

# Brain and Brain Injury 101

- **T.W. McAllister, MD**

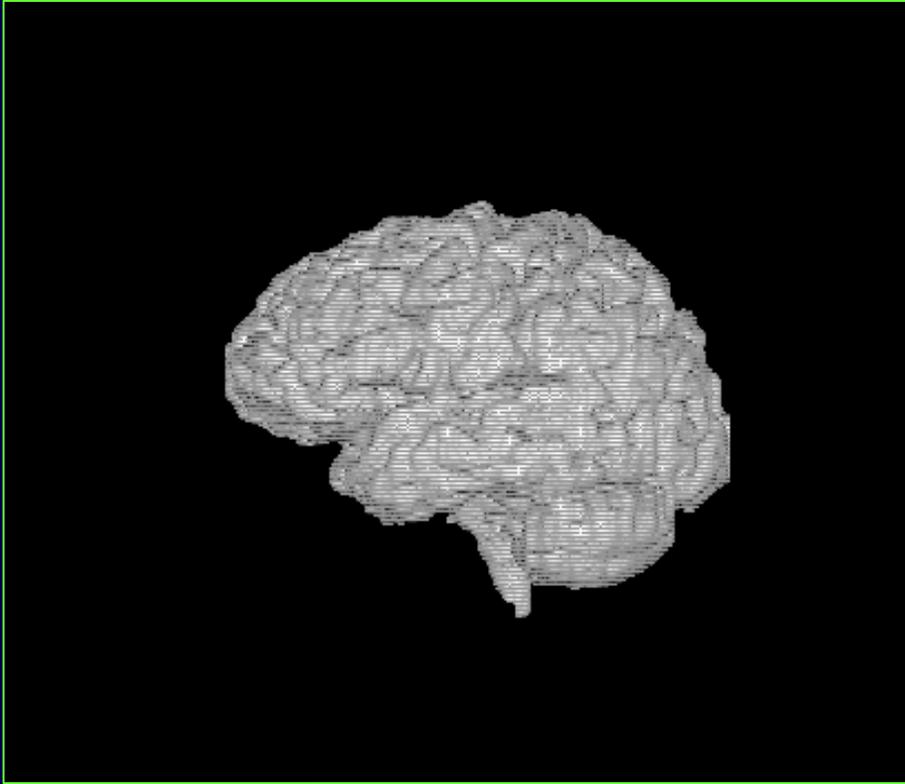
*“From nothing else but the brain come joy, delights, laughter and sports, and sorrows, griefs, despondency, and lamentations...madness and delirium... the fears and frights which assail us... thoughts that will not come, forgotten duties, and eccentricities... All these things we endure from the brain...”*

- 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



**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

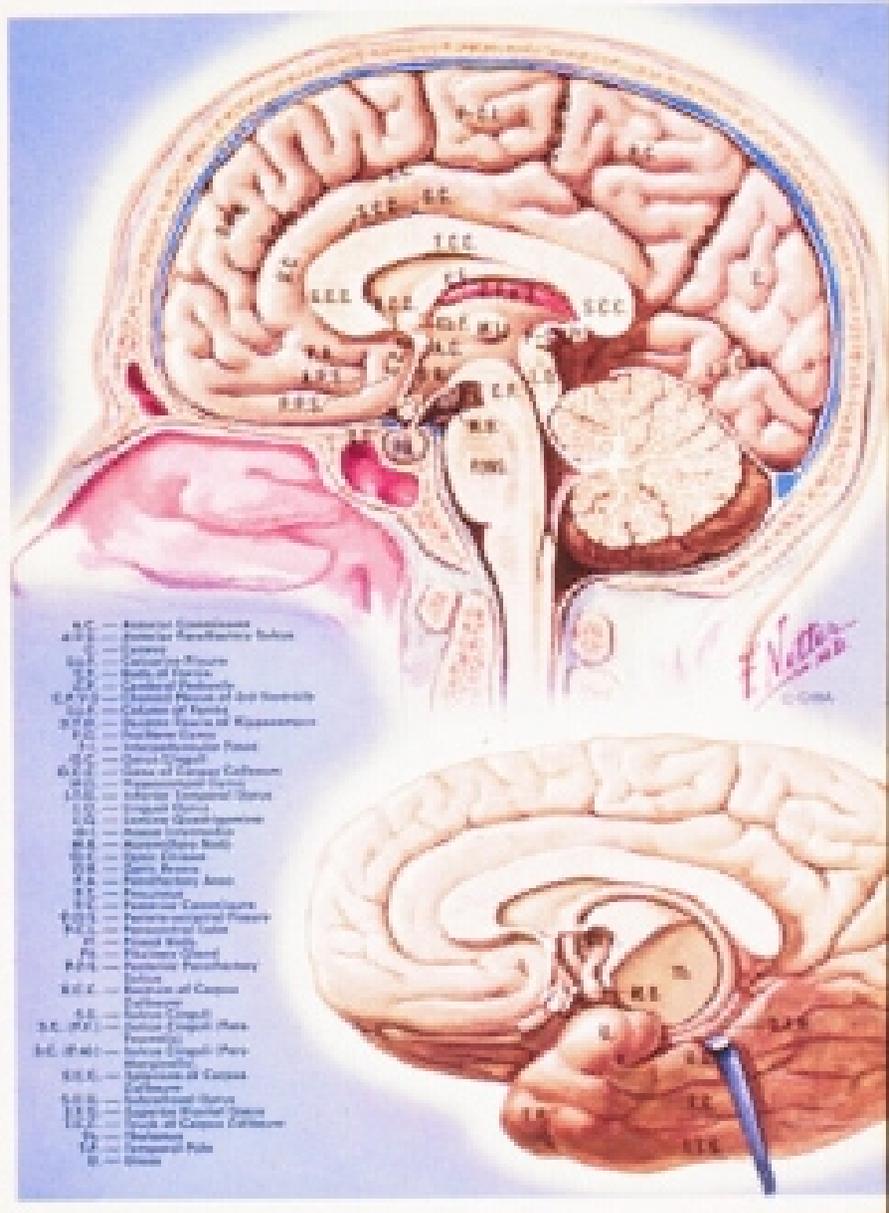
**Myelencephalon** – medulla

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

# Brain Organization

- LOBES
  - frontal, parietal, temporal, occipital
  
- COLOR
  - gray matter (cell bodies)
  - white matter (axons)





- A.C. — Anterior Commissure
- A.C.C. — Anterior Cerebral Cortex
- A.C.G. — Anterior Cingulate Gyrus
- A.C.H. — Anterior Horn of Horn
- A.C.L. — Anterior Commissure
- A.C.P. — Anterior Commissure
- A.C.S. — Anterior Commissure
- A.C.T. — Anterior Commissure
- A.C.V. — Anterior Commissure
- A.C.W. — Anterior Commissure
- A.C.X. — Anterior Commissure
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- A.C.2 — Anterior Commissure
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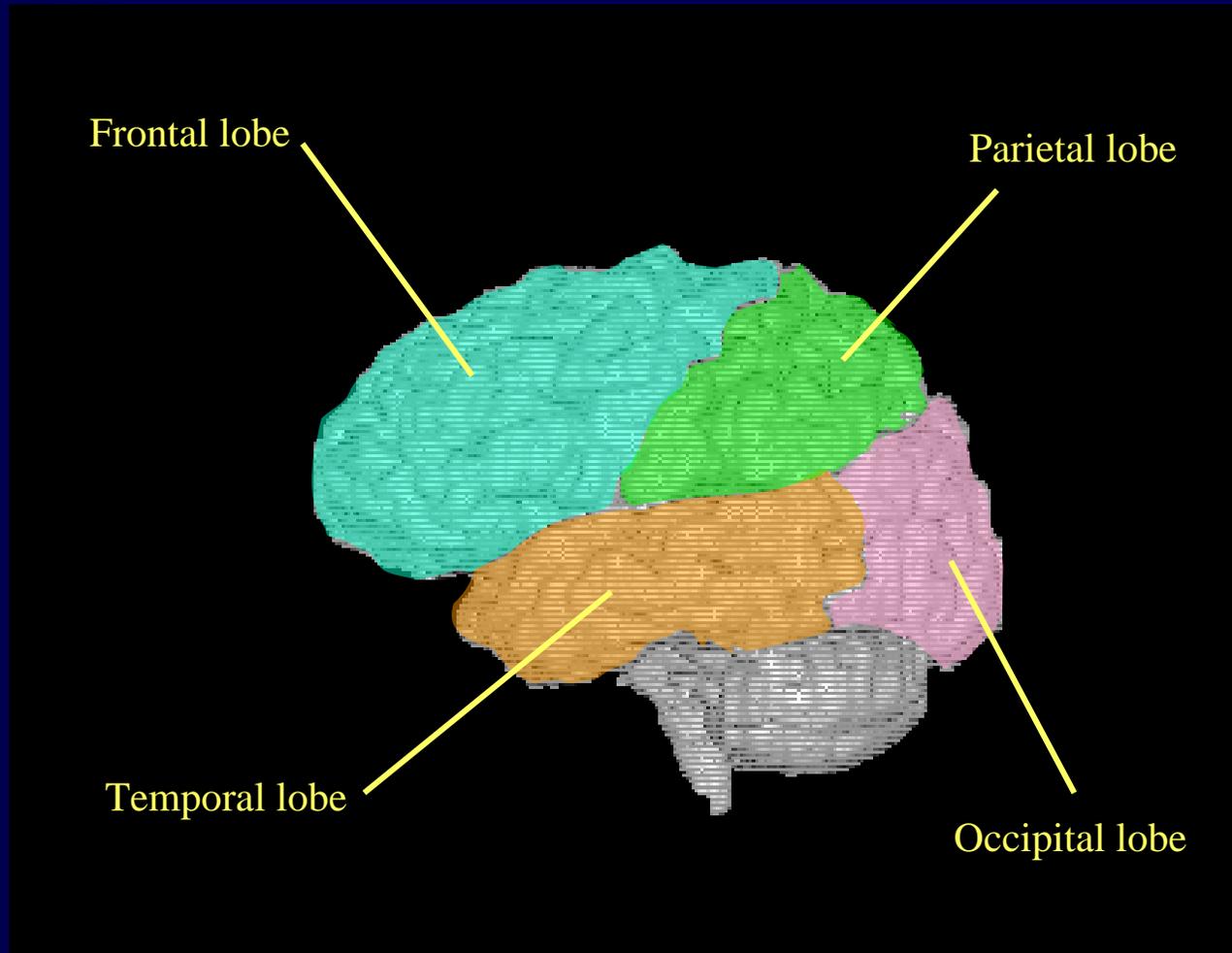
# Brain Organization

- **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)





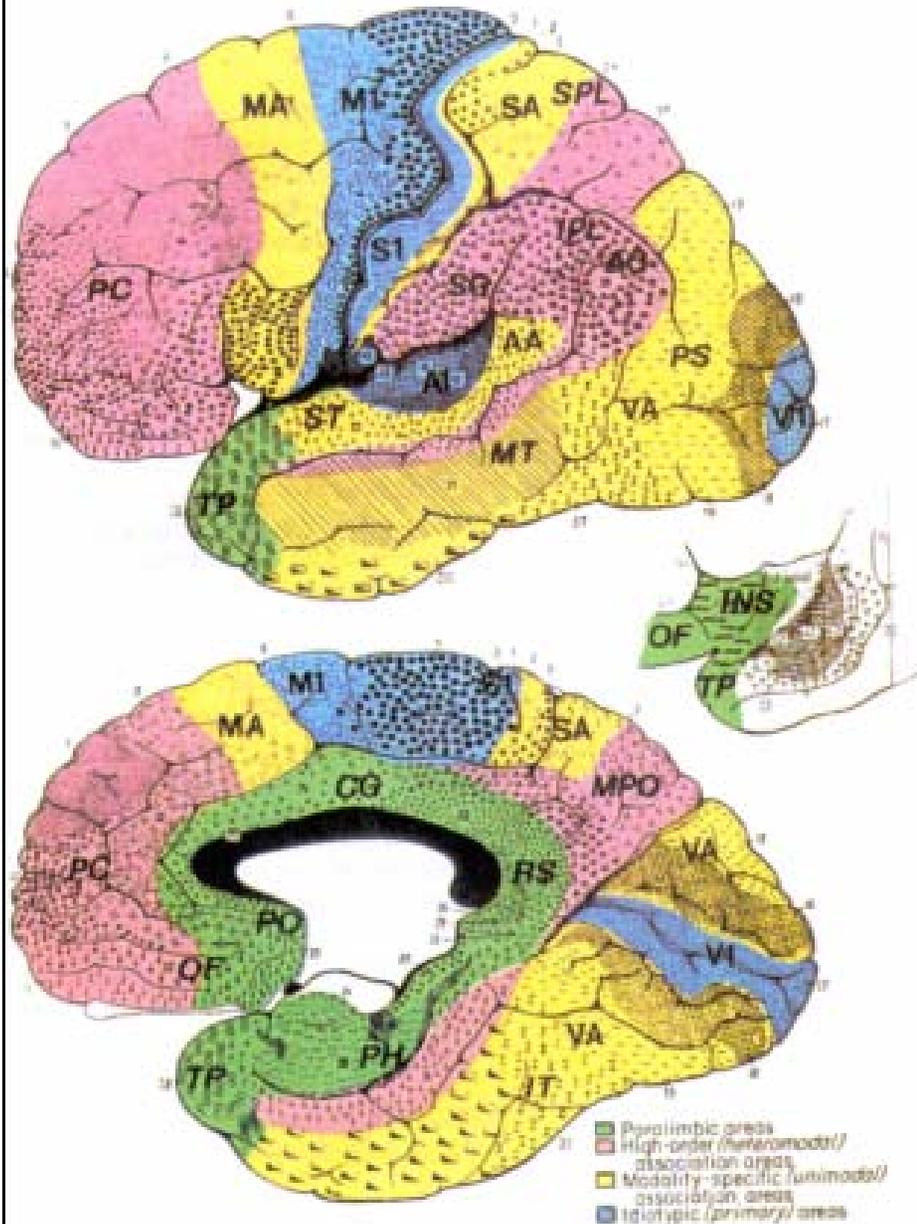


# Neocortex

- 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)

	Frontal	Temporal	Parietal	Occipital
Major	Motor planning	Primary auditory cortex (lateral)	Tactile sensation	Vision
Functions	Voluntary movement	Language recognition (left lateral)	Heteromodal sensory association	Visual perception and association
	Social behavior and judgement	Auditory prosody (right lateral)	Visuospatial function (right)	
	Motivation	Memory (medial)	Some elements of praxis (right)	
	Complex cognition	Fight/flight reactions (medial)	Reading (left)	
	Language production (left)	Smell (medial)	Calculation (left)	
	Language/motor prosody (right)		Stereognosis (left)	

**Table 2.1** The major divisions of the neocortex, including partial lists of the major functions subserved by each neocortical area.

