Brain and Brain Injury 101

• T.W. McAllister, MD
“From nothing else but the brain come joy, delights, laughter and sports, and sorrows, griefs, despondency, and lamentations...madness and delirium... the fears and frights which assail us...thoughts that will not come, forgotten duties, and eccentricities... All these things we endure from the brain...”

- Hippocrates, *On the Sacred Disease*, 4th Century B.C.
OVERVIEW

• Brain Organization (Lobes, Layers, Colors, Cells, Circuits)
• Brain Disorganization
  – sequelae of regional injury
  – sequelae of traumatic brain injury (TBI)
Essential Neuroanatomy

**Figure 2.1** Three-dimensional reconstruction of the brain, view of the left hemisphere.

**Telencephalon** – neocortex, white matter, and subcortical nuclear complexes

**Limbic System** (functional) – collection of medial structures including the cingulate gyrus, hippocampus, amygdala, and other medial temporal gyri, thalamus (esp. dorsal and anteromedial), hypothalamus, and limbic midbrain area

**Basal Ganglia** (functional) – caudate, putamen, globus pallidus (interna and externa), and substantia nigra

**Diencephalon** – thalamus, hypothalamus, pituitary gland, pineal gland

**Reticular Formation** – collection of brainstem nuclei running from the rostral midbrain to the medulla, and with its functional components:

**Brainstem:**
- **Mesencephalon** – midbrain
- **Metencephalon** – pons and cerebellum
- **Myelencephalon** – medulla
Brain Organization

• **LOBES**
  – frontal, parietal, temporal, occipital

• **COLOR**
  – gray matter (cell bodies)
  – white matter (axons)
Brain Organization

- **LAYERS**
  - cortical (top layer, primarily gray matter)
  - subcortical (underneath – white matter and gray matter such as the basal ganglia)

- **CIRCUITS** – specific pathways which span lobes and layers and regulate specific functions
Telencephalon (Neocortex)

- Frontal lobes
  - prefrontal: complex cognition
- Temporal lobes
- Parietal lobes
- Occipital lobes
Telencephalon (Neocortex)
Neocortex

- **Primary cortex**
  - unimodal, serves a single neurobehavioral function
    - primary sensory cortex or primary motor cortex

- **Secondary cortex**
  - unimodal, serves a single neurobehavioral function
    - association cortex for either sensory or motor cortex
Neocortex

• Tertiary cortex
  – heteromodal cortex
    • integrates information from secondary cortices to form multimodal associative representations

• Quaternary cortex
  – heteromodal cortex
    • serves to direct processing resources toward the associative network(s) most relevant to environmental (internal or external) contingencies
## Telencephalon (Neocortex)

<table>
<thead>
<tr>
<th>Major Functions</th>
<th>Frontal</th>
<th>Temporal</th>
<th>Parietal</th>
<th>Occipital</th>
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<tbody>
<tr>
<td>Motor planning</td>
<td>Primary auditory cortex (lateral)</td>
<td>Tactile sensation</td>
<td>Vision</td>
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</tr>
<tr>
<td>Voluntary movement</td>
<td>Language recognition (left lateral)</td>
<td>Heteromodal sensory association</td>
<td>Visual perception and association</td>
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<tr>
<td>Social behavior and judgement</td>
<td>Auditory prosody (right lateral)</td>
<td>Visuospatial function (right)</td>
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<tr>
<td>Motivation</td>
<td>Memory (medial)</td>
<td>Some elements of praxis (right)</td>
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<td>Complex cognition</td>
<td>Fight/flight reactions (medial)</td>
<td>Reading (left)</td>
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<td>Language production (left)</td>
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<td>Language/motor prosody (right)</td>
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<td>Stereognosis (left)</td>
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**Table 2.1** The major divisions of the neocortex, including partial lists of the major functions subserved by each neocortical area.
Neurotransmission at the Synapse

Electro-neurochemical coupling

Illustration by Lydia Kibiuk
Figure 6.9 Events at the synapse.
Dopamine and Norepinephrine

Dopamine Pathways (meso-limbic; meso-cortical)

(From http://www.onu.edu/user/FS/tfaulkner/Dopamine.htm)
Language: Fluency

- **Broca’s Area**
  - motor component of language

- **Supplementary motor area**
  - responsible for generating the motor impulses integrated in Broca’s area

