Biopsychosocial Therapeutic Modalities in the Treatment of Traumatic Brain Injury

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By

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Abstract

Traumatic brain injury (TBI) is a leading cause of death and disability among persons in the United States. Each year, an estimated 1.7 million Americans sustain a TBI. Because the overarching symptomatology of TBI encompasses biological, psychological, and social deficits, a spectrum of intervention to match those needs would be logically imperative to meet holistic therapeutic outcomes. This literature review covers the intrinsic modalities of intervention, which are informed by recent research in acute-phase stabilization, neurobiological assessment, neuroimaging, psychopharmacology, neurocognitive remediation, and psychotherapy. Previously, these modalities were held largely as separate treatment entities in the case of TBI. Yet, with advancement in more precise measurement, a deeper understanding of treating all phases of deficit in the individual in concert between the disciplines proves to yield better therapeutic outcomes for the individuals and their families.
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Biopsychosocial Therapeutic Modalities in the Treatment of Traumatic Brain Injury

We are what our brains allow us to be. Yet that allowance comes with factors both inherited genetically and acquired post-birth. Our reasoning and cognitive awareness are truly remarkable examples of our brains’ evolved higher functions. This can be seen in the ability for the brain to use complex visual, auditory and sensory data from all around it, and, with projection and differential reasoning, to comprehend and operate within those stimuli toward specific goals. Above all goals set out by higher cognitive aspiration, however, is the brain’s primary function of maintaining homeostasis of the body it is entrusted to govern. Through a complex system of autonomic regulatory systems and inbuilt feedback mechanisms, the brain serves as a control center for effective homeostatic balance toward optimal functioning of all somatic and cognitive systems. These systems work in concert to maintain organic balance given their environmental conditions.

To illustrate the complexity of multi-level processing in the brain, consider a simple scenario: Imagine yourself walking up a busy set of stairs, while carrying a parcel, having a lighthearted conversation with a friend on a cell phone, recounting a fond recent memory. This seemingly unremarkable scene to the outside observer takes on the air of the wholly remarkable when viewed from the complex systems required to complete the task. The myriad of visual, spatial, auditory and somatic information processing alone are astoundingly intricate. From the signaling of heart rate increase to supply proper oxygen levels to the muscles, to issuing fine and gross motor corrections to maintain balance, direction, and speed changes to negotiate obstacles, these incredible acts of synchrony are often taken for granted. Pair with this physical task the unrelated higher cognitive functions of memory, emotion and language processing happening simultaneously. When viewed from a biological, psychological, and social perspective, the
entirety of the processing required to complete this seemingly simple task is nothing short of astounding.

Imagine now the same scenario on that same busy staircase, yet due to a prior traumatic brain injury (TBI), you are having difficulty physically negotiating the steep incline, the moving crowd of people around you, the direction of travel or balance due to visual, auditory, or biomechanical deficiencies. You may have consistent painful headaches, requiring powerful medications that may further impair these physical deficiencies. You are unable to move the way other people move. The seemingly simple cell phone call you may have been able to make prior to the injury, has now become a difficult emotional process of incomprehension, confusion and frustration. You may have difficulty remembering the fond memory the person on the other end is recounting, or you may not remember the name of the person speaking to you or the last sentence spoken. At its most extreme, you may not comprehend the utterances coming from the strange device in your hand. This cascading deficiency of function can be seen and felt on biological, psychological and social levels. The interrelatedness of these functions, which make up human sentience, has been corrupted by one traumatic event, through which all processes are either directly or indirectly affected.

Yet, somehow, within the core of the brain-injured individual are pictures stitched together in a patchwork of memory or personal history. They may seem alien, as they include a person who had much higher physical, cognitive and social capabilities. The one experiencing real time events feels a shell of his or her former self; a sensation or faint recollection of an event in childhood reminds the person that things were not always this way. Somewhere within there is sense that they were not always “broken”. Feelings of inferiority, frustration, sadness and fear are common in those suffering, giving rise to psychopathologic disorders, with the possible caveat of
inadequate faculty to rationalize or express these feelings to others. Because of this, a TBI sufferer may feel alone, misunderstood and isolated, feeling relegated to a life within a perception of an uncaring world that may appear frightening, confusing, and altogether inhospitable.

**Statement of Purpose**

Because the symptomatology of TBI encompasses biological, psychological, and social deficits, a spectrum of intervention to match those needs would be a logical imperative to yield a more positive therapeutic outcome for patients. It is the writer’s opinion that, based on research within all three facets of symptomatology relating to TBI, an interweaving of the empirical with the theoretical, the objective with the subjective, reveals a new protocol for treatment. The case for sound holistic intervention parallels much of life’s processes of survival through systemic adaptation and resilience. These processes are both affective and biological, and when faced with challenge in vivo, are tested in vitro with feedback information received and implemented in continuance of systemic homeostasis of the individual. A modality of treatment based on aspects of social learning theory and psychotherapy, coupled with finite neuroscientific data to better serve a patient’s pharmacological and functional rehabilitative needs, has grown to be the accepted treatment model (Grosswasser & Keren, 2007). Previously, prognoses of the brain’s possible healing capabilities were held within the realm of the unknown, yet with verifiable neuroimaging and measurement, gives encouragement and proof to the adaptive or “plastic” nature of the brain post-injury (Shaw & McEachern, 2001). These revelations forge a new protocol for unified intervention, now being applied to the treatment and rehabilitation of brain-injured individuals. Because of the necessarily specific nature of intervention, applied solely to TBI, it is the goal of this paper to provide inquiry and review of biopsychosocial interventions specifically relating to each phase of treatment as it applies to the holistic model. This paper also
serves to provide overview to treatment methods based on both qualitative and quantitative therapeutic outcomes in the treatment of traumatic brain injury within each facet of the treatment protocol.

**Overview of Traumatic Brain Injury**

A traumatic brain injury (TBI) changes a person on an individual level. It impacts their ability to interact with their environment effectively. TBI may cause changes that affect cognition, behavior, somatic function, and neuromedical status. Such an injury can have a negative impact on the complex environmental biological relationships within the body, as well as cognitive processes, limiting normal interaction with the outside world (Bootes & Chapparo, 2002). TBI truly is, for the sufferer, a biopsychosocial issue. While seeing this in a close family member who suffers from a recent TBI, as well working as a mental health practitioner and skills worker with individuals who have sustained traumatic brain injuries over the past three years, I have seen these impacts first hand. Managing the complexities of nuance in life, flexibility in reason and understanding and performing daily tasks I take for granted, such as grooming, managing a schedule, or even recall of the reason for a schedule, these real difficulties have all opened my eyes to the impact a TBI might have on what society may term normal, everyday function.

Though valuable in the expansion of my understanding of psychological dysfunction, the most salient point I have come to realize is that many TBI sufferers demonstrate the capability to recall past experiences, though often the experience was not felt as real, or their own, because of their present state: rather, they experience a split life, so to speak. The faint “shreds” of memory resembled a movie with a character like them, yet the character was married, employed, a mother, a student, a parent, a soldier or a football star. This presented a dualistic sense of self, where awareness of oneself might take the form of a shadow identity. Oftentimes, when in session, we
would work on present moment mindfulness, checking in with oneself, expressing what is being felt in the “here and now”, where consistent self scripting of time and place would guide their awareness toward directed goals.

In addition with TBI clients, there may be a somatic component, which includes a bodily function or dysfunction they must now negotiate. Mindfulness exercises and bodywork can help to strengthen connection with the alien form that they often struggle with. This proves effective in that it helps them ground emotions to the body they are contained within. In other clients, there might be an acute unawareness of deficit, and that the ego is enmeshed in an inability to see differences. Though this would be due to an organic deficiency, the deficiency, which would follow, might in some cases not be of an affective nature, yet may be ego syntonic, warranting a possible diagnosis on Axis II. In either case, though some insight might be gained, the TBI sufferer feels an overarching sense of hopelessness, frustration, and confusion. Different from a disorder, in which the root cause can be seen, addressed and mindfully challenged, the TBI sufferer has the added dimension of biological deficiency affecting awareness. “Is my brain broken?” was a question uttered by more than one of my clients, denoting their felt inferiority, along with the sad fact, though in simplistic terms, of a reality of true disability beyond the affective commentary. It is the experiences of these individuals, and the families and facilities who care for them which inform my growing interest in the scientific disciplines that set out to understand, treat, to affect self-determination and healing in the face of brain trauma.

Brain injury, if experienced during the early stages of life, can create additional and unexpected problems of personality depending on the developmental age of that individual at the time of injury. Gelber and Callahan (2004) state, the sequelae of symptoms during the acute stages of injury differ in adults versus that of a child still within either the growth stage of the
brain (ages 0-5), or the pruning stage of neural networks (ages 5-13), where aspects of personality development and affective reasoning are tested and implemented. Sometimes, these problems may not be readily apparent early after an injury, but may develop in the post-acute phase, as the child “grows into” brain injury difficulty (Gelber & Callahan, 2004). Additional to time of injury being predictor of future symptomatology is the specific region in which the injury occurs. The foci of impact, or more specifically the initial impact site, along with concomitant sites affected, bear much to the determination of symptom sequelae (Al-Adawi, Powell, & Greenwood, 1998). An injury to the rear of the brain would have greater symptoms relating to that of coordination, spatial memory, and possible autonomic function if the brainstem is injured. Al-Adawi, Powell, and Greenwood go on to posit that deficits in motivation, emotional/impulse control, thought differentiation and speech would be likely if the foci of impact were to the frontal lobe, where executive functions of the brain are carried out (1998).

Although, both in history and in the present day, TBI has been associated with assault and war, large numbers of those with TBI now comprise those whose injuries were acquired through vehicular accident or repetitive concussive events in sports (CDC, 2003). Included in these numbers has been the growing diagnoses of acquired brain injury where no external foci of impact are involved, yet where cortical damage is induced by factors of either low blood oxygen for sustained periods (prenatal hypoxia, drowning, stroke) or neuronal damage due to chemical exposure (illicit drug use, environmental toxins, PCBs) or toxic heavy metal (arsenic, lead, mercury) exposure (Albensi & Janigro, 2003). When looked at from a statistical perspective, the numbers are staggering in regard to the incidence and proliferation of both acquired and impact related brain injury. According to the Centers for Disease Control and Prevention (2003):
Approximately 1.7 million people suffer TBI each year in the United States and about 52,000 people die from the injury. Estimates of the number of people who have survived a TBI range from 2.5 million to 6.5 million. The range is broad because mild TBI often goes unreported. The cost of traumatic brain injuries in the United States is estimated at $48.3 billion annually: $31.7 billion in hospitalization costs and another $16.6 billion in costs associated with fatalities. The CDC estimates the total cost of acute care and rehabilitation for TBI victims in the United States is $9 billion to $10 billion per year, not including indirect costs to families and society (e.g., lost earnings, work time, and productivity for family members, caregivers, and employers, or the costs associated with providing social services). It is estimated that over a lifetime, it can cost between $600,000 and $1,875,000 to care for a survivor of severe TBI (CDC, 2003, p. 9).

