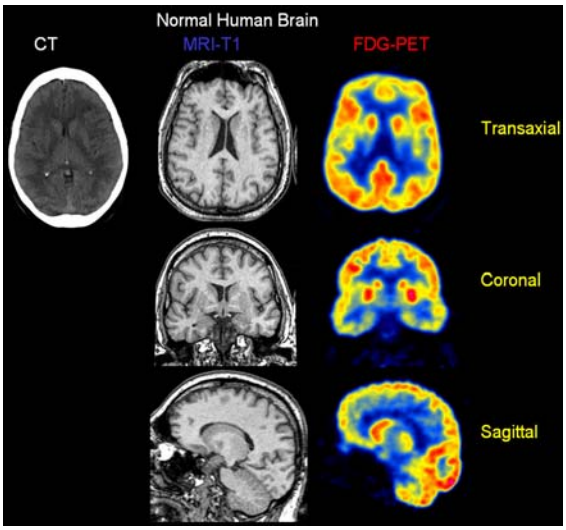
Functional neuroimaging techniques are primarily based on rCBF, rCMRglc, and neuroreceptor signaling and/or neuroreceptor status. These techniques can be used to diagnose metabolic diseases (e.g., dementias), and to study the neurobiology, and cognitive psychology associated with various psychiatric disorders, such as major depression, schizophrenia, and drug abuse (Herholz, Herscovitch, & Heiss, 2004). The most common neuroimaging techniques used to measure neuronal function are functional MRI (fMRI) and two nuclear medicine techniques based on the administration of a radiotracer or a radiopharmaceutical: PET and SPECT. While all three techniques can measure rCBF, only PET is ideally suited to quantitatively assess neuronal function based on rCBF, rCMRglc, or neuroreceptor interaction (Herholz, Herscovitch, & Heiss, 2004).

The specifics of neuroimaging

The term "neuroimage" encompasses computer-generated representations of brain structures or functions. To be more precise, all imaging methods generate a number of slices, or sections of the brain based on tomography. These representations are then used to identify the specific cortical and subcortical structures in the brain that may be

associated with a disorder, disease, or dysfunction. Three directional planes exist in the brain: rostral/caudal, dorsal/ventral, and medial/lateral (Bergman & Afifi, 2005). When sectioning or cutting the brain the planes that are visible depends on the type of section obtained. A transverse (axial) plane is parallel to the ground and separates the dorsal or front from the ventral or back planes. A sagittal (median) plane is perpendicular to the ground and separates left from right or side to side. A coronal (frontal) plane is also perpendicular to the ground and separates the rostral from the caudal and the ventral from the dorsal planes (Bergman & Afifi, 2005). With CT only transverse or axial slices can be obtained, while all other imaging modalities can provide tomographic slices in all three different planes (see Fig. 24).

Figure 24. Neuroimages Displayed in Three Different Planes



Typical neuroimages displayed in three different planes: transaxial, coronal, and sagittal (http://www.med.harvard.edu/).

Whether structural or functional, brain imaging can involve a variety of methods, each of which is designed to detect and measure specific signals of some property related to the brain with a detection device that is usually outside of the brain. These signals vary widely in parameters, including specificity, sensitivity, temporal and spatial

resolution,[31] and in their fidelity towards reflecting the physiological process being studied (Reeves, Mills, Billick, & Brodie, J.D. (2003). More specifically, the spatial resolution can vary from 0.1-10 mm, while the temporal resolution can vary from a few seconds to several minutes (Levin, 2005) (see Fig. 25).

Table 1. Spatial and Temporal Resolution of various Imaging Modalities

Imaging Modality	Form of energy used	Spatial Resolution (mm)	Temporal Resolution Time (s)/frame
CT	X- rays	0.5 – 1.0	1 – 300
MRI fMRI	Radio frequency waves Radio frequency waves	0.2 – 1.0 2.0 – 4.0	60 – 2000 1 – 5
PET	Annihilation photons	4.0 – 8.0	1 – 300
SPECT	γ - photons	5.0 – 10.0	60 – 2000

Regardless of the source, signals result in distinctive patterns that allow for comparisons among individuals. These signals can, as in the case of PET, SPECT, MRI, and fMRI, be fed into a computer that stores the data and uses the information to reconstruct an image of the brain. In order to enhance visual impact, these image produced are generally color-coded along a spectrum from blue and green to red, and yellow to reflect the varying degrees of activity in specific regions of the brain (Brown & Eyler, 2006). A closer look at the currently available imaging techniques used to assess structure and function is presented.

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[31] Temporal resolution refers to how fast images can be produced, while spatial resolution refers to the size of an object that can be imaged clearly.

1. Structural imaging techniques

a. Computerized Axial Tomography (CT)

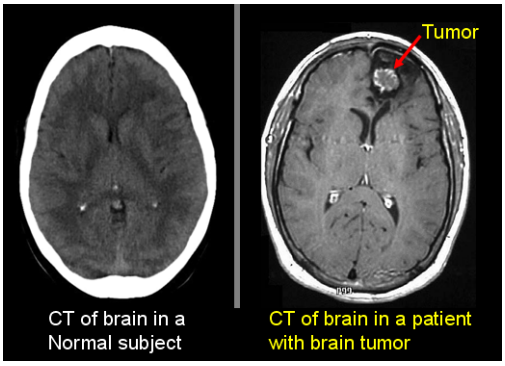
Computerized Axial Tomography is an x-ray procedure that is generally used to define normal and abnormal structures in the brain. As shown in the illustration below, a CT scanner is a large donut-shaped x-ray machine that takes x-ray images at many different angles around the body. These images are processed by a computer to produce cross-sectional pictures (slices) of the brain. When all the slices are added together, a three-dimensional picture of the body or brain can be obtained. This recorded image is called a tomogram (Brenner & Hall, 2007).

Figure 25. Picture of CT Scanner

There are many different types of CT machines used to diagnose neurological conditions and abnormalities; however, most medical facilities use a multislice CT scanner (MSCT). Providing up to 64 image slices in one test, MSCT allows several cross-sections to be taken simultaneously, which can significantly shorten the examination time and reveal internal structures in greater detail than earlier CT scans could. A faster form of CT scan, called a spiral, helical, or volumetric CT, provides continuous pictures, rather than slices, and allows for the entire scan to be taken in one minute, thus eliminating gaps

in the images collected (Bushberg, Seibert, & Leidholdt, 2002). Spiral CT has several advantages over earlier machines. More specifically, because the machine rotates in a continual spiral path it can be used to create three-dimensional images of body areas. Further, because the image acquisition takes place over a shorter period of time, the potential for distortions caused by movement as a patient breathes, which is inevitable during longer scans, is reduced. In addition, this technique's high level of sensitivity makes it a particularly useful tool in diagnosing small blood clots, and cancerous tumors (see Fig. 26) (Grossman & Yousem, 2003).

Figure 26. Non-contrast CT Scans of Normal Brain and Brain Tumor



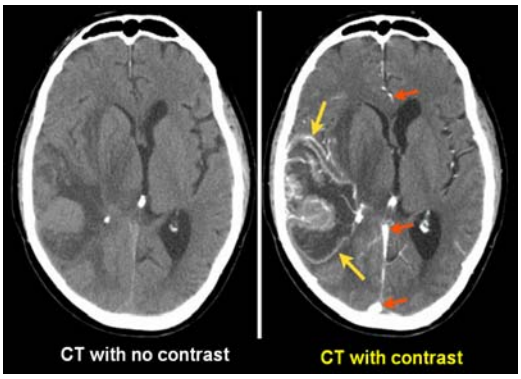
Non-contrast CT scans of subject with a normal brain and a patient with a brain tumor in the left frontal lobe (red arrow) (http://path.upmc.edu/divisions/neuropath/bpath/cases/case19.html).

CT scans can provide more detailed information about brain tissue and brain structures than standard x-rays of the head, thus providing more information related to injuries, and/or diseases of the brain. However, CT can not distinguish gray matter from white matter and can not clearly identify sub-cortical structures, such as the basal ganglia or hippocampus. Nevertheless, CT is useful for the assessment of brain tumors and other lesions, injuries, intracranial bleeding, structural anomalies, such as hydrocephalus, and infections. CT scans can also be used to detect clots in the brain that may be responsible

for strokes and can provide guidance for brain surgery or biopsies of brain tissue (Grossman & Yousem 2003).

In order to improve image contrast, CT contrast agents or dyes are sometimes used to highlight specific areas so that the organs, blood vessels, or tissues are more visible. The most common type of contrast materials used for brain CT contains iodine and is usually injected by means of an intravenous line. Increasing the visibility of all surfaces of the organ or tissue being studied helps the clinician to determine the presence and extent of a disease, or injury (Grossman & Yousem, 2003). As shown in Fig. 27 the blood vessels and tissues containing the dye appear as pure white on the CT scan similar to bone.

Figure 27. Contrast CT Image



Contrast CT scan shows normal blood vessels (red arrows) and tumor enhancement from breakdown of the blood-brain barrier (yellow arrows) (http:/www. knol.google.com/.../brain-ct-mri/).

A newer technique, CT perfusion imaging, can detect the presence and location of blood vessel obstructions. This technique involves taking CT scans while a contrast medium is administered into a patient's vein. More specifically, contrast agents help to identify blood volume and flow in different parts of the brain, as well as the average time it takes blood to travel through the blood vessels. Perfusion CT allows rapid qualitative and quantitative evaluation of cerebral perfusion by generating maps of CBF, cerebral

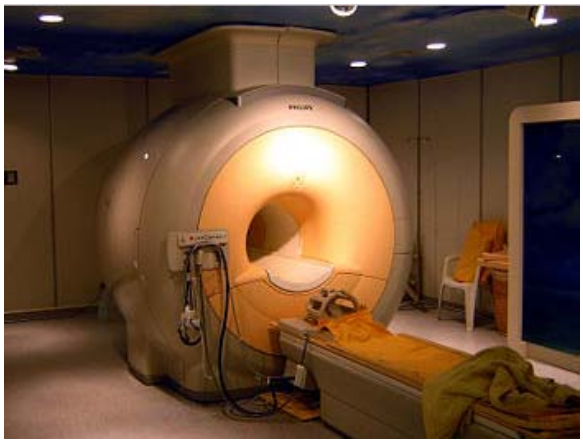
blood volume, and mean transit time (Hoeffner, 2004). Perfusion CT can be performed quickly with any standard spiral a multi-detector row CT scanner. This is accomplished by scanning the patient several times every few seconds before, during and after the intravenous delivery of an iodine-containing contrast agent that absorbs the x-rays. Perfusion CT can be used to assess not only patients with acute stroke, but also a wide range of patients with other cerebrovascular diseases, and may also be helpful in the diagnosis, and assessment of treatment response in patients with a variety of tumors. (Hoeffner, 2004)

b. Magnetic Resonance Imaging (MRI)

Magnetic Resonance Imaging is primarily a medical imaging technique most commonly used to visualize body structure. Unlike CT, MRI provides detailed images of the body with much greater contrast between the different soft tissues, making it especially useful in brain imaging. MRI relies upon signals derived from water molecules, which comprise between 70% and 80% of the average human brain. Each water molecule has two protons, which by virtue of their positive charge act as small magnets on a subatomic scale (Bushberg, 2002). Positioned within the large magnetic field of an MRI scanner, typically 30 to 60 thousand times stronger than the magnetic field of the earth, these microscopic magnets collectively produce a tiny net magnetization that can be measured outside of the body and used to generate very high-resolution images that reveal information about water molecules in the brain and their local environment (Pooley, 2005).