- If these areas are intact and connected, language is fluent
Impact of Regional Injury on Behavior

• Frontal Lobe Syndromes

• Temporal Lobe Syndromes

• Parietal Lobe Syndromes

• Basal Ganglia Syndromes
Frontal Cortex

Dorsolateral prefrontal cortex
Supplementary motor area
Primary motor cortex
Orbitofrontal cortex
Broca’s area
Frontal eye field

Figure 4.2
Sagittal view of the cingulate gyrus and its major division: the infracallosal cingulate or visceral effector region (A), the anterior cingulate or cognitive effector region (B), the skeletomotor effector region (C), and the posterior cingulate or sensory processing region.

A-D: Cingulate gyrus
Frontal-subcortical circuits

• Five major circuits
  – Motor
  – Frontal eye fields
  – Dorsolateral prefrontal (executive)
  – Lateral orbitofrontal (“social intelligence”)
  – Anterior cingulate (motivation)

• All share the same fundamental circuits, and hence dysfunction in one is often associated with dysfunction in one or more of the others
**Frontal-Subcortical Circuits**

**Figure 4.3** General outline of frontal-subcortical circuitry.
Frontal-Subcortical Circuits

• Complex cognition is subserved by three of these circuits
  – Dorsolateral prefrontal: executive function
  – Lateral orbitofrontal: “social intelligence”
  – Anterior cingulate: motivation

• All share the same fundamental circuits, and hence dysfunction in one is often associated with dysfunction in one or more of the others
FRONTAL LOBE SYNDROMES

- APATHETIC OR “PSEUDODEPRESSED”
- DISINHIBITED OR “PSEUDOPSYCHOPATHIC”
- AKINETIC MUTISM
- MIXED
Limbic and Paralimbic Anatomy

Important concepts:

1. The limbic system includes a lateral and medial circuit.
2. Basic (or “unconscious”) emotion is represented in a ventral limbic-paralimbic network.
3. Emotional awareness (“conscious”) emotion is represented in a dorsal limbic-paralimbic-cortical network.
4. Emotion is relatively lateralized in the brain.
   - Left hemisphere = positive emotion
   - Right hemisphere = negative emotion
Limbic System

A. Corpus Callosum
B. Olfactory Bulb
C. Mammillary Body
D. Fornix
E. Ant. Nuc. Hypothalamus
F. Amygdaloid Body
G. Hippocampus
H. Parahippocampal Gyrus
I. Cingulate Gyrus
J. Hypothalamic Nuclei
Limbic System

Figure 2.8 Several important limbic structures. T1-weighted coronal magnetic resonance image of the brain at the level of the hippocampal formation.
Figure 34-4
Composite drawing showing size and location of contusions found in a series of 40 consecutive cases. The tendency to localize in the subfrontal and temporal regions is clearly indicated. (From Courville.)
Temporal Lobe and Limbic Dysregulation Syndromes

• Cognitive Deficit Syndromes
• Emotional Dysregulation
  – Mood disorders
  – Personality changes
• Seizure Disorders
  – complex partial seizures
  – atypical seizures
  – inter-ictal personality syndromes
Non-Classic Ictal Syndromes

1. “Spells” – 100%
   - losing time / staring / trance-like

2. Intense Episodic Mood Swings – 70%
   - panic-like anxiety / depression
   - abrupt onset / “off set”

Non-Classic Ictal Syndromes

3. **Episodic Thought Disturbance** – 60%
   - paranoia/first rank symptoms/delusions

4. **Suicide Attempts / Ideation** – 50%
   - no precipitants

5. **Episodic Hallucinations** – 60%
   - auditory – 50%
   - visual – 40%

Atypical Partial Complex Seizures

- paroxysmal nature of spells
- more normal function in between events
Telencephalon (Neocortex)
Parietal Lobe Syndromes

• Sensory functions
  – Touch, Pain, Temperature
  – Proprioception
  – Stereognosis
  – Graphesthesia
PARIETAL LOBE SYNDROMES
Cognitive Functions

Coordination of Multi-Modal Functions

- Kinesthetic Praxis
- Ideomotor Praxis
- Dressing Praxis
- Facial Recognition
- R/L Orientation
- Spatial Orientation
- Calculation
- Directed Attention
PARIETAL LOBE SYNDROMES