The neuromedical and rehabilitative management of TBI demands vast knowledge from multiple fields including neurophysiology, neuroanatomy, neuropathology, neuropharmacology, psychiatry, psychology, neuropsychology, orthopedics, nursing, speech pathology, and both occupational and physical therapy (Grosswasser & Keren, 2007). However, this complete intervention and rehabilitation network has only come into existence since the mid 1970s, as a response to the growing number of returning veterans of the Vietnam War who were suffering from open or closed head trauma resulting from concussive force. Notable was the consistency of post-acute symptoms, where soldiers suffered similar deficits both biological as well as psychological after returning from combat (Thurman, Coronado, & Salassie, 2007). Additionally, the assumption of the soldier, having undergone such acute resiliency training while in combat, left little for the nuanced non-combat world, giving rise to social impairments regarding
reintegration into society. Factors including isolation from family, employment difficulties, increased criminality and public policy insensitivity, proved to be an unfortunate catalyzing set of circumstances, upon which much of the new treatment protocol of biopsychosocial modeling as it applies to TBI of the last thirty years is based.

Only within the past twenty years, have non-combat related injuries to the brain been researched more extensively even though they have similar symptoms. The data gathered reveals an overwhelming commonality of symptoms; those being biological factors of chronic pain, fatigue, circadian sleep disturbance along with concomitant substance abuse side effects as a result of self medicating (O’Gorman 2006). Coupled with this, the neurocognitive impairments (attention, memory, problem solving, personality change, emotion regulation, mood disorder, psychosis etc.) were seen now as something that was comorbid, versus previous assertions that one did not necessarily precede or require the other for diagnosis. Previous to this, these factors of deficiency both affective and functional previously were often held in separation from the interpersonal functionality of the individual (Grosswasser & Keren, 2007).

Biopsychosocial: Conceptual Beginnings

The late Dr. George Engel developed the biopsychosocial model, in its primacy. Engel, an internist and practicing physician, published a paper in the journal Science in 1977 entitled The Need for a New Medical Model: A Challenge for Biomedicine. In the context of clinical medicine, Engel made the deceptively simple observation that actions at the biological, psychological, and social level are dynamically interrelated and that these relationships affect both the process and outcomes of care (Engel, 1977). Engel goes on to state the need for collaboration between disciplines, citing the thousand-plus year dichotomization of science from art, empiricism from subjectivity, and biology from psychology as being example of need for change (Engel, 1977).
The biopsychosocial perspective involves an appreciation that disease and illness do not manifest themselves only in terms of physical pathology, but also may simultaneously affect many different levels of functioning, from cellular to organ system to person to family to society.

Engel cites, as influence to his widening lens on holistic treatment, progressions in understanding learned behavior, and in part on Social Cognitive Theory, proposed initially by N.E. Miller, and J. Dollard in 1941, and later expanded theoretically and in greater application by psychologist Albert Bandura in the 1970s. It was Bandura’s theory on learning which expanded upon Skinnerian emphasis on individual behavioral reinforcement to include social modeling and imitation as proponents to new behavior generation in humans (Zimmerman & Schunk, 2003). Bandura’s theory of observational learning contained four key components required in the learning process: Attention, Retention, Motor Reproduction, and Reinforcement/Motivation (Bandura, 1997). These processes interrelate in the psychosocial learning realm, which Engel in the past saw and others today in the medical and rehabilitative sciences know now are essential to therapeutic success in treatment of TBI. Engel (1977), continues in stressing the need for a growing understanding of the health care community at large and of the importance of multi-phase care toward treating the biopsychosocial deficits of the individual and their affected families. Other than in the acute phase of treatment, where stabilization immediately, post injury takes precedent for life saving purposes, the biological, psychological and social needs of the individual must be addressed as soon as stabilization of damage has been reached.

**Biological Intervention**

**Neurobiological Acute and Post-Acute Phase Assessment**

The time period from point of injury to stabilization of neuro-biological function comprise the initial acute phase of TBI treatment. This phase specifically requires medical...
intervention, where the primary focus is to assess the extent of damage, and to provide a finite map toward stabilization of possible function. The *Glasgow Coma Scale* (GCS), developed by Teasdale and Jennet (1974) has become the standard in first line assessment of overall measure of patient consciousness, post accident. The scale comprises three tests: *Eye*, *Verbal* and *Motor* responses. The three values separately as well as their sum are considered. The lowest possible GCS (the sum) is 3 (deep coma or death), while the highest is 15 (fully awake).

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<td><strong>Eyes</strong></td>
<td>Does not open eyes</td>
<td>Opens eyes in response to painful stimuli</td>
<td>Opens eyes in response to voice</td>
<td>Opens eyes spontaneously</td>
<td>N/A</td>
<td>N/A</td>
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<td><strong>Verbal</strong></td>
<td>Makes no sounds</td>
<td>Incomprehensible sounds</td>
<td>Utters inappropriate words</td>
<td>Confused, disoriented</td>
<td>Oriented, converses normally</td>
<td>N/A</td>
</tr>
<tr>
<td><strong>Motor</strong></td>
<td>Makes no movements</td>
<td>Extension to painful stimuli (decerbrate response)</td>
<td>Abnormal flexion to painful stimuli (decorticate response)</td>
<td>Flexion / Withdrawal to painful stimuli</td>
<td>Localizes painful stimuli</td>
<td>Obeys commands</td>
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(From Teasdale & Jennett, 1974)

Previous to the GCS, assessment of trauma was left to the subjectivity of the first responder, which carried with it a high degree of inaccuracy, and the possibility for subsequent further damage to the individual due to inadequate acute response. From the point of injury and subsequent stabilization of biological function, this scaling device for cranial trauma assessment,
standardizes a baseline for post-acute care direction, where more finite measuring of specific intact function through can be assessed and implemented (Grosswasser & Keren, 2007).

**Neuroimaging**

Through the methods of scientific inquiry and measurement during the span of the Late nineteenth to the mid-twentieth century, it was understood that the brain was an organ, comprised both of electrical as well as vascular processes; where one is the messenger, and the other, the fuel for the multitude of messages being sent within a hundredth of a second. With much to the credit of advances in neuroimaging and measurement, a more defined understanding of the brain’s physical complexity has been achieved. We now can say with certainty that the brain consists of finite physical parameters. The brain in normal functioning capacity is comprised of 100 billion neocortical neurons, with .16 quadrillion synapses between them, along an electrical pathway, which would total 180,000 kilometers if strung end to end (Pakkenberg & Gunderson, 1997). This network firing at nanoseconds per square millimeter sits within a 3.5 pound environment consisting primarily of water, lipids, protein, carbohydrates and inorganic salts, which require 25% of our total oxygen intake, and requires 70% of the bodies overall glucose in order to carry out these tasks. With such a high biological ratio of need, versus the overall usage by the rest of the body’s systems, the complexity of any injury to the brain, both vascular and electrical, would have its effects resonating throughout the individuals’ entire system (Grosswasser & Keren, 2007). After acute stabilization has been achieved, measurement of both electrical and metabolic processes of brain regions would then be essential to ascertaining overall function as it relates to injury in the post-acute phase of treatment.

Technological advancement of accurate neurobiological imaging, essential to accurate prognosis and progress of post-acute treatment, has evolved considerably within the last twenty
years. So too, the specializations in medicine and psychology have evolved with these technologies; giving rise to the field of neuroscience, a relatively recent boon to the field, which empirically confirms or denies subjectivity in the professional, and avails a better treatment plan for the patient (Thornton & Carmody, 2008). Previous to these advances, imaging was relegated first to use of x-ray, which gave a generalized view of the brain's physical properties, limited mainly to show diffuse vascular anomalies via static view silver emulsion plates by the physician. Though essential in a first-line assessment of trauma, the accuracy of determining specific trauma and subsequent deficit would still be relegated to use of the GCS, and x-ray, while in the post acute phase, the use of affective and motor function assessments to determine treatment progress. The timeframe for measurement would be in a longitudinal scale over the course of a patient's healing, irrespective of possible psychosocial deficits, which may yet be discovered over the lifetime of the patient. Specificity in brain regions affected, and better still, those which have remained intact and have function potential, were still an unknown until the advent of technologies which provided a more accurate portrayal of these specific deficits and or potentials (Brown, 2006).

Most notable technologies for measurement of brain trauma utilize graphical representation of general brainwave activity, site-specific neuronal activity, and vascular flow to those regions affected. Electroencephalography (EEG), and the more advanced; Quantitative Electroencephalography (QEEG), measure electrical waves of activity along the scalp via electrodes to give general brainwave activity corresponding to a specific region (Figure 1), Functional Magnetic Resonance Imaging (fMRI) where pictorial representation of neuronal activity in a specific brain region can be seen in real time (Figure 2), and Single Photon Emission Computerized Tomography (SPECT) where accurate representation of brain metabolism and
blood flow is represented via 3D modeling (Figure 4). These measurement methods have become indispensible in the assessment of function deficits through graphic representation of specific disorder, both vascular and electrical subsequent to injury, and inform better all areas of medical, psychiatric, psychological and psychotherapeutic intervention.