As illustrated below, an MRI scanner consists of a cylinder surrounded by a magnet, receiver, and computer. During the procedure, the patient is placed on a moveable bed, which is inserted into the cylinder.

Figure 28. Picture of MRI Scanner

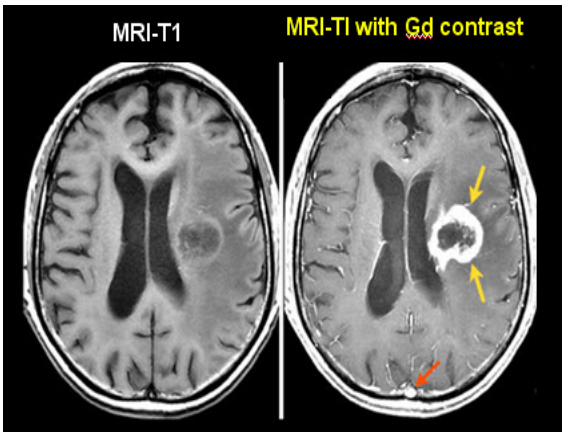


The MRI scanner creates a strong magnetic field, exposing hydrogen atoms in water molecules in the body to radio waves, causing them to move into different positions. The way these hydrogen atoms move and then move back into their original position once the scanner is turned off provides information about tissue density in the area of the body that has been scanned. A computer processes information about how the molecules move and creates a detailed image of internal body structures. Tissue that contains less water, such as bone, appears darker, while tissue that contains more water appears lighter. The image and resolution produced by MRI is very detailed and the test can be used to detect small structural changes in the body. Further, while CT provides good spatial resolution, MRI provides comparable resolution with far better contrast resolution (the ability to distinguish the differences between two arbitrarily similar but not identical tissues). In addition, with MRI different imaging protocols can be designed

to provide different image contrast in order to identify a specific brain area of interest, or a specific structural lesion (Pooley, 2005).

In some cases, a contrast agent is injected intravenously prior to MRI scan to increase the image contrast and accuracy of the images. The illustration below clearly illustrated the difference between a routine MRI scan and an MRI scan obtained following administration of a contrast agent.

Figure 29. MRI Images With and Without Contrast



While the tumor can be identified in the non-contrast MRI (left), the scan with contrast shows the tumor boundary (yellow arrows) as well as the major blood vessel (red arrow) (http://knol.google.com/.../brain-ct-mri/).

Using MRI, scientists can image both surface and deep brain structures with a high degree of anatomical detail, and detect minute changes in these structures that occur over time. For this reason, MRI is particularly useful in evaluating tumors, tissue damage, and blood flow in the brain. In fact, MRI is considered the most sophisticated type of imaging procedure to show brain structure. Further, while MRI cannot confirm a diagnosis of AD, it can be used to detect some changes in the brain structure that are common in people with dementia. For example, MRI can be used to check for shrinkage and atrophy in key memory centers of the brain, and can detect multi-infarcts, or small

strokes that are common in people with vascular dementia, and sometimes occur in people with AD (Grossman & Yousem, 2003).

c. Relative advantages and disadvantages of CT and MRI

As with all currently available tests, measures, and techniques, there are a number of advantages and disadvantages of using CT and MRI. More specifically, it is widely believed that CT is more reliable than MRI in the detection of acute bleeding, but recent studies have shown that MRI is at least as sensitive as CT (Kidwell & Wintermark, 2008). Nevertheless, CT is generally preferred since it can more rapidly identify patients that require emergency treatment. CT is also preferred to MRI in cases of acute head injury when it is necessary to rapidly triage patients who require emergency surgery (Provenzale, 2007). However, in the weeks to months following head trauma, MRI is a more sensitive tool to evaluate for the presence of chronic hemorrhage and subacute shear injury. Further, by virtue of its speed and accuracy, CT continues to be the main method of diagnosis acute stroke (Lev & Nichols, 2000). In addition, since most first-time seizures are caused by metabolic abnormalities or medications, CT is useful in these cases to exclude rare secondary causes for acute seizures, such as brain tumor, brain infection, or stroke. When seizures are longstanding, however, MRI is more sensitive in the assessment for structural sources of the seizures (Bernal & Altman, 2003). In addition, while modern CT scanners produce fine-grained resolution, permitting some differentiation between white and gray matter;[32] even with high-resolution scanners, a CT

[32] Brain tissue is made up of cell bodies ("gray matter") and the filaments that extend from the cell bodies ("white matter"). The density of cells (volume of gray matter) in a particular region of the brain appears to correlate positively with various abilities and skills. The density of cells is

image is almost always less precise than it appears. CT is also inferior to MRI in showing the precise details of brain tissue damage that is common in people with dementia, due to the inferior quality of the images and obstructed views of tissue that result from overlying bone. CT is also not the preferred tool for diagnosing dementia since many people will have normal CT scans, at least in the early stages of this disease (Bernal & Altman, 2003).

There are also a number of basic advantages to using MRI instead of CT: (1) because MRI does not use ionizing radiation it is preferred over CT in patients requiring multiple imaging examinations; (2) MRI has a much greater range of available soft tissue contrast, depicts anatomy in greater detail, and is more sensitive and specific to for assessing abnormalities within the brain itself; (3) MRI scanning can be performed in any imaging plane without having to physically move the patient; and (4) MRI allows the evaluation of structures that may be obscured by artifacts from bone in CT images. Research studies have also shown MRI to be more sensitive for identifying early strokes (Chalela, Kidwell, Nentwich, Luby, et al., 2007). MRI is also the modality of choice in evaluating known primary brain tumors and metastases, since it allows more accurate delineation of tumor margins and provides more information with which to differentiate between various tumor types (Cha, 2006). In addition, MRI is superior in detecting diseased or damaged tissue in people with dementia. The disadvantages of MRI include the amount of time it takes to do the scan and sensitivity to movement. Finally, neither CT nor MRI scans can provide reliable quantitative data. As a result, interpretation of CT

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determined by both genes and environmental factors, such as experience, while the speed with which we can process information is governed by the white matter (Bergman & Afifi, 2005).

or MRI scans is much less objective than the interpretation of scans obtained by other neuroimaging techniques. In addition, the main limitation of CT and MRI techniques is that they cannot detect functional abnormalities in the absence of focal structural lesions (Grossman & Yousem, 2003).

2. Functional imaging techniques

In contrast to structural imaging techniques, functional brain imaging, in particular, plays an important role in our understanding of the relationship between various behaviors and brain processes. More specifically, this technique offers the possibility of coupling an image with emotional and neurocognitive functions and, in the case of PET and fMRI, to further integrate brain function with features of molecular and neural circuitry information. Basic functional imaging can be achieved through a variety of methods, such as fMRI, PET and SPECT, each of which measures a different physical property of the brain. To date, however, among these different imaging technologies, fMRI and PET have been the most technologically advanced and have had the broadest application in the courtroom (Weiller, May, Sach, Buhmann, et al., 2006).

a. Functional Magnetic Resonance Imaging (fMRI)

Functional Magnetic Resonance Imaging is a form of specialized MRI that works by detecting the changes in blood oxygen concentration, and blood flow that occur in response to neural activity. fMRI operates under the principle that changes in the brain's hemodynamics,[33] which relate to mental operations, and can be detected and mapped using basic MRI instrumentation. More specifically, this technique relies on the

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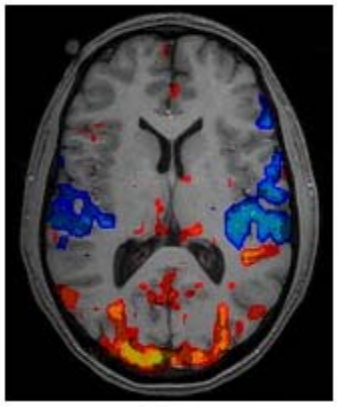
[33] Hemodynamics, meaning literally "blood movement", is the study of blood flow or blood circulation.

magnetic properties of blood to enable scientists to see images of blood flow in the brain as it is occurring. When a brain area is more active it consumes more oxygen and, to meet this increased demand, blood flow increases to the active area. Since blood oxygenation varies according to the levels of neural activity these differences can be used to detect brain activity. This form of MRI is known as blood oxygenation level dependent (BOLD) imaging (Detre, 2006). This technique depends on the observation that the properties of hemoglobin[34] in a strong magnetic field are dependent upon its state of oxygen saturation. The underlying physiological notion is that increased neural activity in a particular brain region results in more consumption of oxygen from the blood near these neurons. Accompanying the increased oxygen consumption are increases in blood flow and blood volume of the local vasculature of the activated regions of the brain. The consequence is that the blood near a region of local neuronal activity has a higher concentration of oxygenated hemoglobin than blood in locally inactive regions (Cacioppo, Berntson, Lorig, Norris, et al., 2003).

Figure 30. fMRI Scan of Subject Playing a Violent Video Game

[34] Hemoglobin is the protein molecule in red blood cells that carries oxygen from the lungs to the body's tissues and returns carbon dioxide from the tissues to the lungs. Hemodynamics, meaning literally "blood movement", is the study of blood flow or blood circulation.