Associated Behavioral Syndromes

- Confusional States
- Alienation Experiences
- Prosopagnosia
- Delusional Syndromes
  (Capgras “Impostor” Syndrome)
- Denial of Deficits
- Spatial neglect
Basal Ganglia

- Caudate
- Putamen
- Globus pallidus
  - interna
  - externa
- Substantia nigra
- Subthalamic nucleus
Figure 2.6 The basal ganglia. Also shown is the thalamus.
Basal Ganglia

- Initiation, coordination, and cessation of neurobehavioral functions
  - Fine motor, postural, and facial (incl. eye) movement
  - Executive function, social behavior, motivation

- Plays an important role in many neuropsychiatric disorders (e.g., Parkinson’s, Huntington’s, OCD)
Primary Basal Ganglia Disorders

- Parkinson’s Disease
  - Depressive Syndromes (47-71%)
  - Dementia (30-40%)
  - Psychotic Syndromes
- Huntington’s Disease
  - Depressive Syndromes (28-63%)
  - Dementia
  - Schizophreniform Syndromes
- Wilson’s Disease
  - Schizophreniform Syndromes
  - Depressive Syndromes
Basal Ganglia Injury And OCD

• Assoc. between OCD sx’s and diseases affecting the basal ganglia
  
  – Metabolic Insults (carbon monoxide, manganese toxicity)
  – Traumatic brain injury (several case reports) following even minor TBI)
  – Sydenham’s Chorea
  – Encephalitis lethargica
  – Idiopathic Basal Ganglia Calcification
  – Huntington’s Disease
  – Functional imaging data
Basal Ganglia Injury And OCD

• Primary neuronal circuitry links the basal ganglia with orbito-frontal cortex, limbic system and thalamus

• Metabolic studies, lesion studies, and circuitry strongly suggest an important role for the basal ganglia in the genesis and maintenance of OCD symptoms
IMPACT OF LATERALITY of INJURY on BEHAVIOR
Depression in Neurologic Disorders

• Variety of mood disorders common after all disorders of the CNS

• Complex interaction between profile of brain injury, genetic vulnerabilities, and meaning of sequelae
The Bluebird of Happiness long absent from his life, Ned is visited by the Chicken of Depression.
Summary of Findings

- 60% of CVA patients will suffer a depressive syndrome in the subsequent two years

- RATE and SEVERITY of depressive symptoms is higher than in population of patients with orthopedic injuries and similar functional disability
LATERALITY OF CVA AND DEPRESSION

• RATE and SEVERITY of depressive symptoms is higher in left hemispheric CVA patients, and varies directly with proximity of the lesion to the anterior frontal pole.

• Phenomenology similar to non-CVA depression with respect to
  – Symptom picture
  – Natural history
  – Response to treatment

• May be related to disruption of asymmetrically distributed neurotransmitter tracts
Depressive Syndromes in TBI

- 66 TBI patients without spinal cord or other significant injuries
- 20% had GCS of 12-15
- Lesions categorized by CT scan
- Followed at 1 month and 1 year

From: Federoff et al., 1992; Jorge et al., 1993; Jorge et al., 1994
Depressive Syndromes in TBI

• 1 Month:
  – 27% had major depression
  – No difference in rates of family psychiatric illness
  – Strongest correlate: presence of left anterior and/or left basal ganglia lesion
Depressive Syndromes in TBI

• 1 Year:
  – 20-30% currently depressed
  – Duration of depressive episodes: 4-5 mos
  • i.e., Percentage is same, but different individuals depressed
  – No longer correlated with lesion location
Mania

- Sustained Euphoric or Irritable mood and:
  - grandiosity
  - decreased need for sleep
  - pressured speech
  - racing thoughts
  - distractibility
  - increased psychomotor activity
  - excessive involvement in pleasurable activities
Mania

- Can present with periodic increase in the frequency and intensity of assaultive or dyscontrolled behavior
- Grandiosity can be context specific
- Can see increase in sexual behavior
Mania

• Usual pattern is periodic bursts or clusters of sustained manic behaviors
• Some have reported “chronic mania”
• Can see rapid cycling pattern (4 or more episodes in a 12 month period)
Mania in TBI