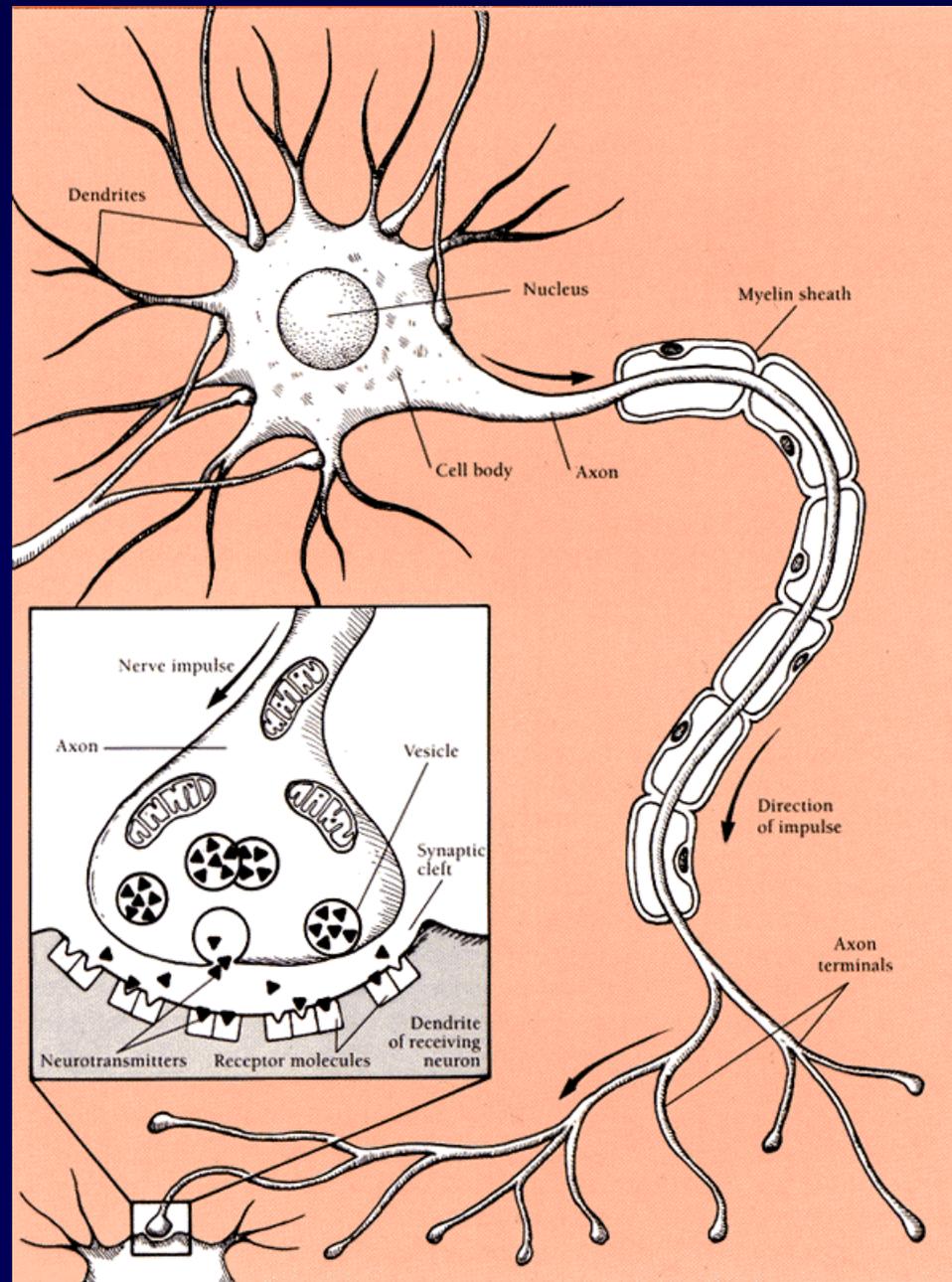
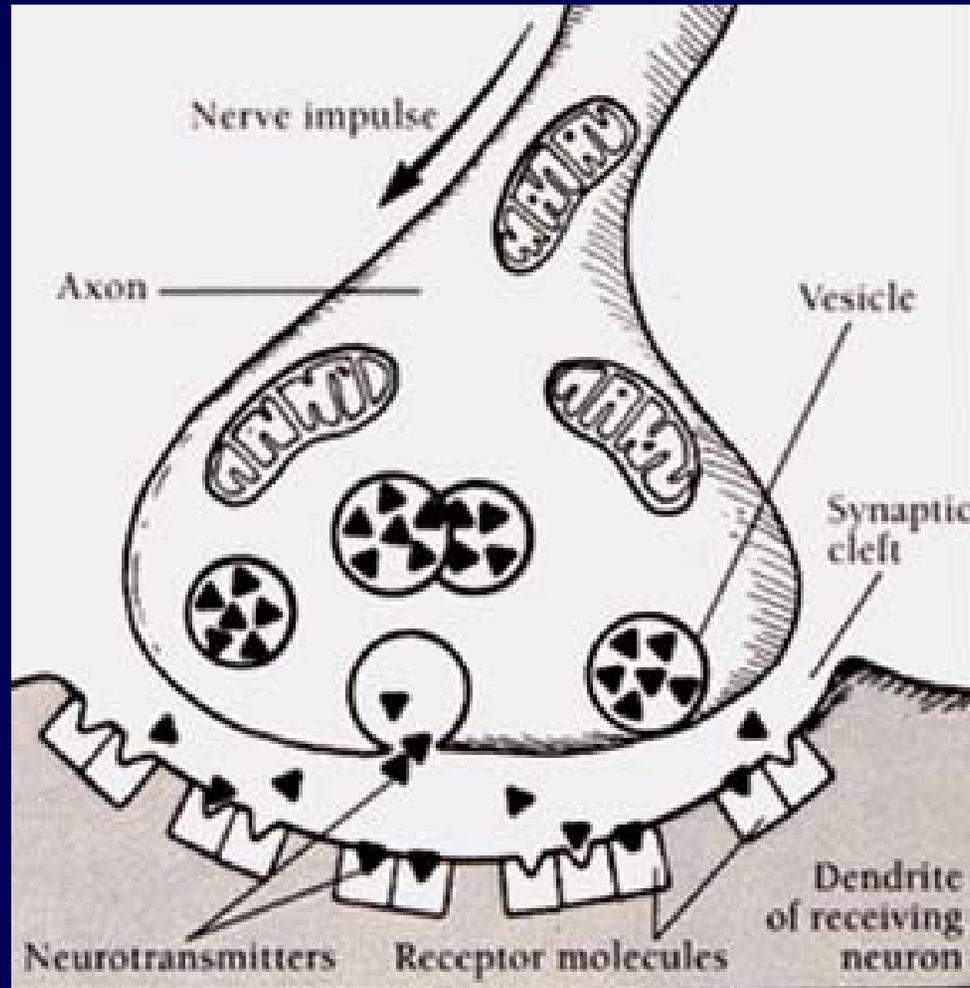


Illustration by Lydia Kibiuk

# Neurotransmission at the Synapse



Electro-  
neurochemical  
coupling

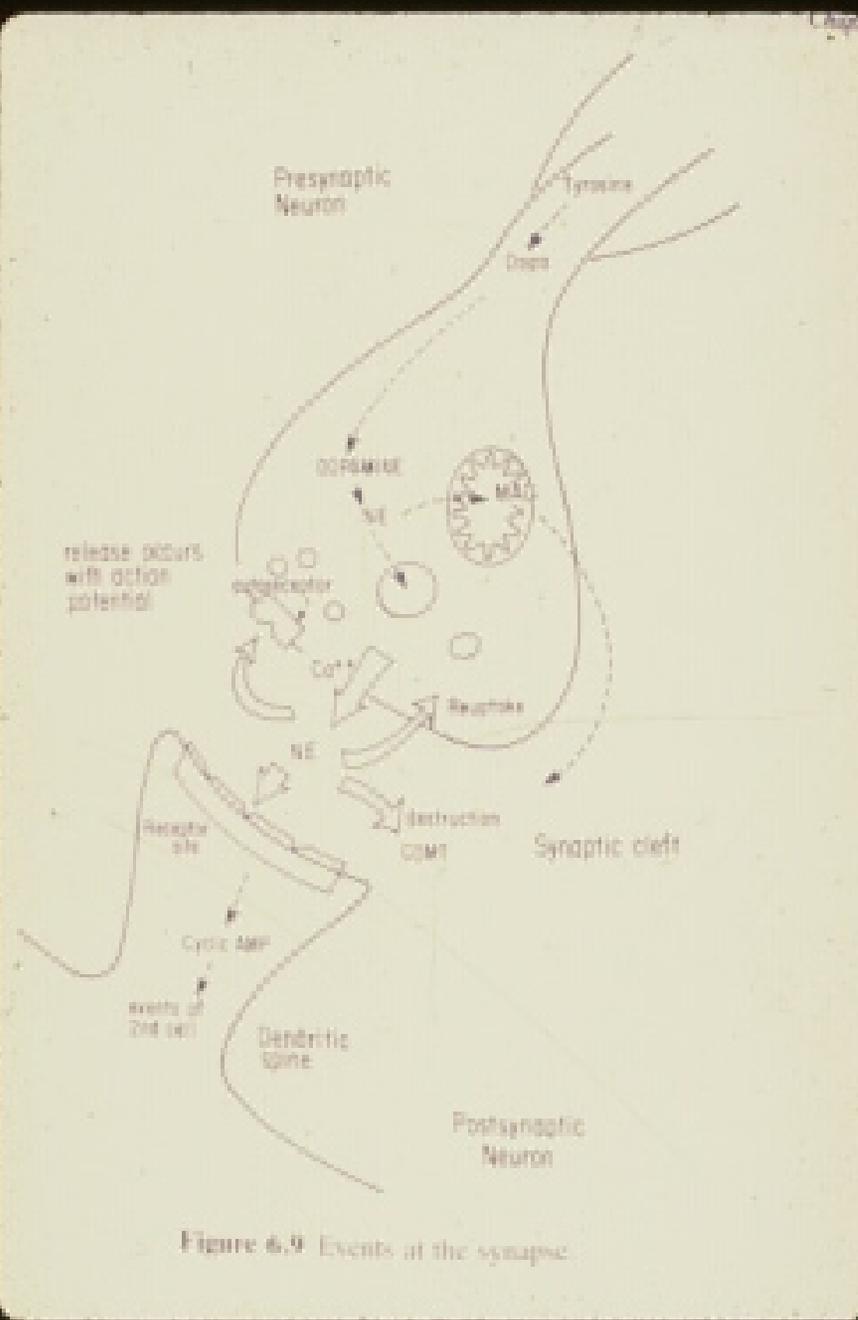
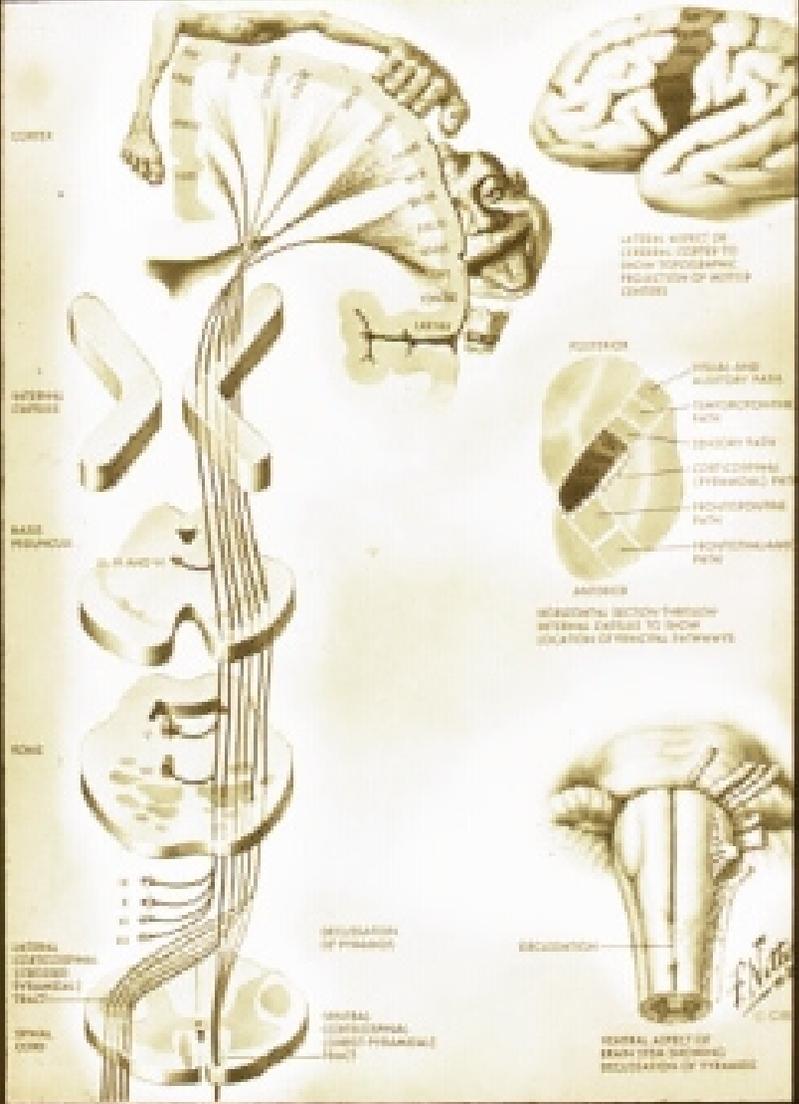
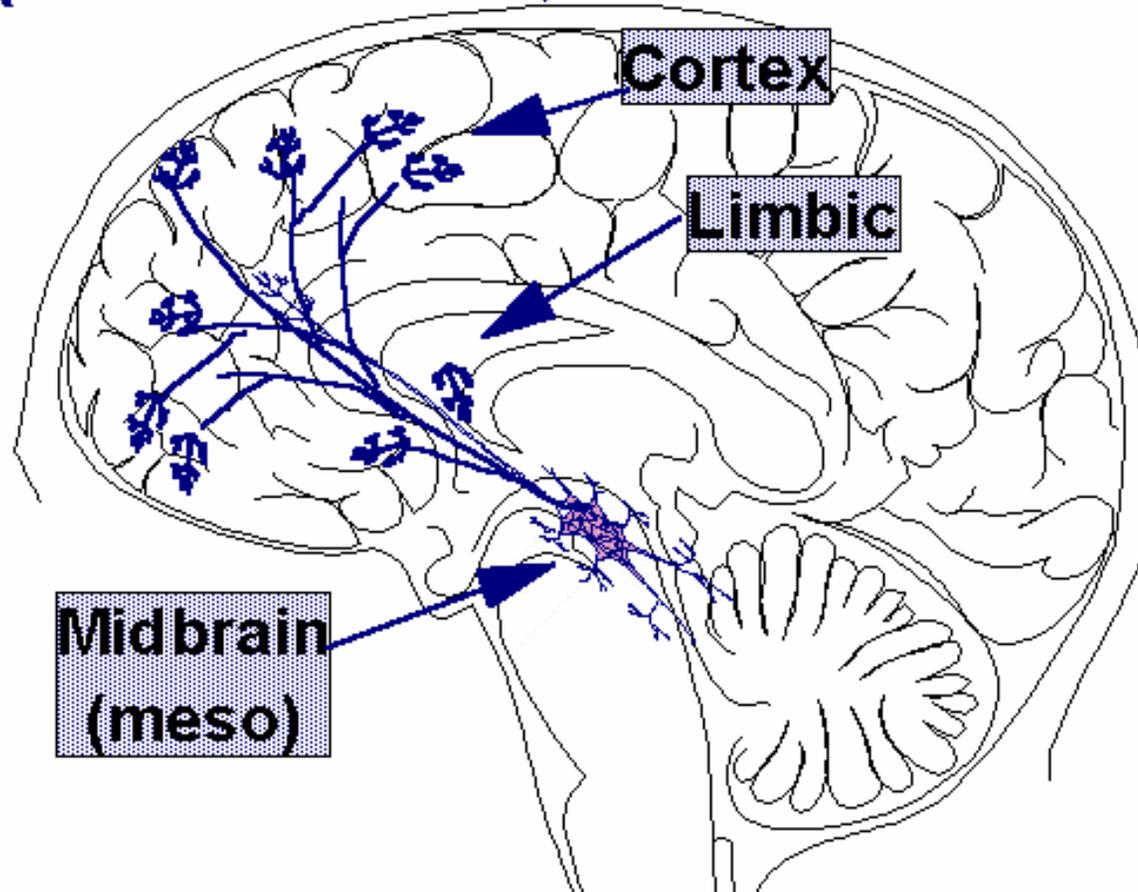


Figure 6.9 Events at the synapse



# Dopamine and Norepinephrine

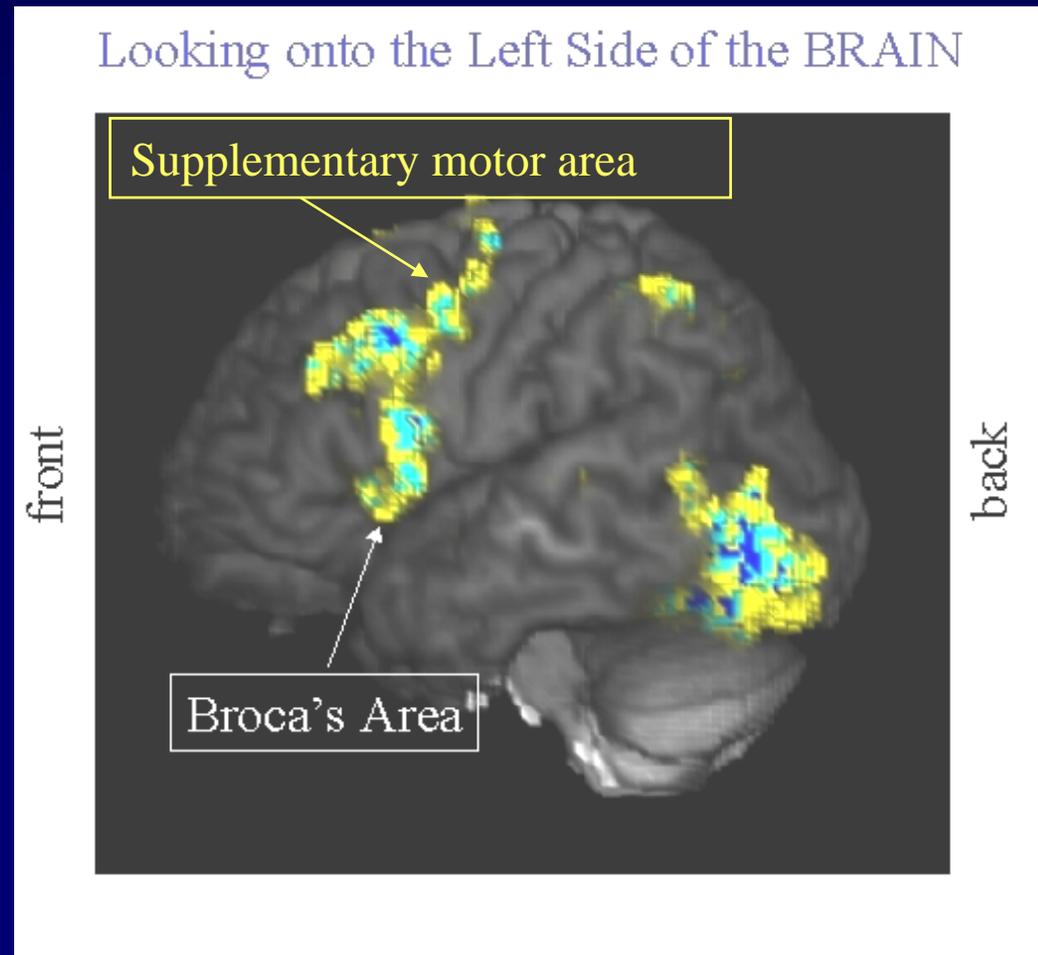
## Dopamine Pathways (meso-limbic; meso-cortical)

