INSTITUTE FOR CLINICAL SOCIAL WORK

Summer 2010

Course: CL ??? , **Learning Disorders and Self-Deficits: Diagnostic and Treatment Considerations**

Instructor: *Joe Palombo*, email: jp@josephpalombo.com

Welcome!

PLEASE READ THE FOLLOWING CAREFULLY.

Attached are:

1. The description of the requirements and your assignment for the courses, and
2. The outline and bibliography for the course.

All reading materials and handouts will given to you on a CD in the first class to distribute to every student. I strongly suggest that you print out all the handouts, put them in a binder, and bring them to EVERY CLASS. We will be referring to them frequently.

To facilitate the integration of some of the complex and novel materials to which you will be exposed, I will be using PowerPoint presentations. You will have copies of the PowerPoint.

I have tried to limit the number of references, but in spite of that there are still too many for you to be able to read during the semester. I realize that the amount of time that you have to read is limited, but I decided to include the relevant references so that you may have them available to read later on, i.e., after you graduate! In the meantime, choose an area of interest and read that material in as much depth as you can.

If you have any question, you may e-mail me at: jp@josephpalombo.com

See you then!

C:\1Data\1Data Dev\007Current Inst Courses\Summer 2010\1 READ FIRST\01 Welcome 10 0515.wpd

INSTITUTE FOR CLINICAL SOCIAL WORK

Summer 2010

Course: CL , **Learning Disorders and Self-Deficits: Diagnostic and Treatment Considerations**

Instructor: *Joe Palombo*, email: jp@josephpalombo.com

The following cover the requirements for both courses:

1. At the beginning of each class, students will take turns summarizing the materials presented in the previous class. These presentations should not exceed 10 minutes. They are meant to remind students of what was covered and where we left off during the last class. Since we will have a total of 5 classes, depending on how many students are enrolled, that will mean that we will need 10 presenters, one for each half of the class. We will discuss the details of the implementation this plan during the first class.

2. Since this the format of this class is highly condensed, you may do **one of the following** to fulfill the written requirements for the course:

    a. Submit your class notes, clearly written out and properly formatted, ***with comments on what has helped you and what you found difficult about every class and a summary of at least TWO papers you have read related to the topic we discussed*** .
    b. Submit, by mid-term, a one page proposal of a topic on which you would like to write a 15 page paper on one of the disabilities we discussed, i.e., dyslexia, ADHD, Executive Function Disorders, Nonverbal Learning disabilities, or Asperger's Disorder. I will review the proposal and discuss it with you. This paper should not include case material, but should review the literature. You may append a case, if you wish.

Assignments are due one week after the last day of class on Wednesday, July 28, 2010. You may email th assignment.

C:\1Data\1Data Dev\007Current Inst Courses\Summer 2010\1 READ FIRST\02 Assingn10 0515.wpd

Institute for Clinical Social Work

Learning Disorders and Self-Deficits: Diagnostic and Treatment Considerations

Summer 2010

Instructor: Joe Palombo

The goal of this class is:

1. To familiarize students with the emerging knowledge in the neurosciences;
2. To delineate the major controversies this new paradigm raises; and
3. To discuss the relevance of this body of knowledge to the development of childhood psychopathology.

The objectives are:

a. To integrate into clinical social work the body of knowledge contributed by the neurosciences.
b. To review some learning disorders of the self that result from learning disabilities in children, adolescents, and adults with learning disorders.
c. To understand the impact of learning disorders on development.
d. To develop the skills to formulate the psychodynamics and a treatment plan for each case based on the type of specific learning disorder.

Course Outline & Bibliography

Texts: [I suggest you purchase one book with * as a basic text on brain function and one with ** as a basic text on clinical relevance]

**Applegate, J. S., & Shapiro, J. R. (2005). Neurobiology for Clinical Social Work: Theory and Practice. NY: W.W. Norton. [Deals primarily with the neurobiology of attachment]

*Bloom, F. E., Beal, M. F., & Kupfer, D. J. (Eds.). (2003). *The Dana Guide to Brain Health*. New York: The DANA Press. [Excellent guide, highly recommended]

**Cozolino, L. (2002). The neuroscience of psychotherapy: *Building and rebuilding the brain*. Second Edition. N.Y.: W. W. Norton. [An eclectic approach to psychodynamic theory]

Mayr, E. (2001). *What Evolution Is*. NY: Basic Books. [Elementary text on evolution, recommended reading]

*Palombo, J. (2001). *Learning disorders and disorders of the self in children and adolescents*. NY: W.W. Norton.
*Palombo, J. (2006). *Nonverbal learning disabilities: A clinical perspective*. NY: W. W. Norton.
PDM. (2006). *Psychodynamic Diagnostic Manual (PDM). A collaborative effort of the American Psychoanalytic Association, International Psychoanalytic Association, Division of Psychoanalysis (39) of the American Psychological Association, American Academy of Psychoanalysis and dynamic Psychiatry, and National Membership Committee on Psychoanalysis in Clinical Social Work.* Silver Springs, MD: Alliance of Psychoanalytic Organizations.
**Ratey, J. J. (2001). A user's guide to the brain: Perception, attention, in the for theaters of the brain. New York: Pantheon Books. [You can read selectively and get a great deal out of this book]
**Siegel, D. J. (1999). The developing mind: Toward a neurobiology of interpersonal experience. NY: Guilford Press. [Very popular, very readable, and informative book]
**Solms, M., & Turnbull, O. (2002). The brain and the inner world: An introduction to the neuroscience of subjective experience. NY: Other Press. [An update of Freud's project for a neurology for psychologists]

Course outline

Class I.

A. Introduction

1. PDM

PDM. (2006). *Psychodynamic Diagnostic Manual (PDM). A collaborative effort of the American Psychoanalytic Association, International Psychoanalytic Association, Division of Psychoanalysis (39) of the American Psychological Association, American Academy of Psychoanalysis and dynamic Psychiatry, and National Membership Committee on Psychoanalysis in Clinical Social Work.* Silver Springs, MD: Alliance of Psychoanalytic Organizations. **Pp. 173-318.**

a. Major Sections Part II: Classification of Child and Adolescent Mental Health Disorders:
   (1) Profile of Mental Functioning for C & A (MCA).
   (2) C & A Personality Patterns and Disorders (PCA Axis)
   (3) C & A Symptom Patterns: The Subjective Experience (SCA Axis)

b. Case Illustrations of the PDM Profile with C & A Mental Health Disorders. Pp. 297-318.
c. Consultation Report: see Palombo's Outline

2. The Emergence of a New Paradigm

a. Freud's project revisited
   (1) Revival of Freud's Project
   (2) Can psychoanalysis and neuroscience be integrated?
      (a) Two strategies have emerged for bridging neuroscience and psychoanalysis
         i) The Rosetta Stone strategy
         ii) The Neurodevelopmental Model

Palombo, J. (2001). *Learning disorders and disorders of the self in children and adolescents*. NY: W.W. Norton.