MRI, or Magnetic Resonance Imaging measures brain electrical activity through taking “slice” images of the brain, usually at the thickness of 5 mm per slice. These slices are then pieced together to form a more complete static image of the brain. The difference between standard MRI, and the more advanced Functional MRI (fMRI), is that the “slices” are read via computer in rapid succession, at a constant rate while the patient is lying in the scanner, whereby, a “flipbook” is generated of the real time functioning of the brain. This method of imaging is especially informative to the nature of a patient’s in vivo deficiencies when a stimulus is applied through having the patient perform a task while scanned, to measure electrical anomaly, or deficit in the presence of TBI (Thornton & Carmody, 2008). Most notable in these technologies, are their abilities to be used throughout treatment of the individual. Technological advances in brain imaging give physicians a better window into specific electrical pathways affected versus healthy controls (Figure 3).
Fig 1. EEG waveforms and correlating electrical potentials, using 36 channel sensor unit (Niedermeyer E. & Da Silva F.L. 2004).
Fig 2. MRI slices displaying electrical over-activity in right dorsolateral prefrontal cortex of TBI patient with symptoms of impulsivity and anger control (Scheibel, et al. 2003).

Fig 3. Differences in electrical pathway stimulus in primary (M1) and secondary (SM*) motor cortex between hemispheres (Hauk, Johnsrude & Pulvermiller, 2004).
Fig 4: SPECT imaging examples of TBI

SPECT images of normal brain, 23 year old female.

26 year old rollerblade accident, no helmet executive function problems, suicidal ideas

16 year old fell down stairs at age 3, school failure, aggression in jail for rape

48 year old male roofer who fell 25 feet off a roof. Problems with speech, listening, memory, concentration and temper problems. His wife divorced him (Amen, 2004).
Imaging Data Informing Psychiatric Intervention

Deficits, which pose significant clinical difficulties within treatment of symptoms following TBI have, with the help of technology been better assessed with approaches more tailored to specific needs. An example of assessing motivational deficits in individuals sustaining damage to the frontal cortex would manifest symptoms of apathy, indifference, lack of verbal output, lowered libido, and poor initiation (Arcinieagas & Silver, 2006). Some facts on the prefrontal cortex (PFC) are that it is the key area of executive or supervisory processes. Its function also consists of analyzing and directing behavior with respect to desired outcomes or reward (Norman & Shallice, 1986). Put specifically to an example, if an individual has a goal, but has difficulty generating or activating goal directed behavior, than he or she might exhibit symptoms related to, but not directly products of that difficulty, and in turn, might seek reward outcomes, which may exacerbate initial deficits (Al-Adawi, Powell, & Greenwood, 1998). This holds the patient within a loop of a pseudo-depressive syndrome and as well might misinform the attending psychiatrist as to the real deficit to be addressed.

Past psychiatric data indicated an increase of the neurotransmitter dopamine in the presence of reward, and that in turn would set the goal directedness of the behavior, and its subsequent continuation. Using two developed measurement tools based on imaging of the dopamine-reward model, Al-Adawi, Powell, & Greenwood developed neuropsychological tests specific to measurement of motivational deficits, as well as subsequent dopaminergic deficits in the case of PFC damage (1998). Using the Percent Participation Index (PPI), developed by Al-Adawi, Powell, & Greenwood to determine a patients overall clinical incentive response to the Card Arranging Reward Responsivity Objective Test (CARROT), a test used to measure deficits in incentive motivation through comparison of speed in a psychomotor task with and without
financial reward in 167 patients with mild to moderate TBI to the PFC (1998). The findings of the test were based on subjects with the prescribed dopamine agonist drug Bromocriptine, and those without. Those with the dopamine inducement showed better than 71% increase in CARROT scores, with subsequent higher interest in clinical participation in goal directedness. These findings were corroborated through the use of neuroimaging to show increases in PFC activity while under test (Al-Adawi, Powell, & Greenwood, 1998).

Another example of neuropsychological testing informing better allopathic intervention involves the role of assessing modulated activity in memory formation and retrieval in patients with TBI to the left temporal basal forebrain (Leon-Carrion, Dominguez-Roldan, & Murillo-Cabesas, 2000). This area is associated with memory and is dense with acetylcholine receptors, the neurotransmitter associated with memory formation. Through the use of SPECT imaging to determine regional cerebral blood flow in that area affected, as well as EEG to specifically measure electrical activity, the researchers administered CDP choline, a precursor to the neurotransmitter acetylcholine, to determine effects using concurrent neuropsychological training meant to increase working memory and learning. The research findings show that neuropsychological testing using the Benton Visual Retention Test with the group administered CDP choline, proved upwards of 15 points standard deviation from the placebo group (Leon-Carrion, Dominguez-Roldan, & Murillo-Cabesas, 2000). The results show, first, that patients with chronic severe memory deficits present a deficiency in the left basal temporal area, and that an activation of that area increases working memory. The second and possibly most important finding was that a patient with specific memory deficits can increase retention if provided increased support neurochemically, as well with specific neuropsychological task to increase memory capability.
Pharmacotherapy Considerations

Pharmacotherapy of posttraumatic cognitive impairments requires consideration far and above normal intervention procedure. Three types of induced trauma are: hypoxic, or low oxygen to brain cells, cytotoxic, or that which chemically destroys brain cells, and axonal projection, or those where there is high speed, long duration deceleration injury common in motor vehicle and sports related TBI (Arciniega & Silver, 2006). In all cases, there is a cascade of injurious processes to the delicate neurotransmitter balance within the brain from within. During normal operation, the amino acid glutamate serves as the primary excitatory neurotransmitter to all neurons in the central nervous system. It activates receptors in a controlled fashion, allowing for measured transmission of specific neurotransmitters (dopamine, serotonin, norepinephrine, gaba, acetylcholine) to their receptors for uptake. In the face of either hypoxic, cytotoxic, or axonal projection injury, the activity of glutamate becomes increased in other areas of the brain. This over activity, otherwise known as excitotoxicity destroys neurons by increasing an influx of calcium, which initiates oxidation of the neuron, eventually destroying it in rapid fashion (Sugden, Kile, & Farrimond, 2006). Traumatically-induced glutamate excitotoxicity lies at the heart of TBI whether through hypoxia, cytotoxic exposure, or axonal projection, in that both injuries trigger this cascading response, when in great volume, causes the resulting decrease in regional brain function (Arciniega & Silver, 2006).

Within biopsychosocial modeling, pharmacology, has taken on a new face of neuropsychiatry, where combinations of pharmacologic therapies and neurocognitive rehabilitation are seen no longer as mutually exclusive, but linked inextricably. Although a thorough medical history is necessary for the prescription of medication for the common contributors of posttraumatic problems, the consideration of the synergistic cascade of action in
the brain is of utmost importance (Arciniegas & Silver, 2006). Common in earlier treatments were further neurotoxic complications due to polypharmacy symptom interventions. For example: a TBI patient would possibly be given an anticonvulsant (phenytoin, carbemazepine) for posttraumatic seizures, along with an antipsychotic (haloperidol, thoridiazine, etc.) to decrease limbic over-activity, while also given a psychostimulant (methylphenidate, amphetamine) to combat extreme lethargy; a common side effect with anti psychotics. Added to this, the likely continuance of narcotic pain medication for headaches, as well as possible others for premorbid conditions such as high blood pressure and the like, decreased livelihood, and shortened the life spans of many TBI patients due to neurotoxic interaction (Kim & Ko, 2006).

With the advent of cross-communication between disciplines of neuroscience, nutrition and the medical community, understanding of root actions of cytotoxicity in the brain informs a better treatment protocol. For example, Arciniegas and Silver (2006) cite research on glutamate channel agonists such as Memantine, used to modulate calcium oxidation in the neurons of the hippocampus, as well as gaba antagonists such as Modafinil for increased glutamate release. There is also citation of the use of over the counter nutritional supplements, citing CDP choline’s role, as well as other neurotransmitter precursors, common in certain foods, and pharmaceutical grade supplements, which may provide equal if in select cases better results with fewer symptomatic side effects. Arciniegas and Silver’s MRI and neuropsychological data uncover that these act as double or in some cases triple-action agents performing in service to cause in biochemical deficiency at the cellular level versus a symptom palliative (2006).

Pain Assessment

In the majority of TBI sufferers, pain oftentimes presents as both first line symptom in both acute and post acute phases of treatment. Traumatic brain injury is frequently associated with
painful complaints in patients. Persistent pain may arise from a combination of physical and psychological factors, and while pharmacotherapy provides a temporary relief, a long-term solution including a multidisciplinary approach ensures better outcomes for the patient (Tyrer, 2003). Pain is a subjective experience, associated with real physical tissue damage. Acute pain occurs at the time of injury, while chronic pain is maintained after the healing process has been completed, and oftentimes associated with depression, lassitude, and lack of motivation (Tyrer, 2003). The incidence of pain following a TBI varies according to the interval of time following brain injury. Tyrer notes that headache is reported as occurring in 80% of patients at some stage in the recovery process, where 30% complain of additional pain throughout the body more than 6 months post injury. Acute pain often accompanies the initial stages of brain damage, particularly in more severe cases of axonal projection TBI, as multiple injuries may be present. Conversely, however injuries to the head may be overlooked in the clinician’s concerns for injury to other parts of the body, this occurs most often in closed head injuries (Tyrer, 2003).

Since pain is a wholly subjective experience, assessing TBI patients with chronic pain aims to discover the origin, site, and intensity of felt pain on that basis. Both physical and psychological factors affect the perception and long-standing consequences of pain. A simple 1-10 scale of intensity gives the clinician ample idea of the patient’s subjective experience of pain, while more finite questions might uncover pain strength and duration, and perceived loss of biomechanical function due to pain. Confounding factors in the proper assessment of pain affect proper pharmacotherapeutic intervention. The prevalence of depression following TBI varies from 5% to 25% depending on the population studied (Fleming & Ownsworth, 2006). In patients with persistent pain, depression is more frequent; the greater the degree of comorbidity, the greater the impairment of function. It is important to differentiate between depressive
syndrome because of loss of mobility, and depressive illness for prescribing purposes, where an appropriate agent can be properly administered (Payne 2000). Additionally, Posttraumatic Stress Disorder can be another confounding element in accurate assessment. As many incidents of brain injury occur in either threatening or trauma laden environments, especially in combat situations, and other intense sudden impact emotion-laden scenarios, pain as a cofactor to PTSD must be ascertained for proper treatment (Tyrer, 2003). A multidisciplinary approach to pain management requires healthcare providers from several disciplines. A typical assigned team for an individual would likely involve a pain management physician, a psychologist, a clinical nurse specialist, and a physiotherapist. Additionally vocational advisors as well occupational therapists, and neuropsychologists may become a part of the treatment spectrum as management needs arise (Fleming & Ownsworth, 2006).