- Very similar to DSM-IV description in mild TBI
- Characteristic change in sleep and activation are the most helpful signs in more severely impaired
- Often associated with increase in frequency and intensity of aggression/irritability
SUMMARY OF CNS SECONDARY MANIA

- Based on 106 cases abstracted from the literature
MANIA SUBSEQUENT TO TBI

• At least 16 reports representing 44 patients
Parietal Syndrome

- 53 yo psychiatrist driving to his workplace of 12 years
- Sudden sense of unfamiliarity, became lost
- Able to find phone, describe landmarks, recognized aspects of environment, but did not feel familiar
- Right parietal infarct on CT scan
Parietal Syndrome

- Right Parietal Infarct
- 3 weeks later, developed a manic syndrome
IMPACT OF TYPE OF INJURY ON BEHAVIOR
What happens in a TBI?

• Mechanism – Acceleration/Deceleration
  – Differential movement of partially tethered
  – Brain within the skull

• Results in – Bruising of the brain surface
  against rough areas of the skull
  – Stretching and twisting of nerve axons
Biomechanical Injury: Translation

Coup site

Contre-Coup site

Force vector

Coup site
Biomechanical Injury: Rotation
Biomechanical Injury: Angular Acceleration

Rotation vector

Force vector
CT Scanning: Examples
http://nanonline.org/nandistance/mtbi/modules/patho/pathophysiology.html

Normal Brain

TBI with Depressed Skull Fracture
CT Scanning: Examples

Hemorrhage w/mass effect

Subdural Hematoma
MRI: Cerebral Hemorrhage

http://www.med.harvard.edu/AANLIB/home.html
MRI (T1): Hemorrhage & Hydrocephalus
MRI (T1): Hematoma & Hemorrhage

L frontal extra-axial subdural hematoma

R temporal hemorrhagic contusion; L hematoma
Composite drawing showing size and location of contusions found in a series of 40 consecutive cases. The tendency to localize in the subfrontal and temporal regions is clearly indicated. (From Courville.)
Neuropathophysiology of TBI

• Two Major Categories

• PRIMARY INJURY: Direct result of the force applied to the brain
  – Contusions
  – Hemorrhages
  – Diffuse Axonal Injury (DAI)
Neuropathophysiology of TBI

- **SECONDARY INJURY**: Occurs after the initial trauma, & as a result of the primary injury
  - Cerebral edema, cerebral infarcts
  - Cerebral anoxia
  - Cell injury and death from release of toxins from injured cells
Biomechanical Injury

Pre-Injury

Acute Injury
Biomechanical Injury: Diffuse Axonal Injury

Pre-Injury

Acute Injury
Cytotoxic Injury

- Cytoskeletal injury
- Axonal swelling and lysis
Gradient Echo Scans:
Shear Hemorrhages
Neuropathophysiology of TBI

SECONDARY INJURY:

• Categories of Cell Toxins
  – Excessive Excitatory Amino Acids
  – Excessive Cholinergic Activation
  – Release of Cytokines
TBI Related Neurotransmitter Changes

- **Catecholamines/Indoleamines**
  - Both peripheral and central effects
  - Prolonged (1 week) elevation of hypothalamic NE and DA
  - Can alter peripheral and central vasculature
  - Appear to play a role in recovery
  - May have implications for use of psychotropics during recovery
TBI Related Neurotransmitter Changes

- **Acetylcholine** – increased release/turnover (thalamus, amygdala, cingulate)
- **Functional Role**: makes certain regions more vulnerable to further injury (e.g., hippocampus)
- **Treatment Implications**: use of anticholinergic agents
Effects of Secondary Injury

- Altered brain blood flow
- Altered blood-brain barrier
- Altered local metabolic environment
- Kills neurons and glial cells (neurotoxicity)
Neuropathology of TBI

• Combination of:
  – Diffuse and focal
  – Primary and secondary
  – Immediate injury and evolution of damage over time

• Non-random geographic distribution of injury burden within the brain
Diffuse Axonal Injury

- Particularly effects:
  - Sub-cortical white matter
  - Corpus callosum
Regional Cortical Vulnerability to TBI Predicts Neuropsychiatric Sequelae