3. From Neuroscience to a Neuropsychodynamic Perspective

a. The agenda for a new paradigm: The making of minds–the sense of self
b. A theory of development: The neurodevelopmental perspective
c. A theory of psychopathology: Deficit model, innate factors contribute to dysfunctional states
d. A theory of treatment: Brain changes may result from talk therapy
e. Methodology:
   (1) Non-linear dynamic systems
   (2) Evolutionary perspective

4. A Neurodevelopmental Perspective of the sense of self

a. Neuropsychological systems and their functions
b. The relational components
   (1) Sociality & social communication
   (2) Emotional communication
   (3) The attachment system
c. The intrapersonal experience: Meaning of experience
   (1) The sense of self cohesion
      (a) Mindsharing
      (b) Emotional communication
   (2) Coherent self-narratives
      (a) Personal and shared meanings
      (b) Central coherence

Palombo, J. (1991). Neurocognitive Differences, Self Cohesion, And Incoherent Self Narratives. *Child & Adolescent Social Work Journal, 8*(6), 449-472.
Palombo, J. (1992b). Narratives, Self-Cohesion, And The Patient's Search For Meaning. *Clinical Social Work Journal, 20*(3), 249-270.
Palombo, J. (1993). Neurocognitive Deficits, Developmental Distortions, And Incoherent Narratives. *Psychoanalytic Inquiry, 13*(1), 85-102.
Palombo, J. (1994). Incoherent self-narratives and disorders of the self in children with learning disabilities. *Smith College Studies in Social Work, 64*(2), 129-152.

5. Psychopathology: Deficits and disconnections

a. Neuropsychological deficits:
   (1) Constraints on competence and functioning
   (2) Constraints on social relationships, affect integration, and attachments
   (3) Constraints on integration of the meaning of experiences
b. Relational deficits
   (1) Problems in sociability & social communication
   (2) Problems in emotional communication & processing
   (3) Insecure attachments
c. Self deficits
   (1) Selfobject function deficits
   (2) Deficits in self-cohesion
   (3) Incoherent self-narratives
d. Remediation & Treatment
   (1) Complementary functions:
   (2) Remediation of neuropsychological deficits:
      (a) provide adjunctive functions
      (b) O.T., Tutoring
   (3) Provide group therapy, social skills and support groups
   (4) Treatment of disorders of the self:
      (a) provide selfobject functions and
      (b) a coherent self-narrative

6. The making of minds: Of brains and selves

a. Neuropsychological systems and their functions

Eliot, L. (1999). *What's going on in there? How the brain and mind develop in the first five years of life.* N.Y. Bantam Book.
Gilkerson, L. (2001). Integrating an understanding of brain development into early chidhood education. *Infant Mental Health Journal, 22*(1-2), 174-187.
Pally, R. (1997a). I. How brain development is shaped by genetic and environmental factors. *International Journal of Psycho-Analysis, 78*, 587-593.

Siegel, D. J. (1999). *The developing mind: Toward a neurobiology of interpersonal experience*. NY: Guilford Press.

b. Theories of brain functions
(1) Localization theories
(2) Neural network theories
(3) The embodied mind

c. Brain structures
(1) Neuron, neuronal networks, neurotransmitters
(2) Hemispheric specialization and functions & dysfunctions associated with each lobe
(3) Functions and dysfunctions associated with the limbic system and the basal ganglia

Ratey, J. J. (2001). *A user's guide to the brain: Perception, attention, in the for theaters of the brain*. New York: Pantheon Books.
Harris, J. C. (1998). *Developmental Neuropsychiatry* (Vol. I). Oxford: Oxford University press.

d. Cerebral Cortex
(1) Brain Hemispheres
(2) Contralateral Representation
(3) Corpus Callosum
(4) Motor & somatosensory cortex

e. Brain Anatomy & Related Dysfunctions
(1) Left Hemisphere ----------Language disorders
(2) Right Hemisphere----------Nonverbal LD
(3) Lobes and associated disorders
  (a) Frontal----------ADHD, Executive Functions
  (b) Parietal----------Body in space, spatial orientation
  (c) Temporal----------Dyslexia, memory
  (d) Occipital----------Visual processing
(4) Limbic System----Anxiety disorders
(5) Basal Ganglia----------Motor movements, Tourette's

B. Psychopathology I. Left hemisphere dysfunctions

1. Language-based disorders

a. Receptive language

(1) Phonological level–dyslexia
(2) Morphological level–reading problems
(3) Syntactical level–grammar disorders
(4) Semantic level–comprehension disorders

b. Expressive language

(1) Speech problems (motor impairments)
(2) Level of Pragmatics (i.e., usage)

c. Central Auditory Processing Disorders
d. Other language-based disorders

2. Dyslexia:

a. Characteristics

(1) Defining Features: "Dyslexia is one of several distinct learning disabilities. It is a specific language-based disorder of constitutional origin characterized by difficulties in single word decoding, usually reflecting insufficient phonological processing abilities. These difficulties in single word decoding are often unexpected in relation to age and other cognitive and academic abilities: they are not the result of generalized developmental disability or sensory impairment. Dyslexia is manifested by variable difficulty with different forms of language, often including, in addition to problems in reading, a conspicuous problem with acquiring proficiency in writing and spelling." (Orton Dyslexia Society definition adopted by the National Institutes of Health, 1994).
(2) Prevalence rates: Estimates of children in the school population in the U.S. who have readinE difficulties range from 20 to 30%. The percentage varies among whites, African-Americans, and other ethnic groups. Only a subset of this group of children is identified as having dyslexia. The disorder may be present in a range from mild to severe and is thought to occur in approximately 3 to 6% of the school age population [Frost, 1995 #1820].
(3) Sex ratio: Incidence is the same in males as in females, although boys may be affected slightly more severely than girls (Lyon, 1990, p. 50).
(4) Coexisting conditions: At one time dyslexia was thought to be associated with a variety of somatic illnesses, such as allergies. But the association has not been supported by research.

b. Developmental History: Usually unremarkable as most milestones are achieved on time.

c. Disorders of the Self: Self-esteem problems are prevalent

(1) Presenting Symptoms:
(a) Academic: Problems first appear when children begin to read. They have difficulty identifying their letters. They have trouble recognizing and sounding out words. But all have good comprehension of spoken materials. Some appear to be reading, but in reality have memorized the texts of books read to them and have become quite clever at concealing their deficit. Prominent problems with spelling exist.

(b) Social: No social problems are associated with this learning disorder until the children confront situations in which they are required to complete reading tasks. Their embarrassment at not being able to do what other children do easily may interfere with peer relationships.
(c) Emotional: No single set of emotional problems is associated with this learning disability, although the children get embarrassed when asked to perform reading or written task. Their repeated embarrassment may eventually lead to self-esteem problems.

(2) Sense of self-cohesion: Sense of self-cohesion remains reasonably intact, although self-esteem problems are prevalent.
(3) Self-narrative coherence: The self-narrative does not adequately explain the symptoms of the dyslexia unless t e child has had that explained to him. Otherwise, the child remains puzzled as to why she cannot read

d. Interventions:

(1) Remediation: Systematic phonics instruction by a reading specialist.
(2) Psychotherapy: Elective or if indicated because of the secondary effects of the disorders.

McNulty, M. A. (2003). Dyslexia and the life course. *Journal of Learning Disabilities, 36*(4), 363-381.
Palombo, J. (2001). *Learning disorders and disorders of the self in children and adolescents*. NY: W.W. Norton. Ch. 7.
Shaywitz, B. A., Pugh, K. R., Fletcher, J. M., & Shaywitz, S. E. (2000). What cognitive and neurobiological studies have taught us about dyslexia. In L. L. Greenhill (Ed.), *Learning Disabilities: Implications for psychiatric treatment* (pp. 59-98). Washington, DC: American Psychiatric Press.