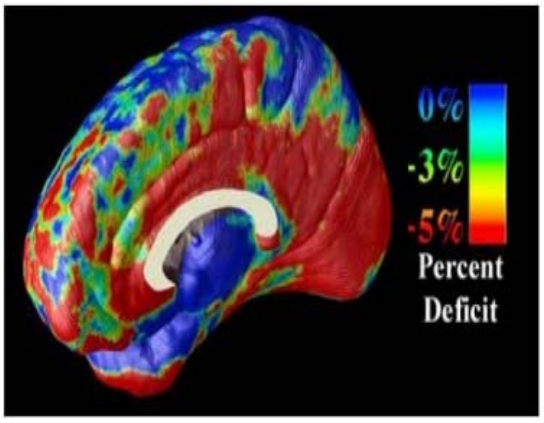
fMRI scan of subject playing a violent video game. Scan shows less activation in the prefrontal area (top) and more activation in the amygdala (bottom) (http://psychcentral.com/news/2006/11/29//).

Almost all fMRI research studies use BOLD to determine where in the brain the activity occurs as the result of various experiences. However, because the signals are relative, and not individually quantitative, some researchers have questioned the rigor of BOLD. One of the main issues in investigating brain function using fMRI rests on the fact that specific brain functions are localized at various sites. This functional specialization can be identified with fMRI and mapped at high spatial resolution. fMRI is similar to PET in that it accurately localizes signal sources thereby more closely identifying regions of the brain in terms of anatomy and function. In fact, its most important application is to map the hemodynamic responses of defined cognitive and affective stimuli to determine the anatomical locations, which subserve specific brain functions in the cognitive, behavioral, and affective domains. The underlying assumption is that cognitive functions are basically located in focal brain regions; however, evidence from brain studies suggests that most complicated behavioral and psychological processes are not located in a single brain center (Sarter, Berntson, & Cacioppo, 1996).

Although, neuroimaging techniques have greatly contributed to our understanding of a wide range of aspects related to neurological diseases, such as the classification and localization of diseases, aside from pre-surgical mapping, the clinical application of

fMRI, to date, is limited (Weiller, May, Sach, Buhmann, et al., 2006); however, research has suggested that fMRI may be useful in the assessment of various neurological, psychiatric, and psychological disorders. For example, one study measuring brain activation in psychotic patients while they experienced auditory and verbal hallucinations found predominant engagement of the right inferior frontal region (Sommer, Diederen, Blom, Willems, et al., 2008). It has also been suggested that reduced functional response in the frontal and inferior parietal regions, as assessed with fMRI, may lead to serious violence in schizophrenia via impaired executive functioning (Kumari, Aasen, Taylor, et al., 2006). Further, bipolar depression was found to be associated with attenuated bilateral orbitofrontal activation, attenuated right DLPFC activation, and heightened left orbitofrontal activation (Altshuler, Bookheimer, Townsend, Proenza, et al., 2008). Functional magnetic resonance imaging may also be useful in evaluating the effects of various types of drug abuse, as suggested by the scan below which depicts the effect of methamphetamine on brain tissue volume.

Figure 31. fMRI Scan Showing the Effects of Methamphetamine



Red and green colors show decreased blood flow in different brain areas (http://neuroanthropology.net/2008/05/).

b. Positron Emission Tomography (PET)

Positron Emission Tomography is a nuclear medicine[35] imaging technique, which produces three-dimensional images of functional processes in the body. The advantage of PET in imaging functional activity of the brain is based on the fact that a number of chemical molecules in the body can be easily radiolabeled with positron emitting radionuclides (Herholz & Heiss, 2004). In the last three decades, hundreds of PET radiotracers have been developed to study various diseases, but the most important radiotracers used in brain imaging are listed below.

Table 2. List of Commonly Used Radiotracers

Radio-nuclide	Half-life (min)	Radiotracer	Use
^{11}C	20.4	[^{11}C]Raclopride	Brain dopamine D2 receptors
^{15}O	2.0	[^{15}O]Water	Brain perfusion or blood flow
^{18}F	110	2-[^{18}F]Fluo-2-deoxy-D-glucose	Brain glucose metabolism

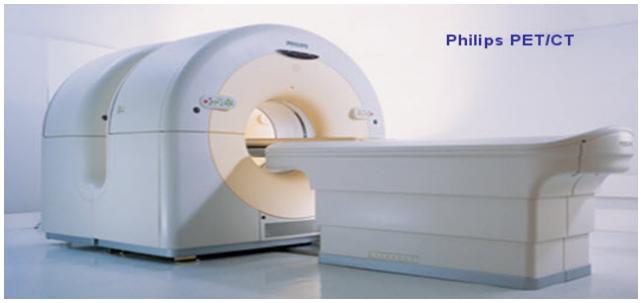
PET measures emissions from radiotracers that have been injected into the bloodstream and uses the data to produce tomographic images of the distribution of the chemicals throughout the brain and body. Using different radiotracers, such as 2-[^{18}F]FDG (FDG), PET can image blood flow, oxygen, and glucose metabolism in the tissues of the working brain. Since PET measures the differences in metabolism of oxygen and glucose used by various tissues in the body, it is also regarded as a "metabolic imaging" technique. Further, unlike other brain imaging techniques, PET is the only true quantitative imaging technique providing metabolic rates, and neuroreceptor

[35] Nuclear medicine or radionuclide imaging procedures are noninvasive tests that help physicians diagnose medical conditions. These imaging scans use radioactive agents called radiopharmaceuticals or radiotracers.

concentrations in absolute numbers, rather than relative amounts (Herholz & Heiss, 2004).

A typical PET scanner consists of many rings of detectors surrounding a subject (see below). Following injection of a positron emitting radiotracer into the patient, the PET scanner obtains images of the radiotracer distribution in the body. These images are then corrected for the differences in the absorption of high energy photons in the body and then mathematically reconstructed or processed to obtain tomographic images of the brain (Levin, 2005).

Figure 32. Picture of a PET Scanner



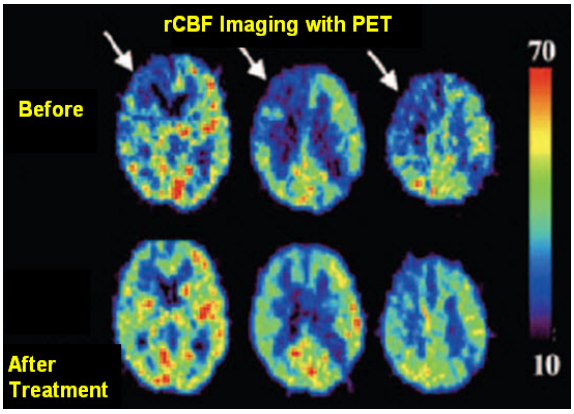
As previously noted, structural imaging techniques show the brain's anatomy or structure. PET, in contrast, produces brightly colored "maps" of the brain's activity. As a functional brain imaging technique, PET allows physicians to detect the presence of disease long before any structural damage is done to the brain. Further, in brain research, PET is frequently used to identify the brain sites where drugs and naturally occurring neurotransmitters act and to show how quickly drugs reach and activate a neural receptor system such as the dopamine receptor systems. In addition, using PET in patients with schizophrenia and depression, physicians are able to determine the appropriate treatment dose for a specific patient in order to minimize the side effects often associated with

antipsychotic drugs. PET is also frequently used to assess brain changes following chronic drug abuse, during withdrawal from drugs, and while the subject is experiencing drug craving (Herholz & Heiss, 2004).

PET - blood flow

The most accurate and comprehensively validated approach to the measurement of rCBF is based on PET using ^{15}O labeled radiotracers, such as [^{15}O]Water. Since rCBF depends on neuronal function, the measurement of rCBF during various physiological and cognitive tasks has become a major tool for the study of functional brain organization (Salinas & Sejnowski, 2001). For example, the PET scan shown below clearly identifies the decreased rCBF in the frontal lobes (white arrows) of a patient with Moyamoya disease, a rare disorder in which major blood vessels are blocked leading to ischemia and strokes. Following treatment, the PET scan shows significant improvement in rCBF in the frontal lobes (Wintermark, Sesay, Barbier, Borbe´ly, et al., 2005).

Figure 33. PET Scan of Patient with Moyamoya Disease



PET blood flow studies have been extensively used to study a number of neuropsychiatric diseases, such as chronic cerebrovascular disease (stroke), dementias, epilepsy, depression, and schizophrenia (Herholz, Herscovitich, & Heiss, 2004). These

studies are often performed at rest, or while a subject is performing a basic task, such as a neuropsychological attention task. However, PET blood flow can also be employed in activation studies where the brain areas of interest are activated by means of a cognitive task.

PET- glucose metabolism

PET can detect abnormalities in cellular activity generally before there is any anatomical change and, in many cases, can identify diseases earlier and more specifically than, for example, MRI. In the case of PET, the most frequently studied biological process has been glucose metabolism. This is primarily because energy metabolism is closely linked to brain function, although in a very complex way (Brodie, 1996). The assessment of glucose metabolism begins with an injection of FDG. Since FDG is similar to glucose it is used as a tracer to study glucose metabolism, unlike glucose, however, FDG once transported into the cells is not metabolized completely and so gets trapped in the cells. The amount of FDG accumulated in the cells reflects the metabolic rate of the cells (Herholz & Heiss, 2004).

The power of FDG-PET lies in its ability to measure the rate of glucose metabolism in absolute units and, to date, it is the only radiotracer approved by the Food and Drug Administration for diagnosing brain tumors, epilepsy, AD, and other forms of dementia (Silverman, 2008). Indeed, a recent study, comparing FDG-PET with histopathology, has reported relatively high predictive accuracy (84% sensitivity, 74% specificity, 81% PPP, and 78% NPP) for the assessment of AD (Jagust, 2007). FDG-PET also plays a significant role in evaluating patients with epilepsy and its predictive accuracy of doing so is relatively high. More specifically, in patients with temporal lobe

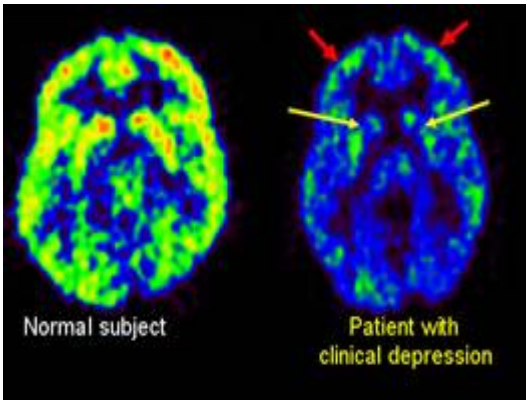
epilepsy who had surgically and pathologically confirmed lesions the reported specificity and sensitivity of FDG-PET for localizing the seizure was found to be 89% and 91%, respectively (Herholz, et al., 2004; Kim, et al., 2003,). Further, in patients with structural lesions sensitivity was found to be 73%, while the sensitivity was only 36% in patients without any structural lesions (Kim, et al., 2003). FDG-PET has also been used in the assessment of brain tumors although the current clinical "gold standard" for the diagnosis of brain tumors is MRI, which provides superior structural detail but poor specificity. However, since FDG-PET may show increased or decreased glucose metabolism depending on tumor pathology, it has the advantage of being able to identify different tumor types and tumor grade with much better specificity, than other imaging modalities (Chen, 2007).

