Neurobiological research that confirms Freud's trauma theory and its implications for treatment with children diagnosed with reactive attachment disorders

Carla Gerber-Weintraub
Smith College

Follow this and additional works at: https://scholarworks.smith.edu/theses

Part of the Social and Behavioral Sciences Commons

Recommended Citation
https://scholarworks.smith.edu/theses/1063

This Masters Thesis has been accepted for inclusion in Theses, Dissertations, and Projects by an authorized administrator of Smith ScholarWorks. For more information, please contact scholarworks@smith.edu.
ABSTRACT

This theoretical thesis argues that children diagnosed with reactive attachment disorder are best served with psychoanalytic treatment. Four major components are examined to support this argument. First, the progression of reactive attachment disorder criteria set forth in the 1980 DSM-III up through a recent DSM V proposal are reviewed. Secondly, a history and development of Freud’s trauma theory is discussed. Thirdly, neurobiological research of the social brain and limbic system that confirm certain hypotheses of Freud’s trauma theory are outlined. Lastly, current treatment modalities used with children diagnosed with reactive attachment disorder are scrutinized. Each of these components leads to the conclusion that psychoanalytic treatment is the most effective treatment for children diagnosed with reactive attachment disorder.
NEUROBIOLOGICAL RESEARCH THAT CONFIRMS FREUD’S TRAUMA THEORY AND ITS IMPLICATIONS FOR TREATMENT WITH CHILDREN DIAGNOSED WITH REACTIVE ATTACHMENT DISORDER

A project based upon an independent investigation, submitted in partial fulfillment of the requirements for the degree of Master of Social Work.

Carla Gerber-Weintraub
Smith College School for Social Work
Northampton, Massachusetts 01063
2010
Acknowledgements

This thesis would not have been possible without the guidance and inspiration of many individuals. I am most grateful for each person’s contribution concerning this exhaustive endeavor.

First, I would like to acknowledge Smith College School for Social Work who required the completion of this thesis. The process of researching and writing taught me the joy of independent learning; a gift that will continue throughout my life.

Special thanks go to Holly Simons, research professor, for inspiring and solidifying my topic. Joyce Everett, thesis advisor, for giving patient, kind, and informative guidance. Dennis Meihls, second reader, for giving me the opportunity to present my neurobiological chapter during our senior seminar class.

Appreciation goes to Smith College School for Social Work BSW Advance Standing 2010 cohorts who rallied and cheered me throughout this intensive process. I want to thank Rebecca Weintraub, my daughter, for her success as the first college graduate in our family which paved my way; Mary Russell, my friend, for her encouragement and hours of proof reading, Sara Weinberger and Mordi Kamel, my Northampton family, who were continuously in my corner; Linda Gerber, my sister, for her support and perseverance throughout the editing process; and Roger Gordy, my dearest friend, who unwaveringly walked with me every step of the way.
# Table of Contents

ACKNOWLEDGEMENTS ........................................................................................................ ii  

TABLE OF CONTENTS ...................................................................................................... iii 

LIST OF FIGURES ........................................................................................................ iv 

CHAPTER 

I INTRODUCTION ............................................................................................................. 1 

II REACTIVE ATTACHMENT DISORDER ........................................................................... 6 

III FREUD’S TRAUMA THEORY ...................................................................................... 23 

IV NEUROBIOLOGICAL RESEARCH OF THE SOCIAL BRAIN AND THE LIMBIC SYSTEM AND HOW ITS FINDINGS SUPPORT FREUD’S TRAUMA THEORY ........................................................................................................... 33 

V IMPLICATIONS FOR TREATMENT OF REACTIVE ATTACHMENT DISORDER ................................................................. 59 

REFERENCES .................................................................................................................. 72
List of Figures

Figures

1. MAJOR STRUCTURES WITHIN THE LIMBIC SYSTEM..............................  34
2. DIAGRAM OF THE HYPOTHALAMUS-PITUITARY-ADRENAL AXIS.....  40
CHAPTER ONE

INTRODUCTION

The purpose of this theoretical study is to argue that psychoanalytic treatment is most effective when working with children diagnosed with Reactive Attachment Disorder (RAD).

There are many reasons why this theoretical exploration of RAD is important. First, RAD is a relatively new diagnosis. Secondly, there seems to be confusion around defining and diagnosing RAD. Thirdly, there are many different treatments used today with children diagnosed with RAD. The question is; which treatment is most effective?

One of the major criteria that define RAD is pathogenic care of children under the age of five. Children diagnosed with RAD have had an early childhood history of trauma. In the late 19th century, Sigmund Freud began his medical journey by treating patients with “Hysteria.” Patients with Hysteria presented with unexplainable physical symptoms such as blindness or paralysis that apparently had no biological cause. While working with patients diagnosed with hysteria, Freud discovered that early childhood trauma was a common factor. At the time, Freud was a neurophysiologist, and from that perspective he developed theoretical hypotheses regarding the treatment of trauma. Today, neuroscientists through empirical studies and the use of modern technology have revealed that many aspects of Freud’s theory are supported by their findings. Since 21st century neurobiology is confirming many of Freud’s trauma related hypotheses, it has given resurgence to the effectiveness of psychoanalytic treatment.

During the mid to late 20th century, behavioral psychology took precedence over psychoanalytic theory. A focus upon symptom reduction with measurable and quantifiable short
term methods replaced traditional long term psychoanalysis. It was during a time when science became Truth and Freud was irreverently referred to as Sigmund Fraud. This thesis argues that children with infant and early childhood attachment disorders are more effectively treated with psychoanalysis. Symptom reduction may extinguish unwanted behaviors, but many times the quenched behavior would be replaced with a new negative behavior. For instance, through behavioral modification a person might stop smoking however; in many cases the smoking was replaced with overeating. When we concentrate on a child’s presenting behaviors and ignore the underlying cause for those behaviors, we may have success with symptom reduction, but have we effected healing? This thesis proposes that children diagnosed with RAD need longer term treatment where a positive attachment can form within the therapist/patient dyad which can replace and heal an earlier primary-caretaker/child maltreatment. The hypothesis examined in this thesis is that by giving the child a new opportunity to experience a healthy attachment then the previous drive for negative behaviors is removed.

This thesis focuses on four major components; the definition of RAD, a description of Freud’s Trauma Theory; an overview of neurobiological findings that support Freud’s Trauma Theory; and a description of how psychoanalytic treatment can be effectively applied to working with children diagnosed with RAD. The importance of determining an effective RAD treatment is due to the increase of reported child abuse and neglect cases over the last decades. Many of these children are at high risk for a lifetime of psychopathology.

Reactive Attachment Disorder was introduced in 1980 in the DSM III. Since then the criteria have gone through many changes. With the anticipated 2013 edition of the DSM V, prominent researchers have proposed a major change to the criteria set forth for Reactive
Attachment Disorder in Infancy and Childhood. A thorough review of those changes will be addressed in the RAD chapter.

Some clinicians believe that RAD is over-diagnosed; others believe that it is under-diagnosed. RAD research has only been conducted during the last twenty years. Although some clarification has been gained, there are still many questions that need to be explored. Professional agreement upon the definition of RAD is still in question. For instance, should the RAD diagnosis have two sub-types, inhibited and disinhibited, or should the two sub-types become two separate diagnoses? Should Attachment Theory be incorporated into the diagnostic criteria? Should disorganized attachment be considered an attachment disorder? If a child presents with a disorganized attachment, does this mean that the child’s symptoms fit the criteria for RAD? If criteria for Attention-deficit/hyperactivity disorder and Oppositional Defiant Disorder consistently present with children diagnosed with RAD, should those criteria be incorporated into the RAD diagnosis or should a comorbid diagnosis be given to the child? These questions just begin to explore the diagnosis of Reactive Attachment Disorder.

Methodology

The basic methodology used to prepare this theoretical thesis was to review pertinent publications regarding Reactive Attachment Disorder, Freud’s Trauma Theory, Social Brain and the Limbic system, Neurobiological findings that support Freud’s Trauma Theory, and current Treatment of Reactive Attachment Disorder. Psychological literature from 1850 to 2010 was reviewed. Google scholar was used to initiate the search. The Smith College Library Journal Finder was most effective in retrieving full text PDF articles. Many articles were located and retrieved from the Psychoanalytic Electronic Publishing site. Required and suggested readings,
both textbooks and articles, in the Syllabi of Smith College School for Social Work classes were a great aid. The process of reading the literature also provided additional resources.

RAD literature is limited due to the fact that it is a relatively new diagnosis. Definition, history, and treatment of RAD written by researchers and clinicians were reviewed. A DSM V proposal suggesting radically new criteria was summarized. The history of neurology and Freud’s early medical training as a neurologist was incorporated into this thesis. Both Freud’s original writings and literature review of his writings were used to understand Freud’s Trauma Theory. Knowledge of neurobiology of the social brain and limbic system was gathered from articles and textbooks of today’s most prominent trauma neuroscientists.

Each chapter individually discusses a major component of this thesis. The second chapter of this thesis provides an overview of Reactive Attachment Disorder. This chapter discusses the prevalence of child abuse and neglect, the history of the diagnosis, newly proposed definitions, research findings, and a DSM V RAD proposal. The third chapter presents a history of Freud’s early medical training under Charcot and Janet. Freud’s *Project for Scientific Psychology* and *On Aphasia* are discussed to illustrate how Freud’s neurological perspectives influenced psychoanalysis. The *Study of Hysteria* written with Breuer is also discussed. The development of Freud’s Trauma Theory is traced. The fourth chapter addresses current neurobiological findings. It provides an overview of the limbic system describing the amygdala, hypothalamus, hippocampus, and pituitary gland and how this specific neuroanatomy responds to trauma. The hypothalamus-pituitary-adrenal axis is also described in detail to illustrate how trauma is processed through the brain. This neurobiological chapter concludes with outlining the intersection of modern neurobiology with Freud’s trauma hypotheses and how Freud’s Trauma Theory is supported by modern neurobiological technology. The last chapter is dedicated to an
overview of current treatments used with children diagnosed with Reactive Attachment Disorder and attempts to integrate all of the previous components. It is in this chapter that through recent literature, an argument can be made that psychoanalytic treatment is the most effective way to heal Reactive Attachment Disorder.

The next chapter begins with a description of the history and definition of RAD. The history of the diagnosis is outlined from the original submission to the DSM III right up to the most recent DSM V proposal. This historical discussion discloses the complexity of the diagnosis and the challenges clinical social workers have when making a treatment decision.
CHAPTER TWO

REACTIVE ATTACHMENT DISORDER

The purpose of this chapter is to provide an overview of Reactive Attachment Disorder in children. Those children who are at risk for developing Reactive Attachment Disorder (RAD) may have been institutionalized, placed in multiple foster homes, or chronically and severely abused and/or neglected by a primary caregiver. The onset of this disorder occurs while the child is under the age of five. The chapter begins with a summary of the U.S. Department of Health and Human Services 2008 Report on child abuse and neglect as one way to describe the potential incidence of RAD. Reactive Attachment Disorder is a relatively new diagnosis which was introduced in the 1980 Diagnostic and Statistical Manual of Mental Disorders (DSM III). Since that time, the definition of RAD has been continuously challenged and changed. To fully understand the current definition of Reactive Attachment Disorder, an outline of Attachment Theory is provided. The criteria set forth in the Diagnostic and Statistical Manual of Mental Disorders (DSM IV TR) and the World Health Organization International Classification of Disorders (ICD) 2007 version of RAD are described and compared. A critique of those definitions are discussed which lead to a new DSM V proposal. The new proposal makes such significant changes that it replaces the DSM IV TR (2000) RAD criteria with two suggested separate disorders: Reactive Attachment Disorder in Infancy and Childhood and Disinhibited Social Engagement Disorder. Both of these newly proposed diagnoses are reviewed.
In the chapters that follow, Freud’s Trauma Theory and Neurobiological Research of the Social Brain and Limbic System are compared and contrasted. Those Freudian hypotheses supported by neuroscience are outlined. Once the intersection of Freud’s Trauma Theory and neurobiological findings are identified, treatments currently used with children diagnosed with Reactive Attachment Disorder are analyzed. The intention of this analysis is to show that psychoanalytically based practice supported by neuroscience is the most effective treatment for children diagnosed with Reactive Attachment Disorder.

**Reactive Attachment Disorder**

The definition of Reactive Attachment Disorder is very broad. According to Hanson & Spratt (2000), there is “considerable disagreement about what RAD actually entails, and in particular, what types of assessments and interventions to use with effected children and families” (p. 137). Disagreements range from behaviors and characteristics of RAD symptoms to whether or not it is either under-diagnosed or over-diagnosed (Zilberstein, 2006). Since the parameters of Reactive Attachment Disorder tend to be nonspecific, data about the incidence of reactive attachment disorder among children is limited. Therefore in an attempt to define RAD, this chapter will begin with statistics regarding child abuse and neglect. These statistics are significant because one of the DSM IV TR (2000) criteria is that pathogenic caretaking before the age of five must occur.