3. Other language-based disorders: Central Auditory Process Disorder

Ferre, J. M. (2002). Managing children's central auditory processing deficits in the real world: What teachers and parents want to know. *Seminars in Hearing, 23*(4), 319-326.

Class II. Psychopathology II: Frontal Lobe Dysfunctions

A. Frontal lobe functions

1. The **motor cortex,** which lies immediately anterior to the central sulcus, is involved in the control of voluntary movements.
2. Primary and secondary levels of motor control, verbal fluency and design fluency, and spelling.

3. The remainder of each frontal lobe is referred to as **prefrontal association cortex.** This region is involved in complex behaviors such as **perception, emotion, memory, language, and thinking**
4. The original form of psychosurgery, prefrontal lobotomy, involved disconnecting the frontal lobes from the rest of the brain to reduce constant agitation in mental patients and to relieve intractable pain.

B. Frontal lobes dysfunctions

1. Attention Deficit/Hyperactivity Disorder

a. Characteristics:

(1) Defining Features: According to DSM-IV, there are three components to AD/HD: inattention, impulsivity and hyperactivity. DSM-IV identifies three subtypes of AD/HD: combined type; predominantly inattentive type; and predominantly hyperactive-impulsive type.

(2) Prevalence Rates: The National Institutes of Health studies fmd that 3 to 5% of all children, perhaps as many as 2 million American children, suffer from the disorder (NIMH, NIH Publication #96-3572, 1994; NIH, 2000), Cantwell states that as "much as 50 % of the child psychiatric clinical populations" have attention deficit disorders (Cantwell, 1996, p. 978).

(3) Sex Ratio: The ratio of male to female ranges from 2:1 to 9:1 (Pennington, 1991, p. 84.) Barkley states "epidemiological studies fmd the proportion to be approximately 3:1 among nonreferred children displaying [these] symptoms" (Barkley, 1989, p. 44).

(4) Coexisting Conditions: Among those conditions found to coexist with AD/HD are: mood disorders, anxiety disorders, oppositionality and aggression, obsessive-compulsive disorder, executive dysfunction, and substance use disorder.

b. Developmental History: Most milestones are achieved on time. Some children's level of activity is noticed to be higher than average at a very early age. Most frequently, the child's hyperactivity manifests around the time he begins to walk..

c. Disorders of the Self: Self-esteem problems and problems with impulsivity and self-regulation predominate.

(1) Presenting Symptoms:

(2) Academic: Academic performance may be impaired, but the impairment is secondary to the impulsivity or inattentiveness.

(a) Social: Social relationships and relationships within the family may be impaired because of the child's disruptiveness, bossiness, or oppositional behaviors.

(b) Emotional: Demoralization and self-esteem problems may be present secondary to the effects of others' reactions to the child's disruptiveness. Problems with self-regulation and affect regulation are often present.

(3) Sense of Self-cohesion: Sense of self remains reasonably intact, although self-esteem problems are prevalent. Regulation of affect states, particularly anger, is problematic.

(4) Coherence of Self-narrative: The self-narrative does not adequately explain the symptoms. Children may justify their behaviors by displacing the responsibility for their actions onto others.

d. Interventions:

(1) Remediation: Stimulant medications are often prescribed for the attenuation of the symptoms. These medications present a complication for children with a coexisting Tourette's disorder because the medication exacerbates the tics. Behavior modification sometimes produces positive results.

e. Psychotherapy: Family therapy or individual therapy may be used selectively, depending on the situation.

APA, A. P. A. (1994). Attention deficit hyperactivity disorder in adults: Editorial. *American Journal of Psychiatry, 152*(5), 633-638.

Barkeley, R. A. (2000). Genetics of childhood disorders: XVII. ADHD, Part 1: The executive function of ADHD. *Journal of the American Academy of Child & Adolescent Psychiatry, 39*(8), 1064-1068.

Castellanos, F. X., & Tannock, R. (2002). Neuroscience of Attention Deficits Hyperactivity Disorder: The search for endophynotypes. *Nature reviews. Neuroscience, 3*, 617-628.

Denckla, M. B. (2000). Learning disabilities and Attention-Deficit/Hyperactivity Disorder in adults: Overlap with executive dysfunction. In T. E. Brown (Ed.), *Attention-deficit disorders and comorbities in children, adolescents, and adults.* (pp. 297-318). Washington, DC: American Psychiatric Press.

Palombo, J. (2001). *Learning disorders and disorders of the self in children and adolescents*. NY: W.W. Norton. Ch. 8.

2. Executive Function Disorders

a. Characteristics:

(1) Defining Features: Executive Functioning Deficits involve a complex set of deficits that include: Difficulties with management of time. Difficulties conceptualizing the goal for a task, assessing its feasibility, lacking the organizational capacities to carry out the goal lacking ability to translate the plan into productive activity, or the capacity to self-monitor and self-regulate to measure progress toward the goal, being ineffective in the performance of the tasks involved.

(2) Prevalence Rates: No data are available on prevalence rates.
(3) Sex Ratio: No data are available on sex ration.
(4) Coexisting conditions: AD/HD often accompanies this condition.

b. Presenting Symptoms:

(1) Academic: The child underachieves because homework assignments are lost or not turned in. The child has poor study skills, he is inefficient in doing class assignments and appears scattered and disorganized. The child is described as procrastinator.
(2) Social: Social relationships appear unaffected by the disorders. As the child gets older, caregivers and teachers, get increasingly impatient with the child's disorganization as well as the reasons for the underachievement. Some children become oppositional in reaction to caregivers' attempts at structuring and organizing tasks for the child.
(3) Emotional: No distinctive emotional problems are associated with this disorders, although a pattern emerges of not being able to put order and sequence into life occurrences. A sense of bewilderment as to why things do not work out overtakes the child and erodes self-esteem. Patients are generally ineffectual in adapting to social and life situations, reflecting perhaps an absence of psychic structure.

c. Developmental History: Generally unremarkable with milestones achieved on time.

d. Interventions:

(1) Remediation: Intense tutoring to help the child develop habits that minimize the effects of the deficit.
(2) Psychotherapy: Parent guidance to supply structure for the child. Individual psychotherapy is elective.

Barkley, R. A. (1996). Linkages between attention and executive function. In G. R. Lyon & N. A. Krasnegor (Eds.), *Attention, Memory, and Executive Function* (pp. 307-325). Baltimore: Paul H. Brookes Pub.
Denckla, M. B. (1996). A theory and model of executive function: A neuropsychological perspective. In G. R. Lyon & N. A. Krasnegor (Eds.), *Attention, Memory, and Executive Function* (pp. 263-278). Baltimore: Paul H. Brookes Pub.
Palombo, J. (2001). *Learning disorders and disorders of the self in children and adolescents*. NY: W.W. Norton. Ch. 9.

e. Working memory

(1) Phonological loop:
(a) Brief storage associated with representational code. It involves the encoding of information as part of the process of reception. But "sensations" without "concepts" are meaningless. To the extent that sensations become perceptions, they involve a form of long-term retrieval. Speech forms are auditory signals that require translation for

comprehension to occur. Does the phonological loop involve comprehension or mere sensory reception?

(b) Left parietal area holds information in place (good digits forward), rehearsal goes to frontal (poor digits backwards).