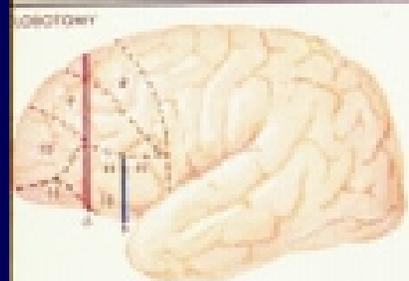
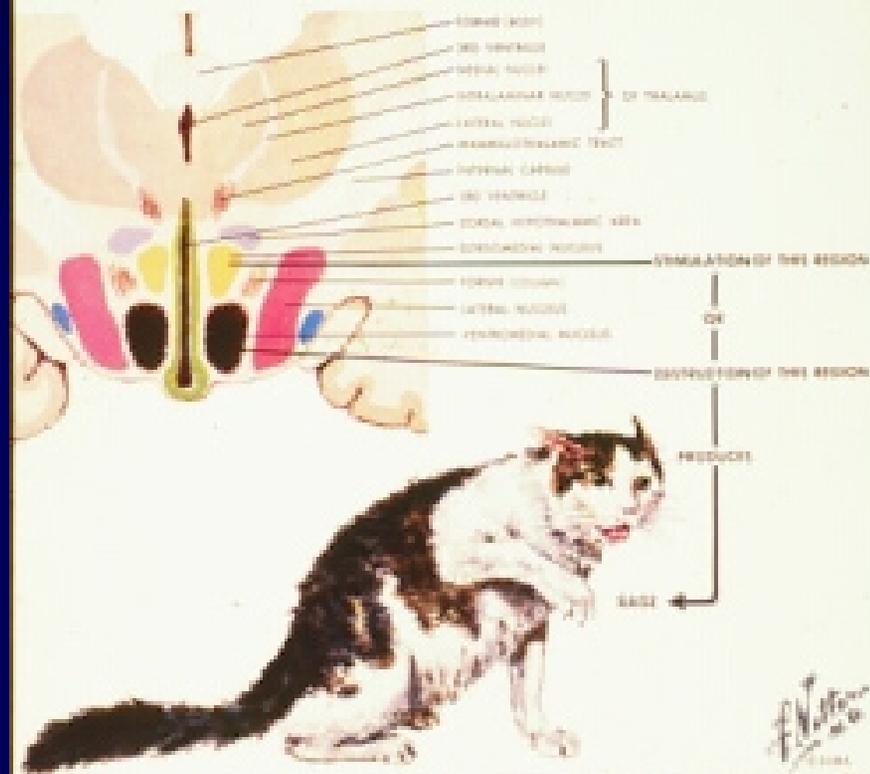


(From <http://www.onu.edu/user/FS/tfaulkner/Dopamine.htm>)

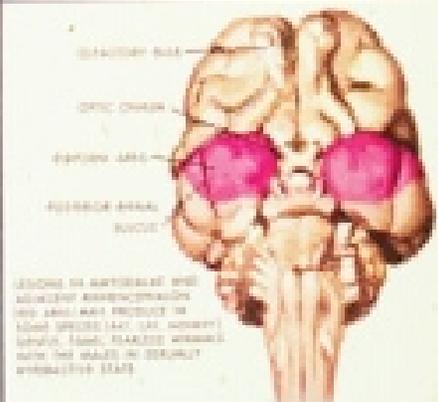
# Language: Fluency

- **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



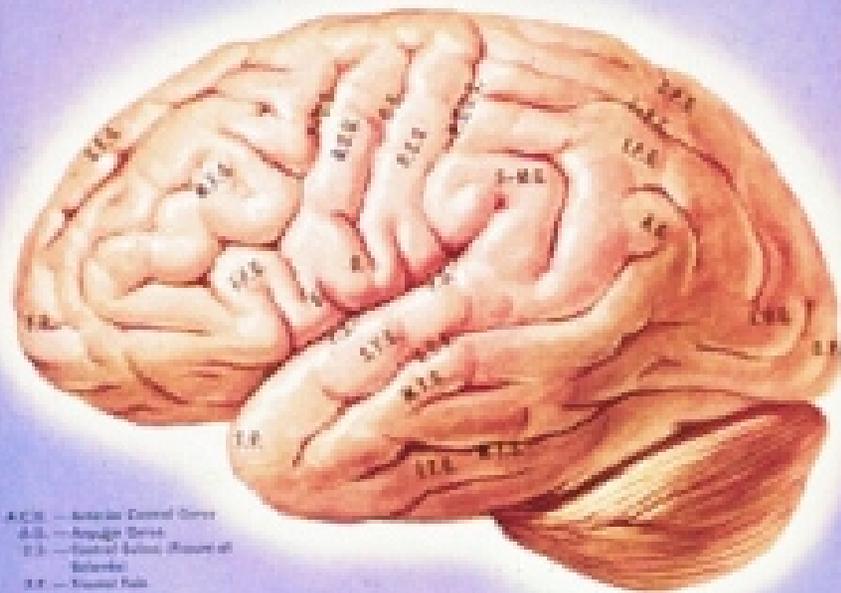


1. ANATOMICAL SITE OF THE LATERAL CINGULATE GYRUS  
 INDICATED WITH RED LINE  
 2. ANATOMICAL SITE OF THE INTERNAL CAPSULE  
 INDICATED WITH BLUE LINE  
 3. ANATOMICAL SITE OF THE MAMMILLOTHALAMIC TRACT  
 INDICATED WITH GREEN LINE  
 4. ANATOMICAL SITE OF THE MAMMILLARY NUCLEUS  
 INDICATED WITH YELLOW LINE  
 5. ANATOMICAL SITE OF THE MEDIAL NUCLEUS  
 INDICATED WITH PURPLE LINE  
 6. ANATOMICAL SITE OF THE LATERAL NUCLEUS  
 INDICATED WITH LIGHT BLUE LINE  
 7. ANATOMICAL SITE OF THE CORPUS CALLOSUM  
 INDICATED WITH DARK BLUE LINE

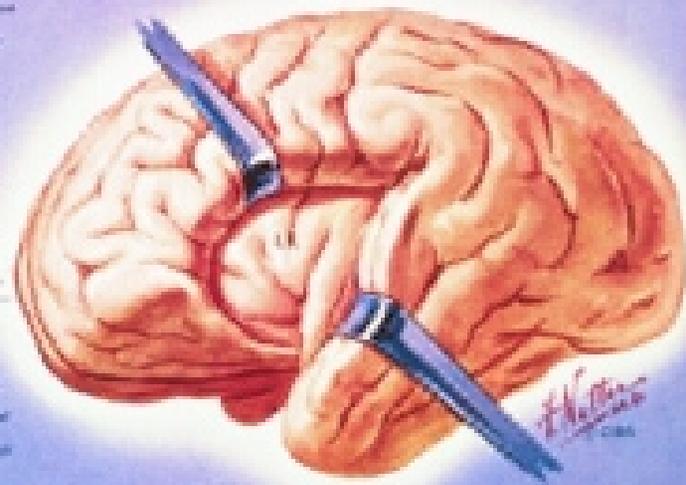


# Impact of Regional Injury on Behavior

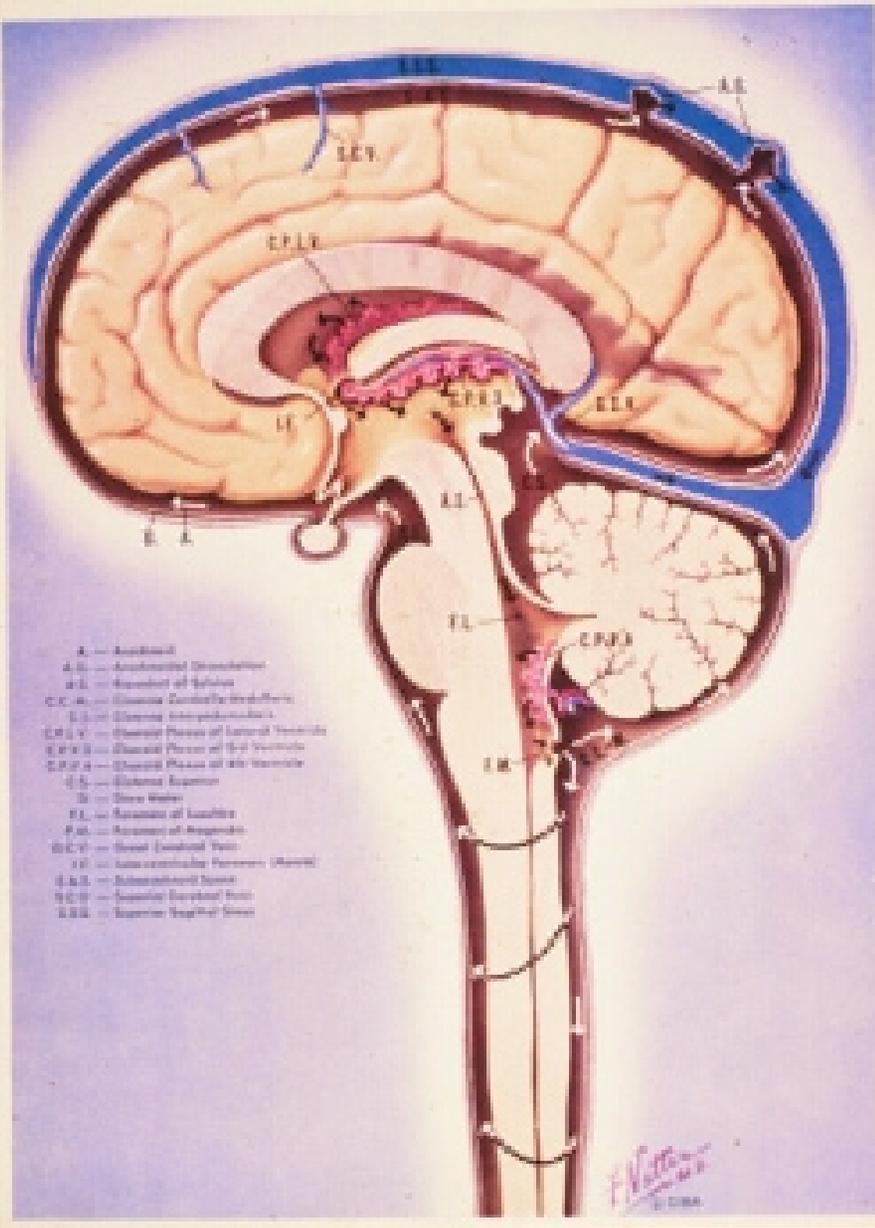
- **Frontal Lobe Syndromes**
- **Temporal Lobe Syndromes**
- **Parietal Lobe Syndromes**
- **Basal Ganglia Syndromes**



- A.C.G. — Anterior Central Gyrus
- A.S. — Angular Gyrus
- C.S. — Central Sulcus (Fissure of Rolando)
- C.P. — Central Pole
- C.S. — Fissure of Sylvius
- L.P.G. — Lateral Frontal Gyrus
- L.P.S. — Lateral Parietal Gyrus
- L.S. — Lower Parietal Sulcus
- I.P. — Island of Reil
- L.T.C. — Lateral Temporal Gyrus
- L.O.G. — Lateral Occipital Gyrus
- M.F.G. — Middle Frontal Gyrus
- M.T.G. — Middle Temporal Gyrus
- M.T.S. — Middle Temporal Sulcus
- O. — Occipital Pole
- O.P. — Occipital Pole
- P.C.G. — Posterior Central Gyrus
- P.C.S. — Pre Central Sulcus
- P.O.C.S. — Post Occipital Sulcus
- S.P.S. — Superior Parietal Sulcus
- S.M.S. — Superior Marginal Sulcus
- S.P.G. — Superior Frontal Gyrus
- S.T.G. — Superior Temporal Gyrus
- S.T.S. — Superior Temporal Sulcus
- S.P. — Superior Pole



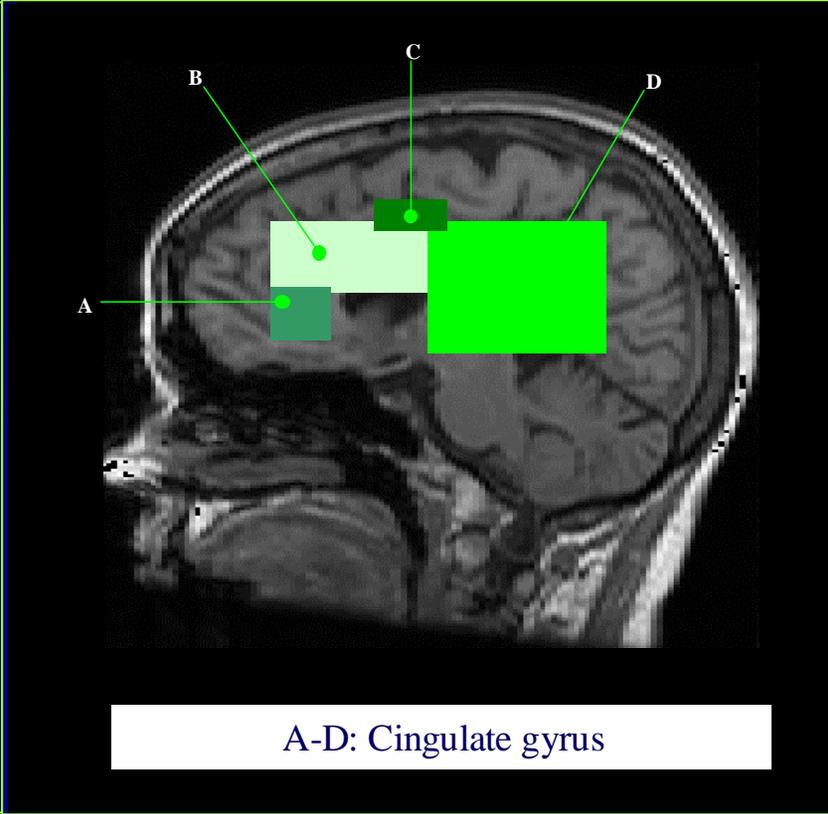
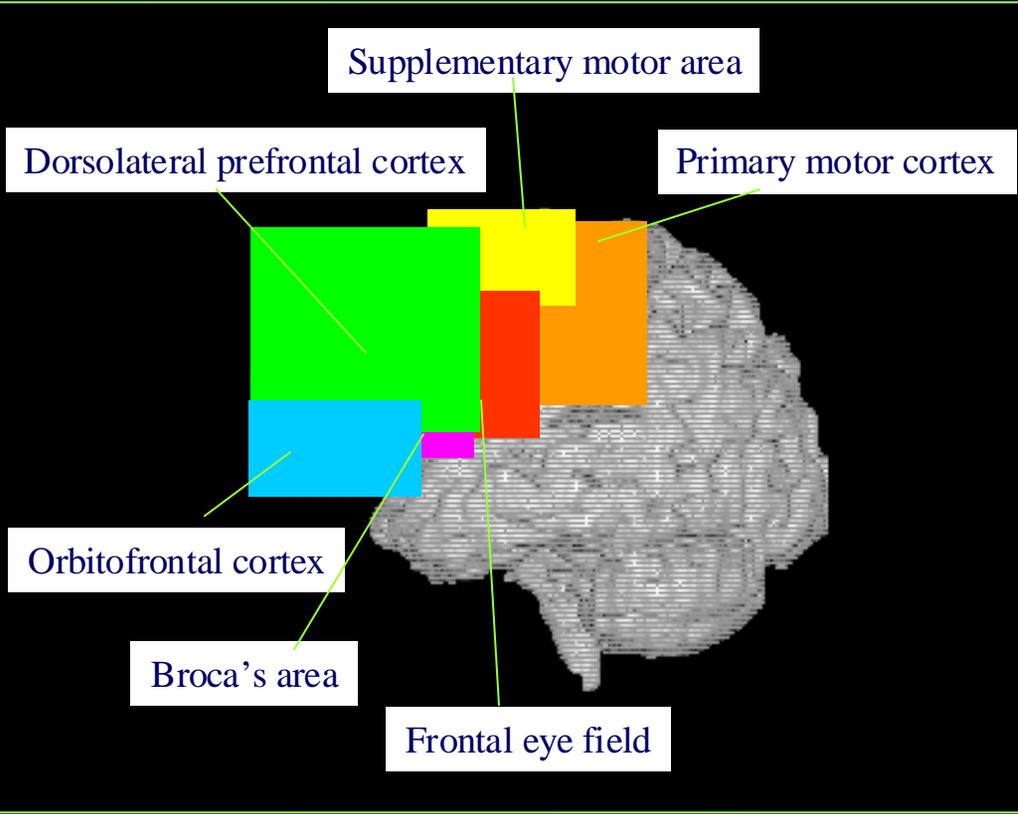
*W. H. Jones*  
1906