Dorsolateral prefrontal cortex
(executive function, including sustained and complex attention, memory retrieval, abstraction, judgement, insight, problem solving)

Orbitofrontal cortex
(emotional and social responding)

Anterior temporal cortex
(memory retrieval, face recognition, language)

Amygdala (emotional learning and conditioning, including fear/anxiety)

Hippocampus (only partially visible in this view - declarative memory)

Ventral brainstem (arousal, ascending activation of diencephalic, subcortical, and cortical structures)
Neuropsychiatric Sequelae

• Combination of changes in:
  – Personality, emotional control, mood regulation
  – Cognition
Personality Changes

• Two I’s
• Three A’s
Personality Changes

• **Impulsivity**
  – Speak before thinking
  – Act before thinking
  – Poor judgement
  – Sexual indiscretions
Personality Changes

- Irritability
  - "snippy"
  - Angry outbursts
  - Aggression
Personality Changes

• Affective Instability
  – “more emotional” (emotional incontinence)
  – Pathological affect (laughing, crying)
  – Predictably unpredictable
Affective Lability

...I was not as upset or as sad as my crying would imply, nor as uproariously amused as my uncontrollable laughter would indicate.

You have no idea how terrible it is when the crying is fully triggered and takes hold like a seizure. I can’t control any of it. I simply disintegrate and it isn’t only emotionally horrible with me, it is physically painful and debilitating.

Personality Changes

• Apathy
  – Loss of interest
  – Loss of initiative
  – Loss of drive
Personality Changes

• Awareness
  – Lack of awareness of personality changes
  – Unable to self-monitor
  – Unable to predict impact of their behavior on others
  – Unable to predict impact of deficits on life plans
PERSONALITY CHANGES

- Lower frustration tolerance, mood swings, irritability, impulsivity
- Inability to perceive deficits
- Difficulty understanding impact of behavior on others
- Apathy, lack of motivation
- Above related to typical Frontal and Temporal lobe injuries
PERSONALITY CHANGES

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Cognition

• Complex cognitive functions can be roughly localized to:
  – brain regions
  – Interactive circuits

• Much of our information comes from studying individuals with localized and regional injury
Cognitive Deficits After CNS Injury

- Executive Function
- Attention
- Memory
- Speed of Information Processing
- Speech and language
Executive Functions

• Volition
  – Must be able to conceptualize wants/needs

• Planning
  – Visualization, sequencing

• Purposive Action
  – Initiate, maintain, switch, stop

• Performance
  – Monitor, integrate feedback
Executive Functions

• Also encompass concepts of:
  – Mental flexibility
  – Awareness
  – Judgement

• Require intact:
  – Attention
  – Memory
  – Impulse Control
Medial View of Substrate of Frontal Executive Function

- Superior frontal gyrus
- Precentral gyrus (Motor Strip)
- Supplementary motor cortex
- Anterior cingulate gyrus
- Orbitofrontal cortex
- Medial View of Substrate of Frontal Executive Function
Attention

- **Arousal**, or the general level of neuronal responsivity
- **Orientation**, or the realignment of sensory processing to novel stimuli
- **Selective Attention**, or the preferential allocation of processing resources to a specific stimulus
- **Sustained Attention**, or vigilance and working memory
- **Divided Attention**, or simultaneous attention to and processing of several stimuli
Memory

• Many different types:
  – Working memory
  – Episodic memory
  – Procedural
  – Long term/short term

• Many different components
  – Registration
  – Encoding
  – Retrieval
Memory After TBI

• Most common cognitive complaint
• All types vulnerable, but most common deficits:
  – Working memory
  – Short term memory
  – Encoding
Memory Deficits After TBI

• Recently, attention has focused on working memory (WM) impairment as a core problem encountered by many TBI patients.
• Working memory refers to the ability to hold information in mind, or “online” while retrieving or processing other relevant information [Baddeley, 1986;1994].
Letter-Number Sequencing

- First numbers in ascending order
- Then, letters in alphabetical order

7-L-2
R-6-B-4-9-M
8-C-5-X-A-3-T-1

2-7-L
4-6-9-B-M-R
1-3-5-8-A-C-T-X
Surface Rendered Projections on Standardized Atlas Brain