(2) Visual-spatial sketch pad
   (a) Memory for objects
   (b) Memory for spatial locations

(3) Central executive
   (a) Attention
   (b) Executive function

(4) "Binding" brings together diverse information and places them in relationship to each other.

f. Complex set of behaviors reflecting metacognitive and control organizational processes

(1) Component Processes of EF
   (a) Involve attentional and regulatory acts
   (b) Inhibition
   (c) Delayed Responding - Intentionality
   (d) Shifting Focus/Set

(2) Each component process serves as a response or action that has as its primary function the alteration of the probability of a subsequent response by the individual:
   (a) Inhibition is a conscious act at deferring or deterring a specific behavioral response
   (b) Intentionality refers to underlying motivation and drive
   (c) Shifting focus or set refers to the act of redirecting attention to aspects of the environment that are most salient at a given point in time

(3) As Self-Regulatory behaviors: Interact with developing capacities and innate aspects of self to promote behavioral expression in service of goals and drives:
   (a) Developmental trajectories: -
      i) Infancy: Affective attunement
      ii) Toddlerhood: Socialization & moral awareness ("shame")
      iii) Middle Childhood: Cognitive competencies & social learning
      iv) Adolescence: Self-concept & refining independence
      v) Adulthood: Establishing goal-orientation & forming primary adult roles
   (b) Across the Lifespan: Identifying sources of disruption in EF and cognitive acts that translate into behavioral disorders:
      i) Deficits in attention, inhibition, affect regulation,
      ii) Comprehension of rules, language usage (expression, comprehension), social attunement -

iii) Failures in moral reasoning
iv) Disruptions in behavioral fluency and action
v) Deficits contribute to specific problems:
vi) Lack of focus or tendency to overfocus and become locked into a particular pattern of response
vii) Difficulty shifting attention to new sources of information
viii) Prone to act impulsively and without attention or awareness of consequences and implications
ix) Respond in a rigid and inflexible manner, even with feedback that rule being followed is incorrect or inappropriate
x) Fail to respond to environmental contingencies
xi) Require immediate reward or gratification
xii) Fail to attend to and make use of internally represented information, like past experience

g. **Where do they originate?**

(1) Arise from the development of neural networks within the frontal lobes of the brain and their connections with other brain regions directing thought and action -
(2) Dorsolateral prefrontal cortex
(3) Subcortical region, including basal ganglia, thalamus, and limbic structures

Class III. Psychopathology III.

A. Sociability and object relatedness

1. Introduction

a. Object relations and neuroscience
b. The sense of self
c. The neurobiological underpinning of the sense of self
d. Relational Component: The neurobiological underpinnings of object relationships.

[Much of what I discuss in this is contained in my two books. I therefore recommend that you use them as texts:
Palombo, J. (2001). *Learning disorders and disorders of the self in children and adolescents*. NY: W.W. Norton.

Palombo, J. (2006). *Nonverbal learning disabilities: A clinical perspective*. NY: W. W. Norton.]

2. Social communication

a. The mirror neurons system
   (1) ***How does the mirror neuron system contribute to the development of communicative abilities in humans?***
   (2) Mirror neuron theory
      (a) Mirror neurons are located in the pre-frontal region but probably exist in other regions of the brain.
      (b) They fire during specific activity related to the observations or enactment of hand-mouth activity. In humans they fire if the person thinks of the activity or watches someone performing the activity.
      (c) They are implicated in gestural communication, and more generally in nonverbal communication.
      (d) It is hypothesized that they are related to early evolutionary forms of communication.
      (e) The relevance of these findings to humans
      (f) Relationship between MNS and development

b. Theory of mind
   (1) The term theory of mind refers to the ability of typically developing children to attribute mental states such as desires, beliefs, and intentions, to themselves and others, as a way of making sense of and predicting the behavior of others (Tager-Flusberg & Baron-Cohen, 1993). The ability entails understanding that other people's behaviors are intentional or purposive, that a desire either to express their state of mind or to communicate with others motivates them. A pathological deficit in theory of mind abilities is believed to underlie autism. Autistic children have no difficulty understanding causal sequences, or even social routines and interactions, but they show significant impairment when asked to understand intentional tasks. Investigators who study autism consider this construct critical to understanding that disorder. (Baron-Cohen, 1997; Baron-Cohen, Tager-Flusberg, Cohen, Eds., 1993; Frith, 1989a; 1989b; Happe, 1993; Tager-Flusberg & Baron-Cohen, 1993).

c. Mentalization
   (1) Theory of mind describes how it is possible to attribute false beliefs to others, whereas mentalization or reflective function denotes the understanding of one's own as well as others behavior in mental state terms. In typical children, theory of mind develops by the ages of three-and-a-half to four. Mentalization or reflective function is the developmental acquisition that permits children to respond not only to another person's behavior, but to the child's conception of others' attitudes, intentions, or plans. Mentalization enables children to "read" other people's minds. By attributing mental states to others, children make people's behavior meaningful and predictable. As children learn to understand

other people's behavior, they can flexibly activate, from the multiple sets of self-object representations they have organized on the basis of prior experience, the one(s) best suited to respond adaptively to particular relationships (Fonagy & Target, 1998, p. 92).

d. Relevance, gist, and coherence
   (1) Relevance Theory and theory of mind
   (2) Central Coherence

3. Reciprocal social interactions

a. Complementarity and mindsharing
   (1) *Mindsharing may be defined as a form of intersubjectivity in which one person provides psychological functions that complement, and are essential to maintenance the integrity of, the sense of self of the other person. The interchanges between such dyads, at times, may be reciprocal. The experience of "being with" or feeling intimacy with another person is constitutive of the experience of mindsharing.*
   (2) Examples of this sense of the term mindsharing are the use of transitional object (Winnicott, 1953), the performance of auxiliary ego functions (Spitz, 1965), and selfobject functions (Kohut, 1971). In each of these functions either the presence of a person or an internal representation of a function that person performs is necessary for the other person to be able to maintain a sense of inner psychological stability and integrity, to which I refer as self-cohesion. I assume that understanding what is on another person's mind, explicitly or implicitly, is a necessary condition for self-other complementing.
   (3) Mindsharing as a form of mental state sharing and tuning serves not only to complement but also to transform the inner state of another person. Such are the dual functions that empathy performs. It not only serves as an instrument through which we can grasp another person's inner state, but it also provides a human milieu that is experienced a benignly caring.
   (4) The concept of complementarity has its roots in several intellectual traditions. In psychoanalysis,among complementary functions are transitional object functions, auxiliary ego functions, selfobject functions, and adjunctive functions.
   (5) *Transitional object functions:*
   (6) *Selfobject functions:*
   (7) *Adjunctive Functions:* Another group of phenomena that appears to operate in a manner similar to selfobject functions and that have been called auxiliary ego functions (Spitz, 1965), I designate as adjunctive functions (Palombo, 2001). These also serve to complement the sense of self, but they are conceptually different. While all selfobject functions provide complementarities to the sense of self, not all complementary functions are selfobject functions. Some examples may help clarify this distinction. Children often need to be reminded to perform a task or to prepare themselves for school in the morning.

Sometimes directions must be repeated to them, because either they were inattentive or because their auditory processing of verbal information is weak, e.g., they may have poor auditory memory skills. In performing these functions, caregivers are providing auxiliary ego functions or adjunctive cognitive functions for the child. They are complementing an area of immaturity or deficit, but are not necessarily providing the emotional complements that are embedded in selfobject functions.