- A — Axonhilus
- A.S. — Arachnoid Granulation
- A.L. — Arachnoid of Lohse
- C.C.M. — Corpus Callosum
- C.P.L. — Corpus Callosum
- C.P.V. — Corpus Callosum
- C.P.S. — Corpus Callosum
- C.P.I. — Corpus Callosum
- S — Sinus
- S.V. — Sinus
- F.L. — Funiculus
- F.M. — Funiculus
- S.C.V. — Sinus
- X — Xanthochloric Serum
- S.S.S. — Subarachnoid Space
- S.C.S. — Superior Sagittal Sinus
- S.C.P. — Superior Sagittal Sinus
- S.S.P. — Superior Sagittal Sinus

F. Netter  
M.D.

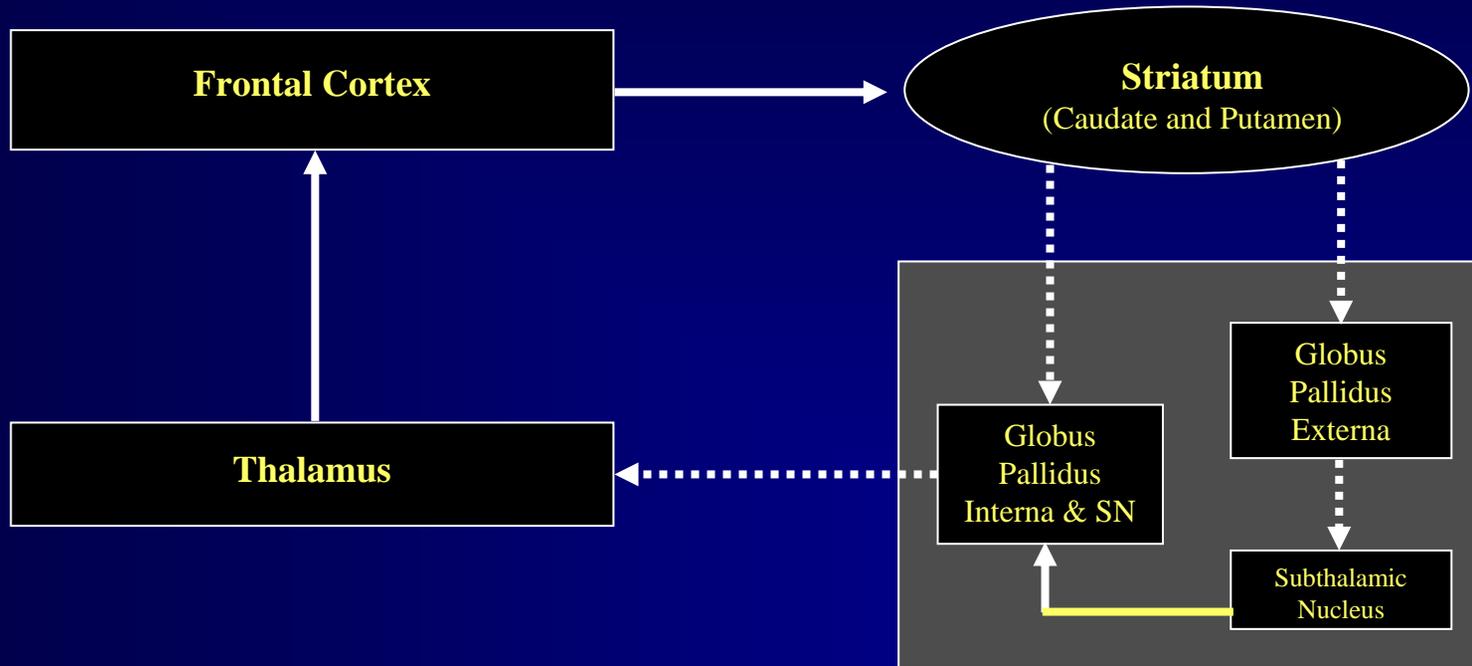
# Frontal Cortex



# 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



## Key:

-  Excitatory Pathway
-  Inhibitory Pathway

**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**

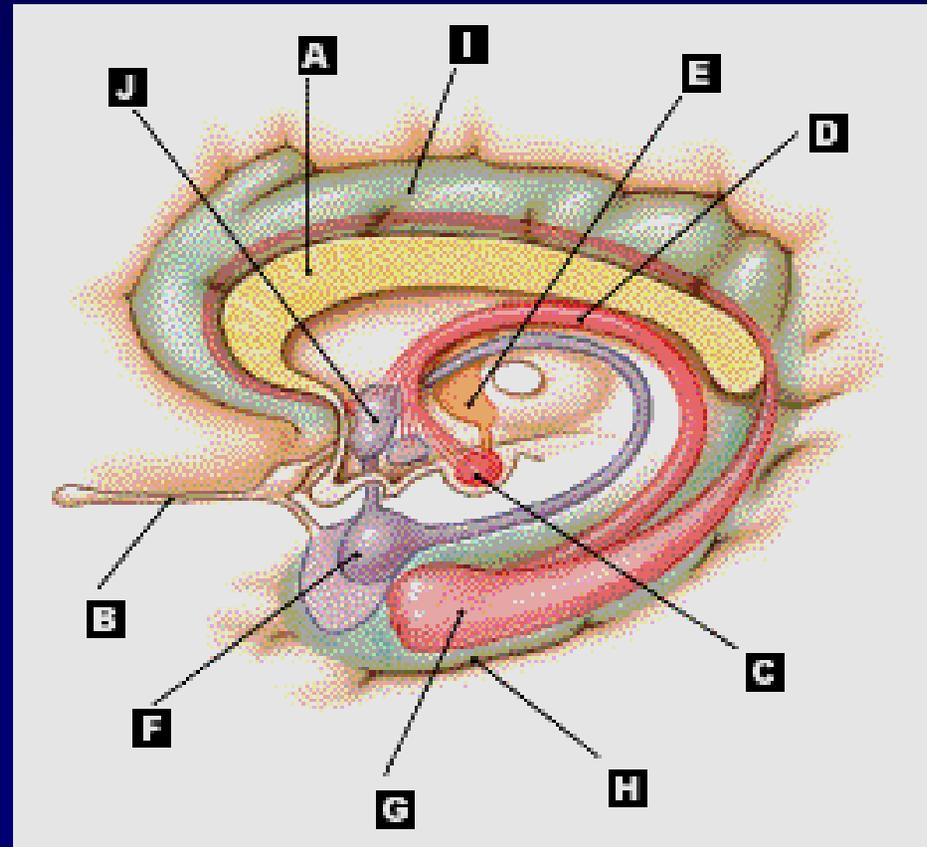


# Limbic and Paralimbic Anatomy

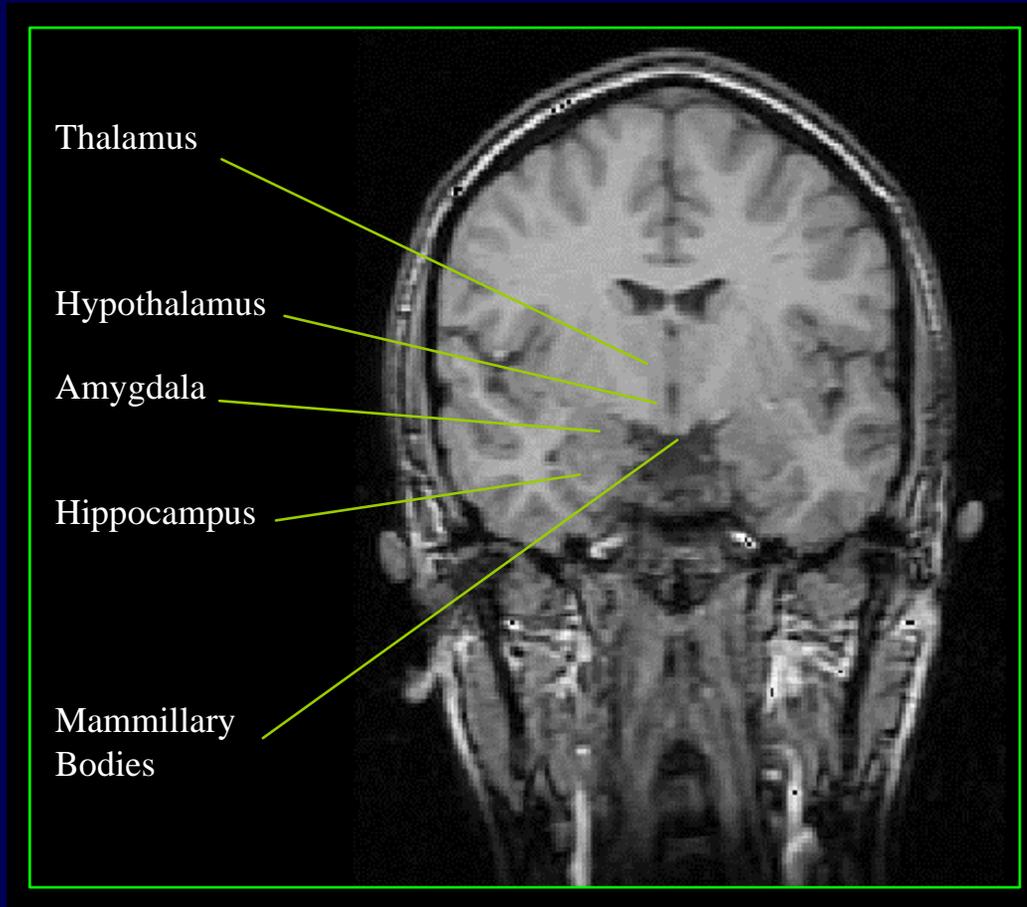
- Important concepts:
  - (1) The limbic system includes a lateral and medial circuit
  - (2) Basic (or “unconscious”) emotion is represented in a ventral limbic-paralimbic network
  - (3) Emotional awareness (“conscious”) emotion is represented in a dorsal limbic-paralimbic-cortical network
  - (4) 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.

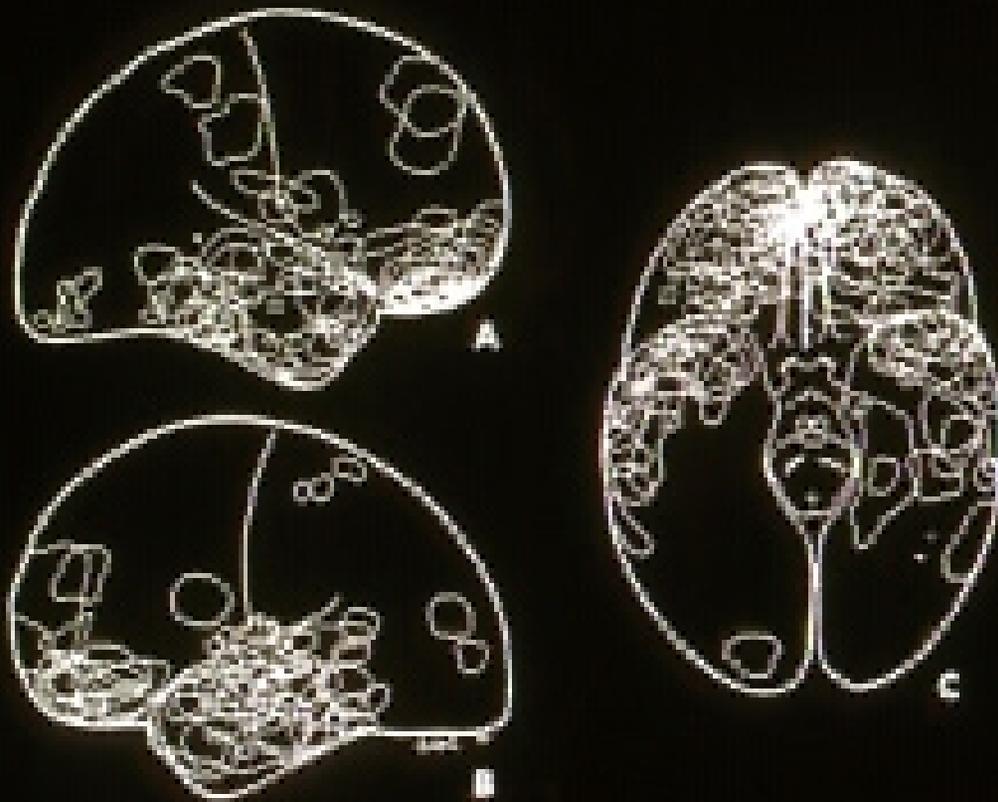


Figure 34-4

*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 Cowwille.)*

# 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”

Tucker, G.J., Price, T.R.P., Johnson, V.B., McAllister, T.W.:  
Phenomenology of temporal lobe dysfunction: A link  
to atypical psychosis---A series of cases. J of Nervous  
and Mental Disease, 174(6):348-356, 1986.

# 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%

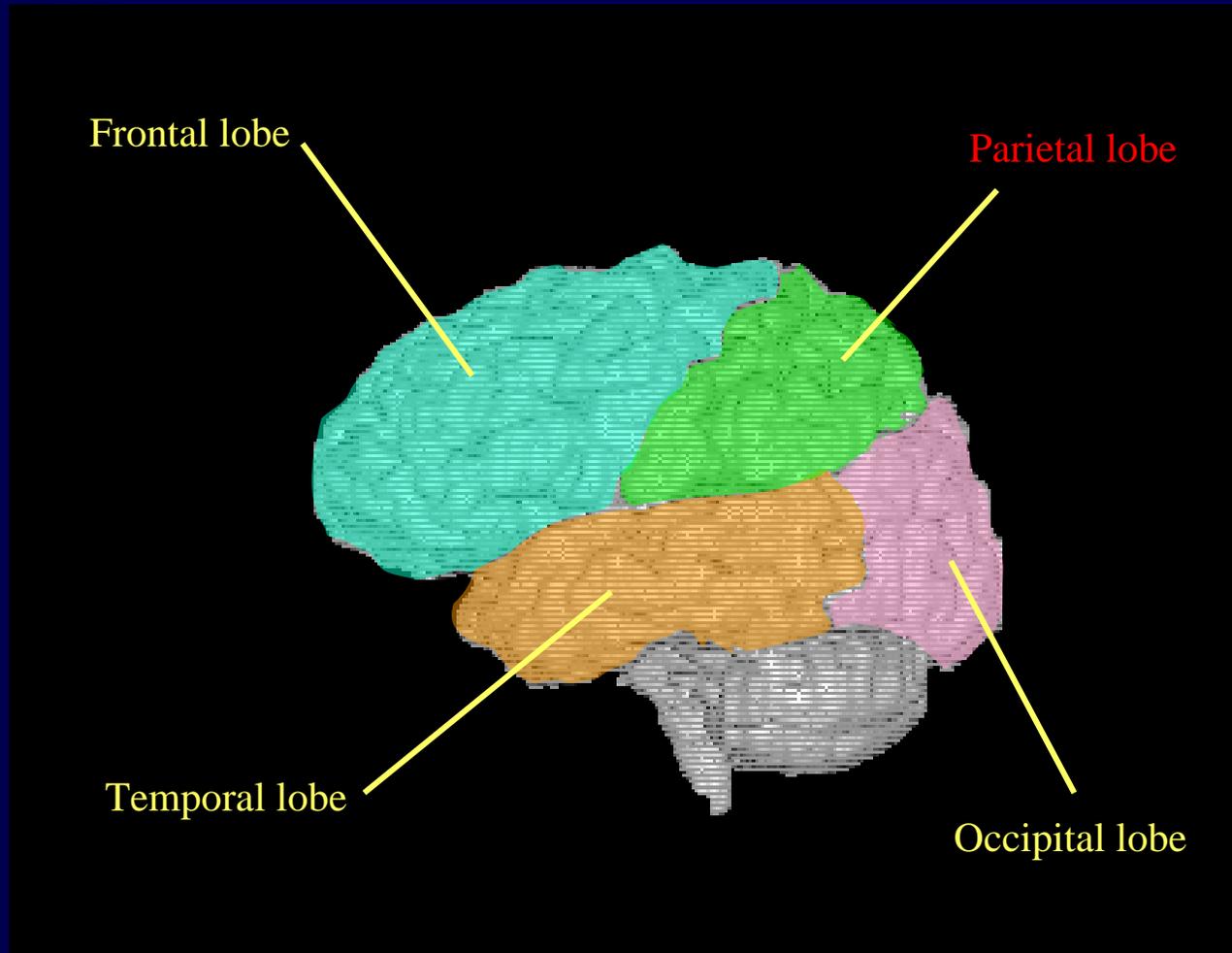
- visual – 40%

Tucker, G.J., Price, T.R.P., Johnson, V.B., McAllister, T.M.: Pharmacology of temporal lobe dysfunction: A link to atypical psychosis---A series of cases. J of Nervous and Mental Disease, 174(6):348-356, 1986.