**Neuroplasticity**

*Neuroplasticity* is a neuroscientific term applied generally to the ability of the brain and nervous system in all species to change structurally and functionally as a result of input from the environment (Bach-Y-Rita, 2003). Plasticity occurs on a variety of levels, ranging from cellular changes involved in physical development and motor acuity to large-scale changes involved in *cortical remapping* in response to injury (Albensi & Janigro, 2003). During most of the 20th century, the general consensus among neurologists and practitioners held that brain structure is relatively immutable after a critical period during early childhood. This belief has been challenged by new findings, revealing that many aspects of the brain remain plastic even well into adulthood (Shaw & McEachern, 2001). Evidence has been accumulating that the synapses between neurons can reorganize extensively after damage, and that reorganization can be obtained even many years after the trauma with appropriate rehabilitation. When neuroplasticity, or more specifically
*synaptic plasticity* is viewed within the biopsychosocial lens of intervention, the emergence of remapping cortical function requires synchrony of all three areas of the model (Bach-Y-Rita, 2003).

**Historical Concepts of Plasticity**

The concept of neuroplasticity has had anecdotal affirmation as far back as the early 20th century, when observation in both science and in the growing field of psychology lent much to the development of technology and evolved into a more finite understanding of the brain's processes today in the 21st century. Early vestiges of plasticity in adaptation can be found in the work of Charles Darwin in the late 19th century. Darwin proposed the formative notion that there exists an inherent mechanism of adaptation in life forms, and that adaptation is indeed a changeable process in regard to environmental influence and internal feedback mechanisms. Darwin’s work on the whole was largely based on observation of naturalistic behavior of animals, yet his published 1872 work, *The Expression of Emotions in Humans and Animals*, introduced the concept of Human and animal behavior as being the product of a life-preserving stimulus response. These evolved actions, tested and retested become behaviors for survival when kept; others conversely are extinguished for their needlessness (Bowlby, 1990). This theory of evolution of behavior, and the understanding of behavior in terms of natural selection of those behaviors, helped to inform the medical community of the day, and was a catalyzing foundation to the relatively new discipline of psychology, unleashing the cascade of theoretical positions on the bases of human cognition.

The notable work of Sigmund Freud expanded on the theory of inbuilt mechanisms of determined behavior. In his work *Beyond the Pleasure Principle* (1920), Freud asserted that human instinct is a product of human *life drives* (survival, propagation, hunger) and adaptation, or
maladaptation, a result of human death drives (conservation of instinct) (Freud, 1920). “Emotion occurs,” Freud states, “when one is beset with the conflict between the two, and adaptation of behavior evolves to meet that influence” (27). Though seen by some, as an exercise in ambiguity by Freud, and that Freud himself did not include therapeutic protocol in the work specific to brain trauma, it does speak clearly of the concept of plasticity, in that it infers inherent adaptation and growth from influence as its subtext.

Alfred Adler, a colleague of Freud, physician and founder of individual psychology, discusses the aspect of adaptability to stress and stressful stimuli through organ systems. In a passage of his work, Organ Inferiority and its Psychical Compensation (1917), Adler both acknowledges and disagrees with Freud’s assertion of pathologic behavior as being “simplistic in his valuation of the basis for problem drives as inbuilt unconscious, yet necessary in understanding that there is a compensation at hand” (59). Adler, the physician, stressed the interrelatedness of physical dysfunction with psychological compensation, and that “disorder only comes when an improper adaptation or solution - a conscious choice - is used toward the safeguarding from organ inferiority” (60). He further says, “the result depends on the capability of development of the fundamentally inferior superstructure, and on the congenital growth of cerebral cells in ‘moral compensation’ to the inferiority. In the case of overcompensation response, the motor portion of the compensating superstructure arise all form of neuroses” (61).

Though formative in their approach, Adler and Freud both addressed adaptation of consciousness as being a reflexive act in humans. The concept of plasticity of thought based on environmental and psychogenic forces, though new in its assertion to science, became the foundation for what would be a lasting mainstay in further research and therapeutic intervention to come.
Though still non-technologically based, yet important in its uncovering of plasticity and adaptation, were the works of behaviorists in the early to mid 20th century. The work of notable behaviorists Ivan Pavlov in the 1920s, with his famous experiments in classical conditioning, as well as behavior modification experiments performed by B.F. Skinner in the 1950s, related reinforcement strategies toward voluntary use/non-use of behavior (Dayan, Kakade, & Montague, 2000). Also worthy of mention is the work of developmental psychologists John Bowlby and Mary Ainsworth in the 1960s researching human adaptive or maladaptive responses in social attachment patterns. Along the evolution of theory, the concept of systems theory and cybernetics, headed by notable theoreticians Bateson, Bowen, Assagioli and others, helped further expand an understanding of interrelatedness between instinct and the function of acquired behavior in the service of social learning. The concept of cybernetics, as Dayan, Kakade, and Montague (2000) point out, is “the last piece to the puzzle bringing together the micro-organization of the human, in relation to its macro-influential stimuli surrounding it” (1218). It is “the human organism’s drive toward homeostatic equilibrium of communication, self organization, and response regulation is at the heart of modern neuroscience” (1218). It is these concepts, paired with measurement technologies, upon which the biopsychosocial modality is based, and more specifically in the treatment of traumatic brain injury.

**Synaptic Plasticity in TBI**

While plasticity, as its presented in most research literature regarding rehabilitation, often denotes functional improvements after brain injury, it may also generate unwanted effects, such as spasticity, seizure, and aphasia related to the specific regions affected (speech, memory, motor), as well as general diffuse functions involving more regions of the brain (motivation, mood, sleep) (Bach-Y-Rita, 2003). The complex relationships within TBI, including the alterations in synaptic
plasticity and cognitive impairment, are slowly being unraveled. Recent research has shown that recuperative processes are more apt to be successful, whether the result of impact is focal or if the damage is diffuse among brain regions of children versus adults (Levin, 2003). Levin goes on to say that instances of focal injury in infants (falls, etc.), diffuse axonal injuries (sudden acceleration/deceleration, shaking), as well as hypoxic instances (oxygen deprivation, malnutrition) have developmental impacts on formation of language processing, autonomic regulation and executive processing. Longitudinal research has shown that though the physical recuperation is relatively rapid, the overall effects are more devastating, in that during the period between 3 to 36 months, the brain is undergoing an essential process of synaptic growth in the formation of gray matter, as well as the formation of the essential protective fatty acid myelin around nerve cells in the formation of white matter (Levin, 2003). As the child reaches the age of five, where formative language acquisition and social feedback processes are necessary in development of later stages of introspection and metacognition in the teenage years, a disruption of normal processing due to TBI is apparent in contrast to one’s peers which, in turn can have detrimental stigmatizing effects, giving rise to felt inferiority and possible affective disorder. This, as Levin points out, is a process of plasticity in the forming brain, where compensation of neural use is not the factor, but rather wholesale gaps in growth of receptors are present (2003). These gaps have the probability of dramatically altering social learning and personality development as the child grows into the world around him or her, possibly unaware of any instance of past injury because of its occurrence at such an early growth stage (Levin, 2003).

**Mechanisms of Plasticity**

The well studied mechanisms by which homeostatic adaptation, post-injury are employed to continue life in humans is a remarkable process giving credence to Darwin’s initial assertions.
The autonomic nervous system operates via two pathways toward these ends: *afferent*, or impulses directed toward the central nervous system (CNS), and *efferent* or those signals directed away from the CNS (Taub, 2004). After injury occurs to the CNS, the initial deficit in behavior, perception, or cognitive ability is frequently followed by a spontaneous recovery of function. The difference in qualitative pre- and post-morbid dysfunction of an individual certainly factors into the definition of “spontaneous recovery”, yet adaptive processes of behavior and physical function give ample evidence to plasticity overall. Previous to the 1970s, commonly held belief was that recovery is solely reliant upon afferent/efferent autonomic processes engaged in recovery (Laatsch, Little, & Thulborn, 2004). Present-day fMRI and SPECT information reveals a much denser, more multi-modal set of systems at work in regard to recovery of function in TBI individuals. At the heart of recent discoveries is a greater understanding of brain function in *synaptic transmission, extracellular space volume fraction, and neuronal multiplexing*. Bach-Y-Rita lends a useful way to visualize the three mechanisms: *signal type, quality of transmission, and complexity of signal*. The coordination of recovery processes, both afferent and efferent, are dependent on these three mechanisms. By understanding which mechanism is deficient, more finite and appropriate therapy can then be administered (Bach-Y-Rita, 2003).

Understanding the mechanisms by which these and other changes occur should, as Bach-Y-Rita continues, inform clinicians as to therapeutic direction (2003). The research of Bach-Y-Rita proposes two mechanisms by which neural transmission and plasticity are activated. There is, as Bach-Y-Rita states, ample proof that information is transmitted by both *synaptic transmission*, and by non-synaptic transmission, or *volume transmission* (2003). Bach-Y-Rita, a professor and researcher, uses an example apropos to his profession to describe the two processes where volume transmission is like students leaving a classroom, scattering to different parts of the
school (receptor sites). They flow out into the halls and the grounds between buildings (extracellular fluid), where they mix with other students (neurotransmitter molecules) from other classrooms. They walk (diffuse) to their specific classrooms (receptors), which they enter (bind). In contrast, synaptic transmission students would be propelled along enclosed walkways connecting the point-of-origin classroom with each target classroom (2003). Neural mechanisms related to processes such as arousal or motivation, as well as sleep, hunger and mood, are related to volume transmission, where diffusion across brain regions affects complex coordinated processes. Activities that require great selectivity and rapid initiation, such as fine motor tasks (playing a musical instrument, writing, sport activities, etc.) are dependant on synaptic transmission for their purposes.