(8) ***The Ontogeny of Mindsharing in Childhood***

b. Mutual regulation
c. Spectrum Disorders
d. Attachment
   (1) Secure attachments
e. I nsecure attachments

B. Disorders of Reciprocal Social Interaction: Attachment Disorders

[For this class, please read the chapters from our new forthcoming book *Guide to Psychoanalytic Developmental Theory*, which are available on the web:
Section VII: Attachment Theories: Part 1: Traditional Attachment Theories, John Bowlby, Mary Ainsworth & Mary Main; Part 2: Neuropsychological Attachment theories: The return to psychoanalysi, Allan Schore, Peter Fonagy.]

1. **Bowlby**
2. **Ainsworth/Main**
3. **Fonagy**

4. **Allen Schore**
   a. Schore presents a psychoneurobiological point of view that specifies the structural systems of the developing unconscious in terms of recent brain research.
   b. Attachment redefine
      (1) For Schore, attachment theory is a regulatory theory[1]. Attachment occurs as a result of the emotional interchanges between the infant and its caregiver and the attachment process mediates the social construction of the social brain. Schore's theoretical program became that of elucidating the neurobiological underpinnings of the emotional interchanges between infant and caregiver and of the processing and regulation of emotional information by the infant's brain.

[1] An idea first introduced by Sroufe (1996)

To that end, Schore explored the functions of the right hemisphere, the frontal and prefrontal cortex and the subcortical systems associated with these processes.

(2) Bowlby had maintained that attachment behavior is instinctive. It reflected evolutionary processes that operate in the service of survival through seeking proximity to a caregiver that provides a secure base. In contrast, Schore's formulation goes beyond Bowlby's and proposes that attachment theory is a regulatory theory. Schore states: Aattachment is instinctive behavior with a biological function, ... emotional processes lie at the foundation of a model of instinctive behavior, and ... a biological control system in the brain regulates affectively driven instinctive behavior. This control system can now be identified as the orbitofrontal system and its cortical and subcortical connections. This >senior executive of the emotional brain' acts as a regulatory system, and is expanded in the right hemisphere, which is dominant in human infancy and centrally involved in inhibitory control. ." (2000, p. 23) "[A]ttachment [is] now defined as the interactive regulation of states of biological synchronicity between organisms*"* (Schore 2002, p. 443). He also states: "[R]esonant emotional transactions involve synchronized and ordered directed flow of energy in the infant's and mother's brains." (2002. P.444)

5. **Secure attachments**
   a. *Group B. Securely Attached*: The **Securely Attached** child will greet mother with a smile on reunion, seeming to welcome her return. Whereas the child may or may not interact with the stranger, the child actively seeks interaction with mother, seeking proximity and contact with her. He or she will wish for the contact to be maintained and protests if put down after being picked up. The child is distressed during the separation from mother and clearly wants to be reunited with her.
   b. The neurobiology of secure attachments
      (1) Right Brain Function
      (2) The Orbitofrontal region
6. **Insecure avoidant attachments:** *Group A. Anxious/Avoidant:* The **Anxious/Avoidant** child avoids interacting with mother during the reunion episodes, may avert his or her gaze, and is not distressed by the separation. He or she does not resist contact with mother and treats the stranger in the same way.
7. **Insecure ambivalent attachments:** *Group C. Anxious/Resistant**:*** The **anxious/resistant** child exhibits clear-cut ambivalence toward mother during the reunion episodes, seeking proximity while at the same time resisting the contact that is made. With the stranger, he or she will exhibit more anger or passivity than children in the other groups.
8. **Disorganized/disoriented attachments**
   a. Children with Type D attachments
      (1) As Main discovered, children with Type D attachments experience have low stress tolerance and instead of finding a haven of safety in the relationship are alarmed by the parent. According to Schore, when caregivers either neglect or

maltreat a child, the child's response to such a trauma is comprised of two separate response patterns, *hyperarousal* and *dissociation*

(2) One sequella of the trauma is an interference is the capacity to perceive others' emotional states as well as an impaired capacity to regulate and modulate their own internal emotional states. The accompanying developmental failures produce severe difficulties in forming attachments to others as well as serious psychopathology.

9. The Neurobiology of Insecure Attachments
10. Bereavement

Palombo, J. (1981). Parent loss and childhood bereavement: Some theoretical considerations. *Clinical Social Work Journal, 9*(1), 3-33.

Class IV. Disorders of Social communication disorders: NLD

A. Right hemisphere functions: Nonverbal Learning Disabilities (Social learning disabilities)

1. **Characteristics:**
   a. *Defining features*: Patients exhibit a complex set of neurocognitive strengths and weaknesses: strengths in rote verbal memory, in reading decoding and spelling; weaknesses in tactile and visual perception and attention, in concept formation, reading comprehension of complex materials, problem solving, and dealing with novel materials; academic problems in math and science; and a pattern of socioemotional difficulties involving with the reception and expression of modulated affects and of nonverbal modes of communication.
   b. The four subtypes.
   c. *Prevalence rates*: No data are available on prevalence.
   d. *Sex ratio*: Unknown, but clinically the rate of referral to the Rush Neurobehavioral Center is 10:1 boys to girls.
   e. *Coexisting conditions*: Chronic anxiety, ADD-like symptoms, and depression.
2. **Developmental History**: As infants they are passive, fail to engage in exploratory play, and do not respond as expected. They appear clumsy and poorly coordinated. They have difficulties interacting with other children in groups. They are unable to form friendships or to sustain being with other children even for brief periods of time without an eruption ensuing.
3. **Disorders of the Self**:
   a. *Presenting symptoms*:
      (1) *Academic*: They have poor handwriting and are deficient in arithmetic skills. Their reading comprehension is not on a par with their verbal skills, although they are good readers, they have great difficulty with tasks required by art

classes. They also have problems with attention, dealing with novel materials, and adjusting to new situations.

(2) *Social*: Their social functioning is often problematic; they interact well with adults but not with peers. They are unable to decode social cues, failing to "reading" other people's body language, facial expressions, and vocal intonations. They are inept in social situations.

(3) *Emotional*: The area of affective communication is problematic. They appear unable to decode feelings associated with prosodic or vocal intonations; they have difficulty reading the feelings conveyed by facial expressions. They have problems in modulating or regulating certain affects; they lose control and have temper tantrums, when frustrated.

b. *Sense of self-cohesion*: The child's sense of self is prone to injury and vulnerable to fragmentation manifested as rage attacks or meltdowns.

c. *Self-narrative coherence*: In their self-narratives they provide explanations for their thoughts and behaviors that exclude the significance of the learning disorders. These self-narratives have elements of incoherence that are rationalized and attributed to the child's experiences.

4. Interventions:

a. *Remediation*: Specialized social-skills groups that address problems with social interactions.

b. *Psychotherapy*: Long-term supportive psychotherapy can be helpful.

5. NLD and Affects, Emotions, and Feeling

a. Reception of emotional communications

(1) The children's inability to read others' feelings has often led to the misconception that they lack empathy, that is, the capacity to understand and appreciate how another person feels. On further examination, however, it is clear that these children are not insensitive to others. In discussions with them, they demonstrate the capacity to understand others' feelings; where they fail is in the correct interpretation of what transpired. Their misreading of others leads to responses that are considered insensitive and therefore unempathic.

b. The Expression of Emotions

(1) One of the more frustrating experiences of caregivers in their interactions with some of these children is that it is difficult to know how the children feel. Because they often give little indication of their feeling states through their facial expressions, caregiver cannot reliably know what is going on within the child. If they guess at the child's feeling, they find themselves in an argument with the child who claims not to feel that way at all. The child ends up feeling misunderstood and further isolated. Yet the children expect to be understood in spite of their expressive difficulties.

c. Dispositional states

(1) Feelings about self: Self-esteem, Oversensitivity and overreactivity to criticism.

