32nd Annual Virginia EMS Symposium

Traumatic Brain Injury Seminar

November 9, 2011

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Due to an emergency situation involving my position as Chairman of the Board of Directors of the Brain Injury Association of America, I am unable to deliver this seminar in person.

I sincerely apologize for this unanticipated conflict and appreciate those professionals who have graciously agreed to present this in my absence using my notes.

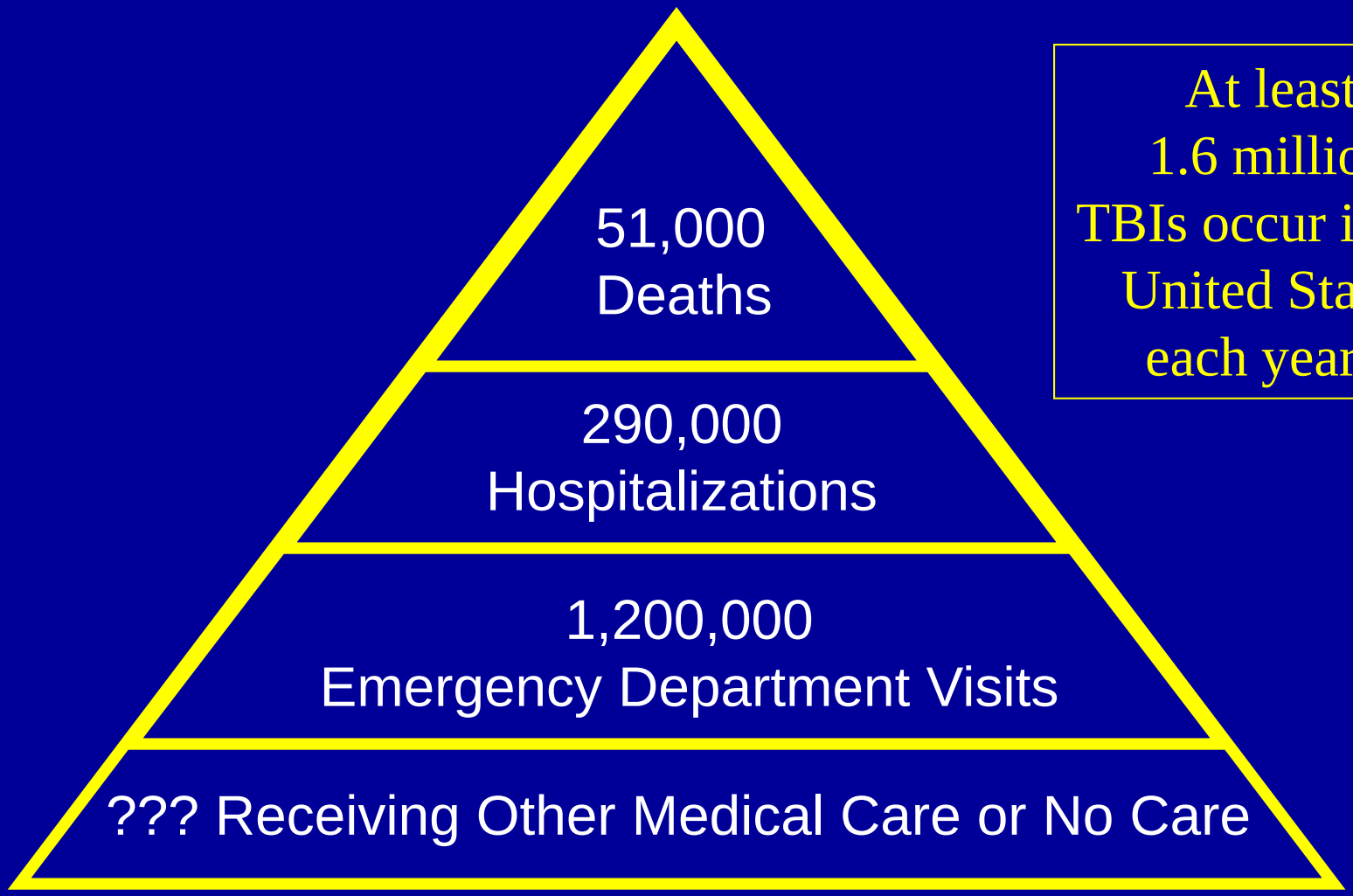
In the unlikely event that questions arise during this seminar that my esteemed colleagues are unable to address, please do not hesitate to contact me by email at

Gregory.OShanick@CenterForNeurorehabServices.org.

I will be here tomorrow (11.10.2011) in person to deliver my lecture at 1:30pm entitled, “Brain Injury Medicine for the 21st Century.”

TBI EPIDEMIOLOGY

TBI in the United States



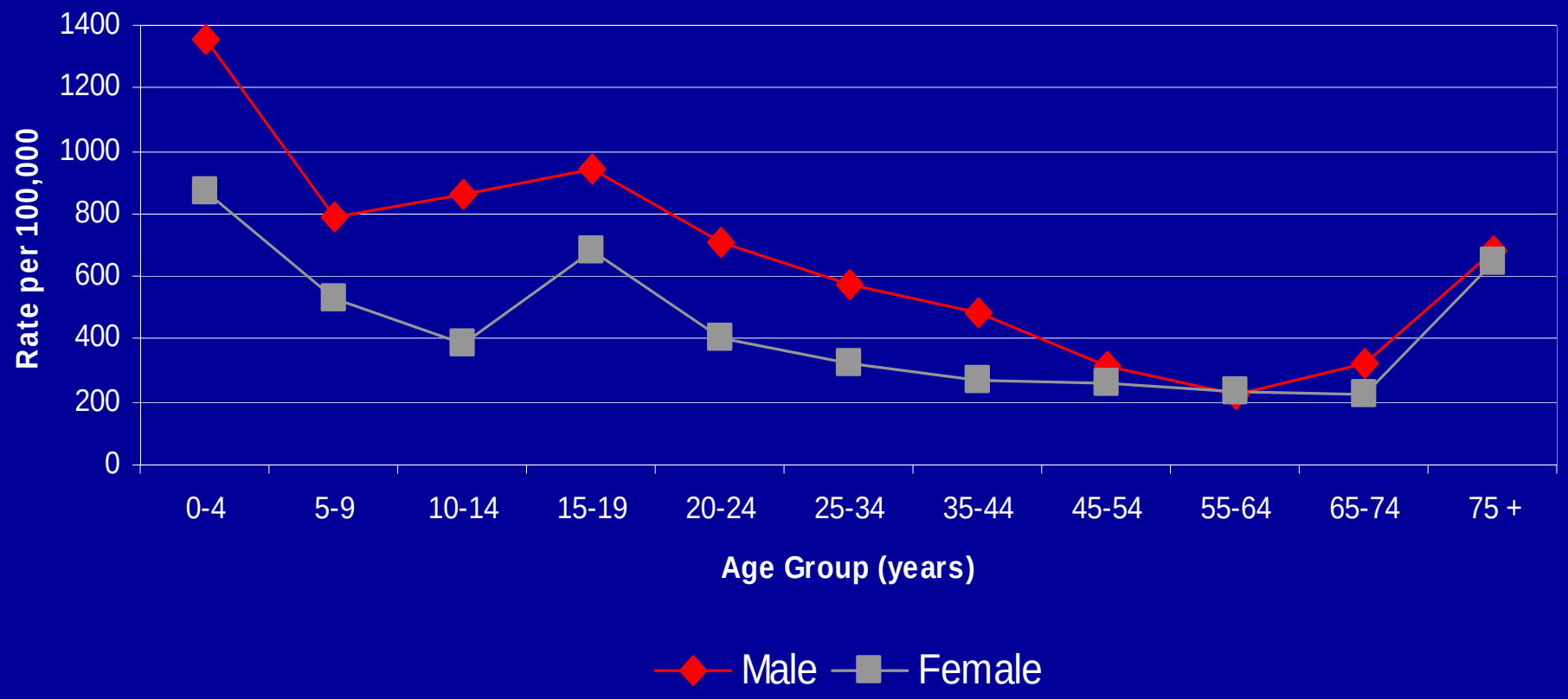
At least
1.6 million
TBIs occur in the
United States
each year.*

* CDC, 2008

From the 2004 CDC Report: TBI in the United States: ED Visits, Hospitalizations, and Deaths

TBI in the United States

TBIs by Sex*

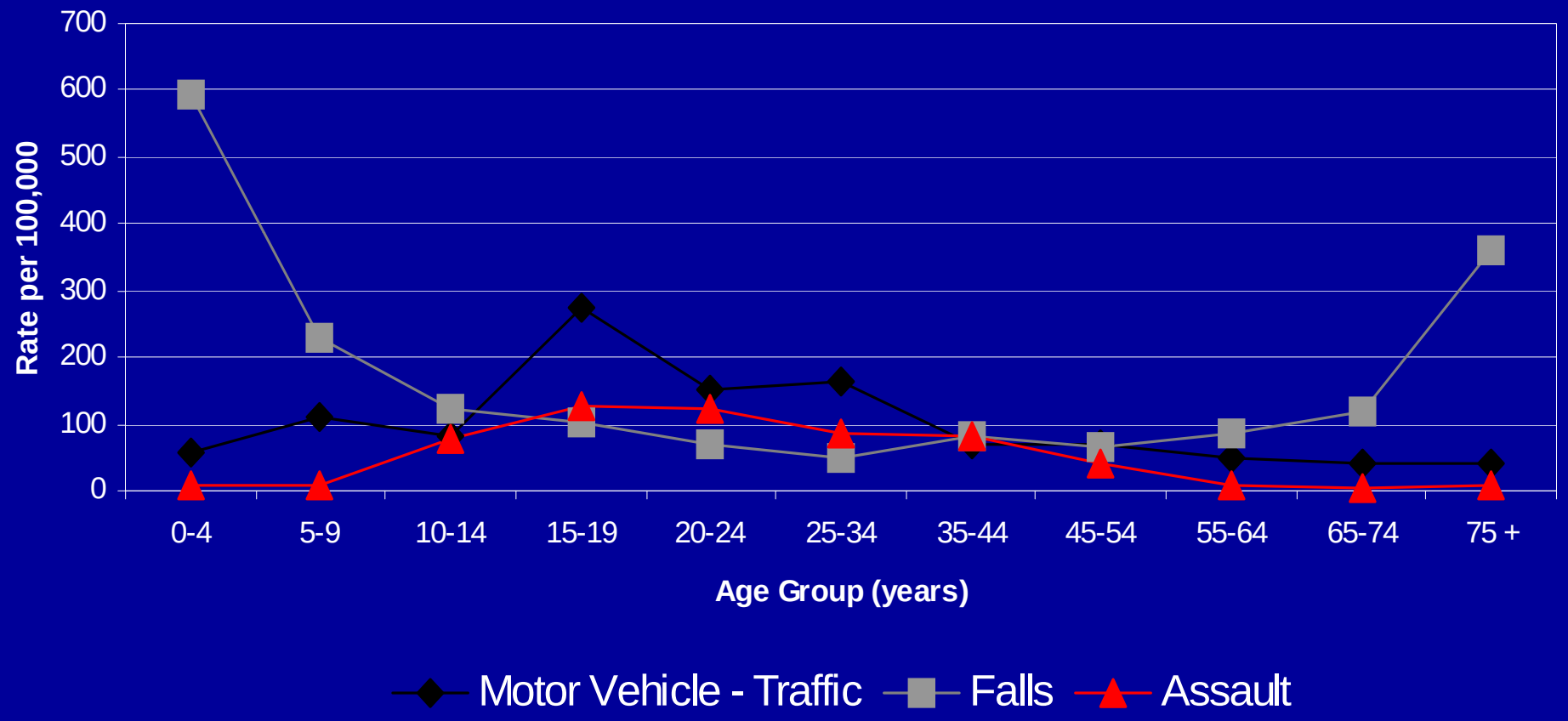


* Average annual rates, 1995-2001; ED visits, hospitalizations, and deaths combined

From the 2004 CDC Report: TBI in the United States: ED Visits, Hospitalizations, and Deaths

TBI in the United States

TBIs by Cause*

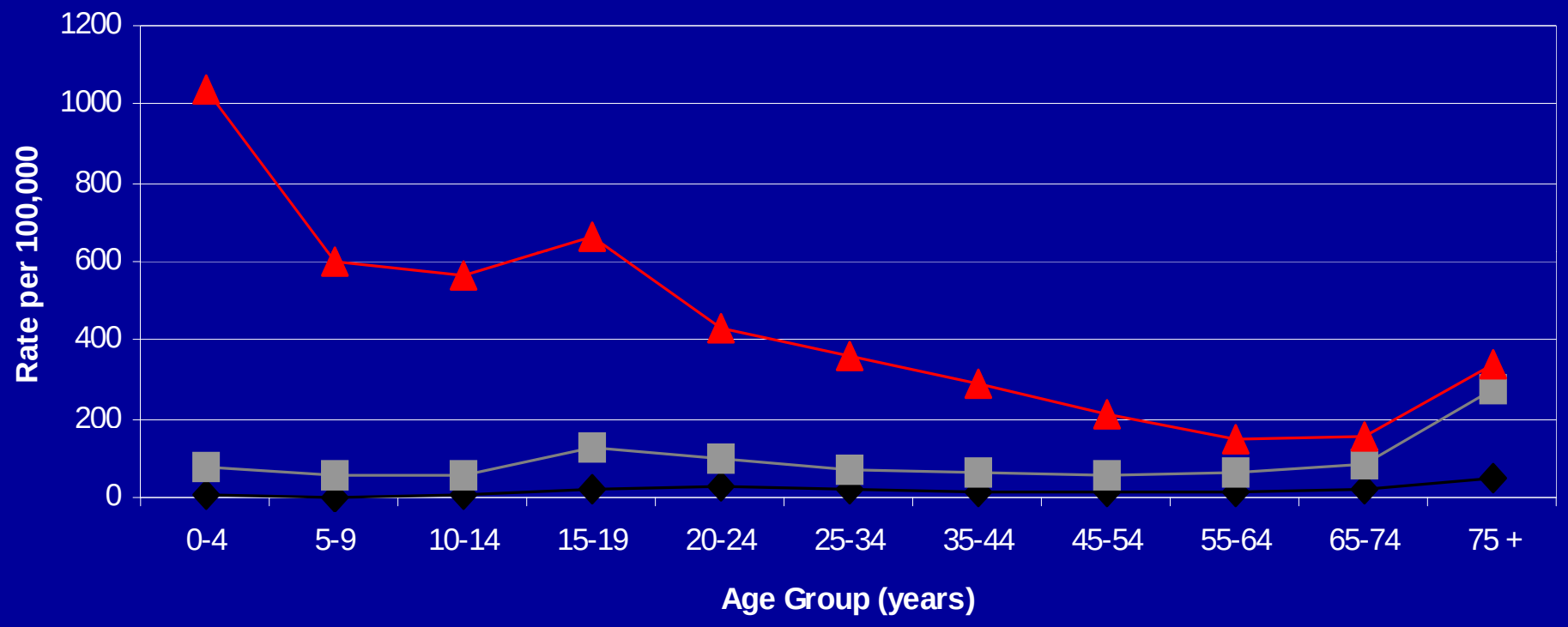


* Average annual rates, 1995-2001; ED visits, hospitalizations, and deaths combined

From the 2004 CDC Report: TBI in the United States: ED Visits, Hospitalizations, and Deaths

TBI in the United States

TBIs by Age Group*



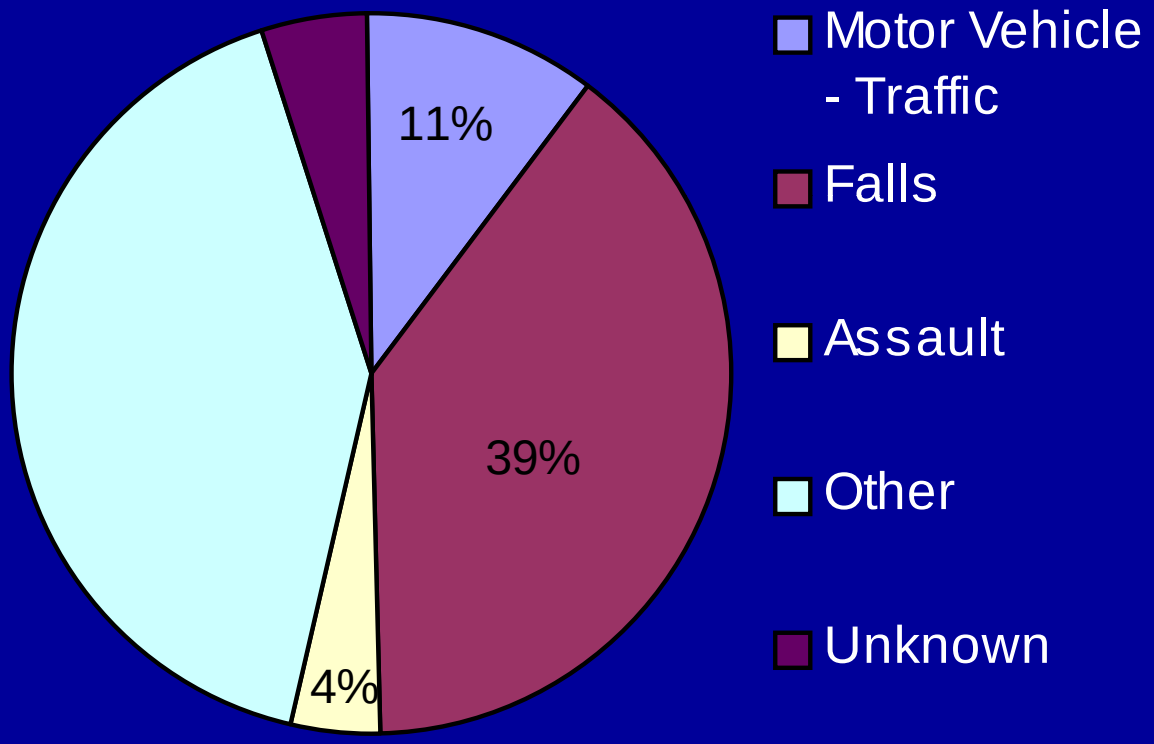
Deaths Hospitalizations ED Visits

* Average annual rates, 1995-2001

From the 2004 CDC Report: TBI in the United States: ED Visits, Hospitalizations, and Deaths

TBI in the United States

TBIs Among Children (0-14 years) by Cause

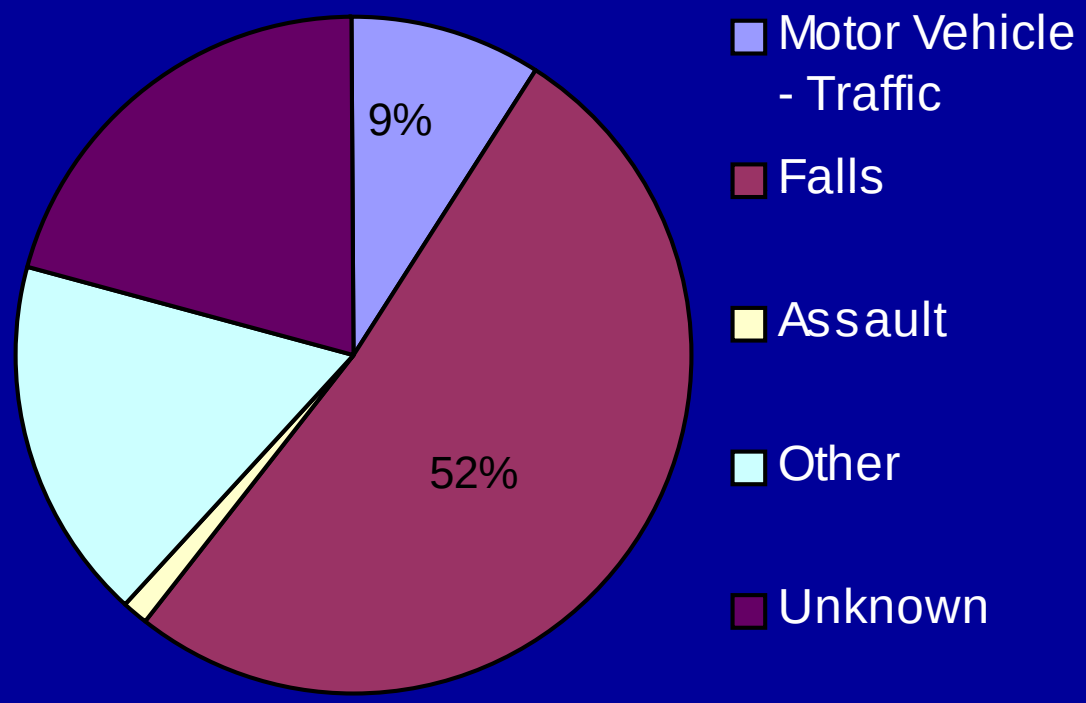


* Average annual percents, 1995-2001; ED visits, hospitalizations, and deaths combined

From the 2004 CDC Report: TBI in the United States: ED Visits, Hospitalizations, and Deaths

TBI in the United States

TBIs Among Older Adults (≥65 years) by Cause



* Average annual percents, 1995-2001; ED visits, hospitalizations, and deaths combined

From the 2004 CDC Report: TBI in the United States: ED Visits, Hospitalizations, and Deaths

MILITARY TBI EPIDEMIOLOGY

Military Incidence of TBI

7000 peacetime admissions annually

Active duty males: 225 per 100,000

Active duty females: 150 per 100,000

Combat Statistics

(DVBIC, January 2008)

32% of patients evacuated to WRAMC had TBI

5591 patients with TBI seen at DVBIC sites

90% of combat-related TBIs are closed head injuries (most are mTBI or concussion)

>50% blast related

From 01/03-01/31/08

Combat Statistics

30,906 servicemembers wounded

3486 killed in action in OIF/OEF

(As of 31 January 2008)

10-20% of Marines & Soldiers returning from Afghanistan & Iraq might have experienced brain injuries

(Army's TBI Task Force)

(5/5/2007)

Improvised explosive devices

Terrorism

ATF	VEHICLE DESCRIPTION	MAXIMUM EXPLOSIVES CAPACITY	LETHAL AIR BLAST RANGE	MINIMUM EVACUATION DISTANCE	FALLING GLASS HAZARD
	COMPACT SEDAN	500 Pounds 227 Kilos *(In Trunk)*	**100 Feet 30 Meters**	**1,500 Feet 457 Meters**	1,250 Feet 381 Meters
	FULL SIZE SEDAN	1,000 Pounds 455 Kilos *(In Trunk)*	**125 Feet 38 Meters**	**1,750 Feet 534 Meters**	1,750 Feet 534 Meters
	PASSENGER VAN OR CARGO VAN	4,000 Pounds 1,818 Kilos	**200 Feet 61 Meters**	**2,750 Feet 838 Meters**	2,750 Feet 838 Meters
	SMALL BOX VAN *(14 FT BOX)*	10,000 Pounds 4,545 Kilos	**300 Feet 91 Meters**	**3,750 Feet 1,143 Meters**	3,750 Feet 1,143 Meters
	BOX VAN OR WATER/FUEL TRUCK	30,000 Pounds 13,636 Kilos	**450 Feet 137 Meters**	**6,500 Feet 1,982 Meters**	6,500 Feet 1,982 Meters
	SEMI-TRAILER	60,000 Pounds 27,273 Kilos	**600 Feet 183 Meters**	**7,000 Feet 2,134 Meters**	7,000 Feet 2,134 Meters

Improved individual survival
(Killed to wounded ratio)

American Revolution: 1:0.7

War of 1812: 1:0.5

Civil War (Union): 1:0.7

World War I: 1:1.8

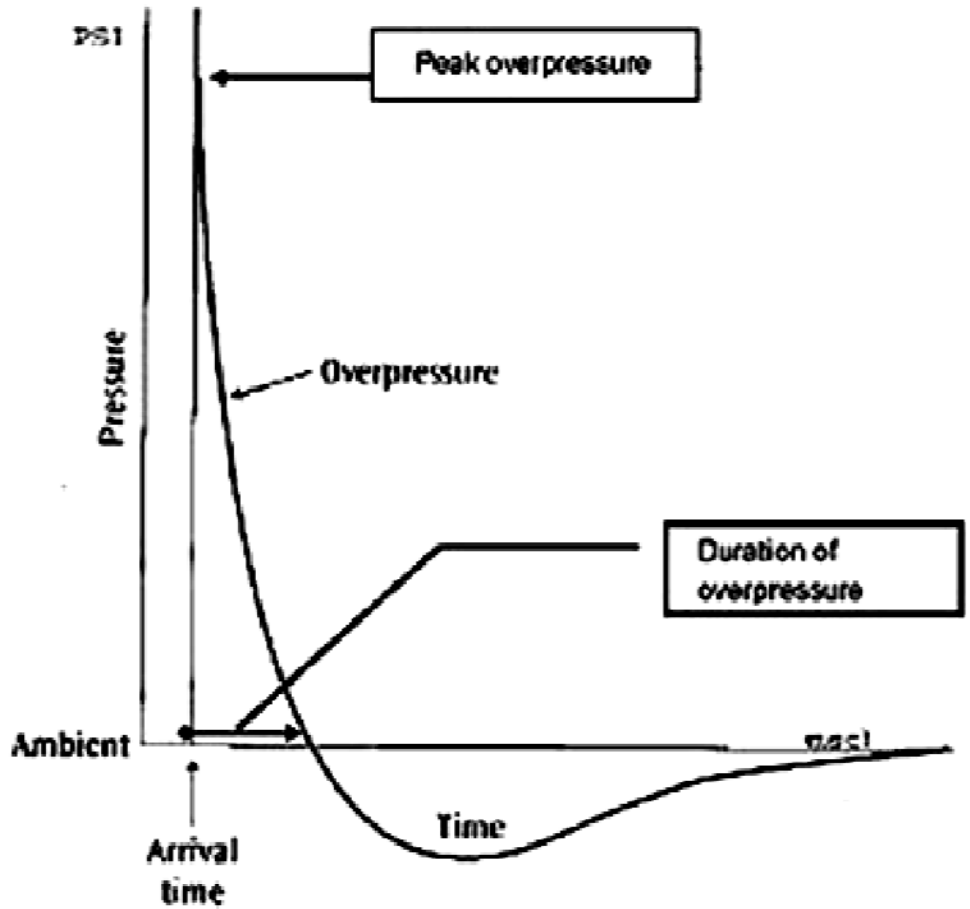
World War II: 1:1.6

Korean Conflict: 1:2.8

Viet Nam: 1:2.6

OIF/OEF: 1:16

Pressure wave dynamics



Improved individual survival

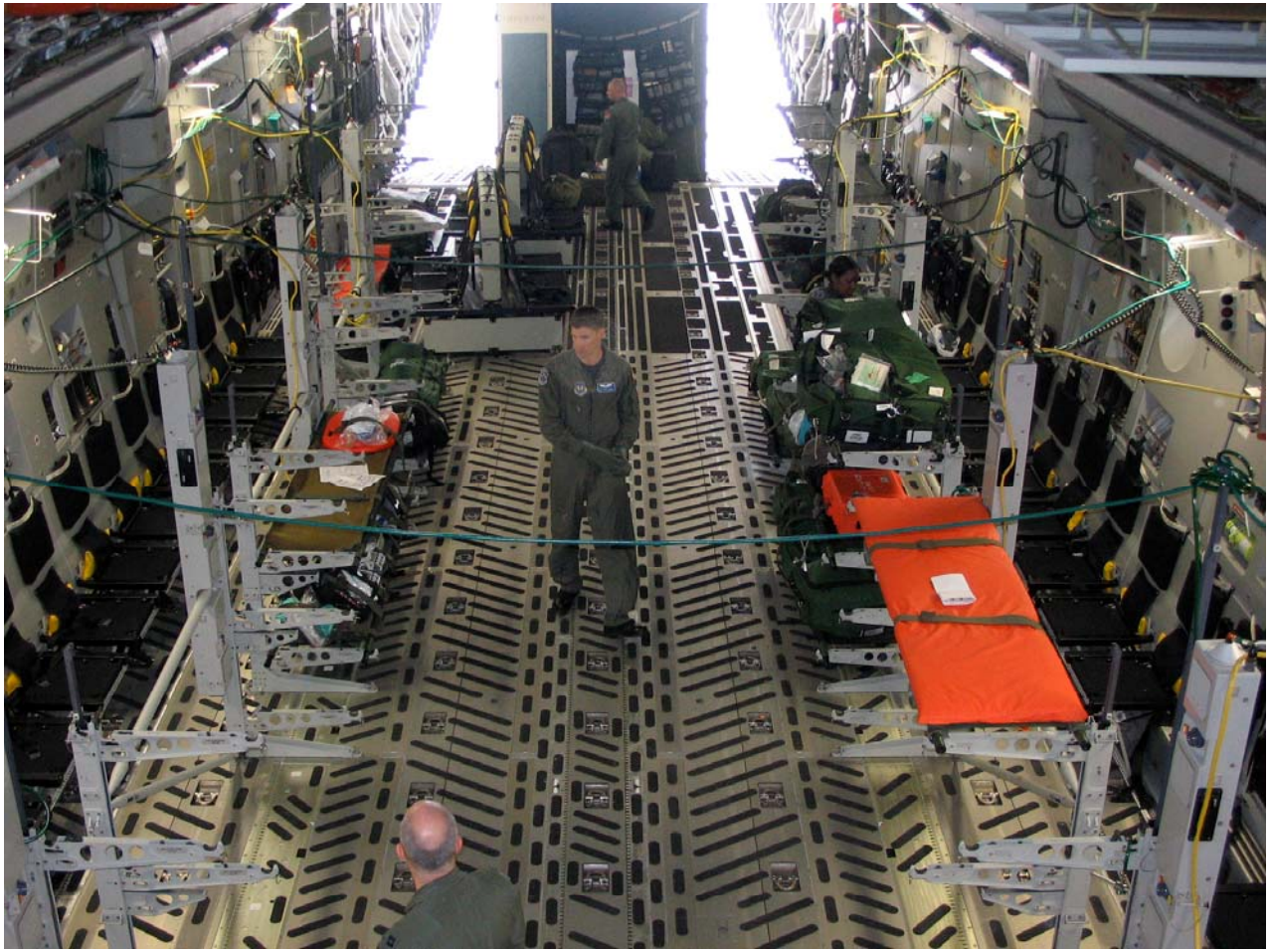
Mandarin collar worn in the up position to keep out debris and protect the neck when wearing the OTV

Infrared-feedback American Flag that provides both day and night recognition for friend or foe identification

Permanent IR feedback squares affixed to each shoulder for nighttime identification

ACU design is maximized for use with the OTV, with easy-access pockets and maximized storage space

C-17 Air Transport



C-17

C-17

NEUROANATOMY
&
NEUROPHYSIOLOGY

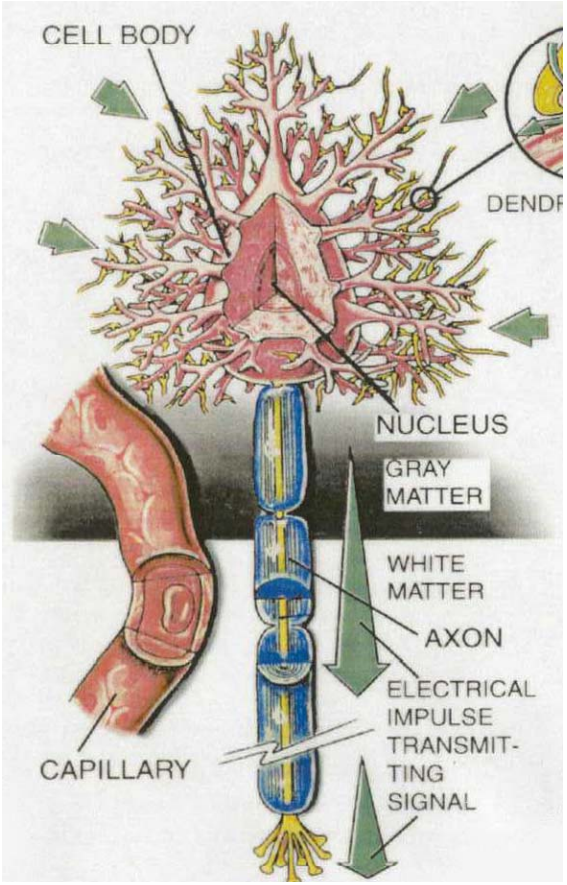
The neuron is the basic unit of brain function

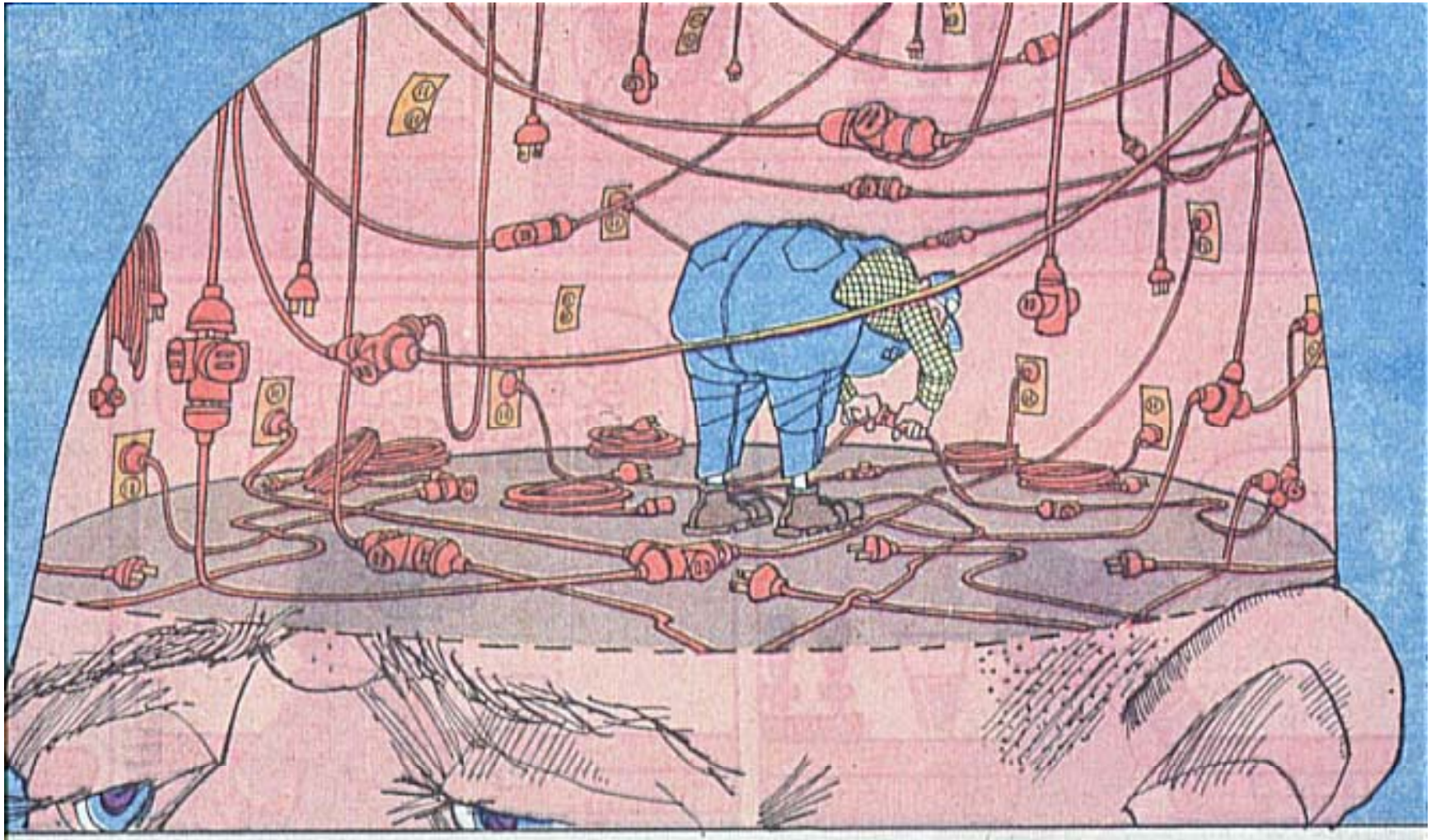
10,000 neurons can fit in a space the size of the eye of a needle (1mm)

Mature neurons have specialized functions

Neurons are supported by a "skeleton" (glial cells)

Electrical activity triggers neurotransmitter release





How the brain works.

Neurons interact through the release of neurotransmitters

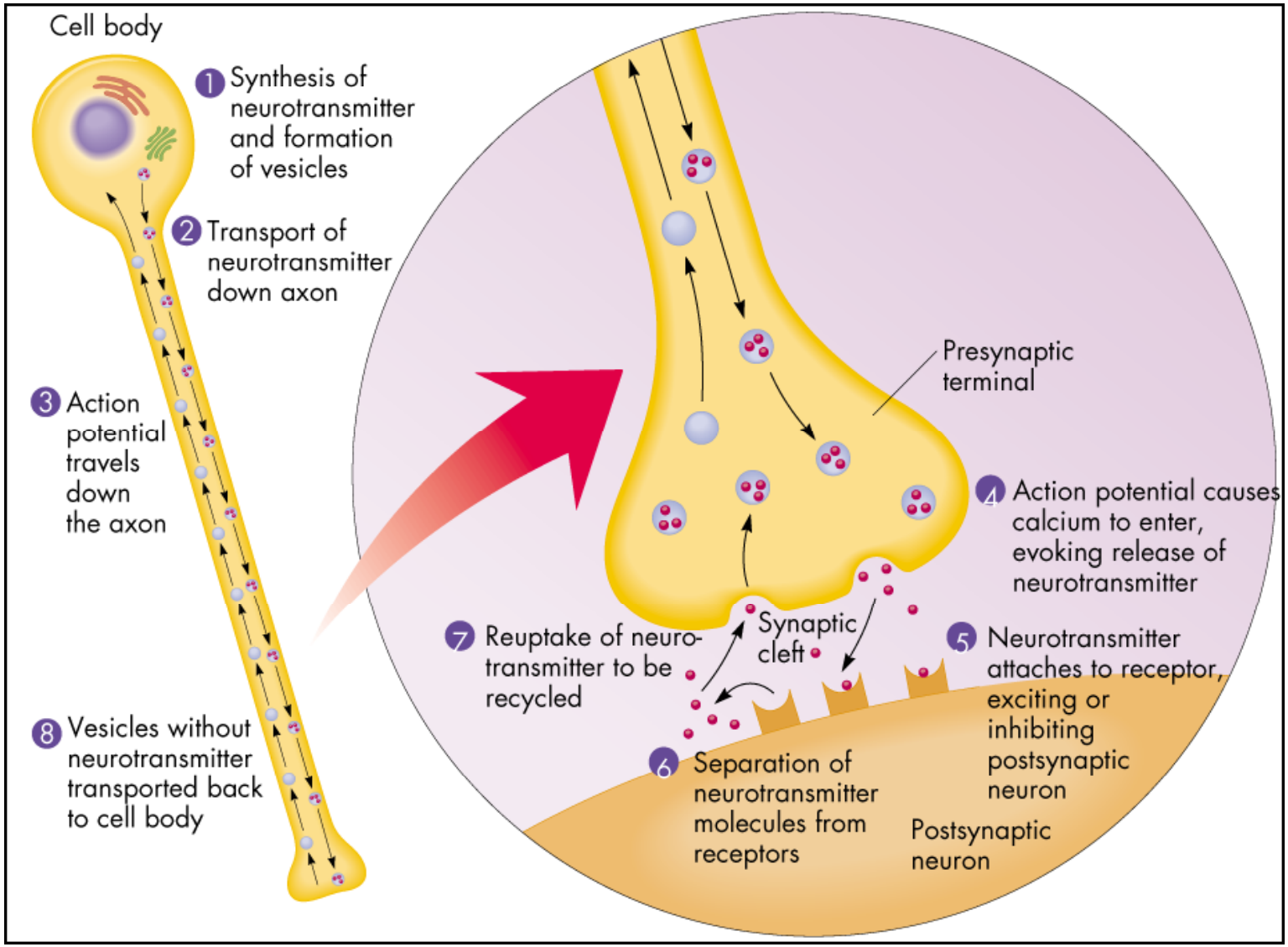
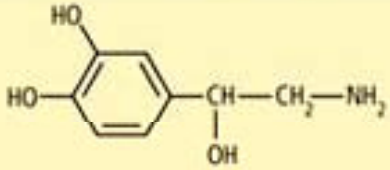
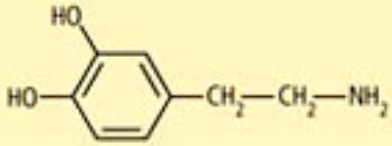
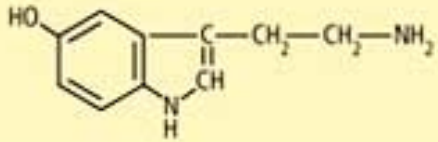
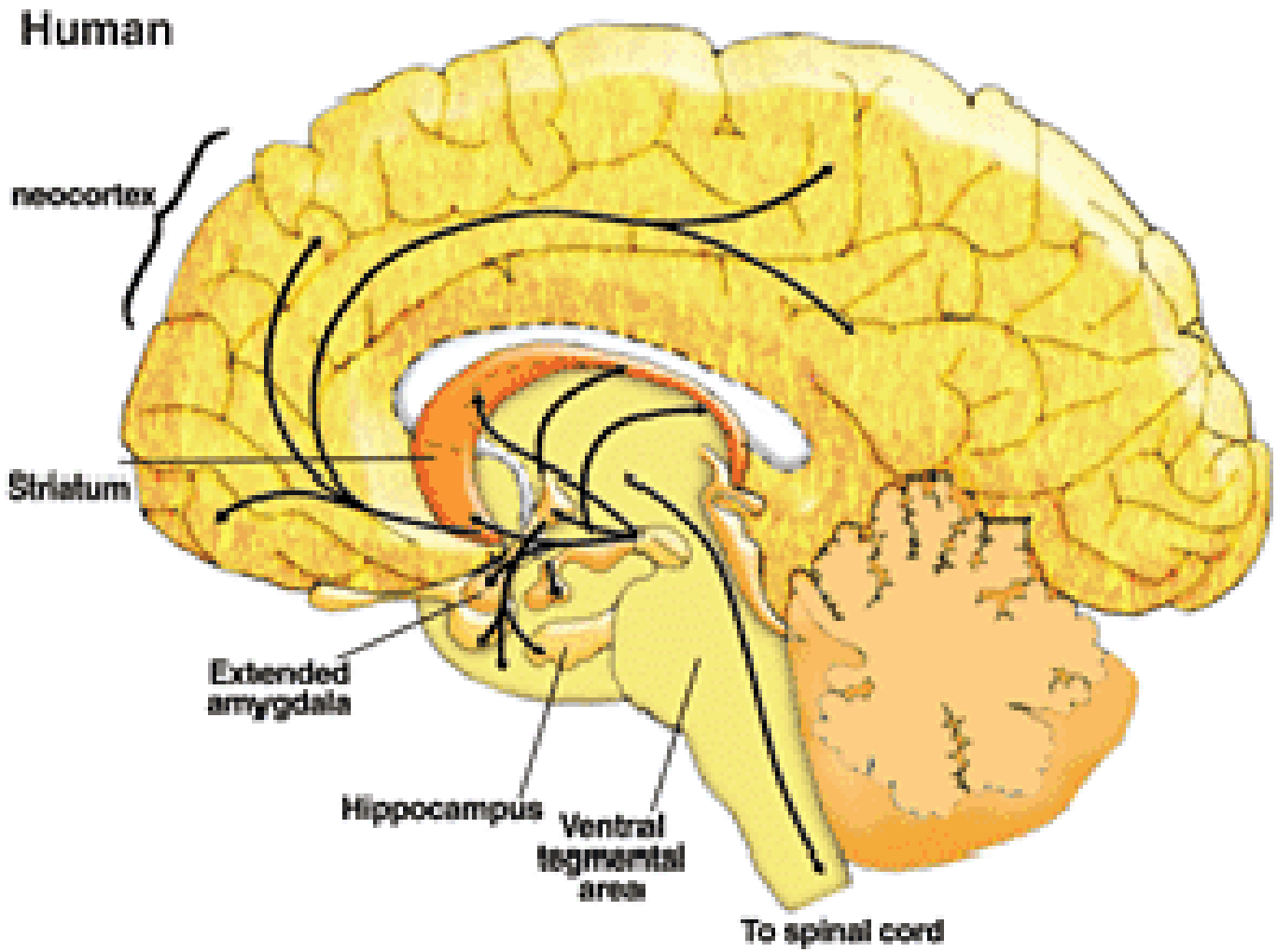
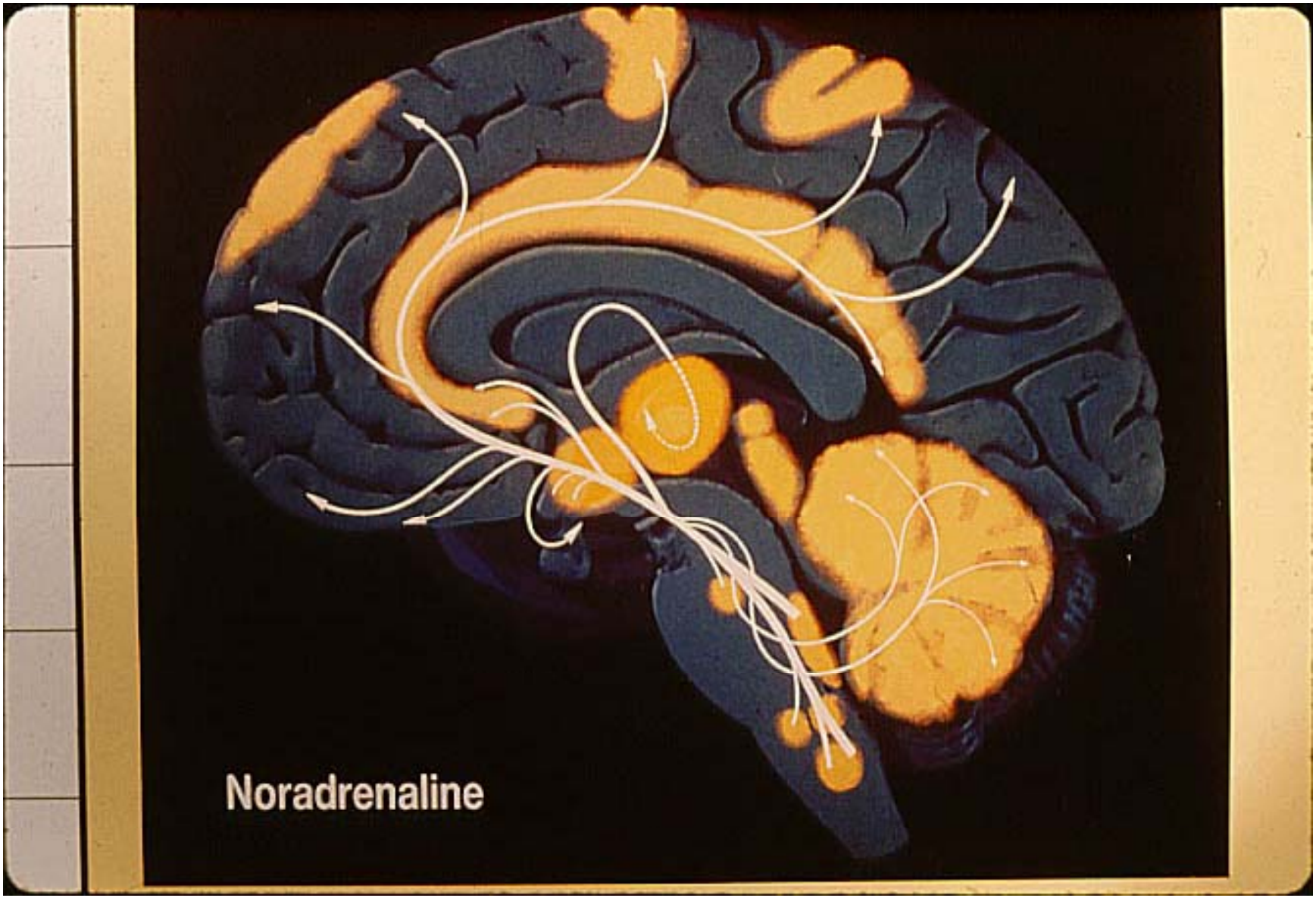