Although FDG-PET is not yet an integral part of clinical practice in psychiatry, the potential value of FDG-PET in neuropsychiatric diseases has been extensively evaluated. In fact, the core of neuroimaging research in psychiatry consists of functional imaging studies of the prefrontal cortex and the limbic system with specific focus on schizophrenia, major depression, and drug abuse (Zipursky, Meyer, & Verhoeff, 2007). For example, it has been shown that patients with major depression have hypometabolism in the prefrontal cortex, temporal lobes, and cingulated cortex, and hypermetabolism in the ventrolateral prefrontal cortex (Parsey & Mann, 2003; Herholz, et al, 2004).

FDG-PET studies are generally performed at rest, or while a subject is performing a basic task, such as a neuropsychological attention task. FDG-PET is also frequently used in activation studies where the brain areas of interest are activated by means of a pharmacological agent or a cognitive task. For example, in assessing frontal lobe

functioning in the context of violent and aggressive behavior a neuropsychological test known to activate the frontal lobes may be employed.

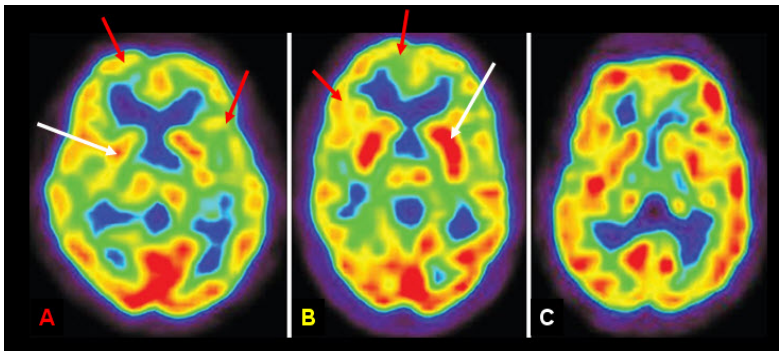
Figure 34. FDG-PET Scan showing Clinical Depression



Comparison of normal subject (left) and patient with depression (right). The color blue represents less activity while red represents more glucose metabolism (more activity) (www.musc.edu/fnrd/petdep.htm).

Further, studies of patients with schizophrenia have also found hypometabolism in the prefrontal cortex at rest or during a simple attention task (Herholz, et al 2004). This classical hypofrontality finding has been confirmed by frontal lobe tests, such as the WCST (Riehemann, Volz, Stutzer, Smesny, et al., 2001). In addition, studies have reported increased metabolism in unmedicated patients with schizophrenia (Soyka, Koch, Möller, Rüther, & Tatsch, 2005). A recent study has also suggested that chronicity and severity of illness do not influence cerebral glucose metabolism. Finally, hypofrontality has been associated with negative symptoms schizophrenia (Potkin, et al 2002).

Figure 35. FDG-PET Scans of Subjects With Schizophrenia

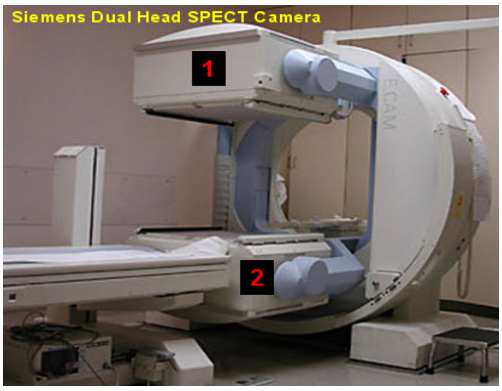


(FDG-PET scans in resting state demonstrate hypometabolism in cortical (red arrows) and basal ganglia (white arrows) in unmedicated schizophrenics (A), hypermetabolism in basal ganglia (white arrow) of a patient on typical antipsychotic drug (B), and normal or hypermetabolism in cortical areas of a patient on atypical antipsychotic drug (C) (Seethalakshmi, Parkar, Nair, Batra, 2007).

c. Single Photon Emission Computed Tomography (SPECT)

Single Photon Emission Computed Tomography is based on the detection of gamma rays emitted by radiotracers and a special camera, known as "gamma camera," is used for the detection of these rays. The SPECT camera may consist of a single-head, dual-head, or triple headed gamma camera, but the most common SPECT camera, at present, is a dual-headed gamma camera (see below).

Figure 36. Pictureof SPECT Scanner



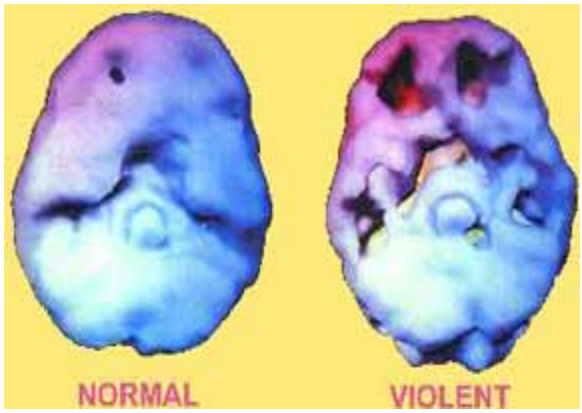
The most common brain SPECT scan allows physicians to visualize brain function such as brain perfusion or rCBF. In order to generate a typical brain SPECT scan, a radiotracer is first injected intravenously into a subject (O'Connor & Kemp, 2006). In the last three decades, a number of commercially available and experimental radiotracers have been used for SPECT imaging studies to assess rCBF, however, the most commonly used radiotracers are ^{99m}Tc-HMPAO and ^{99m}Tc-ECD. Following the intravenous administration, these radiotracers are rapidly transported into the brain tissue, usually within 2-3 minutes. As the blood travels through the brain, or perfuses the brain,

the amount of radiotracer taken up by different areas in the brain reflects the rCBF. More specifically, as the radiotracer travels, the gamma cameras heads, which are rotating around the patient, take pictures to monitor its uptake in different areas of brain. The cameras detect the gamma rays emitted by the radiotracer and record the distribution, position, and rate of flow of the tracer through the brain. A computer collects the information and translates them into two-dimensional cross-sections. These cross-sections can be added back together to form a three-dimensional image of brain. In general, brain areas with structural and functional abnormalities show decreased radiotracer levels compared to healthy brain tissue (O'Connor & Kemp, 2006).

As noted above, brain SPECT provides three-dimensional information on the perfusion status of brain tissue. While this information is often complementary to the anatomic details provided by structural neuroimaging techniques, brain perfusion SPECT has clinical value by itself since functional impairment in cerebral diseases often precedes structural changes. Consequently, SPECT can be used to define a patient's pathologic status when neurological or psychiatric symptoms cannot be explained by structural neuroimaging findings (Catafeu, 2001). More specifically, SPECT is more sensitive to brain injury than either MRI or CT because it can detect reduced blood flow to injured sites. Similarly, in the early phase of frontal lobe dementia, CT or MRI may show normal findings or only mild frontal cerebral atrophy, disproportionate to the degree of hypoperfusion, while SPECT usually shows symmetric hypoperfusion of the frontal lobes (Miller, Cummings, Villanueva-Meyer, J., et al., 1991). Further, since blood flow is tightly coupled to local brain metabolism and energy use, the ^{99m}Tc-HMPAO and ^{99m}Tc-ECD tracers are often used to assess brain metabolism regionally in an attempt to

diagnose and differentiate the different causal pathologies of various types of dementia. In fact, the accuracy of SPECT in diagnosing DAT was found to be as high as 88% (Bonte, Harris, Hynan, Bigio, et al., 2006). SPECT is also useful in localizing seizure focus in epilepsy, especially in temporal lobe epilepsy, and has been found to be more sensitive than CT or MRI for revealing lesions caused by head injury (Camargo, 2001; Devous, Thisted, Morgan, Leroy, & Rowe, 1998; Gray, Ichise, Chung, Kirsh, et al., 1992). In addition, SPECT is valuable in assessing psychiatric, mood, anxiety, substance abuse, psychotic, and behavioral disorders. More specifically, as illustrated below, SPECT has been used to assess blood flow in violent individuals.

Figure 37. SPECT Scan of Normal and Violent Subject

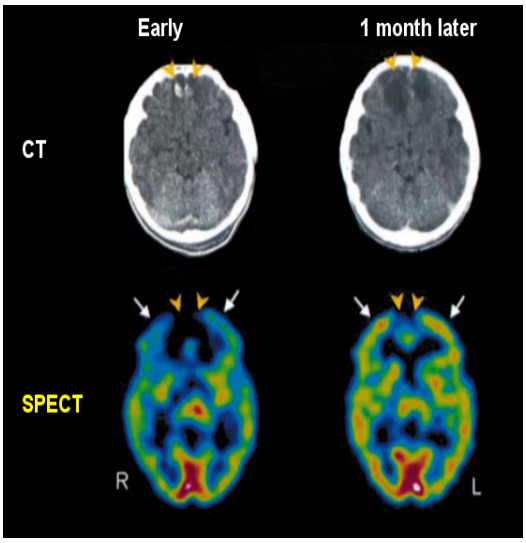


These views of the human brain illustrate the extent of blood flow. Note that brain of the violent individual has greatly reduced levels of activity compared to the normal subject. Areas of chronic dysfunction appear as "holes" in the brain. Note especially in the violent case [top of image], these regions occur in the front of the brain, the region of the brain that normally provides a filter against impulsive, aggressive, and violent behavior. The lesions thus may be expected to cause significant loss of impulse control, decision making, and moral reasoning (http://www.istpp.org/bri/reversing_violence.html).

Finally, SPECT is frequently used to assess (1) changes in CBF in response to traumatic injury, and ongoing trauma, and (2) the effectiveness of medication and therapy on brain function by monitoring changes in CBF in response to treatments rendered. The

potential value of brain SPECT in the detection of focal brain abnormalities is clearly illustrated by the image below.