(2) Feeling states

(a) The anxiety is generally pervasive, deserves special consideration.

(b) The constant frustrations to which children with NLDs are exposed lead them to feel helpless and enraged.
(c) Many children with NLDs vacillate between exited hyperactivity and low-keyed sadness.

(3) Affect regulation

B. Disorders of Reciprocal Social Interaction & Social Commincation: Asperger's Disorder

1. Characteristics:
   a. *Defining features*: *DSM-IV* criteria include the following:
      (1) Qualitative impairment in social interaction.
      (2) Restricted repetitive and stereotyped patterns of behavior, interests, and activities.
      (3) Stereotyped and repetitive motor mannerisms, and
      (4) Persistent preoccupation with parts of objects
   b. *Prevalence rates*: Klin and Volkmar quote studies as finding the prevalence rate to be 3.6 per 1,000 (1997, p. 106).
   c. *Sex ratio*: The ratio of male to female is reported to be 4:1.
   d. *Coexisting conditions*: Tentative data, based on case studies, document the presence of depression, obsessive-compulsive disorder, and AD/HD (Klin & Volkmar, 1997, p. 114).
2. **Developmental History**: Wing finds that in infancy and early childhood children with Asperger's lack normal interest in people, their babbling is limited, their shared interests and activities are very reduced, they lack the drive to communicate verbally and nonverbally, speech acquisition is delayed, and they do not engage in imaginative play.
3. Disorders of the Self:
   a. *Presenting symptoms*:
      (1) *Academic*: The children appear as average or above average in intelligence, often have unusual capacity for rote memorization, and learn to read quite early. They may have difficulties with written work because of their motor problems. As they progress to higher grades, they are less responsive to academic demands and consequently begin to fall behind. This is not because of their inability to do the work, but because they cannot conform to expectations.
      (2) *Social*: Their social functioning is impaired as they present as odd, cold, stiff, egocentric and immature. Their capacity for social communication is impaired in the nonverbal area and in the area of pragmatics. In the latter area, they are tangential, engage in long monologues that are unrelated to the context in which they find themselves, and seem to be conducting one-way conversations.
      (3) *Emotional*: They appear to lack the capacity for empathy, seem not to appreciate that others have feelings, and intentions.
   b. *Sense of self-cohesion*: They maintain a tenuous sense of self-cohesion and are prone to meltdowns or fragmentation when stressed. Among the defenses they use

to maintain self-cohesion is that of collecting facts in an area where they become expert, although the knowledge is not put to any use.

c. *Self-narrative coherence*: The children appear unable to generate a self-narrative. This may reflect their fragmented or noncohesive sense of self or it may represent their difficulties with pragmatic language.

4. Interventions:
   a. *Remediation*: Extraordinary interventions are recommended to begin at an early age. These include occupational therapy, speech therapy, and socialization groups
   b. *Psychotherapy*: Intervention with mother and child to deal with the child's attachment issues, as the child gets older, supportive therapy for the family, and possibly individual therapy for the

C. Case presentation

Class V. Disorders of Emotional communication: Anxiety Disorder

[For this class, I strongly recommend one book, which covers the topic in simple and understandable language. LeDoux, L. (1996). *The Emotional Brain: The mysterious underpinnings of emotional life*. New York: Simon & Schuster.]

A. Anxiety disorders, phobias, and the amygdala

1. **Conditioned fear can occur when an *unconditioned stimulus* is paired with a *conditioned stimulus* resulting in a conditioned response or when a *natural trigger* is paired with a *learned trigger* resulting in fear conditioning.**

2. **The amygdala is like the hub of a wheel.**
   a. The amygdala processes low-level inputs from sensory-specific regions of the thalamus, higher level information from sensory-specific cortex, and still higher level (sensory independent) information about the general situation from the hippocampus formation. The amygdala is, in essence, involved in the appraisal of emotional meaning.
   b. *If in some individuals (for genetic or acquired reasons) thalamic pathways are dominant or otherwise uncoupled from the cortical pathways these persons might form emotional memories on the basis of stimulus events that do not coincide with the ongoing conscious perceptions of the world mediated by the cortex. Such people would have very poor insight into the emotions.* At the same time, *if the hippocampus system were uncoupled from the thalamic or cortical projections to the amygdala, we might have persons who express emotions that are inappropriate to the immediate context, including possibly the social context.*
   c. Destruction of the amygdala illuminates the fear response
   d. By way of the amygdala and its input and output connections, the brain is programmed to detect dangers, both those that were routinely experienced by our

ancestors and those learned about by each of us as individuals, and to produce protective responses that are most effective for our particular body type, and for the ancient environmental conditions under which the responses where selected. P. 175

e. One reason that cognition is so useful a part of the mental arsenal is that it allows the shift from reaction to action in the face of danger of stimuli. Cognitive resources are directed to "risk assessment." By sizing up situations and planning how to maximize our gains and minimize our losses our survival is enhanced.
f. Emotional coping represents the cognitive planning of voluntary actions once we found ourselves in the midst of an involuntarily elicited emotional reaction. Evolutionary programming sets the emotional ball rolling, but from then on we are very much in the drivers seat.

3. The low and the high road:

a. ***The low road***:
   (1) The thalamic system has important advantages over the cortical input pathway to the amygdala. The advantage is time. The cortex system's job is to prevent the inappropriate response rather than to produce the appropriate one. The thalamo – amygdala and cortico– amygdala pathways converge in the lateral nucleus of the amygdala.
   (2) Information about external stimuli reaches the amygdala by way of direct pathways from the thalamus (the low road) as well as by way of pathways from the thalamus to the cortex to the amygdala. The direct thalamo-amygdala path is shorter and is a faster transmission route than the pathway from the thalamus through the cortex to the amygdala. However, because the direct pathway bypasses the cortex, it is unable to benefit from cortical processing. As a result, it can only provide the amygdala with a crude representation of the stimulus.
   (3) The central nucleus of the amygdala has efferent projections to the brainstem that controls heart rate and to the autonomic nervous system that controls the freezing responses, the release or stress hormones, blood pressure, the suppression of pain, the potentiation of reflexes, such as the startle reflex, and the fight/flight response

b. ***The high road:***
   (1) The sensory thalamus sends the information to the hippocampus and the cortical circuits
   (2) The hippocampus
      (a) Conditioning occurs not only to the immediate stimulus directly associated with trauma but also to other stimuli that just happened to have been there. These makeup the ***context*** in which the trauma took place, the context is conditioned by the traumatic experience. The context is made up of all the stimuli present, other than the explicit conditioning stimulus.
      (b) The integration of individual stimuli into a context that no longer contains the individual elements is a function of the hippocampus. Unlike the

amygdala, the hippocampus does not get information from brain regions that process individual sensory stimuli. Instead, the sights and sounds of a place are thrown together before reaching the hippocampus, and one job of this brain region is to create a representation of the context. It contains not individual stimuli but the relations between stimuli. A hippocampal lesion selectively eliminates fear responses elicited by contextual stimuli without affecting fear response that elicited by the stimuli.

(c) The hippocampus and the amygdala organize memories.