Table 48.1 Major Neurotransmitters

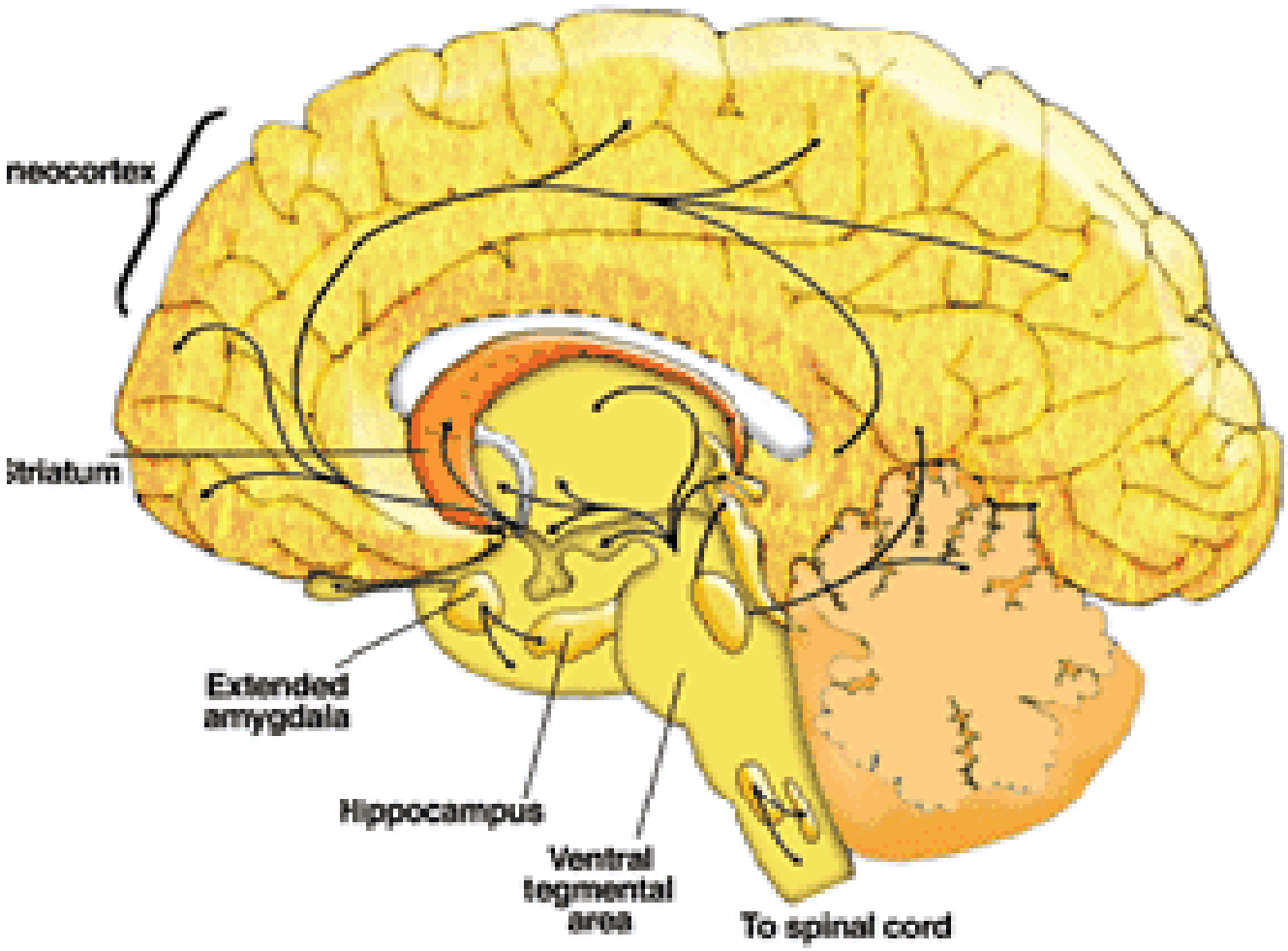
Neurotransmitter	Structure	Functional Class
Acetylcholine	$H_3C—C(=O)—O—CH_2—CH_2—N^+—[CH_3]_3$	Excitatory to vertebrate skeletal muscles; excitatory or inhibitory at other sites
Biogenic Amines		
Norepinephrine		Excitatory or inhibitory
Dopamine		Generally excitatory; may be inhibitory at some sites
Serotonin		Generally inhibitory
Amino Acids		
GABA (gamma aminobutyric acid)	$H_2N—CH_2—CH_2—CH_2—COOH$	Inhibitory
Glycine	$H_2N—CH_2—COOH$	Inhibitory
Glutamate	$H_2N—CH(COOH)—CH_2—CH_2—COOH$	Excitatory
Aspartate	$H_2N—CH(COOH)—CH_2—COOH$	Excitatory
Neuropeptides (a very diverse group, only two of which are shown)		
Substance P	Arg—Pro—Lys—Pro—Gln—Gln—Phe—Phe—Gly—Leu—Met	Excitatory
Met-enkephalin (an endorphin)	Tyr—Gly—Gly—Phe—Met	Generally inhibitory

Dopamine circuits

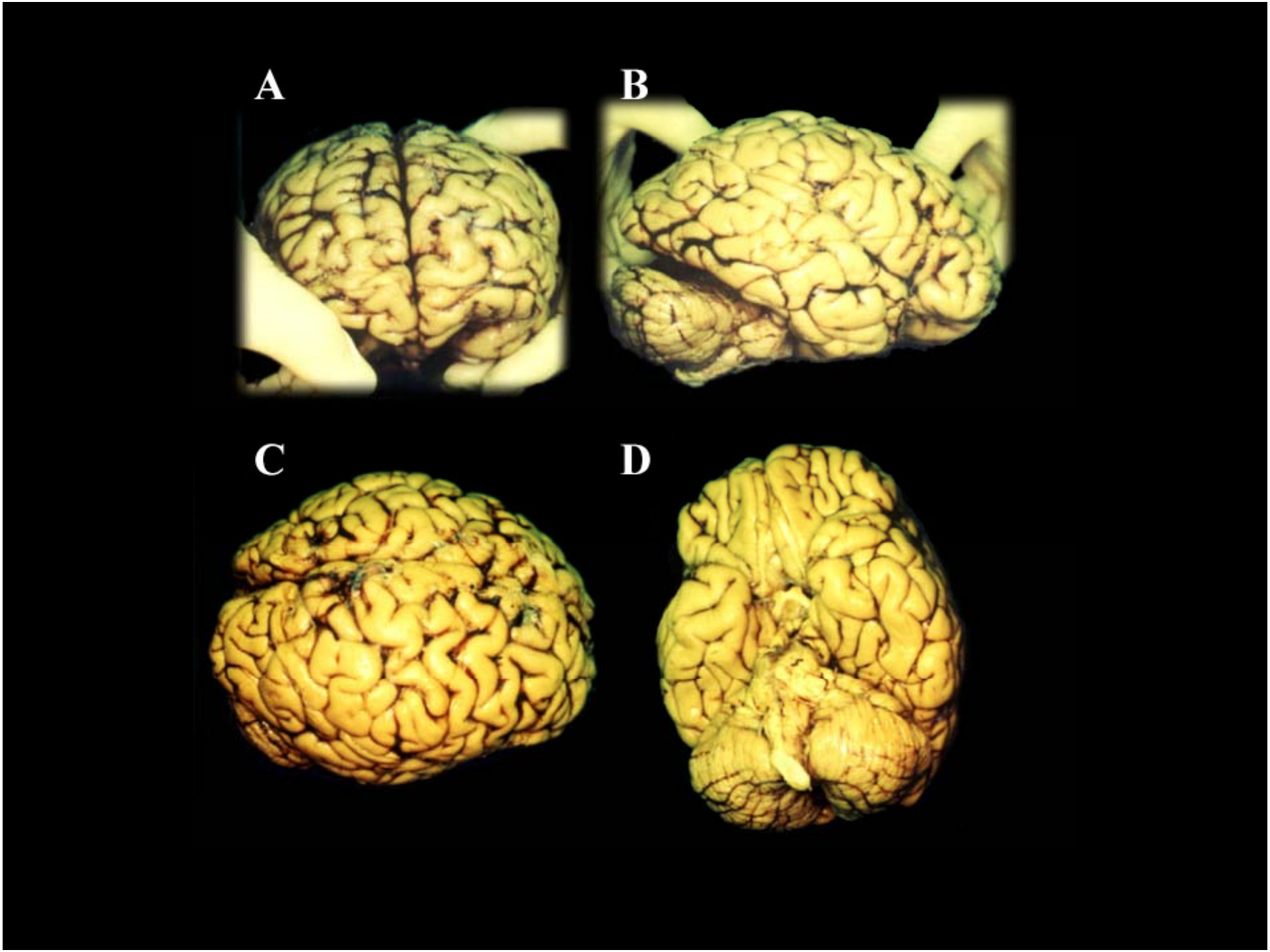



Noradrenaline

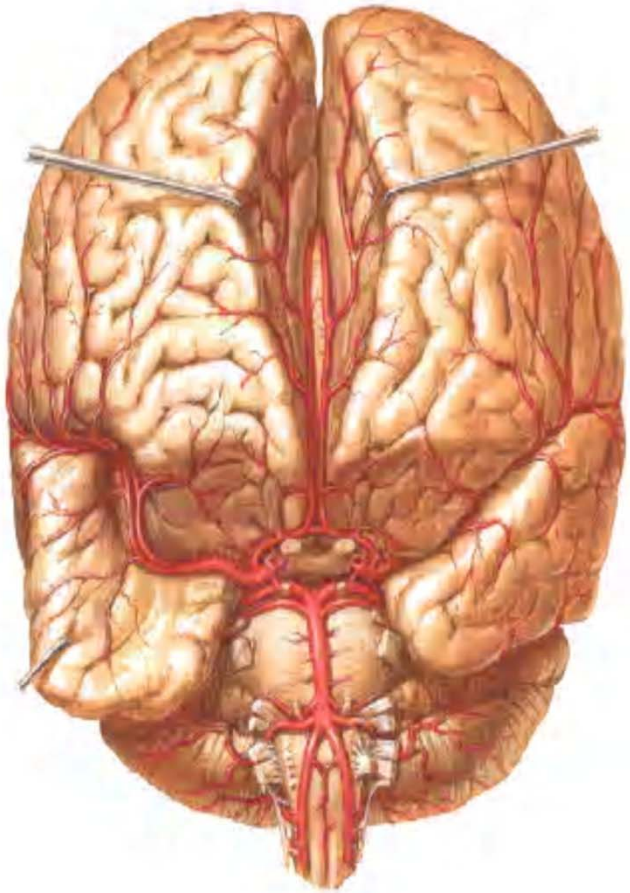
Serotonin circuits

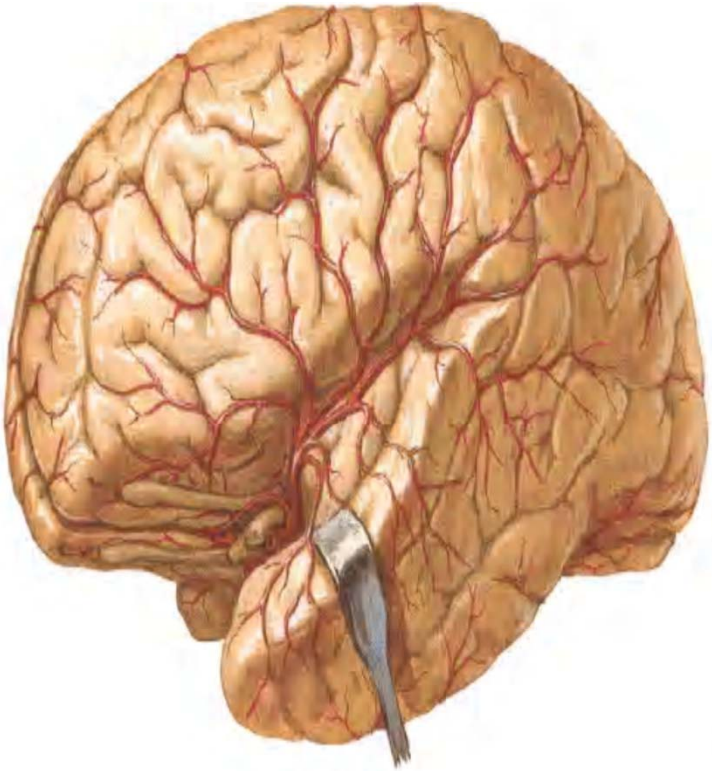


GROSS NEUROANATOMY

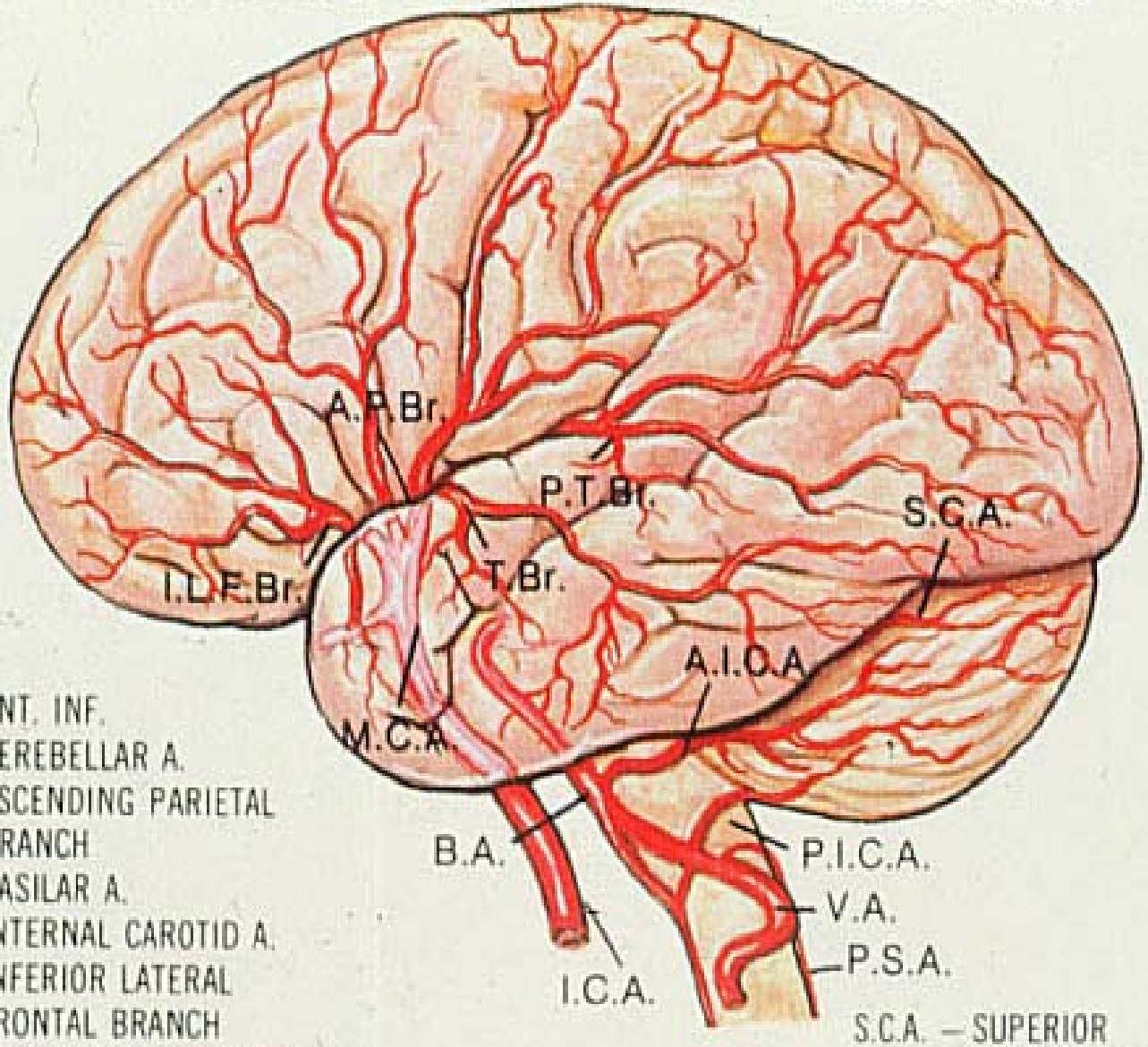

A
B
C
D

Vascular supply to the brain



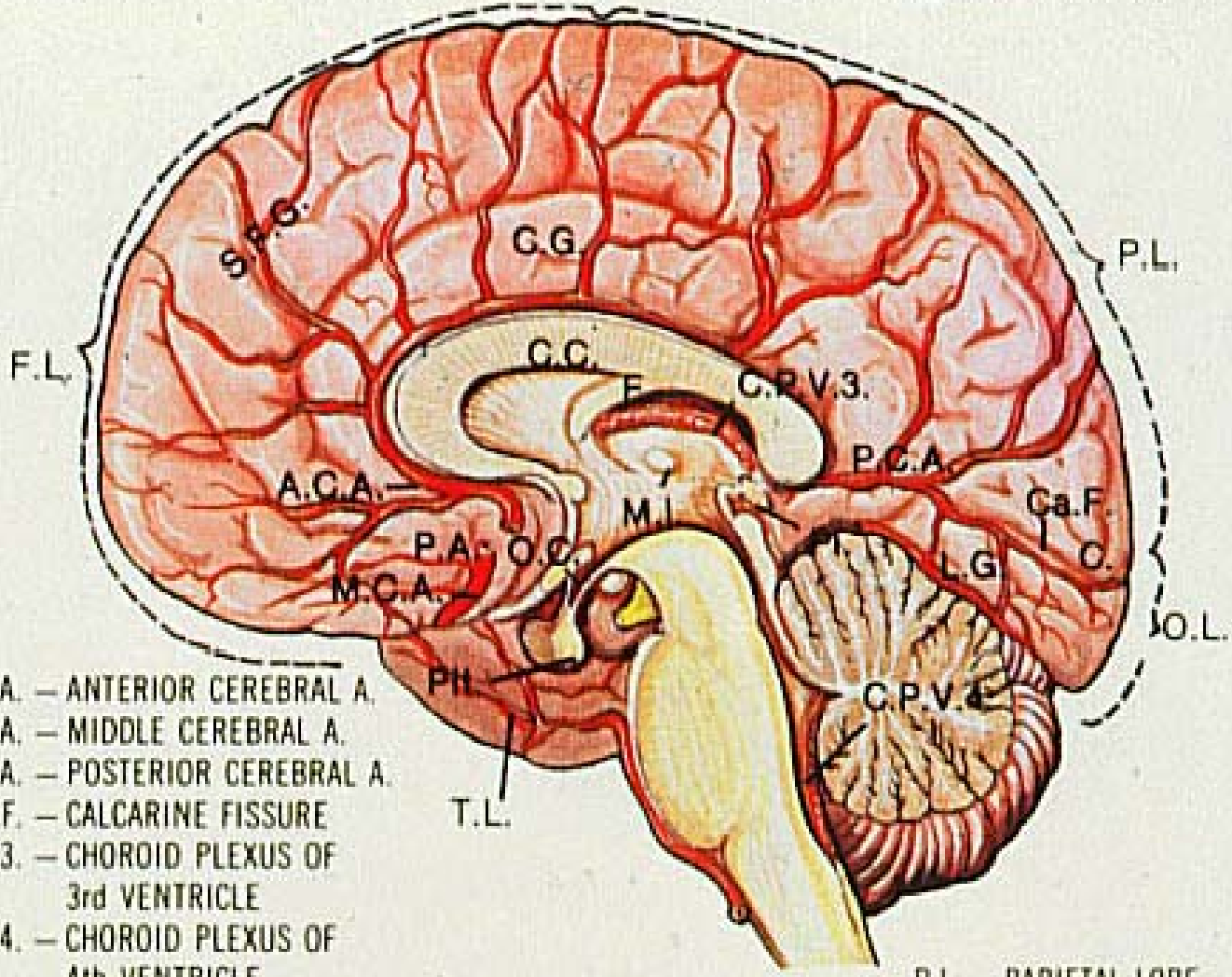


ARTERIES OF THE BRAIN — lateral view



A.I.C.A. – ANT. INF. CEREBELLAR A.
A.P.Br. – ASCENDING PARIETAL BRANCH
B.A. – BASILAR A.
I.C.A. – INTERNAL CAROTID A.
I.L.F.Br. – INFERIOR LATERAL FRONTAL BRANCH
M.C.A. – MIDDLE CEREBRAL ARTERY
P.I.C.A. – POST. INF. CEREBELLAR A.
P.S.A. – POSTERIOR SPINAL A.
P.T.Br. – PARIETO-TEMPORAL BRANCH
S.C.A. – SUPERIOR CEREBELLAR A.
T.Br. – TEMPORAL BRANCH
V.A. – VERTEBRAL A.

ARTERIES OF THE BRAIN — median sagittal section

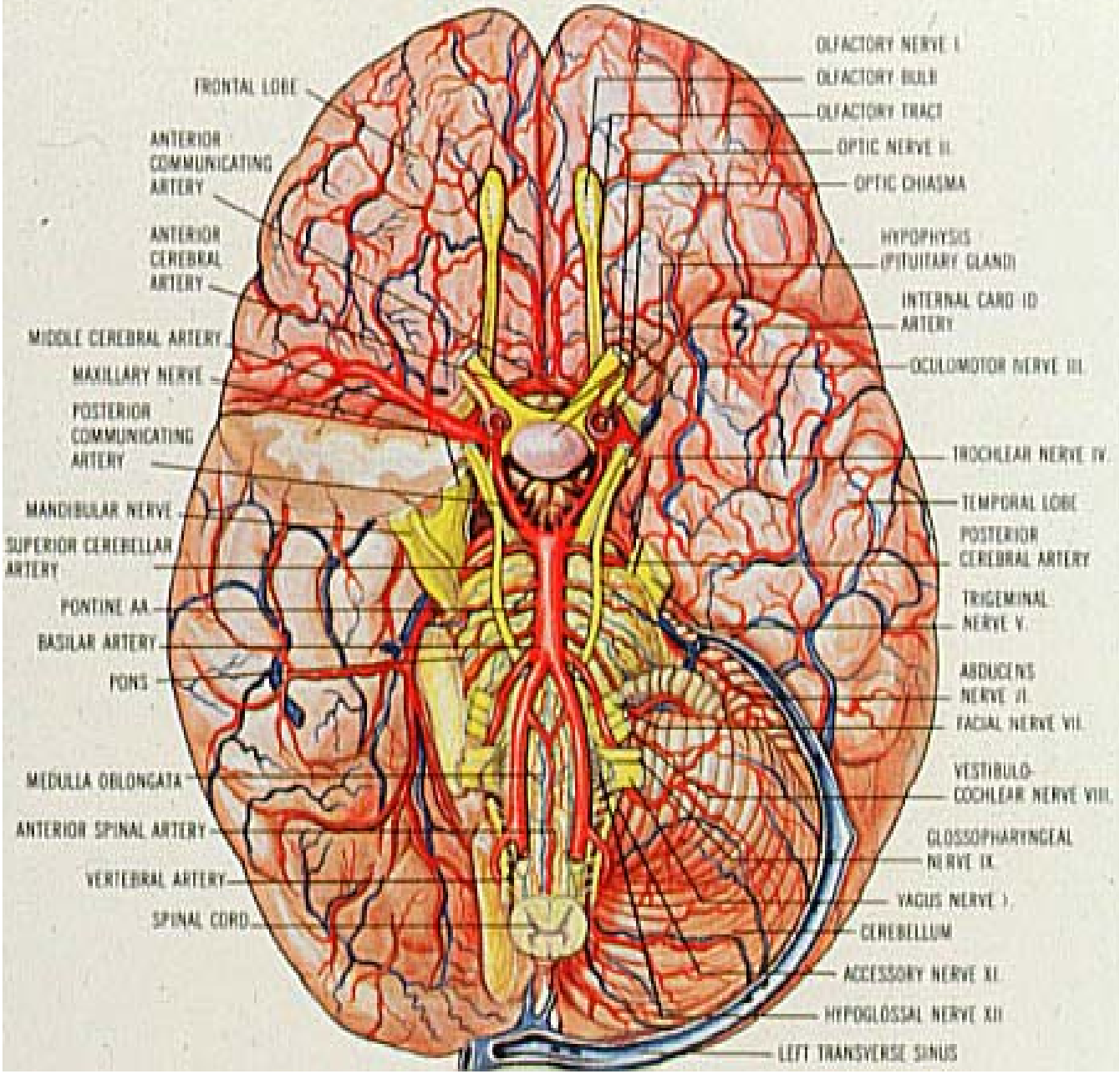


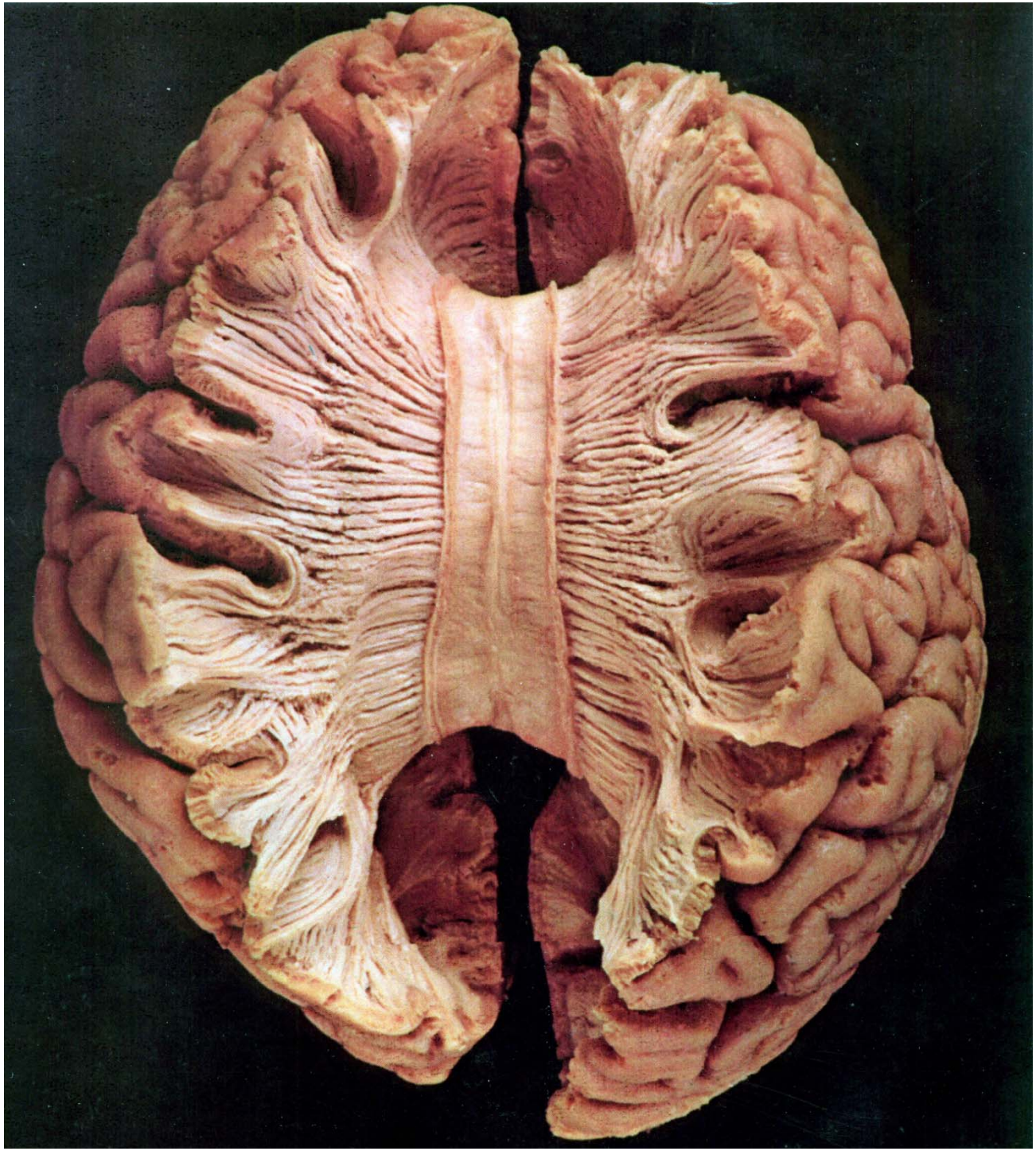
A.C.A. — ANTERIOR CEREBRAL A.
M.C.A. — MIDDLE CEREBRAL A.
P.C.A. — POSTERIOR CEREBRAL A.
Ca.F. — CALCARINE FISSURE
C.P.V.3. — CHOROID PLEXUS OF 3rd VENTRICLE
C.P.V.4. — CHOROID PLEXUS OF 4th VENTRICLE
C.C. — CORPUS CALLOSUM
C.G. — CINGULATE GYRUS
C. — CUNEUS
F. — FORNIX
F.L. — FRONTAL LOBE

L.G. — LINGUAL GYRUS
M.I. — MASSA INTERMEDIA
O.L. — OCCIPITAL LOBE
O.C. — OPTIC CHIASMA
P.A. — PAROLFACTORY AREA

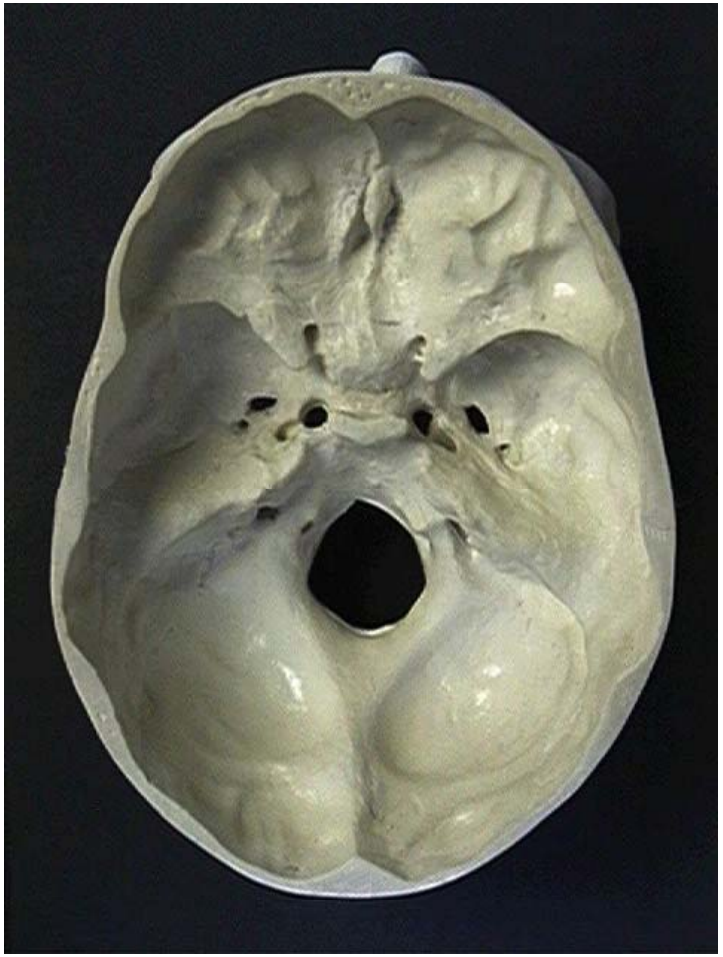
P.L. — PARIETAL LOBE
Pi. — PINEAL BODY
Pit. — PITUITARY GLAND
P.C. — PRECUNEUS
S.F.G. — SUPERIOR FRONTAL GYRUS
T.L. — TEMPORAL LOBE

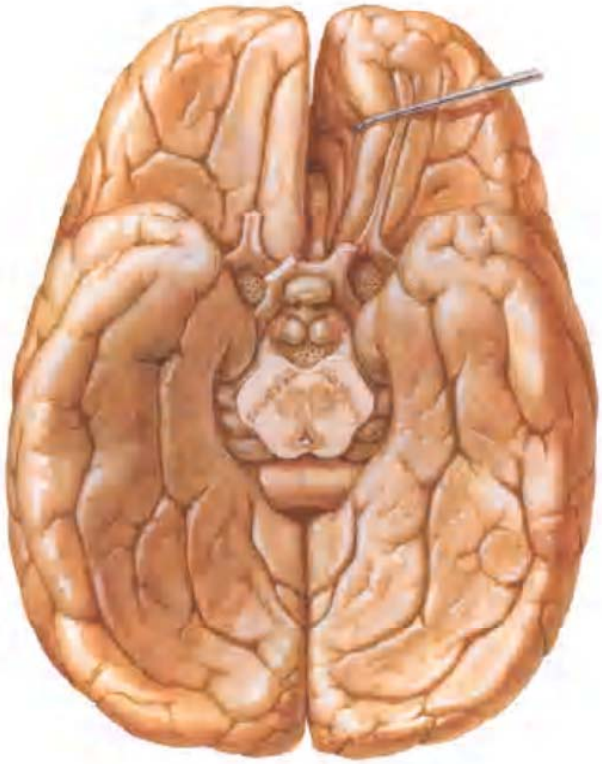
INFERIOR SURFACE OF THE BRAIN WITH BLOOD VESSELS



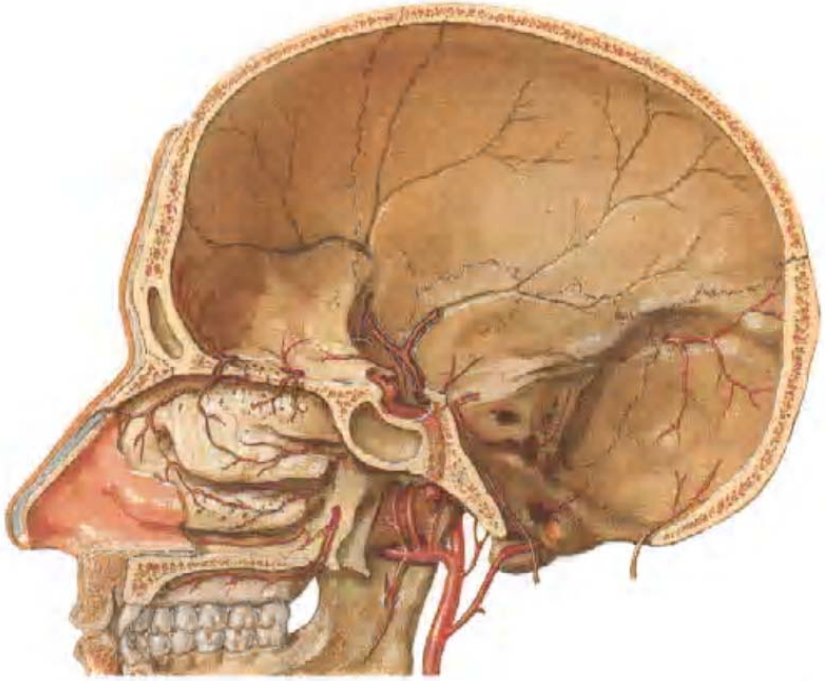


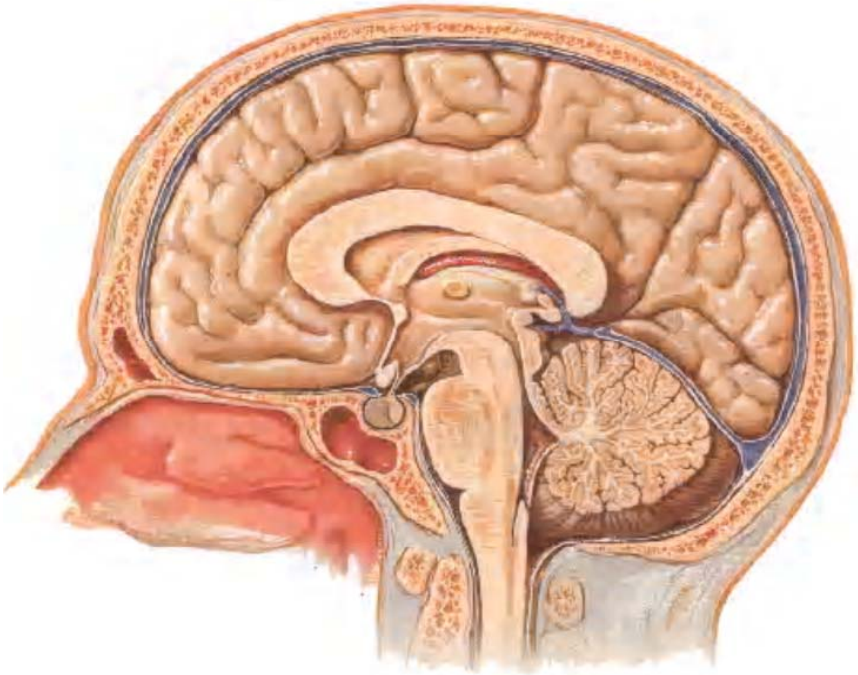
Skull base & brain proximity





Brain – skull proximity

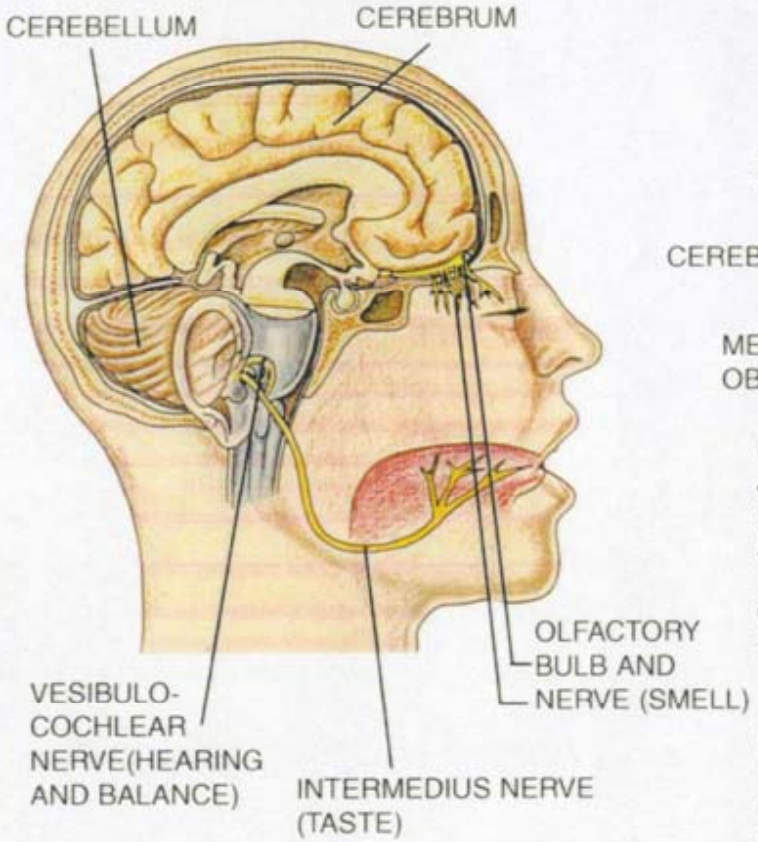




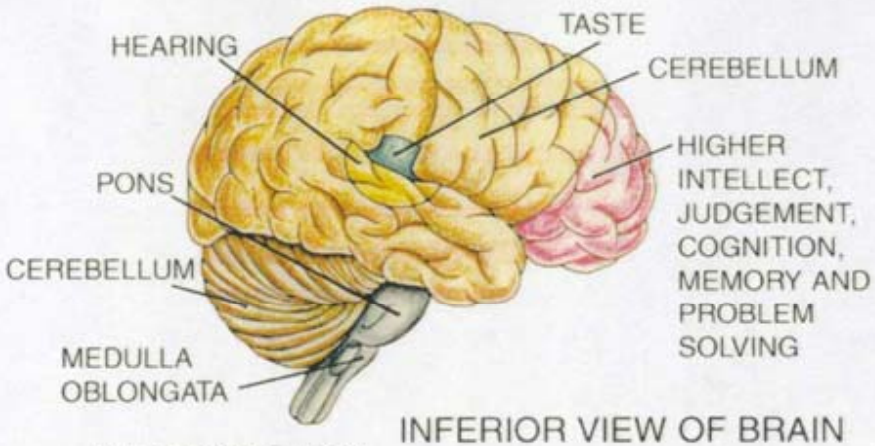
BRAIN FUNCTION BY REGION

LOCATION OF BRAIN AND CRANIAL NERVE FUNCTIONS

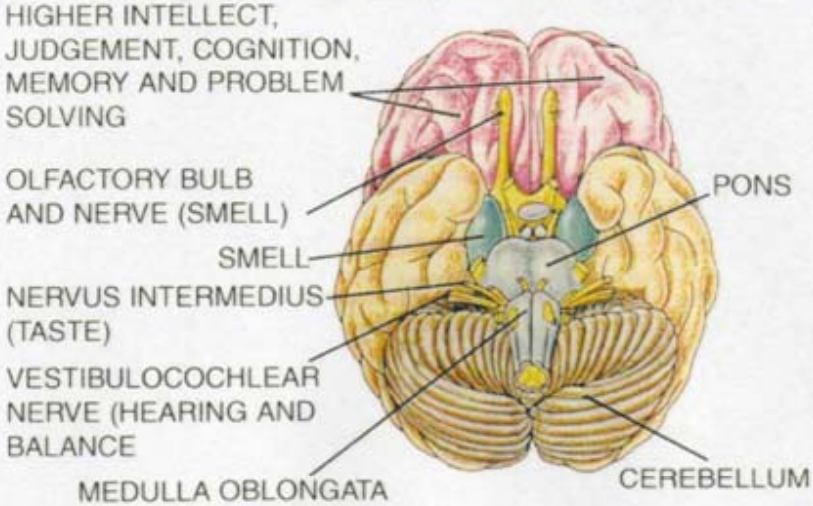
SAGITTAL VIEW OF BRAIN



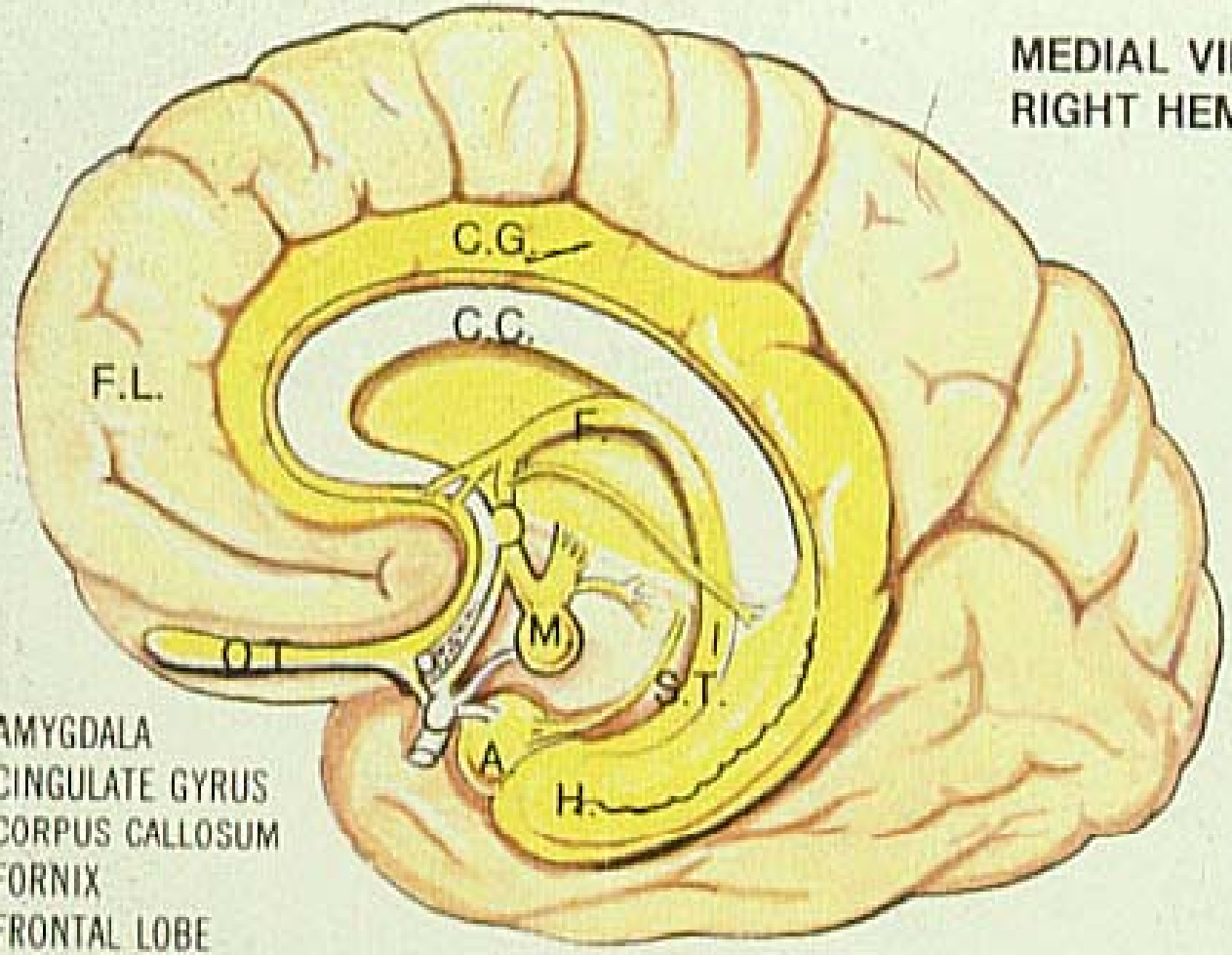
LATERAL VIEW OF BRAIN



INFERIOR VIEW OF BRAIN



THE LIMBIC SYSTEM



A. – AMYGDALA
C.G. – CINGULATE GYRUS
C.C. – CORPUS CALLOSUM
F. – FORNIX
F.L. – FRONTAL LOBE
H. – HIPPOCAMPUS
M. – MAMMILLARY BODY
O.T. – OLFACTORY TRACT AND BULB
S.T. – STRIA TERMINALIS

The Limbic system includes the thalamus, the hypothalamus, amygdala, parts of the reticular formation in the brain stem, and the limbic region of the cerebral cortex. Its functions deal primarily with behavior and emotions.

Brain regions associated with personality traits

Adapted from Carter R, Aldridge S, Page M, and Parker S: The Human Brain Book. London, Dorling Kindersley Limited, 2009.

Aggression: Reduced cingulate cortex volume and activity

Decision values: Central orbitofrontal activation

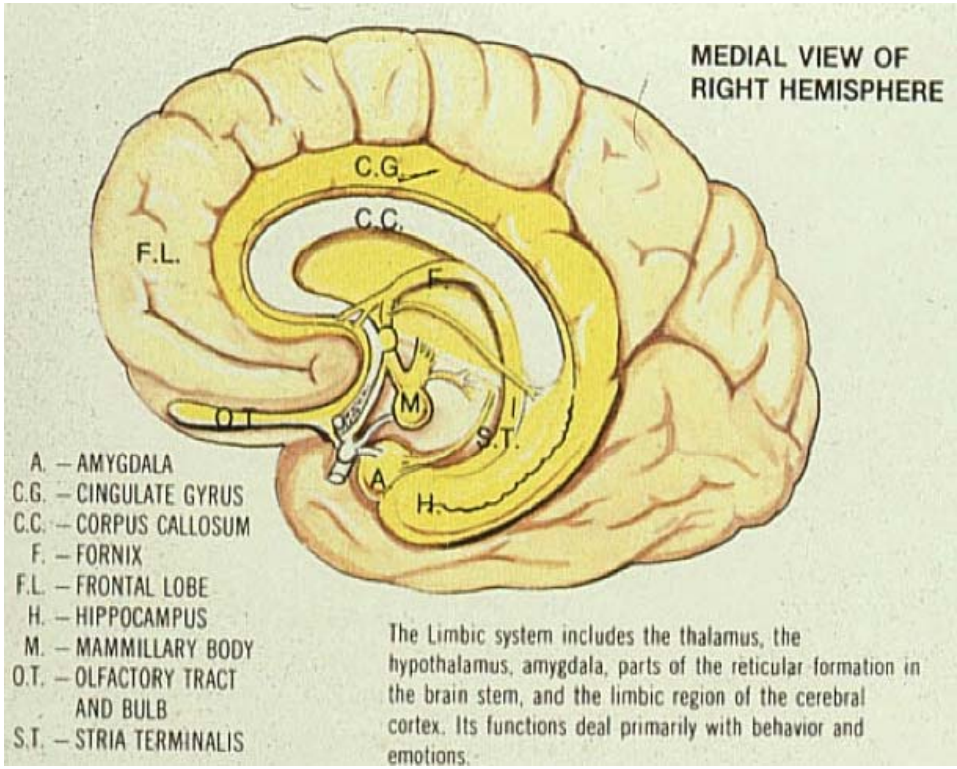
Dispassionate analysis: Increased anterior cingulate activity

Emotional bias in moral decisions: Ventromedial and orbitofrontal prefrontal cortices activation

Emotional memory storage: Amygdala

Goal values: Medial orbitofrontal activation

Extroversion: Reduced dorsolateral prefrontal cortex, anterior cingulate and thalamus activity



The Limbic system includes the thalamus, the hypothalamus, amygdala, parts of the reticular formation in the brain stem, and the limbic region of the cerebral cortex. Its functions deal primarily with behavior and emotions.