While the prevalence of RAD is estimated at 1% of all children under the age of five ([http://www.minddisorders.com/Py-Z/Reactive-attachment-disorder-of-infancy-or-early-childhood.html#ixzz169AWxGJ3](http://www.minddisorders.com/Py-Z/Reactive-attachment-disorder-of-infancy-or-early-childhood.html#ixzz169AWxGJ3)), given the prevalence of child abuse and neglect, the number of children at risk for a diagnosis of reactive attachment disorder is actually much higher. According to the U.S. Department of Health and Human Services (2008) child abuse and neglect
are most prevalent between the ages of birth to three; the younger the child – the higher the risk. According to the Child Welfare Information Gateway 2008 Report, [http://www.childwelfare.gov/pubs/factsheets/fatality.pdf](http://www.childwelfare.gov/pubs/factsheets/fatality.pdf) research indicates that 33% of all maltreatment reported cases were children of age birth to three. Age one had the highest percentage of maltreatment with 12.3 percent. These statistics are highly relevant due to the fact that the onset of RAD is before the age of five. ([http://www.childwelfare.gov/pubs/factsheets/fatality.pdf](http://www.childwelfare.gov/pubs/factsheets/fatality.pdf))

Also in 2008, the U.S. Department of Health and Human Services reported that the most prevailing type of maltreatment is neglect and accounts for 71.1 percent of all cases reported where physical abuse accounts for only 16.1 percent (U.S. Department of Health and Human Services [http://www.childwelfare.gov/pubs/factsheets/fatality.pdf](http://www.childwelfare.gov/pubs/factsheets/fatality.pdf)). Hanson & Spratt (2000) report behavioral differences between a child who has been abused and a child who has been neglected. Abused children tend to exhibit “confusion, fear, ambivalence, an inability to explore their environment effectively, aggression, and hypervigilance in their interactions with others” (p. 139). In contrast, neglected children seem to show other characteristics such as “difficulty communicating, appear to expect a lack of cooperation from their parents, and may become prematurely independent” (Crittenden & Ainsworth, as cited in Hanson & Spratt, 2000. p. 139). Studies have also shown that the interaction between a caretaker and neglected child may contain negative verbal feedback and interpersonal avoidance (Hanson & Spratt, 2000).

There are two major considerations when diagnosing a child with RAD. First, one must assess how the child relates in social situations and whether or not their behaviors are developmentally appropriate. A child’s social interactions have a wide range. A child may show inhibited behaviors such as being overly cautious, withdrawn, and hypervigilant or a child may
exhibit disinhibited behaviors such as excessive friendliness with strangers and exercising poor character judgment. Secondly, one must consider the quality of caretaking the child received before the age of five. The quality of the interpersonal relationship between the caretaker and the child is important history to gather during a clinical assessment particularly with those children considered to have a high risk for developing RAD. According to Hanson & Spratt (2000), the relationship between the caretaker and the child must be reciprocal. They distinguish the difference between the terms bonding and attachment. They claim that bonding is the caretaker’s feeling toward the child, as opposed to attachment which is the child’s feeling toward the caretaker. In the case of a child diagnosed with RAD, Hanson & Spratt (2000) argue that both the caretaker’s bonding and the child’s attachment must be problematic. Hanson & Spratt (2000) express concern that the criteria set forth in the DSM IV TR do not distinguish between bonding and attachment and instead uses the term pathogenic care too broadly.

Hanson & Spratt caution the clinician not to over-diagnose reactive attachment disorder. A child having a history of abuse and neglect does not necessarily indicate a RAD diagnosis, but a “history of neglect and/or abuse compromises the ability to form stable attachments with a primary caregiver, and therefore might result in RAD in some cases” (p. 139). According to the APA (1994) DSM IV there are two sub-types of RAD; inhibited and disinhibited. These subtypes will be discussed more specifically in the DSM IV TR (2000) definition section of this chapter. However, according to Zeanah & Gleason (2010) “emotionally withdrawn/inhibited RAD is exceptionally rare – even among young children raised in institutions” (p. 8). In fact, Zeanah & Gleason’s (2010) conclusion seems to be that both sub-types do occur but are actually exceedingly rare (Zeanah & Gleason, 2010). On the other hand, Zeanah & Gleason (2010) also report that many studies using continuous and categorical measures affirm that “the two types of
RAD can be reliably identified in maltreated, institutionalized, and formally institutionalized children” (p. 1). Zeanah & Gleason (2010) are not necessarily questioning the possibility of over-diagnosing as much as whether or not the DSM IV TR RAD criteria are comprehensively written. Their concern seems to be that the narrow scope of the APA (1994) DSM IV TR RAD criteria may exclude children who exhibit RAD behaviors but do not necessarily meet the criteria as currently written.

**Attachment Theory**

RAD is the first childhood diagnosis added to the *Diagnostic and Statistical Manual of Mental Disorders* that is considered a disorder of attachment. Therefore, an overview of Attachment Theory will help give a more comprehensive understanding of RAD. Attachment Theory was introduced in the 1960’s by John Bowlby whose view of attachment was influenced by observations of young children separated from familiar caregivers. According to Davies (2004) a child seeks out one specific person to meet their needs. The child’s discrimination for one or two specific caretakers begins between the seventh and ninth month (Zeanah & Gleason, 2010). The primary goal of the relationship is to create a sense of security for the child. Davies claims that a primary care taker/child relationship is a universal phenomenon. From the early weeks of birth, a baby engages in a caretaker/child dyadic. The infant recognizes the caretakers face, voice, and smell. (Davies, 2004). Through the continued interaction between caretaker and child, the child develops several healthy behaviors such as affect regulation, communication, emotional reciprocity, and interpersonal play. Eventually the child’s curiosity motivates them to explore the world around them. When the child has experienced a positive attachment, the trusted caretaker becomes a “*base for exploration*” (Davies, 2004. p. 10).
Ainsworth (1978) proposed two types of attachment; secure and insecure. Currently attachment theory sub-divided insecure attachment leaving us with four different classifications of attachment: 1) Secure; 2) Insecure – Avoidant; 3) Insecure – Ambivalent/Resistant; 4) Insecure – Disorganized/Disoriented (Davies, 2004 p.12). A child who has not developed a healthy attachment (i.e., secure attachment) to their primary caretaker typically forms an insecure attachment. Children with RAD usually present with the fourth type, Insecure – Disorganized/Disoriented but not all children exhibiting a disorganized/disoriented attachment necessarily have RAD. Secure attachment in a young child resembles safety, security, confidence, and trust. Children who have had the disadvantage of neglect, abuse, multiple foster homes, or inadequate institutional care are prone to feeling unsafe, insecure, doubtful, and distrustful. According to Hardy (2007) “Infants are intrinsically driven to form attachments” (p.28). Due to this intrinsic drive, the infant will attach to the primary caregiver regardless of the caregiver’s treatment. In the case of disorganized/disoriented attachment, the child may exhibit a mixed reaction by perceiving the caregiver’s attention as giving both comfort and distress. The child will then express this inner conflict by behaviorally reaching out and pulling away simultaneously (Hardy, 2007). This disorganized/disoriented attachment many times leads to psychopathology throughout the child’s life. Corbin (2007) summarizes several theorists regarding the impact of abuse and neglect in infants quite eloquently:

In the absence of appropriate dyadic attachment experiences during infancy, children grow into adulthood exhibiting difficulties in their ability to self-soothe, self-organize, regulate affect, and engage in healthy relationships (Bowlby; Kradin, Winnicott ; as cited in Corbin 2007 p. 540).
History of the Development of the DSM RAD Definition

Reactive Attachment Disorder (RAD) was introduced to the *Diagnostic and Statistical Manual of Mental Disorders, Third Edition* (DSM-III) in 1980 (American Psychiatric Association, 1980). A RAD diagnosis presumably provides a clinical description or picture of the context in which certain behaviors develop and problems that a child might have in relating to others (Hanson & Spratt, 2000). Over the course of thirty years the nosological definition of RAD has changed and evolved to its present criteria found in the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision* (DSM-IV-TR) (American Psychiatric Association, 2000). According to Richters & Volkmar (1994), the criteria set forth in the DSM III “included symptoms such as failure to thrive, a lack of developmentally appropriate social responsiveness, apathy, and onset before eight months” (p. 328). In 1987 changes were made in the DSM III R (1987). Most importantly a change from eight months to five years was set forth. The “failure-to-thrive symptoms were no longer defining features” (p. 328). The new criteria emphasized that RAD was caused by pathogenic care and it was stated that with treatment the condition could improve (Richters & Volkmar, 1994). Despite the effort to improve the criteria in the DSM III R (1987), there remained controversies amongst some practicing clinicians. Set factors for the cause of RAD needed to be more specific. Also, the definition for RAD in the World Health Organization (ICD) was inconsistent with the DSM III R’s (1987) definition. It was also suggested that the term “maltreatment syndrome” may be more suited than the term RAD (Richters & Volkmar, 1994).

According to Zeanah & Gleason (2010) the sub-types, inhibited and disinhibited, were influenced by a 1975 London study conducted by Tizard & Rees with children raised in residential nurseries where caregivers were discouraged from developing attachments with them.
The study was constructed to concentrate on one variable; care-giver/child relationships. Tizard & Rees observed three outcomes: 1) Some children were emotionally withdrawn and unresponsive; 2) Some children were indiscriminately social with both strangers and familiar people. These children were attention seeking and clingy; and 3) Some children managed to select a caregiver and subsequently developed an attachment. Tizard & Rees’ description of these phenotypes, inhibited and disinhibited, were incorporated into the DSM III R (1987) definition of RAD (Zeanah & Gleason, 2010).

When RAD was introduced in the DSM III in 1980 it focused upon growth failure of the child and an inability to be socially responsive (Zeanah & Gleason, 2010). The onset was set at the age of eight months. The child’s behaviors could not be supported by biological or medical reasons. A history of gross neglect and abuse was a criterion (Zeanah & Gleason, 2010). Criticisms of the RAD DSM III (1980) definition mainly focused upon the onset of eight months being developmentally premature for infants to form attachments, and there were no empirical studies supporting the idea that growth failure or failure to thrive had a direct link to poor attachment. Too many behaviors such as “poor tone, weak cry, excessive sleep, lack of interest in the environment, and weak rooting and grasping when feeding” (p. 3) were overlapping criteria with other childhood disorders such as non-organic failure to thrive, a diagnosis that has since been removed from the DSM-III-R (1987). When the DSM III R (1987), that is a revised edition of the DSM III (1980), was published, it changed the onset from eight months to five years or younger. Also significant, the addition of the two sub-types derived from the Tizard & Rees study was incorporated into the criteria.
Currently Published Reactive Attachment Disorder Definitions

**DSM IV TR.** *American Psychiatric Association (2000) Diagnostic and Statistical Manual of Mental Disorders IV Text Revision* (DSM-IV-TR) criteria for 313.89 Reactive Attachment Disorder of Infancy or Early Childhood are “markedly disturbed and developmentally inappropriate social relatedness” (p. 130) caused by various insecure attachments by the age of five; the symptoms and behaviors should not be accounted for by mental retardation or from the autistic spectrum; and pathogenic care caused by abuse, neglect, or frequent changes of primary caretaker. The DSM IV TR (2000) also requires a clinician to specify whether or not the child’s symptoms are inhibited (shy and withdrawn) or disinhibited (indiscriminate social ability).

**World Health Organization ICD Version 2007.** WHO ICD categorizes two separate disorders, Reactive Attachment Disorder of Childhood and Disinhibitive Attachment Disorder of Childhood. The description set forth in the ICD-10 (2007) Reactive Attachment Disorder of Childhood includes consistent abnormalities of the child’s social responses to its environment and the people in it. Some of the behaviors cited are hypervigilance and fearfulness that cannot be comforted by another person, aggressive behavior toward self and others, difficulty relating to their peers and others, a pervasive depression, flat affect, and in some cases delayed growth. Typically these children have been exposed to chronic abuse, neglect, or mishandling before the age of five. The ICD-10 (2007) Reactive Attachment Disorder of Childhood behaviors and symptoms coincide with the inhibited subset of the DSM IV TR (2000) Reactive Attachment Disorder of Infant and Childhood. The children have not experienced an attachment to a specific caretaker. The ICD’s (2007) exclusions are similar to the DSM IV.TR (2000) (World Health Organization, 2007) [http://apps.who.int/classifications/apps/icd/icd10online/](http://apps.who.int/classifications/apps/icd/icd10online/)
The second disorder in the ICD-10, Disinhibitive Attachment Disorder of Childhood, is defined as an abnormal social functioning with an onset before age five. It does specify, however, that symptoms persist even with a positive environmental change. Children exhibiting Disinhibitive Attachment Disorder symptoms typically have been raised in institutions, however, other living situations can have the same effect. The key is the number of caretakers prior to age five where a child has formed no consistent attachment. Very young children will present as clingy and scattered. As the child matures the clinging behavior is typically replaced with attention seeking behavior. The child can be overly friendly with strangers but, in contrast, have very poor peer relations. Some children may also have emotional and behavioral issues. (World Health Organization, 2007)  

http://apps.who.int/classifications/apps/icd/icd10online/  

**Similarities and Differences of the DSM-IV-TR (2000) and ICD (2007) Definitions**

Both the DSM-IV-TR (2000) and ICD (2007) definitions agree that the onset of RAD occurs during the first five years of a child’s life. There is also agreement that severe parental neglect and/or abuse, and many environmental changes, such as multiple foster homes or inferior institutional care, may be responsible for causing the disorder.

The main difference between the DSM-IV TR (2000) and the ICD (2007) is how they define inhibited behavior as opposed to disinhibited behavior. The DSM-IV-TR (2000) incorporates the two differences and requires the clinician to specify the type of Reactive Attachment Disorder. The ICD (2007), however, separates the two types as two different diagnoses. ICD (2007) Reactive Attachment Disorder resembles the type specified in the DSM-IV TR (2000)as Inhibited type. The DSM IV TR (2000) Disinhibited type is referred to as the ICD (2007) Disinhibited Attachment Disorder of Childhood . The DSM-IV TR (2000) gives a general statement that symptoms should not be accounted for by mental retardation or the autistic
spectrum. In contrast, the ICD (2007) excludes very specific disorders and syndromes as noted above.