Figure 38. CT Scan of Subject with TBI



CT of patient with TBI, at time of admission, shows focal defects due to hemorrhage in frontal lobes (orange arrows), while SPECT shows markedly decreased rCBF in frontal lobes (orange and white arrows). One month later, following clinical recovery, CT shows resolution of the hematomas while SPECT still shows focal abnormalities (orange arrows) (Catafau, 2001).

d. Relative advantages and disadvantages of fMRI, PET, and SPECT

As is the case with structural imaging techniques, each of the functional imaging modalities has its advantages and disadvantages. More specifically, fMRI has better temporal and spatial resolution than PET and SPECT. Further, since fMRI can produce images of brain activity as fast as every second, scientists can determine with greater precision when brain regions become active and how long they remain active. In contrast, both PET and SPECT take 30 seconds to several minutes to obtain an image. In addition, fMRI can produce an image that distinguishes structures less than a millimeter apart, whereas the latest commercial PET scanners can resolve images of structures only within 4mm of each other. In summary, fMRI provides superior image clarity along with the ability to assess blood flow and brain function in seconds (Matthews, 2006). However, FDG-PET can (1) detect disease, such as AD, prior to changes in structure, (2) reveal

metabolic changes in the brain, such as changes due to cancer, and (3) show the extent of disease of various types of brain cancer. Further, PET may detect changes in brain chemistry or functioning before symptoms appear (Herholz & Heiss, 2004). To date, PET also retains the significant advantage of being able to identify which brain receptors are being activated by neurotransmitters, drug abuse, and potential treatment compounds. Because it is a tracer method, PET has the distinct advantage of being thus far the best modality for the detection of a wide variety of biochemical processes. Another advantage of PET is that it has a high degree of quantification accuracy regarding changes pre- and post- intervention in brain regions with altered brain perfusion or metabolism (Wintermark, Sesay, Barbier, Borbe´ly, et al., 2006). In addition, because [^{15}O]water-PET is quantitative and considered to be the "gold standard" for the measurement of rCBF, assessment of brain regional activations under different cognitive tasks, provides physiologically relevant information, which can not be obtained with fMRI (Raichle, 2007).

Although SPECT is widely available, it has the lowest spatial and temporal resolution of all currently available neuroimaging techniques. In addition, SPECT is not a quantitative technique although its images can be processed to provide a semi-quantitative estimation of brain activity. However, SPECT can compete with PET in providing information about local brain damage from many processes, such as tumors and strokes (Catafau, 2001). Generally, SPECT tracers are more limited than PET tracers in the types of brain activity they can monitor and deteriorate more slowly than many PET tracers. Therefore, SPECT studies require longer test and retest periods than PET

studies do. Further, the quality of the pictures generated by SPECT is not as good as that generated by more advanced functional scans, like PET (Catafau, 2001).

Accurate anatomical localization of functional abnormalities obtained with PET or SPECT is known to be problematic. More specifically, while these techniques can visualize certain normal anatomical structures, the spatial resolution is generally inadequate for accurate anatomic localization of pathology. Combining PET or SPECT with a high-resolution anatomical imaging modality such as CT or MRI can resolve the localization issue as long as the images from the two modalities are accurately co-registered (Cherry, Louie, Jacobs, & Townsend, 2008). Prior to the introduction of hybrid imaging techniques, various software algorithms were established to allow for the fusion of anatomic and functional images. Although current software algorithms permit highly accurate co-registration of anatomic and functional datasets, motion artifacts can markedly affect image fusion and overall quality of the images. In addition, differences in patient positioning and respiratory motion make the correct alignment of anatomic and functional images even more complicated (Forster, Laumann, Nickel, Kann, et al., 2003).

e. Hybrid imaging techniques

Hybrid imaging techniques allow for the direct fusion of structural and functional information and have become an attractive strategy due to their ability of providing accurate anatomical and functional information, simultaneously (Cherry, Louie, Jacobs, & Townsend, 2008). The combination of CT and PET was fist introduced commercially in 2001, followed by CT and SPECT in 2004, and PET and MRI in 2008. While PET/CT and SPECT/CT are in routine clinical use, PET/MRI is still under clinical evaluation. PET/CT and SPECT/CT can provide physicians with a more complete picture of what is

occurring in the body, both anatomically and metabolically, at the same time. Consequently, these systems are more accurate and have higher sensitivity and diagnostic accuracy than PET, SPECT, or CT alone (Branstetter, 2005, Townsend, 2008). At this time, PET/CT hybrid systems are routinely used in the clinic while PET/MRI hybrid systems are under extensive evaluation.

f. Electroencephalography (EEG) and Quantitative EEG (QEEG)

Although not a true neuroimaging technique, EEG can be used to assess global brain function. EEG refers to the recording of the brain's spontaneous electrical activity over a short period of time, as recorded from multiple electrodes placed on the scalp. The main diagnostic application of EEG is in the case of epilepsy, as epileptic activity can create clear abnormalities on a standard EEG study. In addition, EEG can be used as a first-line method for diagnosing, strokes, tumors, and other focal brain disorders. Further, EEG is useful in differentiating organic from functional brain diseases (Dimitrios, Sahu, & Treloar, 2005).

Quantitative EEG is used to aid in identifying mental health conditions by means of statistical evaluations of the EEG. QEEG is useful as an adjunct to traditional clinical assessment, as it provides a sensitive and specific method to detect subtle variations in the activity of the brain, which might otherwise go unnoticed by the clinician. Unlike EEG, QEEG does not assess the structure of the brain, but rather, evaluates the manner in which a particular person's brain functions (Thornton & Carmody, 2009). In other words, QEEG is not designed to diagnose tumors, epilepsy, or other structural impairments, such as brain tumors (Hughes & John, 1999). Quantitative EEG, however, can help to identify mild dementia and mild cognitive impairment and can increase

diagnostic accuracy when used with other imaging techniques. It has also been suggested that QEEG is useful in assessing FLD in patients with OCD (Tot, Özge, Çömelekoglu, Yazici, et al., 2002). Although readily available and easily administered EEG and QEEG are not suitable for the assessment of subcortical structures, such as the ACC, and due to poor spatial resolution provide little detailed information.

Functional neuroimaging and activation

An emerging practice is to conduct functional imaging while the patient is challenged with various neuropsychological tests and neurobehavioral probes that activate the brain area of interest. More specifically, functional neuroimaging began in the 1980s with PET blood flow studies using the brain's responses to carefully controlled sensory, cognitive and motor events. The problem, however, is that one must have a "baseline state" against which to measure stimulus, or task-induced changes in brain regional activities. When functional images acquired during the baseline (resting state)[36] or control state[37] scan are subtracted from those acquired in a "task state," the vast majority of changes observed are activity increases or "activations".[38] While the term physiologic baseline or rest state is only applicable to PET studies, the terms control state and control condition may be applied equally to PET and fMRI imaging techniques.

Baseline is an important issue in cognitive neuroscience especially when functional brain imaging techniques are used to measure changes in brain activity that are

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[36] Baseline is defined as a rest state (subject is lying quietly with eyes closed and ears plugged during image acquisition) (Roland, 1997).
[37] Control state is defined as a state when the transient changes in neuronal activity are due to a control task (subject is performing a task during image acquisition).
[38] Activation is defined as a transient increase in neuronal activity due to motor, sensory or cognitive stimulation task (subjects is performing a task that is known to activate brain region of interest).

presumed to be associated with specific mental operations, such as executive functions. In fact, it has been contended that left unconstrained the baseline activity would vary unpredictably and consequently affect the specificity of a functional activation scan (Gusnard & Raichle, 2001). Further, both PET and SPECT at the resting (baseline) state are clinically useful for the detection of both structural and functional abnormalities in a number of neuropsychiatric disorders, such as dementia, and schizophrenia. The major limitation with resting state studies, however, is that the processes involved in cognitive tasks can not be linked to specific brain regions. The resting state is also of limited use due to its variability across individual subjects (Weinberger, Berman, & Zec, 1986). Variations in rCBF may, therefore, simply reflect the subjective experience of the procedure itself, rather than indicate an underlying pathology. It has also been contended that the "resting state" is a misnomer:

> "The brain does not become inactive or empty of thought in the absence of specific experimental tasks or instructions; on the contrary, patients report after scans that when at 'rest' they typically recalled past experiences, or made future plans" (Andreasen, O'Leary, Flaum, et al., 1997, p. 1732).

In addition, because resting state studies are usually associated with single measurements, they provide no clues about how a brain region may respond to the challenge posed by a cognitive task, such as the WCST (Frith, Friston, Herold, et al., 1995). Nevertheless, obtaining a baseline is crucial since it provides information regarding FLD due to major structural and functional abnormalities.

As previously discussed, many neuropsychiatric disorders are associated with functional brain abnormalities. The metabolism and blood flow in specific impaired brain areas can be activated by carefully designed "activation" tasks, such as a cognitive task.

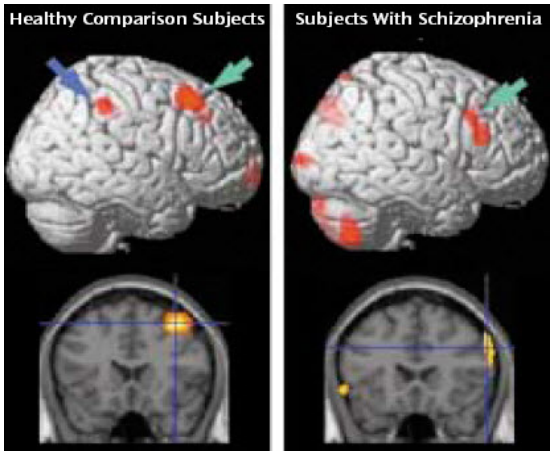
More specifically, in functional neuroimaging techniques that employ a cognitive task, cognitive "activation" refers to a specific psychological state or occurrence that is deemed to be active in specific areas in the brain relative to the baseline state. In the interpretation of neuroimaging data it is important to remember that activation in a given brain region means that this region is associated with the performance of that particular activation task. In other words, a cognitive brain activation study is a cortical stress test in that it imposes a selective physiological load on a specific brain area. This analogy is particularly important in light of research findings showing that functional abnormalities evident during the performance of cognitive tasks are not found during resting baseline rCBF (Zemishlany, Alexander, Prohovnik, et al., 1996). Consequently, many studies attempt to assess brain activity while the subject is engaged in a specific mental task. For example, in the case of FLD in the context of aggressive behavior, one would want to use a neuropsychological test known to activate the prefrontal cortex.