# Atypical Partial Complex Seizures

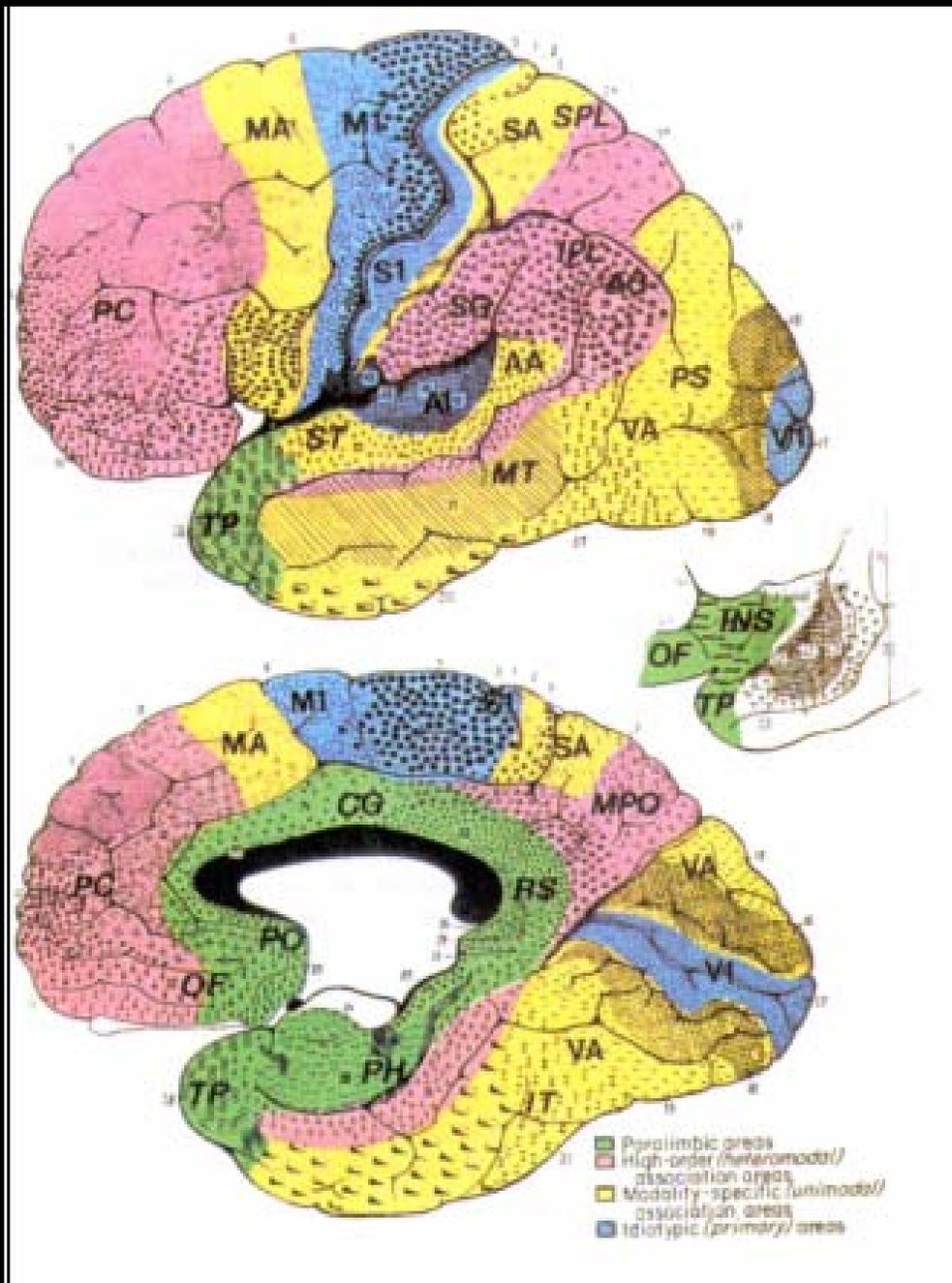
- paroxysmal nature of spells
- more normal function in between events

# 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

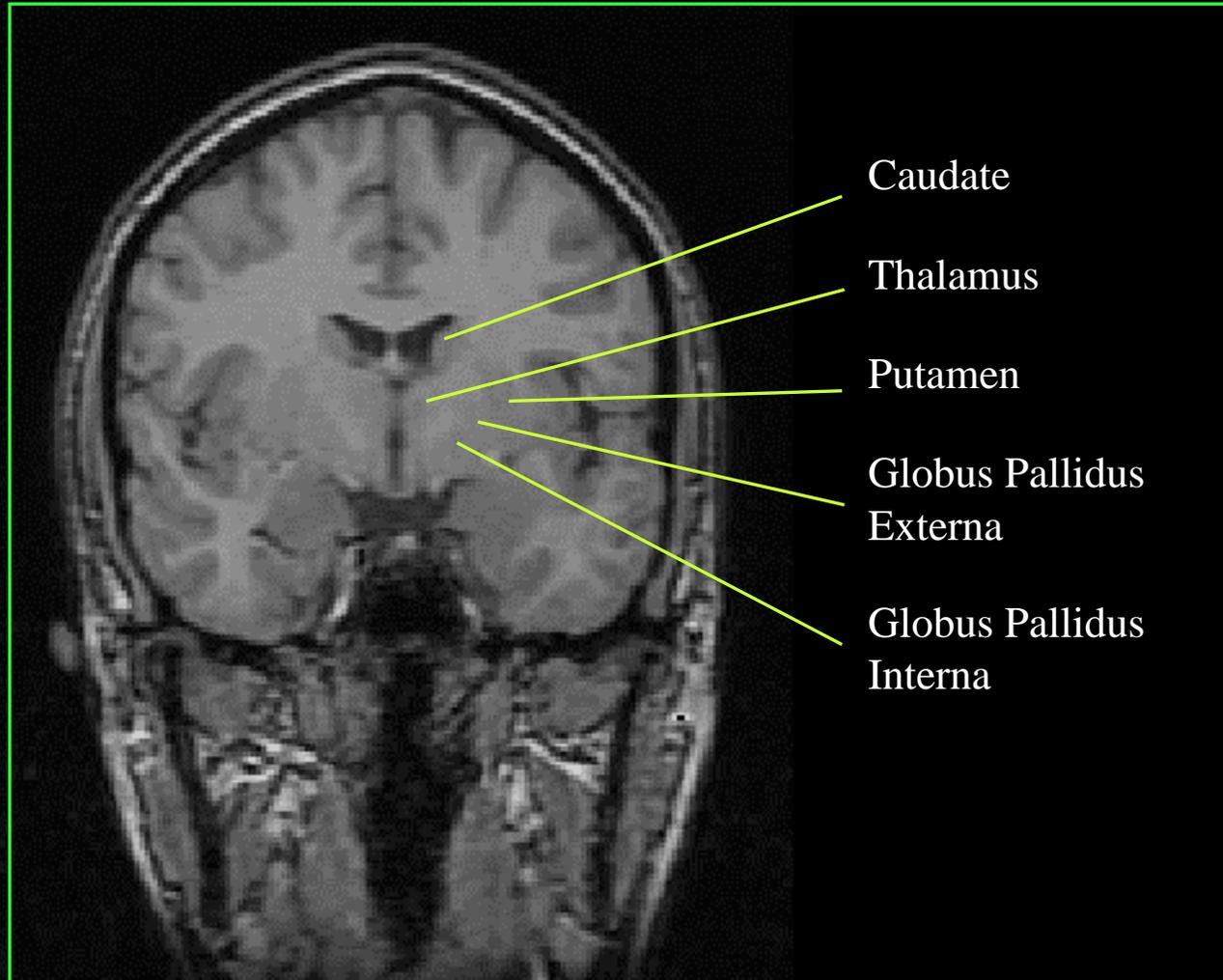
- **Confusional States**
- **Alienation Experiences**
- **Prosopagnosia**
- **Delusional Syndromes**  
(Capgras “Impostor” Syndrome)
- **Denial of Deficits**
- **Spatial neglect**



# Basal Ganglia

- Caudate
- Putamen
- Globus pallidus
  - interna
  - externa
- Substantia nigra
- Subthalamic nucleus

# Review of Neurobehavioral Anatomy



**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)

# Basal Ganglia & Other Sub-Cortical Regions

## 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

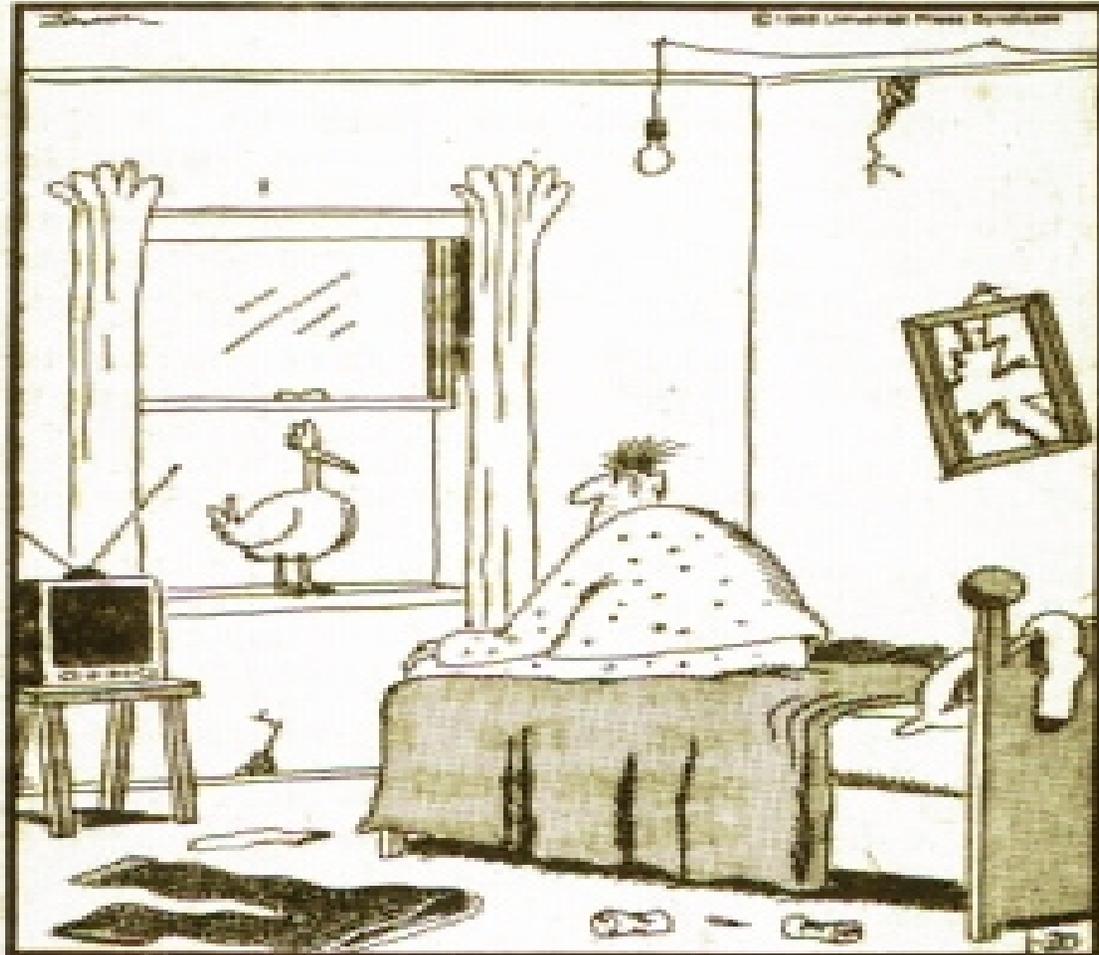
- Primary neuronal circuitry links the basal ganglia with orbito-frontal cortex, limbic system and thalamus
- Metabolic studies, lesion studies, and circuitry strongly suggest an important role for the basal ganglia in the genesis and maintenance of OCD symptoms

# 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 FAR SIDE



The Bluebird of Happiness long absent from his life,  
Ned is visited by the Chicken of Depression.

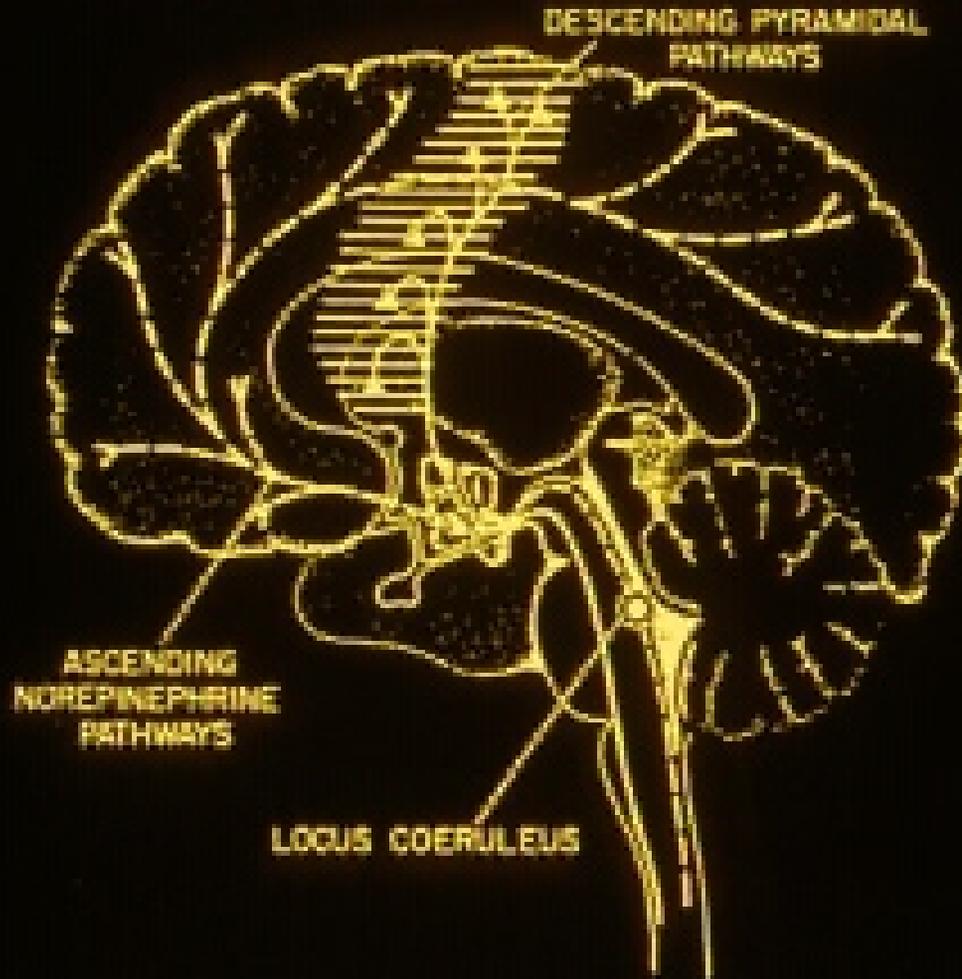
# LATERALITY OF CVA AND DEPRESSION

## 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

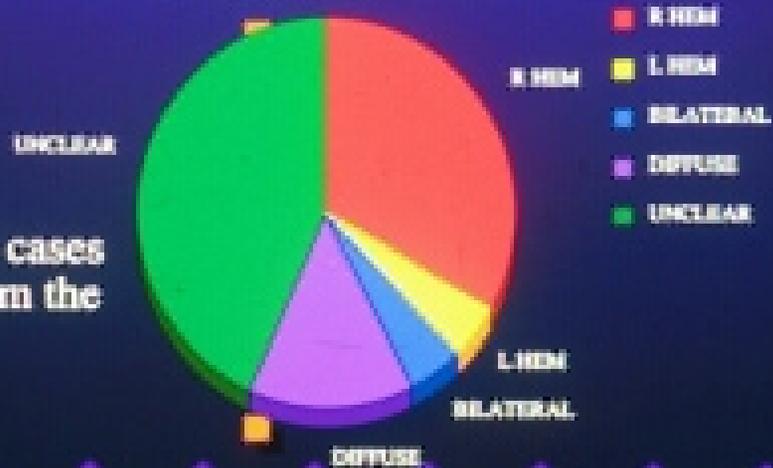
- Usual pattern is periodic bursts or clusters of sustained manic behaviors
- Some have reported “chronic mania”
- Can see rapid cycling pattern (4 or more episodes in a 12 month period)