**Critiques of DSM-IV TR and ICD (2007) RAD Criteria**

The central concern with the diagnostic criteria set forth in the DSM-IV TR (2000) and the ICD (2007) is that it is narrow in its scope. Viewing secure and reactive attachments with such a black and white perspective leads to many questions. For instance, what about children who form attachments with caretakers but the relationship is pathological? Many children who have disruptive attachments in foster care and the adoptive community are not recognized as having RAD by the DSM-IV Tr (2000) or by many clinicians (Zilberstien, 2006). Yet, poor and disruptive attachment in children “may present differently at different developmental ages” (Zilberstien, 2006. p. 57). For treatment purposes, particularly psychodynamic treatment, having four specific descriptions of attachment is more effective than being bound by the DSM-IV TR’s (2000) inhibited/disinhibited choice (Corbin, 2007).

From a behavioral perspective, behaviorists emphasize identifying specific behaviors and personal characteristics those children with RAD exhibit. Behaviorists agree that psychologically healthy children have formed strong and secure attachments to their caregivers. They also assent to the concept that children with RAD do not have the ability to have healthy attachments due to their early neglect and abuse. Though an understanding of attachment is important, their focus and treatment concentrates on a child’s behavior patterns. There are many anti-social behaviors observed in children with RAD. Some examples are the inability to give eye contact, lying for no reason at all, fire-setting, cruelty to animals, lack of remorse, eating disorders, promiscuity, overly friendly with strangers but standoffish with family members, inappropriate sexual activity with self and others, hyperactivity, developmental delays, unusual
speech patterns, and pre-occupation with violent acts (Hall & Geher 2003). The DSM-IV TR (2000) addresses specific criteria where maladaptive behaviors are easily identified. In this sense Hall & Geher (2003) agree with the definition found in DSM-IV TR (2000). They believe that the observation of attachment is much more difficult than focusing on the obvious behavior. Hall & Geher (2003) summarize their position thusly:

Because both behaviors and attachment patterns are integral to the diagnosis of RAD, it may be most appropriate for researchers to address the behavioral difficulties of these children and for the diagnostic criteria to include identified common behaviors in addition to an exploration of attachment patterns (Hall & Geher, 2003 p. 147).

From a developmental consideration another question one might ask is how does poor and disruptive attachment reveal itself during different developmental stages? Zilberstien (2006) reports that RAD research up to this point in time has mainly focused upon young children under the age of six. What effect does RAD have upon middle childhood and adolescence? This is an area that has not been studied and clinicians many times may misdiagnosis older children (Zilberstien, 2006).

In addition to Attachment Theory, Developmental Theory, and Behavioral Theory, Neurobiology of RAD is also important to consider. Neurobiological findings show that early neglect and abuse actually alters brain development, particularly in the right hemisphere of the brain. These brain alterations, even if attachment is repaired as the child becomes older, will affect personality and behavior throughout adulthood (Hardy, 2007). Research indicates that the dyadic connection between infant and caretaker “is the most important factor in the infant’s early development” (Hardy, 2007, p.28). The connection can be measured by energy patterns between the infant and caretaker. Hardy continues to describe the neurobiological significance of attachment during infancy:
When the energy expenditure fails, the right brain is unable to maintain coherent neural connections and goes into a state of shock that leads to dissociation and stalls normal development. When both internal and external regulatory systems fail, a sense of helplessness (inability to self-maintain) and hopelessness (loss of predictable outer structure) could result (Hardy, 2007, p. 29).

Since neurobiology is supporting the significance of attachment between infant and caretaker, it would follow that the DSM IV TR’s (2000) criteria for RAD is lacking due to the fact that it does not take attachment into consideration. Another complicating factor to diagnosing RAD and developing a comprehensive set of criteria, is comorbidity. Many of the behaviors and symptoms seen in children with RAD overlap with such diagnoses as Pervasive Developmental Disorder (PDD; Autistic spectrum), Attention-Deficit/Hyperactivity Disorder (ADHD), developmental delays, and neurologic deficits to name a few. The challenge for clinicians will be to glean other diagnoses from the presenting RAD symptoms. The guidelines written in the DSM-IV TR (2000) clearly state that “when an individual has more than one Axis I disorder, all of these should be reported” (APA, DSM-IV, 2000, p. 27). Zilberstien sums the importance of comorbidity nicely, “Considering attachment to be comorbid with other conditions rather than encompassing them deepens our formulation and broadens our repertoire of available treatments” (Zilberstien, 2006, p. 62).

**Proposed DSM-5 Criteria of Reactive Attachment Disorder**

Development website [www.dsm5.org/ProposedRevisions/Pages/proposedrevision.aspx?rid=120](http://www.dsm5.org/ProposedRevisions/Pages/proposedrevision.aspx?rid=120) had accepted a proposed revision of 313.89 Reactive Attachment Disorder in Infant and Early Childhood. An extensive review was prepared by Charles H. Zeanah, MD and Mary Margaret Gleason, MD of the Department of Psychiatry and Behavioral Sciences at Tulane University School of Medicine. Zeanah & Gleason (2010) have prepared and submitted a comprehensive document outlining the history of RAD and summarizing research conducted in the last twenty years with children diagnosed with RAD. They have proposed to the APA DSM-5 Committee many changes, the most significant of which is influenced by the ICD-10. As opposed to using the limited phenotypes, inhibited and disinhibited, Zeanah & Gleason (2010) suggest having two different diagnoses: Reactive Attachment Disorder in Infancy and Early Childhood and Disinhibited Social Engagement Disorder.

**Proposed Reactive Attachment Disorder in Infancy and Early Childhood**

Zeanah & Gleason (2010) propose six criteria for RAD in Infancy and Early Childhood:

1. A child under the age of five who exhibits “disturbed and developmentally inappropriate attachment behaviors” particularly when the child is in distress. The child will either refuse or not respond to comfort from an adult when distressed; or the child will simply not seek comfort when distressed. The child’s overall presentation is that of “inhibited, emotionally withdrawn behavior.”

2. Social and emotional disturbance characterized by at least two of the following behaviors: (a) social and emotional response to others is minimal or non-existent; (b) an overall flat affect; and/or (c) an exaggerated response of irritability, sadness, or fear when interacting with adult caregivers.
3. The symptoms observed in the child should not meet the criteria for Autistic Spectrum Disorder (ASD).

4. A history of neglectful caretaking with any one of the following: (a) ignoring basic emotional needs for nurturing and comfort; (b) absent or limited attention to physical needs; (c) disruptive placements, such as multiple foster homes, which prevents the child from forming consistent attachments; and/or (d) living environments such as residential institutionalized care with a high rate of staff turnover.

5. Pathogenic care is responsible for any “disturbed and developmentally inappropriate attachment behaviors.

6. The developmental age of the child is at least nine months.

**Proposed Disinhibited Social Engagement Disorder**

Zeanah & Gleason (2010) propose five criteria for Disinhibited Social Engagement Disorder:

1. If a child engages with strangers with at least two of the following concerns: (a) little or no caution when interacting with unfamiliar adults; (b) verbal or physical behavior which is too demonstrative; (c) unaware of the presence or need for the presence of a familiar adult when interacting with the stranger; and/or (d) exercising poor judgment by being too willing to venture out with the stranger.

2. The above-described behavior with strangers should not be confused with the impulsivity of ADHD. The behavior must be socially disinhibited.

3. A history of abuse or neglect by at least one of the following: (a) absent or limited response to child’s emotional needs when distressed; (b) physical and psychological
safety is ignored; (c) severe and harsh punishment; (d) unstable and inconsistent primary
caretaking; and/or (e) residential institutional care with high staff turnover.

4. If pathogenic care is responsible for the child’s indiscriminate and inappropriate
interpersonal relations with a stranger.

5. The developmental age of the child is at least nine months.

Zeanah & Gleason (2010) report that there are many more differences between the two
disorders than similarities. Some examples are: (1) inhibited and emotionally withdrawn
children have little interest in engaging with adults, as opposed to disinhibited children have an
indiscriminant need for adult interaction; (2) quality of caregiving is related to inhibited
behaviors, whereas disinhibited children will exhibit criteria despite their caretaking history; and
most significantly, (3) inhibited children are very responsive to intervention, whereas
disinhibited are either marginally responsive or not responsive at all. According to Zeanah &
Gleason (2010) an attachment disorder can be diagnosed when a child appears to be perfectly
capable of forming an attachment, but does not form an attachment due to a history of
maltreatment. This definition can be applied to Reactive Attachment Disorder in Infancy and
Childhood; however, it does not best describe Disinhibited Social Engagement Disorder. Zeanah
& Gleason (2010) report that the major difference between the two disorders is that in the case of
Disinhibited Social Engagement Disorder, the child may or may not have developed an
attachment. In fact, some children presenting with the criteria of Disinhibited Social
Engagement Disorder have had secure attachments. Zeanah & Gleason (2010) claim that “it is
not necessarily attachment behaviors that are disinhibited, but rather social engagement or
affiliative behaviors that are expressed non-selectively” (p. 28).
Defining Reactive Attachment Disorder takes many aspects into consideration: different theories; multitude of research with different findings; and different diagnostic criteria. Added to this complexity are the many different treatment approaches. Treatments will be reviewed in the last chapter. Since this thesis’ hypothesis is that psychoanalytic treatment is the best choice for children diagnosed with Reactive Attachment Disorder, the next chapter describe Freud’s Trauma Theory.
CHAPTER THREE

FREUD’S TRAUMA THEORY

This chapter traces how Freud’s trauma theory ultimately resulted in the development of psychoanalysis. During Freud’s work with patients diagnosed with hysteria, Freud developed hypotheses into a formal discipline now referred to as Freud’s trauma theory. Freud’s transition from neurologist to psychoanalyst is reviewed and defined. Neurobiological findings that support Freud’s findings are discussed in the next chapter.

Jean-Martin Charcot and the Hôpital du Salpetrière in Paris

Freud’s trauma theory was first formulated during the late 19th century while he studied under the neurologist Jean-Martin Charcot in the Hôpital du Salpetrière in Paris. Charcot was considered the patriarch of the etiology of hysteria and highly regarded in the field of neurology. It was Charcot who hypothesized those patients somatic systems stemmed from a trauma.

Hysteria during Charcot’s time was never systematically defined since the term hysteria was commonly used (Herman, 1992. p.10). It is believed that the term hysteria dates back to Hippocrates 466 – 377 B.C.E. (Huopainen, 2002; Laplance & Pontalis, 1973). Many definitions have been applied to the term hysteria. One such definition is “for twenty-five centuries, hysteria has been considered a strange disease with incoherent and incomprehensible symptoms. Most physicians believe it to be a disease proper to women and originating in the uterus” (H. Ellenberger, as cited by Herman, 1992, p.10.). Hysteria was also thought of as a catch-all phrase that physicians used to describe mysterious symptoms found in women (Herman, 1992). Modern definitions that have been formulated include: “An hysteric…is someone who goes
through life pretending to be who he really is” (Bromberg, 1996; Laing, 1962) and “an hysteric is like a glass of water without the glass” (Bromberg, 1996, p. 55). During the late 1800’s Charcot proposed two types of hysterical trauma, “la grande hystérie” and “la petite hystérie” the latter of which had less severe symptoms (Huopainen, 2002). Interestingly, this trauma theory construct is still considered today (Huopainen, 2002; van der Kolk et al., 1996).

**Pierre Janet’s Influence upon Freud’s Trauma Theory**

During Freud’s tenure at the Hôpital du Salpetrière, he was greatly influenced by Pierre Janet’s work with hysterical patients. Under Charcot’s supervision, Janet managed the hospital’s laboratory where he worked directly with hysterical patients. He studied 591 patients of whom 257 cases experienced some sort of trauma (van der Kolk, 2000; crocq and Le Verbizier, 1989). Janet based his work on Charcot’s hypothesis that the somatic symptoms patients presented with stemmed from a trauma. Janet found that “intense emotional experiences can dramatically affect the way that people process information” (van der Kolk, 2000, p. 238). According to van der Kolk (2000), Janet determined that when the capacity to integrate information becomes overwhelmed it causes abnormal memory processing. The processing actually splits off the memory from the person’s conscious awareness. The trauma is then stored in the memory as an “unconscious fixed idea.” Once the trauma is no longer in the patients’ consciousness, the trauma may be stored in the sensory and emotional memory and intrude as “terrifying perceptions, obsessive preoccupations, and as somatic re-experiences such as anxiety reactions” (Janet, 1889; van der Kolk & van der Hart, 1989, 1991; van der Kolk, 2000). Central to Janet’s theory is dissociation.

Modern trauma theorists describe dissociation as an identity which has two personalities, one which is considered normal, and the other which is considered emotional. When a patient
cannot flee or fight, they instead dissociate by either separating themselves from the event, referred to as derealization, or psychically disconnecting from their body, referred to as depersonalization (Huopainen, 2002; Myers, 1940; van der Kolk et al., 1996). Freud, Charcot, Janet, and Breuer did not use the modern terminology of derealization and depersonalization; however, their written works describe the phenomena. Janet also proposed that unless the patients integrated their trauma into their consciousness that they were at a risk of personal and occupational deterioration (Janet, 1889; van der Kolk & van der Hart, 1989; 1991; van der Kolk, 2000. p. 238). Janet’s description of the hysterical patient’s process of their traumatic experience seems to fit today’s criteria for post traumatic stress disorder (van der Kolk, 2000. p. 238).