Despite its usefulness, a major limitation of FDG-PET is the relatively long uptake period ($\geq$20 minutes) required to record the metabolic increase related to functional activity by FDG accumulation. Therefore, this excludes the use of activation tasks that can not be maintained over that period of time. On the other hand, the robustness of the method gives it a clear advantage over other more rapid activations methods (Roland, 1997). With [^{15}O]Water PET studies of rCBF, multiple replications of conditions in the same subject can be performed sequentially in a short period of time (over a period of 30-60 min) and, as a result, PET blood flow studies are widely used for activation studies.

In recent years, fMRI has become the dominant imaging technique because it does not involve ionizing radiation and allows more rapid signal acquisition; however, there are practical considerations limiting the routine use of fMRI as a clinical tool (Matthews, et al., 2006). SPECT can also be used in activation protocols to identify FLD. However, SPECT tracers are more limited than PET tracers in the type of brain activity they can detect and because SPECT tracers are longer lasting than PET tracers, baseline and activation studies must be performed on different days. In addition, the spatial resolution of SPECT is poor (8-10mm) compared to PET which is 4-6mm (Levin, 2005).

Neuroimaging activation procedures are frequently employed for the assessment of FLD and hypofrontality has been observed in patients with neuropsychiatric disorders, not only at rest but also during activation of the frontal cortex. For example, better performance on the WCST was found to correlate with rCBF increase in the prefrontal cortex of control subjects and in the parahippocampal gyrus of schizophrenic patients (Ragland, 1998). Further, as shown below, during a working memory task, activation (green arrows) was observed in the right DLPFC (green arrow) and in the inferior parietal area (blue) of healthy subjects. In contrast, in schizophrenics, activation was observed in the right ventrolateral prefrontal cortex (green arrow) and in several other brain regions (Kim, et al., 2003).

Figure 39. PET Scan showing Activation during Memory Task



In addition, patients with depression also showed less activation in prefrontal brain regions similar to the schizophrenic patients, suggesting that impairment of executive functions is not unique to schizophrenic subjects (Hugdahl, Rund, Lund, et al., 2004). Also, Kirsch and colleagues (2006) have suggested that using PET with fMRI is a suitable method to quantify PFC dysfunction in patients suffering from schizophrenia and other neuropsychiatric disorders. However, because fMRI BOLD signals, for reasons currently not understood, do not remain constant some researchers have concluded that the fMRI baseline cannot be defined (Raichle & Snyder, 2007). Unlike PET activation studies, fMRI, as it is conventionally practiced using BOLD imaging, does not offer a similar absolute reference and is not truly quantitative. Therefore, estimated changes in parameters such as oxygen consumption must be viewed with caution until further work is done to determine their validity (Raichle & Snyder, 2007).

Detection of frontal lobe impairment in the context of violent and aggressive behavior can be difficult, especially if only traditional methods are used. Indeed, changes can often be discerned only with reference to the previous personality and behavior of an

individual and not with regard to standardized and validated behavioral norms based on population studies. A further complication is that abnormal behaviors may (and often do) fluctuate from one testing occasion to another. Based on the foregoing, brain structure will, therefore, be assessed using MRI, functional baseline will be determined with FDG-PET/CT, and frontal lobe functioning will be assessed using [^{15}O]Water-PET.

The issue of radiation exposure

Every year, millions of individuals benefit from nuclear medicine procedures used to diagnose and treat a wide variety of diseases. In fact, the use of radiation in these procedures provides clinicians with information that would (1) not be available otherwise, (2) necessitate more costly and invasive procedures, and (3) necessitate the use of less reliable tests and measures. However, since working with ionizing radiation can present significant safety risks, its use is closely regulated. More specifically, in the United States, the Nuclear Regulatory Commission (NRC) is responsible for protecting workers, the public and the environment from the effects of radiation. The NRC is an independent agency established by the Energy Reorganization Act of 1974 to regulate civilian use of nuclear materials. Indeed, the NRC has developed a system of radiation protection that reflects the world's improved understanding of the effects of radiation. In particular, the NRC ensures that users of radioactive materials keep radiation exposures within the agency's specified dose limits and as low as reasonably achievable. In addition, users must obtain a license from the NRC and be inspected to ensure that they are following the agency's regulations and safely use radioactive materials (Towson, 2005).

For some common diagnostic x-ray and nuclear medicine procedures the radiation dose can be estimated. However, since radiation doses differ for each person because of

(1) differences in imaging machines and their settings, (2) the amount of radioactive material given in a nuclear medicine procedure, and (3) the patient's metabolism, these are only typical values (see below for dose estimates). The scientific unit of measurement for radiation dose, commonly referred to as effective dose, is the millisievert (mSv). For comparison, in the United States we receive approximately 3.0 mSv (300 mrem) of exposure from natural background radiation every year.

Table 3. Examples of Radiation Exposure

Exam	**Effective dose mSv (mrem)**
CT Head	2.0 (200)
MRI Head	No radiation exposure
PET [^{15}O]Water	0.34 (34)
SPECT ^{99m}Tc HMPAO	6.9 (690)
PET [^{18}F] FDG	7.0 (700)

As the foregoing suggests, the risks of not performing a needed medical exam may be much greater than the risks of the radiation exposures associated with the exam.

Functional neuroimaging and data analysis

After the data has been obtained from the scanner, image processing is required to extract the quantitative information from the images as requested by the clinician. This information may include brain volume, blood flow, size and shape of brain structures, thickness of cortex, or functional activation. The major steps of data analysis include: (1) data acquisition, (2) pre-processing, (3) model estimation, and (4) analysis of results. Although, these steps are not standardized from one technology, machine, or laboratory to the next, there are two major methodological approaches for analyzing functional imaging data, which address the long-standing debate about functional specialization versus functional integration in the brain (Cohen & Tong, 2001).

The first approach, brain mapping, produces three-dimensional images of neuronal activation, showing which areas of the brain respond to a cognitive challenge. This method is also known as the study of functional specialization and generally proceeds using some form of Statistical Parametric Mapping (SPM). SPM is a voxel-based approach, which employs topological inference and classical statistics to explain regionally specific responses to experimental factors (Ashburner & Friston, 2000). Using SPM, functional neuroimaging data is spatially processed so that it conforms to an established anatomical space, in which responses are characterized, typically using the General Linear Model. To accommodate the spatial nature of the imaging data, and also account for the multiple statistical comparisons made, SPM techniques make use of Random Field Theory and/or other statistical procedures. The SPM technique can also be employed with structural data to find brain regions containing a higher gray matter density. This statistical method is known as Voxel-Based Morphometry (Ashburner & Friston, (2000).

The second approach is known as functional integration, where models are used to describe how different brain regions interact. A wide range of statistical techniques are available to measure inter-regional connectivity. For example, both unsupervised (e.g., Independent Component Analysis) and supervised techniques (e.g., support vector machines) are frequently used. Other models attempt to directly measure the causal connectivity based on statistics, statistical constraints (e.g., Structural Equation Modeling, SEM) or dynamic, more bio-physically motivated assumptions (e.g., Dynamic Causal Modeling, DCM) (Ashburner & Friston, 2000).

CHAPTER 7. ALGORITHM FOR ASSESSING FRONTAL LOBE FUNCTION

> "A science of the mind must reduce … complexities (of behavior) to their elements. A science of the brain must point out the functions of its elements. A science of the relations of mind and brain must show how the elementary ingredients of the former correspond to the elementary functions of the latter" (James, 1890, pg. 103).

As the previous chapters illustrate, FLD is a complex phenomena and the assessment of FLD is difficult, at best. More specifically, since all currently available tests, measures, and techniques lack predictive and diagnostic accuracy when used in isolation, the only way to reliable determine the presence/absence of FLD requires a multimodal and multidisciplinary approach. To that end, the algorithm discussed in detail below, which is based on a complete review of all relevant literature, attempts to provide a "gold standard" for assessing frontal lobe functioning that employs the most reliable measures currently available and emphasizes the importance of relying on a sound methodology in reaching a conclusion of FLD.

When a CT or MRI scan shows a gross structural abnormality such as, for example, a brain tumor, the distinction between normal and abnormal is clear since the presence of a brain tumor, at least statistically, points to an abnormal state. Similarly, when a subject scores significantly below the norm on neuropsychological tests impairment can easily be inferred. The problem arises when one attempts to determine the relationship between an abnormal neuroimage and/or below normal test performance and an individual's behavior, thoughts, or feelings (Brown & Eyler, 2006). There is no doubt that neuroimaging techniques can provide powerful visual evidence of FLD; however, because results may differ as a function of the conditions under which the scan was obtained, or the imaging technique employed, differentiating normal from abnormal

results is difficult at best (Knight & Stuss, 2002). Further, as previously noted, an image of diminished CBF in the frontal lobes, indicating hypometabolism, is simply a computer simulated reflection of physiological signals picked up by a camera geared to that specific signal. This image does not and can not fully tell us what is happening in that particular region of the brain, nor does it reveal the impact of the diminished blood flow on neighboring tissue or connected circuitry. Consequently, the image is subject to the imprecision of interpretation, so that it is highly inferential that the specific abnormal condition relates to a particular set of behaviors. In addition, just as an abnormal image may not necessarily relate to a brain deficit, a normal image does not necessarily mean that there are no functional deficits (Uttal, 2001).

Clearly, neuroimaging techniques alone cannot establish a definitive relationship between aggression and brain functioning, or forensically relevant abilities, such as competency to stand trial. Unfortunately, however, to many the concrete image stands on its own right and it is this dimension of the power of images that can result in distortions of scientific and legal truths. Therefore, while neuroimaging techniques can be helpful in the identification brain injuries or dysfunctions, the behavioral consequences of an injury or dysfunction can only be fully appreciated in the context of other information, such as a clinical interview and neuropsychological testing (Reeves, et al., 2003).