Brain regions associated with personality traits

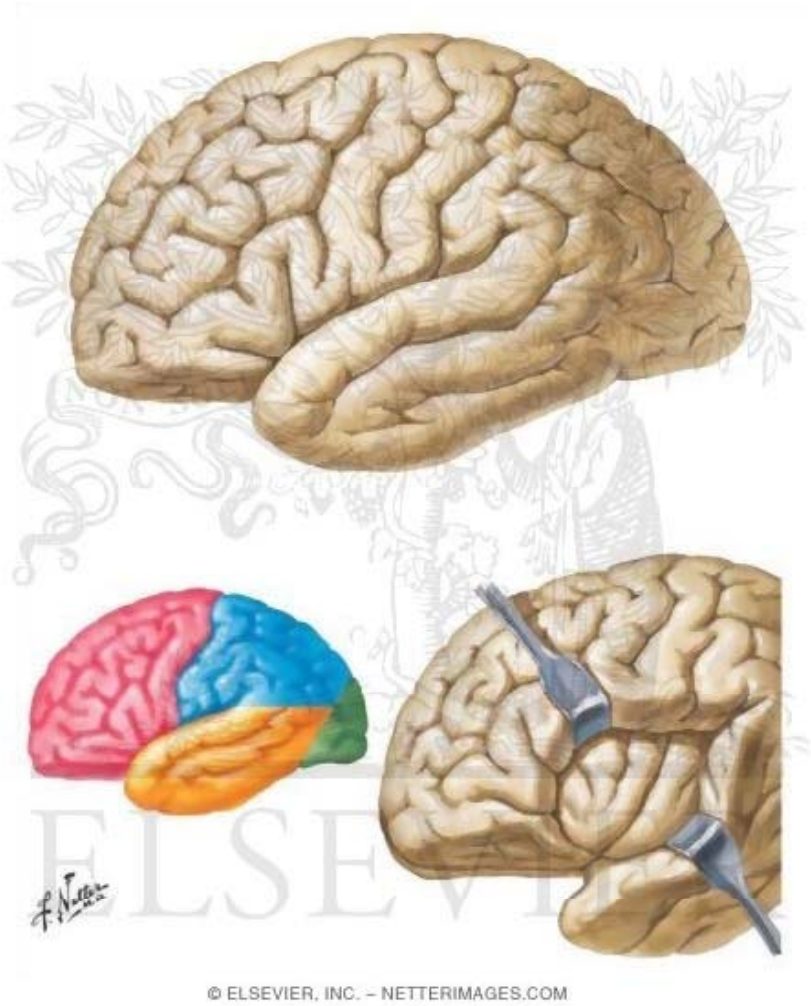
Empathy/self-reflection: Insula activation

Insightful/"Eureka" moments: Increased superior temporal gyrus activity

Mistrust/disbelief: Reduced insula activity

Novelty seeking: Increased hippocampus and striatum activity

Conditioned memory storage: Cerebellum



Brain regions associated with personality traits

Personal awareness of mental state/character: Medial prefrontal cortex

Personal space boundaries: Motor, somatosensory, cingulate and parietal cortices

Prediction errors: Ventral striatum (caudate-putamen) activation

Punctuality/subjective time sense: Substantia nigra, basal ganglia and prefrontal circuits

Reflective/comparing past-present situations: Lateral prefrontal cortex activation

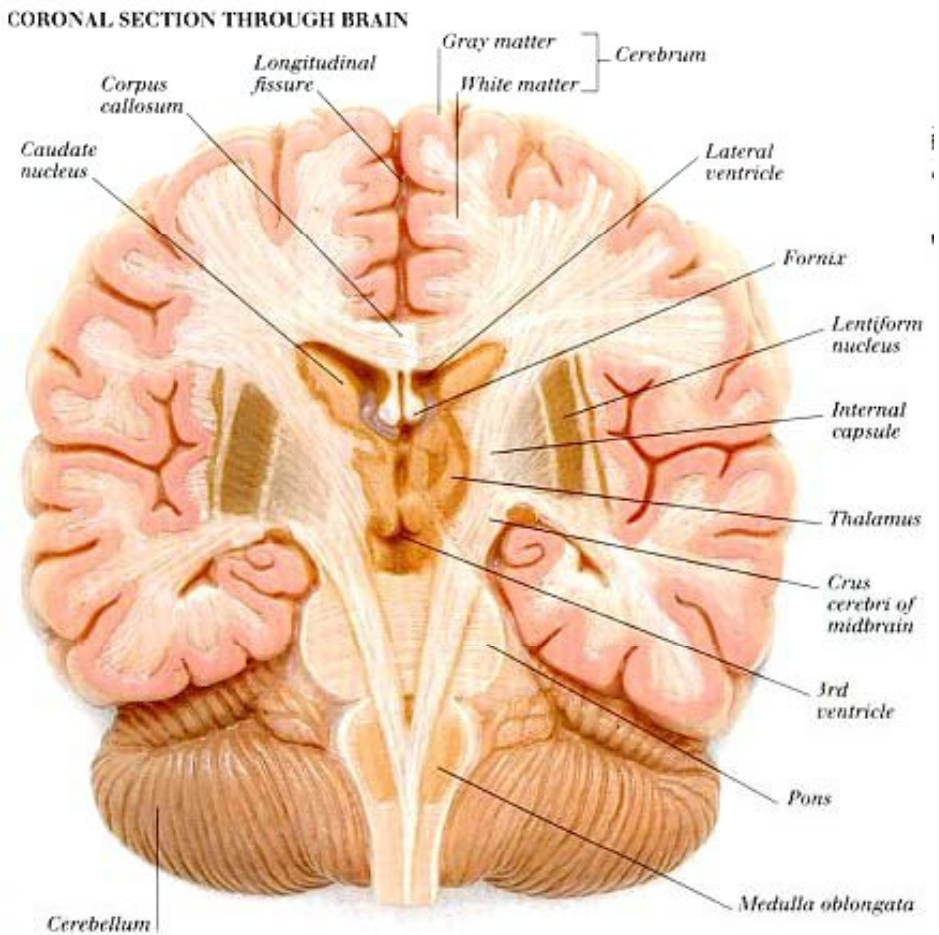
Social avoidance/fear/anxiety: Increased amygdala activity

Self-monitoring/guiding behavior: Cingulate cortex

Social comfort/safety: Increased striatal activity

Trust/belief: Increased ventromedialprefrontal cortex activity

Unfairness: Increased insula activity



Guide to Brain Anatomy and Function

Frontal Lobe

Function: Integral to personality, involved in tracking, sense of self, arousal, and awareness of environment. Responsible for executive functioning & judgments, emotional response & stability, language, and memory for habit

Symptoms of Impairment

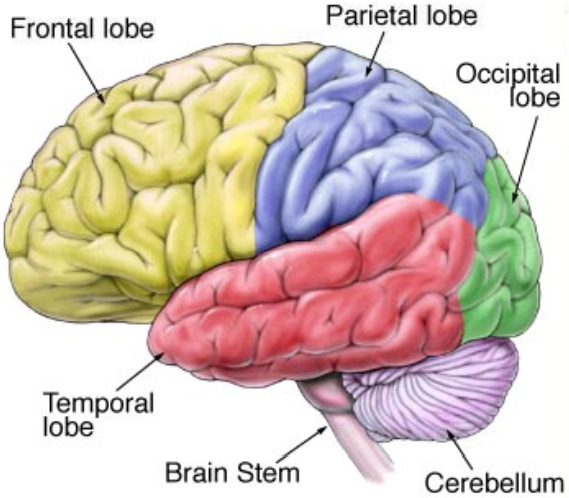
- Changes in personality & social behavior
- Loss of spontaneity in interacting with others
- Sequencing (difficulty planning and completing complex tasks in correct order)
- Perseveration (repeating same actions and comments over without conscious awareness of having done so)
- Loss of flexibility in thinking (mental rigidity)
- Distractibility
- Mood swings
- Diminished abstract reasoning
- Difficulty with problem solving
- Language difficulties (usage and word finding)
- Loss of simple movement of various body parts

Brain Stem

Function: Plays role in heart rate, swallowing, reflexes to sight and sound, sweating, blood pressure, digestion, temperature levels of alertness, ability to sleep, and balance

Symptoms of Impairment

- Decreased vital capacity in breathing
- Swallowing food & water
- Difficulty with organization/perception of environment
- Problems with balance and movement
- Dizziness and nausea
- Sleeping difficulties



Temporal Lobes

Function: Play key role in intellect, as well as auditory perception (hearing), long-term memory and some visual perception

Symptoms of Impairment

- Difficulty remembering names and faces
- Difficulty understanding spoken words
- Difficulty with identification of, and verbalization about objects
- Difficulty with concentration
- Short-term memory loss
- Interference with long-term memory
- Aggressive behavior
- Change in sexual interest
- Persistent talking (right lobe damage)
- Difficulty locating objects in environment
- Inability to categorize objects
- Religiosity
- Seizure disorders, auras and strange reveries

Parietal Lobes

Function: Involved in visual perception, tactile (touch) perception, integration of sensory information that allows for understanding of concepts, and goal-directed voluntary movements

Symptoms of Impairment

- Difficulties naming objects
- Difficulties writing words
- Inability to attend to more than one object at a time
- Inability to focus visual attention
- Problems with reading
- Poor hand-eye coordination
- Confusion left-right orientation
- Difficulty performing math calculations
- Difficulty drawing
- Poor visual perception
- Lack of awareness of certain body parts and/or surrounding space

Occipital Lobe

Function: Visual perception system

Symptoms of Impairment

- Impaired vision

Cerebellum

Function: Involved in coordination and control of voluntary movement, balance and muscle tone

Symptoms of Impairment

- Tremors
- Nystagmus (Involuntary movement of eye)
- Ataxia, lack of coordination
- Weak muscles (hypotonia)
- Inability to judge distance and when to stop (dysmetria)
- Inability to perform rapid altering movements
- Slurred speech (ataxic dysarthria)

© 2004, MediVisuals Inc.

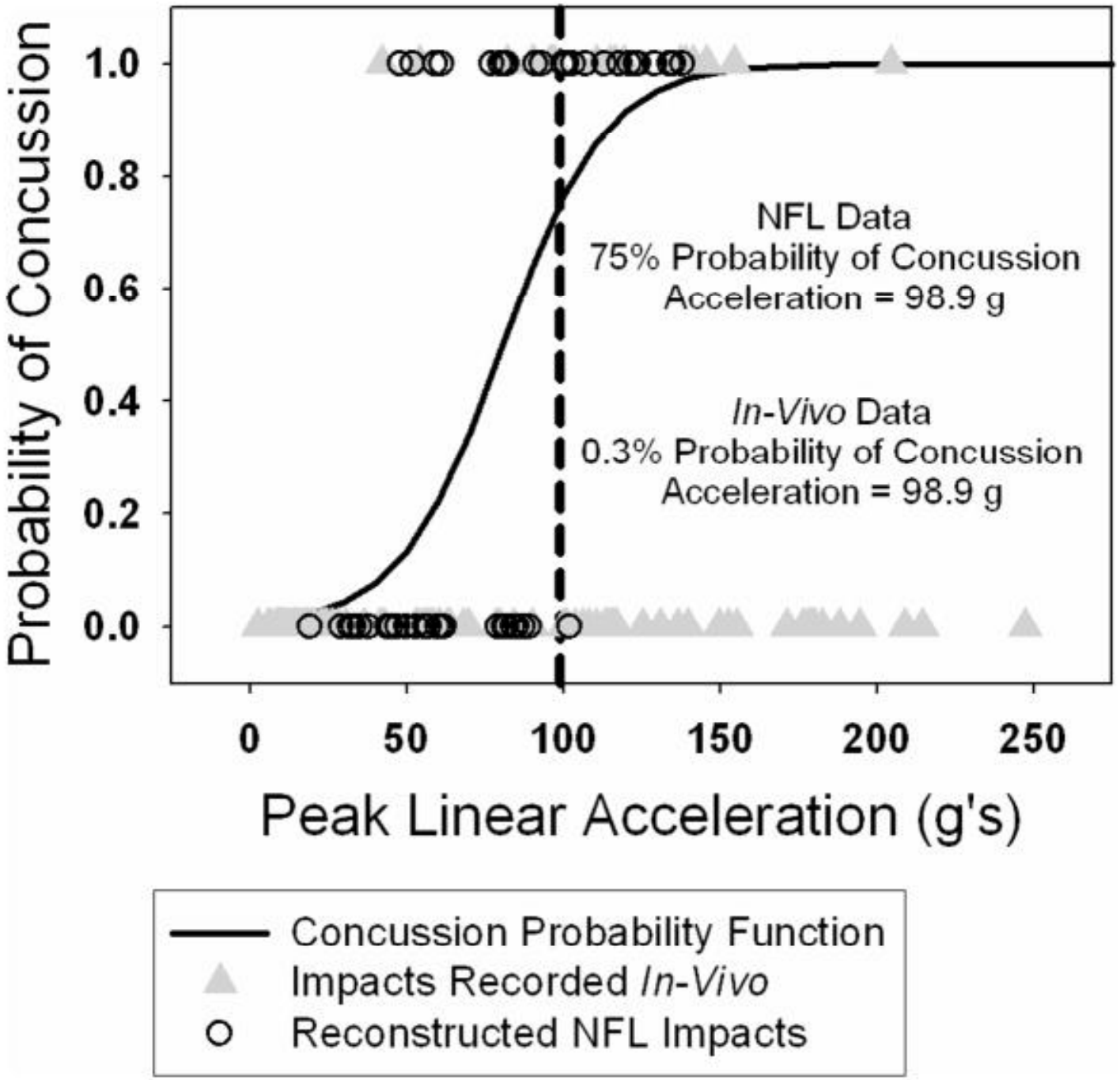
BIOMECHANICS OF TBI

External Forces

(DoD, 2007)

- the head being struck by an object
- the head striking an object
- the brain undergoing an acceleration/ deceleration movement without direct external trauma to the head
- a foreign body penetrating the brain
- forces generated from events such as a blast or explosion
- other force yet to be defined

Blunt force trauma


Probability of Concussion
1.0
0.8
0.6
0.4
0.2
0.0
0
50
100
150
200
250
Peak Linear Acceleration (g's)
NFL Data
75% Probability of Concussion
Acceleration = 98.9 g
In-Vivo Data
0.3% Probability of Concussion
Acceleration = 98.9 g
Concussion Probability Function
Impacts Recorded In-Vivo
Reconstructed NFL Impacts

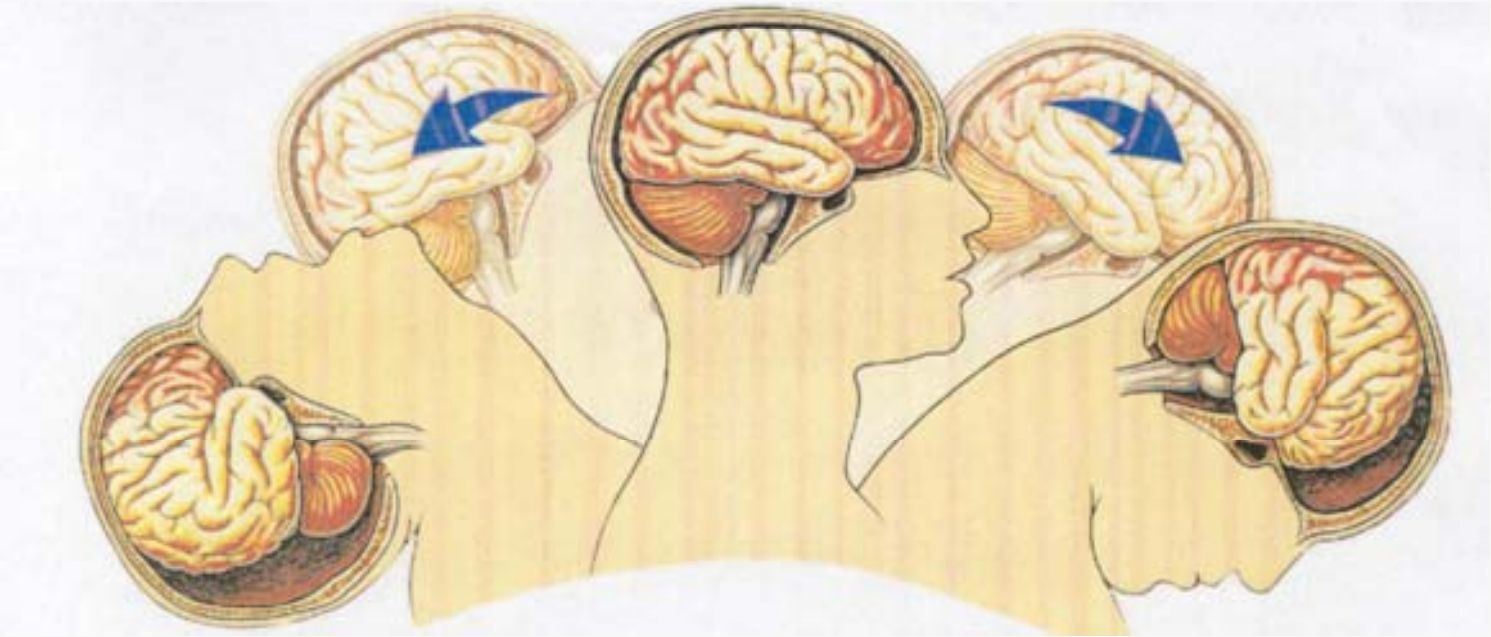
Position	No. of Cases	Incidence(%)	No. of Game Positions	Risk per 100 game-positions[a]
High Risk				
Offensive				
Quarterback	62	7.9%	3,826	1.62(1.22, 2.02)
Wide Receiver	94	11.9%	7,652	1.23(0.98, 1.48)
Tight End	36	4.6%	3,826	0.94(0.63, 1.25)
Running Back	69	8.8%	7,652	0.90(0.69, 1.11)
Defensive				
Secondary	143	18.2%	15,304	0.93(0.78, 1.08)
Moderate Risk				
Offensive				
Offensive Line	56	7.1%	19,130	0.29(0.21, 0.37)
Defensive				
Linebacker	52	6.6%	11,478	0.45(0.33, 0.57)
Defensive Line	67	8.5%	15,304	0.44(0.34, 0.54)
Special Team				
Return Ball Carrier	22	2.8%	3,826	0.58(0.34, 0.82)
Kick Unit	131	16.6%	38,260	0.34(0.28, 0.40)
Low Risk				
Special Team				
Punter	7	0.9%	3,826	0.18(0.05, 0.31)
Return Unit	33	4.2%	38,260	0.09(0.06, 0.12)
Kicker, FGA	1	0.1%	3,826	0.03(–0.02, 0.08)
Kicker, PAT	1	0.1%	3,826	0.03(–0.02, 0.08)
Holder	1	0.1%	3,826	0.03(–0.02, 0.08)
Unknown/Undesignated	12	1.5%		
Total	787	100%		8.08

Source: Neurosurg Focus © 2006 American Association of Neurological Surgeons

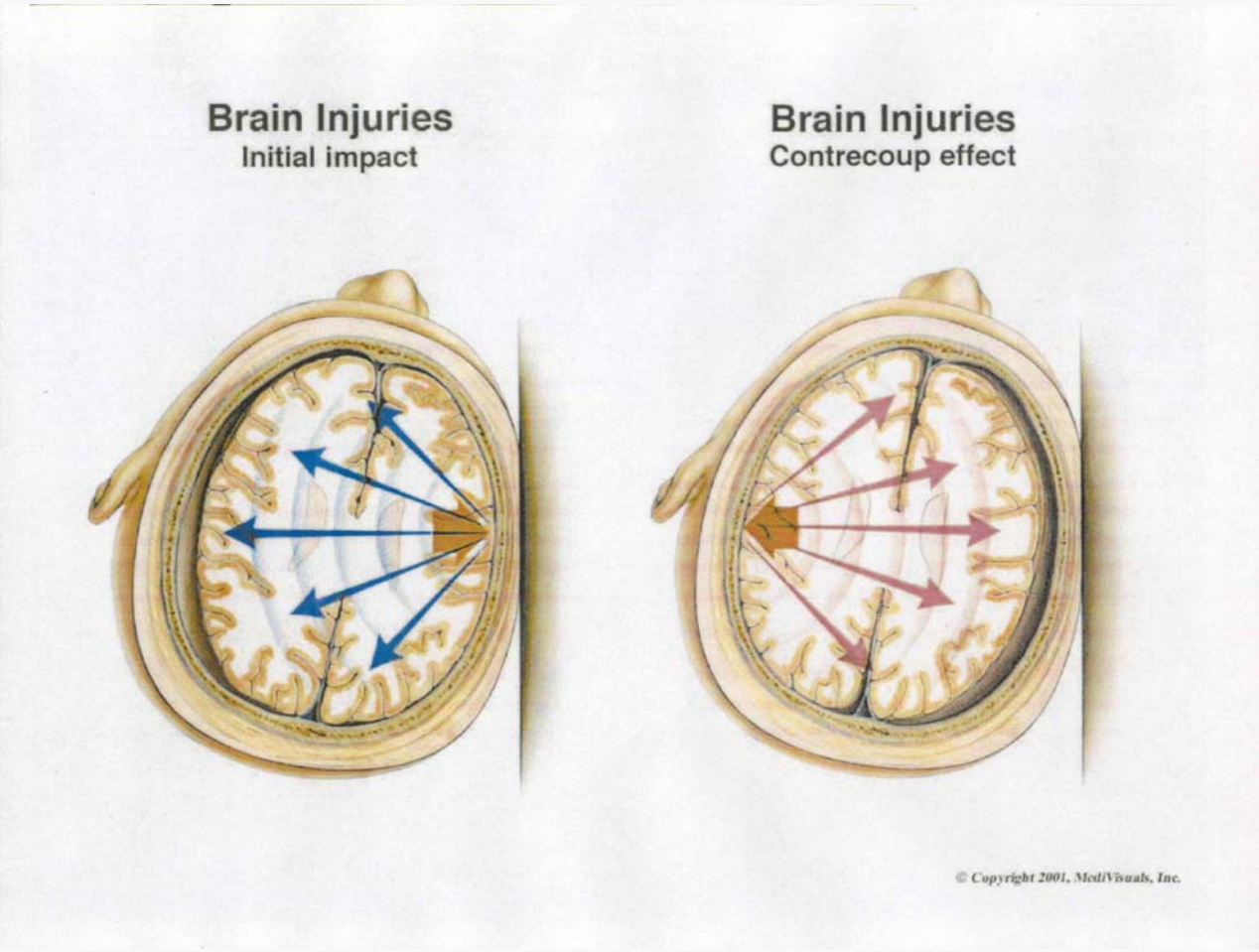
Levels Of Consciousness In Relation To Head Injury Criteria

Head Injury Criteria	AIS Code	Level Of Brain Concussion And Head Injury
135 – 519	1	Headache or dizziness
520 – 899	2	Unconscious less than 1 hour – linear fracture
900 – 1254	3	Unconscious 1 – 6 hours – depressed fracture
1255 – 1574	4	Unconscious 6 – 24 hours – open fracture
1575 – 1859	5	Unconscious > 25 hours – large hematoma
> 1860	6	Non survivable

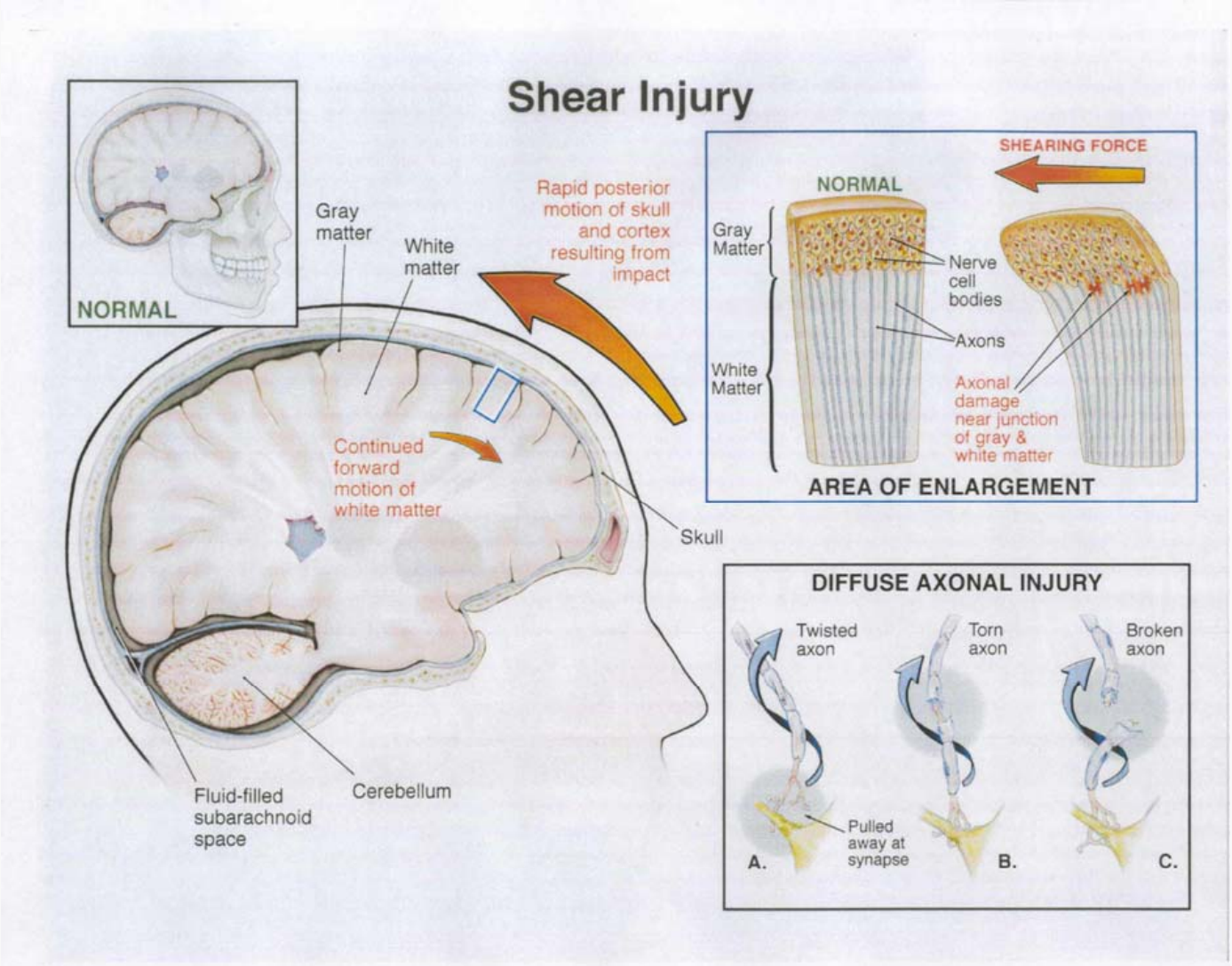
Mechanics of traumatic brain injury



Mechanics of traumatic brain injury



Mechanics of traumatic brain injury



PATHOPHYSIOLOGY

DIFFUSE AXONAL INJURY

Primary neuropathological change in any TBI

- Microscopic post-mortem finding

Since 1980's, recognized 2 causes in severe TBI:

- Acute: mechanical shearing
- Delayed: cellular metabolic alteration due to trauma

Search for interventions to improve clinical outcome in severe TBI by reducing delayed cellular metabolic injury

- BTF Guidelines
- Fluid resuscitation
- ICP monitoring; decompressive craniectomy
- Metabolic interventions: hypothermia; tirilizide; progesterone

DIFFUSE AXONAL INJURY

ACUTE = CELL MURDER

Mechanical disruption of axon

Due to differential densities

Myelinated (white matter)

Non-myelinated (gray matter)

Tearing damages vascular supply to axon

Releases Hgb -> hemosiderin

DELAYED = CELL SUICIDE

Metabolic process

Mismatch between energy demands & resources

Mitochodrial injury

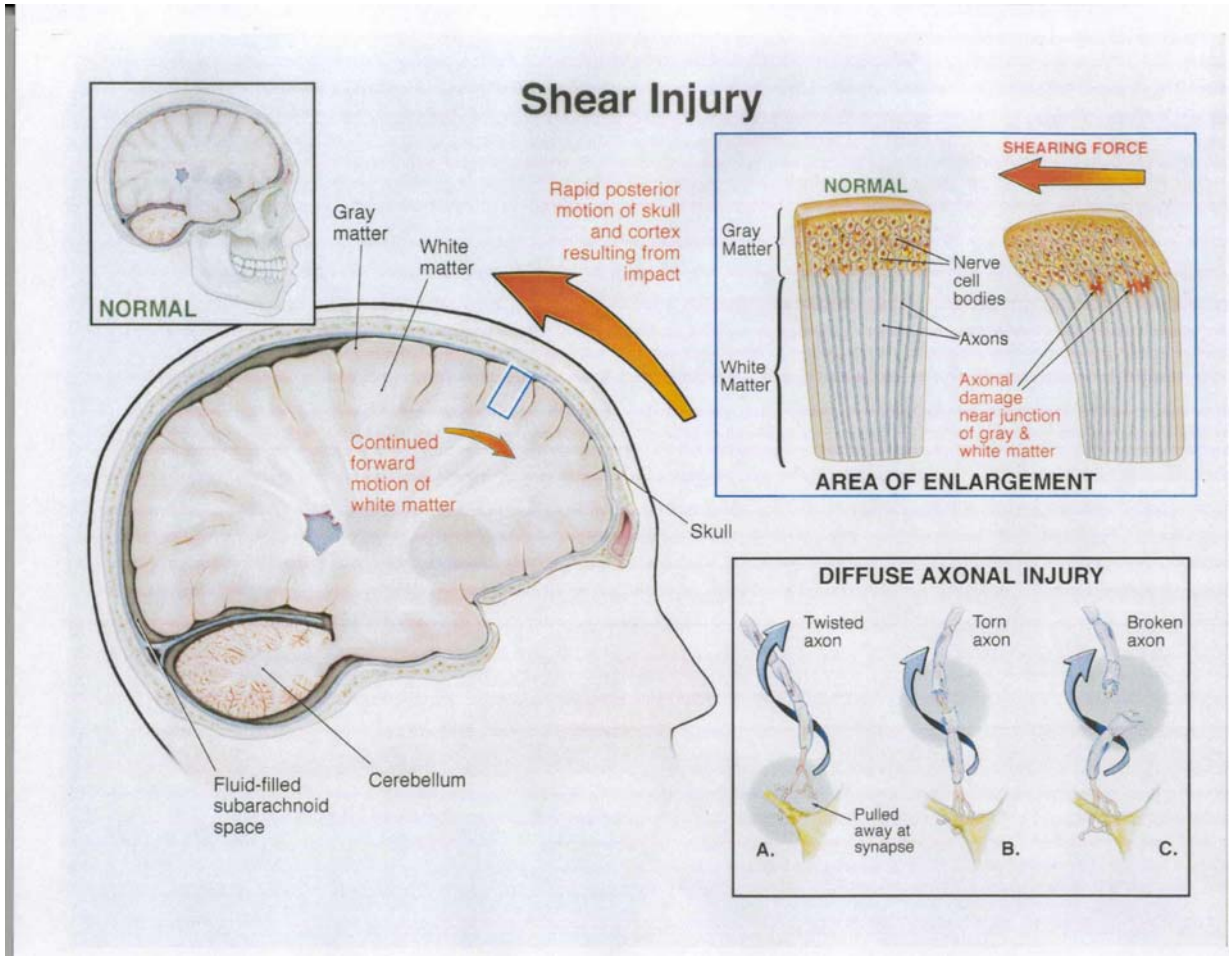
Loss of synthetic capacity of neuron

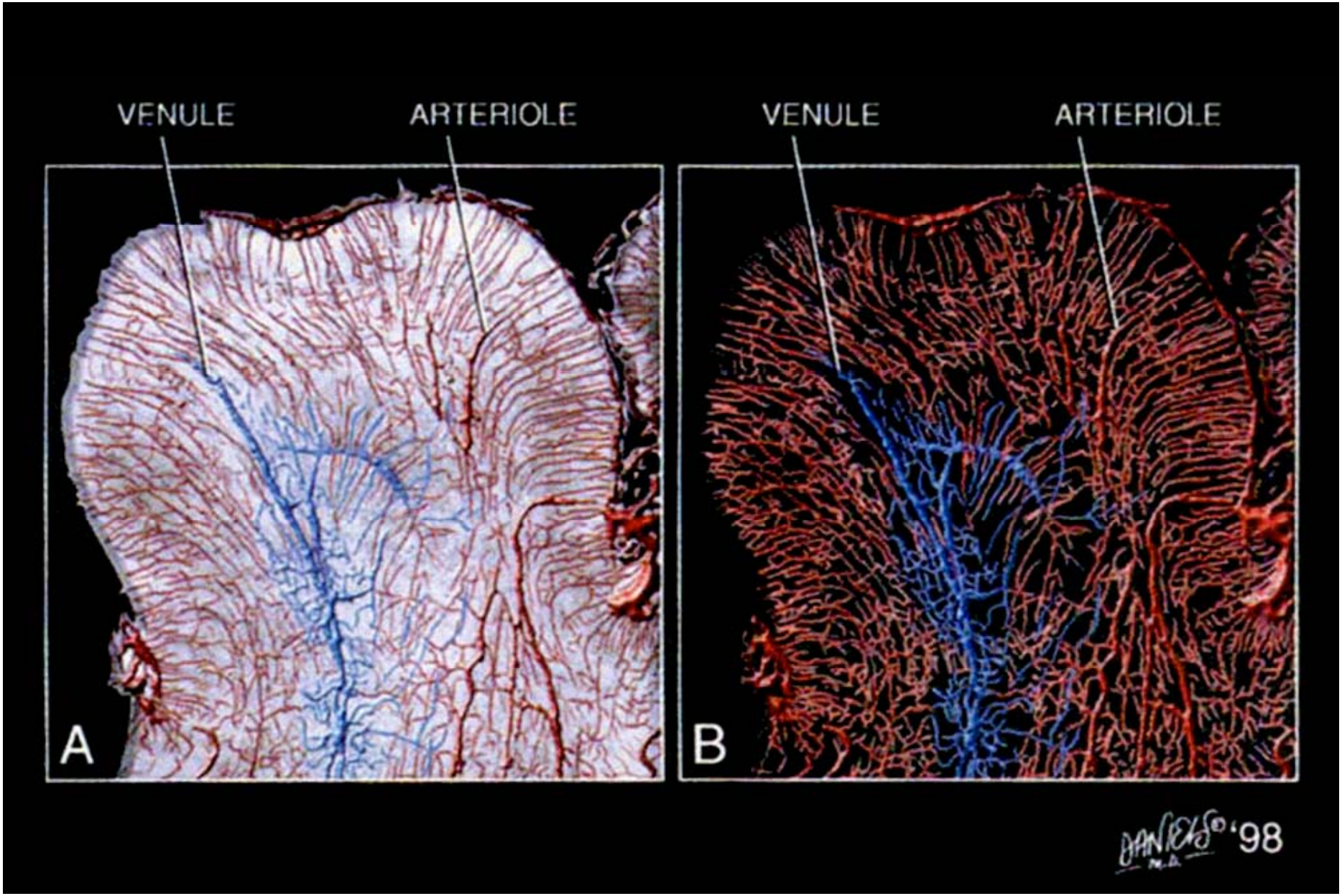
Internal loss of structure

Apoptosis

No damage to vascular supply

Mechanics of traumatic brain injury
“cell murder”




VENULE
ARTERIOLE
A
VENULE
ARTERIOLE
B
DANIELS © '98

“Cell murder” on CT

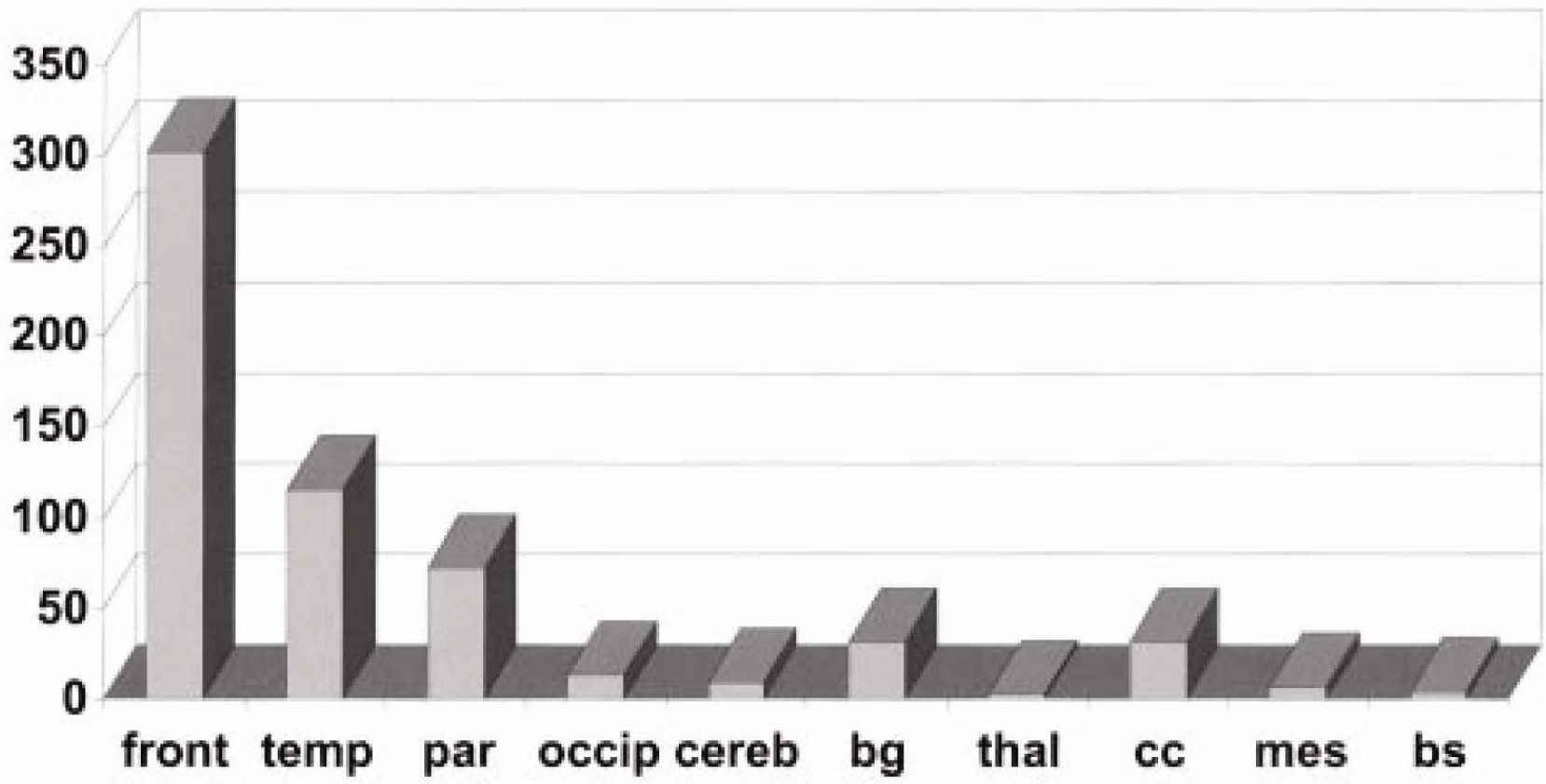
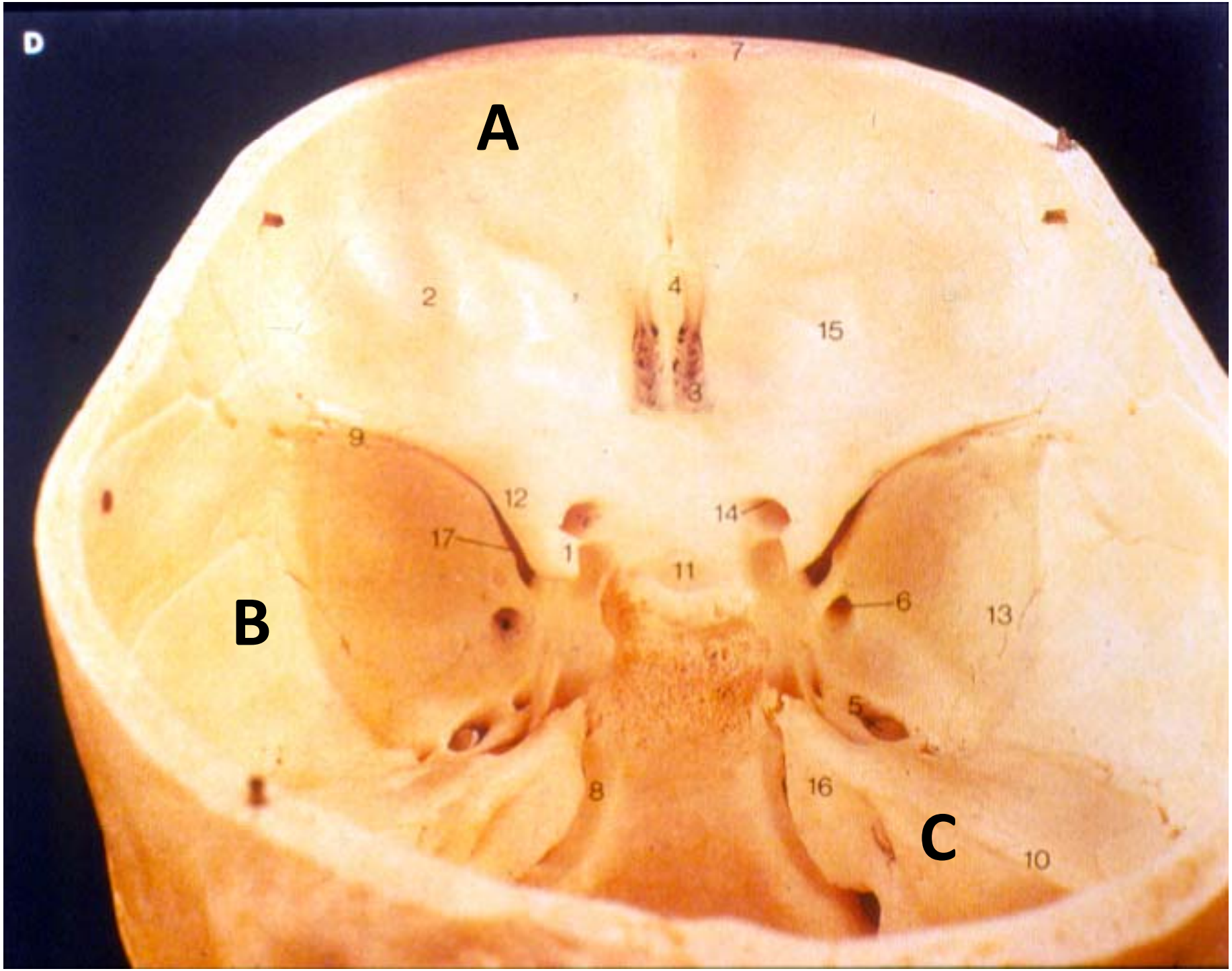


FIG 1. Frequency and site of traumatic microbleeds according to 10 brain areas. Shown is the total number of traumatic microbleeds in each brain area (*front*, frontal lobe; *temp*, temporal lobe; *par*, parietal lobe; *occip*, occipital lobe; *cereb*, cerebellum; *bg*, basal ganglia; *thal*, thalamus; *cc*, corpus callosum; *mes*, mesencephalon; *bs*, brain stem).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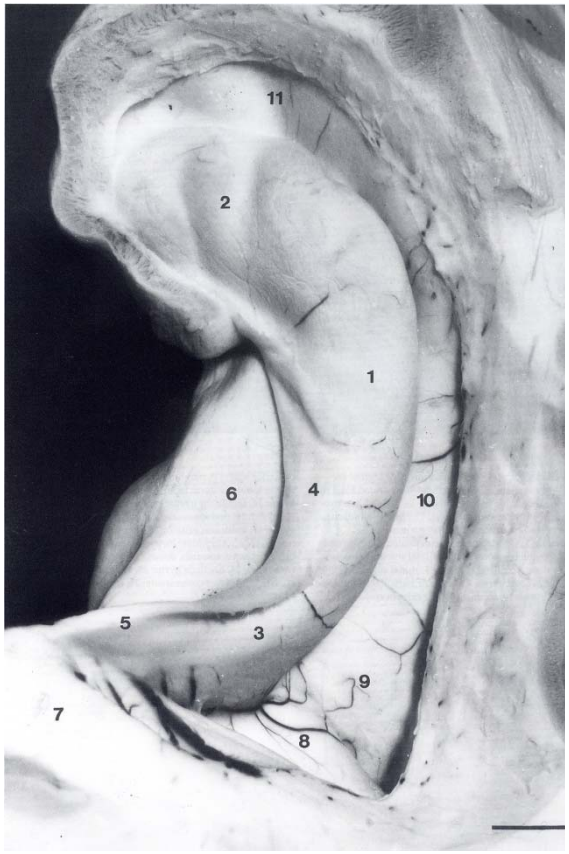
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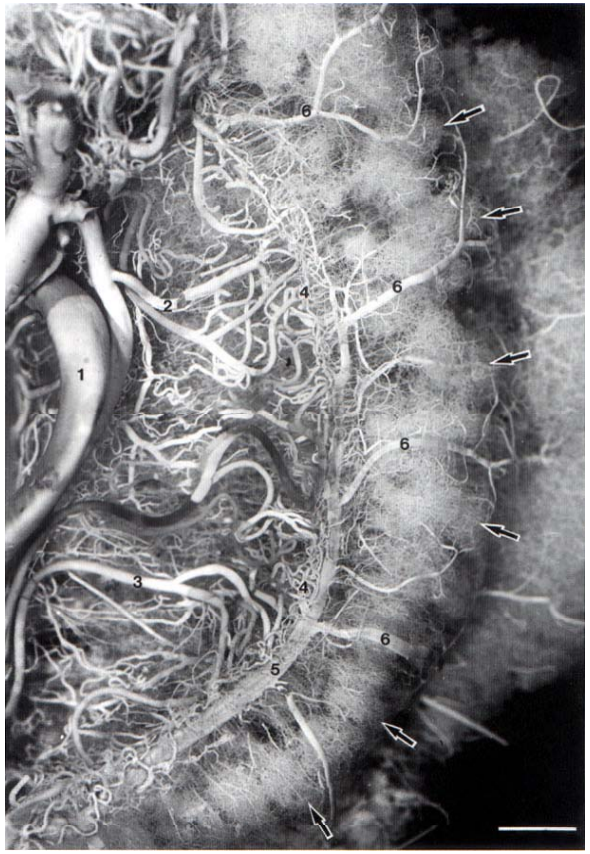
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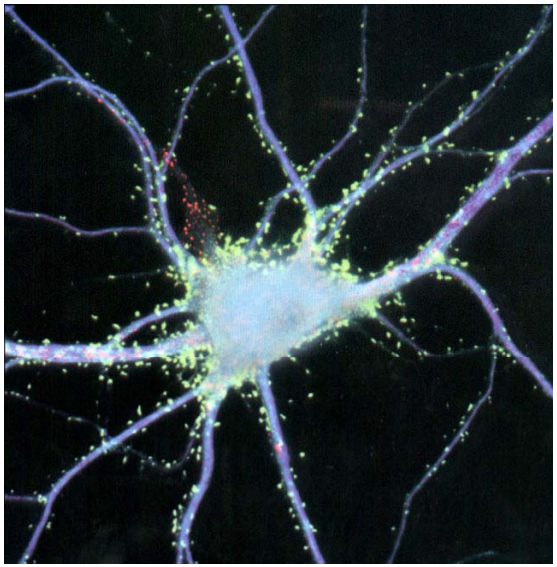

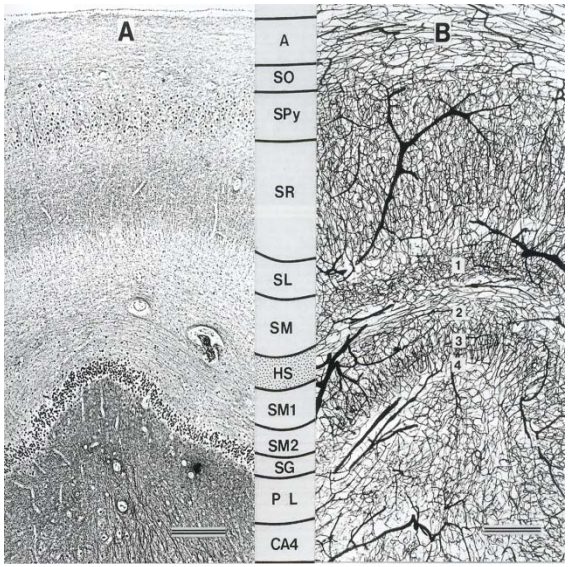
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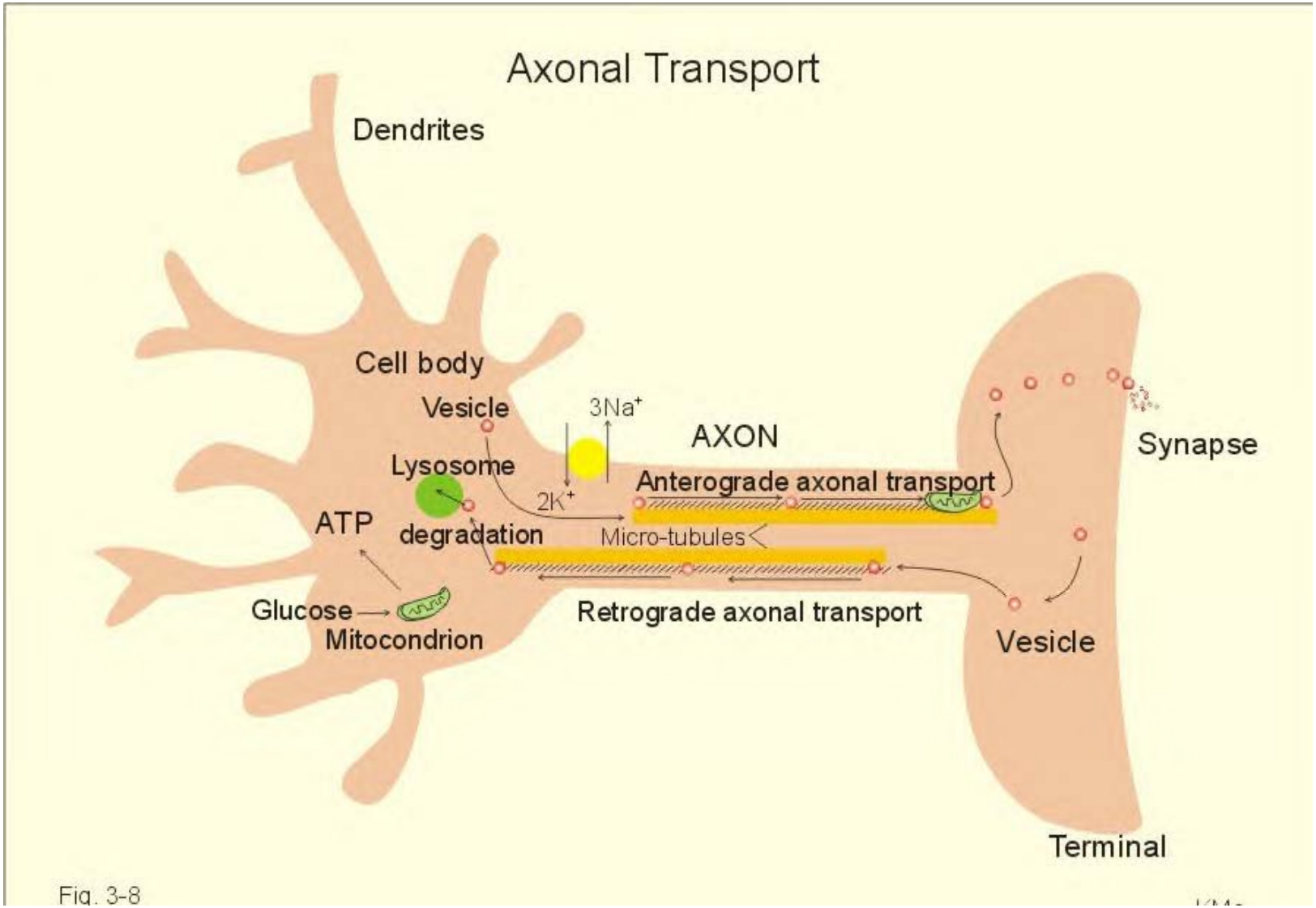
Powering the neuron



DIFFUSE AXONAL INJURY

ACUTE = CELL MURDER

Mechanical disruption of axon

Due to differential densities

Myelinated (white matter)

Non-myelinated (gray matter)