B. Anxiety disorders reflect the operation of the fear system of the brain.

***1. Fear and loathing in anxiety*:**

a. ***Anxiety and fear are closely related. Anxiety is usually distinguished from fear by the lack of an external stimulus that elicits the reaction – anxiety comes from within us, fear from the outside world. Fear is related to the behavioral acts of escape and avoidance in threatening situations, and when these actions are thwarted, fear becomes anxiety.***

b. DSM classifies anxiety disorders as including: ***panic, phobias, PTSD, obsessive-compulsive disorder, and generalized anxiety.*** Anxiety disorders reflect the operation of the fear system of the brain.

(1) ***Phobias*** are fears of specific stimuli or situations that are in excess of the actual threat they pose.

(2) ***Panic attacks*** involve discrete periods of intense anxiety and discomfort.

(3) ***PTSD*** involves severe anxiety elicited by stimuli that were present during some extreme trauma or that somehow are related to stimuli that occurred during the trauma.

(4) ***OCD*** involves intrusive, repetitive, and persistent thoughts and/or repetitive behaviors that are performed in a very precise way in response to obsessive thoughts.

(5) ***Generalized anxiety***, also known as free-floating anxiety, involves excessive worry about unrelated things for long period of time.

2. Learning theories of anxiety state that:

a. The genesis of pathogenic anxiety ***involves fear conditioning.*** This theory is based on Pavlov's condition reflexes.

b. Another form of learning, called ***instrumental conditioning*** proposes that an arbitrary response is learned if it is reinforced, which means it is either followed by the presentation of a reward or the omission of a punishment.

c. Ready to fear: **[preparedness theory: a genetic/heritable component to aversive reactions to some objects–which varies from individual to individual]** Seligman suggested that phobias reflect our evolutionary preparation to learn about danger and to retain the learned information especially strongly.

d. **These theories fail to explain Anxiety Disorders**. They do not explain why people would forget the event that triggered the trauma, and they do not explain the problems with the extinction of the anxiety.

3. Traumatic learning: Two things occur in traumatic learning:

a. ***conscious memories are laid down by a system involving the hippocampus and related cortical areas,*** and

b. unconscious memories established by fear conditioning mechanisms operating through an amygdala-based system.

c. Forgetting:

(1) Recent work showing that stressful events can cause malfunctions in the hippocampus suggests that the failure to recall may be due to a stress-induced breakdown in hippocampal memory function. (See p. 241)

(2) ***Stress does not appear to interfere with the workings of the amygdala and may even to enhance its function. It is thus possible that one might have poor conscious memory of a traumatic experience, at the same time form very powerful implicit, unconscious emotional memories through amygdala mediated fear conditioning. Such memories become very resistant to extinction, and there is no way to convert these memories into explicit memories.***

d. Extinction:

(1) *Brain malfunction can make unprepared learning resistant to extinction:* ***Extinction appears to involve the cortical regulation of the amygdala, and even unprepared condition fear can be resistant to extinction and the mental is freed from these cortical controls. The prefrontal cortex, like the hippocampus, may be altered by stress. A stress-induced shutdown of the prefrontal cortex might release the brakes on the amygdala, making new learning stronger and more resistant to extinction, and possibly allowing previous be extinguished conditioned fear is to be expressed the new.***

e. *Gone but not forgotten-the indelibility of emotional memory*:

(1) ***The finding that when the medial prefrontal cortex is damaged routine fear conditioning becomes resistant to extinction has another important implication. It suggests that extinction prevents the expression of conditioned fear responses but does not erase the implicit memories that underlie these responses. An explanation for this finding may be that the extinction decreases the firing of neurons but does not undo the strengthening of the connections between them. It is these connections event are reactivated in the revival of the fear response. Unconscious fear memories established through the amygdala appear to be indelibly burned into the brain. We may therefore not be able to get rid of the implicit memories that underlie anxiety disorders. The best we can hope for is to exercise control over them (see diagram p. 253).***

C. Anxiety Disorders

1. Phobias

a. ***Contemporary ideas about phobias continue to be centered around the notion of preparedness.*** Normally, the strength of conditioning is determined mainly

(though not exclusively) by how traumatic the unconditioned stimulus is. But in prepared fear conditioning, the conditioning stimulus also contributes some of the emotional impact. As a result, given two conditioned stimuli, one that is biologically prepared to be conditioned to danger and the other not, the same unconditioned stimulus should support the establishment of a stronger conditioned responds for the prepared stimulus.

b. ***It may be that because of genetic predisposition or past experiences, phobic learning may involve the subcortical pathway to a great extent than the cortical pathway, especially for prepared stimuli.***

c. Although amygdala – mediated fear conditioning is a form of implicit learning, phobic's are consciously afraid of their phobic stimuli. This means that they have and explicit conscious memory, performed through their temporal lobes memory system, that reminds them that they are afraid of snakes, heights, or whatever (See page 255). This memory might be established during the initial traumatic learning situation, but some phobic's do not recall such a learning experience, possibly because of the stress-induced memory loss. In such instances, the conscious memory of the phobically afraid could be established in later experiences with the phobic object. When the object is encountered, the amygdala will unconsciously detect the stimulus and produce the bodily expression of fear. Upon becoming aware of this by the response, the person attributes the arousal to the most likely object and forms a memory that they are afraid of object of that type. Once this explicit memories created, its retrieval into consciousness becomes a potent stimulus that is itself capable of activating the amygdala and producing anxieties by way of connections from cortical areas (including the hippocampus) to the amygdala. Even if one does not have the conscious memory of the initial learning, there is likely to be an awareness of the phobic condition stored in explicit memory.

D. **Traumatic stress (PTSD)**

1. ***The difference between a fear conditioning theory of phobia and PTSD revolves around the issue of where the conditioning process gets strength.*** In the case of prepared phobic learning, the conditioned stimulus makes the learning especially strong. The unconditioned stimulus is typically unpleasant and maybe even painful, but is not necessarily extraordinary.
2. ***However, in the case of PTSD, the conditioned stimulus events are less notable than the unconditioned stimulus (the actual traumatic events). PTSD involves a trauma that is far outside the realm of experience in ordinary life.***

E. ***Panic***

1. ***While phobic and PTSD responses occur in the presence of external stimuli, panic attacks appear to be more related to internal stimuli.*** For that reason it is more difficult for a person to avoid the stimuli that brings it on. A panic attack can be induced by having a patient hyperventilating or inhale a gaseous mixture rich in carbon dioxide, or giving the patient and intravenous injection of sodium lactate. These procedures give rise to internal signals similar to those that are typically present during a naturally occurring attack.