**Breuer and Freud Collaborate**

After Freud’s work with Charcot and Janet at the Hôpital du Salpetrière, Freud left Paris and moved to Vienna. It was at this time that he collaborated with Joseph Breuer and together they wrote a significant work *Studies in Hysteria*. In this work we are introduced to the famous case study, Anna O. Breuer administered intensive psychotherapy daily to Anna O. This intensive work resulted in a transference/countertransference experience, though Breuer had no understanding of this psychoanalytic phenomenon at the time. In the end, Anna believed that she was pregnant with Breuer’s child, though it was psychosomatic. Breuer abruptly ended his treatment with her. Anna ended up hospitalized due to the devastation of the abrupt separation. Freud and Breuer used the case of Anna O. to illustrate their hypotheses regarding their work with hysteria. In the case of Anna O., her symptoms, like many of Freud’s trauma patients, were rooted with early childhood sexual abuse.

In both written works, Freud & Breuer stayed true to Pierre Janet’s understanding of dissociation in the hysterical patient (van der Kolk, 2000). Breuer and Freud believed that the
patient’s traumatic memories were too painful to be kept in the conscious mind. Instead, the memories were stored in the patient’s body and were expressed as physical symptoms. These symptoms appeared to have no medical cause. Because Breuer and Freud considered the somatic symptoms of patients as having no organic cause instead they believed that the mind has an unconscious life that is hidden from the hysterical patient. They also believed that traumatic memories were held in the patient’s unconscious.

According to van der Kolk (2000), Breuer and Freud’s *The Phenomenology of Hysteria* documented “some remarkable insights…[that are]…supported by contemporary psychological and neuroscientific studies of traumatic phenomena” (van der Kolk, 2000. p. 239). For example, one insight is the splitting of the conscious after a hysteric has experienced a profoundly frightening trauma. The patient dissociates during the traumatic event. The memory of the traumatic event is outside of the consciousness but is still psychically present. Consequently, the memory remains, and the patient’s experiences are affected by the past traumatic memory. Breuer and Freud asserted that if these dissociated memories are not released by way of abreaction then the memory pathology persists. In fact, the memory can persist for a long time, and the original profound fright will still be experienced by the hysteric as if the original trauma was reoccurring. Breuer and Freud make note that most memories will fade, however, in the case of a traumatic memory, the incident is outside of the consciousness, but the psychic affect remains as intensely as it was first experienced. This intensity continues to occur because the patient has not reacted to the trauma with reactive affect, instead the reaction is suppressed but the affect of the trauma is psychically attached to the memory (Breuer & Freud, 1893; van der Kolk, 2000).
When the patient does not have a strong affective reaction to the initial trauma, not only does the memory split from the consciousness and become psychically attached with its original intensity, the emotion attached to the traumatic memory becomes strangulated. In other words, the patient becomes emotionally stuck. Breuer and Freud encouraged their patients to re-tell the trauma with strong affect. They believed that releasing the memory and the strangulated affect through speech was an effective cure. It was clearly stated, however, that if a patient recalls her/his trauma without affect, there will be no results. The patient must recall the event with detail and with strong affect, then the hysterical symptom will disappear completely. These were Breuer and Freud’s principles of the “talking cure” (Breuer & Freud, 1893; van der Kolk, 2000).

Breuer and Freud stayed true to their description of the “talking cure” in their second publication *Studies of Hysteria* by stating that:

> each individual hysterical symptom immediately and permanently disappeared when we had succeeded in bringing clearly to light the memory of the event by which it was provoked and in arousing its accompanying affect, and when the patient had described that event in the greatest possible detail and had put the affect into words. Recollection without affect almost invariably produces no result (Breuer & Freud, 1883-1895; Strachey, J., 1955, p.4).

Breuer and Freud also discovered with their patients that their hysteria was not based upon one event; it was an accumulation of many events. They go on to say:

> In the case of common hysteria it not infrequently happens that, instead of a single, major trauma, we find a number of partial traumas forming a group of provoking causes” (Breuer & Freud, 1883-1895, Strachey, J., 1955, p.4).

The quotes below are significant for two reasons: (1) This chapter addresses whether or not Freud’s trauma theory is applicable to the understanding and treatment of children diagnosed with reactive attachment disorder; and (2) Freud’s discovery that many of his hysterical patient’s were victims of child sexual abuse takes him in a different theoretical direction:
The disproportion between the many years' duration of the hysterical symptom and the single occurrence which provoked it is what we are accustomed invariably to find in traumatic neuroses. Quite frequently it is some event in childhood that sets up a more or less severe symptom which persists during the years that follow (Breuer & Freud, 1883-1895, Strachey, J., 1955, p.4).

van der Kolk et al. (1996) also brought attention to the importance of Freud’s discovery of a high rate of childhood trauma. According to van der Kolk et al, they indicate that most of the childhood trauma concerned child sex abuse.

Early in their work [Breuer & Freud] these two clinicians firmly adhered to Janet's notion that traumatic experiences, especially when they occurred early in life, were at the origin of psychological conflict and symptom-formation: in 1896 Freud published his observations of 18 consecutive hysterical patients, in all of whom he attributed the origins of their symptoms to a history of childhood sexual trauma (van der Kolk et al., 1996).

Freud’s patients with a history of child abuse seemed to have no conscious memory of the childhood trauma. Freud needed to find a way to tap into the patient’s repressed memory. As a neurologist, Freud came to understand that there was a neurological disconnect between the conscious and the unconscious (Berzoff, 2010). This led him to experiment with hypnosis as a way to delve into the patient’s unconscious mind. Eventually, his work with hypnosis morphed into what Freud termed “the cathartic method” and what we refer to as free association. The discovery of the cathartic method actually occurred somewhat accidentally. According to Bromberg (1996), while Freud was treating Emmy von N, he tended to concentrate upon asking her the cause of her symptoms. She experienced his questioning as annoying which prompted her to tell Freud to stop asking her questions and just let her talk about whatever she felt like saying. Her request became Freud’s “fundamental rule.” This cathartic free association was considered by Freud as the raw data which the analyst would “then link together and transform into the substance of [a] cure” (Bromberg, 1996. p. 57). Free association and the fundamental
Freud (1904) described the sessions as proceeding "like a conversation between two people equally awake, but one of whom is spared every muscular exertion and every distracting sensory impression which might divert his attention from his own mental activity" (p. 250) (Freud as cited in Kanzer, 1972, p. 249)

The object was to have the patient through either hypnosis or free association to remember the traumatic memory with affect. It appeared to Freud and Breuer that the emoted memory erased away the somatic symptom.

Freud believed that the somatic symptoms of hysterical patients were repressed traumatic memories. He believed that if the patient could talk through their memory, the somatic symptom would cease to exist. However, it was important that the patient remember the memory with affect in order for the “talking therapy” to remove the somatic symptom(s). The goal of the therapy was for the patient to gain self-knowledge and through that self-knowledge unearth repressed memories. By exploring and remembering the past, the patient would ultimately work through the original trauma which would result in relieving the patient of those somatic symptoms associated with the patient’s traumatic event (Berzoff, 2010).

Through the hypothesis that somatic symptoms – such as paralysis, limited speech, deafness, and blindness – are physical expressions of past and forgotten trauma, Freud began to see the somatic symptom as a symbol which represented the trauma. For instance, in the case of Anna O, her sexualized transference to her therapist Breuer expressed itself as a hysterical pregnancy, the pregnancy being a symbol of Anna’s sexualized feelings and love for Breuer. Another possible example might be if a child shared a room with an older sibling and that child
observed the older sibling being sexually abused by their father, then perhaps the patient’s symbolic symptom may be blindness, as the patient may no longer want to “see.”

Freud also believed that feelings of the repressed memory were strangulated. The hysterical patient could not express the emotions attached to the memory. It was as if the patient were stuck emotionally and psychologically at the age the trauma occurred. Freud hypothesized that if the memory could be consciously realized with affect, then the emotions associated with the trauma would be able to be freely expressed by the patient. By freely expressing the emotion attached to the trauma, then according to Freud, the patient would become unstuck and emotions would no longer be strangulated (Berzoff, 2010).

Breuer and Freud’s collaboration came to an end when Breuer fell in love with Anna O. According to Judith Herman (1992), Breuer may have discontinued Anna O’s sessions because his wife was resentful that he and Anna’s sessions were so intense. For over two years, Breuer saw Anna daily and then abruptly discontinued the sessions when he became aware that she was passionately attached to him. This abrupt disruption resulted in Anna O needing to be hospitalized. Freud continued his work with hysterics; Breuer never practiced again. However, Freud did convince Breuer to write the Anna O case study in *Studies of Hysteria* (Herman, 1992, p. 18).

According to Kihlstrom & Hoyt (1986), Janet and Freud agreed that the traumatic memory of a hysteric does not remain in the patient’s conscious mind. Both also agreed upon the existence of a psychological unconsciousness where the traumatic memory is stored. Their theoretic departure concerned the mechanism responsible for the loss of the traumatic memory from the conscious mind. Janet theorized that dissociation split the memory from the conscious mind. Freud hypothesized that the mechanism was repression of the memory from the conscious
mind to the unconscious mind. Throughout the decades, psychodynamic theorists and clinicians have argued the difference between dissociation and repression. There seems to be a minor difference, but to Janet and Freud, it was enough for them to break ties. Later, when Freud introduced his theory of childhood sexual fantasy in place of childhood sexual abuse, Janet chastised Freud publically by stating, “We all know that in hysteria we sometimes find sexual fixed ideas, but one should not ground a general theory for hysteria on these cases” (Bromberg, 2002. p. 96; Ellenberger, 1970). Once Freud was pressured by Viennese intellectuals’ icy receptions of his “Aetiology of Hysteria” presentation at the Vienna Society for Psychiatry and Neurology, his theory shifted from childhood sexual abuse to childhood sexual fantasy. Even the “world famous sexologist Richard von Krafft-Ebing condemned Freud’s original theory claiming an epidemic of child sexual abuse as having presented a “scientific fairy tale” (Bromberg, 2002. p. 95; Masson, 1992). For Freud this was the end of his theory based in the teachings of Charcot and Janet at the Hôpital du Salpetrière in Paris.

**Freud’s Personal Reasons for Abandoning Trauma Theory**

According to Huopainen (2002), historians came upon a letter from Freud to his friend Fliess. The letter is dated September 21, 1897. Freud (1892 – 1899, pp 259 -260) tells his friend that there were four basic reasons why he has lost faith in his trauma work with hysterics. First, he was discouraged by many patients who dropped out of therapy and by “poor treatment results” (p. 97) Secondly, Freud found that an increase of hysterical patients had early childhood trauma that pointed to fathers sexually abusing their daughters. Thirdly, he was frustrated with trying to distinguish his patients’ “internal and external realities found in their fantasies, memories and affect” (p. 97). Lastly, the fourth reason was his challenge with penetrating the patients’ unconscious to reveal the hysterics repression (Huopainen, 2002. p. 97).
A direct translation of Freud’s letter is as follows:

So I will begin historically from the question of the origin of my reasons for disbelief. The continual disappointments in my attempts at bringing my analysis to a real conclusion, the running-away of people who had for a time seemed most in my grasp, the absence of the complete successes on which I had reckoned, the possibility of explaining the partial successes in other ways, on ordinary lines (Freud, 1892 – 1899, p 259 as cited in Huopainen, 2002. p. 97)

In the next chapter, this thesis summarizes neurobiological research and findings that inform our understanding of effects of trauma. Bessel van der Kolk is a leading psychiatrist who, for decades, has been studying Post Traumatic Stress Disorder (PTSD) with brain imaging. His scientific findings have supported many of the Charcot, Janet, Breuer, and Freud’s early work with hysterics. In addition to modern technology, treatment of PTSD and reactive attachment disorder (RAD) in children is enhanced with psychopharmacology. Though cognitive behavioral therapy has proven to be helpful with PTSD and RAD, a resurgence of psychoanalytic theories has re-emerged. Attachment theory is based upon psychoanalytic theory and is used particularly with children diagnosed with RAD. Brain technology has confirmed that traumatic memory causes impairment in the brain. The next chapter summarizes neurobiological findings confirming particular tenets of Freud’s trauma theory. Before doing so, the next chapter will give an overview of the neuroanatomy of the social brain and limbic system.
CHAPTER FOUR

NEUROBIOLOGICAL RESEARCH OF THE SOCIAL BRAIN AND THE LIMBIC SYSTEM AND HOW ITS FINDINGS SUPPORT FREUD’S TRAUMA THEORY

This chapter concentrates on neurobiological research on early childhood attachment. It explores how early childhood abuse and neglect affects the growth and development of the brain, which has particular relevance for the development of children diagnosed with RAD. Since the 1990’s researchers have discovered how traumatic experiences have affected the brain’s limbic system. Through cutting edge medical technology, such as magnetic resonance imaging (MRI) and positron emission tomography (PET), researchers have come to understand the physiological affect of the flight/fight/freeze response during a traumatic experience. A new understanding of long-term dysregulation and the lasting effects of trauma on the brain are discussed. The research has also given new insight into how traumatic memory is stored. This chapter also provides an overview of the anatomy of the brain’s limbic system and the direct influence it has upon memory. Both these aspects, the limbic system and memory function, are crucial to understanding infant attachment, early childhood environment, and primary care relationships. Recent neurobiological research and a comprehension of the social brain and limbic system seems to play a significant role in clinical social work treatment of children diagnosed with reactive attachment disorder. Once the social brain and limbic system have been reviewed, the chapter then integrates Freud’s Trauma Theory with recent neurobiological findings.
The Social Brain and the Limbic System

The brain is a complex organ consisting of many parts. There are three major sections to the brain, the hindbrain, midbrain, and forebrain (Wolman, 1996, p. 81). According to van der Kolk, (1996) the brain also has “three interdependent subanalysers” (p. 214). First there is the “brainstem and the hypothalamus which are primarily associated with the regulation of internal homeostasis” (p.214) meaning that it helps regulate emotion. Second in line is the limbic system, “which is charged with maintaining the balance between the internal world and external reality” (p. 214). The last of the three interdependent subanalysers is “the neocortex, which is responsible for analyzing and interacting with the external world” (p. 214).