Tearing damages vascular supply to axon

Releases Hgb -> hemosiderin

DELAYED = CELL SUICIDE

Metabolic process

Mismatch between energy demands & resources

Mitochodrial injury

Loss of synthetic capacity of neuron

Internal loss of structure

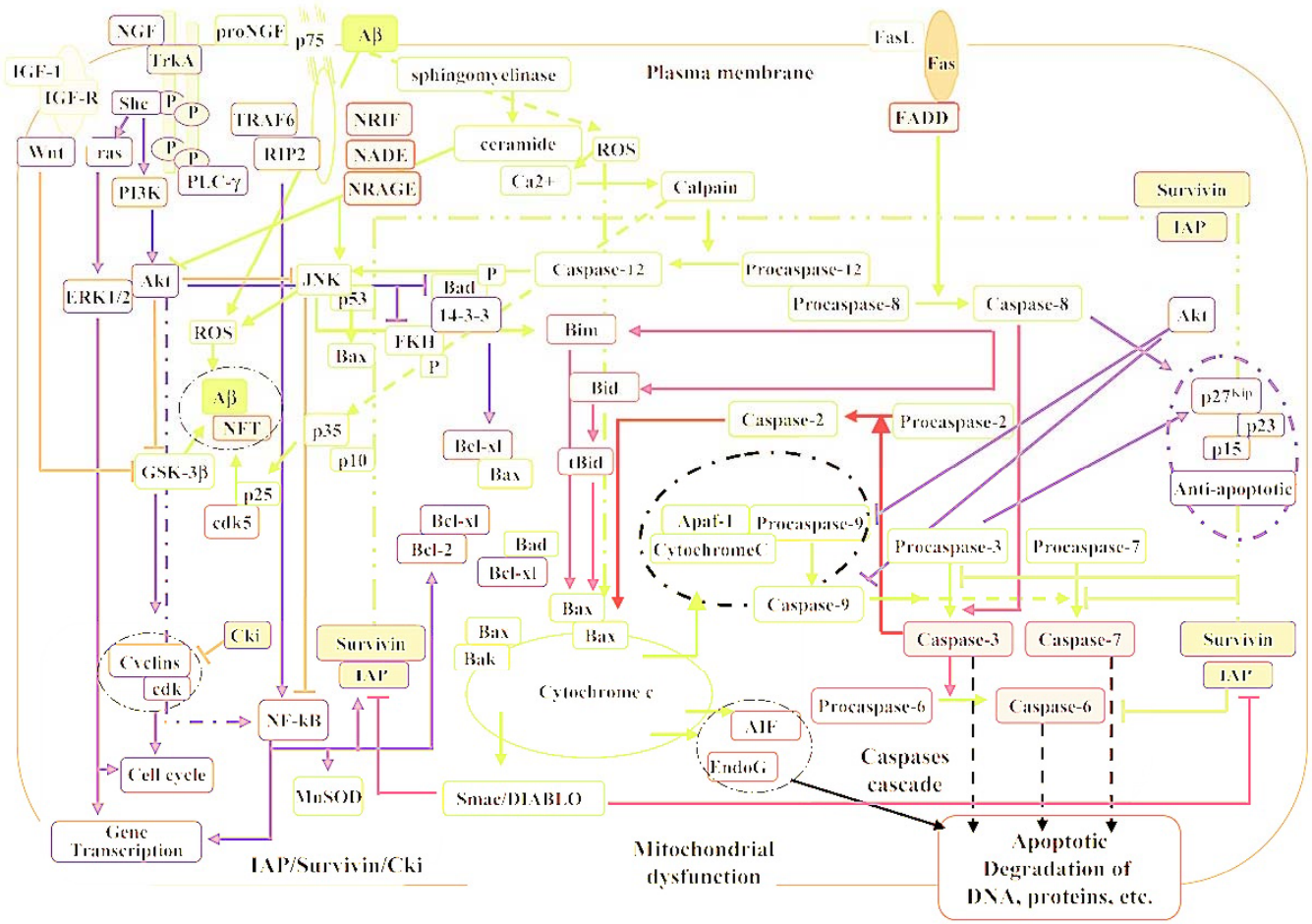
Apoptosis

No damage to vascular supply

Energy crisis in TBI
"cell suicide"

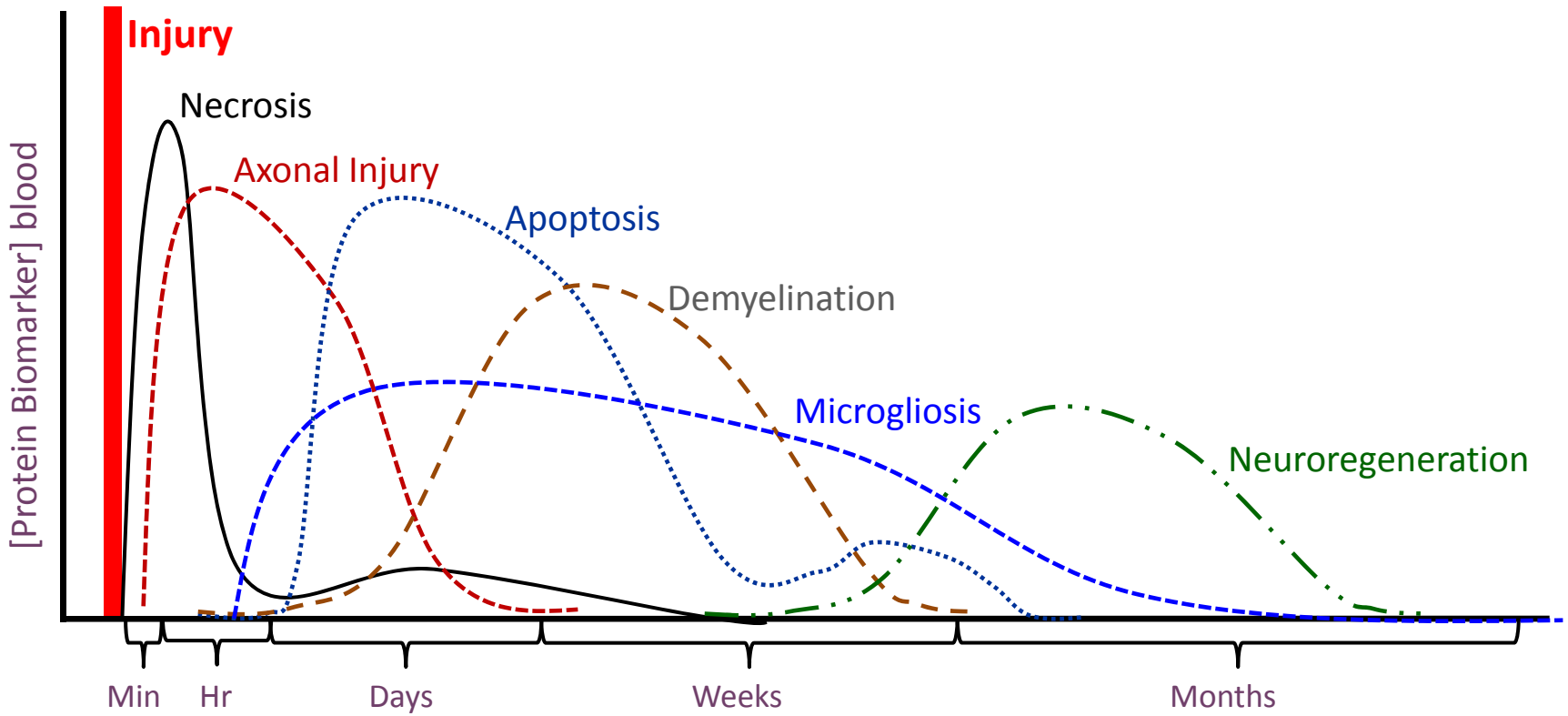
- Oxygen and glucose needed for neuronal metabolism
- With TBI, release of excitatory neurotransmitter (glutamate) causes increase in metabolism
- Blood flow to neuron unable to keep up with demand
- Net effect reduces neuronal metabolism (metabolic depression)
- Reduced metabolism makes the neuron "leaky" and less able to manufacture proteins needed for stability
- Gradual disconnection of axon from neuronal body (apoptosis)
- No corresponding damage to adjacent blood vessels

Mechanics of traumatic brain injury
“cell suicide”



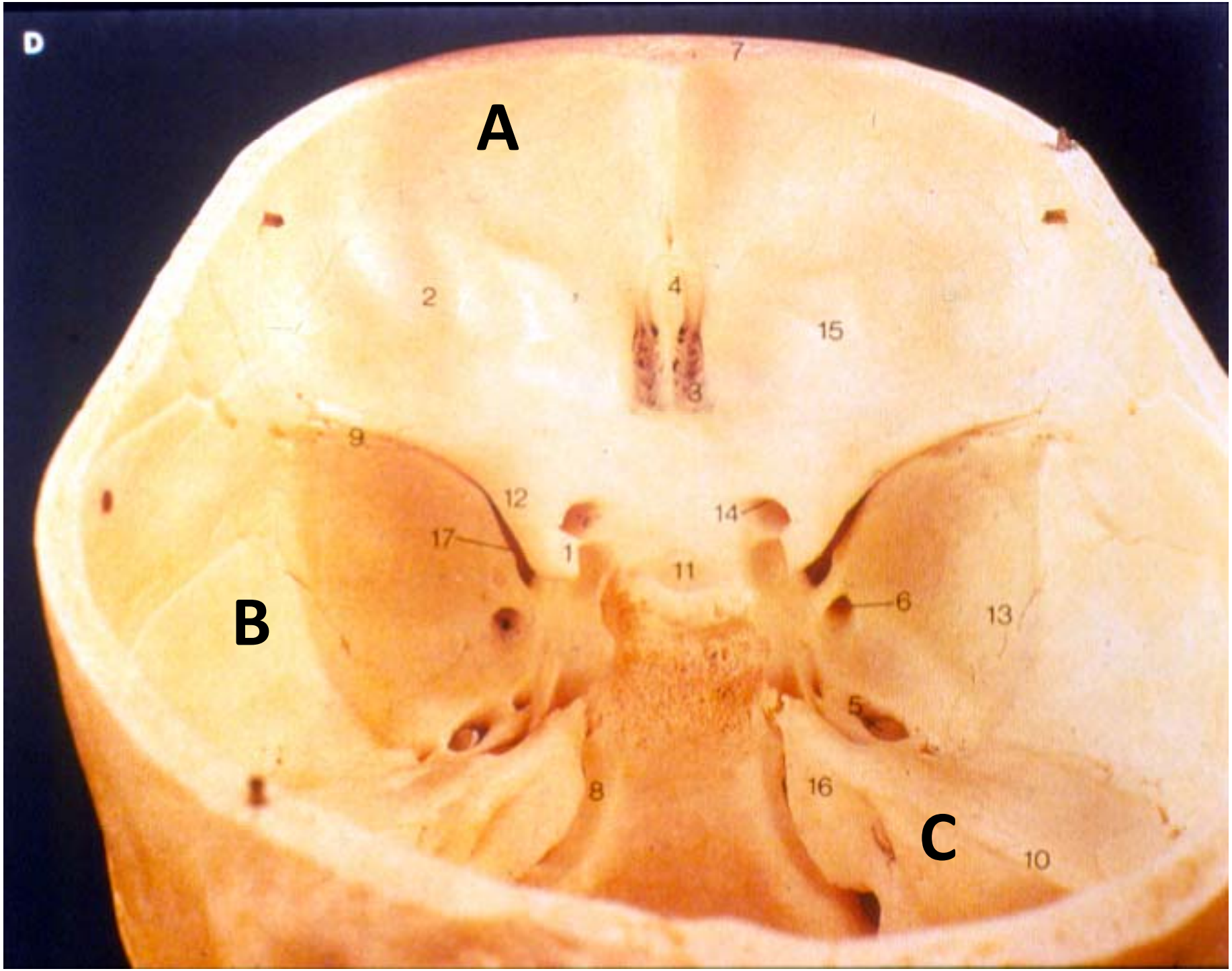
BIOMARKER BACKGROUND

What happens after brain injury?



Pathophysiology of TBI

- Diffuse axonal injury:
  - mechanical – early (cell murder)
  - metabolic – late (cell suicide)
- Focal contusions and bony prominences
- Increased intracranial pressure/edema
- Mass effects:
  - Epidural hematoma
  - Subdural hematoma
  - Intracerebral hematoma
- Excitotoxic (glutamate) cascade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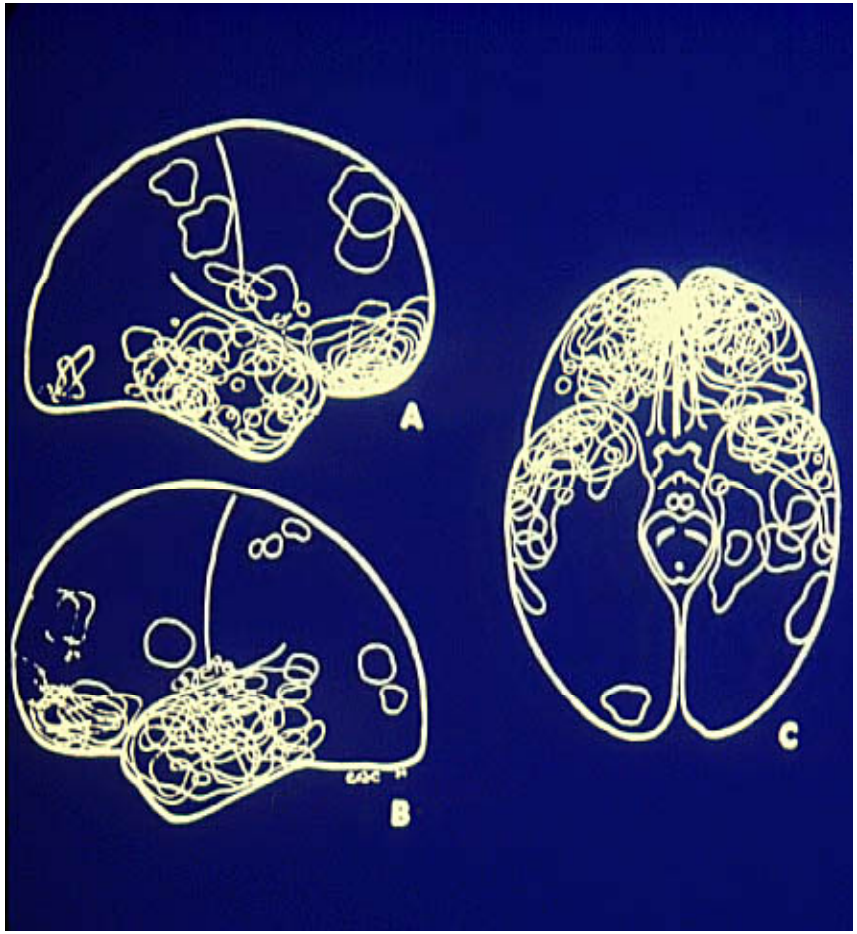
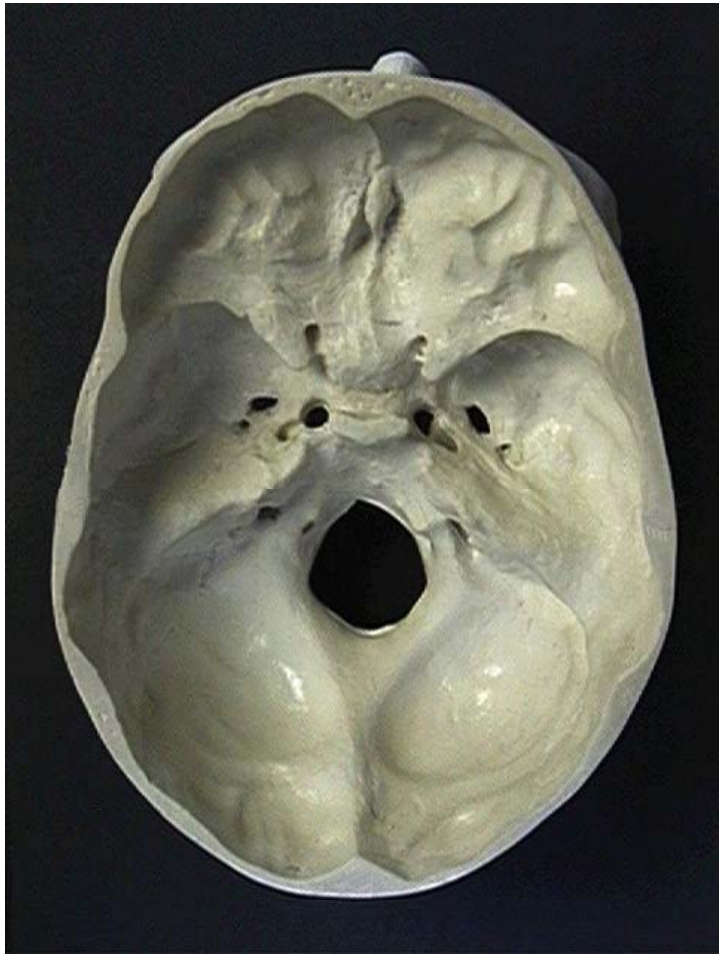
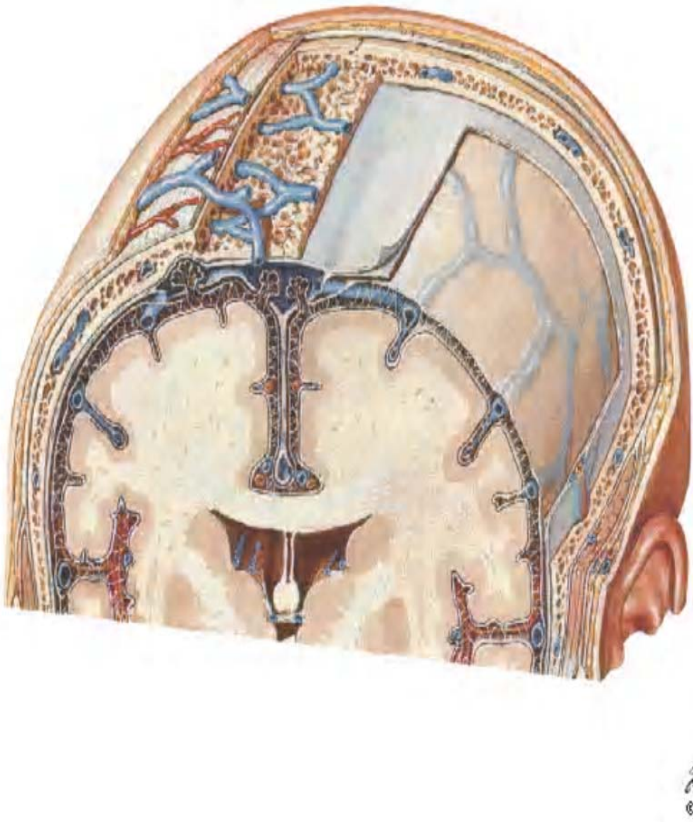
Internal view of human skull

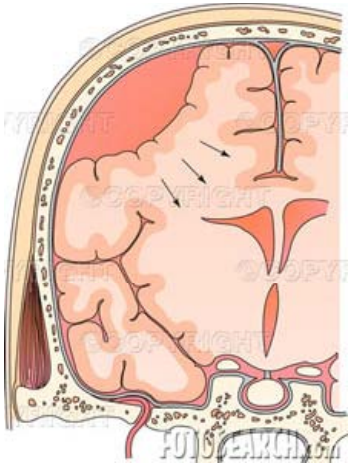


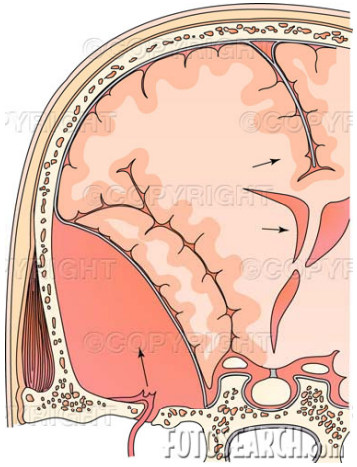


Hematoma (blood clots)

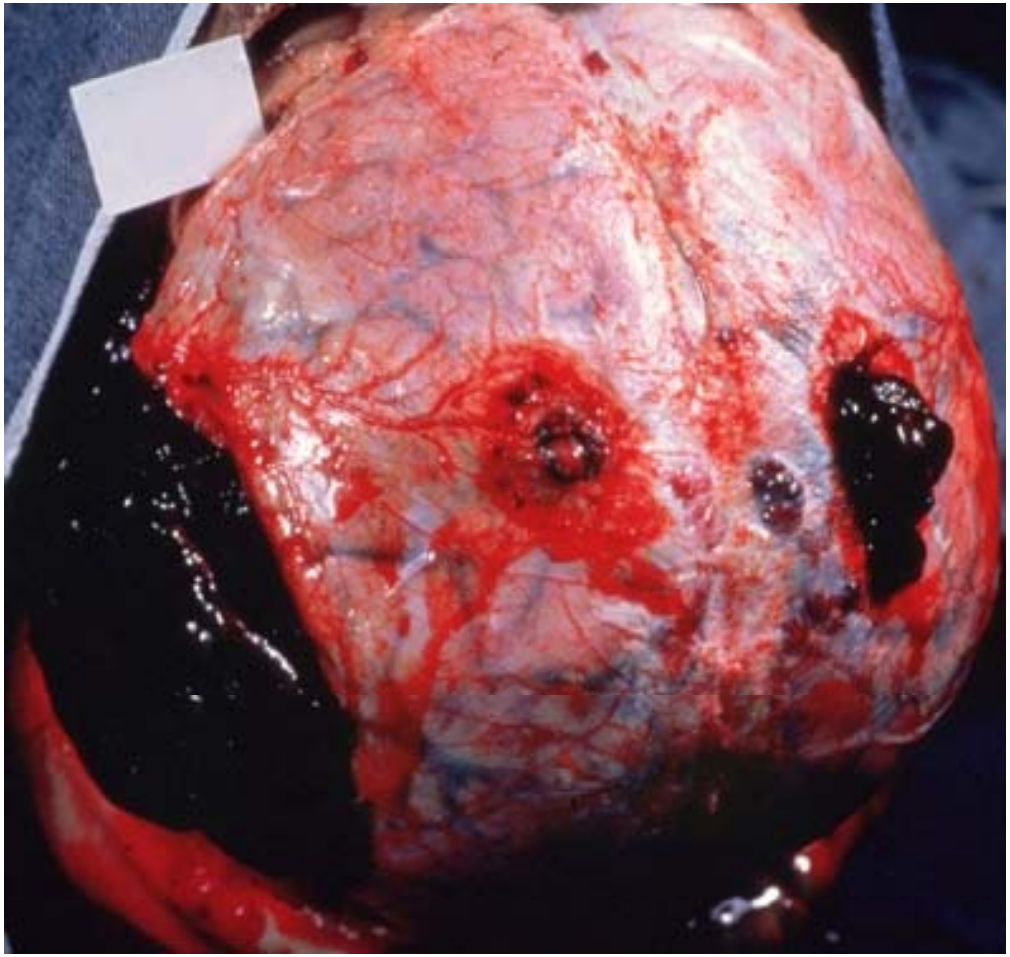
Subdural & Epidural





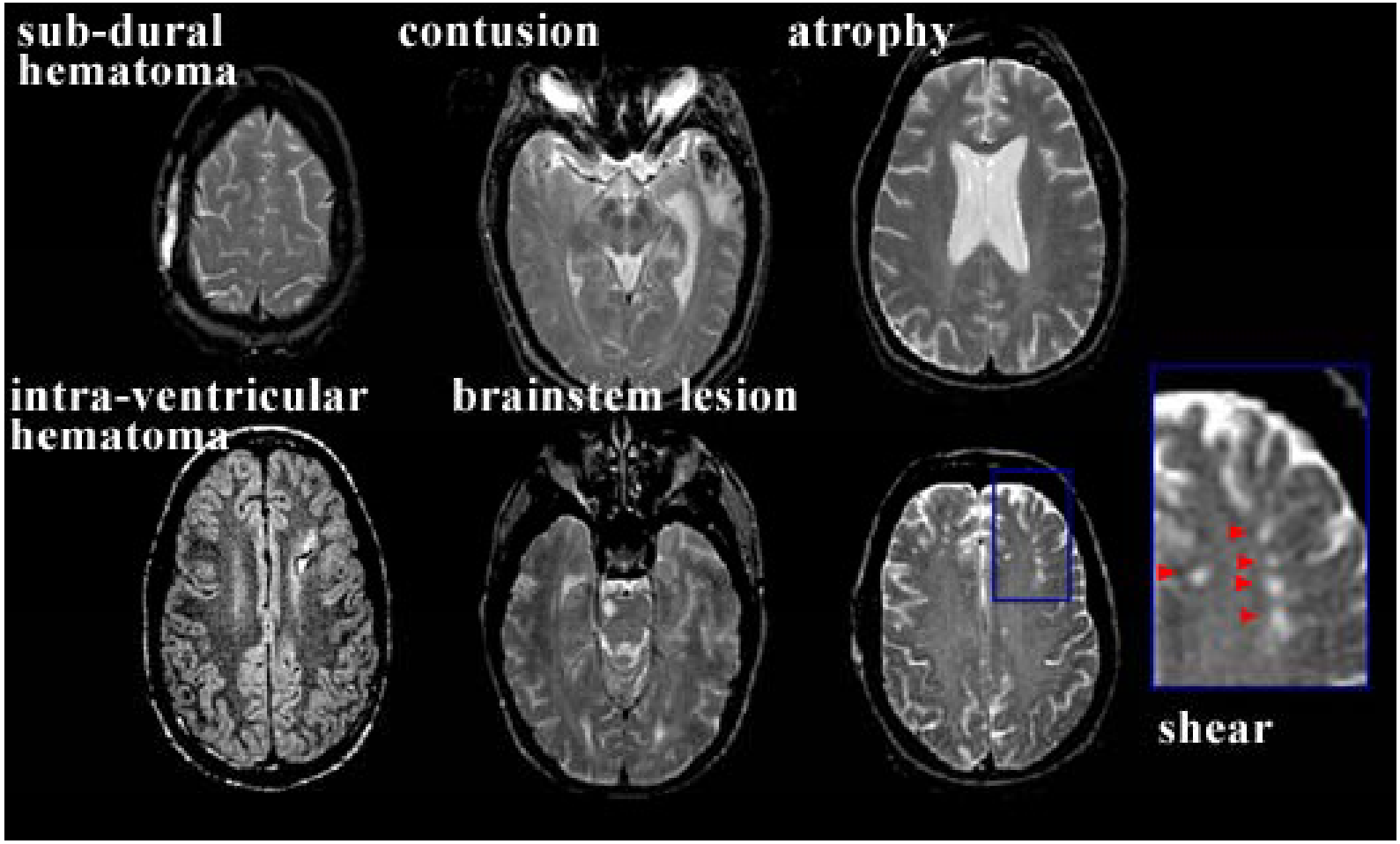


Epidural hematoma



Cerebral compression due to epidural hematoma

Hemorrhagic trauma (MRI)



TBI SEVERITY

Injury Severity (Acute)

(DoD, 2007)

Injury severity is determined at the time of the injury, but this severity level while having some prognostic value does not necessarily reflect the patient's ability to function.

It is recognized that serial assessments of the patient’s cognitive, emotional/behavioral and social functioning are required.

Glasgow Coma Scale

1974

Graham Teasdale &
Bryan J. Jennett

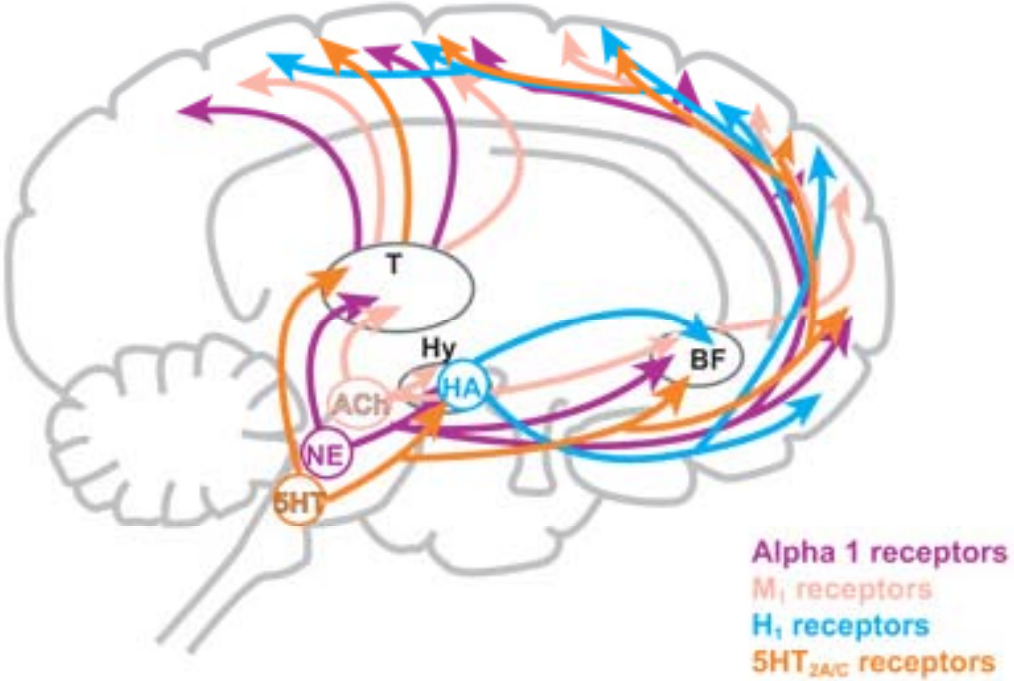
Outcome studies in coma

Glasgow Coma Scale	
Motor Responses:	Score
Obeys commands	6
Localizing responses to pain	5
Generalized withdrawal to pain	4
Flexor posturing to pain	3
Extensor posturing to pain	2
No motor response to pain	1
Verbal Responses:	
Oriented	5
Confused conversation	4
Inappropriate speech	3
Incomprehensible speech	2
No speech	1
Eye opening	
Spontaneous eye opening	4
Eye opening to speech	3
Eye opening to pain	2
No eye opening	1
TOTAL (Add above 3 scores)	Range = 3 - 15

Table 1

Ascending Reticular Activating System

FIGURE 1.
Cortical Arousal



T=thalamus; Hy=hypothalamus; ACh=acetylcholine; HA=histamine; BF=basal forebrain; NE=norepinephrine; 5-HT=serotonin; M=muscarinic; H=histamine.

Stahl SM. *CNS Spectr.* Vol 13, No 12. 2008.

Severity: Acute Classifications

(DoD, 2007)

Mild	Moderate	Severe
Normal structural imaging	Normal <u>or</u> abnormal structural imaging	Normal <u>or</u> abnormal structural imaging
LOC = 0-30 min	LOC >30 min to 24 hours	LOC > 24 hrs
AOC = a moment up to 24 hrs	AOC >24 hours Severity Based on Other Criteria	
PTA = 0-1 day	PTA >1 to 7 days	PTA > 7 days

MTBI Diagnostic Criteria

American Congress of Rehabilitation Medicine

Center for Disease Control and Prevention

World Health Organization

Veterans Administration/Dept of Defense

National Academy of Neuropsychology

ACRM (1993)

Mild Traumatic Brain Injury Committee of the
Head Injury Interdisciplinary Special Interest Group

A traumatically induced physiological disruption of brain function, as manifested by *at least* one of the following:

- any loss of consciousness
- any loss of memory for events immediately before or after the accident
- any alteration in mental state at the time of the accident (e.g., feeling dazed, disoriented, or confused) and
- focal neurologic deficit(s) that may or may not be transient

But where the severity of the injury does not exceed the following:

- loss of consciousness of approximately 30 min or less
- after 30 min, an initial Glasgow Coma Scale score of 13–15 and
- post-traumatic amnesia not greater than 24 hr

CDC (2003)

Report to Congress on Mild Traumatic Brain Injury in the United States:

Steps to Prevent a Serious Public Health Problem

September 2003

Recommended Definitions for Mild Traumatic Brain Injury

Incident Cases of MTBI

The **conceptual definition** of MTBI is an injury to the head as a result of blunt trauma or acceleration or deceleration forces that result in one or more of the following conditions:

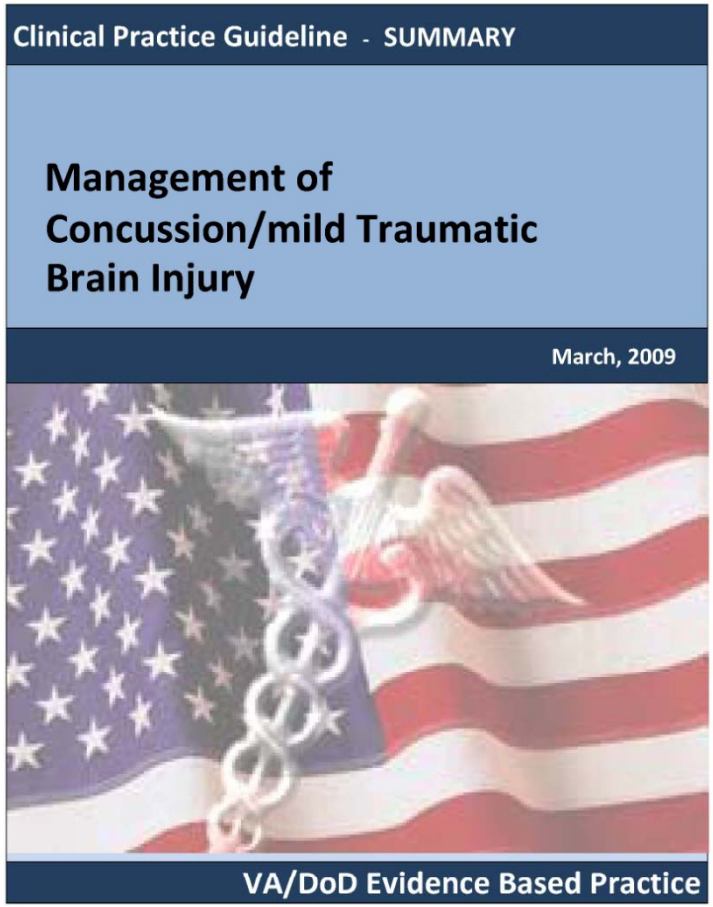
- Any period of observed or self-reported:
  - Transient confusion, disorientation, or impaired consciousness;
  - Dysfunction of memory around the time of injury;
  - Loss of consciousness lasting less than 30 minutes.
- Observed signs of neurological or neuropsychological dysfunction, such as:
  - Seizures acutely following injury to the head;
  - Among infants and very young children: irritability, lethargy, or vomiting following head injury;
  - Symptoms among older children and adults such as headache, dizziness, irritability, fatigue or poor concentration, when identified soon after injury, can be used to support the diagnosis of mild TBI, but cannot be used to make the diagnosis in the absence of loss of consciousness or altered consciousness. Research may provide additional guidance in this area.

WHO (2004)

Collaborating Task Force on Mild Traumatic Brain Injury. J Rehab Med (Suppl.43), 113-125

MTBI is an acute brain injury resulting from mechanical energy to the head from external physical forces. Operational criteria for clinical identification include: (i) 1 or more of the following: confusion or disorientation, loss of consciousness for 30 minutes or less, post-traumatic amnesia for less than 24 hours, and/or other transient neurological abnormalities such as focal signs, seizure, and intracranial lesion not requiring surgery; (ii) Glasgow Coma Scale score of 13–15 after 30 minutes post-injury or later upon presentation for healthcare. These manifestations of MTBI must not be due to drugs, alcohol, medications, caused by other injuries or treatment for other injuries (e.g. systemic injuries, facial injuries or intubation), caused by other problems (e.g. psychological trauma, language barrier or coexisting medical conditions) or caused by penetrating craniocerebral injury (p. 115).

VA/DoD (2009)



1.3 Diagnostic Criteria for mTBI

BACKGROUND

In the U.S., the most widely accepted criteria for mild TBI are those proposed by the American College of Rehabilitation Medicine (ACRM, 1993). They are "a physiological disruption of brain function as a result of a traumatic event as manifested by at least one of the following: alteration of mental state, loss of consciousness (LOC), loss of memory or focal neurological deficit, that may or may not be transient; but where the severity of the injury does not exceed the following: post-traumatic amnesia (PTA) for greater than 24 hours, after the first 30 minutes Glasgow Coma Score (GCS) 13 - 15, and loss of consciousness is less than 30 minutes." There are other criteria used by other medical groups. However, most agree that common criteria include GCS score of 13-15, brief LOC, brief PTA and negative head computed tomography (CT) scan.

RECOMMENDATIONS

1. A diagnosis of mTBI should be made when there is an injury to the head as a result of blunt trauma, acceleration or deceleration forces or exposure to blast that result in one or more of the following conditions:
    a. Any period of observed or self-reported:
        - Transient confusion, disorientation, or impaired consciousness
        - Dysfunction of memory immediately before or after the time of injury
        - Loss of consciousness (LOC) lasting less than 30 minutes.
    b. Observed signs of neurological or neuropsychological dysfunction, such as:
        - Headache, dizziness, irritability, fatigue or poor concentration, when identified soon after injury, can be used to support the diagnosis of mild TBI, but cannot be used to make the diagnosis in the absence of loss of consciousness or altered consciousness.
2. The severity of TBI must be defined by the acute injury characteristics and not by the severity of symptoms at random points after trauma.

SAC (1996)

McCrea, M; Kelly, J; Randolph C

FIGURE 1

Standard Assessment of Concussion-SAC

FORM A

Name: _____

Team: _____ Examiner: _____

Date of Exam: _____ Time: _____

Exam (Circle One): BLine Injury Post-Px/Game

Day 1	Day 2	Day 3	Day 5	Day 7	Day 90

Introduction:

I am going to ask you some questions. Please listen carefully and give your best effort.

Orientation:

What Month is it? _____	0	1
What's the Date Today? _____	0	1
What's the Day of the Week? _____	0	1
What Year is it? _____	0	1
What Time is it right now? (within 1 hr.) _____	0	1

Award 1 point for each correct answer.

ORIENTATION TOTAL SCORE

Immediate Memory:

I am going to test your memory. I will read you a list of words and when I am done, repeat back as many words as you can remember, in any order.

LIST	TRIAL 1		TRIAL 2		TRIAL 3	
Elbow	0	1	0	1	0	1
Apple	0	1	0	1	0	1
Carpet	0	1	0	1	0	1
Saddle	0	1	0	1	0	1
Bubble	0	1	0	1	0	1
TOTAL						

Trials 2 & 3: I am going to repeat that list again. Repeat back as many words as you can remember in any order, even if I said the word before.

Complete all 3 trials regardless of score on trial 1 & 2. Score 1 pt. for each correct response. Total score equals sum across all 3 trials. Do not inform the subject that delayed recall will be tested.

IMMEDIATE MEMORY TOTAL SCORE _____

Exertional Maneuvers:

If subject is not displaying or reporting symptoms, conduct the following maneuvers to create conditions under which symptoms are likely to be elicited and detected. **These measures need not be conducted if a subject is already displaying or reporting any symptoms.** If not conducted allow 2 minutes to keep time delay constant before testing Delayed Recall. These methods should be administered for baseline testing of normal subjects.

Exertional Maneuvers	
5 Jumping Jacks	5 Push-Ups
5 Sit ups	5 Knee Bends

Neurologic Screening:

Loss of Consciousness/ Witnessed Unresponsiveness	No Length:	Yes
Post-Traumatic Amnesia? Poor recall of events after injury	No Length:	Yes
Retrograde Amnesia? Poor recall of events before injury	No Length:	Yes

	Normal	Abnormal
Strength		
Right Upper Extremity		
Left Upper Extremity		
Right Lower Extremity		
Left Lower Extremity		
Sensation - examples:		
Finger-to-Nose/Romberg		
Coordination - examples:		
Tandem Walk/Finger-Nose-Finger		

Concentration:

Digits Backward: I am going to read you a string of numbers and when I am done, you repeat them back to me backwards, in reverse order of how I read them to you. For example, if I say 7-1-9, you would say 9-1-7.

If correct, go to next string length. If incorrect, read trial 2. Score 1 pt. for each string length. Stop after incorrect on both trials.

4-9-3	6-2-9	0	1
3-8-1-4	3-2-7-9	0	1
6-2-9-7-1	1-5-2-8-6	0	1
7-1-8-4-6-2	5-3-9-1-4-8	0	1

Months in Reverse Order: Now tell me the months of the year in reverse order. Start with the last month and go backward. So you'll say December, November...Go ahead.

1 pt. for entire sequence correct.

Dec-Nov-Oct-Sept-Aug-Jul-Jun-May-Apr-Mar-Feb-Jan 0 1

CONCENTRATION TOTAL SCORE

Delayed Recall:

Do you remember that list of words I read a few times earlier? Tell me as many words from the list as you can remember in any order. *Circle each word correctly recalled. Total score equals number of words recalled.*

Elbow Apple Carpet Saddle Bubble

DELAYED RECALL TOTAL SCORE

SAC Scoring Summary:

Exertional Maneuvers & Neurologic Screening are important for examination, but not incorporated into SAC Total Score.

Orientation	/5
Immediate Memory	/15
Concentration	/5
Delayed Recall	/5
SAC Total Score	**/30**

ACE (2006)

Gerard Gioia & Micky Collins

Acute Concussion Evaluation (ACE)
Physician/Clinician Office Version

Gerard Gioia, PhD[1] & Micky Collins, PhD[2]
[1]Children's National Medical Center
[2]University of Pittsburgh Medical Center

Patient Name:_____
DOB: _____ Age:_____
Date:_____ ID/MR#_____

A. Injury Characteristics Date/Time of Injury_____ Reporter: __Patient __Parent __Spouse __Other_____

1. Injury Description _____

1a. Is there evidence of a forcible blow to the head (direct or indirect)? __Yes __No __Unknown
1b. Is there evidence of intracranial injury or skull fracture? __Yes __No __Unknown
1c. Location of Impact: __Frontal __Lft Temporal __Rt Temporal __Lft Parietal __Rt Parietal __Occipital __Neck __Indirect Force
2. **Cause:** __MVC __Pedestrian-MVC __Fall __Assault __Sports (*specify*)_____Other_____
3. **Amnesia Before (Retrograde)** Are there any events just BEFORE the injury that you/ person has no memory of (even brief)? __Yes __No Duration
4. **Amnesia After (Anterograde)** Are there any events just AFTER the injury that you/ person has no memory of (even brief)? __Yes __No Duration
5. **Loss of Consciousness:** Did you/ person lose consciousness? __Yes __No Duration
6. **EARLY SIGNS:** __Appears dazed or stunned __Is confused about events __Answers questions slowly __Repeats Questions __Forgetful (recent info)
7. **Seizures:** Were seizures observed? No__ Yes___ Detail_____

B. Symptom Check List* Since the injury, has the person experienced any of these symptoms any more than usual today or in the past day?
Indicate presence of each symptom (0=No, 1=Yes). ***Lovell & Collins, 1998 JHTR***

PHYSICAL (10)		COGNITIVE (4)		SLEEP (4)	
Headache	0 1	Feeling mentally foggy	0 1	Drowsiness	0 1
Nausea	0 1	Feeling slowed down	0 1	Sleeping less than usual	0 1 N/A
Vomiting	0 1	Difficulty concentrating	0 1	Sleeping more than usual	0 1 N/A
Balance problems	0 1	Difficulty remembering	0 1	Trouble falling asleep	0 1 N/A
Dizziness	0 1	**COGNITIVE Total (0-4)** _____		**SLEEP Total (0-4)** _____	
Visual problems	0 1	**EMOTIONAL (4)**			
Fatigue	0 1	Irritability	0 1		
Sensitivity to light	0 1	Sadness	0 1		
Sensitivity to noise	0 1	More emotional	0 1		
Numbness/Tingling	0 1	Nervousness	0 1		
PHYSICAL Total (0-10) _____		**EMOTIONAL Total (0-4)** _____			
(Add Physical, Cognitive, Emotion, Sleep totals) Total Symptom Score (0-22)			_____		

Exertion: Do these symptoms worsen with:
Physical Activity __Yes __No __N/A
Cognitive Activity __Yes __No __N/A

Overall Rating: How different is the person acting compared to his/her usual self? (circle)
Normal 0 1 2 3 4 5 6 Very Different

C. Risk Factors for Protracted Recovery *(check all that apply)*

Concussion History? Y___ N___	√	Headache History? Y___ N___	√	Developmental History	√	Psychiatric History
Previous # 1 2 3 4 5 6+		Prior treatment for headache		Learning disabilities		Anxiety
Longest symptom duration Days__ Weeks__ Months__ Years__		History of migraine headache __ Personal __ Family_____		Attention-Deficit/ Hyperactivity Disorder		Depression
						Sleep disorder
If multiple concussions, less force caused reinjury? Yes__ No__				Other developmental disorder_____		Other psychiatric disorder _____

List other comorbid medical disorders or medication usage (e.g., hypothyroid, seizures)_____

D. RED FLAGS for acute emergency management: Refer to the emergency department with sudden onset of any of the following:

* Headaches that worsen	* Looks very drowsy/ can't be awakened	* Can't recognize people or places	* Neck pain
* Seizures	* Repeated vomiting	* Increasing confusion or irritability	* Unusual behavioral change
* Focal neurologic signs	* Slurred speech	* Weakness or numbness in arms/legs	* Change in state of consciousness

E. Diagnosis (ICD): __Concussion w/o LOC 850.0 __Concussion w/ LOC 850.1 __Concussion (Unspecified) 850.9 __Other (854) _____
__No diagnosis

F. Follow-Up Action Plan Complete ***ACE Care Plan*** and provide copy to patient/family.
___ **No Follow-Up Needed**
___ **Physician/Clinician Office Monitoring**: Date of next follow-up _____
___ **Referral:**
___ Neuropsychological Testing
___ Physician: Neurosurgery____ Neurology____ Sports Medicine____ Physiatrist____ Psychiatrist____ Other_____
___ Emergency Department

ACE Completed by:_____ MD RN NP PhD ATC

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This form is part of the "Heads Up: Brain Injury in Your Practice" tool kit developed by the Centers for Disease Control and Prevention (CDC).

MACE

(7/2007)

Military Acute Concussion Evaluation (MACE)

Defense and Veterans Brain Injury Center

Patient Name: _____

SS#: _____-_____-_____ **Unit:** _____

Date of Injury: ____/____/____ **Time of Injury:** _____

Examiner: _____

Date of Evaluation: ____/____/____ **Time of Evaluation:** ____

History: (I – VIII)

I. **Description of Incident**
Ask:
a) What happened?
b) Tell me what you remember.
c) Were you dazed, confused, "saw stars"? ❒ Yes ❒ No
d) Did you hit your head? ❒ Yes ❒ No

II. **Cause of Injury** (Circle all that apply):
1) Explosion/Blast 4) Fragment
2) Blunt object 5) Fall
3) Motor Vehicle Crash 6) Gunshot wound
7) Other _____

III. **Was a helmet worn?** ❒ Yes ❒ No Type _____

IV. **Amnesia Before: Are there any events just BEFORE the injury that are not remembered? (Assess for continuous memory prior to injury)**
❒ Yes ❒ No If yes, how long _____

V. **Amnesia After: Are there any events just AFTER the injuries that are not remembered? (Assess time until continuous memory after the injury)**
❒ Yes ❒ No If yes, how long _____

VI. **Does the individual report loss of consciousness or "blacking out"?** ❒ Yes ❒ No If yes, how long _____

VII. **Did anyone observe a period of loss of consciousness or unresponsiveness?** ❒ Yes ❒ No If yes, how long _____

VIII. **Symptoms** (circle all that apply)
1) Headache 2) Dizziness
3) Memory Problems 4) Balance problems
5) Nausea/Vomiting 6) Difficulty Concentrating
7) Irritability 8) Visual Disturbances
9) Ringing in the ears 10) Other _____

Examination: (IX – XIII)

Evaluate each domain. Total possible score is 30.

IX. **Orientation:** (1 point each)

Month:	0	1
Date:	0	1
Day of Week:	0	1
Year:	0	1
Time:	0	1

Orientation Total Score _____/5

X. **Immediate Memory:**
Read all 5 words and ask the patient to recall them in any order. Repeat two more times for a total of three trials. (1 point for each correct, total over 3 trials)

List	Trial 1	Trial 2	Trial 3
Elbow	0 1	0 1	0 1
Apple	0 1	0 1	0 1
Carpet	0 1	0 1	0 1
Saddle	0 1	0 1	0 1
Bubble	0 1	0 1	0 1
Trial Score			

Immediate Memory Total Score _____/15

XI. **Neurological Screening**
As the clinical condition permits, check
Eyes: pupillary response and tracking
Verbal: speech fluency and word finding
Motor: pronator drift, gait/coordination
Record any abnormalities. **No points are given for this.**

XII. Concentration

Reverse Digits: (go to next string length if correct on first trial. Stop if incorrect on both trials.) 1 pt. for each string length.

4-9-3	6-2-9	0	1
3-8-1-4	3-2-7-9	0	1
6-2-9-7-1	1-5-2-8-5	0	1
7-1-8-4-6-2	5-3-9-1-4-8	0	1

Months in reverse order: (1 pt. for entire sequence correct)
Dec-Nov-Oct-Sep-Aug-Jul-Jun-May-Apr-Mar-Feb-Jan
0 1

Concentration Total Score ____/5

XIII. Delayed Recall (1 pt. each)

Ask the patient to recall the 5 words from the earlier memory test (Do NOT reread the word list.)

Elbow	0	1
Apple	0	1
Carpet	0	1
Saddle	0	1
Bubble	0	1

Delayed Recall Total Score ____/5
TOTAL SCORE _____/30

Notes: _____

_____

Diagnosis: (circle one or write in diagnoses)

No concussion
850.0 Concussion without Loss of Consciousness (LOC)
850.1 Concussion with Loss of Consciousness (LOC)

Other diagnoses _____

_____

Defense & Veterans Brain Injury Center
1-800-870-9244 or DSN: 662-6345

Instruction Sheet

Purpose and Use of the MACE

A concussion is a mild traumatic brain injury (TBI). The purpose of the MACE is to evaluate a person in whom a concussion is suspected. The MACE is used to confirm the diagnosis and assess the current clinical status.

Tool Development

The MACE has been extensively reviewed by leading civilian and military experts in the field of concussion assessment and management. While the MACE is not, yet, a validated tool, the examination section is derived from the *Standardized Assessment of Concussion* (SAC) (McCrea, M., Kelly, J. & Randolph, C. (2000). *Standardized Assessment of Concussion (SAC): Manual for Administration, Scoring, and Interpretation.* (2nd ed.) Waukesa,WI: Authors.) which is a validated, widely used tool in sports medicine. Abnormalities on the SAC correlate with formal comprehensive neuropsychological testing during the first 48 hours following a concussion.

Who to Evaluate

Any one who was dazed, confused, "saw stars" or lost consciousness, even momentarily, as a result of an explosion/blast, fall, motor vehicle crash, or other event involving abrupt head movement, a direct blow to the head, or other head injury is an appropriate person for evaluation using the MACE.

Evaluation of Concussion

History: (I – VIII)

I. Ask for a description of the incident that resulted in the injury; how the injury occurred, type of force. Ask questions A – D.