The two processes of transmission, intermixed with extracellular space-volume, or the number of active neurons in a given area, give those in the rehabilitative fields much information in regards to how the brain multiplexes. Multiplexing is the third dimension of brain process, which consists of the multiple co-functioning of neurons and fibers. (Bach-Y-Rita, 2003). Bach-Y-Rita cites studies which demonstrate the multi-functional aspects of sensory cells, namely in the brain’s ability to differentiate and adapt use of such cells. Lesions, or temporary suppression of a sensory input, can unmask multiple sensory inputs which may be mobilized on motor and sensory recovery (2003). Evidence of this unmasking and adaptation was seen in fMRI studies in the learned ability of human subjects to read Braille after loss of eyesight. The studies demonstrated a re-routing of tactile processing pathways usually linked to somatosensory areas, to occipital cortical regions usually reserved for visual shape discrimination (Albensi & Janigro, 2003).
Studies have shown that this multiplexing happens relatively quickly following injury. Bach-Y-Rita goes on to cite the possible affective or emotional changes in an individual post-injury. Injury, Bach-Y-Rita states, which initiates a rapid, concurrent and consistent change in somatosensory input along the brainstem, can affect thalamic and limbic remapping, where gross alterations in personality and intrapsychic awareness result due to the speed at which the changes occur (2003). This aspect of neuronal plasticity change, causing great shifts in affect and behavior, hearkens to the famous account of Phineas Gage, neuroscience’s first and most famous patient regarding brain injury and plasticity. In April 1848, Gage, a railroad worker, had sustained a massive brain injury by way of a tamping iron which entered his lower left cheek and exited through the right top of his skull. This was a devastating injury, which would be understood in the present to be a massive axonal projection injury to right and left prefrontal cortices (Damaiso, 1994). What was remarkable to physicians and those that knew Gage at the time was his relatively rapid physical recovery, where in a matter of months he was able to return to his job as a railroad construction worker. Equally remarkable, and germane to the topic of neuroplasticity, were the resulting profound changes in personality and cognitive function seen. Gage, who previous to the accident was by accounts an affable, agreeable, and conscientious man, became anger prone, socially apathetic, and impulsive following the accident. Though an account of historical medical and psychological phenomena, Gage’s story is especially prescient in the present day in affirming the process of neuroplasticity, in which rapid recovery can occur through multiplexing new neural pathways. Affective changes can also occur as a result of plastic remapping of sensory and limbic areas (Damaiso, 1994). As a tool for clinicians in the rehabilitative sciences, understanding the processes of neuronal plasticity through the systems
pathways that engage it informs better treatment protocols in functional recovery for brain-injured individuals.

**Rehabilitative Strategies**

**Psychophysical Interventions**

After injury to the central nervous system, cortical changes and plastic compensation are readily seen, yet specific therapeutic strategies addressing physical deficits after TBI are necessary within the treatment protocol. One specific treatment, Constraint-Induced Movement Therapy (CIT) built from models incorporating *forced use* has shown to be successful behavioral intervention in both stroke and TBI, in the rehabilitation of damaged neural pathways to one or more limbs (Page & Levine, 2003). CIT has theoretical basis in the work of neuroscientist Edward Taub, who carried out experiments on rhesus monkeys (Taub, 2004). Taub discovered that when the forelimbs were deafferented, where the sensory input from the limb to the brain was disconnected, a “learned non-use” emerges, in which the animals stop using their forelimbs in the free situation (2004). However, application of operant conditioning, in which the deafferented limb use is forced, overcomes learned non-use over a period of time. It is additionally reported by Taub that humans with TBI or stroke exhibit learned non-use of the more affected limb (2004). CIT’s success is based on the simple principle of constraining the less affected limb by either a cast or immobilizing brace during waking hours, and allowing the more affected limb to be free to negotiate the challenges of normal function. Studies conducted on a group of stroke patients, in a 10-week trial showed that, 1) there were measurable increases in performance of affected limb, and 2) fMRI tests proved that there was an induction of cortical remapping to the affected limb (Page & Levine, 2003). This use-dependant brain reorganization shows promise in rehabilitating
one facet of the TBI sufferer’s symptom sequelae, resulting in strengthening a felt reliance in the patient through this psychophysical intervention.

Biofeedback-assisted relaxation training has provided well-documented and empirically validated benefit for a diverse population of medical patients. Also notable is its efficacy in diminishing behavioral obstacles through increasing physiological awareness for brain-injured patients. Holland, Witty & Lawler (1999) note an increase in anxiety, as well as autonomic deregulation to manage that anxiety as a common symptom in many TBI sufferers. The process of biofeedback essentially monitors, through sensors, galvanic skin resistance (sweat gland activity) and heart rate in real time and is represented to the patient either as a color or an object which responds to increases and decreases in activity. Biofeedback’s success lies in its ability to cultivate a learned relaxation response through mindful means. For the TBI sufferer, somatic disconnection can be problematic in therapeutic rehabilitation, both affective and physical, and the frustration in having lost control of past function can be devastating to the patient. Biofeedback can be useful in mediating resistive anxiety in treatment, as well as used conjointly in cognitive remediation strategies for behavioral skills based intervention (Holland, Witty, & Lawler, 1999).

**Applied Cognitive Therapies**

**Cognitive Remediation**

The evolution of cognitive remediation (CR) as a treatment strategy in TBI has followed, in parallel, the advances in neuropsychological data regarding plasticity and deficit compensation in brain-injured individuals. *Cognitive remediation* is an umbrella term for those treatment strategies which involve rehabilitating lost cognitive skills, whether functional in the form of memory loss and attentional difficulties, or perceptual, in terms of affective self perception and
awareness (Fleming & Ownsworth, 2006). Speech pathology, occupational therapy, and neuropsychology are typical areas where cognitive remediation is practiced. Though previously held in separation from cognitive remediation strategies, psychotherapeutic intervention is now considered a part of these strategies, as it informs an individual’s emotional growth, based on skills gained through more concrete remediation interventions (Gordon & Hibbard, 1992). For the sake of separation between gains of concrete skills and emotional-affective skills, only those concentrations related to concrete function deficit will be discussed in this section, with further expansion on complimentary psychotherapeutic strategies to follow.

The general consensus among the research and therapeutic communities holds that TBI has within it very specific factors of cognitive dysfunction: memory, attention and problem solving, with subsets of problem solving in TBI patients, including concreteness, rigidity in thinking, inefficient concept formation and poorly regulated behavior (Ylvisaker, Shaughnessy, & Greathouse, 2002). Cognitive remediation strategies include a multi-modal approach toward these ends, based on the individual’s specific deficits. Neuropsychological assessments have been the standard by which intervention strategies are based. Based on a review of literature on the associations between neuropsychological measures and outcomes, many neuropsychological tests have a moderate level of validity when predicting everyday cognitive functioning. Standardized measures assessing memory, such as the *Wechsler Memory Test III* (1945), the *Wechsler Adult Intelligence Scale* (1955), assessments of attention, such as the *Stroop Test* (1935), and problem solving tests such as the *Category Test* (1976) and the *Wisconsin Card Sort* (1993) have proven useful in assessing deficits, yet proved problematic in assessing specific gains seen throughout treatment (Thornton & Carmody, 2008). In the area of biopsychosocial intervention, cognitive remediation models must employ evidence-based practice of assessment
and recovery throughout the individual’s life in all three phases (Ylvisaker, Shaughnessy, & Greathouse, 2002).

The growing agreement of providing contextualized intervention by speech pathologists, neuropsychologists, and occupational therapists is seen now as the apogee of effective treatment. Reason indoctrination, the former model for cognitive remediation, had a low success rate due to the lack of applied learning in TBI (Gordon & Hibbard, 1992). Mark Ylvisaker, a prominent speech pathologist and researcher suggests that four conditions must be met for transfer of training to be likely: (1) encoding specificity, (2) adequate organization of the to-be-learned information or skill, (3) discrimination ability, and (4) a psychological set that predisposes the person to seek transfer opportunities (2002). Taken together, these four factors give ample argument for a contextualized approach toward cognitive training.

Cognitive remediation strategies seek first to teach in context those executive function losses in TBI sufferers. The *executive system* can be loosely characterized as the processes needed to formulate goals, plan ways to achieve them, and to execute that plan. When the executive function is deficiently operating, limbic (emotional) override can ensue, causing disruption in self-regulation (Page & Levine, 2003). The goal of cognitive remediation is to develop “executive system routines”, or thinking goals and strategies, as well as “self regulation routines,” or emotional and behavioral strategies in tandem to meet those ends (Ylvisaker, Shaughnessy, & Greathouse, 2002). Ylvisaker, Shaughnessy, & Greathouse (2002) cite an example as effective context based executive system routine with a child of kindergarten age who had sustained a TBI at age two. The child’s behavioral difficulties had grown and escalated to a point where, when given a task in school, she would immediately take it on as she saw her peers doing it. As she discovered she could not complete that task, her reaction would become
explosive, and other adjunct behaviors not fitting her developmental age would emerge. Using an executive system routine requires, 1) clear presentation of the task, 2) self-reflection by the child as to the difficulty of the task or simply stating the perceived difficulty, 3) discussing strategies to succeed in the task, 4) providing contextual support only in that task to avoid confusion, and finally, 5) reviewing all completed tasks, highlighting difficulty level in relation to the child’s abilities and what they did to succeed. Over the year, Ylvisaker, Shaughnessy, and Greathouse state, the girl took on more and more of the responsibility for identifying a task’s difficulty level, the plan for success, and the review of past successes (2002). This, as the researchers continue, uses self-determination with self-reflection to form a feedback loop of an executive function routine, of which she continued to employ to great effect, reducing emotional override and discouragement while increasing resilience (Ylvisaker, Shaughnessy, & Greathouse, 2002).

Within cognitive remediation in adults and children with behavior issues, the main focus is on guiding effective future behavior through means, which befit the deficiencies in executive function. Since memory loss is a consistent issue with TBI, the disconnection of past accomplishment or failure becomes apparent when the behavior is continued without reflection (Freed, 2002). The established protocol for behavior modification in non-TBI patients, focuses on consequences and contingency management, utilizing projection of future problem solving measures based on consistent problematic stress response (Freed, 2002). However children, adolescents, and adults with TBI often respond inefficiently to these common behavior plans because of deficiency in projecting based on memory. Damasio (1994) cites the ventral prefrontal areas of the brain as critical for connecting memories of what we have done (conscious or unconscious) with our “gut level” reactions to these events (somatic markers). Since damage to that area would cause an inability to do so, behavior modification strategies based on memory
of consequence would be ineffective in guiding future behavior. Instead using antecedent control procedures, or what has been known collectively as positive behavior supports with TBI, remediation is more effective. The general goal is to put proactive supports in place, such as a good biopsychosocial therapeutic structure, so that, 1) negative behavior based on impulsiveness is infrequently triggered, 2) negative behavior becomes inefficient and unnecessary for the person in everyday routines, and 3) because of the antecedent supports, the child, student, or adult develops habits of behavior that are positive and generally satisfying (Damaiso, 1994).