The limbic system, which is considered one of the oldest areas in the brain, is located deep within the forebrain hidden from view. Not only was the limbic system the oldest in the evolution of the species, but it is also the first part of the brain to prenatally develop (Cozolino, 2006. p. 51). It controls “emotional responses and the formation of memory” (Doctor & Shiromoto,. 2010. p. 164). According to Cozolino (2006) this hidden primitive structure is central to understanding ourselves and connecting with others (p.50). It is the structure that directly affects “caretaking, relationships, and emotional experience” (p. 52). Cozolino informs us that it is the right side of the brain that controls social and emotional functioning. He also notes that the human brain is never fully developed but that throughout our entire lives, the brain is “undergoing constant development and reconstruction” (p. 50). He describes a study involving new mother rats and her pups. The study demonstrated growth in the mother rat’s hypothalamus and hippocampus which are specific parts of the limbic system. (These parts will be discussed in detail below.) The growth in the hypothalamus and hippocampus was influenced by two factors: the hormone fos during pregnancy; and the interaction between the new mother
rat and her pups. The hippocampus also grew larger during breeding which accommodated the new mother to expand her memory of food sources (Cozolino, 2006).

In addition to the hypothalamus and hippocampus there are several other major components that make up the limbic system: amygdala, corpus callosum, the cingulated gyrus which protects the corpus callosum, and the pituitary gland (See Figure 1). The three major components of the limbic system that processes social information are the hypothalamus, amygdala, and hippocampus (Adolphs, 2003; Cozolino, 2006). The next section describes these three later parts. In order to comprehensively discuss the three major limbic components, an understanding of how memory works is essential. This chapter provides an overview of memory function. Memory plays an important role with regard to diagnosis and treatment of reactive attachment disorder in children. The growth and the development of the brain is a major consideration in how children may or may not process or remember early childhood trauma.
Figure 1. Major structures within the limbic system


**Amygdala**

The amygdala and its pathways are more closely related to emotion and motivation whereas the hippocampus controls the memory function (Nicholi, 1999. p. 104). The hippocampus works in conjunction with the amygdala (Cozolino, 2006). The amygdala is an “almond-shaped structure which is made up of ‘interconnected nuclei’ ” (Cozolino, 2006. p. 56). It is located in front of the hippocampus, but lies within the temporal lobe. Its function is to process “experiences of fear, attachment, early memory, and emotion across the lifespan” (Cozolino, 2006. p. 52). The amygdala is “essential to nurturing-behavior and fear-
conditioning” (Ratey, 2002. p.66). According to Ratey, studies have shown that when the amygdala is removed from animals two significant effects occur: 1) the young are neglected; and 2) they “forget previously formed [associations] with particular stimuli” (p. 66). The middle and bottom sections of the amygdala are more primitive. It is this portion of the amygdala that assesses environmental danger and triggers the fight/flight/freeze response. Interestingly, according to Cozolino, the amygdala can be stimulated by either real or imagined danger. It can be triggered by both conscious and unconscious thought (p. 222). The result is over-stimulation which causes an increase in cortisol. This concept is important when treating children diagnosed with reactive attachment disorder because “sustained high levels of stress partly explain why early negative experiences in parenting and attachment have a lifelong impact on physical health, mental health, and learning” (p.222).

As mentioned above, the limbic system is the first to develop prenatally. The amygdala is close to being fully matured by the eighth month of gestation. (Cozolino, 2006). After birth, when the infant is attaching to its primary caretaker, the amygdala governs the child’s relational experience through “eyes, hands, touch, sound, and smell” (Badenoch, 2008. p, 52). It is through this relational experience that the infant determines whether or not the world is a safe place. This is communicated to the child by the primary caretaker’s response, or lack of response, to the child’s expressed needs. An over-sensitive amygdala may be associated with anxiety, panic disorder, post traumatic stress disorder, and attention deficit hyperactivity disorder (Ratey, 2002).

**Hippocampus**

Also within the temporal lobe is the hippocampus. Its task is for learning and short-term memory. Researchers believe that the hippocampus also converts short-term memory into long-
term memory (Shannon, 2010, p. 6). In addition, both the “amygdala and the hippocampus are responsible for storing the ‘danger’ memory for future recall” (Doctor & Shiromoto, 2010, p. 164). The hippocampus regulates “the organization of spatial, sequential, and emotional learning and memory” (Colozino, 2006, p. 57; Edelman, 1989; McGaugh et al, 1993; Sherry et al, 1992; Zola-Morgan & Squire, 1990). Unlike the amygdala, the hippocampus does not fully develop until early adulthood. It is believed that the slow development of the hippocampus is directly related to consciously forgotten traumatic childhood memories (Cozolino, 2006; Fuster, 1996; Jacobs et al, 2000; McCarthy, 1995).

The hippocampus and the amygdala are directly attached to the olfactory nerves which gives us a sense of smell. Smell, in many cases, can promote memories (Ratey, 2002). Sometimes odors may recall good memories like the smell of an apple pie baking in the oven. However, for some, such as a victim of rape, a particular scent of a man’s cologne can stir up memories of the previous rape causing increased anxiety and fear. Since the hippocampus stores memories, it functions as a regulator by comparing old memories with present moments. If a person is startled by something harmless, the hippocampus will “work as a protective filter” and will calm down the fight/flight/freeze response to danger (Ratey, 2002). When a person experiences a traumatic event, the amygdala responds to the event emotionally. The more intense the emotion, the longer more permanently the memory is stored in the hippocampus. However, if the emotion is truly intense, the stress hormone called cortisol is produced in excess amounts resulting in lasting effects upon the brain. It is also interesting to note that depression may affect a person’s ability to identify different odors. The inability to identify smells is significant because “smells are strong prompters of memories.” According to Ratey, “nerves
that communicate smells to the amygdala and hippocampus are crucial components to a person’s memory” (p. 68).

This increase of cortisol inhibits the hippocampus from categorizing and evaluating the traumatic incident. The memory of the incident becomes fragmented. According to Ratey the person does not remember the whole event. Instead of total recall, the person experiences the traumatic memory through their senses. This sensory recall is separate from other life memories. In order for the person to survive the trauma, they dissociate from the experience which results in fragmented memory. The memory through the senses stays with the person indefinitely. The result is that the person experiences the emotion but cannot attach a memory to it (Ratey, 2002). In the case of young children, “neglect and emotional deprivation in the first years of life lead to left hippocampal shrinkage, corpus callosum damage, and dendritic burnout” (Fosha, D., 2003. p.222). Many studies have shown a slow development of the hippocampus which may account for a loss of conscious memory in early childhood (Cozolino, 2006; Fuster, 1996; Jacobs et al, 2000; McCarthy, 1995). Slow development of the hippocampus and hippocampal shrinkage may be important factors with regard to reactive attachment disorder in children. The slow development of the hippocampus directly affects the child’s memory. Until the hippocampus is fully developed at age two, the child will not be able to retain cognitive facts which is referred to as explicit memory. Explicit memory will be described in more detail in the memory section of this thesis.

**Hypothalamus**

The hypothalamus’ basic function is to regulate specific body functions. According to Ratey, “because it plays a crucial role in the regulation of body systems, the hypothalamus is
often referred to as the brain of the brain” (Ratey, 2006. p. 172). Some of the functions are thirst, hunger, activity level, temperature regulation, aggression, and sexual behavior (Cozolino, 2006).

The hypothalamus is an important component regarding affect regulation. When a primary care taker is responsive to an infant’s stress signals, they are essentially teaching the infant emotional regulation. Learning how to regulate emotions into a homeostatic balance is one of the main functions of the hypothalamus. When the antithesis occurs, that is, when the child’s stress signals are ignored, the infant loses out on a learning opportunity. In addition, if the infant is consistently exposed to abuse, neglect, shame, intense stressful situations, and/or under-stimulation, such as in the case of children diagnosed with RAD, it neurologically decreases levels of endorphins and increases levels of adrenaline. According to Cozolino (2006), “this biochemical environment inhibits plasticity [the brain’s ability to adjust to neural changes throughout life] and creates a vulnerability to psychopathology” (p. 86).

The hypothalamus also governs the pituitary gland. This is a gland that affects distributing hormones to every major gland in the body (Ratey, 2006). This function is particularly important when a person confronts danger. When a person is frightened, the brain’s decision-making process is circumvented and it responds with an immediate reaction to the situation. Neurochemicals are produced which increases anxiety, vigilance, and adrenaline. This neurochemical production leads to an increase in the hormone cortisol, a hormone which is typically stress induced (Ratey, 2006). These physiological changes are more commonly known as the fight/flight/freeze response to danger. There is a specific circuitry sequence that causes the fight/flight/freeze response which is called the Hypothalamic-Pituitary-Adrenal Axis (HPA).
Hypothalamic-Pituitary-Adrenal Axis (HPA)

The HPA axis is the main circuitry that sets the fight/flight/freeze into action. When a person is confronted with a situation, whether it is real or imagined, the amygdala determines whether or not the person is in danger. The amygdala sends a signal to the hypothalamus. Once the hypothalamus is alerted by the amygdala, it immediately releases two peptides (two or more amino acids linked together): “corticotrophin-releasing factor (CRF) and arginine vasopressin (AVP)” (Cozolino, 2006. p. 222). Once released, the peptides CRF and AVP find their way to the pituitary gland. The pituitary gland’s response to the peptides is to release the hormone adrenocorticotropic (ACTH) into the bloodstream. When the ACTH reaches the adrenal glands, then the hormones cortisol, epinephrine, and norepinephrine are produced and distributed throughout the body. It is this last stage of the HPA axis that places a person in a fight/flight/freeze mode (Cozolino, 2006. p. 224).
According to Howe, et al. (2008), trauma can cause long-term dysregulation of stress hormones, such as cortisol. This long-term dysregulation may cause “lasting effects of trauma on the brain” (p. 21). Several early childhood studies were evaluated by Howe’s team. Their conclusion is that “these studies suggest that early abuse is associated with long-term changes in the HPA axis” (p. 25). Howe, et al. further report that “trauma-induced stress hormone dysregulation could lead to memory problems during childhood” (p. 146). They continue to say
that a significant amount of empirical research points to confirming that “stress-related hippocampal damage accounts for the autobiographical memory problems observed in traumatized individuals” (p. 147). According to Schore (2003a), when abuse and neglect occur in early childhood, the experience is “imprinted into amygdalar-hypothalmic-limbic-autonomic circuits” (p.257). The result of this imprinting is that when the child is an adult, even a low-stress situation can trigger an intense emotion and acting out. Schore reports that it is believed by many that early trauma experiences are remembered physiologically in adulthood (Schore, 2003 a).

**Memory**

Memory is a function of the brain that, in essence, allows us to know who we are. Like nerves that carry impulses from the body toward the brain or spinal cord, memory “pulls together learning, understanding, and consciousness (Ratey, 2002. p. 185). Many neuroscientists have set out to pinpoint exactly the part of the brain that stores and controls memory. The truth is, so far, our modern technology has not given us a specific answer. Instead, it appears that memory functions are spread throughout the brain utilizing many different parts to form one memory. To complicate matters further, memory recall can be influenced by a person’s mood at the time of an event. There are times that just the surroundings where the event occurred may trigger past memories. Significant to the complexity of memory is that the same event can be remembered differently by two separate individuals. As a person lives through different life cycles and gathers new life experiences, the memory can take on a new meaning. In essence, over time memories can be altered or distorted. (Ratey, 2002).

The formation of memories occurs in three stages: encoding, consolidation, and retrieval. Encoding begins in the amygdala where the memory is first experienced and cognitively
received. Consolidation which occurs in the hippocampus is when a recent memory transforms to long-term memory. The hippocampus also “assembles bits of information into explicit memories (Badenoch, 2008. p. 16). Retrieval occurs after a significant timeframe when a long-term memory is recalled (Howe, et al., 2008; Schacter, 1996). According to Badenoch (2008), it is in the hippocampus where retrieval of past memories occurs. The hypothalamus’ function is to maintain a homeostatic state by its releasing of neurotransmitters and hormones throughout the body and brain (Badenoch, 2008).

Memories are also categorized by two different types: explicit and implicit (Howe, et al., 2008). Explicit memory develops in a toddler somewhere between the ages of twelve to eighteen months. An explicit memory is where cognitive facts can be recalled. For instance, a child taking a test in history will have to recall recently learned and memorized historical facts. The first sign of explicit memory is expressed as factual memory. When a toddler can recall environmental events that they have witnessed, they are using the form of explicit memory considered to be factual memory. Within the second year of development the child will begin to integrate explicit memory by understanding and telling facts of their own story. This form of explicit memory is referred to as autobiographical memory. This latter form of explicit memory is important to understand when working with children diagnosed with reactive attachment disorder because the parental or primary caretaker’s reflection and interaction is vital to the child’s ability to integrate events into personal meaning (Badenoch, 2008).