II. Indicate the cause of injury

III. Assess for helmet use. Military: Kevlar or ACH (Advanced Combat Helmet). Sports helmet, motorcycle helmet, etc.

IV – V Determine whether and length of time that the person wasn't registering continuous memory both **prior** to injury and **after** the injury. Approximate the amount of time in seconds, minutes or hours, whichever time increment is most appropriate. For example, if the assessment of the patient yields a possible time of 20 minutes, then 20 minutes should be documented in the "how long?" section.

VI – VII Determine whether and length of time of **self reported** loss of consciousness (LOC) or **witnessed/observed** LOC. Again, approximate the amount of time in second, minutes or hours, whichever time increment is most appropriate.

VIII Ask the person to report their experience of each specific symptom since injury.

Examination: (IX – XIII)

Standardized Assessment of Concussion (SAC):

Total possible score = 30
Orientation = 5
Immediate Memory = 15
Concentration = 5
Memory Recall= 5

IX Orientation: Assess patients awareness of the accurate time
Ask: WHAT MONTH IS THIS?
WHAT IS THE DATE OR DAY OF THE MONTH?
WHAT DAY OF THE WEEK IS IT?
WHAT YEAR IS IT?
WHAT TIME DO YOU THINK IT IS?

One point for each correct response for a total of 5 possible points. It should be noted that a correct response on time of day must be within 1 hour of the actual time.

X Immediate memory is assessed using a brief repeated list learning test. Read the patient the list of 5 words once and then ask them to repeat it back to you, as many as they can recall in any order. Repeat this procedure 2 more times for a total of 3 trials, even if the patient scores perfectly on the first trial.
Trial 1: I'M GOING TO TEST YOUR MEMORY, I WILL READ YOU A LIST OF WORDS AND WHEN I AM DONE, REPEAT BACK AS MANY WORDS AS YOU CAN REMEMBER, IN ANY ORDER.
Trial 2 &3: I AM GOING TO REPEAT THAT LIST AGAIN. AGAIN, REPEAT BACK AS MANY AS YOU CAN REMEMBER IN ANY ORDER, EVEN IF YOU SAID THEM BEFORE.
One point is given for each correct answer for a total of 15 possible points.

XI Neurological screening
Eyes; check pupil size and reactivity.
Verbal: notice speech fluency and word finding
Motor: pronator drift- ask patient to lift arms with palms up, ask patient to then close their eyes, assess for either arm to "drift" down. Assess gait and coordination if possible. Document any abnormalities.
No points are given for this section.

XII Concentration: Inform the patient:
I'M GOING TO READ YOU A STRING OF NUMBERS AND WHEN I AM FINISHED, REPEAT THEM BACK TO ME BACKWARDS, THAT IS, IN REVERSE ORDER OF HOW I READ THEM TO YOU. FOR EXAMPLE, IF I SAY 7-1-9, YOU WOULD SAY 9-1-7.
If the patient is correct on the first trial of each string length, proceed to the next string length. If incorrect, administer the 2nd trial of the same string length. Proceed to the next string length if correct on the second trial. Discontinue after failure on both trials of the same string length. Total of 4 different string lengths; **1** point for each string length for a total of **4** points.
NOW TELL ME THE MONTHS IN REVERSE ORDER, THAT IS, START WITH DECEMBER AND END IN JANUARY.
1 point if able to recite ALL months in reverse order.
0 points if not able to recite ALL of them in reverse order.
Total possible score for concentration portion: **5.**

XIII Delayed Recall
Assess the patient's ability to retain previously learned information by asking he/she to recall as many words as possible from the initial word list, without having the word list read again for this trial.
DO YOU REMEMBER THAT LIST OF WORDS I READ A FEW MINUTES EARLIER? I WANT YOU TO TELL ME AS MANY WORDS FROM THE LIST AS YOU CAN REMEMBER IN ANY ORDER.
One point for each word remembered for a total of 5 possible points.
Total score= Add up from the 4 assessed domains: immediate memory, orientation, concentration and memory recall.

Significance of Scoring

In studies of non-concussed patients, the mean total score was 28. Therefore, a score less than 30 does not imply that a concussion has occurred. Definitive normative data for a "cut-off" score are not available. However, scores below 25 may represent clinically relevant neurocognitive impairment and require further evaluation for the possibility of a more serious brain injury. The scoring system also takes on particular clinical significance during serial assessment where it can be used to document either a decline or an improvement in cognitive functioning.

Diagnosis

Circle the ICD-9 code that corresponds to the evaluation. If loss of consciousness was present, then circle 850.1. If no LOC, then document 850.0. If another diagnosis is made, write it in.

MACE Form B

Due to test- retest issues (e.g. service members memorizing words and numbers) validated, alternative versions B or C should be used.

Immediate Memory
Read all 5 words and ask the patient to recall them in any order. Repeat two more times for a total of three trials. (1 point for each correct, total over 3 trials.)

List	Trial 1	Trial 2	Trial 3
Candle	0 1	0 1	0 1
Paper	0 1	0 1	0 1
Sugar	0 1	0 1	0 1
Sandwich	0 1	0 1	0 1
Wagon	0 1	0 1	0 1
Total			

Concentration
Reverse Digits: (go to next string length if correct on first trial. Stop if incorrect on both trails.) 1 pt. for each string length.

5-2-6	4-1-5	0	1
1-7-9-5	4-9-6-8	0	1
4-8-5-2-7	6-1-8-4-3	0	1
8-3-1-9-6-4	7-2-4-8-5-6	0	1

Delayed Recall (1 pt each)
Ask the patient to recall the 5 words from the earlier memory test (DO NOT reread the word list.)

Candle	0 1
Paper	0 1
Sugar	0 1
Sandwich	0 1
Wagon	0 1

MACE Form C

Due to test- retest issues (e.g. service members memorizing words and numbers) validated, alternative versions B or C should be used.

Immediate Memory
Read all 5 words and ask the patient to recall them in any order. Repeat two more times for a total of three trials. (1 point for each correct, total over 3 trials.)

List	Trial 1	Trial 2	Trial 3
Baby	0 1	0 1	0 1
Monkey	0 1	0 1	0 1
Perfume	0 1	0 1	0 1
Sunset	0 1	0 1	0 1
Iron	0 1	0 1	0 1
Total			

Concentration
Reverse Digits: (go to next string length if correct on first trial. Stop if incorrect on both trails.) 1 pt. for each string length.

1-4-2	6-5-8	0	1
6-8-3-1	3-4-8-1	0	1
4-9-1-5-3	6-8-2-5-1	0	1
3-7-6-5-1-9	9-2-6-5-1-4	0	1

Delayed Recall (1 pt each)
Ask the patient to recall the 5 words from the earlier memory test (DO NOT reread the word list.)

Baby	0 1
Monkey	0 1
Perfume	0 1
Sunset	0 1
Iron	0 1

ER Diagnosis of MTBI

56% meeting criteria not diagnosed

65% with LOC were diagnosed

28% with confusion were diagnosed

ORIGINAL ARTICLE

Accuracy of Mild Traumatic Brain Injury Diagnosis

Janet M. Powell, PhD, OT, Joseph V. Ferraro, MD, Sureyya S. Dikmen, PhD, Nancy R. Temkin, PhD, Kathleen R. Bell, MD

ABSTRACT. Powell JM, Ferraro JV, Dikmen SS, Temkin NR, Bell KR. Accuracy of mild traumatic brain injury diagnosis. Arch Phys Med Rehabil 2008;89:1550-5.

Objective: To determine how often emergency department (ED) patients meeting the Centers for Disease Control and Prevention (CDC) mild traumatic brain injury (TBI) criteria were diagnosed with a mild TBI by the ED physician.

Design: Prospective identification of cases of mild TBI in the ED by study personnel using scripted interviews and medical record data was compared with retrospective review of ED medical record documentation of mild TBI.

Setting: EDs of a level I trauma center and an academic nontrauma hospital.

Participants: Prospective cohort of subjects (N=197; mean age, 32.6y; 70% men) with arrival at the ED within 48 hours of injury, Glasgow Coma Scale score of 13 to 15, and injury circumstances, alteration of consciousness, and memory dysfunction consistent with the CDC mild TBI definition.

Interventions: Not applicable.

Main Outcome Measure: ED medical record documentation of mild TBI.

Results: Fifty-six percent of mild TBI cases identified by study personnel did not have a documented mild TBI-related diagnosis in the ED record. The greatest agreement between study personnel and ED physicians for positive mild TBI-related findings was for loss of consciousness (72% vs 65%) with the greatest discrepancy for confusion (94% vs 28%).

Conclusions: The diagnosis of mild TBI was frequently absent from ED medical records despite patients reporting findings consistent with a mild TBI diagnosis when interviewed by study personnel. Asking a few targeted questions of ED patients with likely mechanisms of injury that could result in mild TBI could begin to improve diagnosis and, in turn, begin to improve patient management and the accuracy of estimates of mild TBI incidence.

Key Words: Brain concussion; Craniocerebral trauma; Head injuries, closed; Post-concussive syndrome; Rehabilitation.

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TRAUMATIC BRAIN INJURY is a significant public health problem in the United States. As many as 1.4 million people sustain a TBI each year[1] with the lifetime total costs associated with these injuries estimated at $60 billion.[2] Of all TBIs, approximately 75% to 80% are considered mild.[3] Although the majority of persons with a mild brain injury recover from within days to up to 3 months, others experience long-term impairments that affect their ability to resume their prior social roles and responsibilities.[3-11]

Mild TBI is often treated in the ED with as many as 80% of all adults with a TBI discharged home the same day without being admitted to the hospital.[1] Accurate identification and diagnosis of a mild TBI is the first step toward providing appropriate clinical care. However, accurate clinical identification of patients with mild TBI is complicated with variation in the criteria used for diagnosis[3,12-16] and in diagnostic terminology (ie, concussion, minor head injury, minor brain injury, minor head trauma, and minor TBI all used to refer to a mild TBI).[12,17-19]

Accurate diagnosis and written documentation of the diagnosis of mild TBI in the ED is also important for epidemiologic studies. The CDC mild TBI work group[3] and others[20] have recommended that ED and hospital-based data be given the first priority for surveillance studies (ie, those that provide incidence and prevalence estimates). These studies typically identify cases of mild TBI based on ICD-9-CM diagnostic codes[21] found in administrative datasets.[3] Because the ICD-9-CM codes are based, in part, on the clinical diagnosis, clear documentation of a mild TBI diagnosis in the ED is critical for subsequent identification of cases.[22]

Even though accurate diagnosis of mild TBI is important for appropriate care and accurate incidence estimates, there is limited evidence as to whether persons with mild TBI are accurately diagnosed and identified in ED medical records.[12] As a step toward quantifying the accuracy of mild TBI diagnosis in medical records, this study investigated how often ED patients identified as meeting the CDC mild TBI work group[3] mild TBI criteria by study personnel using scripted interviews

From the Departments of Rehabilitation Medicine (Powell, Dikmen, Temkin, Bell), Neurological Surgery (Dikmen, Temkin), Psychiatry and Behavioral Sciences (Dikmen), and Biostatistics (Temkin), University of Washington, Seattle, WA; and North Florida/South Georgia Veterans Health Care System, Gainesville, FL (Ferraro).

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List of Abbreviations

CDC	Centers for Disease Control and Prevention
CHI	closed head injury
CT	computed tomography
ED	emergency department
GCS	Glasgow Coma Scale
ICD-9-CM	*International Statistical Classification of Diseases, 9th Revision, Clinical Modification*
LOC	loss of consciousness
PTA	posttraumatic amnesia
TBI	traumatic brain injury

NAN (2009)

- LOC
- Retrograde amnesia & PTA
- Confusion & disorientation
- Neuro signs & symptoms

Recommendations for Diagnosing a Mild Traumatic Brain Injury: A National Academy of Neuropsychology Education Paper

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Accepted November 2008

Abstract

A special interest group of the American Congress of Rehabilitation Medicine [ACRM; Mild Traumatic Brain Injury Committee. (1993). Definition of mild traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 8 (3), 86–87.] was the first organized interdisciplinary group to advocate four specific criteria for the diagnosis of a mild traumatic brain injury (TBI). More recently, the World Health Organization (WHO) Collaborative Center Task Force on Mild Traumatic Brain Injury [Carroll, L. J., Cassidy, J. D., Holm, L., Kraus, J., & Coronado, V. G. (2004). Methodological issues and research recommendations for mild traumatic brain injury: the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. *Journal of Rehabilitation Medicine*, (Suppl. 43), 113–125.] conducted a comprehensive review of the definitions utilized in evidence-based studies with mild TBI patients. Based on this review, the WHO task force maintained the same four criteria but offered two modifications. The similarities and differences between these two definitions are discussed. The authors of the ACRM and the WHO definitions do not provide guidelines or specific recommendations for diagnosing the four criteria. Thus, we provide recommendations for assessing loss of consciousness, retrograde and post-traumatic amnesia, disorientation and confusion as well as clarification of the neurologic signs that can be indicative of a diagnosis of mild TBI. Finally, confounding factors mentioned in both definitions that should exclude a mild TBI diagnosis are summarized.

Keywords: Mild traumatic brain injury; Neuropsychology; Treatment; Assessment; Head injury

TBI Assessment : Biochemical markers

Herrmann M, Curio N, Jost S, et al: Release of biochemical markers of damage to neuronal and glial brain tissue is associated with short and long term neuropsychological outcome after traumatic brain injury. J Neurol Neurosurg Psychiatry. 1:70, 95-1000, 2001

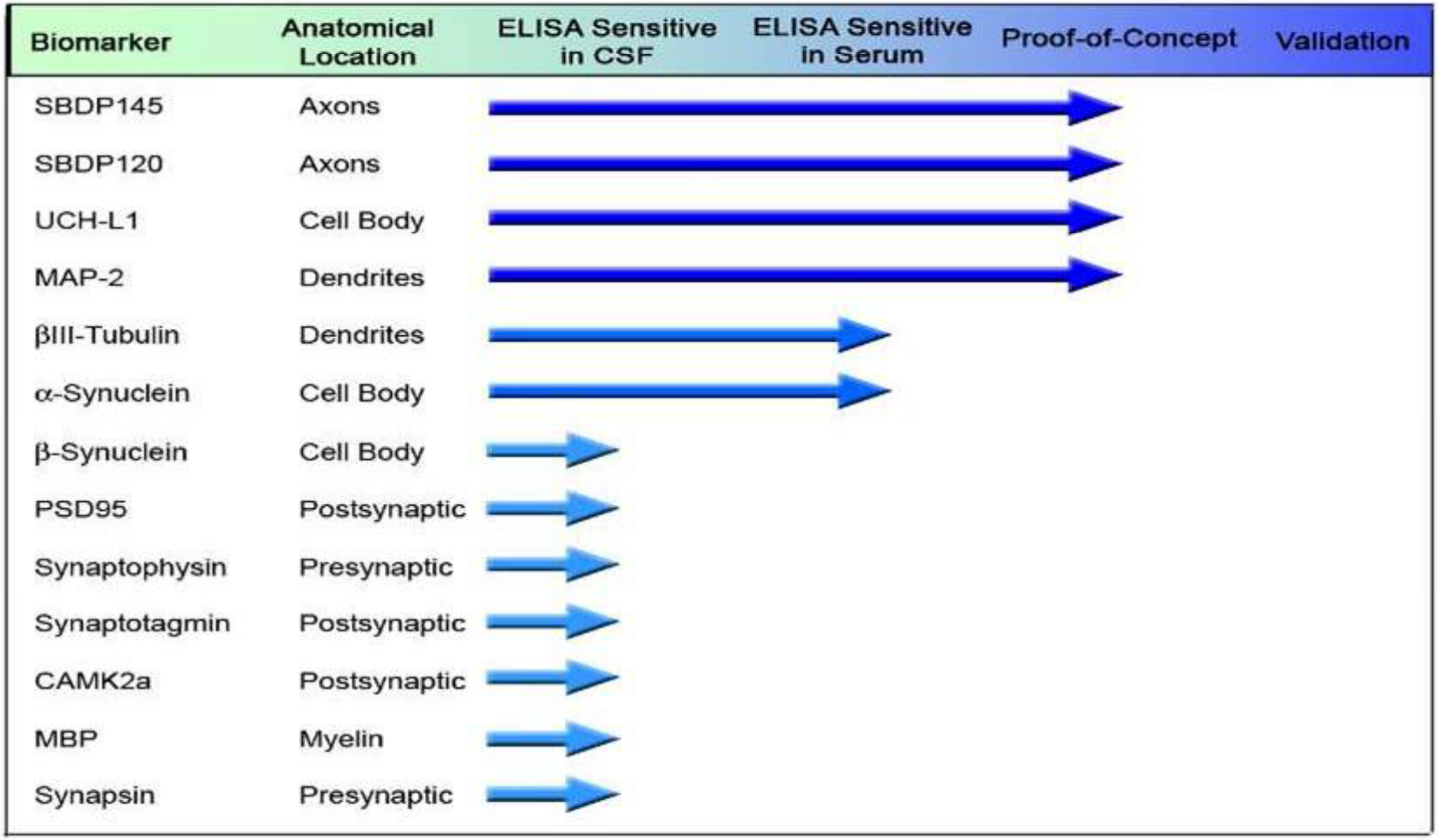
Prospective study of 69 patients measuring blood levels of neuron specific enolase (NSE - formed in neuronal cytoplasm) and protein S-100B (astroglia and Schwann cells) acutely (D1-3)

Neuropsychological evaluation at 2 weeks (39 with GCS 13-15) and 6 months (29 with GCS 13 -15)

TBI Assessment : Biochemical markers

- 74% examined at 2 weeks showed neuropsych abnormalities
  - attentional disorders (88%)
  - executive deficits (46 -74%)
  - memory deficits (30%)
- 69% still displayed deficits at 6 months
  - same profile except memory deficits in 50%
- 48% at 6 months in competitive, transitional or supportive employment
- Median FIM score of 126 (114 -126) - independent in ADLs
- Post-traumatic serum concentrations of NSE and protein S - 100B were highly increased
- Significantly longer lasting and higher S - 100B release in those with abnormalities on 2 week and 6 month evaluation

BRAIN INJURY BIOMARKERS



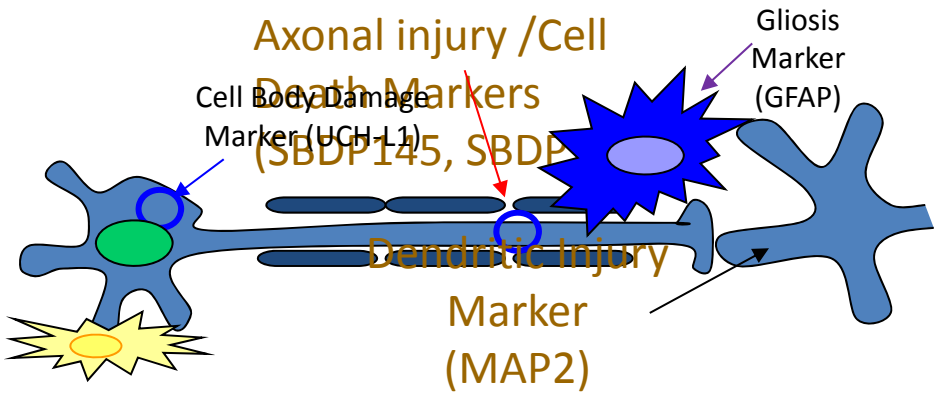
BANDITS PROGRAM

4. Head Injury Assessment

Biomarker Assessment for Neurotrauma Diagnosis & Improved Triage System (BANDITS)

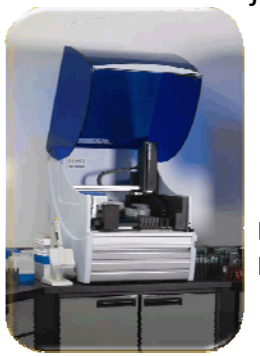
No test is currently approved to objectively diagnose TBI, particularly mTBI. The goal of the BANDITS program is to develop a blood test for brain cell damage, much like the current blood test (Troponin) for heart damage.

BANDITS will be embedded in an automated system available to Level III or lower echelons of care from a open benchtop system to a handheld device.



GOALS

- Minimal- to non-invasive medical device to identify and assess internal brain injuries
- State-of-the-art lightweight, sturdy and reliable diagnostic systems appropriate for far forward screening, assessment, and care
- Designed to diagnose mild, moderate and severe traumatic brain injury



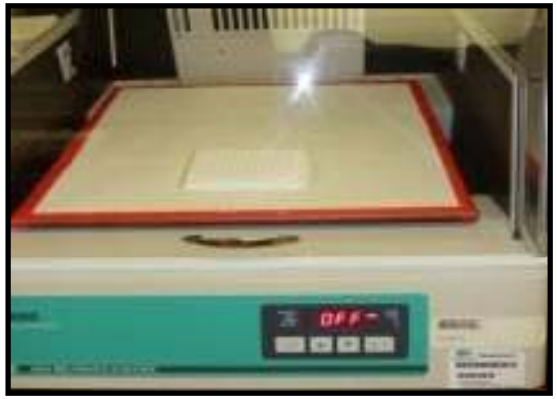
Benchtop System
MedCen/CSH

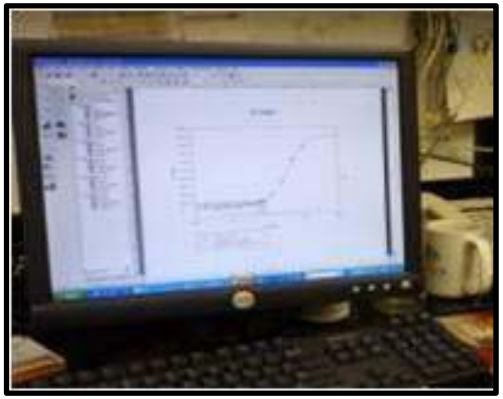
POC System
CSH/FST

Handheld Device
Combat Medic

CURRENT STATUS OF BRAIN BIOMARKERS

- 1st Generation ELISA Assays
  - 2 day assay time
  - All manual steps
  - Complex assay formats
  - Multiple pieces of equipment
  - Not suited for military environment





FUTURE STEPS FOR BRAIN BIOMARKERS

Role 4

Role 3 / Role 2

Role 1

Increment I: Benchtop System

Advantages
- Accuracy
- Sensitivity
- Precision
- Throughput
- FDA approval
- Flexible platform

Disadvantages
- Size & weight
- Need for sample preparation
- Power and space requirements
- Cost of instrument and assay
- Separate and multiple reagent storage

Increment II: POC System

Advantages
- Smaller size/ weight
- No sample prep
- Lower cost
- Assay speed

Disadvantages
- Potentially lower sensitivity, accuracy, and/or precision
- No feasibility data
- Time to FDA clearance

Increment III: Handheld Device

Advantages
- Small size/ weight
- Assay speed
- Low cost
- Stable reagents
- Finger-prick sample

Disadvantages
- Lower sensitivity, accuracy, and/or precision
- Time to FDA clearance
- CLIP/CLIA issues for approval

Increasing developmental cost and time

CONCLUSIONS

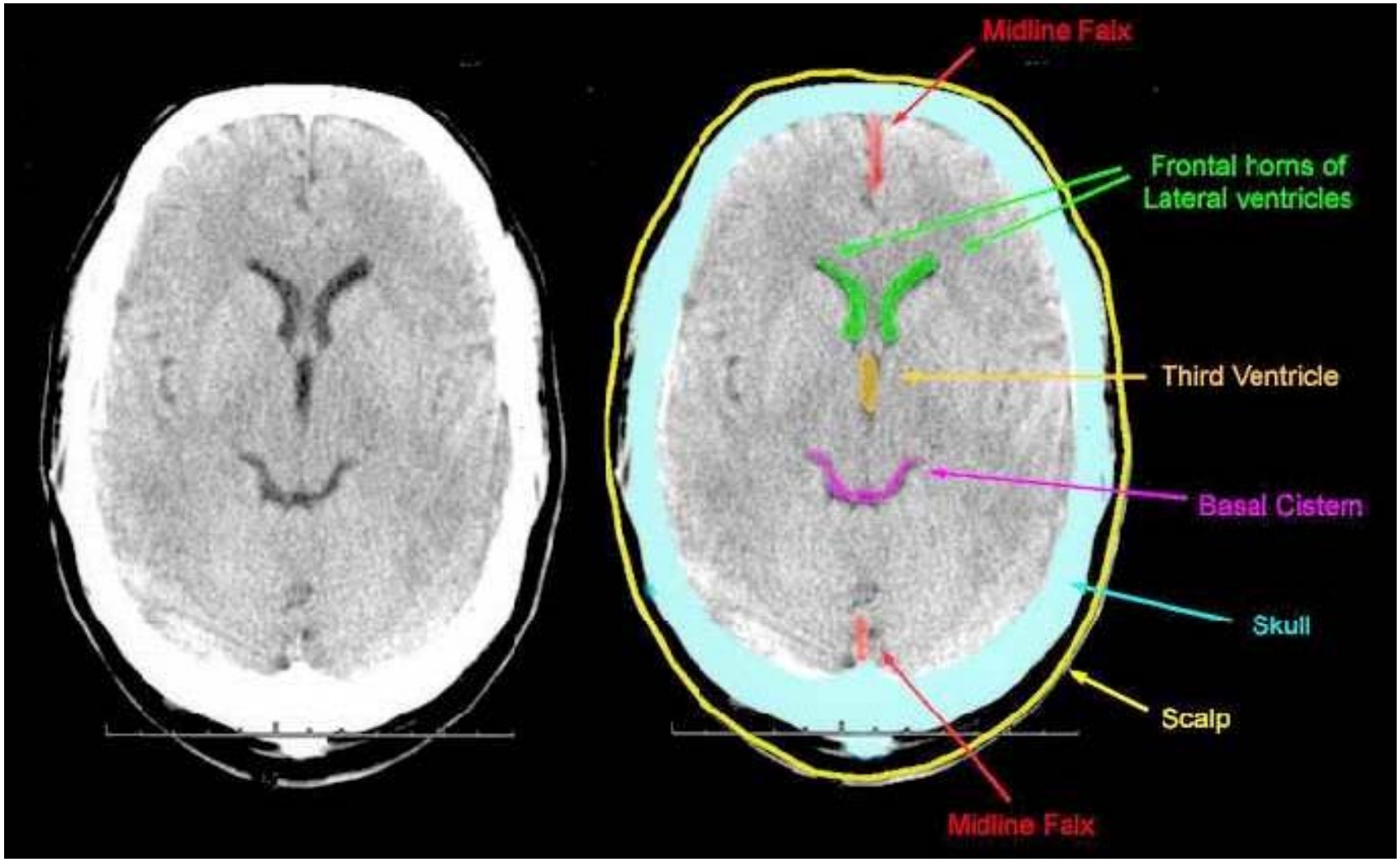
- From Preclinical discovery through clinical validation, the BANDITS program has
  - Identified protein biomarkers of TBI
  - Developed serum assays for lead biomarkers now proven to be specific and sensitive for TBI in over 250 severe TBI patients.
- The BANDITS biomarker platform has been able to objectively distinguish between CT-positiveTBI, CT-negative TBI , and controls in over 94 mild TBI patients
  - A 300 patient mild-moderate feasibility study is underway
- A bench top Increment I device platform with FDA approval should be fieldable for use at a Role 3/4 medical facility within 3 years, with Increments II and III for use further forward to follow

NEUROIMAGING

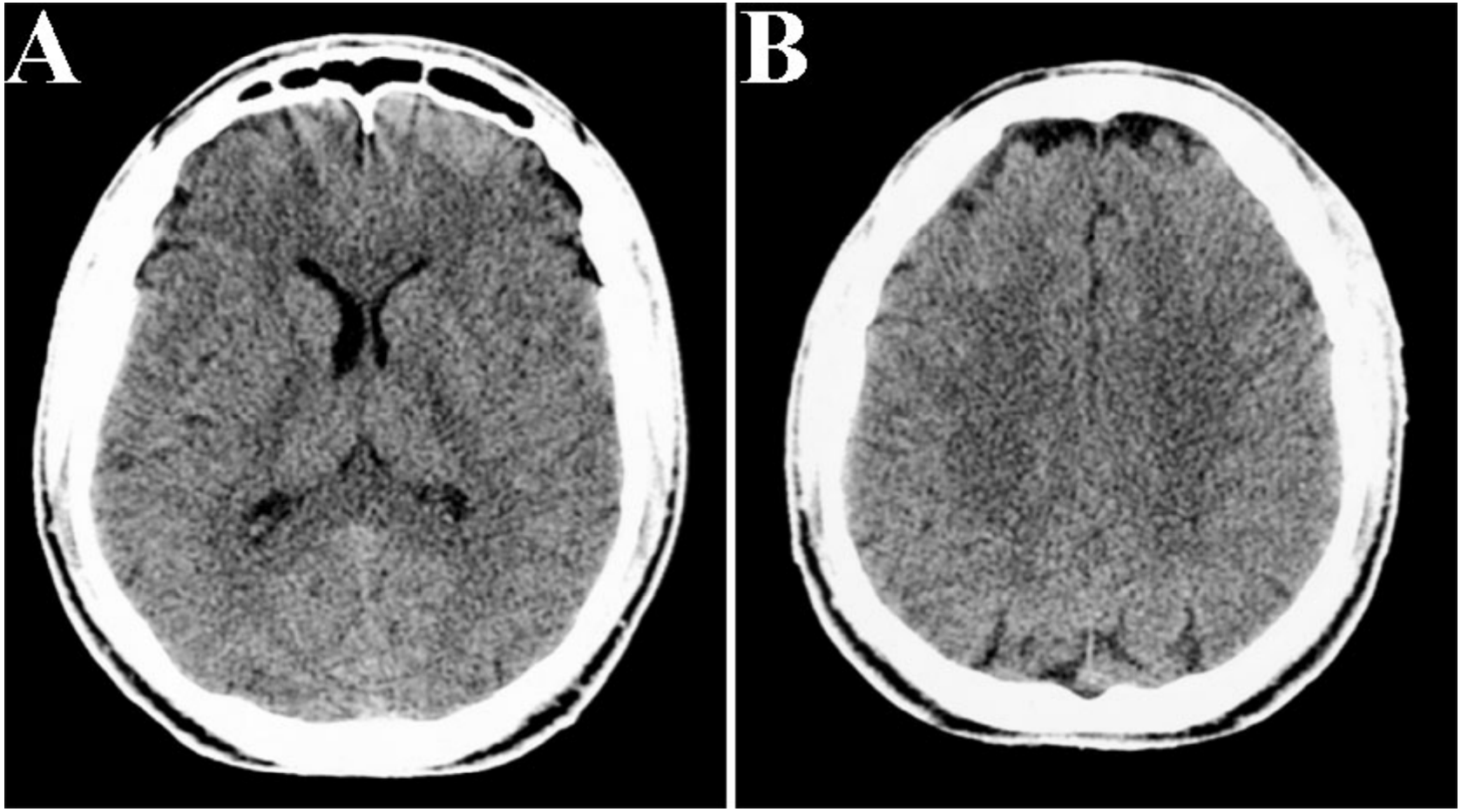
Trephanation

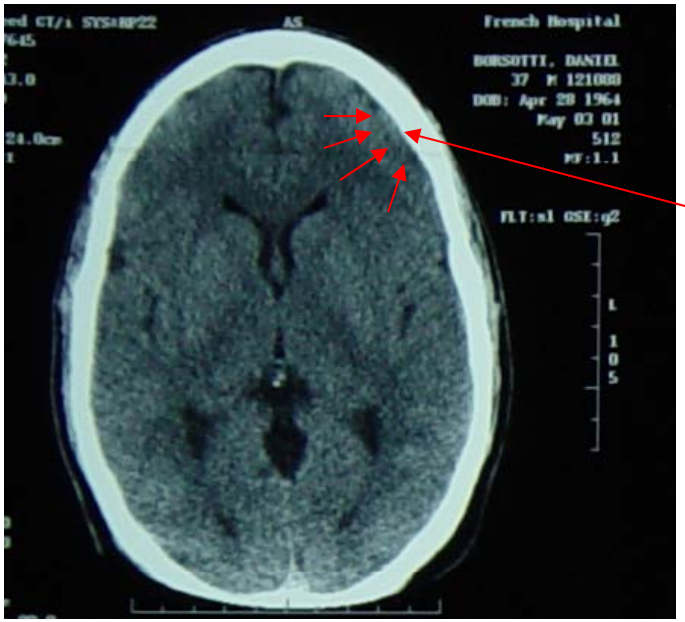
Skull X-rays

Computerized Tomography



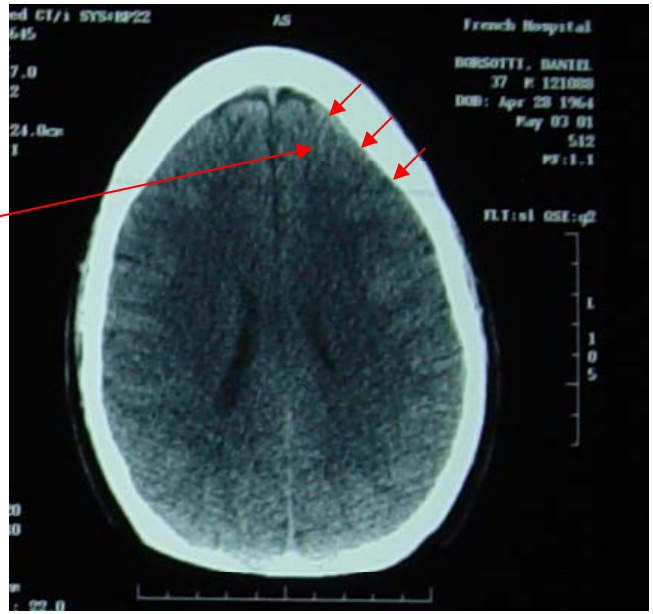
CT - MTBI





CT – 5/3/2001

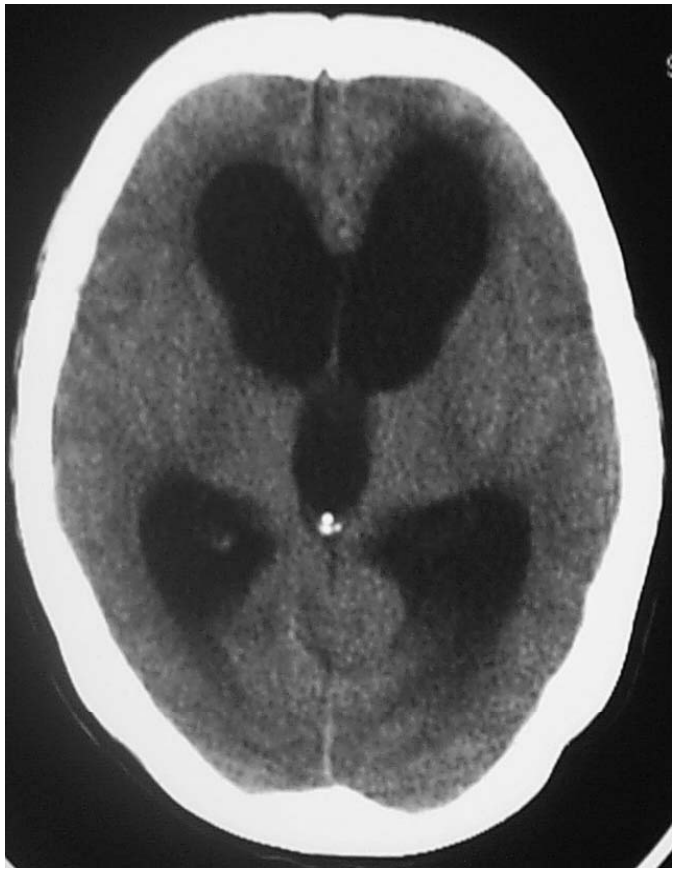
Acute Subdural Hematoma



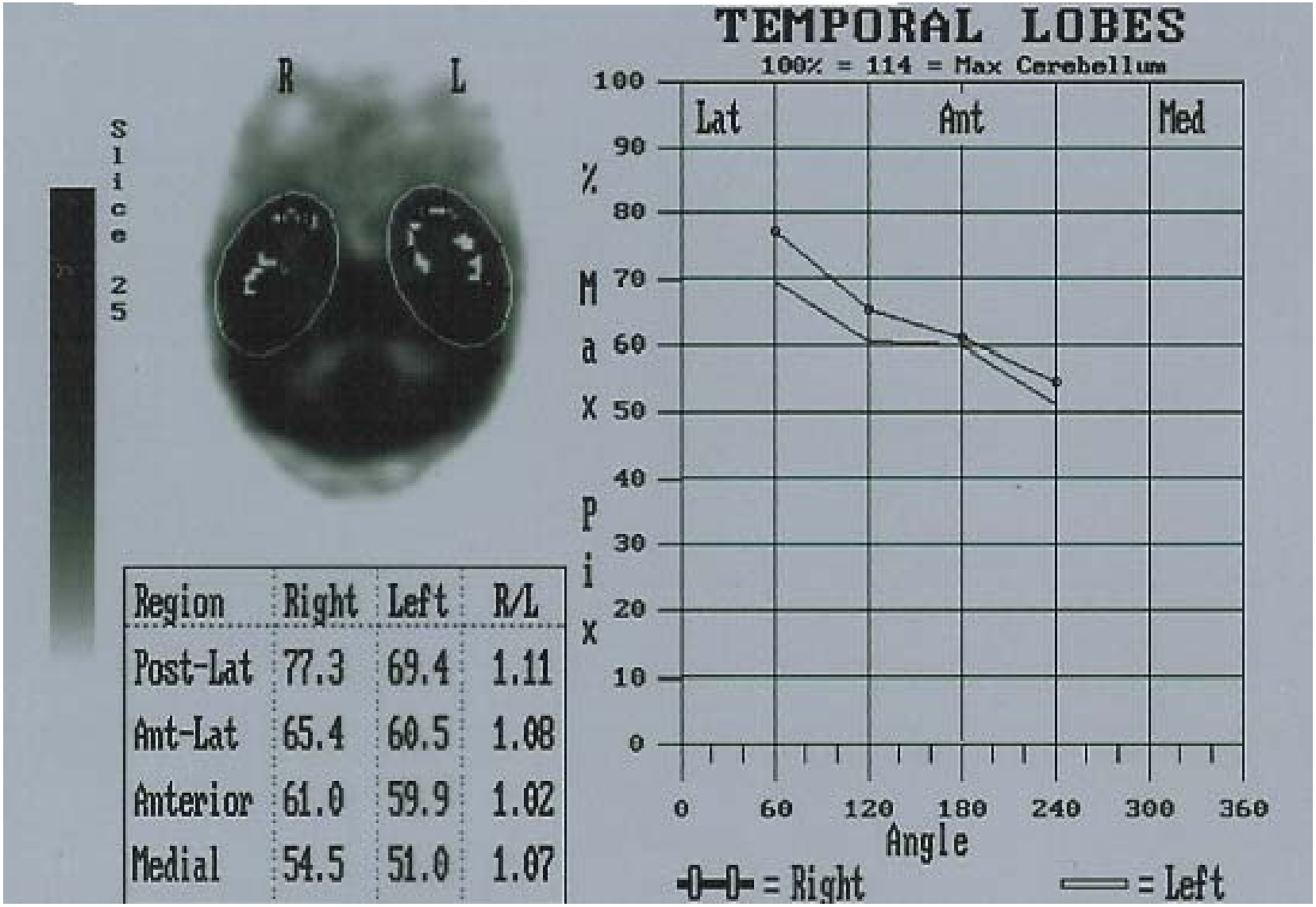
MRI – 2/1/2002

Chronic Subdural Hygromas/Hematoma

Computerized Tomography

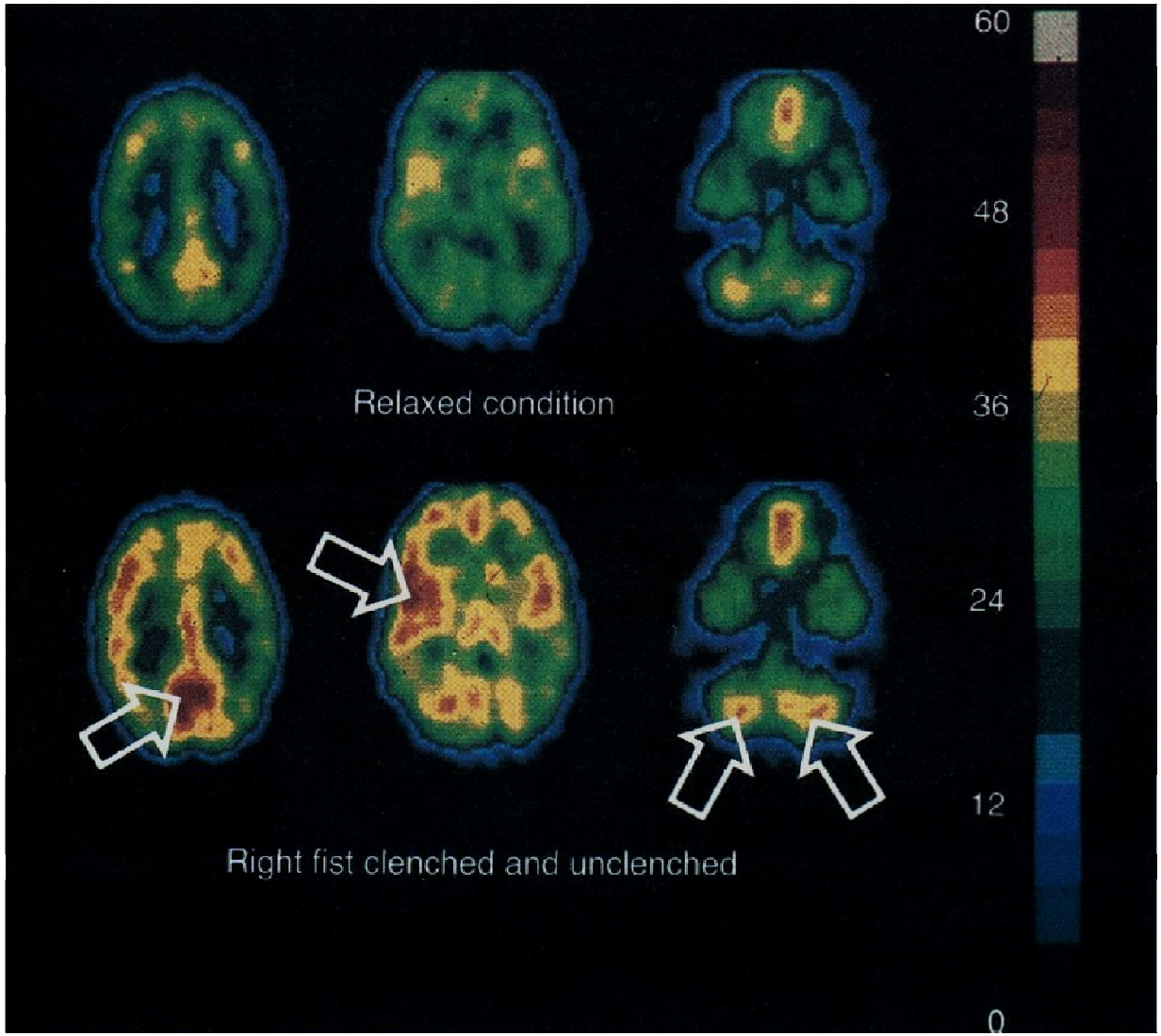


Triple Headed SPECT Scan

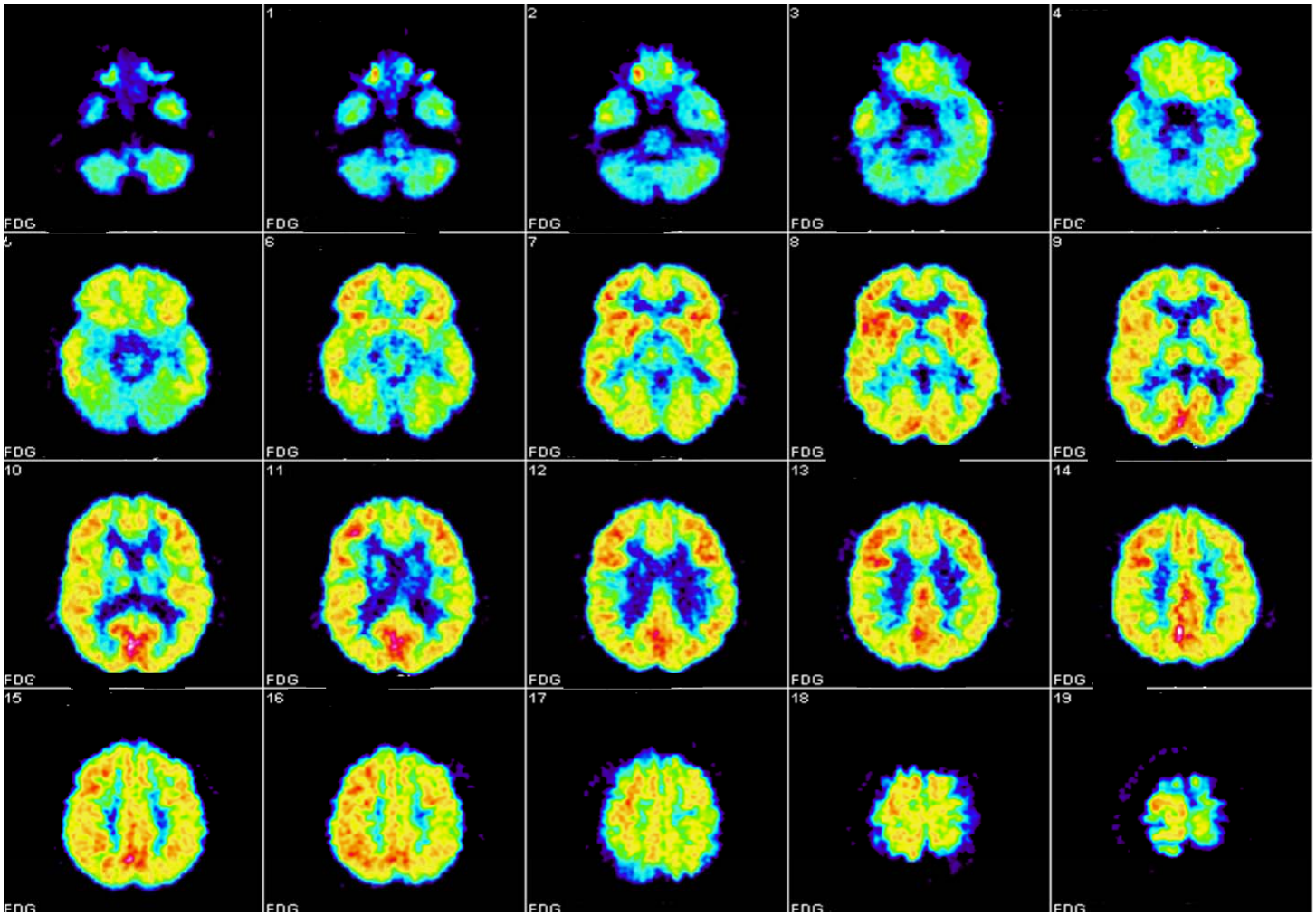


Region	Right	Left	R/L
Post-Lat	77.3	69.4	1.11
Ant-Lat	65.4	60.5	1.08
Anterior	61.0	59.9	1.02
Medial	54.5	51.0	1.07