The algorithm

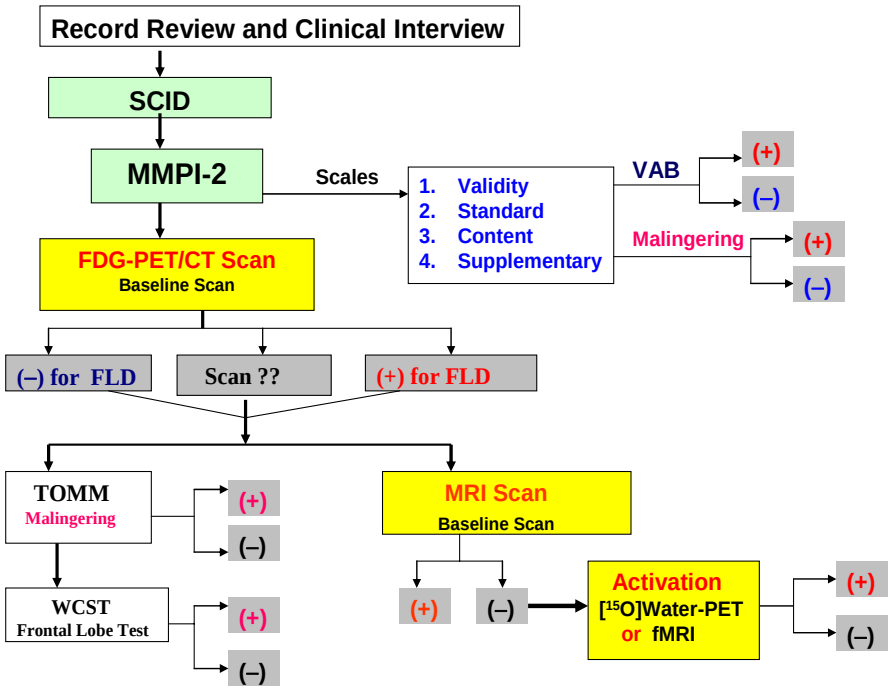
Given the increasing use of brain scans in criminal cases there is the possibility that brain imaging could, in the future, lead to changes in the legal standards used to determine culpability and/or future dangerousness. Consequently, the importance of the scientific validity and evidentiary reliability of tests, measures, and techniques used to

determine the presence or absence of frontal lobe impairment, and the importance of a multimodal assessment approach, cannot be overemphasized.

It has been suggested that many defendants who suffer from FLD fail to disclose symptoms, often because they lack insight that their behavior is maladaptive or socially inappropriate (Damasio & Anderson, 2003). Consequently, a thorough evaluation of frontal lobe functioning must consist of a clinical interview, a thorough review of past medical and behavioral history/records and collateral information sources, psychiatric and neurological examination, neuropsychological testing, and neuroimaging. Further, when used in isolation, neuroimaging and neuropsychological testing can overestimate, under-detect, or fail to detect the type and degree of brain dysfunction present (Brower & Price, 2001); thus, the importance of the clinical interview, psychiatric/neurological exam, and record review is underscored.

In addition, every currently available test, measure, or technique designed to assess frontal lobe dysfunction, malingering, and neurological, psychiatric, and psychological disorders has limitations which not only warrant, but necessitate, a multimodal approach for the assessment of frontal lobe functioning. Therefore, since each technique, test, and measure has limitations with regard to sensitivity, precision, and accuracy, the application of the following multimodal approach for the assessment of frontal lobe functioning is not only suggested, but highly recommended:

Figure 40. Algorithm for Assessing Frontal Lobe Dysfunction



As the algorithm illustrates, the assessment of FLD begins with a thorough record review and clinical interview. To assess the presence of Axis I and Axis II disorders, and the potential for malingering, the evaluation will continue with the SCID, followed by the administration of the MMPI-2. More specifically, since as previously discussed, a number of neurological, psychiatric, and psychological disorders have FLD as part of their symptomatology, employing the SCID and MMPI-2 to establish the presence or absence of such disorders, is crucial. Further, since the SCID is designed to complement and supplement the MMPI-2, the use of the SCID is also important.

Regardless of the results on the SCID and MMPI-2, the presence or absence of any gross brain abnormality, such as a brain tumor, will then be ascertained with FDG-PET/CT. Indeed, even if the SCID and MMPI-2 suggest the possibility of FLD, or suggest no FLD, the assessment process must continue, since these measures alone cannot establish the presence or absence of FLD with a high degree of certainty. Further, FDG-PET/CT is the most appropriate modality to employ, since it allows for the simultaneous assessment of structure and function, thereby eliminating the possibility of artifacts. The assessment will then continue with the administration of the TOMM followed by the WCST, irrespective of whether or not the FDG-PET/CT scan reveals a gross structural abnormality, is questionable, or normal. The decision to continue the assessment even if the FDG-PET/CT scan is abnormal is based on the fact that even if an individual has a gross structural abnormality, FLD can only be inferred. The decision to use the TOMM is based on the facts that (1) the rate of malingering in the forensic setting is presumed to be as high as 30%, (2) unlike the MMPI-2, this test is designed to assess malingering of cognitive deficits (executive function deficits), and (3) this test has high PPP. Further, the advantage of using the WCST is based on the facts that the WCST (1) is known to activate the DLPFC, which is of particular importance for performing executive function tasks, (2) is known to also activate the OFC, which has primarily been linked to aggression, (3) has been widely used to assess FLD, and (4) is the test most frequently used in neuroimaging frontal lobe activation studies.

Irrespective of the score obtained on the WCST, the assessment must continue, since the WCST also cannot establish with a high degree of accuracy the presence or absence of FLD, especially if the TOMM suggests malingering. Since a non-finding of a

gross abnormality with FDG-PET/CT does not imply a lack of structural or functional abnormality, structural abnormality must then be assessed using MRI.[39] If the MRI indicates either no structural or structural abnormality, a brain activation scan based on [^{15}O]Water-PET will then be obtained using an appropriate distractor task (control task) and the WCST to activate the frontal lobes. The absence of any structural abnormality on the MRI does not imply the absence of functional impairment; therefore, frontal lobe functioning must be assessed using a task known to activate the area of interest. The decision to rely on [^{15}O]Water-PET to assess functional impairment is based on the following:

1. PET is currently the only available neuroimaging technique that is truly quantitative.

2. The activation value is obtained by scanning the subject multiple times, while the subject is alternately performing the distractor task and the WCST, and then averaging the values.[40] [^{15}O]Water is the tracer of choice since it has the shortest half-life of all currently available PET tracers. More specifically, because of the short half-life of the tracer, multiple scans can be obtained, relatively quickly.

3. Because of the neuronal activity during activation, blood flow increases. More specifically, blood flow is the first physiological response to the activation and occurs almost instantaneously. Therefore, because activation tasks last only a few minutes,

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[39] It must be remembered that MRI is superior to CT in assessing structural abnormalities. More specifically, MRI has higher image contrast, and higher spatial and temporal resolution than CT.

[40] As previously discussed, obtaining a baseline scan is crucial, since the baseline value will be subtracted from the average activation value. However, with an "at rest" baseline scan there is a lack of control over what the person is thinking. Therefore, a distractor task is used in an attempt to ensure that the subject is not activating the brain area of interest, either willfully or inadvertently.

blood flow, rather than metabolism, is the most appropriate to assess. Further, the FDG-PET metabolism tracer does not allow for multiple activations, in a single setting. More specifically, the activation period has to last a significantly longer time because of the slow FDG uptake. In addition, while SPECT also measures blood flow, this technique is not optimal since the SPECT blood flow tracer has a long half-life.[41] Finally, SPECT has relatively poor spatial and temporal resolution compared to PET and is not a true quantitative measure.

If assessment with PET is not available or cost prohibitive, fMRI is the best alternative to PET. Indeed, the most practical way to assess activation is by means of BOLD fMRI, since (1) this technique is more widely available, and (2) multiple scans can be obtained in a short period of time because no tracers are required. However, in contrast to PET, fMRI BOLD is an indirect measure of blood flow. Further, since fMRI is semi-quantitative and PET is quantitative, PET is arguably a more objective and scientifically valid technique.

It may be argued that, while the algorithm is theoretically sound, its practical application is limited since, for example, PET and fMRI are not yet widely available and relatively expensive to use. However, to be of any real value in the clinical and forensic setting a methodology designed to assess FLD in the context of aggressive behavior must be able to reliably answer all of the following questions:

1. Does the individual/defendant have FLD?
2. If so, what is the FLD due to (e.g., is it due to a psychiatric disorder)?

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[41] Activation studies with SPECT take significantly longer than PET activation studies.

3. What is the effect of the FLD (e.g., cognitive impairment)?

In the clinical setting answers to all of the foregoing questions are needed in order to establish an appropriate treatment plan, while in the forensic setting these answers may be crucial for (a) establishing incompetency to stand trial or waive Miranda rights, (b) providing mitigating factors, (c) supporting a defense of NGRI, GBMI, or diminished capacity, (d) determining future dangerousness, and (e) plea bargaining. Finally, the inability to provide reliable answers to these questions may result in a *Daubert* challenge and possibly inadmissibility of scientific evidence.

It may also be argued that the data derived from a single subject may lack statistical reliability. However, neuroimaging techniques, such as MRI and PET, used to determine structural and functional abnormalities have been shown to have high sensitivity and specificity in routine clinical practice. In addition, since all currently available measures when used in isolation are unreliable, the algorithm was specifically designed to counter the above argument by employing a multimodal/multidisciplinary approach to the assessment of frontal functioning.

CHAPTER 8. CONCLUSION

In 2006, defendant *Williams* and his wife were indicted by a grand jury for felony murder based on their role in the alleged beating and killing of their five-year-old daughter. In July, 2008, the Government filed a motion to (1) exclude defendant's mental health expert witnesses at the guilt-phase of trial and (2) for an evidentiary *Daubert* hearing. The Court subsequently conducted an evidentiary *Daubert* hearing on the motion at which time the defendant presented Dr. Stewart, a clinical psychiatrist, and Dr. Young, a neuropsychologist. Drs. Young and Stewart opined that the defendant suffers from borderline intellectual functioning and "brain damage" which impairs his ability to understand and adapt to highly stressful situations. As a result, Dr. Stewart expressed his opinion that defendant did not have the capacity to form the requisite intent (*United States v. Williams*, 2009).

On rebuttal, the Government presented Dr. Resnick, a forensic psychiatrist, and Dr. Hall, a psychologist and forensic neuropsychologist. Drs. Resnick and Hall offered their opinion on Drs. Young and Stewart's diagnoses and methodology, concluding they were unreliable and unfounded.[42] The Court found Dr. Young's methodology reliable; the Government, however, contended that Dr. Young failed to conduct crucial tests that have a significant impact on the reliability of her overall methodology. More specifically, the Government argued that Dr. Young failed to conduct a functional MRI scan and a

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[42] Dr. Young administered a variety of tests to assess defendant's neural functioning. These procedures tested, among other things, defendant's intellectual functioning, his attention, motor, and learning skills, and his executive functioning, and included the WAIS-III, and the Test of Non-Verbal Intelligence.

quantitative EEG (QEEG) on defendant's brain that could have the potential to definitively prove or disprove defendant's claim of brain damage. In fact, according to the Government's expert, Dr. Hall, the fMRI scan could have increased the chances of detecting the kind of structural and functional damage that Dr. Young was describing (*United States v. Williams*, 2009).