In keeping with specific context based intervention, the aspect of rigidity in thinking being an issue for the TBI sufferer may prove an obstacle for specifics being applied to other aspects of the person’s everyday life. On that point alone, the question that if change in everyday functioning is the goal of cognitive intervention, and the efficacy of the treatment is to be determined by whether or not this goal has been obtained, then generalization must be built in to the training program (Gordon & Hibbard, 1992). Thus, the training curriculum must incorporate sufficient bridges between the skills being remedied and real-life functioning. Ciccerone (1989) expounds on the needs of individuals and the methods of cognitive skills training in four key points, which have direct instruction to all facets of CR practitioners (speech pathology, occupational therapy, psychotherapy), as to the rubric of “generalization” being both the goal and the outcome of cognitive remediation:

1. The carryover of training strategies to diverse settings within the person’s natural environment must be carefully planned and evaluated within the course of treatment. As training proceeds, the stimuli and materials used in remediation must be altered so that they successively approximate everyday functions.
2. Individuals need to be taught the mechanisms that underlie both their cognitive failures and their improved cognitive performances in everyday life. This provides the foundation for teaching the individuals to regulate their own behavior. Clients can be provided with scripts or lists, and be taught inductive or deductive reasoning strategies.

3. Self regulation or self-talk can only be used to enhance generalization when the clients have a sufficient awareness of their cognitive difficulties and how they manifest in day-to-day functioning.

4. Because individuals learn more slowly after brain injury, clients need to be given an adequate number of trials to learn new skills. They need to repeatedly demonstrate successful performance both within and between sessions to assure consistency and retention of new learning. One success on a given trial or a given day of training does not mean the to-be-learned skill has been integrated into the person’s repertoire.

5. Because abstraction skills are often impaired in brain-injured individuals, the failure to generalize strategies taught in cognitive remediation to everyday functioning may be the result of these impaired higher-level abilities. For example, the individual may be unable to decipher the similarities in task response demands across situations, a skill that is necessary to ensure the generalization of new learning. Only through repetition of learning in multiple situations and the repeated demonstration of functional competence in these different settings can the therapist ensure that the learning and generalization have taken place.
**Integration of Self-Perception**

After a brain injury, individuals are faced with the often-difficult task of attempting to integrate three separate perceptions of the self: who they really are (current cognitive and behavioral functioning), what they believe they are (premorbid cognitive and behavioral functioning) and what they want to be (future assumptions about their cognitive and behavioral functioning) (Freed, 2002). The relationships among these perceptions of the self are not static and are quite often at odds in brain injured individuals. Ciccerone (1989) goes on to state that minimizing the discordance between the three self-concepts becomes a major goal in cognitive remediation. The extent of the discrepancies in self-image vary during the course of CR and are dependant on an interaction among 1) the severity of cognitive deficits, 2) the patient’s level of awareness of their limitations, 3) the impact of cognitive deficits on the individuals’ present and future functioning, and 5) the value individuals placed on their premorbid thinking abilities.

**Psychotherapeutic Intervention**

In overview, *psychotherapy* is a general term used to describe the interactive/relational process between the therapist and the client based upon a wide range of differing theoretical frameworks of therapy. Each model proposes its own theoretical conceptualization of the client’s difficulties, their etiology and maintenance which, in turn, directly determines the aim and type of intervention made by the therapist. Specifically, it refers to the use of any technique, procedure or process that has palliative or curative effects upon any mental emotional or behavioral disorder (Reber, 1985). Broadly speaking, the aim is to help a troubled individual reduce their distress and enjoy greater life satisfaction through changing their thoughts, feelings, and behavior. The crucial point, however, is that regardless of theoretical orientation, psychotherapy is a *relational* process where both the therapist and the client collaborate in the therapeutic encounter. This distinction is
important from other forms of cognitive remediation, in that based on research relating to common factors of therapeutic outcome, the largest factor; therapeutic alliance is at the heart of its success (Judd & Wilson, 2005). For psychotherapy to be effective, three components of alliance must be established regardless of theoretical orientation: 1) Goals - mutually agreed outcomes to work towards; 2) Bonds - personal attachments including mutual trust, acceptance and confidence facilitating the therapeutic process; and 3) Tasks - the behaviors and cognitions forming the substance of the therapeutic process (Alderman, 2003).

**Challenges**

Traditionally, individuals with brain injuries were considered poor candidates for psychotherapy because the nature of their cognitive deficits and poor levels of insight were thought to preclude successful therapeutic outcomes (Gordon & Hibbard, 1992). One reason for this, Gordon and Hibbard contend, may lie in the training of the professionals who typically see patients without brain injuries, and that traditionally trained psychotherapists are often uncomfortable or unfamiliar with brain injury (1992). The training of most clinicians does not prepare them to modify their approach to individuals who think concretely, who are memory impaired, or who have poor insight. Conversely, the professionals who render cognitive remediation (i.e. neuropsychologists, speech therapists, and occupational therapists) often are not trained in psychotherapeutic techniques; thus, though they are familiar with the behavioral manifestations of brain injury, they are uncomfortable dealing with patients’ affective responses to brain injury. Consequently, TBI patients’ affective states often remain untreated (Gordon & Hibbard, 1992). In addition, when patients receive both remediation and psychotherapy, they are often seen by two separate individuals who frequently expect that patients are able to learn to generalize between the separate information obtained in one type of therapy to the other (Rohling,
Faust, Beverly, & Demakis, 2009). Though, as was discussed earlier, generalization is a key mode of therapeutic outcome, as it then can be applied to everyday events. However, generalization between therapeutic interventions, Gordon and Hibbard (1992) continue, is beyond the repertoire of many brain-injured individuals. Hence, the long-term efficacy of remediation often is jeopardized by patients’ untreated affective reactions to brain injury. The general assertion is that psychotherapy needs to be embedded within the context of remediation in order to maximize patient’s adjustment.

The lack of psychotherapeutic services for brain injury survivors, according to Judd and Wilson (2005), closely parallels a similar resistance to providing psychotherapy for people with learning disabilities because of the assumption that ‘lack of intelligence’ rendered the individual as an emotional ‘cripple’ and, therefore, incapable of engaging with the therapeutic process. Both learning-disabled individuals and brain injury survivors present the practitioner with obstacles (e.g. cognitive, communicative deficits, disinhibited behavior, emotional lability, etc.), which may make the process of therapeutic engagement and the forming of a sound working alliance particularly challenging. This then requires the therapist to be more interactive within sessions, utilizing transactional skills, which may not be necessary with individuals without impairments (Henry, Knippa, & Golden, 1985).

When trying to reconcile with a permanently altered self, patients must incorporate and eventually accept their residual cognitive and behavioral deficits into their self-perceptions. This process is made more difficult by the fact that cognitive deficits are largely invisible handicaps; that is it is difficult for individuals who think concretely to actually see how they are different (Fleming & Ownsworth, 2006). During the course of remediation, patients’ multiple (i.e. past, present, future) self-perceptions must be carefully evaluated within the context of changes.
Premorbid affective disturbance or personality disorder prior to injury would also be confounding elements in the case of effective assessment and rehabilitation of the individual, post-TBI. As well, disturbances in intrapsychic function in the brain-injured individual can, through deficits in memory function, perception, problem solving, reasoning, and abstraction, cause regression in affective and interpersonal relational processes (Cicerone, 1989; Freed, 2002). These disturbances have an organic basis, yet manifest in strict resemblance to symptom etiology, which may have confounding elements to the cognitive therapist. Aspects of anger, fear, anhedonia, sleep disturbance and thought distortion may give rise to misdiagnosis, without full understanding of the patient’s history. The general consensus among clinicians working with TBI is to see the patient first with an organic neurologic disorder, and to see the problematic symptom as syndromic to that disorder (Miller & Mittenberg, 1998). If, as Miller and Mittenberg (1998) contend, psychotherapeutic technique is applied without this lens, then outcomes will be considerably lower due to clinicians inexperience in understanding the organic mechanisms which propagate the symptom.

**Psychosocial and Personality Disturbances**

Psychosocial and personality disturbances are common after traumatic brain injury during the post-acute treatment phase and psychotherapeutic intervention can substantially reduce patients’ discomfort. Psychosocial sequelae include neuropsychologically based personality changes, emotional reactions to injury and preexisting characterological styles (Cicerone, 1989). Prigatano (1986) has suggested that emotional and personality disturbances after brain injury may be classified as neuropsychologically mediated problems that arise as, 1) a direct consequence of neuropathology, 2) reactive emotional problems related to coping skills deficits or, 3) premorbid characterological propensities as influence in reaction to the injury. An important responsibility
for the psychologist or psychotherapist is to have clarification and differentiation among these possibilities (e.g., frontal lobe-mediated apathy and lack of initiation versus sadness, social withdrawal, and loss of interest as an emotional reaction to injury versus passivity, dependency, and poor motivation as a preexisting personality style) (Cicerone, 1989).

Prigatano (1986) summarizes the personality changes that may result from direct damage to the brain. These include poor social judgment, egocentricity and lack of empathy, increased irritability and aggressiveness, emotional lability and disinhibition, and exaggerated suspiciousness. These symptoms are predominantly related to frontal and temporal lobe injuries, as they directly correlate to executive function and emotional control. Freed (2002) adds that, in addition to executive deficits related to personality change, there are many cases in which there exists a dimension of denial by the individual regarding any cognitive or functional disability, which can have direct consequences in both extra-therapeutic relationships as well as the therapeutic alliance attempts. Cicerone (1989) cites common clinical experience which suggests that patients who exhibit more extensive neuropsychological deficits often show the least awareness of their disability, yet denial does, in most cases, represent an emotional reaction and protective response in the face of increasing recognition of disability and emotional distress.