An implicit memory is more subtle. Typically an implicit memory is one of which we are not consciously aware. A good example may be how a person drives a car. While navigating through traffic, the driver may see that the traffic ahead has stopped. The driver will automatically, without conscious thought, place a foot on the brake to slow down and come to a
At the same time this driver could be engaged in a conversation with a passenger and not notice that their foot depressed the brake to come to a stop. Much of our time throughout the day is spent performing implicit memory such as brushing our teeth, or chopping onions, or riding a bike.

Furthermore, implicit memory is unconscious. “These memories contain elements of behavioral impulses, affective experience, perceptions, sensations, and images” (Seigal as cited in Badenoch, 2008, p. 24). When the amygdala processes this unconscious information, it sets a lifetime tone of how an individual views the world. “Our earliest experiences become the basis of our foundational themes” (Badenoch, 2008, p. 25). Keeping this concept in mind when treating children diagnosed with reactive attachment disorder will be central to a clinical social worker’s treatment. Implicit memory has no timeframe. When a person has an “emotional/visceral/perceptual/behavioral” (Badenoch, 2008) response to a present moment in time, the implicit memory of the past is triggered. The result of having the implicit memory triggered is that the person experiences the present moment with the same emotional intensity of their early childhood experience. This implicit memory never goes away, but through clinical treatment, a person can learn to integrate their past experience with their present. Significant to children with reactive attachment disorder is that “the greater the early wounding, the more power these implicit memories exert in the present” (Badenoch, 2008, p. 26). Having such intense reactions to present moments that are implicitly connected to early childhood experience, sheds a new light on why RAD children have such difficulty with affect regulation.

When a child during the first years of life is exposed to abuse and neglect, the child’s memories can be affected by the child’s overall mood. The mood state has a direct affect upon the memory. According to Ratey, “one’s emotional state at a given instant affects how the
amygdala processes the emotional tag of the memory (p. 186). For instance, a depressed child will remember the event through a negative lens. The child’s memory of a particular event may be very different from a happy child witnessing the same moment.

Most significant to clinical social work treatment with children diagnosed with reactive attachment disorder is how the brain develops in early childhood. According to many, “children do not develop the capacity for long-term autobiographical memory until 2 to 3 years of age” (Howe, et al., 2008, p. 12; Bruce et al, 2005; Eacott & Crowley, 1998; Howe & Courage, 1993, 1997; Usher & Nesser, 1993). A child may have implicit memory prior to age two, but “explicit memories are not retrieved before this time period in later life” (Eacott & Crowley, 1998; Howe, et al., 2008 p. 12).

**Intersection of Freud’s Trauma Theory and Neurobiology**

Children who are diagnosed with reactive attachment disorder typically have early childhood trauma such as abuse, neglect, or under-stimulation which occurs before the age of five. Poor parenting and/or caretaking many times may cause the child to have an insecure attachment. The child may exhibit either inhibited or disinhibited behaviors. This insecure attachment affects the child throughout adulthood and places them at high risk for psychopathology. For instance, a child may have somatic symptoms that cannot be biologically explained. Or, they may present with Oppositional Defiant Disorder which may be driven by repressed and dissociated implicit memory. Viewing the child’s symptoms from a Freud Trauma lens helps the clinician to delve deeper into the child’s psyche as opposed to focusing on surface symptom reduction. Providing a trusting therapist/child dyad will facilitate a secure attachment. The plasticity of the brain will respond to the secure attachment thereby healing the early childhood maltreatment.
The basic foundation of Freud’s trauma theory is that self-knowledge improves health. Freud hypothesized that traumatic experiences were too painful to keep in the conscious mind; therefore, the memory went underground into the unconscious mind. Those repressed and dissociated memories expressed themselves somatically. He believed that the somatic expression was symbolic. His treatment goals were to encourage the patient to uncover the buried traumatic memory and bring them forward to the conscious mind through the use of an affective “talking cure.” By doing so, strangulated emotions could be released which would relieve the biologically unexplained somatic symptoms.

Recent neurobiological studies using MRI (Magnetic Resonance Imagry) and PET (Positron Emission Tomography) scans scientifically reveal the ways with which trauma is processed throughout the brain. Many neuroscientists concentrate on the limbic system and hypothalamus-pituitary-adrenal axis when studying patients with trauma histories. They have shown through empirical means how traumatic memories, particularly those of early childhood, have caused damage to the brain. On a positive note research, has also shown that our brain plasticity, along with clinical treatment, can reverse previous damage.

**Freud’s Transition from Neurologist to Psychoanalyst**

Determining which of Freud’s theories are supported by neurobiology begins with an understanding of Freud’s medical training and experience as a neurologist. Not only was Freud a young student during this medical phase of his professional development, but the development of neurology was at its inception also. During Freud’s neuropysiologic practice he was interested in substantiating and documenting his clinical theoretical hypotheses with neuroanatomy. Unfortunately, Freud was so far ahead of his time that the technology available to him was too archaic to prove his theory (Fullinwider, 1983). Now in the 21st century we have technology that
appears to support Freud’s clinical conjectures, but first let us examine Freud’s neurological development which ultimately lead him to his formulation of trauma theory.

Through historical documentation we know that modern neurology approximately began around 1848 with the famous case study of Phineas Gage. The story of Phineas Gage is an interesting one. As a young man in his twenties, he worked as an explosive expert for the railroad. While working in Vermont an accident occurred that caused Mr. Gage’s tapping rod to shoot straight through his head in the area of his frontal lobe landing about 25 to 30 yards behind him. When examined by a physician it was found that Mr. Gage’s physical health was not compromised in any way. However, it was noted that Mr. Gage’s personality was altered with erratic behavior (MacMillan, 2000). He lived for twelve years after the accident. In the later years he had seizures which ultimately caused his death.

This unusual medical incident initiated the late 19th. and early 20th centuries’. immersion into a new discipline known as neurology. The study of the Case of Phineas Gage essentially set forth a focus on cerebral localization; a neurological method used to pinpoint areas of the brain by analyzing specific behaviors. A good example of cerebral localization is Wernicke’s discovery. Wernicke pinpointed the area of the brain that controlled written and spoken language and to this day is simply referred to as Wernicke’s area. It was during this timeframe that Freud began his studies with major contributors in the medical discipline of neurology (Solms, 2000).

According to Solms (2000), in concert with cerebral localization, neurologists also used a method referred to as clinico-anatomical correlation. This method consisted of observing and recording patient’s behaviors. In order for the neurologists to prove their findings, at the time of their patient’s death autopsies were used to determine the location of the patient’s brain lesion.
The location of the brain lesion was then matched with the observed behaviors. Some patients however, did not have brain lesions. These were typically patients diagnosed with hysteria, the population that interested Freud the most (Solms, 2000).

Solms (1986; 2000) also reported that during the time when Freud was a young medical student, two major neurological schools of thought existed; that is the Austro-German and the French. Freud’s medical education as a neurologist began in Vienna in 1873. He studied under Theodor Meynert and others who held to the tenets of the German school. This school emphasized the importance of neurophysiology and cerebral localization. It was during these years that Freud gained a solid foundation and understanding of the anatomy and functions of the brain (Solms, 2000).

In 1885, Freud continued his education in France with Charcot at the Hôpital du Salpetrière in Paris with patients diagnosed with hysteria. Though both schools were interested in brain anatomy and brain functions, their emphasis of study was different. The German school was most interested in finding the exact part of the brain which governed a particular function. The French on the other hand, were more interested in observing the patient from a clinical point of view. Rather than trying to explain a function of the brain by associating it to a specific cerebral location, Charcot and his students were more interested in observing the patient’s behavior to “identify, classify, and describe them” (Solms, 2000. p. 183). It was during this educational experience that Freud began to approach his clinical work differently (Solms, 2000).

From a researcher’s and neurophysiologist’s perspective, Freud attempted to create an empirical study to support his observations in a document entitled Project for Scientific Psychology (Slipp, 2000). Though much time was dedicated to the Project Freud was not happy with the results and in the end Freud rejected his work. According to Solms (2000), Freud came
to the conclusion that his focus should use clinical observation as opposed to giving strict attention to neuroanatomy.

In an earlier article written by Solms (1986), he reports that a more important and later Freudian work was *On Aphasia*. It was in this work that Freud declared the rejection of cerebral localization and Meynert’s concept of the central nervous system. According to Fullenwider (1983) Freud was influenced by the English neurologist John Hughlings Jackson and his concept that the mind has hierarchal layering. Schore (2003b) acknowledges that Jackson “is unknown to most psychoanalysts (and many neuroscientists)” (p. 208). Though Jackson is little known, his contribution to the understanding of the brain was most influential in developing Freud’s topographic theory that is the conscious, sub-conscious, and unconscious. Jackson’s influence resulted in Freud’s rejection of cerebral localization and his acceptance of the mind having different levels of consciousness. Fullenwider states that “…we have Freud’s 1891 assertion in *On Aphasia* that the Meynertian theoretical scheme was inadequate to handle newly discovered facts about the structure of the central nervous system, and that he was turning to Jackson’s level theory in support of his theoretical departures from Meynert” (p. 151). Jackson’s level theory introduced the concept of hierarchies of the mind. He theorized that speech was in the highest level. According to Fullenwider, Jackson’s theory was mainly influenced by an English philosopher, Herbert Spencer, who supported Darwin’s theory of evolution and introduced the long time catchy phrase of “survival of the fittest.” Fullenwider explains Spencer’s influence upon Jackson:

> Since 1855 Herbert Spencer had been describing a central nervous system featuring integrative levels, the lower levels corresponding to more primitive functions. Man with his higher levels, was said to be able to cognize, thus manifesting a much more refined adjustment to his world than the fight-flight functioning of animals (1983, p. 152).
Fullenwider continues to state:

Jackson’s highest level, the propositional level, superseded lower levels of functioning. It was the highest level of consciousness which by virtue of its functional supremacy renders the lower levels unconscious but not inactive (1983, p. 153).

Schore (2003b) clearly describes Jackson’s pre-verbal lower-level functions as the “earliest stage of cognition” and Jackson believed it to be “closely tied to visceral functions” (p. 209). Schore emphasizes that the “problem of emotion can not be understood with a localizationist perspective, one that still dominates neuroscience” (p. 210). This stratified and integrated concept of the mind rang true to Freud and was one of the key tenets that eventually developed his topographic theory. This departure from Meynert gave Freud his first considerations regarding the idea of a higher level and a lower level of brain functioning. As Freud delved into his study, On Aphasia, he came to embrace Jackson’s findings and theories (Fullenwider, 1983).

While working with patients diagnosed with hysteria, according to Fullenwider (1983) Freud came to realize that active ideas not only lay within the conscious mind but also in the unconscious mind. He integrated his work in On Aphasia with patients who had experienced trauma. Fullenwider (1983) continues to say that Freud “argued that the painful memory is driven out of the consciousness [and] that the ego must exert energy to keep the memory unconscious” (p. 154). Freud also theorized that the patient’s somatic symptoms came from the unconscious and noted that the unconscious memory essentially governed the behavior of the hysterical patient (Fullenwider, 1983). It was this basic concept of the conscious and unconscious that spurred Freud into his trauma theory.

Another of Freud’s work with patients diagnosed with hysteria was his collaboration with Breuer, Studies on Hysteria. Breuer was a devoted student of Meynert and stayed true to Meynert’s teachings while writing Studies on Hysteria. Freud, on the other hand, had already
rejected Meynert’s theories but despite their differences acquiesced to Breuer in this body of work (Fullenwider, 1983). Breuer & Freud did agree, however, that patients with traumatic backgrounds mainly suffer from unconscious memories (van der Kolk, 1996).

**Neurobiological Confirmation of Freud’s Trauma Theory**

There are several specific aspects of Freud’s trauma theory that have been corroborated with empirical neurobiological means. According to Slipp (2000) through scientific evidence there is confidence in the concept of consciousness and unconsciousness. Freud also hypothesized that early childhood emotional memories affect our rational thought throughout life, that is emotional memories in our unconscious induces our conscious behaviors (Slipp, 2000).

Freud also spoke of a primary and secondary process regarding storing memory. According to Slipp (2000), the primary process involves the amygdala and right hemisphere which regulates our implicit memory. Secondary process, or as neuroscientists refer, explicit memory, are governed by the hippocampus and the left hemisphere (Slipp, 2000). However, Schore (2003b) makes a point that cerebral localization does not encompass the inter-relationship between the right and left hemisphere of the brain and the brain function is much more complex than being able to pinpoint specific areas of the brain to explain human behavior.

Slipp points out that Freud’s hypothesis of repetition compulsion has been noted with studies of the amygdala and right brain hemisphere. The enactment of transference is an expression of unconscious relational memory (now referred to as nonconscious by neuroscientists) which is then expressed through emotion and behavior. A further discussion of enactments will follow (Slipp 2000).
Freud’s concept of countertransference has also been researched. Many neuroscientists believe that the relational communication between the patient and therapist takes place between both person’s amygdala and right brain hemisphere. The therapist experiences the countertransference as an emotion through the defense mechanism of projective identification. Projective identification is a psychological process whereby a person projects a thought or belief onto a second person. When this occurs in therapy, it is the clinician’s responsibility to verbalize the patient’s unconscious affect (Slipp, 2000).

**The Conscious and the Unconscious**

Today, neuroscience substantiates Freud’s concept of the conscious and unconscious. Much research on the left and right hemisphere’s of the brain are aligned with the left being the conscious and more cognizant side, and the right being more of an instinctual affective side (Schore, 2003b). According to Slipp, (2000) “research confirms Freud’s monumental discovery that unconscious emotions strongly affect conscious thought” (p. 192). Slipp continues with:

…empirical neurobiological research validates Freud’s discovery that traumatic memories are stored in the unconscious. When perceptions threaten survival, they bypass consciousness and directly go into unconscious implicit memory. Freud’s hypothesis that unpleasant perceptions are repressed from consciousness into the unconsciousness seems to be accurate (p.194).