2. ***One common view is that artificial panic induction procedures lead to bodily sensations that then serve as conditioned stimuli. Having experienced panic before, the patient learns the warning signs. When these internal signals occur the patient feels the panic is starting. This cognitive appraisal of bodily sensations that drives the system into panic. Induced panic, and presumably natural panic, by this way of thinking, is a conditioned response to internal stimuli that occurred during past panic attacks.***

F. ***Bad habits and anxious thoughts***
   1. Avoidance responses that so typify anxiety disorders fall somewhere between innate emotional reactions and voluntary emotional actions. Avoidance responses are instrumental responses that are learned because they often reinforced. They are then performed habitually when the appropriate stimuli appear. These are arbitrarily related to danger. The brain has learned some responses that can be performed in the presence of a learned trigger that short circuits the innate responses.
   2. ***Mower and Miller proposed that avoidance learning is usually thought of as taking place in two stages: First fear conditioning occurs and second a response is learned because it supposedly reduces the learned fear.***

G. **Psychotherapy: just another way to re-wire the brain:**
   1. Psychoanalytic theory and conditioning theories assume that anxiety is a result of traumatic learning experiences that foster the establishment of anxiety producing long-term memories. However each theory leads to different therapeutic approaches. Psychoanalysis seeks to uncover the unconscious origins of the conflict whereas behavior therapy tries to rid the person of the symptoms of anxiety through a form of extinction therapy, such as relaxation training. This approach is called systematic desensitization.
   2. Extinction appears to be involved in interactions between the medial prefrontal cortex and the amygdala. Therapy is just another way of creating synaptic potentiation in brain pathways that control the amygdala. Behavioral (extinction) therapy and psychoanalysis have the same goal-help the person with their problems. In both cases, the effects may be achieved by helping the cortex gain control over the amygdala. However, the neural roads taken may be different. Extinction therapy may take place through a form of implicit learning involving the prefrontal-amygdala circuits, whereas psychoanalysis, with its emphasis on conscious insight and conscious appraisals, may involve the control of the amygdala by explicit knowledge through the temporal lobe memory system and other cortical areas involved in conscious awareness. P 265

Bibliography on Psychoanalysis & Neuroscience

Applegate, J. S., & Shapiro, J. R. (2005). *Neurobiology for Clinical Social Work: Theory and Practice*. NY: W.W. Norton.

Bornstein, R. F. (2004). Reconnecting psychoanalysis to mainstream psychology: An agenda for the 21st Century. In J. Reppen, J. Tucker & M. A. Schulman (Eds.), *Way Beyond Freud: Postmodern psychoanalysis observed* (pp. 1-19). London: Open Gate Press.
Brothers, L. (1997). *Friday's Footprint: How society shapes the human mind*. NY: Oxford University Press.
Cornelius, R. R. (1996). *The science of emotion: Research and tradition in the psychology of emotion*. New Jersey: Prentice Hall.
Cozolino, L. (2002). *The neuroscience of psychotherapy: Building and rebuilding the brain*. N.Y.: W. W. Norton.
Eliot, L. (1999). *What's going on in there? How the brain and mind develop in the first five years of life.* N.Y. Bantam Book.
Gazzaniga, M. S. (1988). *Mind Matters: How Mind And Brain Interact To Create Our Conscious Lives*. Boston: Houghton Mifflin.
Gilkerson, L. (2001). Integrating an understanding of brain development into early chidhood education. *Infant Mental Health Journal, 22*(1-2), 174-187.
Goldberg, E. (2001). *The exceutive brain: Frontal lobes and the civilized mind*. NY: Oxford University Press.
Kandel, E. R. (1998). A new intellectual framework for psychiatry. *American Journal of Psychiatry, 155*(4), 457-469.
LeDoux, L. (1996). *The Emotional Brain: The mysterious underpinnings of emotional life.* New York: Simon & Schuster.
Pally, R. (1997a). I. How brain development is shaped by genetic and environmental factors. *International Journal of Psycho-Analysis, 78*, 587-593.
Pally, R. (1997b). II. How brain actively constructs perceptions. *International Journal of Psycho-Analysis, 78*, 1021-1030.
Pally, R. (1997c). Memory: Brain systems that link past, present and future. *International Journal of Psycho-Analysis, 78*, 1223-1234.
Pally, R. (1998a). Bilaterality: Hemispheric Specialisation and Integration. *International Journal of Psycho-analysis, 79*, 565-578.
Pally, R. (1998b). Emotional Processing: The Mind-Body Connection. *International Journal of Psycho-analysis, 79*, 349-362.
Palombo, J. (1991). Neurocognitive Differences, Self Cohesion, And Incoherent Self Narratives. *Child & Adolescent Social Work Journal, 8*(6), 449-472.
Palombo, J. (1992a). *Learning Disabilities In Children: Developmental, Diagnostic And Treatment Considerations.* Paper presented at the Fourth National Health Policy Forum, Healthy children 2000:Obstacles & Opportunities, April 24-25, 1992, Washington, D.C.
Palombo, J. (1992b). Narratives, Self-Cohesion, And The Patient's Search For Meaning. *Clinical Social Work Journal, 20*(3), 249-270.
Palombo, J. (1993). Neurocognitive Deficits, Developmental Distortions, And Incoherent Narratives. *Psychoanalytic Inquiry, 13*(1), 85-102.
Palombo, J. (1994). Incoherent self-narratives and disorders of the self in children with learning disabilities. *Smith College Studies in Social Work, 64*(2), 129-152.
Palombo, J. (1995). Psychodynamic and relational problems of children with nonverbal learning disabilities. In B. S. Mark & J. A. Incorvaia (Eds.), *The Handbook of Infant, Child, and*

Adolescent Psychotherapy: A guide to Diagnosis and Treatment. Volume I (Vol. 1, pp. 147-176). New Jersey: Jason Aronson.
Palombo, J. (1996a). The diagnosis and treatment of children with nonverbal learning disabilities. *Child & Adolescent Social Work Journal, 13*(4), 311-332.
Palombo, J. (1996b). Paradigms, metaphors, and narratives: Stories we tell about development. *Journal of Analytic Social Work, 4*(3), 31-59.
Palombo, J. (2000). A disorder of the self in an adult with a nonverbal learning disability. In Goldberg (Ed.), *Progress in Self Psychology* (Vol. 16, pp. 311-335). NY: Analytic Press.
Palombo, J. (2001a). *Learning disorders and disorders of the self in children and adolescents*. NY: W.W. Norton.
Palombo, J. (2001b). The therapeutic process with children with learning disorders. *Psychoanalytic Social Work, 8*(3/4), 143-168.
Palombo, J. (2006). *Nonverbal learning disabilities: A clinical perspective*. NY: W. W. Norton.
Palombo, J., & Berenberg, A. H. (1997). Psychotherapy for children with nonverbal learning disabilities. In B. S. Mark & J. A. Incorvaia (Eds.), *The Handbook of Infant, Child and Adolescent Psychotherapy: New Direction in Integrative Treatment.* (Vol. 2, pp. 25-68). Northvale, New Jersey: Jason Aronson.
Plizzka, S. R. (2003). *Neuroscience for the Mental Health Clinician*. NY: Guilford Press
Ramachandran, V. S., & Blakeslee, S. (1998). *Phantoms in the brain: Probing the mysteries of the human mind.* N.Y.: Quill.
Ratey, J. J. (2001). *A user's guide to the brain: Perception, attention, in the for theaters of the brain*. New York: Pantheon Books..
Squire, L. R., & Kandel, E. R. (1999). *Memory: From mind to molecules*. NY: Scientific American Library.
Schore, A. N. (1997). A century after Freud's Project: Is a rapprochement between psychoanalysis and neurobiology a hand? *Journal of the American Psychoanalytic Association, 45*(3), 807-840.
Shapiro, J. R., & Applegate, J. S. (2000). Cognitive neuroscience, neurobiology and affect regulation: Implications for clinical social work. *Clinical Social Work Journal, 28*(1), 9-21.
Siegel, D. J. (1999). *The developing mind: Toward a neurobiology of interpersonal experience.* NY: Guilford Press.

J. Palombo
June 1, 2008
C:\1Data\1Data Dev\007Current Inst Courses\Summer 2010\5 Instructor's file\Dis of Childhood 10 0515 OTL.wpd