**Interpersonal Deficits**

To attain and sustain relationships, brain-injured individuals must regain some semblance of cognitive reintegration. They instinctively look to persons with whom they have had past relationships to provide this function. These designated persons, however, because of feelings of discontinuity with the other, often fail to meet this need (Williams, Evans, & Fleminger, 2003). Fiegelson (1993) described this effect of a loved one’s neurological trauma on prior relationships as a “personality death” that disrupts the core of the self-object dyad. She goes on to describe
how the brain-injured person reactivates primitive identifications and anxieties in the non-injured. This anxiety, as Fiegelson (1993) states, “consists of induced alterations of ego such as a mounting attempt to protect oneself from feelings of hatred toward the injured and subsequent guilt for this hatred, constantly calling on memory of the premorbid individual with unsuccessful results” (336). While the behavior of the injured creates anxiety in the beholder, the injured experience a reciprocal anxiety. They sense, as well, an emotional cutoff from the observer (Freed, 2002). Brain-injured individuals activate their observer’s anxiety mostly because of the radical change in the injured individual’s need for them to compensate for lost ego organization. The observer’s reaction to this need is often negative or evasive, which intensifies anxiety and activates feelings of loss and rejection, reinforcing the injured individuals’ diminished ability to maintain a sense of self among others (Feigelson, 1993).

Added problems may arise when individuals who were significant in the injured persons life at the time of injury have difficulty remaining in these relationships. Even more problematic is that these significant others often project their loss of interpersonal connection onto the injured person. A salient factor regarding interpersonal reintegration problems is that while the non-injured may experience the loss of the brain-injured individual as personality death, the injured may not have the same experience. While they may comprehend a cognitive loss, and may even observe a personality change, they do not necessarily see their own self-experience as altered (O’Gorman, 2006). It is then sometimes necessary, for the sake of meaningful therapeutic movement, that individuals not familiar with them prior to their injuries be entrusted with meeting the needs of their new personality (Freed, 2002).
Mood Disorders

A review of existing evidence reveals that emotional disturbance of various forms is very common following TBI. Research investigating patterns of mood disorders in TBI with the structured clinical interview for DSM-IV, found the most frequent diagnoses were major depression and specific anxiety disorders. Comorbidity was also high, with 44% of individuals having two or more diagnoses (Hibbard, Uysal, Kepler, Bogdany, & Silver, 1998). Specific anxiety disorders (panic disorder, obsessive-compulsive disorder, post-traumatic stress disorder), as well as generalized anxiety disorder, have been reported. It is also important to note aspects of anger control and impulsivity in behavior as being components of disorder commonly seen in brain-injured individuals.

Anxiety

Anxiety, as an anticipatory reaction, signals danger and serves as an essential function of the ego (Freed, 2002). Freud (1920) distinguished two types of anxiety: automatic and signal. Automatic anxiety is a reaction to a traumatic situation that results from excessive stimulation that the organism does not have the capacity to modulate. The ego is overwhelmed, either by an experience of helplessness or by an accumulation of tension. Freud considered automatic anxiety more likely during infancy and childhood, when the ego is immature. Signal anxiety serves to mobilize the ego’s defensive capacities and keep unacceptable thoughts and feelings from being conscious. The infantile psyche moves from automatic to signal anxiety through the gradual internalization of the mother or caregiver’s anxiety-relieving attentions. The infant no longer needs outside mirroring of anxiety relief, but can instead look to his or her own inner strength to regulate the anxiety experience. When signal anxiety fails, Freud (1920) states that more symptomatic manifestations of intrapsychic conflict result, giving rise to compulsions, phobias,
and in the extreme, a loss of self-integration which triggers a “fight-or-flight” response, an intense expression of despondency and helpless, untamed rage.

In many cases, individuals who have sustained brain trauma have difficulty differentiating between expressions of automatic anxiety and failure of signal anxiety. Despite more mature defenses, cognitive failure diminishes the signal anxiety function that wards off emerging impulses and painful feelings from consciousness. Because the anticipatory function of signal anxiety no longer operates in the same way, brain-injured individual’s awareness of potential consequences is variable and inconsistent. Brain trauma not only diminishes reliance on an internal signal, it also inhibits the realization that one can look to others to bring relief from anxiety. Brain-injured individuals do not often have the ability to hold memory-based expectations, and objective reality takes them by surprise, eliciting a constant state of fright, which may present to the therapist as being more disturbed than they are in actuality (Williams, Evans, & Fleminger, 2003).

**Depression**

The symptoms of depression generally fall into four categories: emotional (persistent sad mood), cognitive (feelings of worthlessness or inappropriate guilt), motivational (lack of interest), and physical (appetite or sleep disturbance). Clinical depression contrasts with the normal emotional experiences of sadness, in being extreme, persistent and disabling (Khan-Bourne & Brown, 2003). A review of the literature in brain injury gives a wide variation in the prevalence of clinical depression with rates from 34% to 70% (Kilmer & Demakis, 2006). Depression interacts with both cognitive and motivational processes as evidenced by experimental analogue research in healthy individuals and in depressed individuals with or without brain injury. It is important to note that non-brain-injured individuals with depression showed similar cortical impairment in the
prefrontal and temporal regions to that of TBI patients, causing deficits in cognition and goal-directed behavior (Khan-Bourne & Brown, 2003). The implication then is that depression causes its own problems, which may be superimposed on the primary cognitive deficits resulting from brain injury. Left untreated, studies indicate that depression will increase patient handicap, interfere with participation and rehabilitation, and impact on the recovery of cognitive and psychosocial function (Payne, 2000).

Khan-Bourne and Brown (2003) also examined the influence of depression on the long-term outcome of stroke patients. Patients were assessed on a variety of factors, using various methodologies (e.g. Beck Depression Inventory: BDI, Rankin Scale; assessment of daily living activities, Barthel Index; Neurological evaluation) three months after stroke and then at follow-up 15 months later. It was found that patients with a BDI score of 10 or more at three months was correlated with poorer functional outcome as measured by the Barthel Index. Poorer functional outcome at three months correlated with depression at 15 months. Thus, it is clear that it is necessary to address depression as part of the neurorehabilitative process.

Several factors have been implicated in the development of depression following brain injury, although a comprehensive model has yet to be proposed or tested (Khan-Bourne & Brown, 2003). Poorer initial recovery status (as measured by the Glasgow Coma Scale) is associated with a greater likelihood of depression, while the location of the brain injury and the level of insight into functional deficits may also play a role. Premorbid psychiatric disturbance may be an important predictor of post-injury depression (Payne, 2000). In some cases, the TBI may be an indirect result of premorbid psychiatric problems, for example where the TBI is a consequence of high-risk behavior or a suicide attempt (Khan-Bourne & Brown, 2003). Premorbid personality factors, cognitive assets and family support are also associated with post-brain injury depression.
In particular, it is suggested by Khan-Bourne and Brown (2003) that individuals who coped well with stress before their injury are likely to have some of those facilities intact, and at least the propensity to, with neurorehabilitation and cognitive based treatment, see more protection from developing depression or enhance recovery from it.

**Anger and Impulse Control**

It is known that anger and impulse control are common symptoms with TBI sufferers. Up to 46% of people with TBI report feelings of anger (Demark & Gemeinhardt, 2002). Researchers have investigated the neurological bases of anger and aggression, and have found several brain sites involved. These sites include the limbic: hypothalamus, amygdala, septum, and the cortical: prefrontal cortex. The limbic sites control deep brain function, while the cortical sites control higher executive function. In all humans, the deep limbic structures provide impulse-based activity relegated to functions such as drive, impulse, protection and fear avoidance (Demark & Gemeinhardt, 2002). The prefrontal lobes regulate attention, provide continuity and coherence of behavior over time, and modulate affective behavior in order to satisfy drives. Related to frontal lobe injuries, the cognitive status of the individual is affected in ways, including control of anger expression and impulse control. Tyerman & Humphrey (1984), classified anger categories in the case of TBI. They generated distinctions between *irritability* - the tendency to become annoyed or upset, *agitation* - a condition of uninhibited movement, *anger* - the arousal response which triggers aggression or verbal attack, and finally *episodic control* - attack without provocation. Although anger is a natural emotional reaction in humans, which can be self-preserving, it is problem anger which is at issue. For many with TBI, the inability to differentiate appropriateness of anger or determine sources of anger are key points for intervention purposes to work biopsychosocially in the, 1) physiological manifestations of anger, 2) the cognitive distortions and
impulsive emotionality of anger, and 3) the social implications and treatments of anger (Demark & Gemeinhardt, 2002).

**Psychotherapeutic Intervention Strategies**

Over the past 30 years, there have been considerable developments in psychotherapeutic interventions with brain-injured individuals. Throughout the course of understanding more finitely the neurobiological aspects of TBI, an increased emphasis on the provision of specialized interventions which incorporate a heightened awareness of concomitant physical, emotional, cognitive and behavioral symptoms must be employed. (Judd & Wilson, 2005). The cognitive, emotional and behavioral issues are inextricably linked and virtually impossible to consider in isolation in real world contexts. Judd and Wilson (2005) argue that organic brain damage should be conceptualized within a therapeutic model that recognizes both the physical and the non-physical aspects of existence and in which the organic damage and the psychological damage are inseparable.

The emphasis of psychotherapy after brain injury is on helping patients to make realistic commitments, re-establish a sense of meaning in their lives, and form realistic goals (Eslinger, Zappala, & Chakara, 2003). Ciccerone (1989) has identified four shared features of all psychotherapies:

1) The therapist needs to demonstrate his or her understanding and acceptance of the patient. One should be able to relate to and explain to patients the neurologic, cognitive and social aspects of their condition.

2) The treatment setting establishes the patient’s expectations of help. A specialized brain injury specific program can be a powerful determinants of
patient beliefs about treatment; patients and families can readily be convinced that they will “finally get the therapy they need.”

3) Psychotherapy with TBI promotes a rationale that includes an explanation of illness and health, disability and normality, and should enable the patient to make sense of his or her symptoms.

4) Psychotherapy prescribes a set of tasks and procedures for the patient. All therapeutic techniques demand some activity on the part of the brain-injured patient, and attempt to promote greater autonomy and accountability.

A biopsychosocial approach can be used in guiding the selection of appropriate interventions according to the nature and factors contributing to the individual’s level of unawareness of deficit. This can be achieved using information on brain lesion location, neuropsychological assessment results, the client’s reaction to feedback or difficulties with rehabilitation tasks, collateral information from significant others (e.g. concerning premorbid personality), specific coping style and personality, and analysis of the patient’s social and cultural environment (Fleming & Ownsworth, 2006).