# 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 work place 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

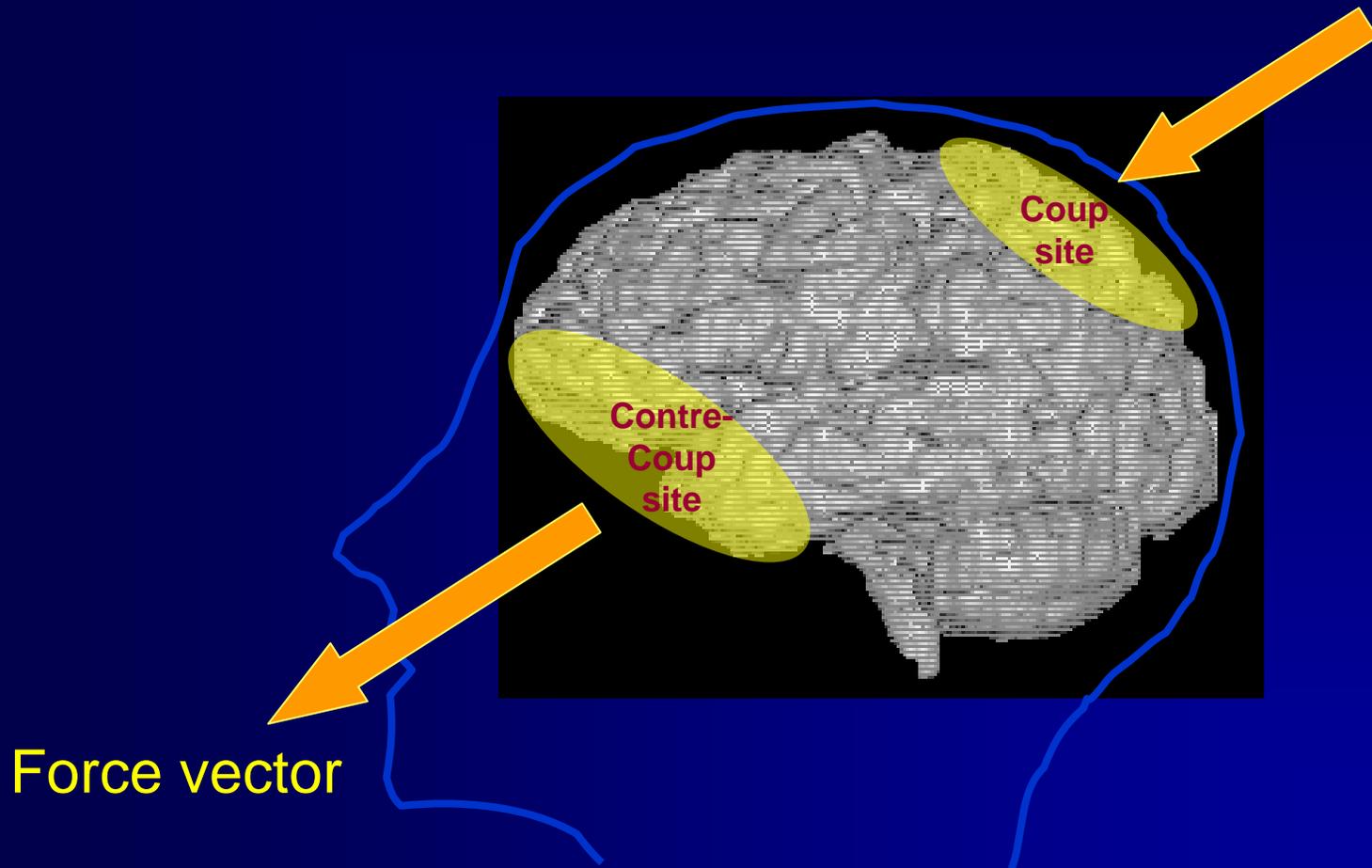
- **Right Parietal Infarct**
- **3 weeks later, developed a manic syndrome**

# IMPACT OF TYPE OF INJURY ON BEHAVIOR

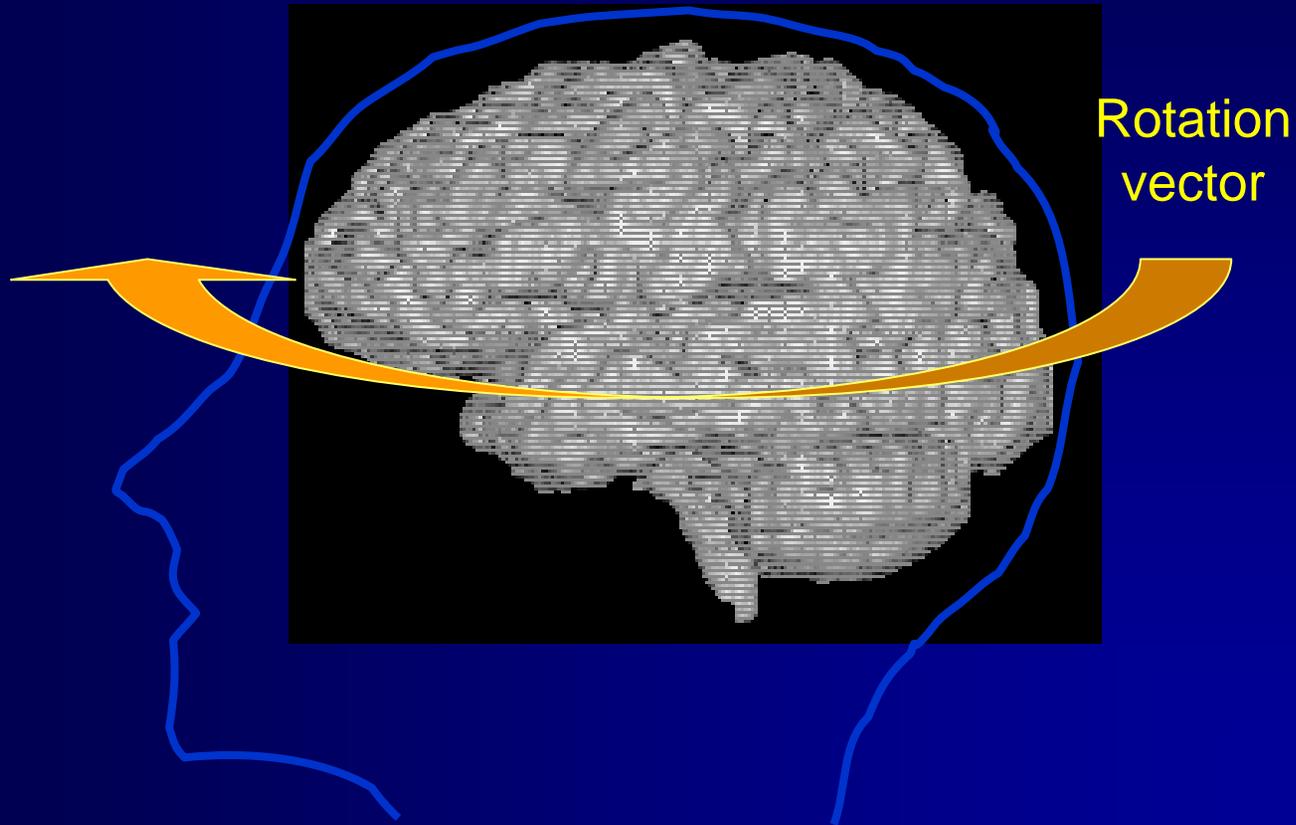
# What happens in a TBI?