At the conclusion of the *Daubert* hearing, the Government requested that the defendant undergo the fMRI and QEEG testing, but the Court made it clear that it would only order the testing upon a joint agreement by both parties. The defendant objected to the Government's request and the Court refused to order the defendant to undergo the tests. The Government subsequently asserted that Dr. Young's methodology remains inadequate and unreliable as a result of her failure to support her opinions with results from an fMRI and QEEG and sought to exclude her testimony (*United States v. Williams*, 2009). The Court concluded that while it may be true that additional testing may have uncovered the extent of defendant's brain damage, there was nothing inherently unreliable about the tests Dr. Young did conduct. Consequently, the Government may use Dr. Young's failure to administer the fMRI and QEEG to go to the weight of her testimony but it does not preclude the admissibility of her testimony (*United States v. Williams*, 2009).

The *Williams* case clearly underscores (1) the importance of employing the multimodal approach, developed here, (2) the need for experts to understand and acknowledge the limitations of neuropsychological tests and neuroimaging techniques, and (3) the difficulty of making a valid and reliable determination of scientific evidence proffered in a court of law. This case also illustrates a number of common

methodological shortcomings, which have prompted the development of the algorithm. For example, defense experts did not administer malingering tests to ensure that the results of the neuropsychological tests were not compromised. Further, the defense experts' sole reliance on neuropsychological tests that formed the basis of their opinion is problematic, since these tests in isolation cannot establish "brain damage" with a high degree of certainty. Since "brain damage" can only be reliably ascertained with neuroimaging techniques, the Government's assertion that the defense expert's methodology was inadequate and unreliable as a result of her failure to support her opinions with results from an fMRI and QEEG was, therefore, especially appropriate. However, with regard to QEEG, the current position of both the American Academy of Neurology and the American Clinical Neurophysiology Society is that, due to its methodological and technical limitations, QEEG is not recommended for use in criminal proceedings (Martell, 2009).

To be sure, neuropsychological or neuroimaging evidence cannot establish a lack of criminal responsibility, which is a legal determination, not a medical one. Taken together, however, this evidence can paint a rich portrait of a defendant's FLD and its potential causal role in the criminal behavior in question. However, while brain injury or dysfunction may impair decision-making or influence violent tendencies, there is no single violent brain region. As the case of Phineas Gage illustrates, it is possible to suffer an extremely traumatic frontal lobe brain injury, even one that results in drastic personality changes, and still refrain from engaging in anything inherently aggressive. Further, while neuroscience has contributed greatly to our understanding of brain structures and functions, there is still no consensus as to whether impairment of a

particular region provides a direct link to behavior. Currently, the best neuroscience can offer is to provide yet another piece of the puzzle (Batts, 2009).

It has been suggested that neuropsychological testing serves as a natural bridge between the structural and functional neurophysiological parameters assessed by neuroimaging, and the actual cognitive and behavioral strengths and weaknesses of an individual that may be relevant in a court of law (Denney & Sullivan, 2008; Martell, 1992). Therefore, because of its ability to link neuroimaging scan patterns with specific behaviors, neuropsychological testing should be routinely incorporated as an integral part of all forensic neuroimaging cases as a means of assisting the trier of fact in understanding the behavioral ramifications of any brain defects identified by either structural or functional imaging (Martell, 2009).

The strengths of neuroimaging are its objectivity and specificity to the individual, while the strengths of psychological testing are its generalizability and its measurement of psychological functions. There is no doubt that, with further research using larger numbers of subjects and with meta-analyses, neuroimaging may eventually achieve clinical utility in assessing forensically relevant mental disorders and their effects on cognitive functioning. However, even with the anticipated improvements, we should never rely on a single measure, however impressive or technologically advanced, to impute a causal connection between violent and aggressive behavior and structural or functional abnormality. Indeed, as has been so aptly argued:

> "It is not the technology that is limiting. Rather it is the failure to recognize that all technologies have limitations that affect the ability of legal and medical practitioners to infer causal relationship" (Tancredi & Brodie, 2007, p. 294).

While studies reviewed for this project and the resulting methodology may appear to be unidimensional in their approach to determining the potential cause of aggressive behavior, they should, in fact, be interpreted within the framework of Barak's (1998) interdisciplinary criminology, where different perspectives relevant to a specific behavior are treated as being complimentary. Understanding that each perspective of violence and aggression, in fact, offers a reality of that behavior from an interrelated, yet different, angle allows us to develop a network of theories that will provide the most accurate information and capture the most dimensions about the phenomena of aggressive behavior. To that end, this appraisal of knowledge from the fields of neuropsychology and neuroscience merely attempts to refine the image of aggression and violence without supplanting, or making obsolete, other paradigms and perspectives. More specifically, the intent is not to reduce violent and aggressive behavior to brain functioning but to inform the scientific community of limitations of current analyses and their importance to future studies of violent and aggressive behavior. The evidence hoped to be gained from future studies relying on this newly developed methodology is valuable, then, not because it may lead to reliable predictions of aggressive behavior, but because it further elucidates the image of aggression and violence (Bufkin & Luttrell, 2005).

Appendix A. Examples of SCID-II Self-Report Questions using a Yes/No format

Antisocial Personality

105. Before you were 15, did you ever bully or threaten other children?

106. Before you were 15, did you ever start a fight?

107. Before you were 15, did you ever hurt or threaten another person with a weapon, like a bat, gun, or knife?

108. Before you were 15, did you ever deliberately torture another person or cause another person physical pain?

109. Before you were 15, did you ever hurt an animal on purpose?

110. Before you were 15, did you ever rob, mug, or take something from a person by threatening that person?

111. Before you were 15, did you ever force someone to have sex with you, get undressed in front of you, or touch you in a sexual way?

112. Before you were 15, did you ever set fires?

113. Before you were 15, did you ever deliberately destroy things that weren't yours?

114. Before you were 15, did you ever break into a car or house?

Borderline Personality

90. How many times have you become frantic when you thought that someone you really cared about was going to leave you?

91. Do your relationships with people you care about have many extreme ups and downs?

92. Did you ever suddenly change your sense of who you are and where you are going?

93. How many times did your sense of who you are change dramatically?

94. Are you sometimes different with different people or in different situations, so that you don't know who you really are?

95. How many times have you suddenly changed your goals, religious beliefs, and so on?

96. How many times have you done things impulsively?

97. Did you ever try to hurt or kill yourself or have threatened to do so?

98. Did you ever burn, cut, or scratch yourself on purpose?

99. Does your mood suddenly change?

Appendix B. Alphabetical List of Abbreviations

AAS	Addiction Acknowledgement Scale
ACC	Anterior Cingulate Cortex
AD	Alzheimer's Disease
ADHD	Attention-Deficit-Hyperactivity Disorder
AMA	American Medical Association
APA	American Psychological Association
APD	Antisocial Personality Disorder
APS	Addiction Potential Scale
BD	Bipolar Disorder
BOLD	Blood Oxygenation Level Dependent
BPD	Borderline Personality Disorder
CRB	Cerebral Blood Flow
CBV	Cerebral Blood Volume
CT	Computed Tomography
COWAT	Controlled Oral Word Association Test
CS	Clinical Scales
CVLT	California Verbal Learning Test
DLPFC	Dorsolateral Prefrontal Cortex
DS	Digit Span
EEG	Electroencephalography
EF	Executive Functions

FBS	Fake Bad Scale
FLD	Frontal Lobe Dysfunction
FIT	Rey 15-Item Memory Test
fMRI	Functional Magnetic Resonance Imaging
FTD	Frontotemporal Dementia
GNG	Go/No-go Task
IED	Intermittent Explosive Disorder
MA	Methamphetamine
MCMI-III	Millon Clinical Multiaxial Inventory-III
MDD	Major Depressive Disorder
MFIT	Memory for 15-Item Test
MMPI-2	Minnesota Multiphasic Personality Inventory-2
MRI	Magnetic Resonance Imaging
MTT	Mean Transit Time
NIM	Negative Impression Management Scale
NPP	Negative Predictive Power
NRC	Nuclear Regulatory Commission
OCD	Obsessive-Compulsive Disorder
OFC	Orbitofrontal Cortex
PAI	Personality Assessment Inventory
PDRT	Portland Digit Recognition Test
PET	Positron Emission Tomography
PFC	Prefrontal Cortex

PMD	Primary Mood Disorder
PMT	Porteus Maze Test
PPD	Primary Psychotic Disorder
PPP	Positive Predictive Power
QEEG	Quantitative Electroencephalography
rCMRglc	Cerebral Metabolic Rate for Glucose
rCRB	Regional Cerebral Blood Flow
RCS	Restructured Clinical Scales
SCID	Structured Clinical Interview for the DSM-IV
SIRS	Structured Interview for Reported Symptoms
SPECT	Single Photon Emission Computed Tomography
SPM	Statistical Parametric Mapping
STROOP	Stroop Color Word Test
SVT	Symptom Validity Test
TBI	Traumatic Brain Injury
TMT	Trail Making Test
TOMM	Test of Memory Malingering
VIP	Validity Indictor Profile
VSVT	Victoria Symptom Validity Test
WAIS-III	Wechsler Adult Intelligence Scale-Third Edition
WCST	Wisconsin Card Sort Test
WMT	Word Memory Test

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Table 4. Calculation of Accuracy Indices

	Actual	
	Malingering	Honest
Predicted		
Malingering	A	B
Honest	C	D
Sensitivity	A/A + C	
Specificity	D/B + D	
PPP	A/A + B	
NPP	D/C + D	

Note: PPP = Positive Predictive Accuracy
NPP = Negative Predictive Accuracy

Table 5. Classification of Malingered Neurocognitive Dysfunction (Slick, et al., 1999)

Criterion B: Evidence from Neuropsychological Testing

Criterion C: Evidence from Self-Report

Criterion D: Behaviors are not fully accounted for by Psychiatric, Neurological, or Developmental Factors

Classification	Criterion A	Criterion B	Criterion C	Criterion D
Definite malingering	X	X	(X)	X
Probable malingering	X	X (two pieces)		X
Or				
Probable Malingering	X	X (one piece)	X (one piece)	X
Possible Malingering	X		X	X

*Must Include Definite Negative Response Bias