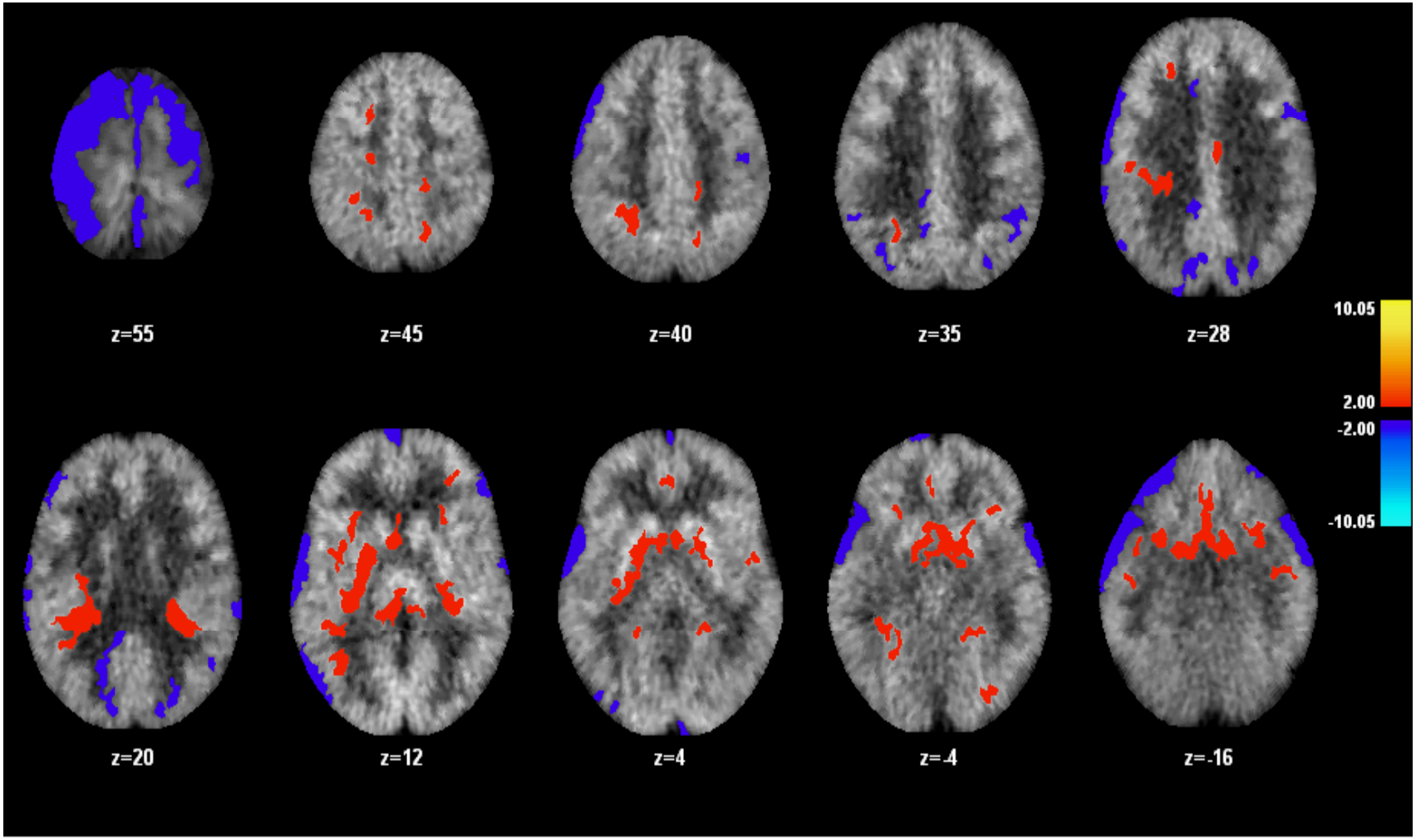
Basic PET with 2DFG



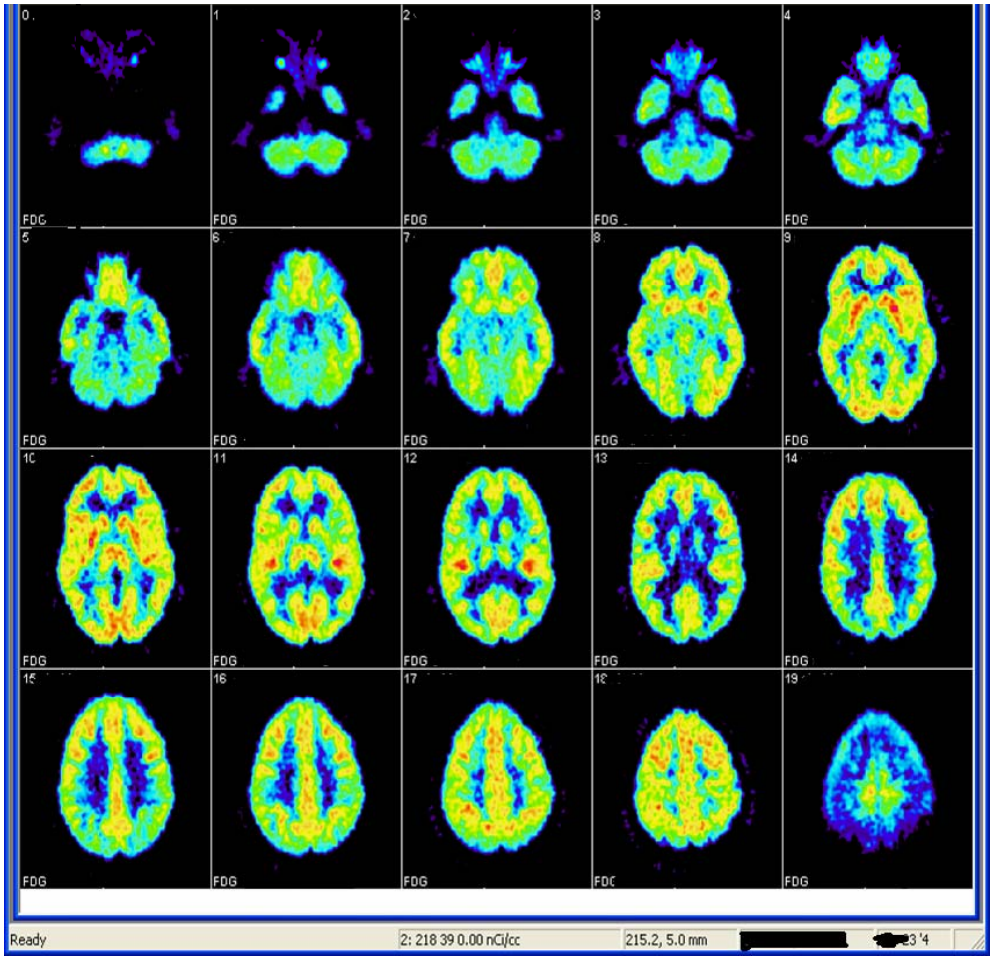
PET in Severe TBI



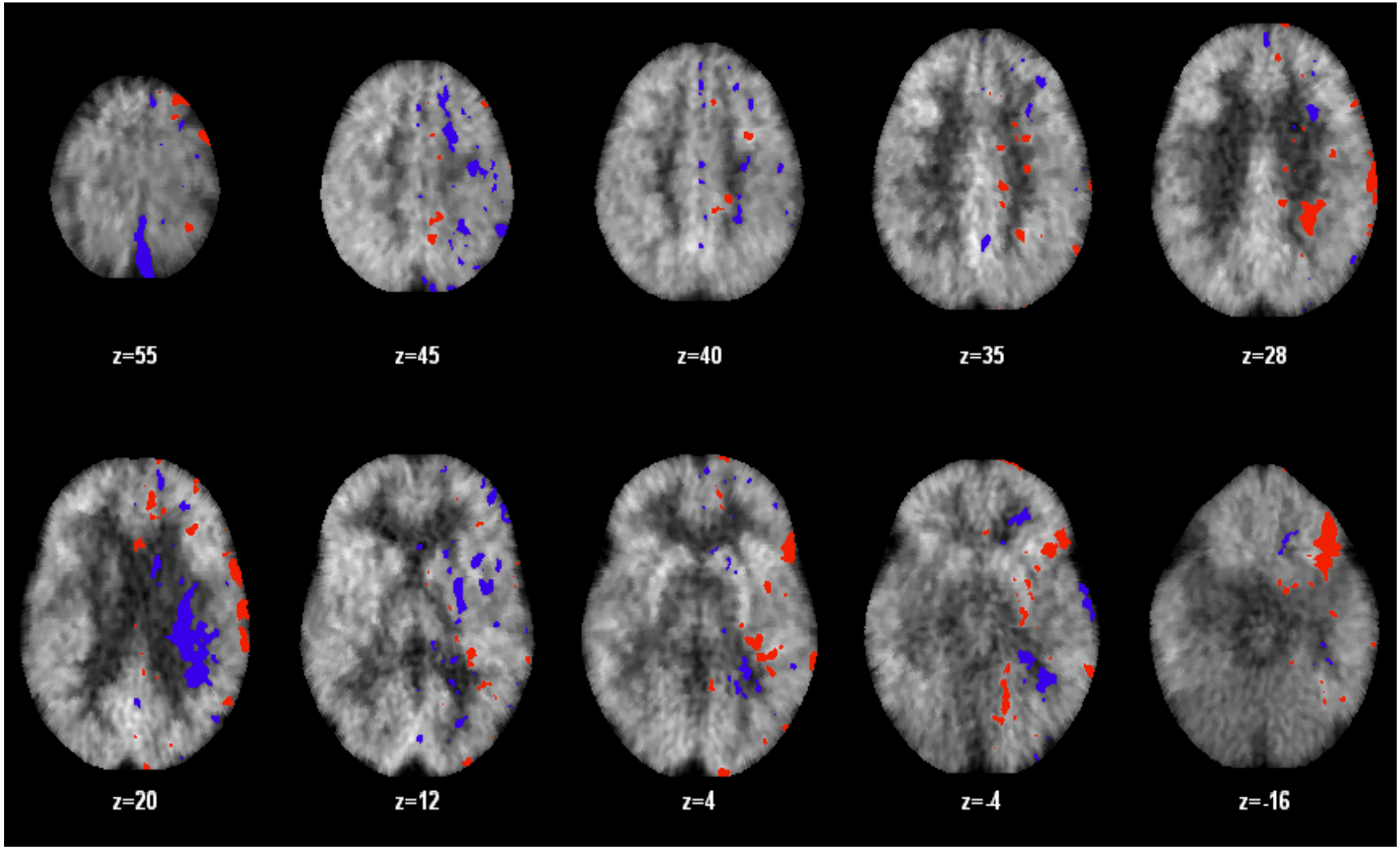
Z Plot-Severe TBI



PET in MTBI

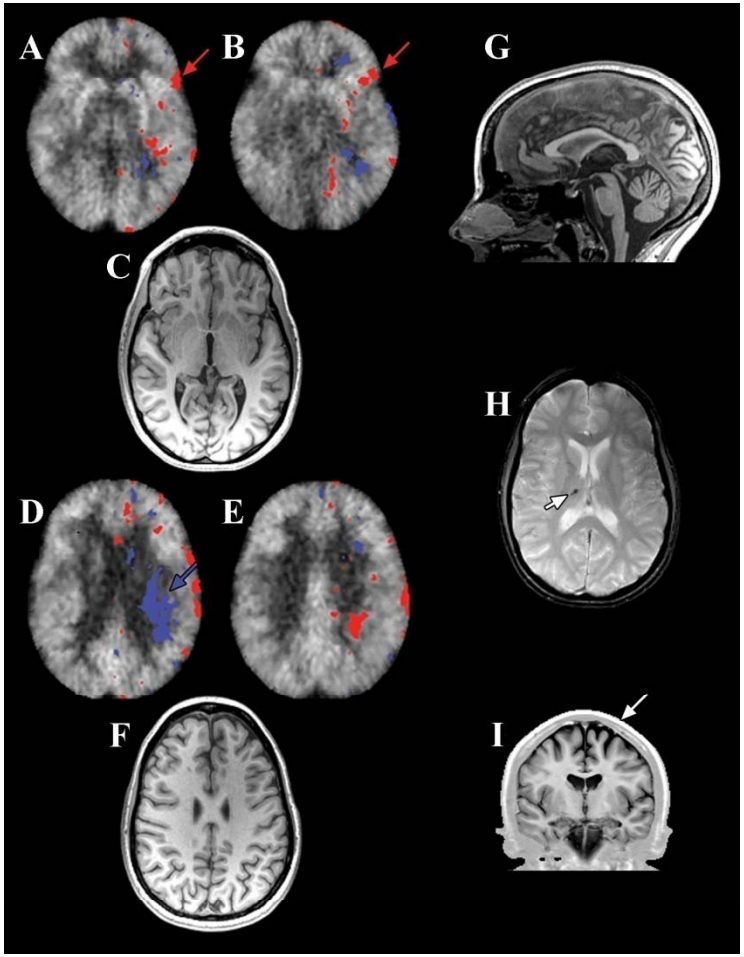


Z Plot-Mild TBI

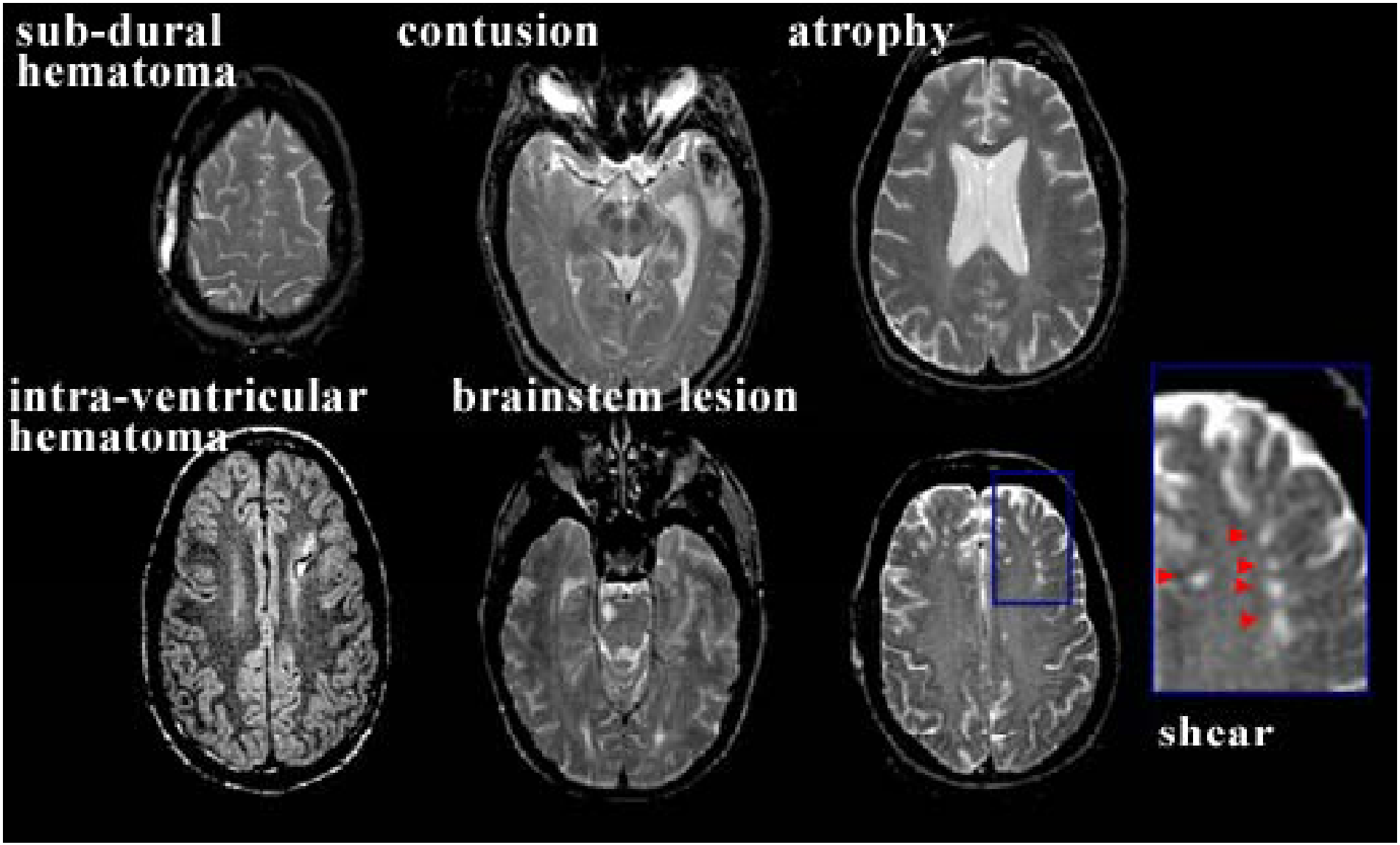


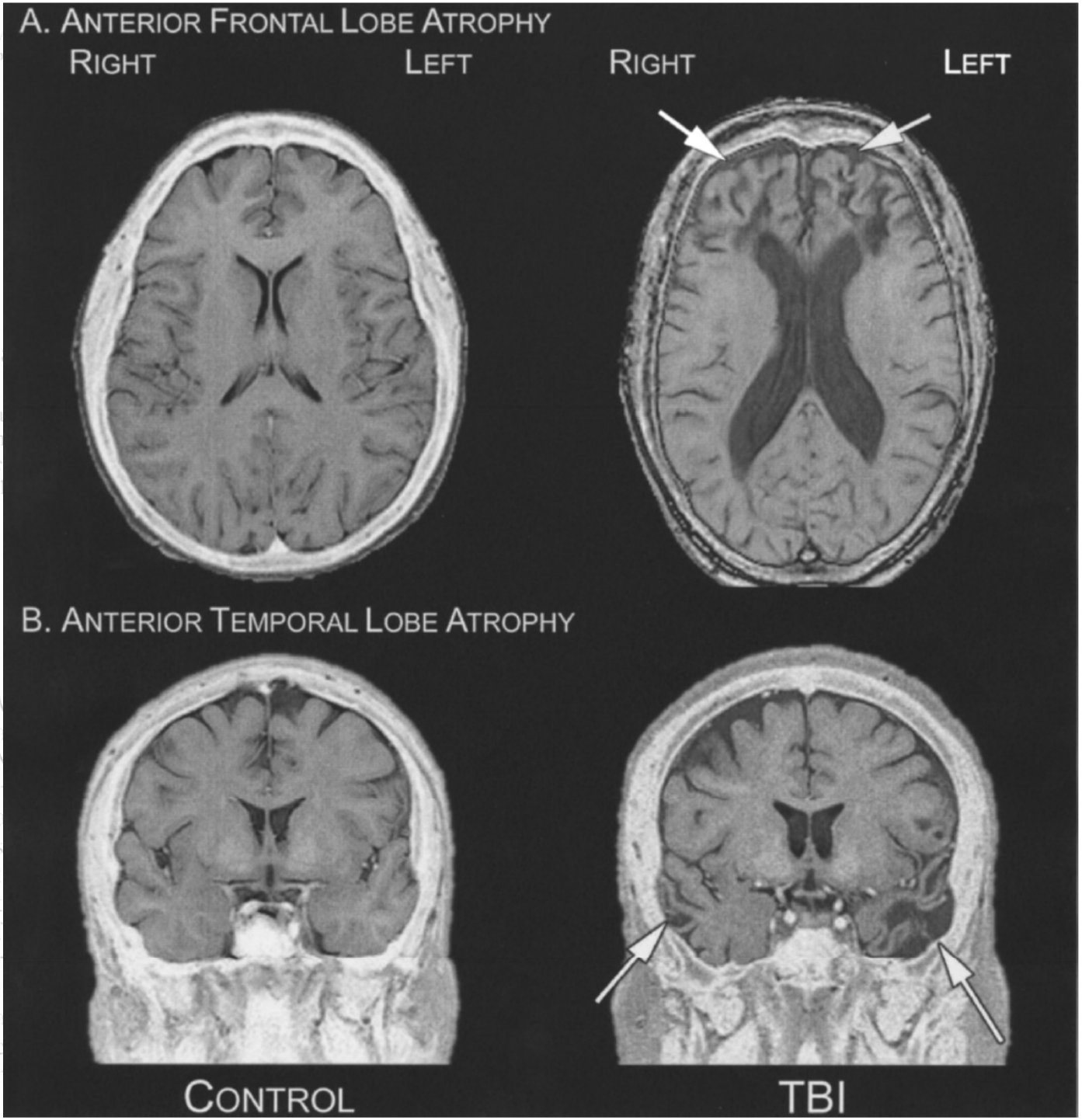
Neurodiagnostic studies

CT vs MRI



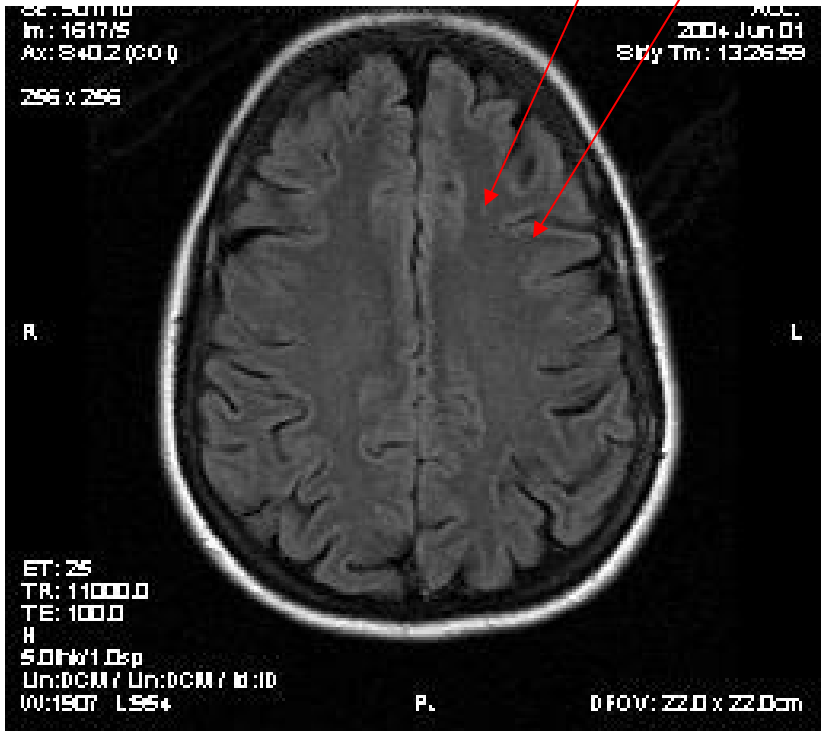
Magnetic Resonance Imaging

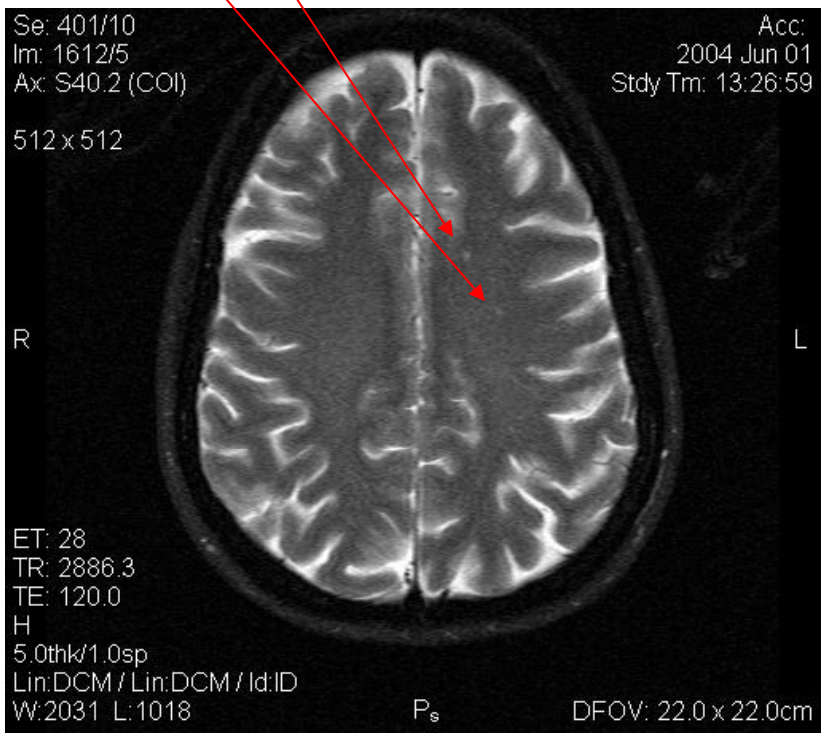



A. Anterior Frontal Lobe Atrophy
Right
Left
Right
Left
B. Anterior Temporal Lobe Atrophy
Control
TBI

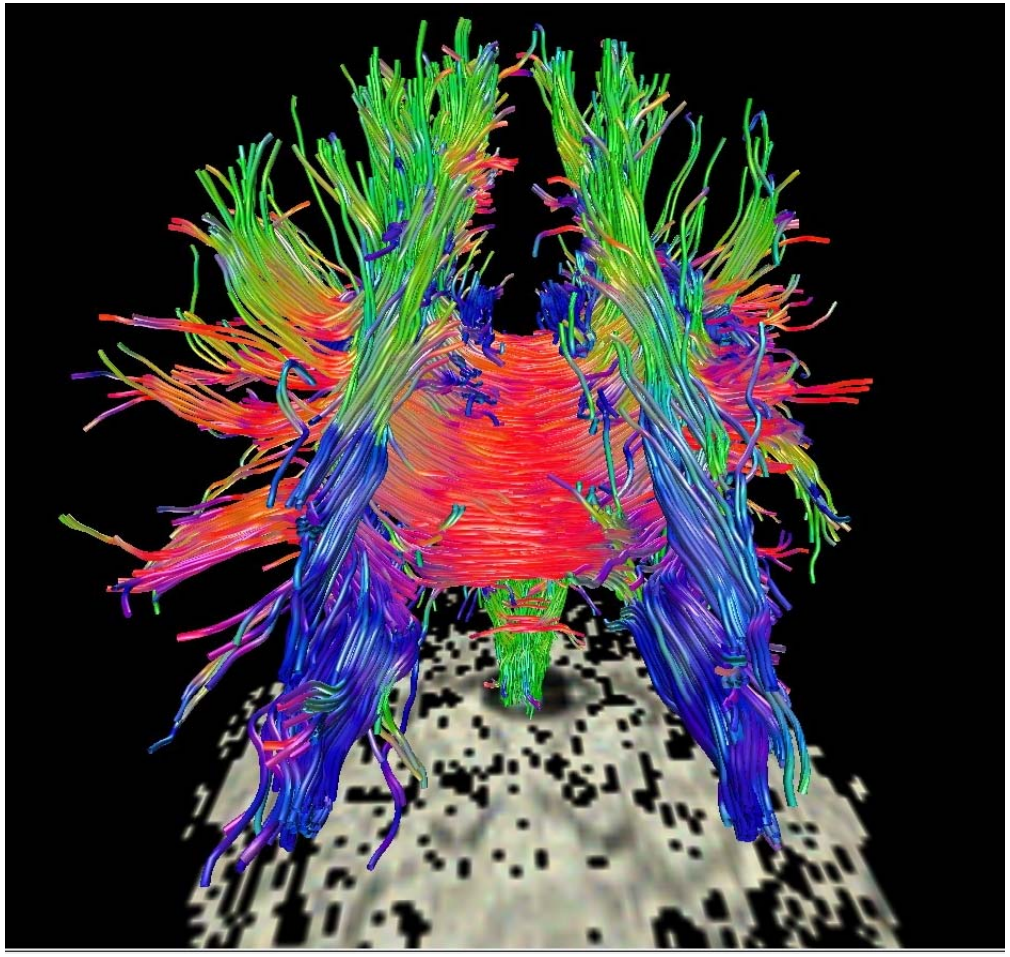
1.5T MRI – 6/1/2004

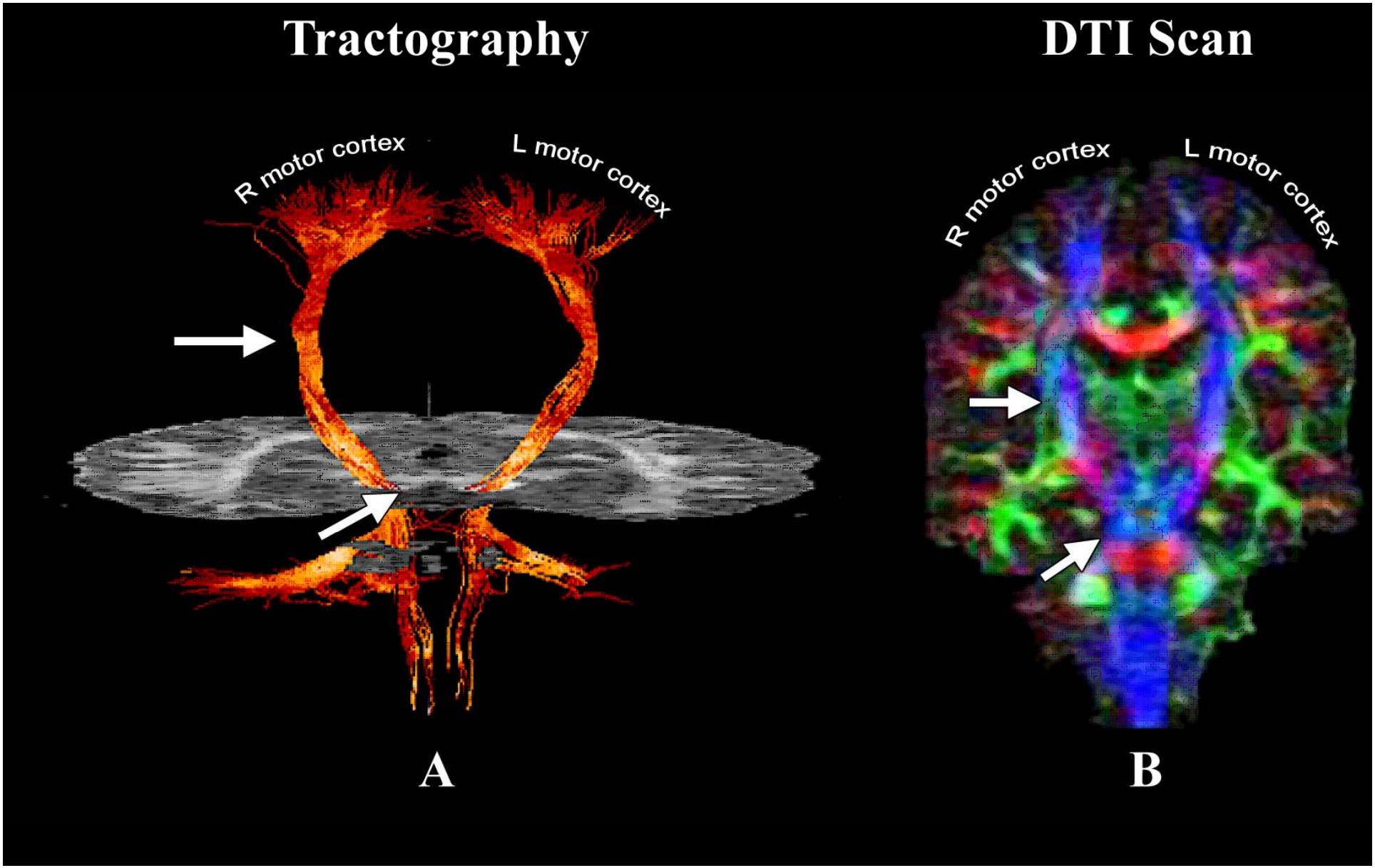
Shearing Injury



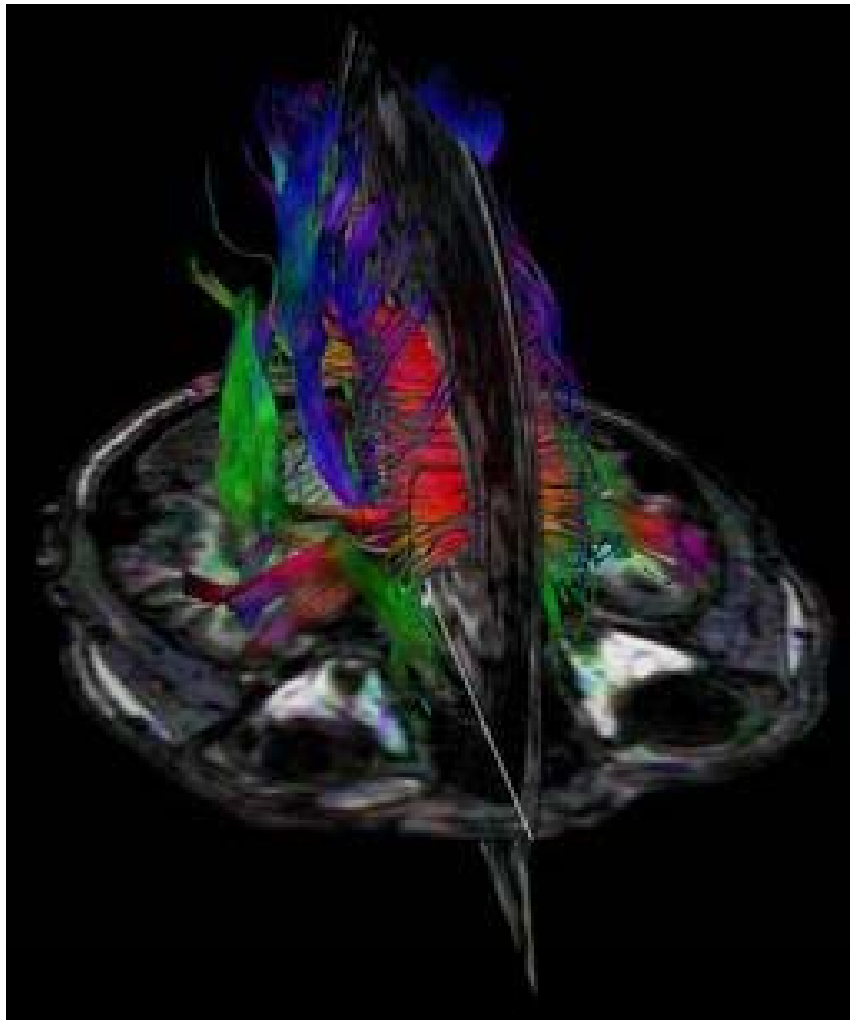


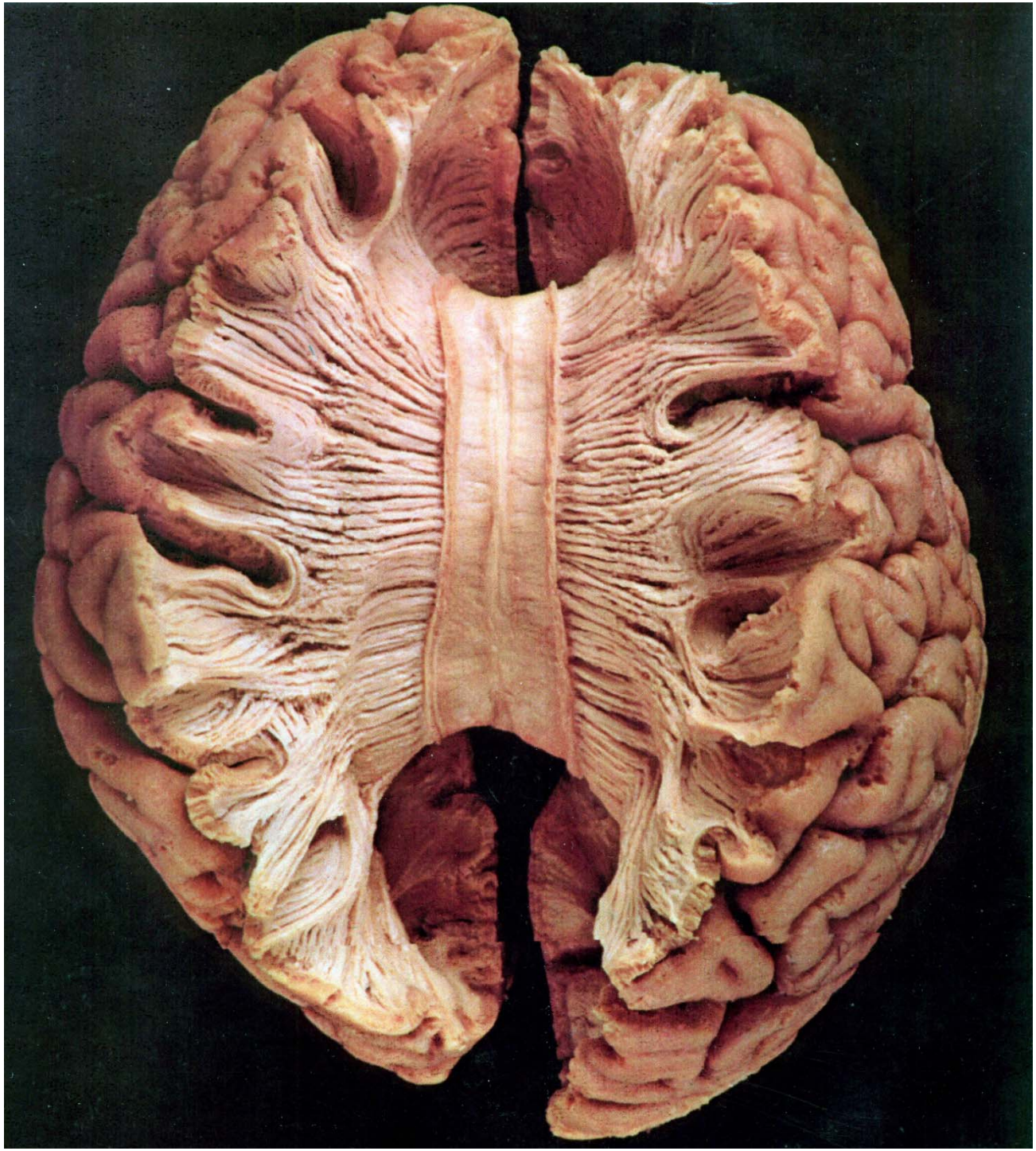
Diffusion Tensor Imaging

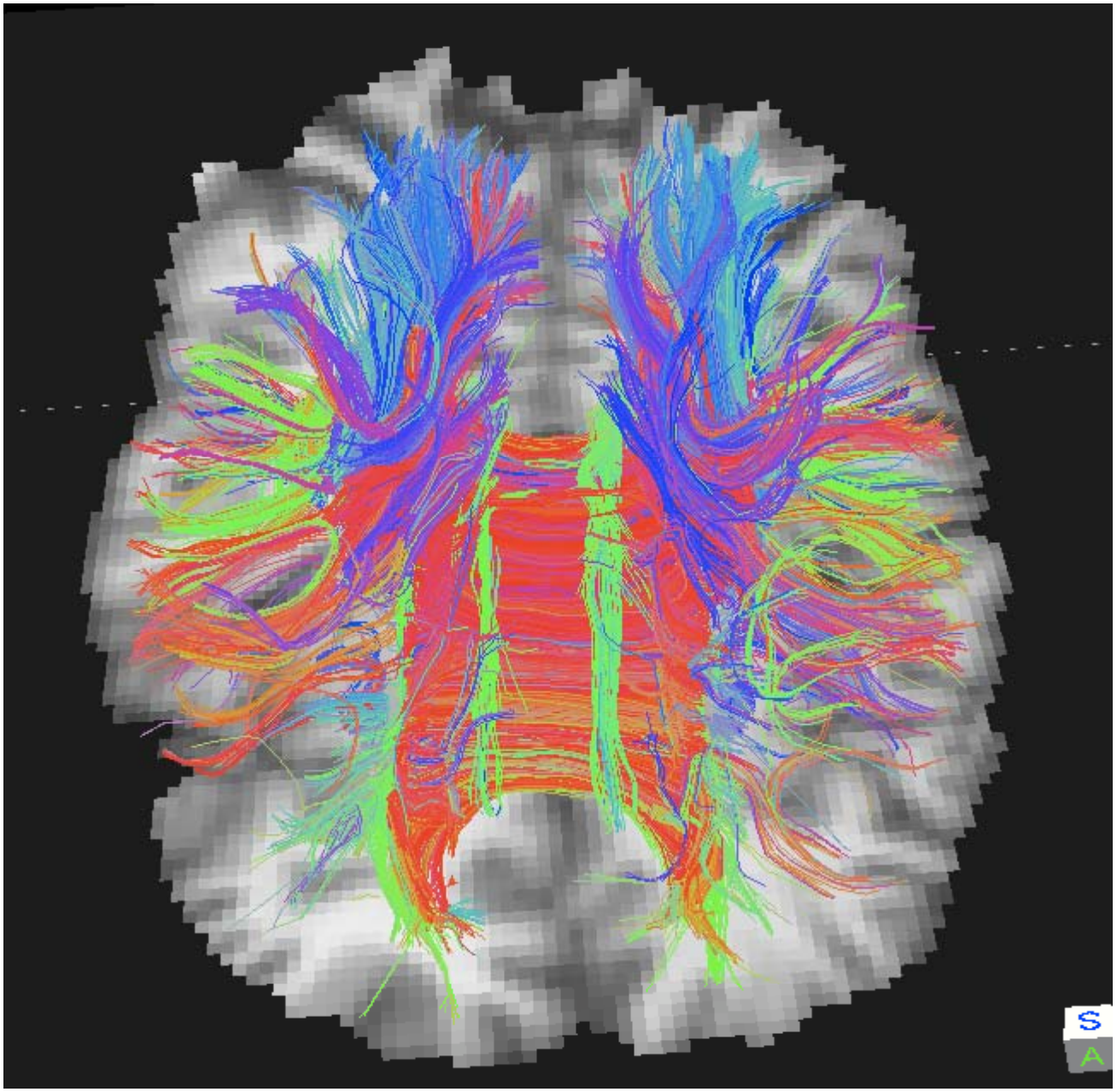


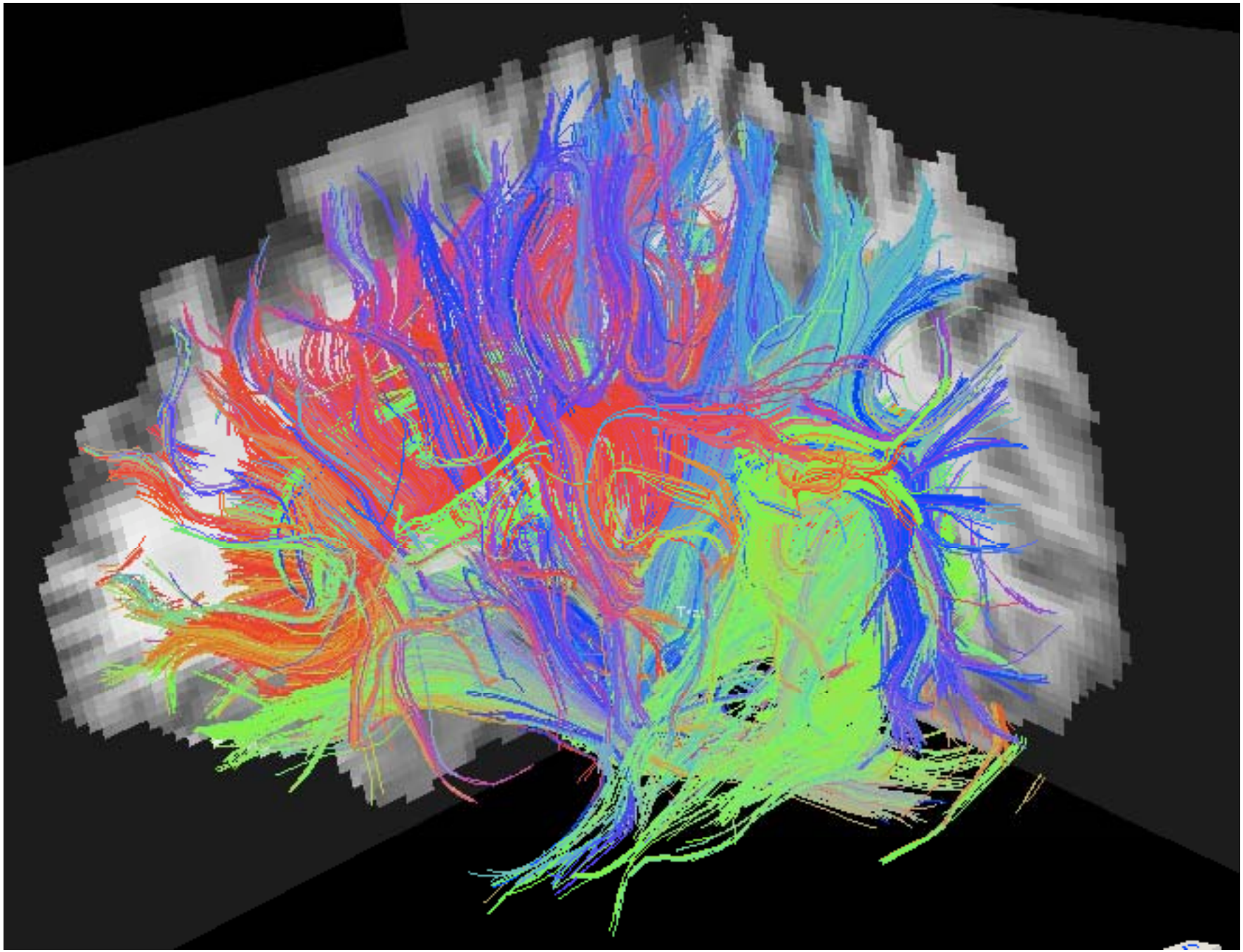

Tractography
DTI Scan
R motor cortex
L motor cortex
R motor cortex
L motor cortex
A
B

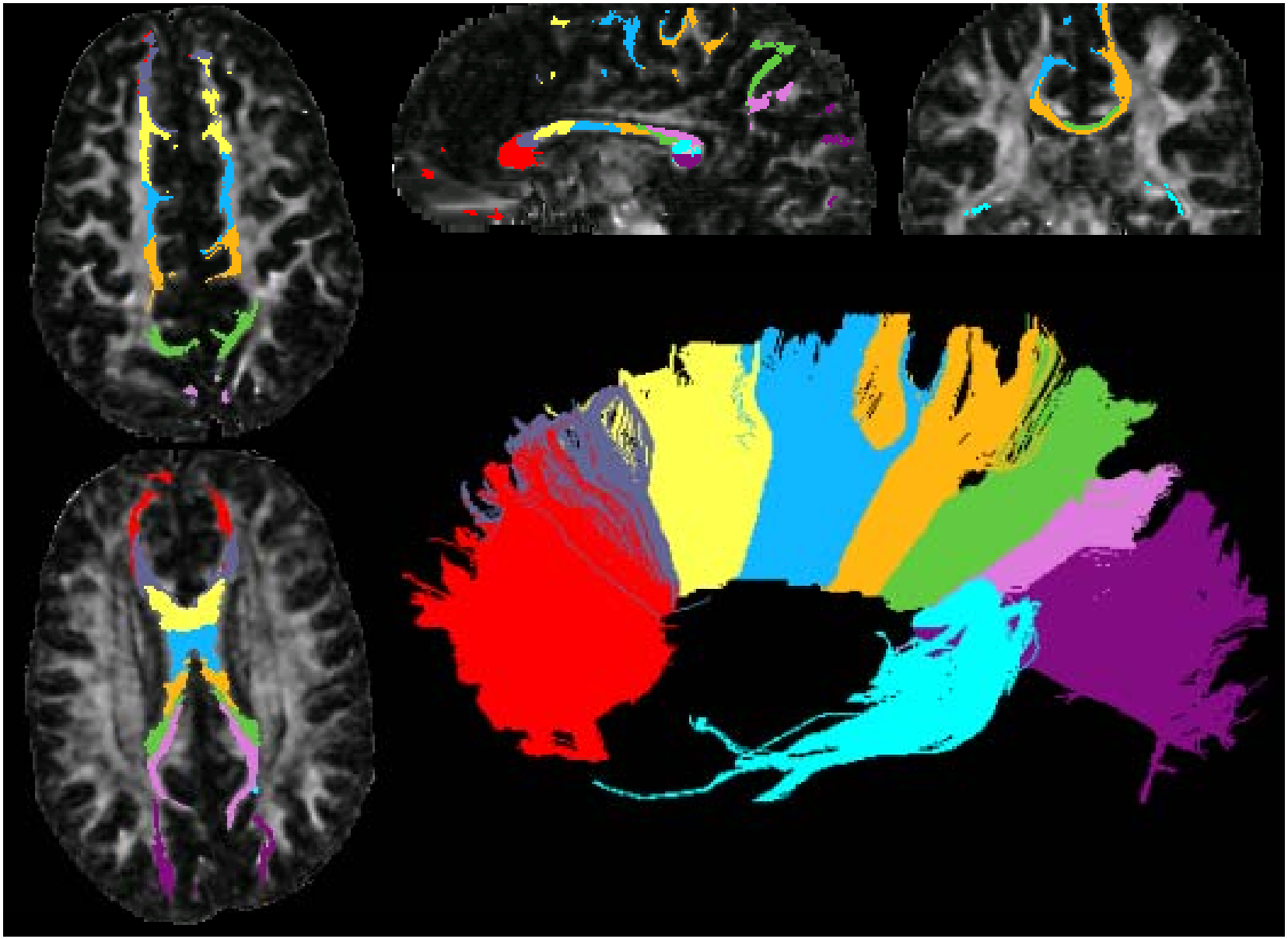
Diffusion Tensor Imaging



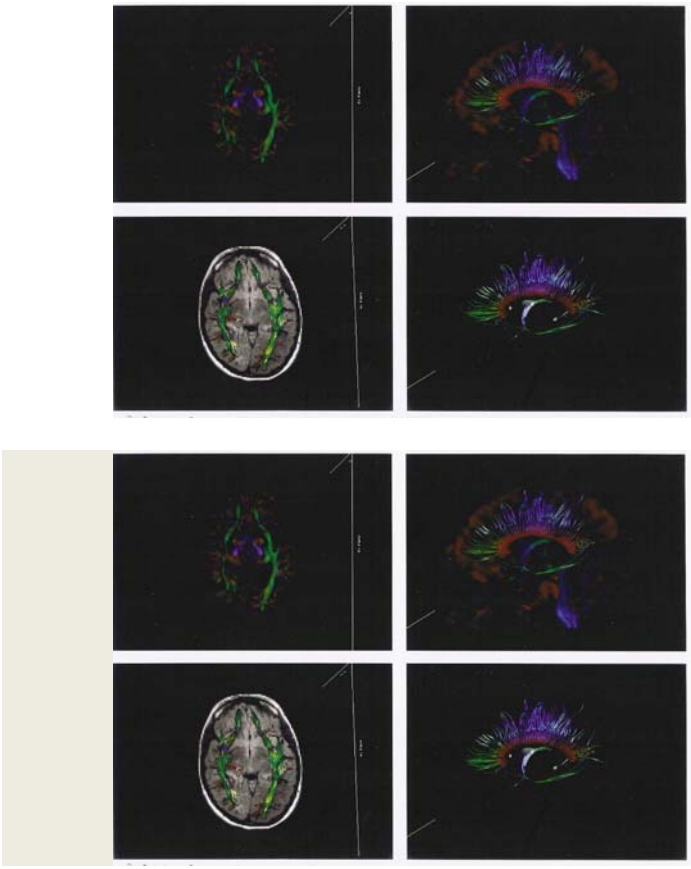


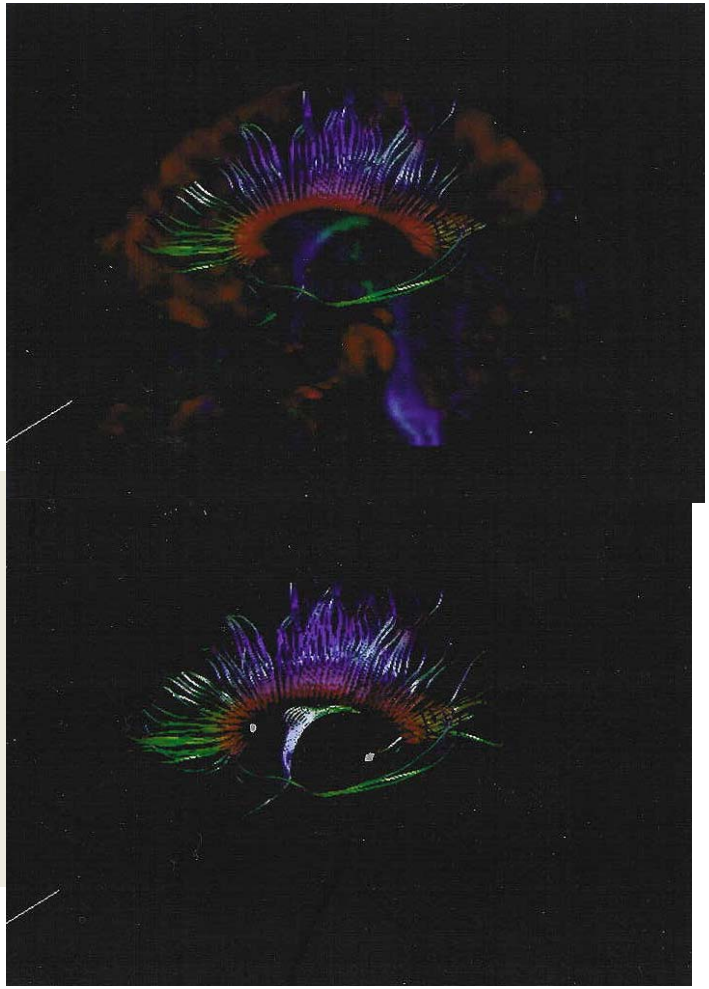

S
A



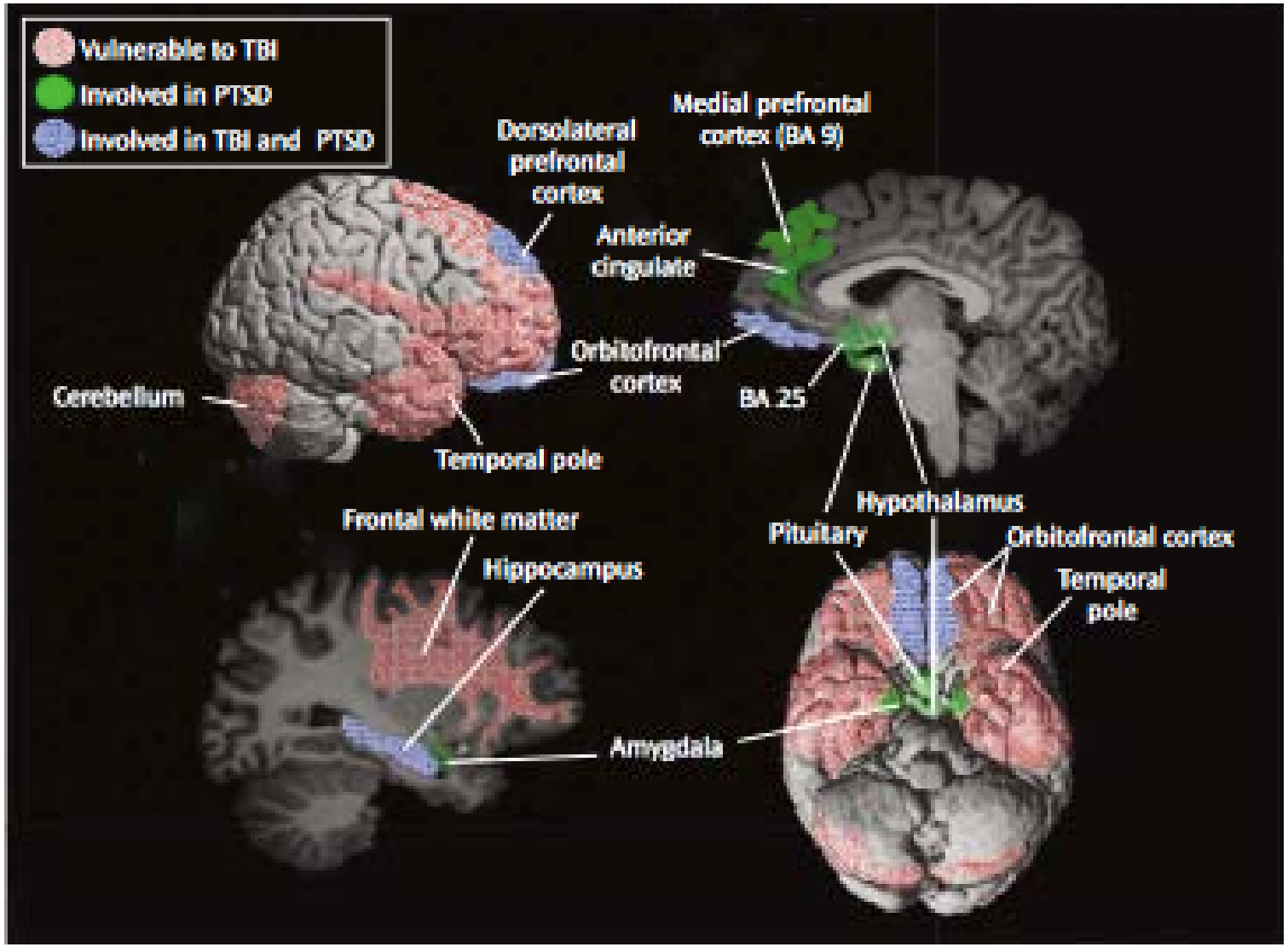


DTI with Occipital Damage





PTSD and MTBI OVERLAP



OUTCOME

Sequelae

(DoD, 2007)

1. Physical: for example, headache, nausea, vomiting, dizziness, blurred vision, sleep disturbance, weakness, paresis/plegia, sensory loss, spasticity, aphasia, dysphagia, apraxia, balance disorders, disorders of coordination, seizure disorder.