- **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

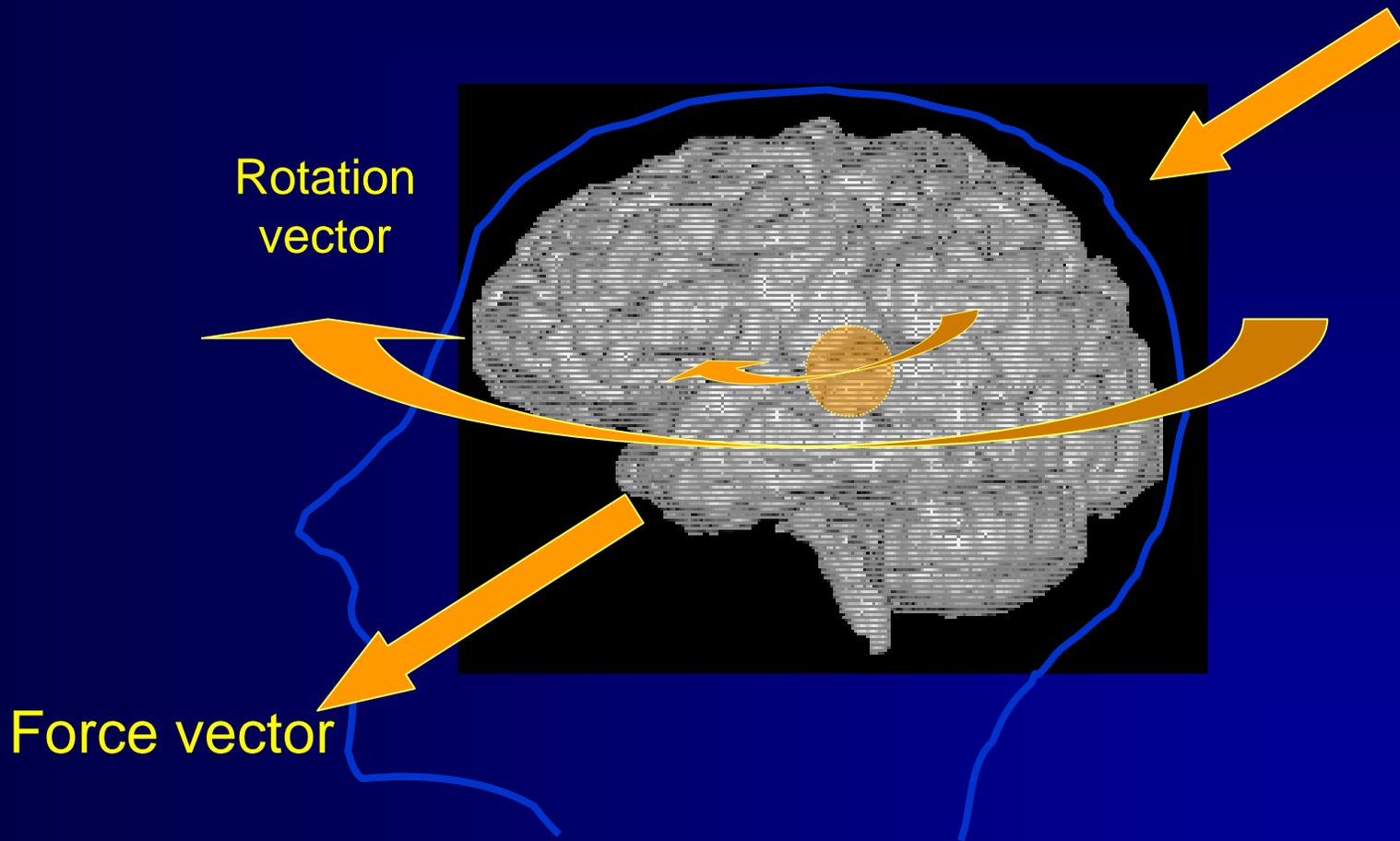
# Biomechanical Injury: Translation



# Biomechanical Injury: Rotation

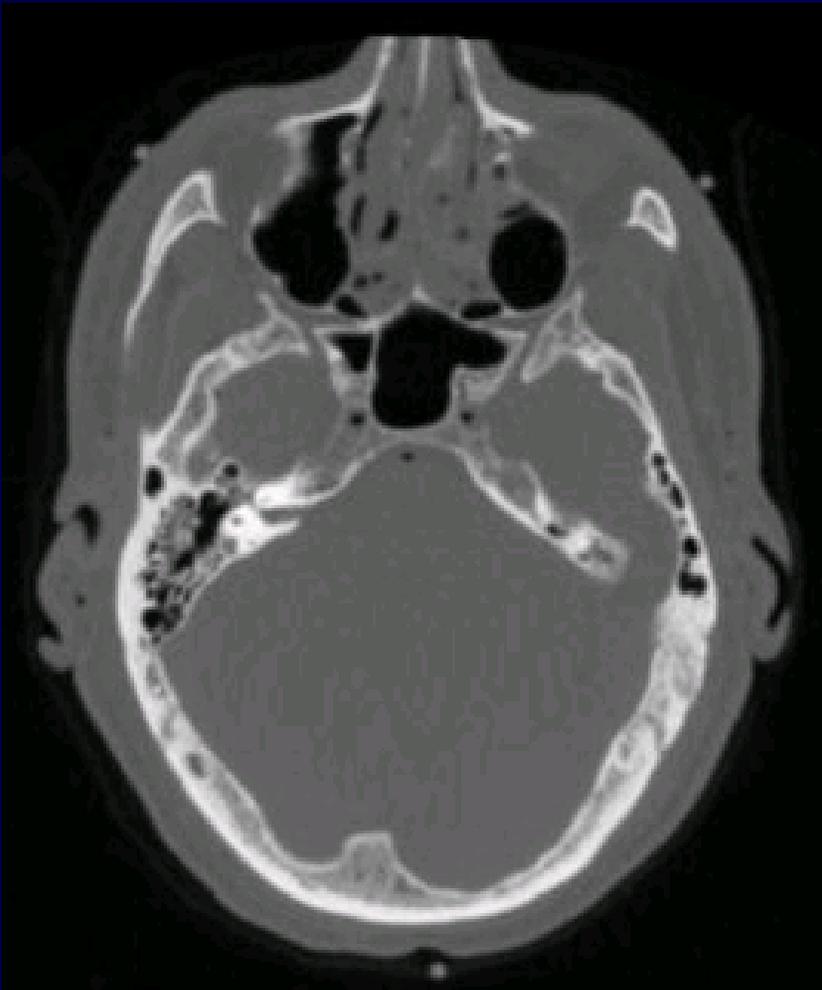


# Biomechanical Injury: Angular Acceleration



# CT Scanning: Examples

<http://nanonline.org/nandistance/mtbi/modules/patho/pathophysiology.html>

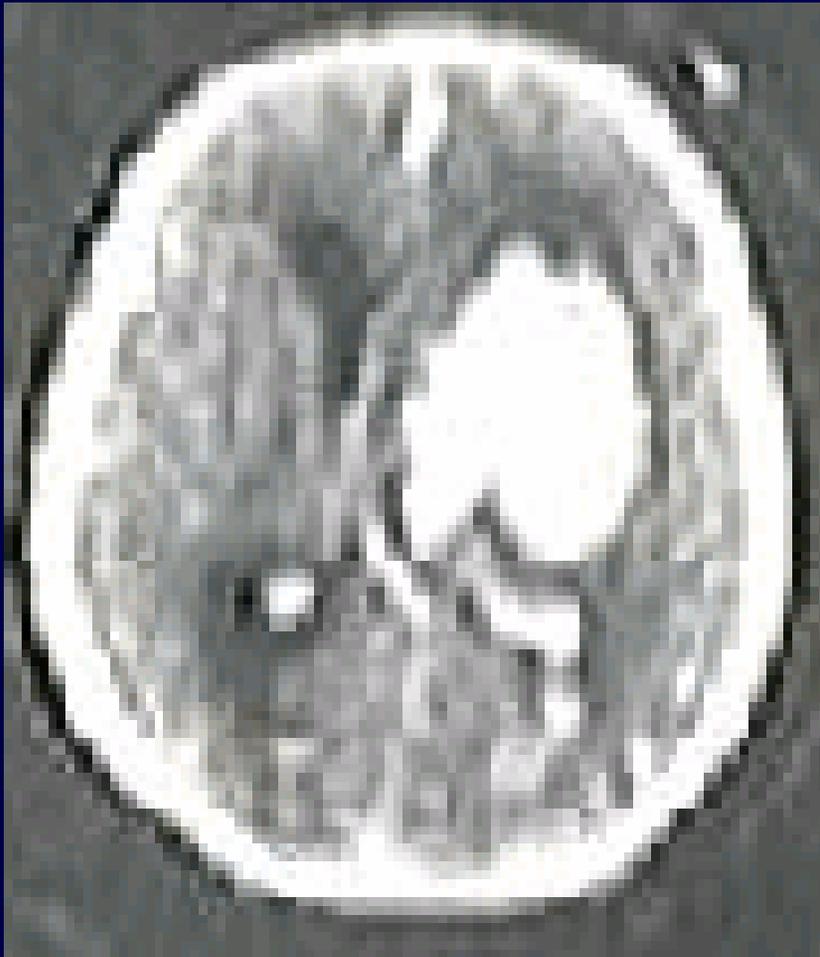


Normal Brain



TBI with Depressed Skull Fracture

# CT Scanning: Examples

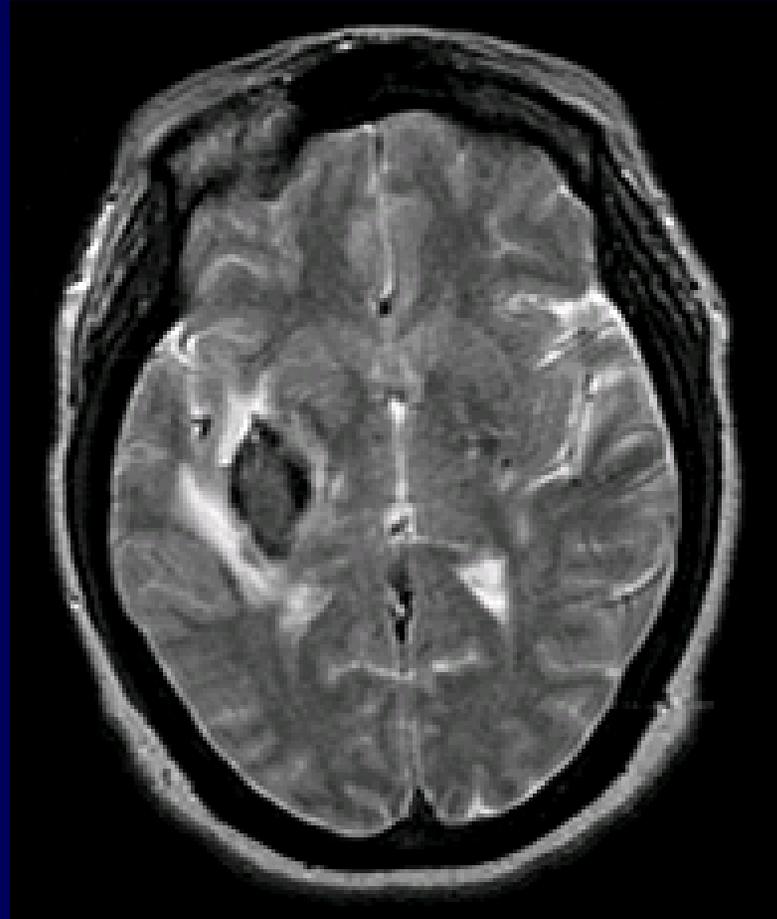


Hemorrhage w/mass effect



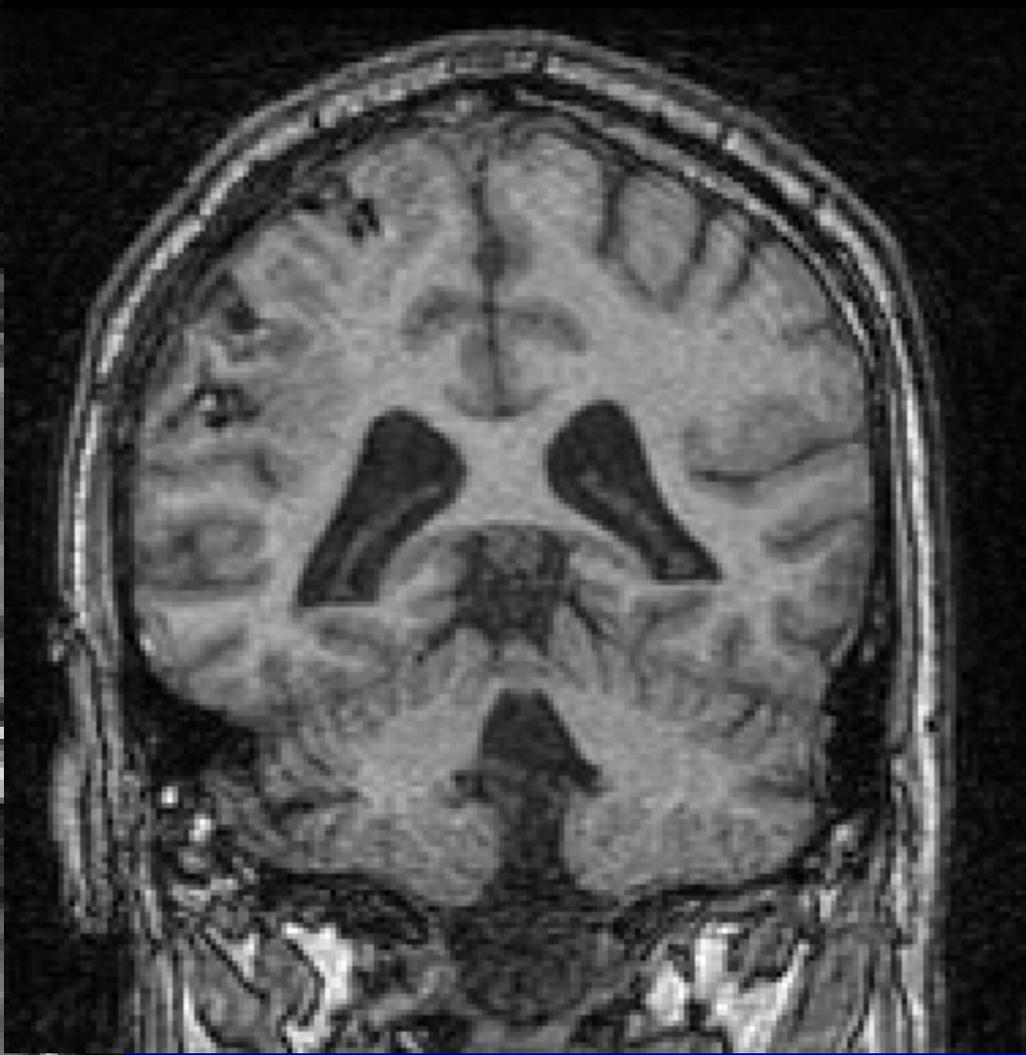
Subdural Hematoma

# MRI: Cerebral Hemorrhage



<http://www.med.harvard.edu/AANLIB/home.html>

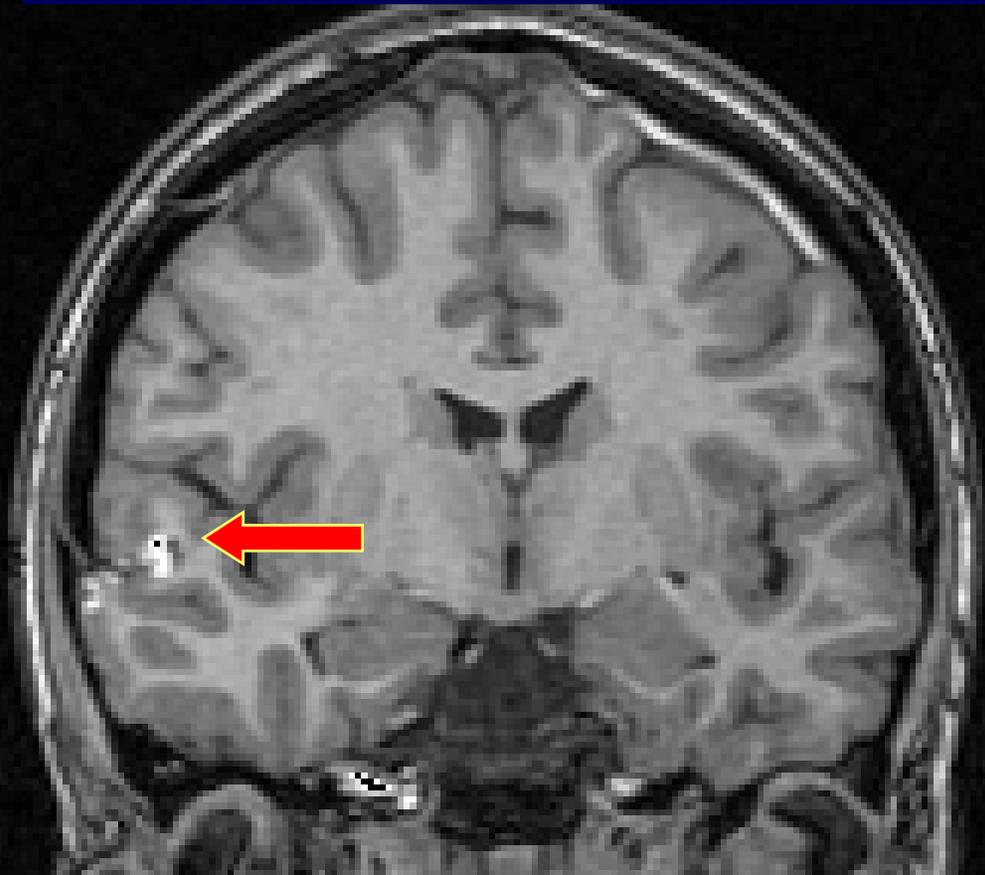
# MRI (T1): Hemorrhage & Hydrocephalus



# MRI (T1): Hematoma & Hemorrhage



**L frontal extra-axial  
subdural hematoma**



**R temporal hemorrhagic  
contusion; L hematoma**



A



B



C

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 Cowwille.)*

# 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

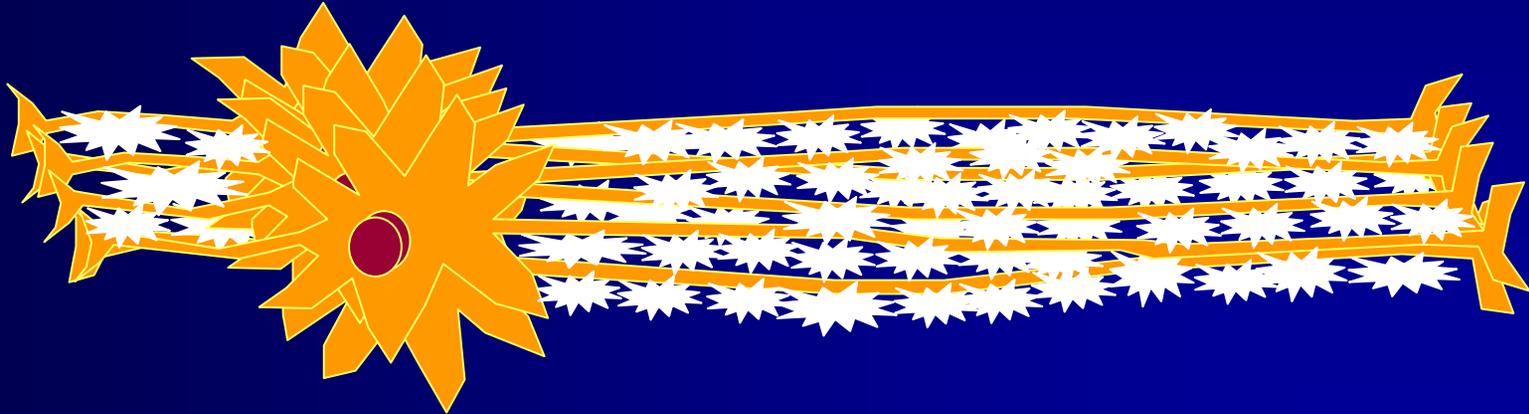
- **SECONDARY INJURY**: Occurs after the initial trauma, & as a result of the primary injury
  - Cerebral edema, cerebral infarcts
  - Cerebral anoxia
  - Cell injury and death from release of toxins from injured cells