Neuroscience uses the terminology of explicit and implicit memory as opposed to conscious and unconscious states of mind. Explicit memory, similar to Freud’s concept of consciousness, is our cognition; that is our awareness of the world around us. It is based in our factual memory such as our autobiographical memory. Implicit memory which is likened to Freud’s unconsciousness is more subtle such as memories that cannot be verbalized but instead are outwardly expressed. van der Kolk (2000) describes memory as a function which does not necessarily remember specific events and facts, but is more prone to remember the memory as an
affective experience. Also according to van der Kolk (2000), “most memory processing occurs outside of conscious awareness” (p. 245). He further reports that due to high levels of adrenaline traumatic events avert linguistic encoding and the memories are stored differently than ordinary memories. These neurological findings support Freud’s theory that painful traumatic memories are held in the unconscious which then drives the conscious mind. For instance, van der Kolk & Fisler (1995) determined that memories of child abuse victims “report[ed] precise images, smells, and physical sensations that they claim[ed] are as clear as they were years or decades before, when the original trauma occurred” (p. 245).

**Transference and Countertransference**

It is today’s neurobiological understanding of the right brain hemisphere that supports Jackson and Freud’s concept of mind stratification. Schore (2003b) states that “an explosion of studies has confirmed the essential role of the right hemisphere in the processing of emotional information, validating Jackson’s insights’ (p. 210). According to Cozolino (2006), “the experience of the personal emotional self…appears to be primarily organized in the right [brain] hemisphere” (p. 67). Cozolino likens the function of the right brain to Freud’s conception of the unconscious. Past experiences that are stored in our unconscious propel our thoughts and behaviors throughout our life. Particularly when a person is stressed, their earlier experiences can be reenacted in later relationships (Cozolino, 2006).

Both adults and children have a strong need to reenact their past trauma. When Freud was studying in Paris, his trauma theory was significantly influenced by Charcot and Janet’s work with hysterical patients. Freud embraced Janet’s hypotheses regarding reenactment. van der Kolk’s contribution in Solomon & Siegel’s (2003) *Healing Trauma*, illustrates Janet’s influential theory thusly, “He [Janet] described how the memories of these traumas tended to
return, not as stories of what happened, but as re-enactments in the form of intense emotional reactions, aggressive behavior, physical pain, and bodily states that could all be understood as the return of elements of the traumatic experience” (p. 174).

According to Judith Herman (1992), traumatic memories are absent of verbal narrative, experienced as bodily sensations, and recalled with imagery. The reenactment may be in many different forms such as dreams, fantasy, and particularly in children’s repetitive play. Often repetitive play is observed when treating children with reactive attachment disorder. As clinicians working with children diagnosed with reactive attachment disorder, understanding that their past trauma is in a sense fixated, or stuck in time, has important implications. In *Studies in Hysteria* Breuer & Freud described fixation thusly: “…the traumatic experience is constantly forcing itself upon the patient and this is proof of the strength of that experience: the patient is, as one might say, fixated upon the trauma” (Breuer & Freud (1893-1895; 1955 as cited by van der Kolk 1996; p. 54). Freud referred to this repetitive need as “repetition compulsion.” Herman (1992) describes the need for reenactment in these terms, “there is something uncanny about reenactments. Even when they are consciously chosen they have a feeling of involuntariness” (p. 41). Freud observed that the unconscious traumatic memory strongly resists change which at times may leave the clinician feeling frustrated. According to Herman (1992) Freud’s frustration with the observation of “repetition compulsion” lead him to the concept of the “death instinct” an impulse for decay or death. Clinicians today do not particularly adopt Freud’s “death instinct” instead they have many different explanations for “repetition compulsion.” In general clinicians working in the 21st century believe that “repetition compulsion” is a patient’s need to master the past traumatic experience (Herman, 1992).
Dissociation and Repression

The two terms, dissociation and repression, are sometimes used interchangeably which, for some, may cause confusion. Some psychoanalysts today using an ego psychology framework define the terms thusly:

1) Dissociation is considered an immature defense mechanism. Many times survivors of incest and adults who experienced childhood abuse rely upon this defense. When a painful memory or idea is brought to consciousness, the patient will protect him/herself by separating, or splitting the memory from the emotion. By doing so, the impact of the painful event is altered (Berzoff et al., 2007); and

2) Repression is considered a neurotic defense mechanism. When a person completely forgets any thoughts, memories, or feelings, it is said to be repressed into the persons unconscious and they are no longer a part of the person’s conscious mind. This defense mechanism simply protects the client from unbearable desires, fears, or disappointments (Berzoff et al., 2007).

Throughout history, these exchangeable terms have been used in psychoanalytic literature. For the purposes of this thesis a focus on Pierre Janet’s concept of dissociation was examined. Freud adopted Janet’s definition while working with patients diagnosed with hysteria. However as Freud’s work advanced and his theory changed, so did his understanding of how painful memories were processed in the unconscious. This new understanding lead him into his theory of repressed wishes and desires. Neurobiological research supports the original understanding of Janet’s hypothesis. According to Huopainen (2002) repression is the term most referred to in both scientific and clinical literature, however the term dissociation is “most supported by [neurobiological] research” (p.102).
van der Kolk (2000) explains Janet’s description of dissociation:

Janet proposed that, when subject to “vehement emotions” people’s integrative capacities may become overwhelmed, resulting in abnormal memory processing. When this occurs, memories of a traumatic event may become split off (dissociated) from ordinary consciousness and from voluntary control and stored as “unconscious fixed ideas.” After having escaped from conscious control, sensory and emotional elements of the traumatic experience may continue to intrude as terrifying perceptions, obsessional preoccupations, and as somatic re-experiences such as anxiety reactions (Janet, 1889 as cited in van der Kolk, 2000, p. 238).

van der Kolk continues to define Janet’s hypothesis by describing how the patients experience the traumatic memory. Janet believed that when his patients dissociated, they actually became attached to the trauma, in essence became developmentally stuck and incapable of having new experiences. It was Janet’s belief that integration of the memory into the patient’s consciousness was necessary in order for the patient to successfully function (van der Kolk, 2000). Janet also postulated that when the patient has no conscious memory of the trauma that the loss of memory “prevents the integration of traumatic events and splits off the traumatic memories from ordinary consciousness” (van der Kolk, 1996, p. 285). In van der Kolk’s et al. 1996 book Traumatic Stress, the Effects of Overwhelming Experience on Mind, Body, and Society, he cites a lecture given by Freud in 1893. Freud at this point agrees with Janet and describes dissociation as “a splitting of the content of consciousness” (p. 54). Freud believed that when a patient diagnosed with hysteria had a recurring hysterical attack that the affect experienced was the same as the feelings when the original trauma occurred (van der Kolk, 1996).

According to Howe, et al. (2008), “understanding how stress affects memory and the brain will have important implications for the treatment of traumatized children throughout their lives” (p. 35). In the late 19th century, Freud theorized many trauma concepts while working with hysterical patients. Now, in the 21st century, we have an opportunity to apply empirical
data to Freud’s hypotheses. By examining Freud’s theory with breakthrough neurobiological research, clinical social workers treating children diagnosed with reactive attachment disorder are able to solidly substantiate their clinical work. During the recent decades, psychoanalysis has been dismissed with cognitive behavioral theories and adaptations. By substantiating Freud’s hypotheses, we may be able to place the “baby back into the bath water” so to speak. An integration of psychoanalytic theory and science will give the clinical social worker firmer ground to administer treatment to children with reactive attachment disorder. The next chapter provides an overview of currently used RAD treatments.
CHAPTER FIVE
IMPLICATIONS FOR TREATMENT OF REACTIVE ATTACHMENT DISORDER

This chapter integrates the three previous chapters, Reactive Attachment Disorder, Freud’s Trauma Theory, and Neurobiological Research of the Social Brain and its Limbic System and how its findings support Freud’s Trauma Theory while investigating currently available treatment modalities. With a comprehensive understanding of reactive attachment disorder, a fresh look at Freud’s trauma theory, and the discovery of recent neurobiological findings this thesis will conclude that the most effective treatment for children diagnosed with reactive attachment disorder is indeed psychoanalytic treatment.

Children who have been diagnosed with APA DSM-IV TR 313.89 Reactive Attachment Disorder of Infancy or Early Childhood (RAD) with a specified sub-type either inhibited or disinhibited will present with many challenges. Many have been misdiagnosed or given a comorbid diagnosis of attention-deficit disorders and/or disruptive behavior disorders such as 314.00 Attention-Deficit/Hyperactivity Disorder; 312.00 Conduct Disorder; 313.81 Oppositional Defiant Disorder; 312.9 Disruptive Behavior Disorder. In addition to these disorders, many children challenged with RAD also present with depression, anxiety, and obsessive-compulsive disorders. Symptoms of 309.81 Posttraumatic Stress Disorder may also be present.

Given the complexity of RAD, what is the most effective treatment? Fosha (2003) is aware of the dichotomy of how trauma and treatment affect the brain. On one hand, neurobiology has documented that the right hemisphere of the brain, the limbic system, and the prefrontal lobe control affect and self regulation. It has also been scientifically determined that
trauma and disorganized attachment can cause damage to the right hemisphere limbic system of the brain. Fosha, however, states that with “therapeutic interventions [that] involve emotion, the body, somatosensory activation, and bilateral information-processing mechanisms are effective in functionally reversing the effects of trauma” (p. 222). Despite Fosha’s declared optimism, research has supported the psychoanalytic hypotheses that maltreatment during the caregiver/infant dyad experience can place the child at high risk for a lifetime of psychopathology. Then again in contrast, neurobiological research has also documented that the plasticity of the brain lends itself to healing of past damage by introducing just one secure, committed, and consistent relationship.

According to Drisko & Zilberstein (2008), there are two major treatments currently used with children diagnosed with RAD; relationship-based attachment therapy and holding therapy. Relationship-based therapy is a psychoanalytic therapy where the client’s growth is due to the therapist/client interpersonal relationship. Holding therapy is broadly defined as a confrontational therapy. The child is held (contained) by parents or professionals to “break” the rage and anger they feel within. Reports of intense expression have been documented such as witnessing the child scream, sob, and yell while being restrained (Drisko & Zilberstein, 2008.). The holding therapy’s basic tenet is that while giving the child permission to release their anger and rage they will breakthrough their resistance and begin to trust and love. Within the therapeutic profession it is a very controversial treatment. Some professionals view holding therapy as an extreme, unnecessary, and coercive measure.

In addition to these relationship-based attachment therapies and holding therapy, according to Howe, Goodman, & Cicchetti (2008), symptom reduction therapy is also an alternative therapeutic intervention when working with children diagnosed with RAD (Howe, et
however, according to Howe, et al. (2008) prior to determining the type of therapeutic treatment, the clinician’s first consideration must be to determine whether or not maltreatment of the child is still continuing. Many times a caregiver may be in denial regarding their own abusive behavior toward the child and their maltreatment may be minimized by the caregiver. If there is maltreatment in the home and it is not addressed, the therapist may be inadvertently supporting the caregiver’s denial. Howe, et al. (2008) clearly state that “It is critical that such denial of trauma not be perpetuated by therapists” (p. 378). The consequence of overlooking the caregiver’s maltreatment could result in the child’s “reluctance to share their abusive experiences” (p. 378) and that “emotional recovery from the trauma may be extremely difficult” (p. 378). While assessing whether or not maltreatment is ongoing, the therapist may be teetering on a slippery slope. The clinician must be cognizant of the child’s suggestibility. Careful attention must be given not to influence the child’s story in any way. This is particularly true with cases involved with criminal proceedings against an alleged perpetrator. Howe, et al. (2008) recommend that early in the treatment the clinician must listen empathically and create a safe environment where the child can freely tell their story and express any possible anxiety the child may be experiencing (Howe, et al., 2008).

Since the child’s recall of its traumatic history is essential in working toward recovery, it is important for a clinician to understand the impact of trauma upon the child’s memory. According to Howe, et al. (2008), this is an area where much more research is needed to determine appropriate treatment “interventions and policy decisions” (p. 370). Howe, et al. (2008), report that after considerable review of literature up to the year 2008, they have determined that memories of maltreated children as compared to non-victimized children appear to be the same. They conclude by summarizing several issues that need further examination.
when treating children with a history of abuse and neglect. Those issues are to accurately determine the child’s developmental maturity, to gauge the timing between the traumatic event and the child’s disclosure, to assess whether or not the primary caretaker is directly involved in the child’s maltreatment, and to decide the exact role of the therapist (Howe, et al., 2008).

Implicit memory is developed from birth to eighteen months. Research, so far, concludes that between birth and eighteen months the hippocampus is not yet fully developed. Therefore, explicit memory recall is not available to newborns up to the age of eighteen months. There are some professionals in the field that question whether or not infants of that age range can internalize trauma. This inquiry raises significant treatment considerations. Howe, et al., (2008) address this concern thusly:

…it becomes particularly challenging as to whether intervention should be directed toward helping a child ‘recall’ a traumatic event that occurred in the early years of life and that may have dissipated over time. If symptoms associated with the trauma are absent, then this question becomes more philosophical than practical, as it is unlikely that the child will be referred to treatment. However, if socioemotional or behavioral problems are present, then the form of intervention to be provided assumes importance (p. 371-372).