Controls

1-back > 0-back

2-back > 1-back

MTBI
TBI and Substance Abuse

• The single greatest risk factor for traumatic brain injury (TBI) is alcohol/drug use
• TBI is often an irreversible adverse consequence of the pharmacological effects and addictive use of alcohol and drugs.
TBI and Alcohol/Drug Abuse

- Prevalence of alcohol dependence (addictive drinking) in patients with TBI ranges from 25%-68%.
- Majority of those involved in TBI had a serious problem with alcohol use prior to the onset of the injury (Edna 1985; Elmer and Lim 1985; Miller and Adams, in press).
Diagnosis of Etoh/Drug Abuse in TBI

- Lethargy, agitation, confusion, disorientation, respiratory depression following acute intoxication and overdose are very similar to those following brain injury.
- Some intoxicated patients discharged from ED with undiagnosed brain injuries.
- In a study of 167 patients (Gallagher and Browder 1968), alcohol obscured changes in consciousness, leading to misdiagnosis or delayed diagnosis of complications of brain trauma.
  - In 21 patients, a subdural hematoma was diagnosed only at postmortem (Galbraith 1976), and others have reported similar results (Rumbaugh and Fang 1980).
    - Miller and Adams, in press
Diagnosis of ETOH/Drug Abuse

• Combination of:
  – blood-alcohol levels (BAL)
  – quantity-frequency of alcohol and/or drug consumption
  – Brief screening instruments
    • Short Michigan Alcoholism Screening Test
    • CAGE
Effect of Alcohol/Drug Use on TBI Outcome

• Individuals drinking/drugging at time of injury:
  – Longer hospital stays and increased expense
  – Increased agitation
  – Poorer cognitive/functional outcomes
  – Increased regional brain atrophy
    • Frontal, temporal, cerebellar regions
Treatment Issues

• Discontinue the active use of alcohol and drugs
• Assess influence of alcohol and drugs on:
  – Mood
  – Cognition
  – Behavior
  – Drug seeking from the addictive disorder
• Rule out coexisting psychiatric disorders apart from the effects of alcohol and drug intoxication and addictive use of drugs
  – (Blankfield 1986; Miller and Mahler 1991; Miller and Adams in press).
Treatment Issues

- Treatment of withdrawal from alcohol and drugs in addicted patients with TBI are similar to those employed in patients without TBI, with some important exceptions.
- The identification of alcohol and drug intoxication and withdrawal follows the general principles of pharmacological dependence.
- The use of blood and urine toxicology is important to identify presence and levels of alcohol and drugs for assessment of intoxication and anticipation of withdrawal.
- The use of vital signs, particularly blood pressure, pulse, and temperature, are critical in determining the presence and severity of the withdrawal state (Miller 1991a).
Treatment Issues

• Usual approaches complicated by:
  – Cognitive deficits (no carry-over session to session)
  – Double Denial (denial of substance abuse and TBI-related deficits)
  – Difficulties in usual treatment settings
    • Group meeting overstimulating
    • Cognitive deficits limit benefits
    • Disorders of social comportment problematic
    • Group resistance to increased use of meds
Treatment Issues

• Basic principles used in working with individuals with TBI work in addiction treatment as well. TBI patients require:
  – concrete and structured settings
  – programs geared to the cognitive level of the individual
  – Importance of team understanding both addiction and TBI to provide a consistent, cogent, and effective treatment plan.
Treatment Issues

• Techniques such as keeping it simple, focused, and concrete are useful in both patient populations (Miller and Adams, in press)

• Being directive and supportive are also useful in patients with addiction and TBI (Sparadeo et al. 1990)
Treatment Issues

• Redirect them using appropriate cues and reinforcers.
• Teach substance use prevention skills that can be used in more than one life setting to maximize generalizability.
• Focus on specific prevention goal.
• Be redundant.
• Never assume understanding or memory from previous session.
• Be redundant.
• Always repeat the purpose, duration, and guidelines for each meeting.
• Be redundant.
• Summarize previous progress and then restate where the previous meeting left off (ie be redundant) (Sparadeo et al 1990; Miller and Adams, in press).
SUMMARY

• Brain Organization (Lobes, Layers, Colors, Cells, Circuits)
• Brain Disorganization (Sequelae of Regional Injury)
• Specific consequences of TBI