**Cognitive Behavioral Techniques**

Cognitive Behavioral Therapy (CBT) may be readily applicable to the TBI patient, as they tend to be relatively structured and directive in nature (Cicerone, 1989). The intrinsic goal of CBT, as well as other therapies, is to engender awareness in the patient (Fleming & Ownsworth, 2006). Eslinger, Zappala, and Chakara (2003) recommend that awareness issues be dealt with early on in the acute phase of treatment. This may involve the performance of meaningful and concrete activities, emphasis on clarification and reframing of the patient’s inaccurate social perceptions and self-appraisals, and problem-solving strategies taught through stages (e.g.,
identification of the problem, review of alternate responses, selection of a response, and 
verification of a solution), which may be taught in relation to a broad range of functional and 
interpersonal situations. Leftoff’s (1983) training on *interpersonal hypothesis formulation* with 
patient’s experiencing anxiety and paranoia are based on specific problem-solving techniques 
relayed in the form of depersonalized hypotheses which require structured feedback.

Other CBT strategies noted in the literature include the use of *guided mastery* proposed by 
Bandura (1997), which involves engaging the client in structured experiences that allow for self-
monitoring and self-evaluation. As the individual exercises some control and gains mastery over 
the task, his or her own self-efficacy or metacognitive knowledge is restructured and 
strengthened. This is most likely to occur with familiar tasks given at the level appropriate to the 
patient’s awareness level. These tasks can then be accompanied by structured methods of self-
questioning and self-evaluation similar to Leftoff’s hypothesis formulation exercises where 
videotaped sessions are then used for later opportunities for self-evaluation (1983). Buskirk 
(1992) has also described structured metacognitive strategies along the lines of personal feedback-
oriented goals. He uses dynamic models within a CBT framework utilizing, 1) anticipation of 
obstacles and outcomes, 2) self-prediction of the level of difficulty and accuracy of performance, 
3) time monitoring, and 4) self-evaluation. Another metacognitive technique proposed by 
Buskirk is the use of *role reversal*, in which the therapist performs the task with errors and the 
client gives feedback (1992). This feedback is then reformatted for the patient’s own awareness 
of his or her own differentiation of the problem and self-determined solution.

**Psychodynamic Therapies**

Dynamically oriented therapies (i.e. those that rely on interpretive statements to develop 
insight) may be used a supportive context of TBI (Bootes & Chapparo, 2002). The interpretation
should be used to make explicit connections that the patient may be unable to make, yet the interpretation would be repeated until connections are made by the patient. The patient’s difficulty in establishing connections need not be based on unconscious factors or deal with past experience; more often, in fact, this difficulty appears related to neuropsychological and reactive emotional factors (Cicerone, 1989). Cicerone (1989) describes some specific psychodynamic techniques for use in TBI:

1) *Limit the range of associations to an interpretation.* The classical open-ended interpretive statement may create cognitive disorganization in the patient. After interpreting a patient’s emotional response the therapist should stay with the patient. For example, the therapist might say: “You’re getting angry now. It seems that you become angry whenever we mention you’re not going back to work. Can you see what I mean?”

2) *“Strike when the iron is cold.”* Interpretations can be postponed until the emotional state has subsided, allowing the patient greater control. For example, a therapist might say: “Do you remember earlier when we spoke about work? I’ve been trying to understand what made you so angry. Let’s talk about that now.”

3) *Maximize the patient’s preparedness and the supportive aspects of the environment.* For example, the therapist might say: “I’ve been thinking about your going back to work again. I’m going to say something that you may not like. You may get upset, but when you’re ready I want you to think about this.”
Psychodynamic TBI therapies also rely on uncovering unconscious defensive measures by the patient used to protect them from awareness of loss and its associated depressive affects. These defenses are of a narcissistic nature, in protection of the ego. The strength of the narcissistic defense depends on ego strength and the containment provided by early object relationships (O’Gorman, 2006). Freed (2002) suggests that narcissistic injury is one of the psychic wounds most common in TBI patients, and this defense is frequently what needs to be addressed in psychodynamic work.

**Group Therapy**

Group psychotherapy is a valuable treatment component. One advantage of group therapy is the placement of the patient in a social situation among peers. This can serve to reduce isolation, and at the same time demand a broader repertoire of social and interpersonal behaviors. For many patients, as Cicerone (1989) notes, sharing experiences with other TBI survivors is a powerful form of alleviating the individual’s sense of alienation and demoralization. Patients also benefit from social feedback from their peers regarding their cognitive limitations and emotional reactions. Group interaction also is effective at dealing with changes in self-concept following injury. Vickery and Gontkovsky (2006) explored the issues relating to self-concept changes and group therapeutic milieu. The study focused on discussion of two types of self-concept in group therapy: *self-complexity*, or the facet of the self which sees that there are many different aspects to the self and that it need not be defined in narrow terms, and *importance differentiation*, or the subjective assessment of importance of self appropriate to situation. The study’s findings were that both dimensions, when discussed metacognitively (i.e. thinking of one’s own self-concept), bolstered understanding of emotional reactions to situations, misappropriated importance placed on loss, ability to grieve loss, and more integration of awareness of others’ self-concepts from
their own. The findings also suggest that this mode of group work can be done relatively soon after acute stage care, as this also boosts awareness for the possibility for other, more insight-based therapies to be employed later on in the patient’s life.

**Family Therapy**

The family experience, which accompanies severe head trauma, parallels in many ways the crises a family might be expected to face with any natural disaster. Several sources of stress that families of TBI patients might encounter would include: little or no time to prepare, little sense of control, no sense of anticipation, no previous experience, few sources of guidance, great sense of loss, feelings of anger, and denial of problem (Henry, Knippa, & Golden, 1985).

Consistent with family systems theory, the family will restructure in response to behavioral changes of one member of the system. With a brain injury, the family realizes that the brain-injured person is unique from the pre-trauma family member. How this new individual is integrated into the reorganized family system, and how family members choose to participate with the injured person are often the basis for therapy. Henry, Knippa, and Golden (1985) consider the *Strategic Therapeutic Approach* in working with the brain-injured adult and the family:

1) The therapist’s principle task is to determine how the family system has responded to the brain-impaired individual. The therapist must determine the family’s definition of the problem and the attempts they have made at solutions.

2) The therapist’s next major task is to determine reasonable goals for therapy. This might require a reframing of the problem in a manner commonly acceptable to family members and conducive to change. Caution in approaching goals with TBI families must be taken, as unreasonable goals such as “returning him to the way he
was” may highlight discouragement. More realistic goals such as “finding a way to get the laundry done successfully,” keeps importance to reason in session.

3) A therapeutic contract is then set up which outline specific goals and length of therapeutic involvement. This contract is always open to re-negotiation if more time is needed or new problems arise.

4) Once the contract is agreed upon, the therapist tracks interactional patterns and important themes (e.g. husbands should always be the “breadwinner”, etc.) which help create or maintain the problems within the family system.

5) The therapist must have ample training, as well as communication with all facets of the patients care network (psychiatrist, neuropsychologist, speech pathologist, etc.), to act as arbiter of information, answer questions, or confront denial of limitations when needed.

**Therapy with Children and Adolescents**

In any one year, according to the CDC (2003), an estimated 250 in 100,000 children and adolescents experience traumatic brain injury. Of particular concern is the dynamic nature of brain development during childhood and adolescence. The impact of TBI on social and emotional adjustment varies greatly, in part as a function at age of injury. Efforts to provide structure and protection for a child who has sustained a TBI may decrease the child’s opportunities to develop autonomy. Problems with stable identity formation, particularly if family functioning was disrupted as a result of the injury, may be a further complication (Fry & O’Brien, 2002). In many instances, multi-modal therapies involving both individual behavioral interventions, as well as family and group work, contribute greatly to increased success. Effective therapeutic
interventions include insight-oriented psychotherapy, cognitive behavioral therapy, art therapy, relaxation training, and group therapy.

Indirect approaches, such as art therapy, are very successful in TBI patients of all ages, but most particularly with children, where concept formation and maturity of word use is not fully present. Plotts and Lasser (2008) identified four aspects of the use of art as therapeutically valuable in the treatment of TBI in children. The first is the multidimensional nature of the method, or its ability to present multiple layers in a single scene whether depicted through music, sandplay, paint or clay. A second therapeutic value stems from the narrative and dynamic possibilities of art therapy. Third, Plotts and Lasser assert that art therapeutic methods give access to preverbal content that would otherwise be inaccessible (2008). Finally, the fact that the method of intervention does not require a high degree of patient skills makes the intervention appealing. Upon completion of an expression through art, the client is typically encouraged to talk about the production. The therapist may ask open-ended questions about the creation (e.g. “Tell me about what you have made here”) or more pointed direct questions (e.g. “How does the cat feel about being surrounded by the high wall?”). Those clients with language impairments may experience difficulty with this verbal process. However, they may still benefit from the non-verbal process of creation and expression.

**Summary**

The therapeutic relationship, setting, rationale, and procedures together provide the TBI patient with opportunities for new learning experiences. In addition, patients develop awareness of their presumed and actual abilities, as well as confronting discrepancies between biased world perceptions and actuality through cognitive therapy. Impressive in importance are the emotional and motivational factors, irrespective of techniques. The therapist’s attitude of caring and
empathetic understanding toward making the person whole are probably of greatest therapeutic value, not only in cognitive therapeutic settings, but at all levels of care with survivors of traumatic brain injury.

**Conclusion**

Brain injury, regardless of chronological age, catapults individuals into the second half of life. That is, life is no longer the same after brain injury. The facts are clear: individuals function with less intelligence, less capacity for memory, less physical strength, less ability to engage in meaningful work, and have a greater inability to feel valued in an oftentimes uncaring world. This is indeed a sobering comment with which to conclude this paper. Yet, those deficits incurred by individuals who have suffered traumatic brain injury are, and have been, greatly decreased with the encouraging prospects of multi-modal therapies utilizing the biopsychosocial lens. With its first inception as a necessary holistic imperative in providing better care in treatment of the whole person, biopsychosocial treatment modalities exist now not merely as components of health care, but rather as a unified body of health care for the TBI patient, as well as for others. They require the cross-communication between disciplines, as well as deeper expansion of knowledge and empathetic understanding by all involved to bring those who have suffered the shattering effects of a traumatic brain injury closer to whole: more able to remember, feeling stronger, enjoying a vocation, having meaningful relationships, and feeling included in a more understanding world which will not allow them to be forgotten.
References