2. Cognitive: for example, attention, concentration, memory, speed of processing, new learning, planning, reasoning, judgment, executive control, self-awareness, language, abstract thinking.

3. Emotional/Behavioral: for example, depression, anxiety, agitation, irritability, impulsivity, aggression.

Sequelae

(DoD, 2007)

- Sequelae of traumatic brain injury may resolve quickly, within minutes to hours after the neurological event, or they may persist longer.
- Some sequelae of TBI may be permanent.
- Most signs and symptoms will manifest immediately following the event. However, other signs and symptoms may be delayed from days to months (e.g., subdural hematoma, seizures, hydrocephalus, spasticity).
- Signs and symptoms may occur alone or in varying combinations and may result in a functional impairment.
- These signs and symptoms are not better explained by pre-existing conditions or other medical, neurological, or psychological causes except in cases of an exacerbation of a pre-existing condition.

Prognosis

Mild TBI

65% recover by 3 months

85-90% recover by 12 months

Poor outcome findings

More severe injury; collateral injuries; possible structural-histological lesions; longer LOC+ PTA

Pain

Age: older adult (or young child)

Poor general health

Low premorbid intellectual/cognitive abilities (exception: very high functioning)

Previous head injury; other neurologic disorder

History of alcohol/drug abuse and/or use at time of injury

Previous psychiatric history and /or present depression

Poor psychosocial, economic and vocational support system

No head injury information provided at hospital/no follow-up

Pending litigation/continuing PCS

Prognosis

Moderate & Severe

- Natural recovery: 18 – 24 months
- Length of coma is strongest predictor
- Length of hospitalization
- Associated injuries
- GCS
- Type/amount intracranial diagnosis
  - (+/- bleeding,edema)

Seizure Disorder

Seizure Disorder

Non-penetrating TBI

Early:

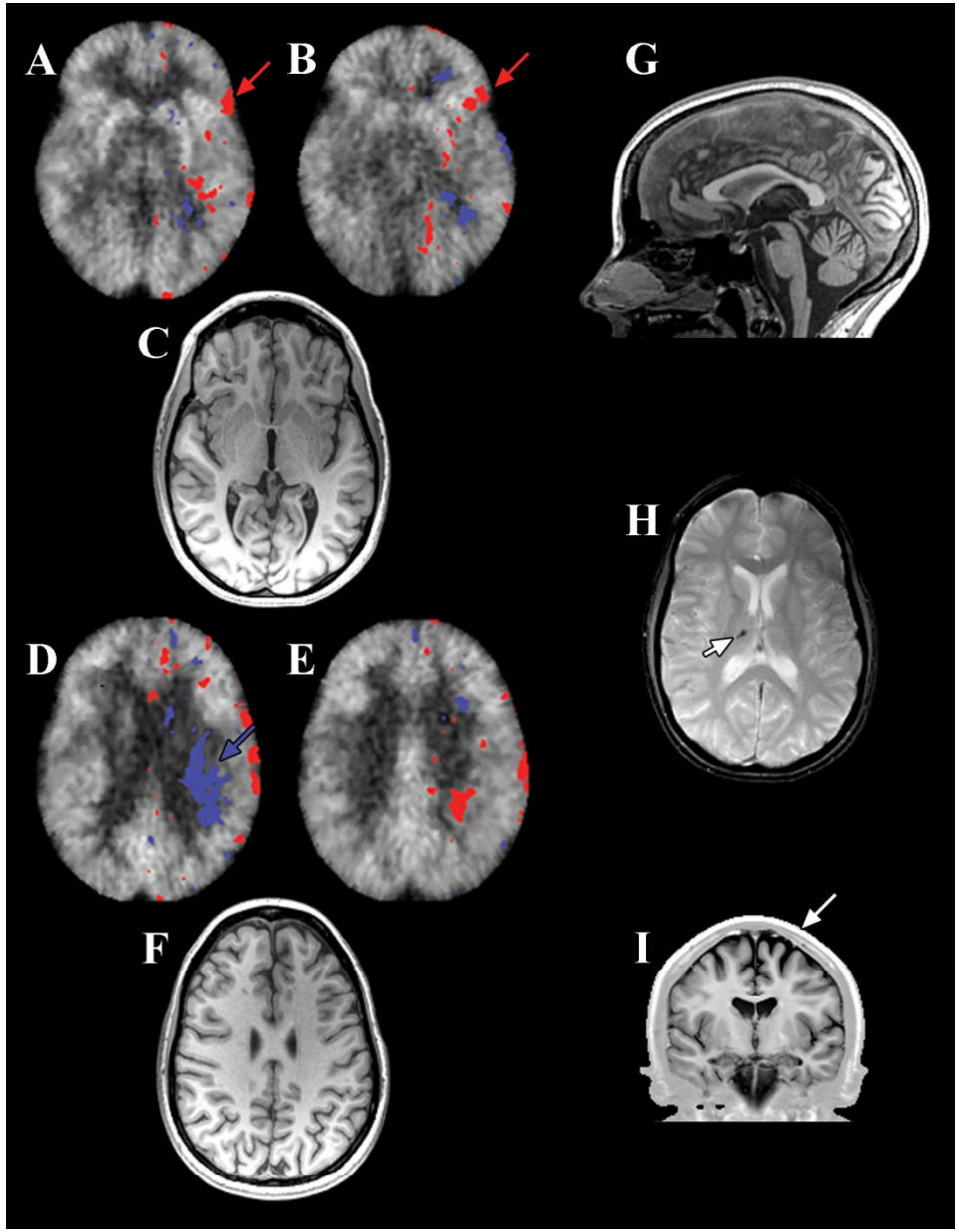
Physiologic reaction
May enhance outcome
Doesn't predict later epilepsy

Late:

Microshearing & hemorrhage
Hemosiderin deposition
Early AED's don't prevent

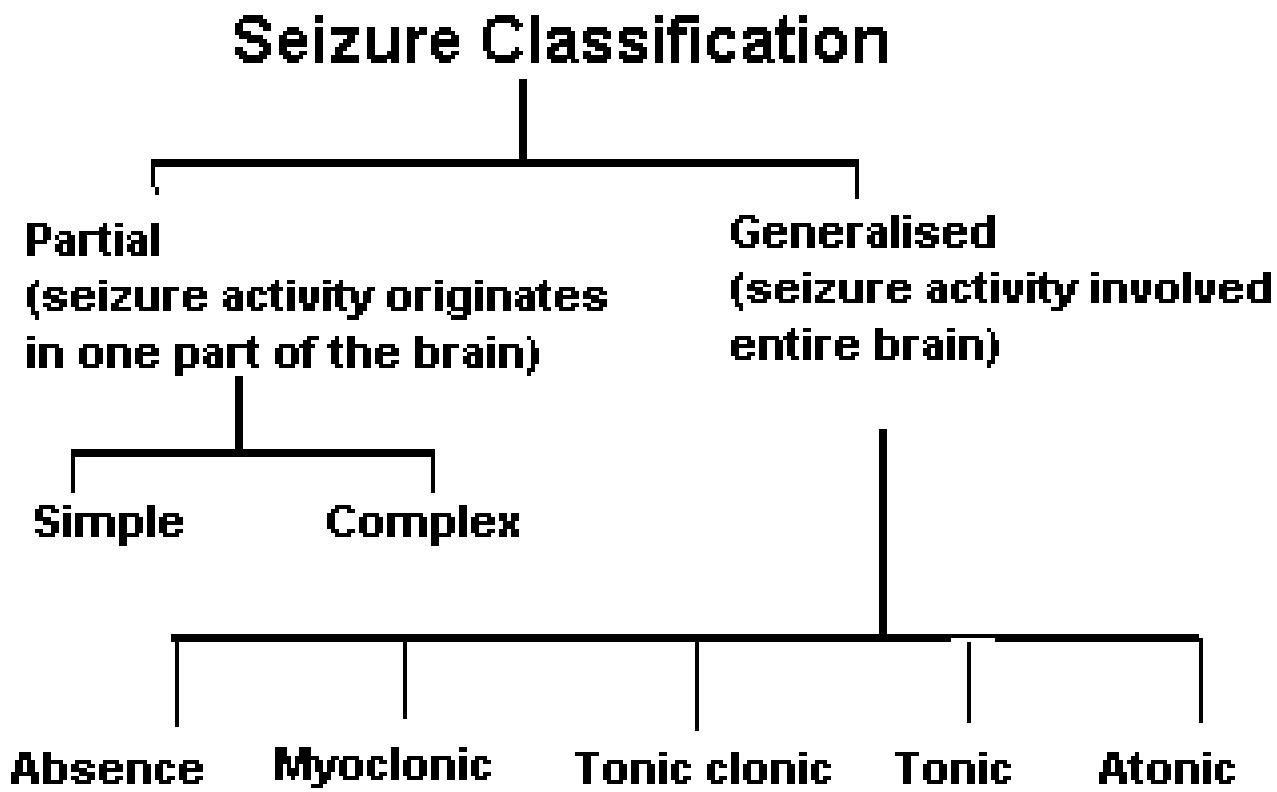
Penetrating TBI

Persistent risk w/o initial sz



Neurologic Consequences

Seizure Disorder



Seizure Disorder

The Lancet, Volume 373, Issue 9669, Pages 1105 - 1110, 28 March 2009
doi:10.1016/S0140-6736(09)60214-2

Long-term risk of epilepsy after traumatic brain injury in children and young adults: a population-based cohort study

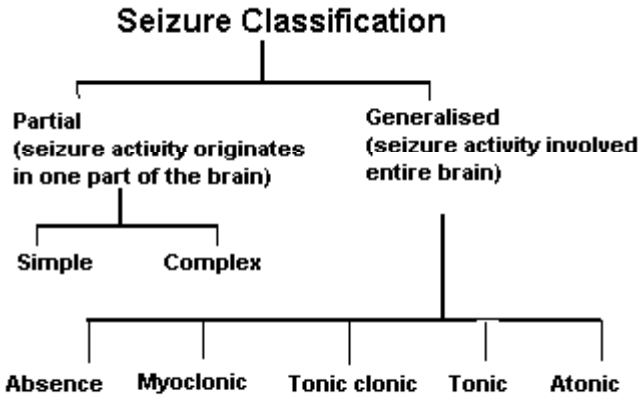
Dr Jakob Christensen MD [a b], Marianne G Pedersen MSc [c], Carsten B Pedersen MSc [c], Per Sidenius MD [a], Jørn Olsen MD [d e], Mogens Vestergaard MD [f]

Summary

Background
The risk of epilepsy shortly after traumatic brain injury is high, but how long this high risk lasts is unknown. We aimed to assess the risk of epilepsy up to 10 years or longer after traumatic brain injury, taking into account sex, age, severity, and family history.

Methods
We identified 1 605 216 people born in Denmark (1977–2002) from the Civil Registration System. We obtained information on traumatic brain injury and epilepsy from the National Hospital Register and estimated relative risks (RR) with Poisson analyses.

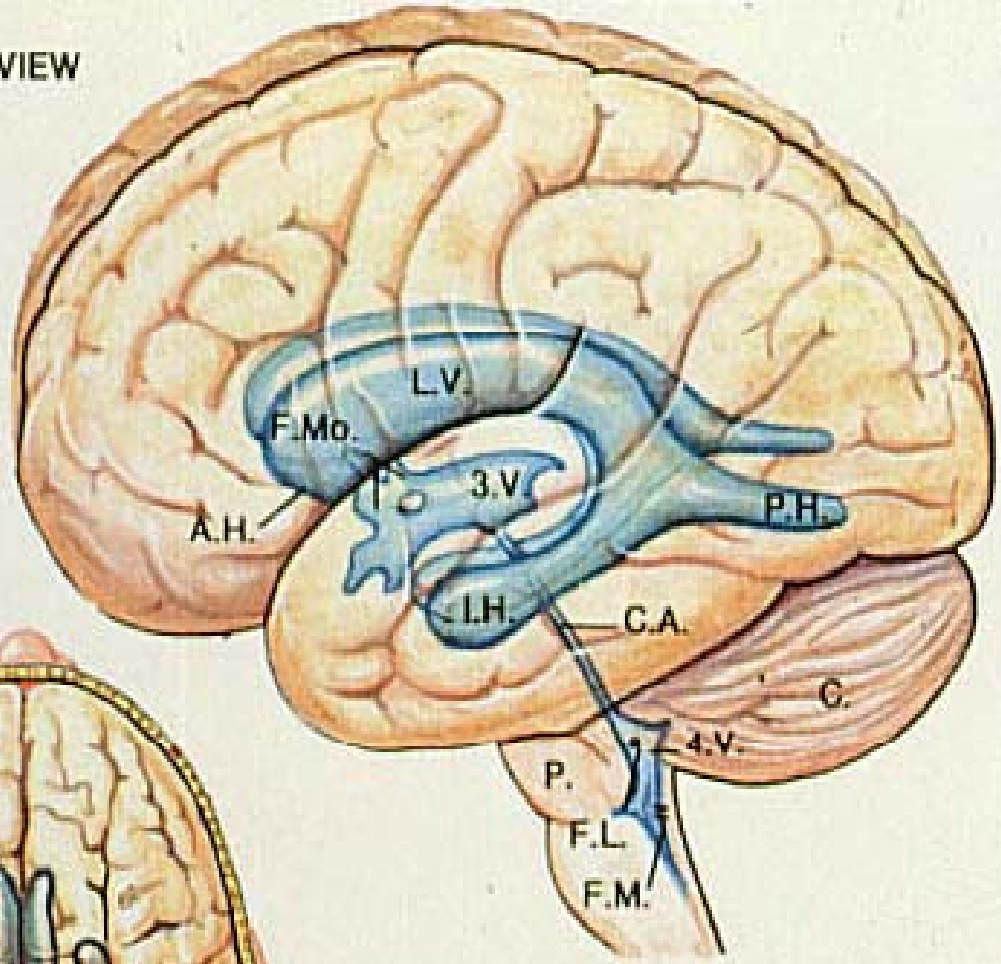
Findings
Risk of epilepsy was increased after a mild brain injury (RR 2·22, 95% CI 2·07–2·38), severe brain injury (7·40, 6·16–8·89), and skull fracture (2·17, 1·73–2·71). The risk was increased more than 10 years after mild brain injury (1·51, 1·24–1·85), severe brain injury (4·29, 2·04–9·00), and skull fracture (2·06, 1·37–3·11). RR increased with age at mild and severe injury and was especially high among people older than 15 years of age with mild (3·51, 2·90–4·26) and severe (12·24, 8·52–17·57) injury. The risk was slightly higher in women (2·49, 2·25–2·76) than in men (2·01, 1·83–2·22). Patients with a family history of epilepsy had a notably high risk of epilepsy after mild (5·75, 4·56–7·27) and severe brain injury (10·09, 4·20–24·26).

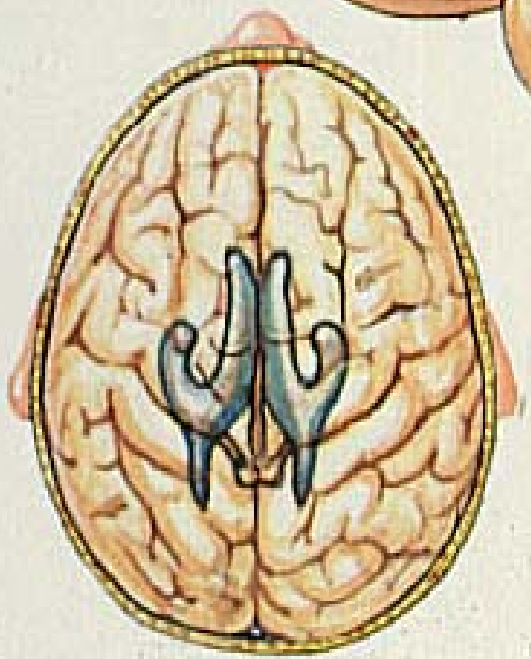


Hydrocephalus

VENTRICLES OF THE BRAIN

LATERAL VIEW



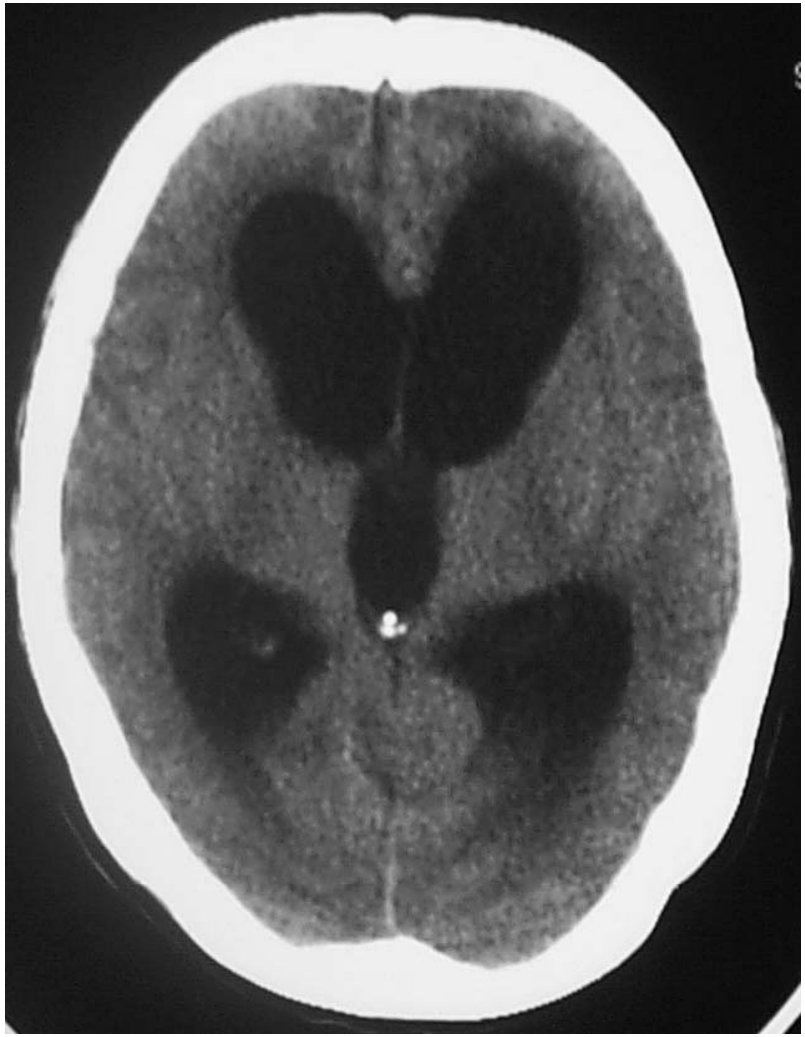


TOP VIEW

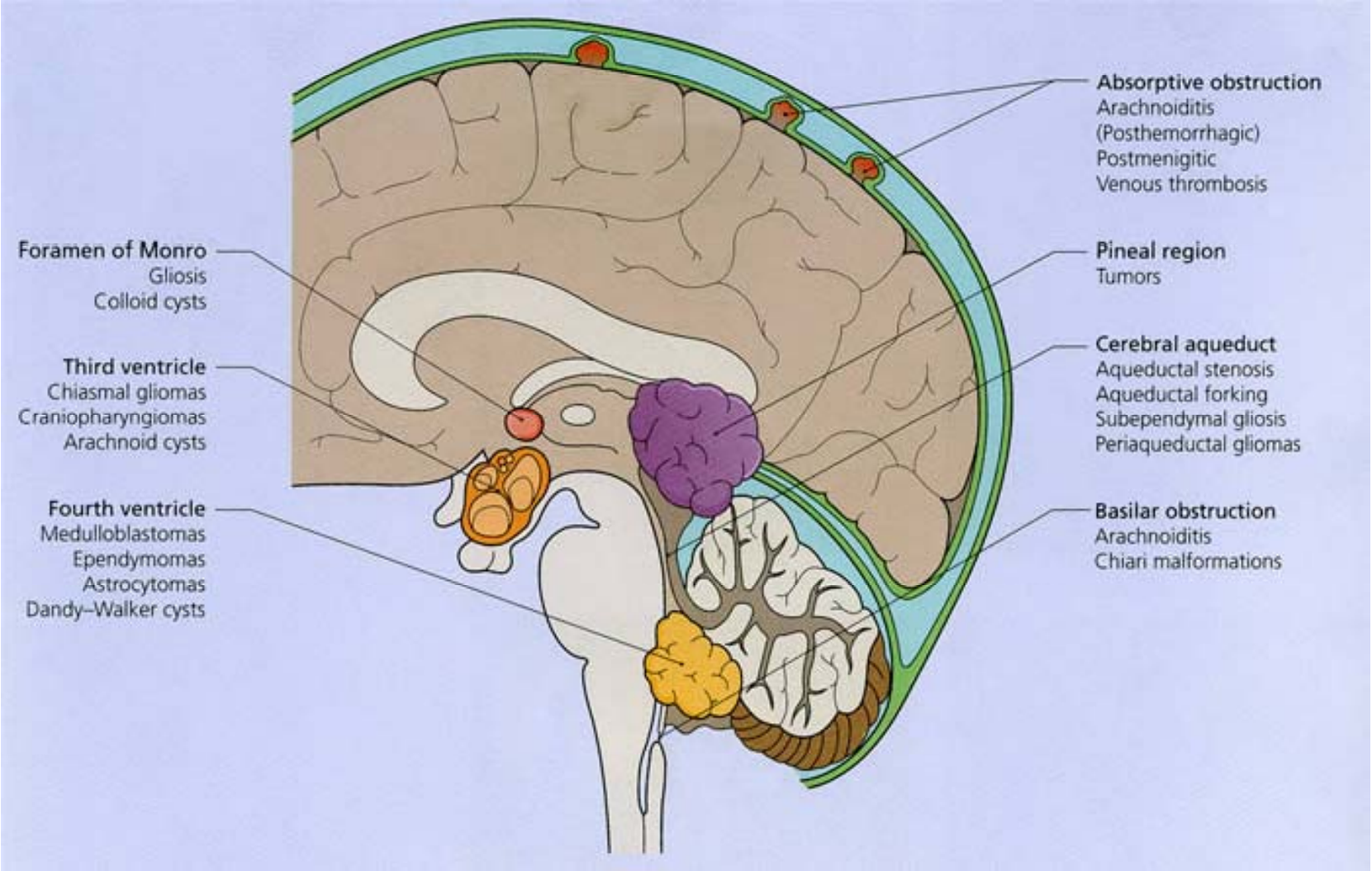
A.H. – ANTERIOR HORN OF LATERAL VENTRICLE
C. – CEREBELLUM
C.A. – CEREBRAL AQUEDUCT
F.L. – FORAMEN OF LUSCHKA
F.M. – FORAMEN OF MAGENDIE
F.Mo. – FORAMEN OF MONRO
4.V. – FOURTH VENTRICLE

I.H. – INFERIOR HORN OF LATERAL VENTRICL
L.V. – LATERAL VENTRICL
P. – PONS
P.H. – POSTERIOR HORN LATERAL VENTRICL
3.V. – THIRD VENTRICLE

Hydrocephalus



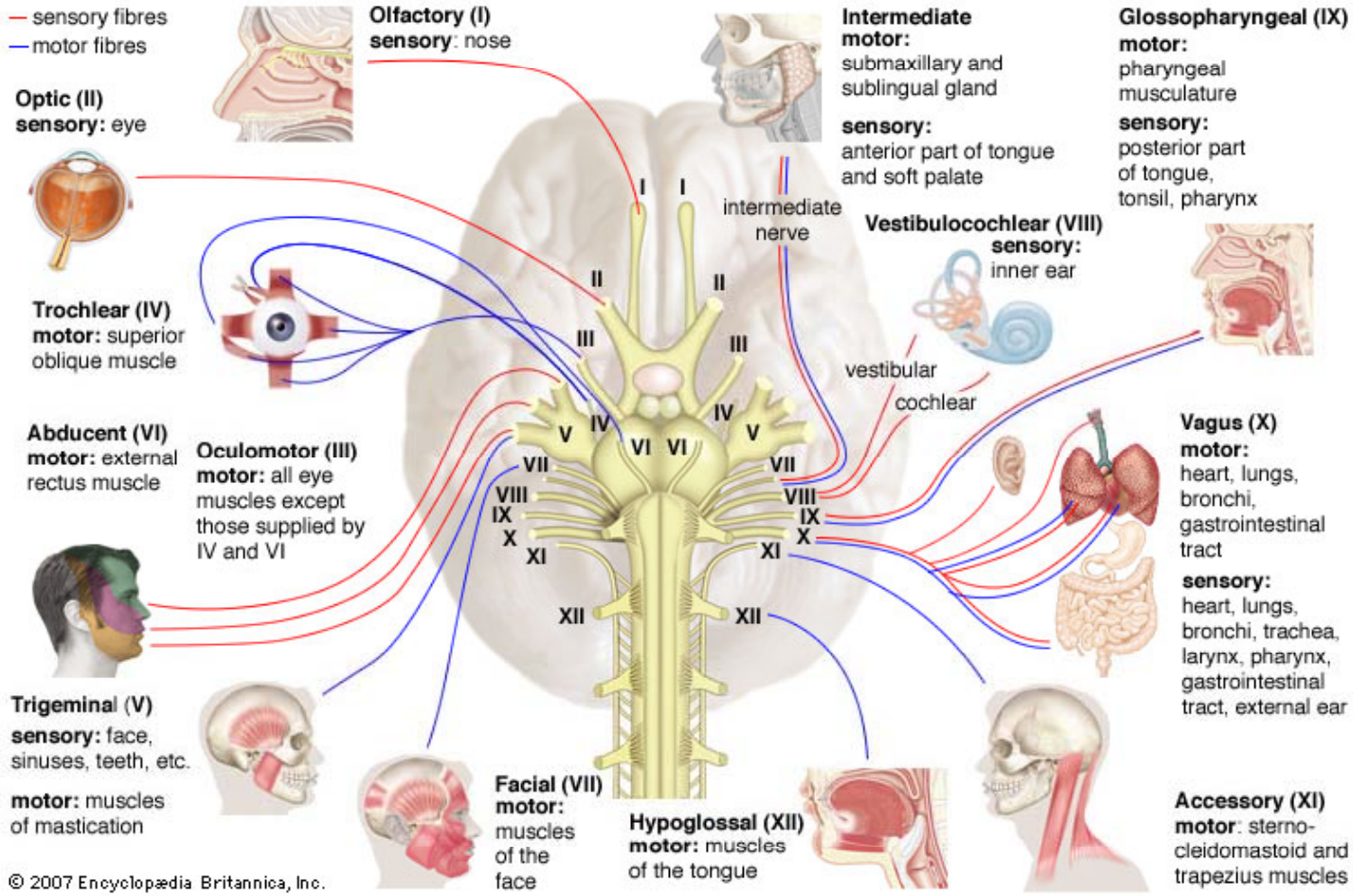
Pathophysiology of hydrocephalus



Cranial Nerve Damage

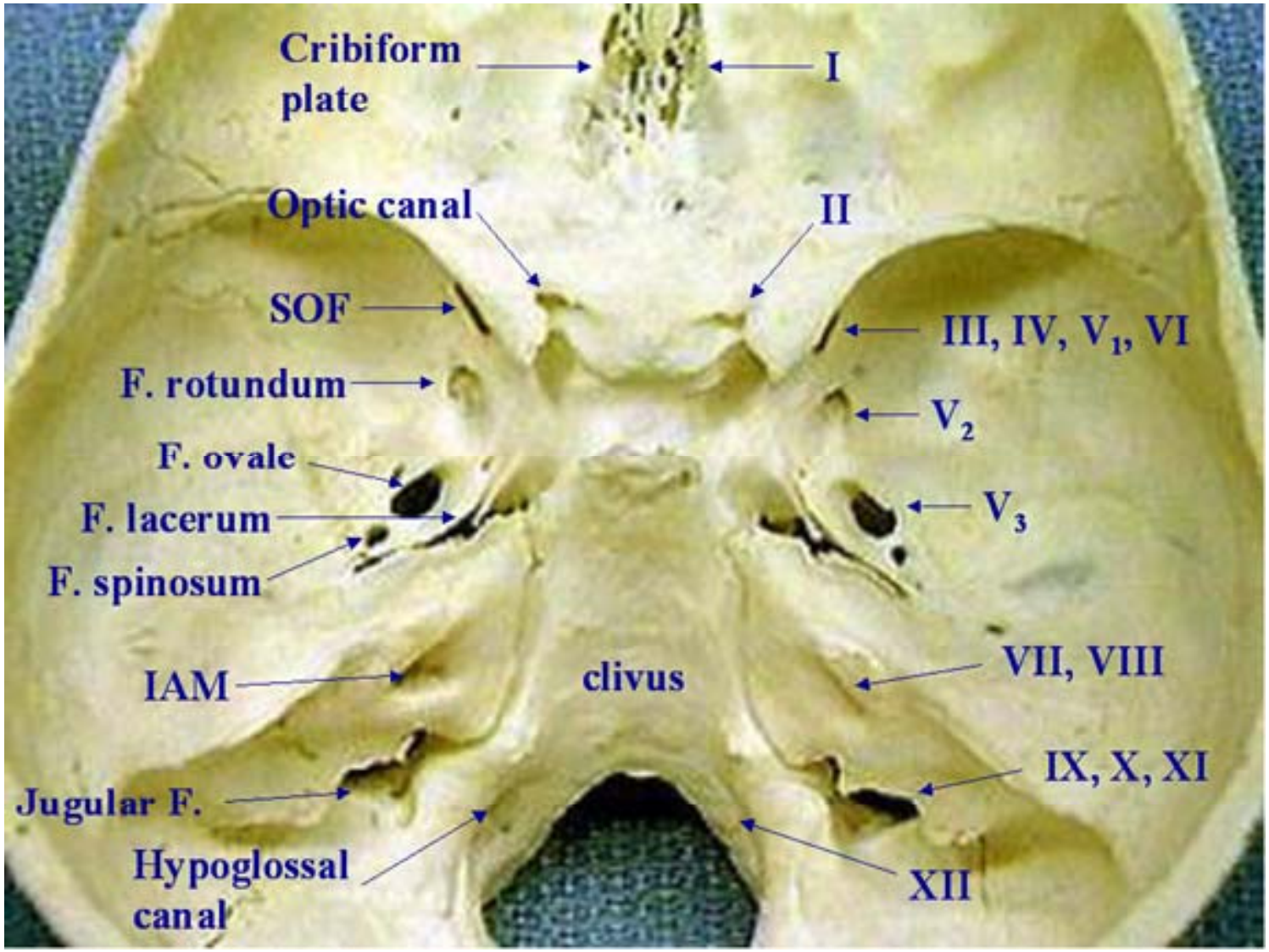
Neurologic Consequences

Cranial Nerves



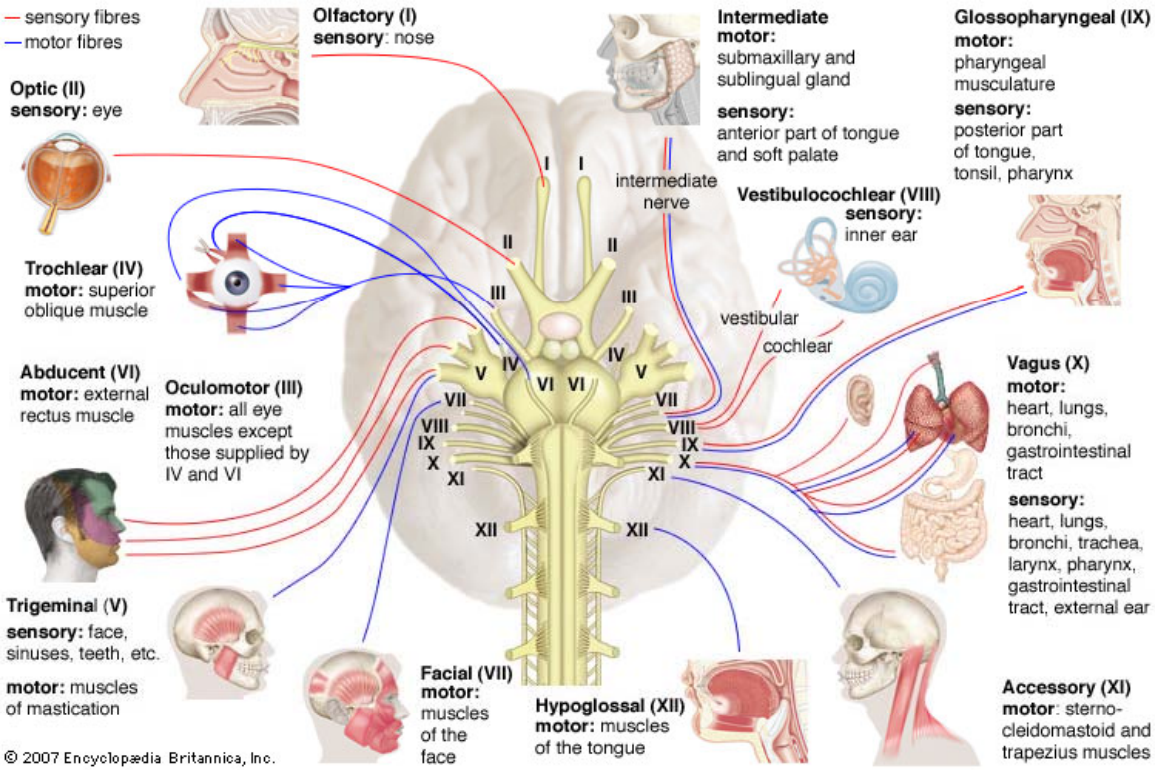
Neurologic Consequences

Cranial Nerve Exit Pathways



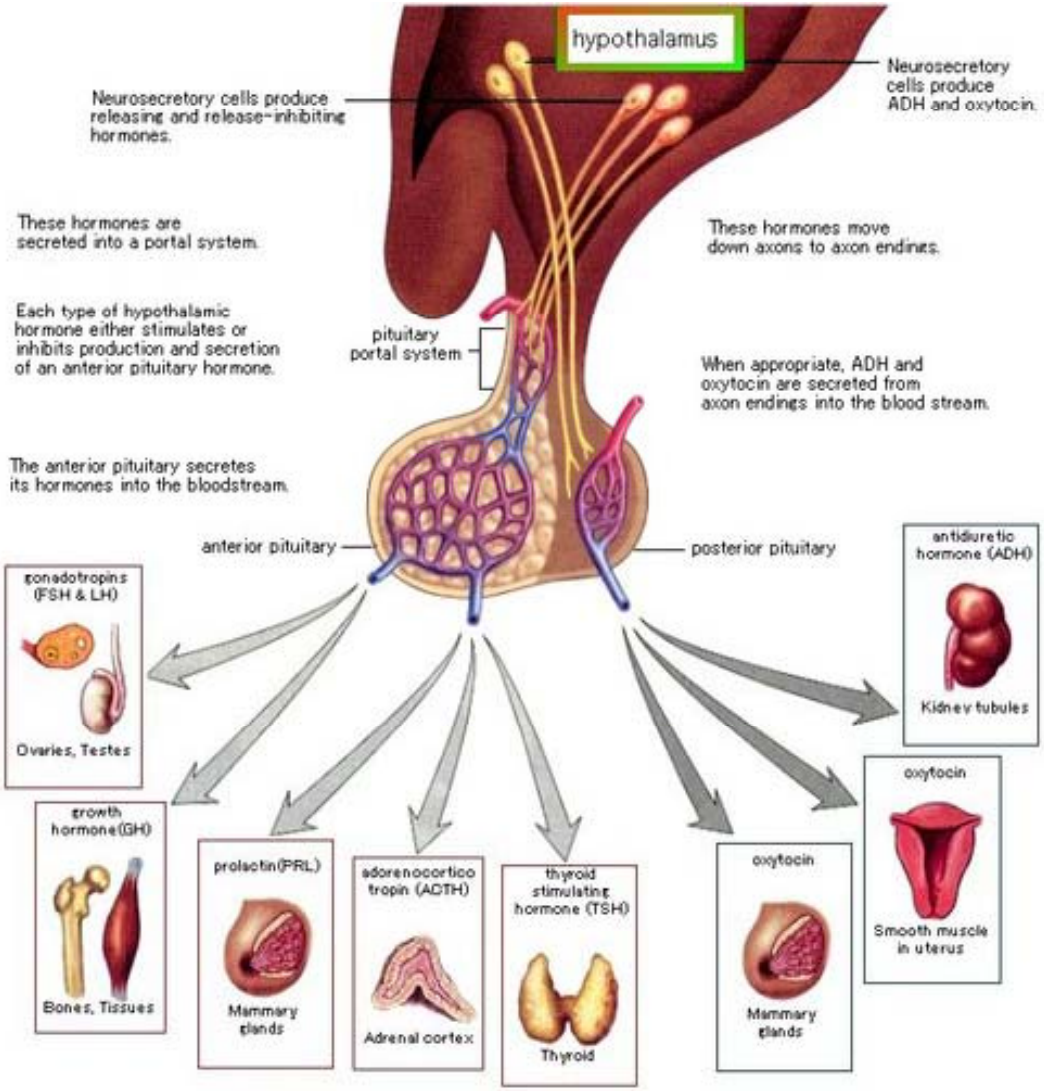
Cranial Nerve Injuries

I: Anosmia; parosmia; ageusia
II: Blindness; Blurry vision
III: Double vision;
Dysconjugate gaze;
Loss of yoking/PTVS
IV: Loss of upward tracking;
Double vision/PTVS
V: Loss of sensation in face;
Weakness chewing
VI: Inability to look laterally/PTVS
VII: Facial weakness
Hypersensitivity to loud noise
VIII: Tinnitus; sensorinerual deafness;
Auditory processing deficit;
Vertigo; visual dependency;
Nystagmus
IX: Dysphagia; ageusia
X: Autonomic changes
XI: Shoulder drop;
Reduced neck ROM
XII: Dysarthria; dysphagia



Post-traumatic pituitary damage

Hypothalamic-pituitary axis



Hormones & Biologic Action

Origin	Hormones	Function
Hypothalamus	Stimulators Inhibitors (**see Table 2**)	Pituitary hormone regulation
Pituitary, anterior	ACTH FSH Growth hormone Prolactin LH TSH	Adrenal control Gonad regulation Growth regul./milk prod. Milk production Gonad regulation Thyroid control
Pituitary, posterior	ADH Oxytocin	Water conservation Uterus contraction & milk ejection
Thyroid	Thyroxine	Metabolic rate control
Parathyroid	PTH Calcitonin	Calcium regulation Calcium regulation
Gut	Gut hormones	Food digestion
Pancreas	Insulin Glucagon	Nutrient metabolism Nutrient metabolism
Adrenals	Cortisol Aldosterone Epinephrine	Body preservation Salt conservation Stress response
Ovaries	Estradiol Progesterone	Female characteristics
Testes	Testosterone	Male characteristics

Neuroendocrine

- Cerebral salt wasting
- Hypoadrenalism
- Hypogonadism
- Hypopituitarism
- Hypothyroidism
- Diabetes insipidus
- Impotence
- Libido
- Obesity
- Osteoporosis
- SIADH

Hypothalamic dysfunction

Hypothalamic Dysregulation
Sleep

PTA/Delirium

Sleep wake cycle disruption

Initial insomnia

Middle insomnia

Phase shift syndromes

Phase forward

Phase backward

Sleep apnea syndrome

Central

Obstructive

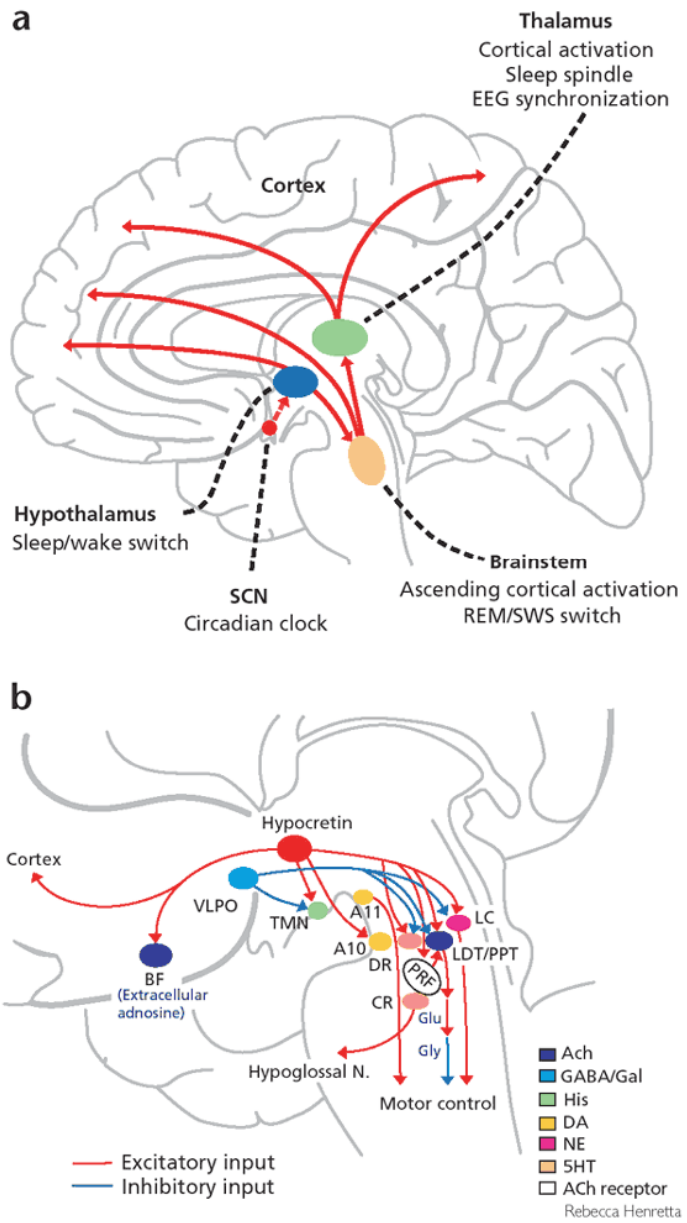
Mixed

Inefficient sleep

Reduced total sleep time

Inadequate SWS (Stage 3 and 4)

Excessive daytime drowsiness

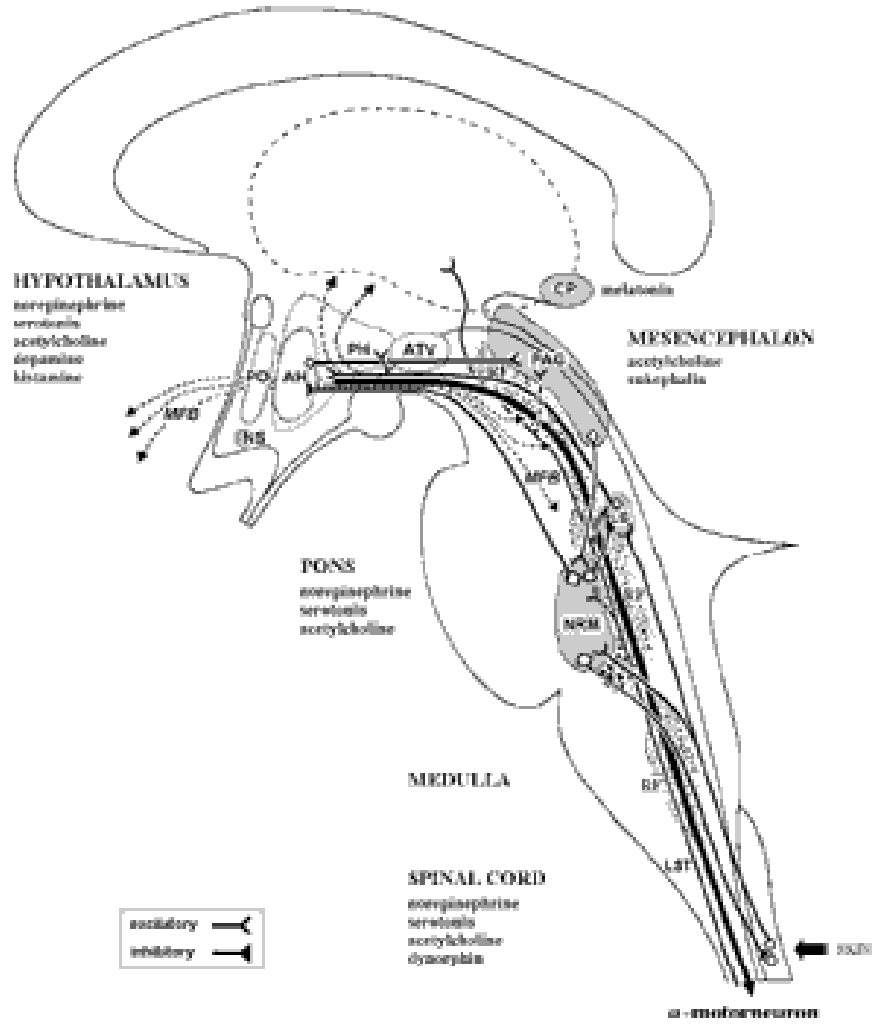


Hypothalamic Dysregulation
Temperature

"Neuro sweats"

"Hypothalamic storms"