# Biomechanical Injury

Pre-Injury

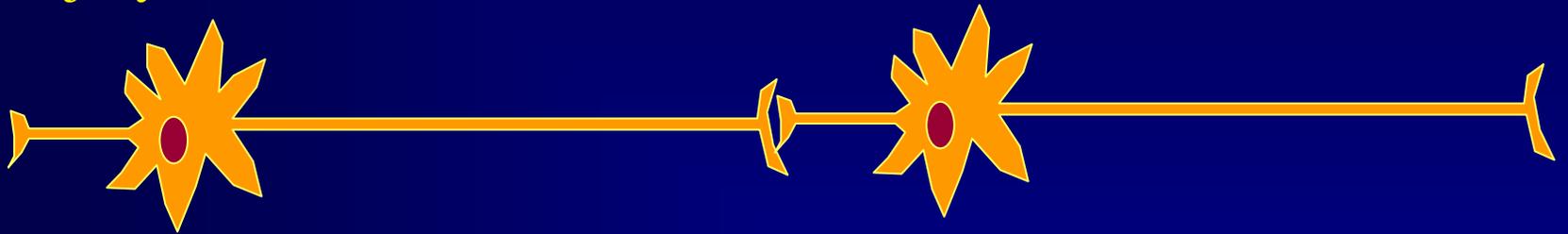


Acute Injury

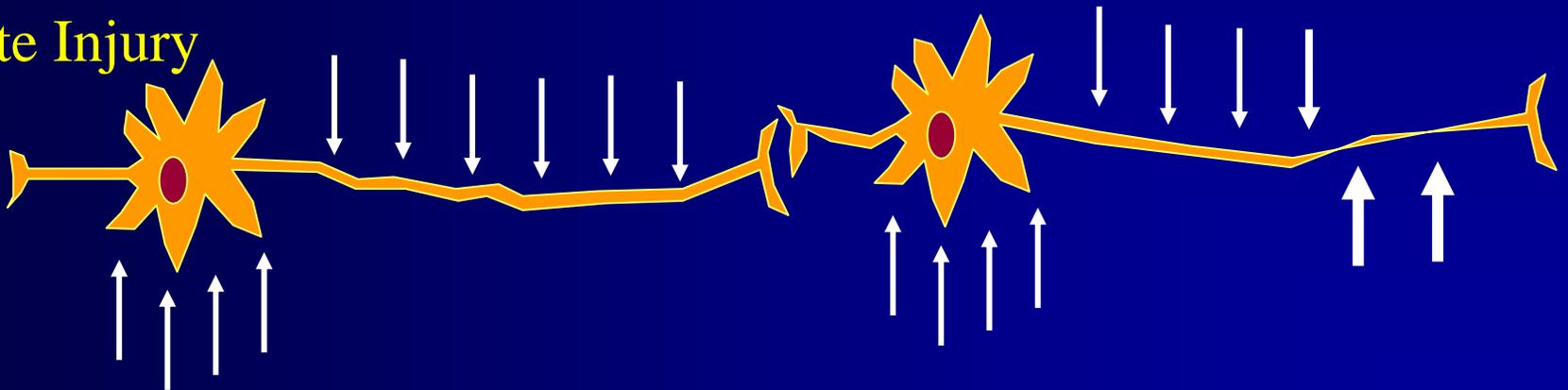


# Biomechanical Injury: Diffuse Axonal Injury

Pre-Injury

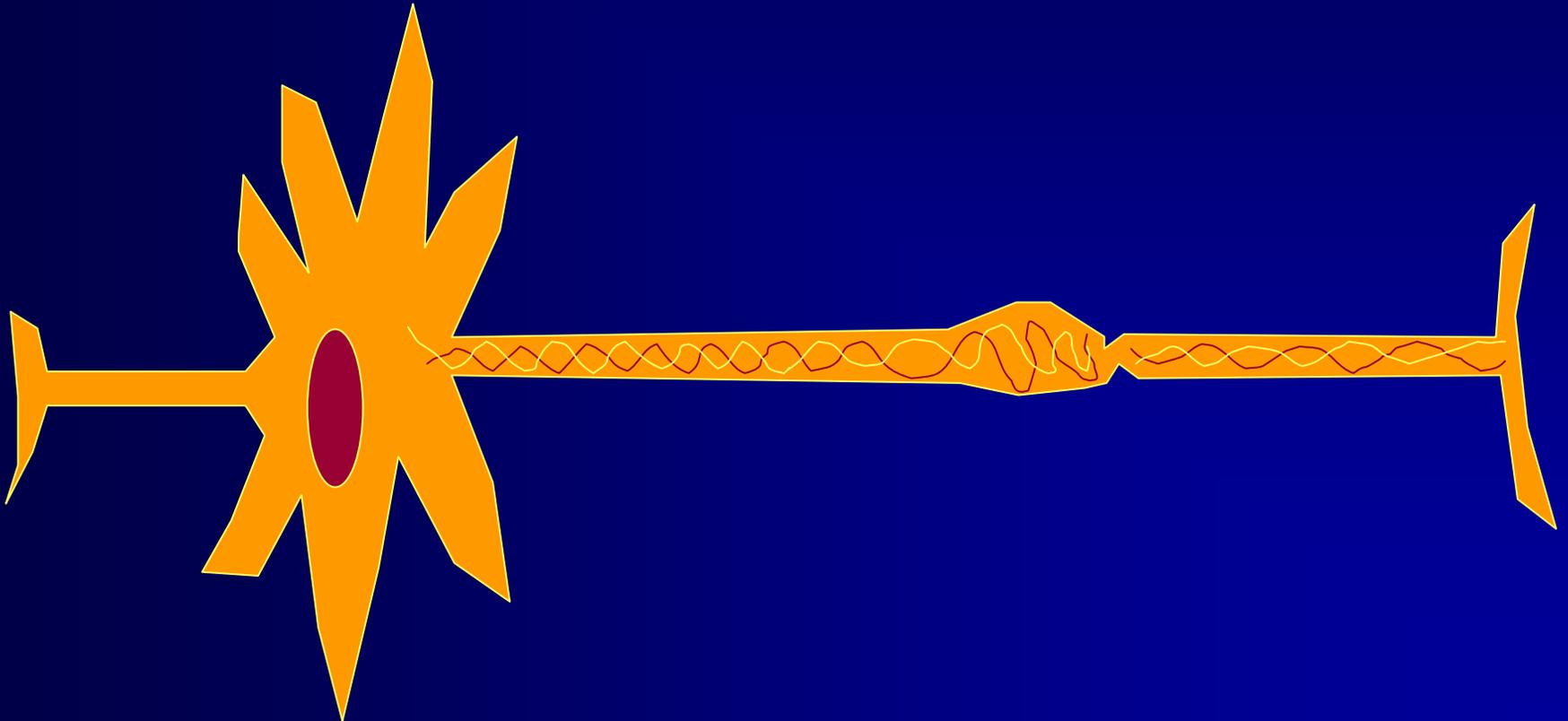


Acute Injury

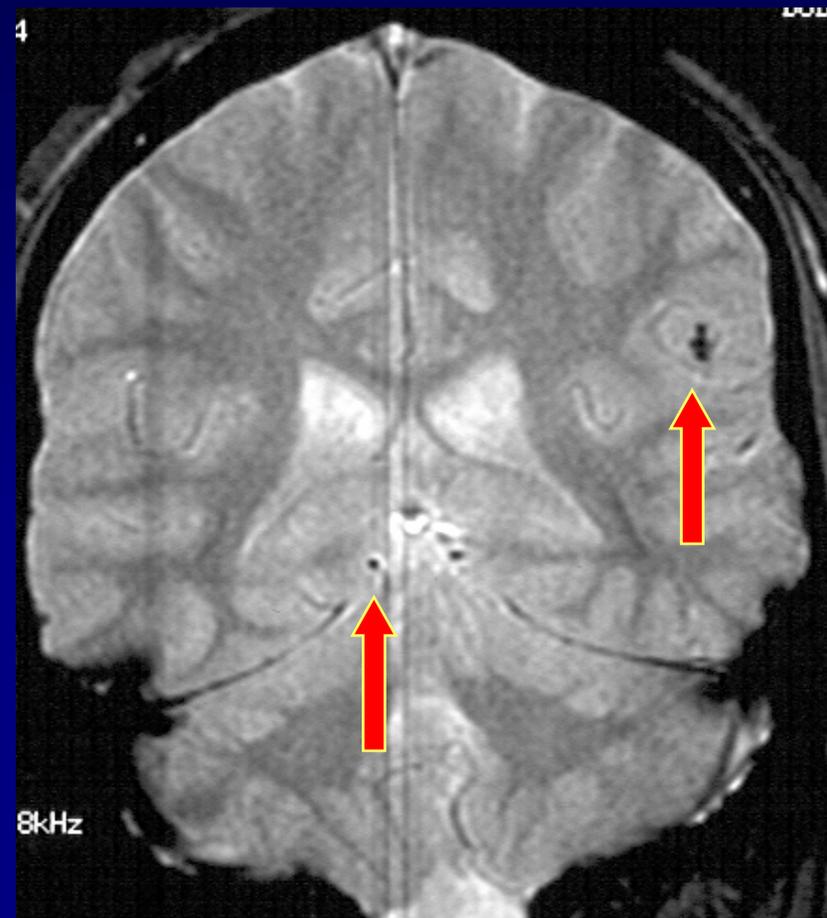
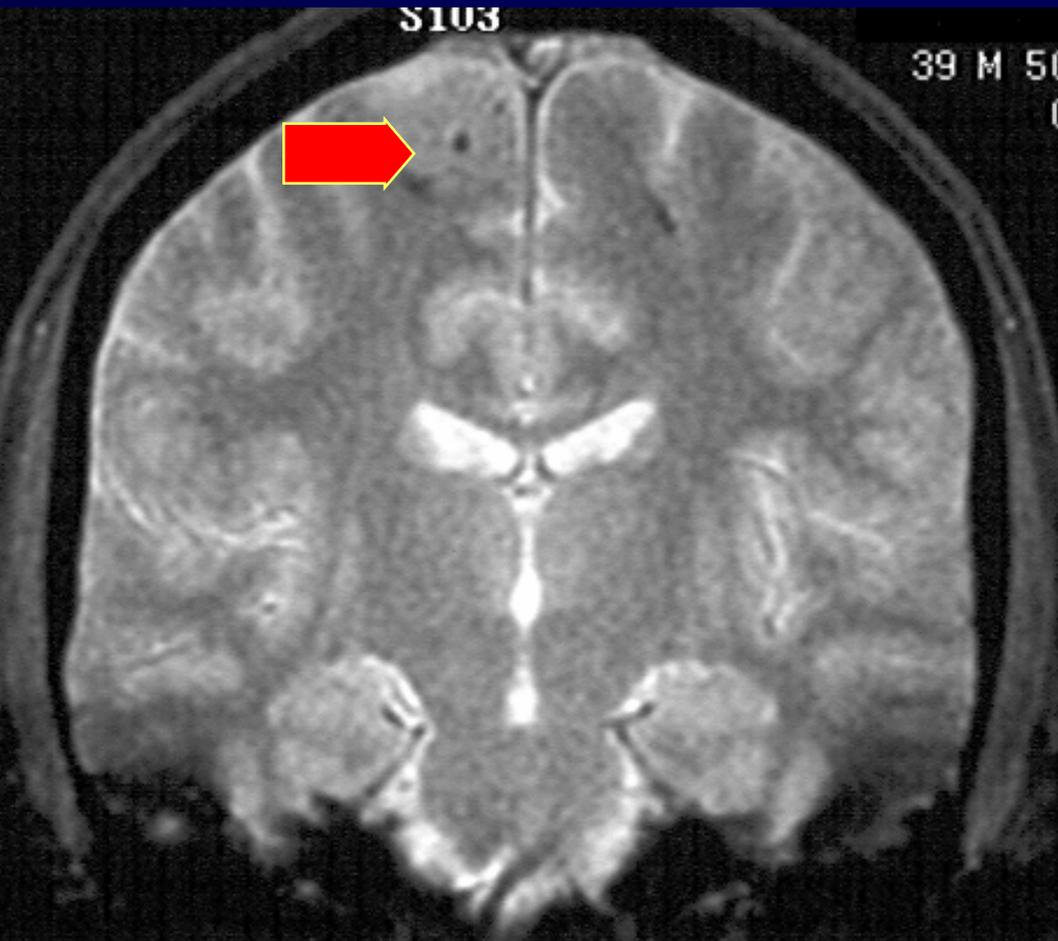


# Cytotoxic Injury

- 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)**

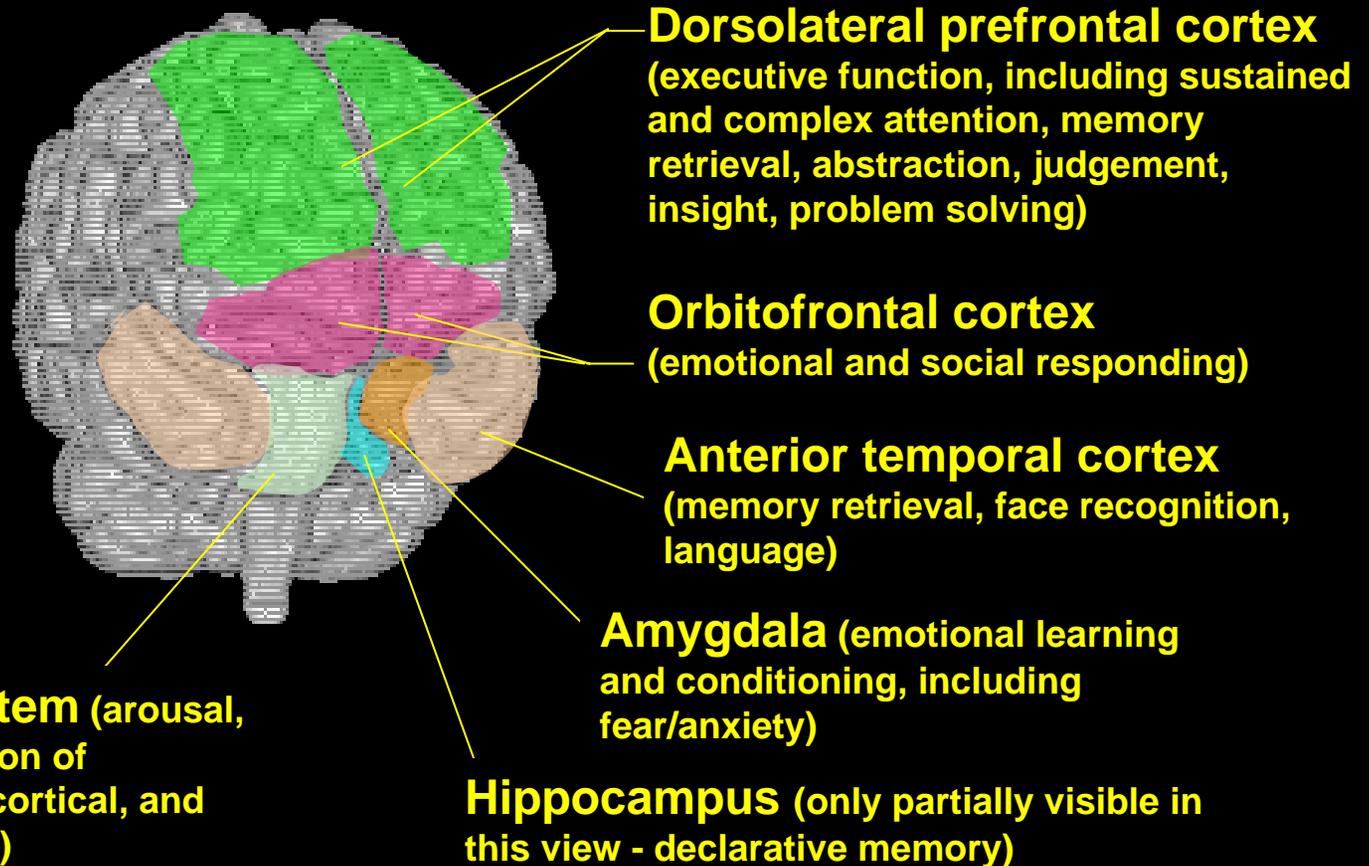
# Neuropathology of TBI

- 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



# 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.**

---

Lieberman A, Benson DF: Control of emotional expression in pseudobulbar palsy: A personal experience. Archives of Neurology 34:717-719, 1977.

# 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

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

# 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

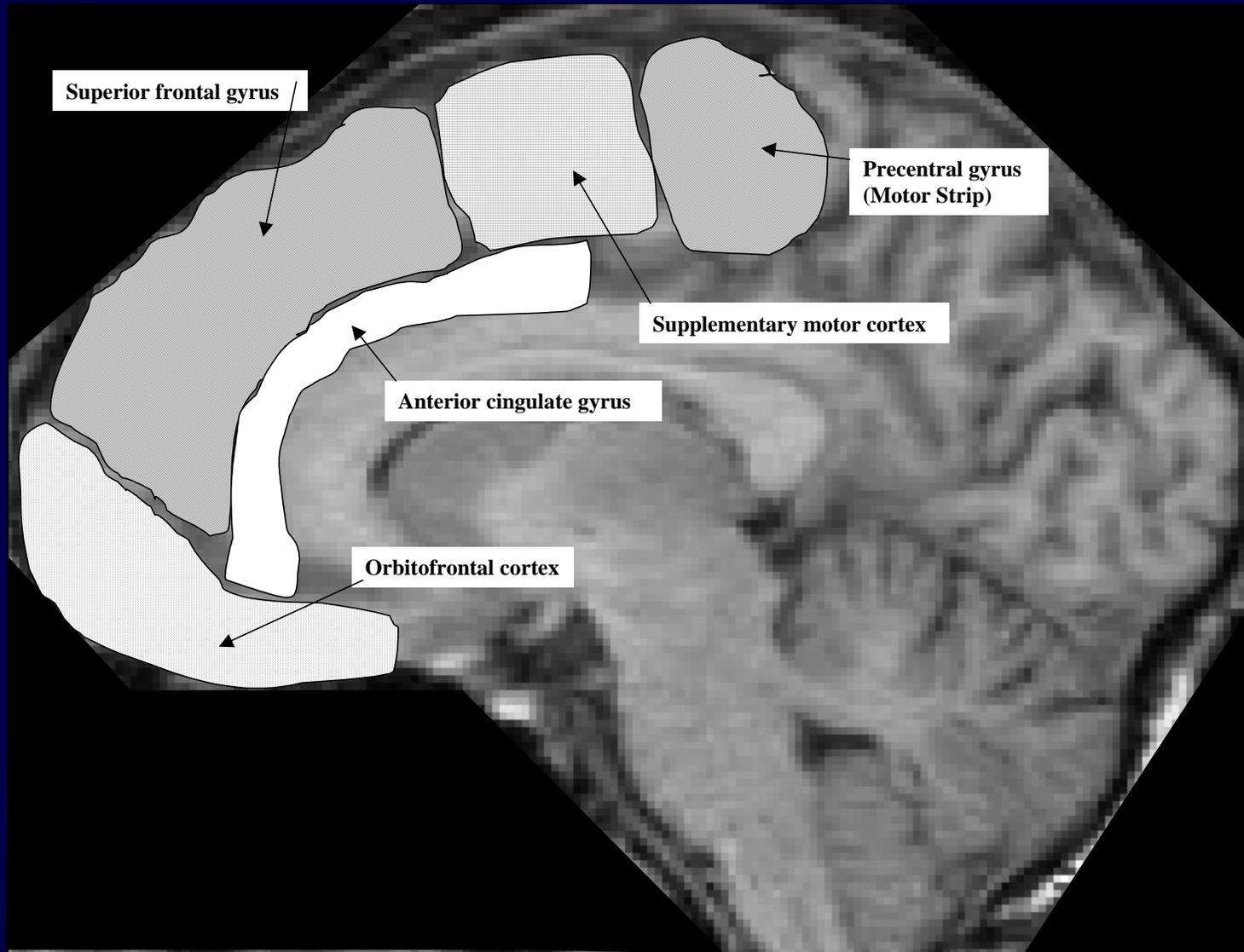
# Executive Functions

- 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



# Attention

- **The components of attention include:**
  - ***Arousal***, or the general level of neuronal responsiveness
  - ***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

2-7-L

R-6-B-4-9-M

4-6-9-B-M-R

8-C-5-X-A-3-T-1

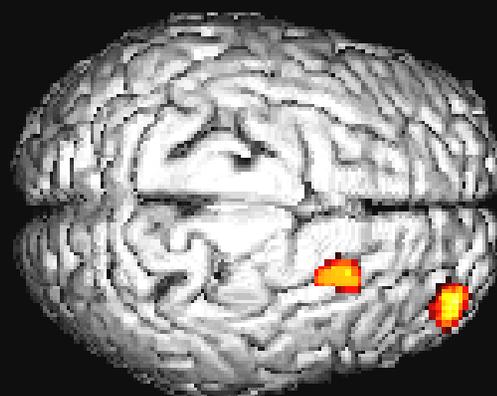
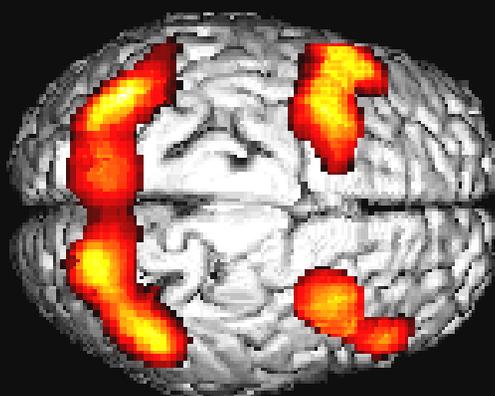
1-3-5-8-A-C-T-X

# Surface Rendered Projections on Standardized Atlas Brain

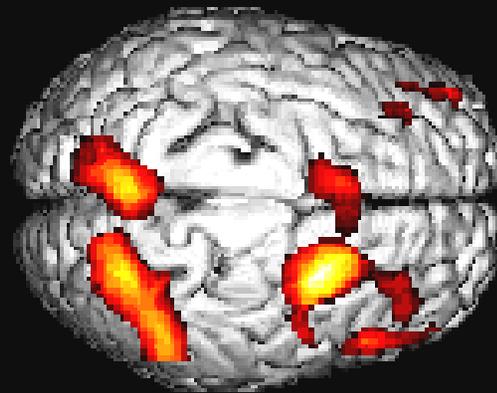
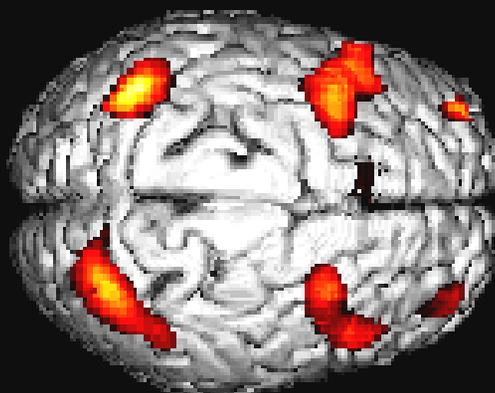
1-back > 0-back

2-back > 1-back

Controls



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