When we consider Freud’s trauma theory, reenactment plays an important role in healing. Many clinicians believe that telling the story through repetitive play is a child’s way to acquire mastery over their past traumatic experience. However, Howe, et al., (2008) disagree. Their recommendation for those children with early childhood trauma is to work on eliminating unwanted behaviors through symptom reduction. If this direct approach is ineffective, then the clinician can engage in more trauma-focused interventions. Howe, et al. (2008) place secondary importance upon telling the story with affect in order to process the emotions of the traumatic experience. In essence, Howe, et al. (2008) seem to be recommending Freud’s “talking cure” as a legitimate intervention but only after symptom reduction treatment has been declared.
ineffective. From a neurological perspective, Howe, et al. (2008) do give caution regarding children diagnosed with RAD to repeatedly tell their traumatic story. Their concern with repetitive reenactment is that the recall “may inadvertently result in the consolidation of maladaptive neural pathways. In other words, telling the story over and over again may actually cause damage to the brain as opposed to it being cathartic and healing” (Howe, et al., 2008, p. 373).

Due to the complexity of the RAD diagnosis and the probability of additional comorbid diagnoses as well, Drisko & Zilberstein (2008) caution clinicians and social service workers to treat each child individually by focusing upon their unique needs. In addition to the child’s individual needs being considered, it is important to assess and therapeutically support the relationship between the child and caregiver. Daniel Siegel, M.D. makes the distinction “that insecurity of attachment is not a characteristic of an individual, but rather of a relationship” (cited by Fosha, 2003.) Drisko & Zilberstein (2008) utilize a family-centered treatment that encourages co-creating a new secure attachment between the caregiver and the child. They claim that a child diagnosed with RAD must be placed in a “solid consistent adoptive family placement” (p. 485) with the understanding that the caregiver’s commitment to the child may become intense, difficult, and prolonged. Drisko and Zilberstein (2008) suggest that parent-counseling, caregiver/child therapy, and individual child therapy may be applied in concert with one another. When considering an adoptive family for the child diagnosed with RAD, the most salient elements are that the child feel safe and psychologically supported (Drisko & Zilberstein, 2008).

Drisko & Zilberstein (2008) designed a small qualitative study with nine children diagnosed with reactive attachment disorder. Overall, the findings of their research emphasized
the following effective interventions: 1) providing a safe environment; 2) parental commitment and persistence; 3) parental recognition of the child’s strengths and small gains; 4) keeping a positive outlook; 5) consistent, almost constant parental availability; 6) daily home structure with the use of behavior management techniques; 7) intersubjective attunement (specifically described in the Modern Attachment Theory section below); 9) surrounding parents with professional advocacy and social supports; and 10) parental use of effective and accepted physical contact to contain the child (Drisko & Zilberstein, 2008). Part of the study’s testing was to evaluate the effectiveness of holding therapy. Parents who participated in this study chose not to use the coercive and confrontational therapy as they found no need for such extreme measures.

Another suggested treatment is dyadic developmental psychotherapy (DDP). This treatment is another example of relationship-based attachment theory. According to Becker-Weidman (2006), DDP “facilitates the development of a healthy attachment between child and caregiver, enables the child to actively trust the caregiver, and allows the child to secure comfort and safety from the caregiver” (Becker-Weidman, 2006. p.169). Becker-Weidman conducted a study with sixty-four children whose backgrounds included early childhood maltreatment and who met the DSM-IV TR criteria for 313.89 reactive attachment disorder. The children during the time of treatment were between the ages of five and sixteen. All of the children were either in foster care or adopted. There were thirty-four patients in a treatment group and thirty patients in the control group. All of the sixty-four patients were treated in an outpatient clinic. (Becker-Weidman, 2006).

The treatment group conducted two-hour weekly sessions with both the child and the parent, twenty-three sessions over a period of eleven weeks. Parents were administered psychoeducation regarding healthy attachment (attachment theory). The treatment was both child and
family focused. The emphasis was in developing attunement between the parent/child and therapist/child. Treatment centered upon non-verbal work since the trauma was experienced during the pre-verbal stage of development. This pre-verbal stage is often dissociated from explicit memory. Eye contact, tone of voice, and touch were important components to the therapeutic endeavor. The work revolves around the acronym PACE which stands for playful, acceptance, curious, and empathic. By using psychodrama re-enactments, the child is taught to integrate past and present feelings of the traumatic experience (Becker-Weidman, 2006).

The control group made up of thirty patients engaged in traditional treatments such as individual therapy, play therapy, family therapy, residential treatment, and intensive outpatient treatment. The data collected from both the treatment group and the control group resulted in supporting DDP as a more effective treatment method (Becker-Weidman, 2006).

Another proposed treatment is called modern regulation theory, also referred to as modern attachment theory. This theory joins neurobiology and psychoanalysis together from the perspective of attachment theory. According to Schore & Schore (2008) affect and self regulation stem from the early primary caregiver/child “affective attachment communication” (p.9). This type of communication, affective attachment communication, occurs when the primary caregiver’s right hemisphere brain non-verbally and non-consciously connects with the infant’s right hemisphere brain. The linking of the caregiver’s right hemisphere brain to the infant’s right hemisphere brain is also referred to as empathic attunement (Badenoch, 2008). Communication between the caregiver and infant occur through eye contact, facial expressions, and vocal tone (Cozolino, 2006). Schore & Schore (2008) refer to the power of the right hemisphere communication as processing “the music behind the words” (p.14). It is through empathic attunement, also coined intersubjective attunement, that the caregiver teaches the infant
affect and self regulation. Empathic attunement is “critical to the development of structural right brain neurobiological systems involved in processing emotion, modulation of stress, [and] self-regulation” (Schore & Schore, 2008, p.10). Schore & Schore also inform us that the early dyadic relationship between caregiver and infant may affect the child in opposite ways. If the relationship contains empathic attunement then the child will most probably experience regulated affect and a positive sense of self therein having the fortune of a secure attachment. If the caregiver/child dyad contains inconsistency and neglect, then the child is at risk for dysregulation of affect and a poor sense of self most likely resulting in an insecure attachment (Schore & Schore, 2008). In the case of children diagnosed with RAD, the degree of neglect they experience places them at a very high risk of never experiencing empathic attunement from the primary caregiver. The lack of empathic attunement may then be a major contributor to the RAD child’s dysregulation of affect and self.

Can the lack of empathic attunement and inconsistent or neglectful caregiver/infant dyad be addressed in clinical treatment and successfully treated with children diagnosed with RAD? Schore (2006b) eloquently answers this question:

The attuned, intuitive clinician, from the first point of contact, is learning the non-verbal moment-to-moment rhythmic structures of the patient’s internal states, and is relatively flexible and fluidly modifying his/her own behavior to synchronize with that structure [affective attunement] thereby creating a context for the organization of the therapeutic alliance (p. 52).

Badenboch (2008) answers this question more directly, “Empathic attunement is the key factor that fosters this all important integrative step in children, and it is also part of the reason that solid therapy can rewire the brain so efficiently” (Badenboch, 2008, p. 28, italics in original).
Also, according to Schore (2003b), the empathically attuned therapist can repair the imprinting of insecure attachment and affect dysregulation (Schore, 2003b).

Schore & Schore (2008) describe affective attachment communication thusly: “psychotherapy is not the ‘talking’ cure but the ‘communicating’ cure” (Schore & Schore, 2008. p.14). Though this may be a catchy phrase, it is important to remember that Freud clearly stated that the “talking cure” must be done with affect in order to effect healing. However, in an earlier resource Schore does give credit to Freud’s belief that “psychotherapy is always concerned with affect” (Schore, 2003b. p.52) and that non-verbal moments during psychoanalysis are equally significant (Schore, 2003b).

Transference and countertransference continue to play a dominant role in modern attachment theory. According to Schore & Schore (2008) transference and countertransference are directly related to the “nonconscious nonverbal right brain-mind-body communications” (p. 15). Describing transference and countertransference in these terms implies that implicit memory and implicit perceptions underlie affective attachment communication. Schore & Schore (2008) also remind us that psychodynamic and psychoanalytic treatments rely heavily upon the “therapist’s unconscious right mind” (p. 16). It takes time to develop an attachment within the therapeutic alliance. Schore & Schore (2008) indicate that developing a secure attachment when a client has an insecure attachment takes even longer. Since many children diagnosed with reactive attachment disorder have an insecure attachment it would be reasonable to say that the therapeutic period necessary to promote healing with a RAD child would need a significant amount of therapeutic time. It will be important for the clinician to recognize the client’s original reflection of their early experience with their primary caretaker in order to co-create a new secure attachment (Schore & Schore, 2008). A considerable amount of time is also
necessary to effectively treat a child diagnosed with RAD since the healing takes place by administrating psychotherapeutic affective attachment communication as a treatment for healing the “right-hemispheric unconscious system” (p. 16).

**Conclusion**

The purpose of this thesis is to argue that psychoanalytic treatment for children diagnosed with reactive attachment disorder is the most effective therapeutic choice when working with maltreated children. To support this argument, four major sections were researched and analyzed: (1) a detailed examination of the reactive attachment disorder DSM criteria, from its onset in 1980, up to a proposal submitted for the 2013 DSM V publication; (2) a chronicle of Freud’s early medical training as a neurologist which lead him to the development of Freud’s trauma theory and psychoanalysis; (3) recent neurobiological findings that support many of Freud’s trauma hypotheses; and (4) an overview of current treatment modalities used with children diagnosed with reactive attachment disorder.

The exploration of reactive attachment disorder’s history and development revealed that there is significant confusion and disagreement amongst clinicians concerning the RAD definition and diagnostic criteria. The diagnosis has undergone several revisions since its introduction to the DSM III in 1980. Within the reactive attachment disorder portion of this paper, a detailed overview of Zeanah & Gleason’s (2010) DSM V proposal is set forth. Zeanah & Gleason (2010) have suggested considerable changes to the existing DSM IV TR criteria. The most noteworthy is their suggestion to eliminate the sub-types inhibited and disinhibited. In their place they are suggesting two separate diagnoses, Reactive Attachment Disorder of Infancy and Childhood and Disinhibited Social Engagement Disorder (Zeanah & Gleason, 2010). Until
professionals agree upon a universal definition of reactive attachment disorder, then the most effective treatment choice for maltreated children may continue to be debated.

The next chapter examines the roots of psychoanalysis by tracing Freud’s distinguished early medical journey. The reader is asked to concentrate exclusively upon Freud Trauma Theory and to consider a fresh look at those hypotheses he developed while training under Charcot and Janet at the Hôpital du Salpetrière in Paris. When Freud returned to Vienna, his disclosure to the medical community that child sexual abuse appeared to be a common factor of hysteria was vehemently rejected by his colleagues. Thereafter Freud’s psychoanalytic theories drastically changed. During the late 20th century, feminists took issue with his retraction that early childhood sexual abuse was at the root of hysteria. As a supporter of feminist theory, this writer chose to compartmentalize Freud’s professional development. By doing so, an objective, non-judgmental view of his early trauma work became possible. Freud’s hypotheses regarding the concept of the subconscious holding painful memory; expressing repressed memory through somatic symptoms; dissociating when triggered with unconscious memories; using the “talking cure” with affect and empathy as a treatment modality; and introducing the concept of transference and countertransference were nothing short of being brilliant. Freud’s foresight is not only revolutionary, but now in the 21st century, his hypotheses are being supported by empirical neurobiological research.

It is essential for 21st century clinicians to understand the neuroanatomy of the social brain and limbic system. In the neurobiology chapter the fight/flight/freeze response to trauma is described. The functions of the amygdala, hippocampus, and hypothalamus are discussed. An overview of the hypothalamus-pituitary-adrenal (HPA) axis describes how chronic early childhood trauma can directly effect the over-production of the hormones cortisol, epinephrine,
and noradrenaline. This hormonal over-production causes a dysregulation of the limbic system’s function. The result of this dysregulation may be anxiety, depression, and debilitating fear. In addition to the limbic system and the HPA axis, memory function, and childhood development of implicit and explicit memory is reviewed. Understanding that explicit memory does not begin until a child reaches eighteen months, is most significant to clinical work with children diagnosed with RAD, and it explains some behaviors that are rooted in the child’s implicit memory.

The last chapter of this thesis attempts to integrate RAD, Freud’s Trauma Theory, and Neurobiology in support of the argument that psychoanalytic treatment is the most effective treatment for children diagnosed with reactive attachment disorder. The literature clearly supports that early childhood primary caregiver/child experience plays a major role in the child’s lifetime mental health. Currently there are three common ways to approach RAD treatment: symptom reduction, holding therapy, and relationship-based attachment therapy. Concentrating upon symptom reduction of RAD symptoms may appear to be therapeutic on a surface level; but, is that good enough? The controversial treatment, Holding therapy, requires “forced” containment of the child; but, could that be re-traumatizing? Despite the pressure of managed care and measurable treatment goals, a child presenting with RAD symptoms needs time to develop a trusting and positive therapist/child dyad. The therapist/child dyad can re-create a healthy secure attachment which, according to recent neuroscience, allows the plasticity of the brain to heal damage caused by early childhood maltreatment. Integrating neurobiology and psychoanalytic theory to the treatment of reactive attachment disorder is crucial for effective and lasting treatment. It is in this psychoanalytic longer-term healing process that unwanted
behaviors will be extinguished. Therefore, psychoanalytic treatment with children diagnosed with reactive attachment disorder is the most effective treatment.
REFERENCES


Siegel, D. J. (1999). *The developing mind: How relationship and the brain interact to shape who we are*. NY: Guilford Press