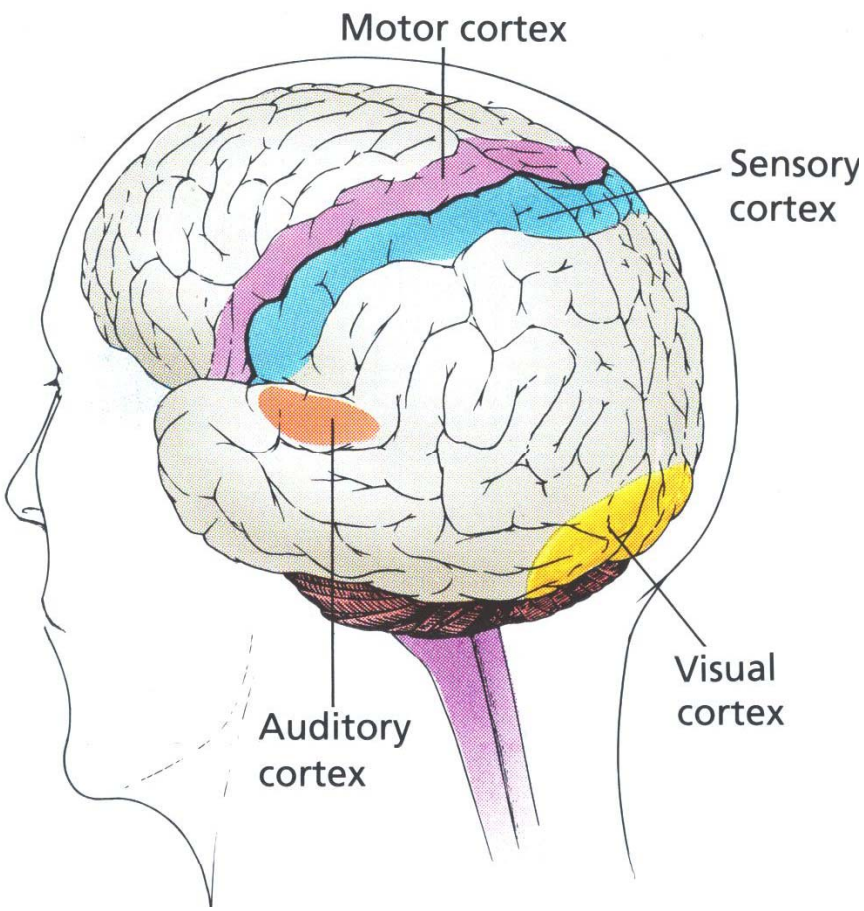
Subjective coldness



Neuromuscular & skeletal syndromes

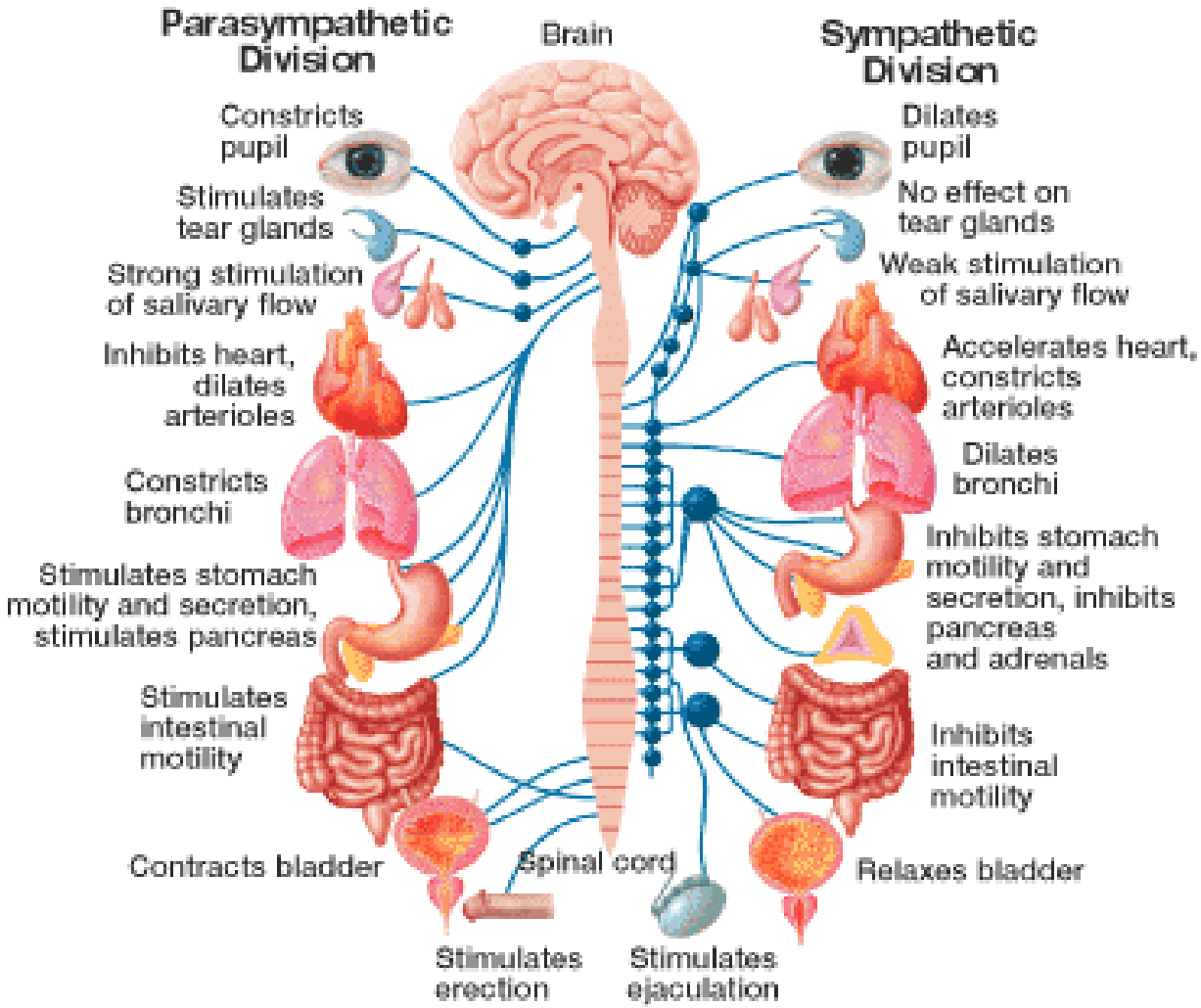
Musculoskeletal

- Spasticity
- Contractures
- Flacidity
- Tremor
- Extrapyramidal
- Pressure points
- Heterotopic ossification
- Myositis ossicans



Secondary autonomic dysfunction

Autonomic Nervous System



Autonomic Dysfunctions

Cardiovascular

Dysphonia

Infections

Pneumonia

Gastric emptying

Ileus

GERD

Bladder dysfunction

Impotence

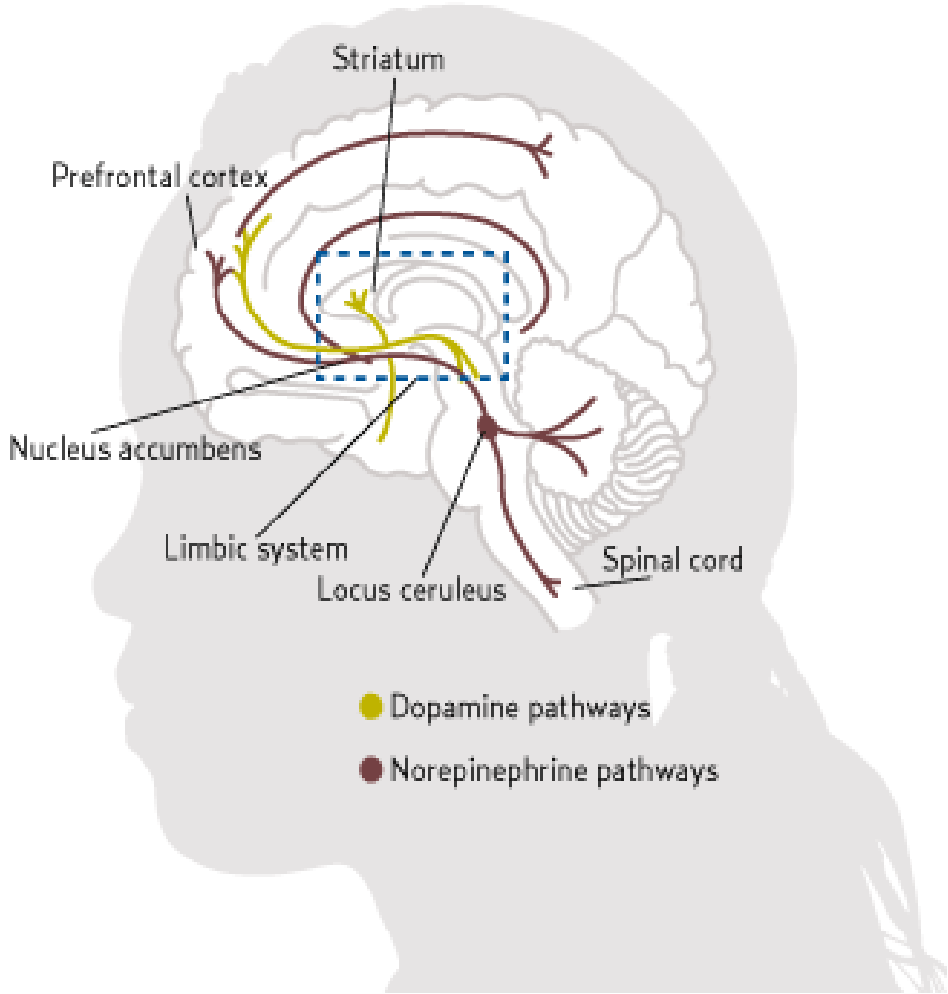
Neurocognitive dysfunction

Neurocognitive

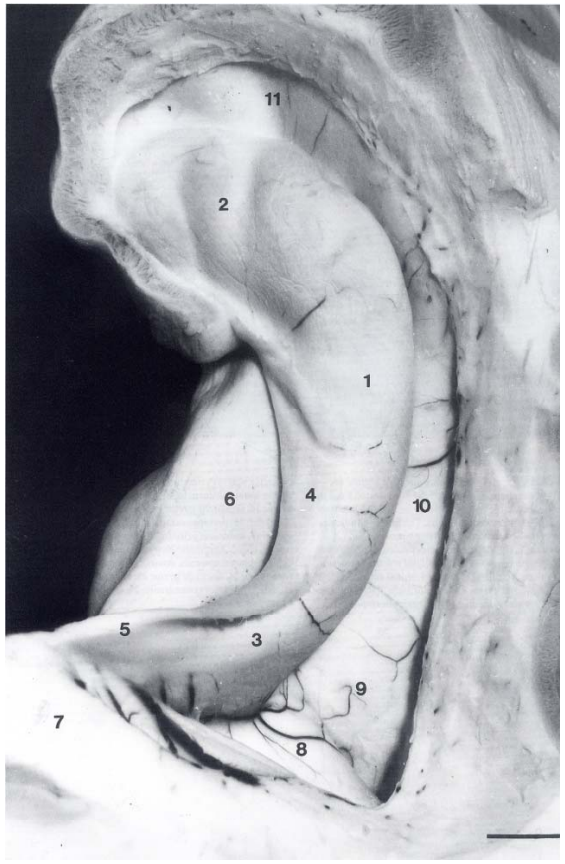
Attention/Concentration

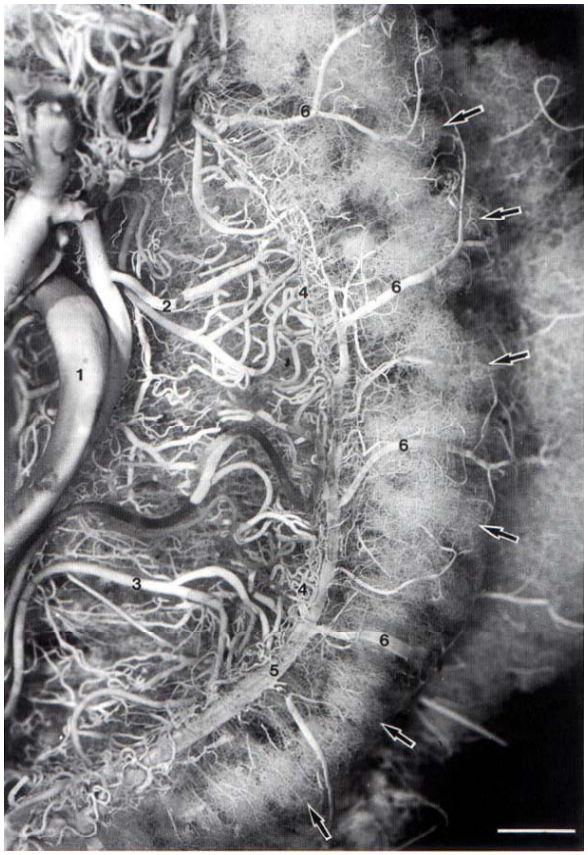
Working memory

Speed of information processing



Hippocampus





AJNR Am J Neuroradiol 24:1049–1056, June/July 2003

Diffuse Axonal Injury

Diffuse Axonal Injury Associated with Chronic

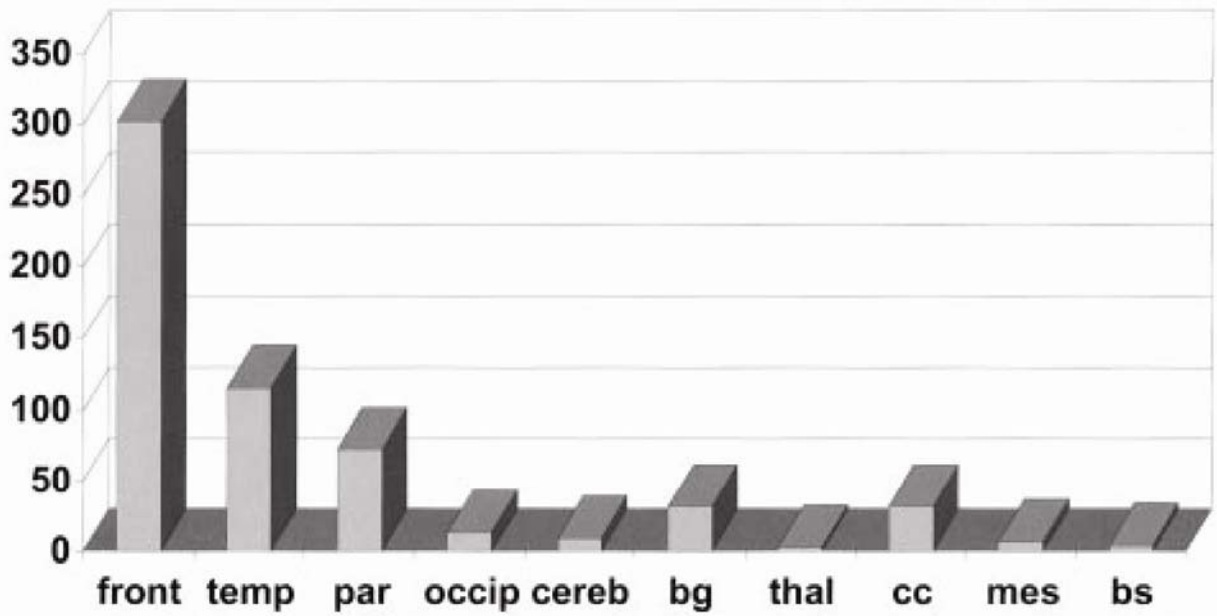


FIG 1. Frequency and site of traumatic microbleeds according to 10 brain areas. Shown is the total number of traumatic microbleeds in each brain area (*front*, frontal lobe; *temp*, temporal lobe; *par*, parietal lobe; *occip*, occipital lobe; *cereb*, cerebellum; *bg*, basal ganglia; *thal*, thalamus; *cc*, corpus callosum; *mes*, mesencephalon; *bs*, brain stem).

CONCLUSION: T2*-weighted gradient-echo imaging at high field strength is a useful tool for the evaluation of diffuse axonal injury during the chronic stage of traumatic brain injury. Diffuse axonal injury-related brain lesions are mainly hemorrhagic. The relevance of diffuse axonal injury for long-term clinical outcome is uncertain.

Guide to Brain Anatomy and Function

Frontal Lobe

Function: Integral to personality, involved in tracking, sense of self, arousal, and awareness of environment. Responsible for executive functioning & judgments, emotional response & stability, language, and memory for habit

Symptoms of Impairment

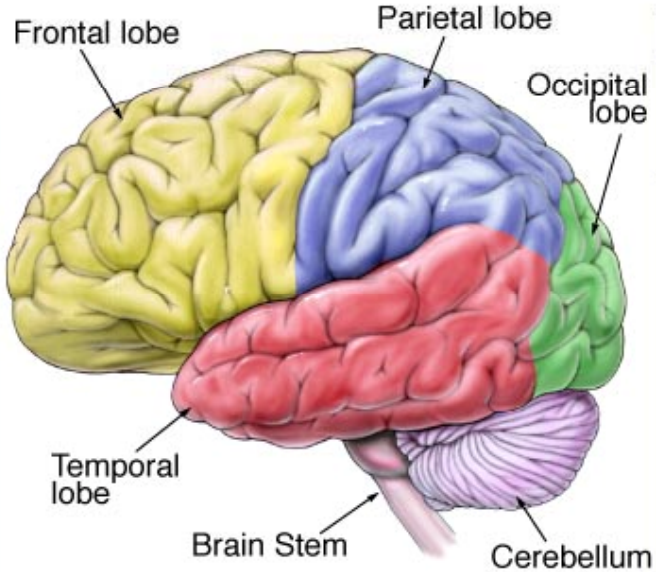
- Changes in personality & social behavior
- Loss of spontaneity in interacting with others
- Sequencing (difficulty planning and completing complex tasks in correct order)
- Perseveration (repeating same actions and comments over without conscious awareness of having done so)
- Loss of flexibility in thinking (mental rigidity)
- Distractibility
- Mood swings
- Diminished abstract reasoning
- Difficulty with problem solving
- Language difficulties (usage and word finding)
- Loss of simple movement of various body parts

Brain Stem

Function: Plays role in heart rate, swallowing, reflexes to sight and sound, sweating, blood pressure, digestion, temperature levels of alertness, ability to sleep, and balance

Symptoms of Impairment

- Decreased vital capacity in breathing
- Swallowing food & water
- Difficulty with organization/perception of environment
- Problems with balance and movement
- Dizziness and nausea
- Sleeping difficulties



Temporal Lobes

Function: Play key role in intellect, as well as auditory perception (hearing), long-term memory and some visual perception

Symptoms of Impairment

- Difficulty remembering names and faces
- Difficulty understanding spoken words
- Difficulty with identification of, and verbalization about objects
- Difficulty with concentration
- Short-term memory loss
- Interference with long-term memory
- Aggressive behavior
- Change in sexual interest
- Persistent talking (right lobe damage)
- Difficulty locating objects in environment
- Inability to categorize objects
- Religiosity
- Seizure disorders, auras and strange reveries

Parietal Lobes

Function: Involved in visual perception, tactile (touch) perception, integration of sensory information that allows for understanding of concepts, and goal-directed voluntary movements

Symptoms of Impairment

- Difficulties naming objects
- Difficulties writing words
- Inability to attend to more than one object at a time
- Inability to focus visual attention
- Problems with reading
- Poor hand-eye coordination
- Confusion left-right orientation
- Difficulty performing math calculations
- Difficulty drawing
- Poor visual perception
- Lack of awareness of certain body parts and/or surrounding space

Occipital Lobe

Function: Visual perception system

Symptoms of Impairment

- Impaired vision

Cerebellum

Function: Involved in coordination and control of voluntary movement, balance and muscle tone

Symptoms of Impairment

- Tremors
- Nystagmus (Involuntary movement of eye)
- Ataxia, lack of coordination
- Weak muscles (hypotonia)
- Inability to judge distance and when to stop (dysmetria)
- Inability to perform rapid altering movements
- Slurred speech (ataxic dysarthria)

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Neurolinguistic dysfunction

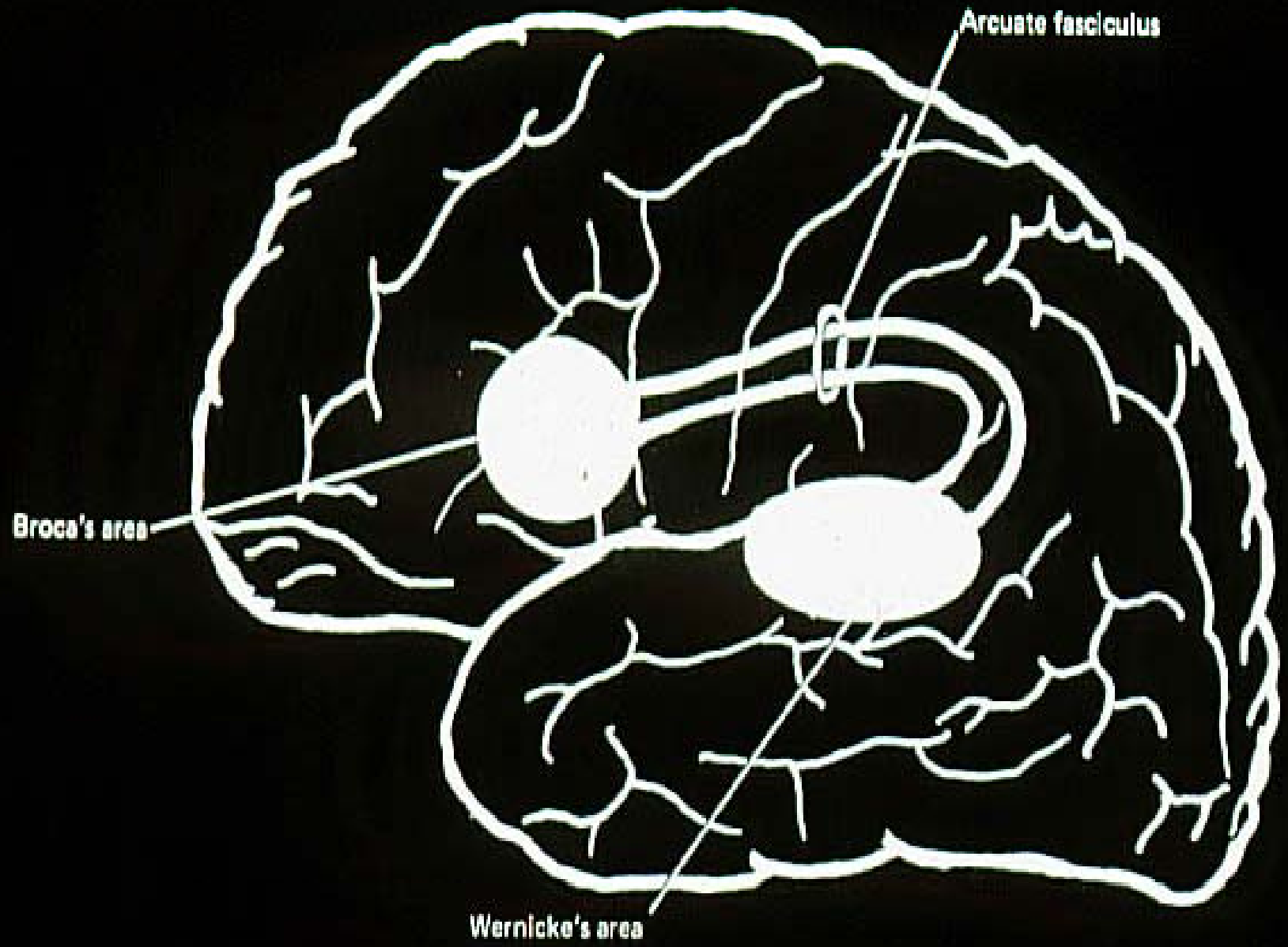
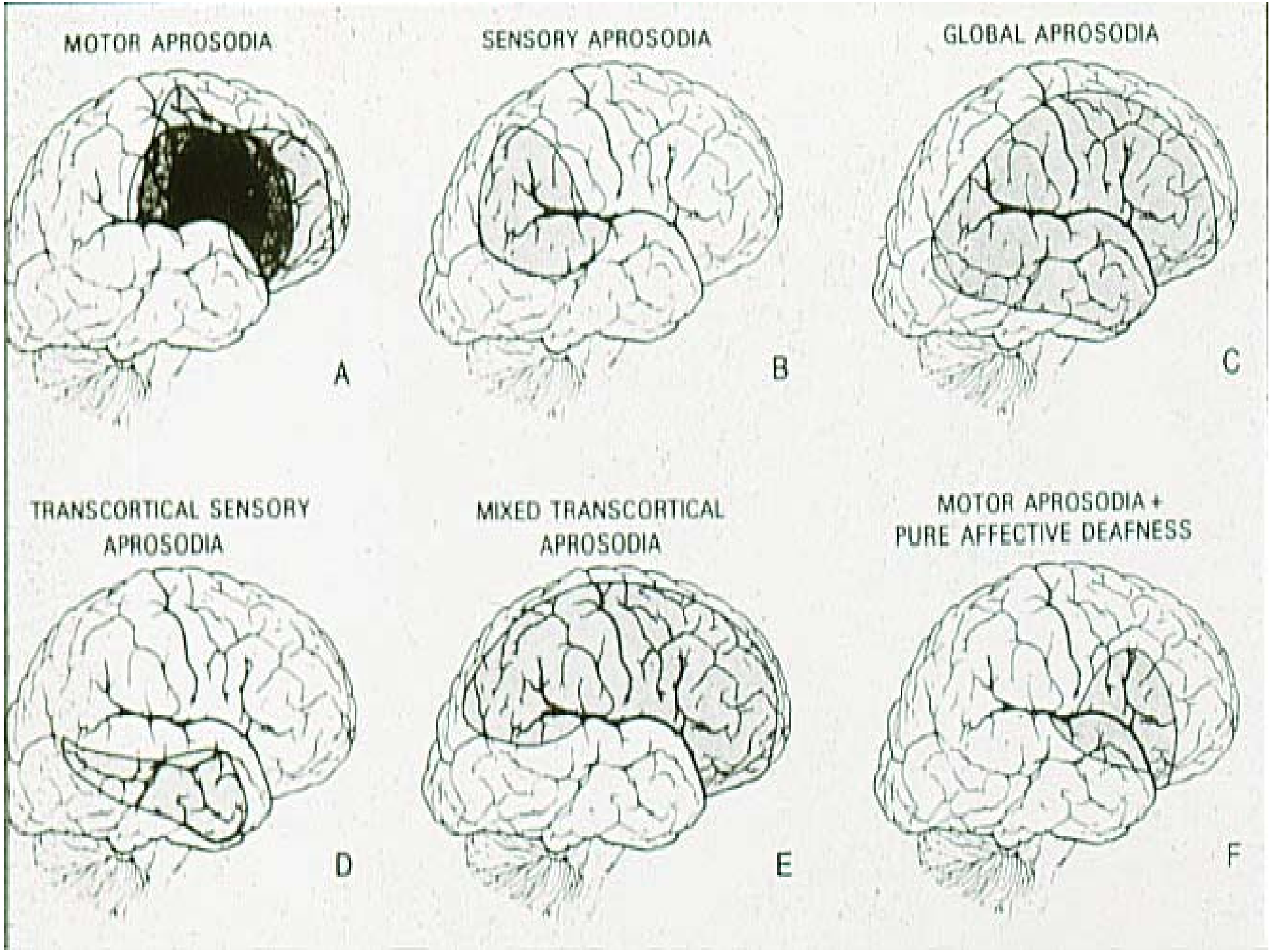
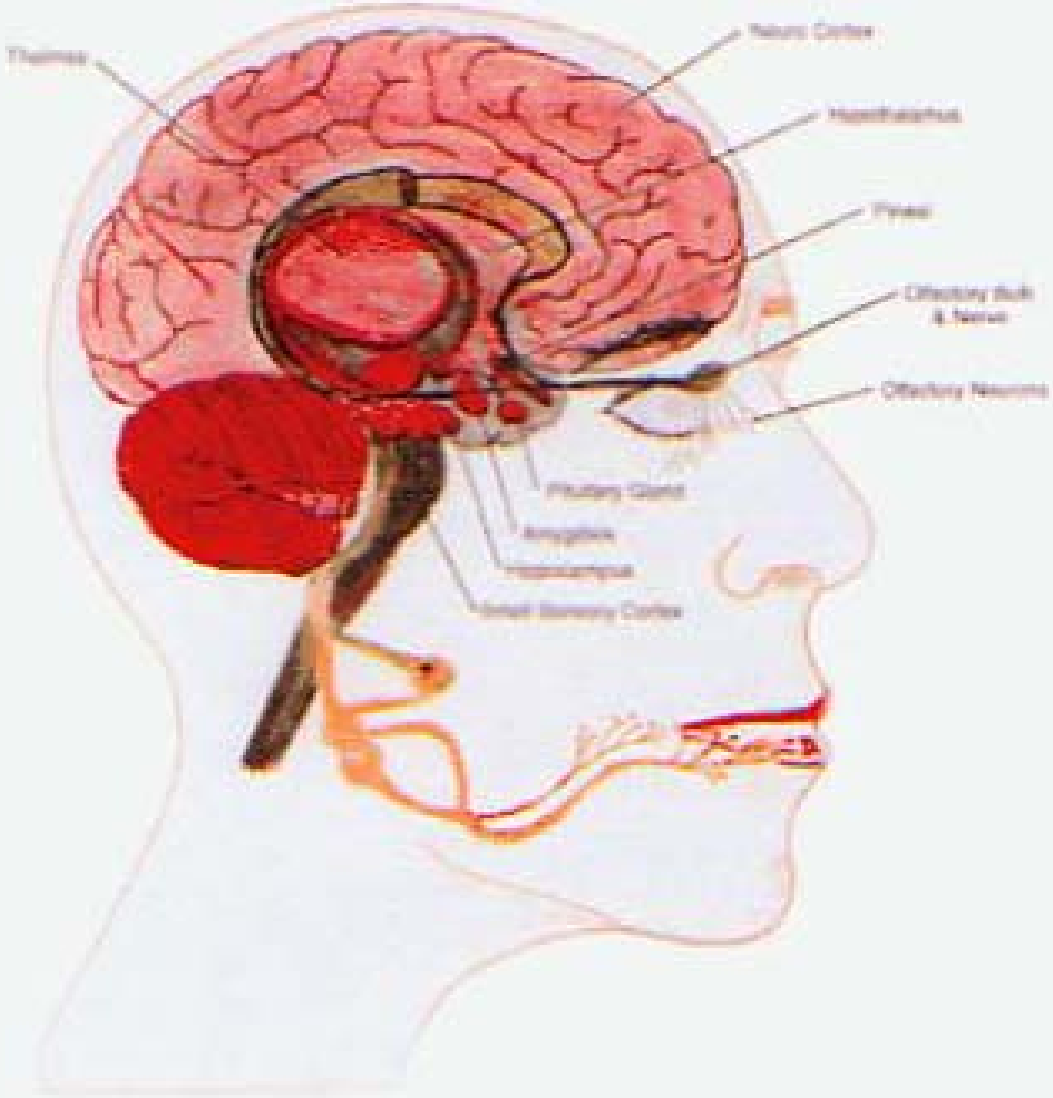


Figure 10–10. Perisylvian language circuitry.


MOTOR APROSODIA
A
SENSORY APROSODIA
B
GLOBAL APROSODIA
C
TRANSCORTICAL SENSORY APROSODIA
D
MIXED TRANSCORTICAL APROSODIA
E
MOTOR APROSODIA + PURE AFFECTIVE DEAFNESS
F

Neurobehavioral dysfunction

Limbic System
Smell and Emotional Responses



Neurobehavioral Consequences

Abulia

Depression

Suicide

Mania

Psychosis

Hallucinations

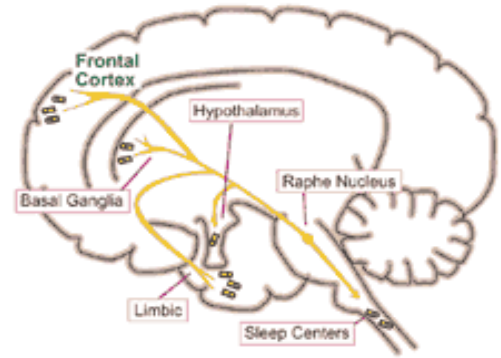
Anxiety

Personality change

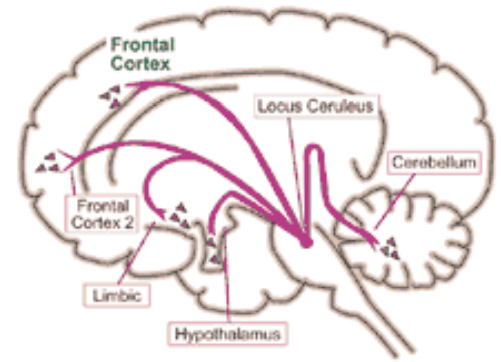
Substance abuse

Neurodegenerative

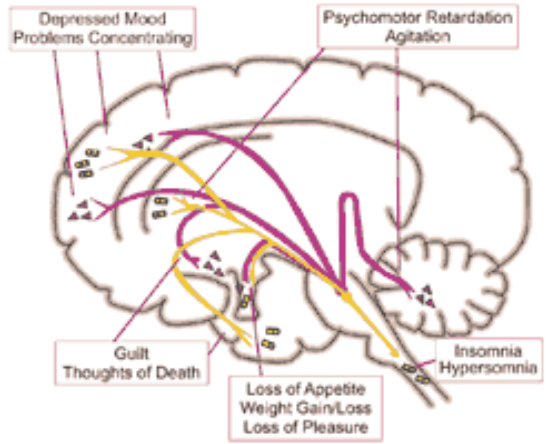
A. Serotonin Pathways in the Brain



B. Norepinephrine Pathways in the Brain



C. Symptoms Associated With Serotonin and Norepinephrine Pathways



TBI vs Community Base rates: Axis I

	Pre TBI	Post TBI	Community[1]
Major Dep.	17%	58%	6%
S/U Disorder	38%	16%	17%
PTSD	6%	18%	8%
OCD	1%	14%	3%
Panic	4%	11%	2%
GAD	1%	8%	4%
Phobias	4%	6%	13%

[1]Bourdon et al (1992)

Neurodegenerative disorders

Neurodegenerative

Encephalomalacia

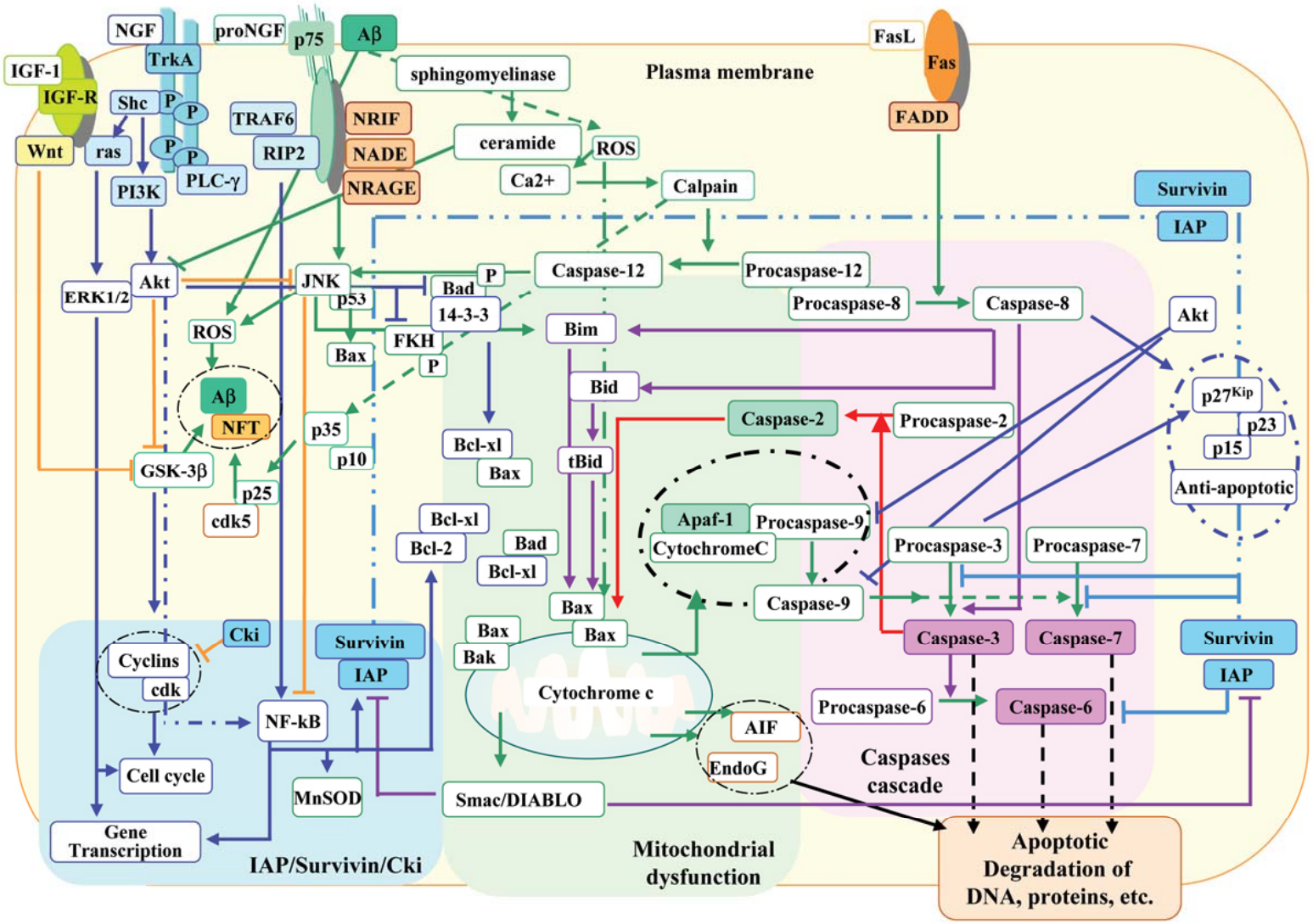
Alzheimers Disease

- Moderate and severe TBI confer increased risk
- Multiple mild TBI appears to increase risk
- APOE-4 genotype may play a role

Demyelinating

- ?Increased frequency of MS in females

Apoptosis cascade



Special populations

Infants

Children

Intimate partner violence

Recurrent TBI

Military personnel/veterans

Elderly

Developmentally disabled/Intellectually disabled

Prisoners

Glasgow Coma Scale

1974

Graham Teasdale &
Bryan J. Jennett

Outcome studies in coma

Glasgow Coma Scale	
Motor Responses:	Score
Obeys commands	6
Localizing responses to pain	5
Generalized withdrawal to pain	4
Flexor posturing to pain	3
Extensor posturing to pain	2
No motor response to pain	1
Verbal Responses:	
Oriented	5
Confused conversation	4
Inappropriate speech	3
Incomprehensible speech	2
No speech	1
Eye opening	
Spontaneous eye opening	4
Eye opening to speech	3
Eye opening to pain	2
No eye opening	1
TOTAL (Add above 3 scores)	Range = 3 - 15

Table 1

Pediatric GCS

Table 1 **Modified Glasgow coma score**

Score	Response	Response	Response
Eye opening	> 1 year	0–1 year	
4	Opens spontaneously	Opens spontaneously	
3	Opens to a verbal command	Opens to a shout	
2	Opens in response to pain	Opens in response to pain	
1	No response	No response	
Best motor response	> 5 years	2–5 years	0–23 months
5	Oriented and able to converse	Uses appropriate words	Cries appropriately
4	Disoriented and able to converse	Uses inappropriate words	Cries
3	Uses inappropriate words	Cries and/or screams	Cries and/or screams inappropriately
2	Makes incomprehensible sounds	Grunts	Grunts
1	No response	No response	No response
Best verbal response	> 1 year	0–1 year	
6	Obeys command	Spontaneous	
5	Localizes pain	Localizes pain	
4	Flexion withdrawal	Flexion withdrawal	
3	Flexion abnormal (decorticate)	Flexion abnormal (decorticate)	
2	Extension (decerebrate)	Extension (decerebrate)	
1	No response	No response	

Diffusion tensor imaging of acute mild traumatic brain injury in adolescents

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ABSTRACT

Background: Despite normal CT imaging and neurologic functioning, many individuals report postconcussion symptoms following mild traumatic brain injury (MTBI). This dissociation has been enigmatic for clinicians and investigators.

Methods: Diffusion tensor imaging tractography of the corpus callosum was performed in 10 adolescents (14 to 19 years of age) with MTBI 1 to 6 days postinjury with Glasgow Coma Scale score of 15 and negative CT, and 10 age- and gender-equivalent uninjured controls. Subjects were administered the Rivermead Post Concussion Symptoms Questionnaire and the Brief Symptom Inventory to assess self-reported cognitive, affective, and somatic symptoms.

Results: The MTBI group demonstrated increased fractional anisotropy and decreased apparent diffusion coefficient and radial diffusivity, and more intense postconcussion symptoms and emotional distress compared to the control group. Increased fractional anisotropy and decreased radial diffusivity were correlated with severity of postconcussion symptoms in the MTBI group, but not in the control group.

Conclusions: In adolescents with mild traumatic brain injury (MTBI) with Glasgow Coma Scale score of 15 and negative CT, diffusion tensor imaging (DTI) performed within 6 days postinjury showed increased fractional anisotropy and decreased diffusivity suggestive of cytotoxic edema. Advanced MRI-based DTI methods may enhance our understanding of the neuropathology of TBI, including MTBI. Additionally, DTI may prove more sensitive than conventional imaging methods in detecting subtle, but clinically meaningful, changes following MTBI and may be critical in refining MTBI diagnosis, prognosis, and management. ***Neurology*® 2008;70:948-955**

Case Reports

Diffusion Tensor Magnetic Resonance Imaging of Microstructural Abnormalities in Children with Brain Injury

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Disclosures:

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DOI: 10.1097/01.PHM.0000073830.15643.6A

ABSTRACT

Lee ZI, Byun WM, Jang SH, Ahn SH, Moon HK, Chang Y: Diffusion tensor magnetic resonance imaging of microstructural abnormalities in children with brain injury. *Am J Phys Med Rehabil* 2003;82:556–559.

We present two pediatric cases demonstrating that diffusion tensor imaging is more efficient at revealing microstructural abnormalities of the brain than conventional magnetic resonance imaging because it enables measurements of the directionality and integrity of white matter fiber tracts. One patient suffered from left hemiparesis, and the other had right hemiparesis. However, whereas conventional magnetic resonance imaging showed only the findings of traumatic contusional hemorrhages in the left temporal and parietal lobes of the first patient and focal encephalomalacia in the left anterior thalamus of the second patient, diffusion tensor imaging successfully disclosed microstructural abnormalities in the right cerebral peduncle of the midbrain of the first patient and in the posterior limb of the left internal capsule of the second. Theses two cases demonstrate that diffusion tensor imaging is more capable than magnetic resonance imaging at detecting the microstructural pathologic lesions that are responsible for clinical motor weakness, especially when conventional magnetic resonance imaging has failed to detect subtle structural abnormalities.

Key Words: Diffusion Tensor Imaging, Microstructural Abnormality, Pediatric, Brain Injury, Rehabilitation

ELSEVIER

Archives of Clinical Neuropsychology 22 (2007) 555–568

Archives of CLINICAL NEUROPSYCHOLOGY

Neurocognitive and neuroimaging correlates of pediatric traumatic brain injury: A diffusion tensor imaging (DTI) study

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Accepted 14 March 2007

Abstract

This study examined the sensitivity of diffusion tensor imaging (DTI) to microstructural white matter (WM) damage in mild and moderate pediatric traumatic brain injury (TBI). Fourteen children with TBI and 14 controls ages 10–18 had DTI scans and neurocognitive evaluations at 6–12 months post-injury. Groups did not differ in intelligence, but children with TBI showed slower processing speed, working memory and executive deficits, and greater behavioral dysregulation. The TBI group had lower fractional anisotropy (FA) in three WM regions: inferior frontal, superior frontal, and supracallosal. There were no group differences in corpus callosum. FA in the frontal and supracallosal regions was correlated with executive functioning. Supracallosal FA was also correlated with motor speed. Behavior ratings showed correlations with supracallosal FA. Parent-reported executive deficits were inversely correlated with FA. Results suggest that DTI measures are sensitive to long-term WM changes and associated with cognitive functioning following pediatric TBI.
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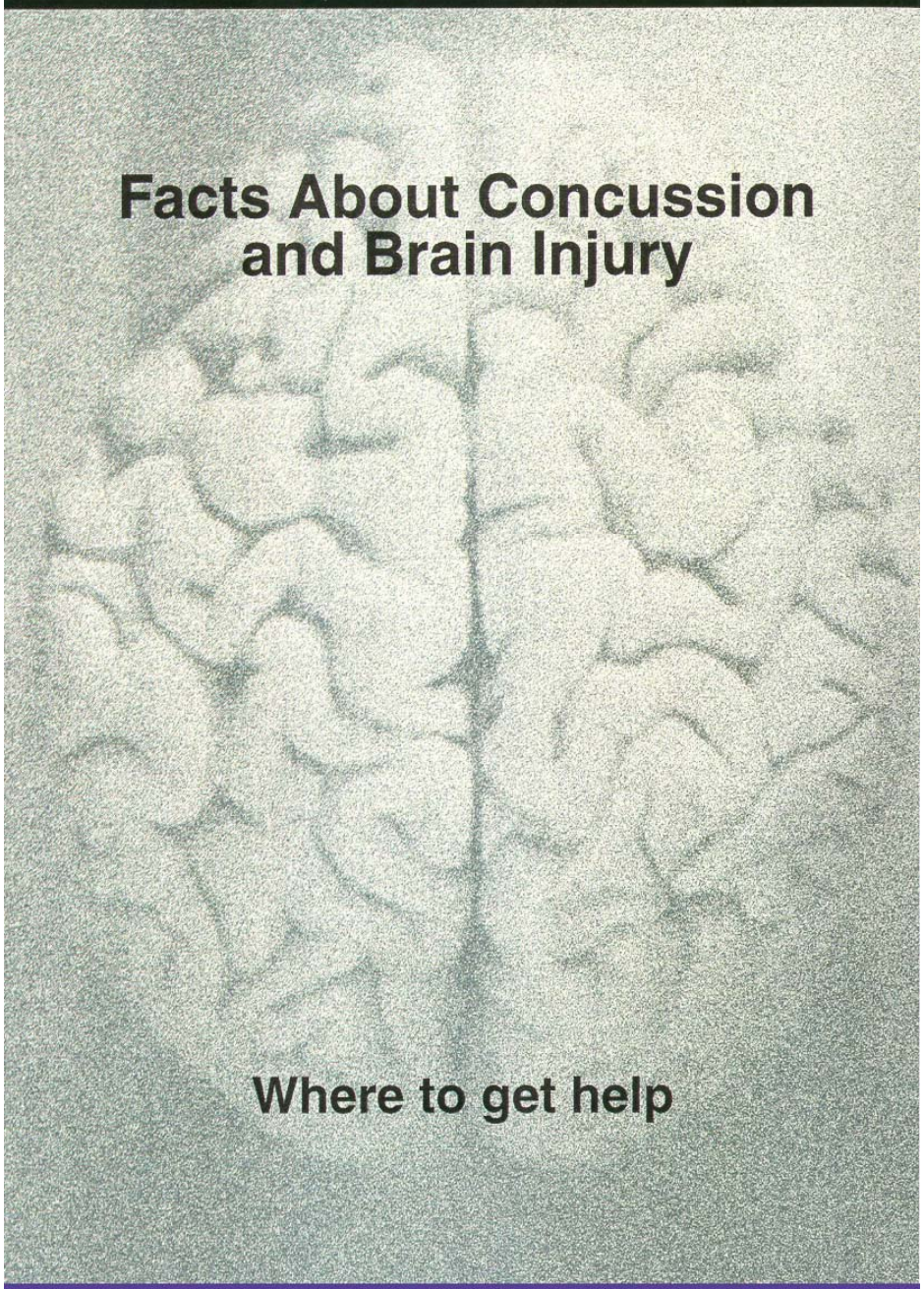
Keywords: Traumatic brain injury (TBI); Diffusion tensor imaging (DTI); White matter; Pediatric

Facts About Concussion and Brain Injury



Where to get help

Keep this...

Margaret Kennard, 1936

- Child's brain was incredibly resilient to trauma, much more "plastic" than the adult brain and could grow new cells and circuits that enabled the child to overcome the effects of TBI.
- Based on very little evidence.
- Published in the American Journal of Physiology and is known as the "Kennard Principle."
- Chimpanzees with focal lesions in the motor cortex
- Found that younger chimps more likely to recover their ability to perform skilled action than older ones.
- **Attributed to the younger chimps were still growing. She developed this into a universal principle that she applied to human children and adults.**
- Long term follow up showed that younger chimps described as fully recovered developed spasticity & motor control deficits later as brains matured & more complex motor tasks were needed.

Outcome: Age at injury

Poorer cognitive & academic outcomes <8yo

(Taylor & Alden, 1997; Verger, et al, 2000)

More effects on post-TBI development

(Anderson, et al., 2005a; Ewing-Cobbs, et al., 2004a)

Expressive language & academics

(Ewing-Cobbs & Barnes, 2002)

Word decoding: TBI <6 ½ yo worse than >6 ½

Reading comprehension: TBI <9 worse than >9

(Barnes, et al., 1999)

Pediatric (moderate & severe) TBI Issues

- Damage to frontal lobe networks will not be seen until middle school/high school
- TBI < 4 yo → Dx of Conduct Disorder by 12 yo
- Girls with TBI → increased risk of victimization
- Boys with TBI → increased risk of risk taking and disciplinary problems
- Never catch up with non-injured agemates

Adult survivors of Ped TBI

Educational failure

Restricted vocational options

Psychological adjustment difficulties

Poor quality of life

(Anderson et al, 2009; Cattelani et al, 1998; McKinlay et al, 2002)

Special populations

Infants

Children

Intimate partner violence

Recurrent TBI (sports)

Military personnel/veterans

Elderly

Developmentally disabled/Intellectually disabled

Prisoners

Intimate partner violence

- Victim with concussion (or multiple) may appear hesitant, give poor history, or be amnestic for the assault
- Substance abuse (self-medication) co-morbidity issues
- Increased irritability/agitation may be sx of TBI

TBI in Prisoners

- 25-87% of inmates have h/o TBI vs 8.5% gen'l population
- More likely to have disciplinary problems in jail
- Inmates with >1 TBI have increased SA in year preceding incarceration
- Females convicted of violent crime more likely to have h/o TBI
- Children/teens convicted more likely h/o TBI

TBI in Active Duty or Veterans

- Frontal lobe injuries mimic depression (apathy) or sociopathy (impulsivity/poor planning)
- Reduced initiation may be seen as laziness
- Loss of external structure of military/garrison life requires increased self-direction as civilian
- Risk-taking behavior is generally incompatible with combat survival

TBI in Cognitively Vulnerable Individuals

- Diminished cerebral reserve pre-injury worsens outcome
- If your brain is not “wired to code” pre-injury, recovery is more prolonged & less robust
- Psychosocial support more critical in data collections (Think: Pediatric approach)

General Principles in Dealing with patients with h/o TBI

1) Attention deficits make it hard to focus on questions or respond to directions
2) Memory impairment makes obtaining detailed history (STM) difficult
3) Delays in processing speed increases the time it takes to respond to questions or directions(especially with increased environmental chaos)
4) Impulsivity or disinhibition results in inappropriate comments or behavior
5) Irritability & poor problems solving issues
6) Get information from a collateral informant

   (think: “pediatric” approach)

THANK YOU
(see you tomorrow!)