TBI: Clinical Issues, Controversies, and Learning from Patients

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DVHIP

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What is Neuropsychology?

- the study of brain-behavior relationships
- employs a standardized, normative approach to describing behavior, cognition, and emotion.
- NOT a set of techniques
- there a NO "neuropsychological" tests or techniques

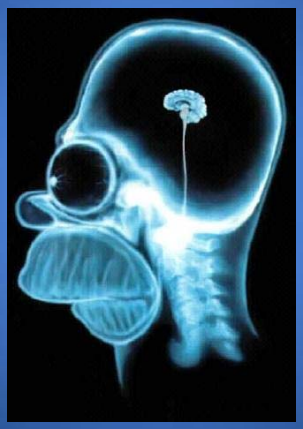
Neuropsychological Evaluations are an assessment process, not just testing.

Vanderploeg. R. (2000)

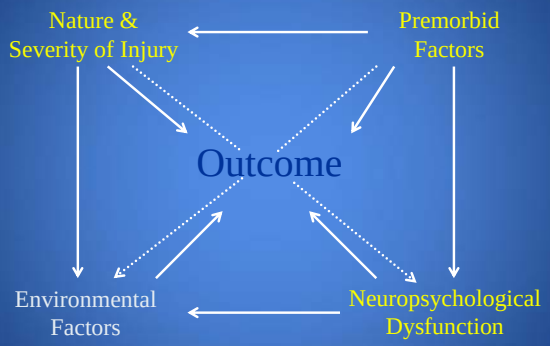
"…it is a way of thinking about behavior, often expressed as test scores; in essence, it is a paradigm for understanding behavior."

Cecil R. Reynolds & Joan W. Mayfield (2011)

Object of Investigation



TBI: An Interactional Model



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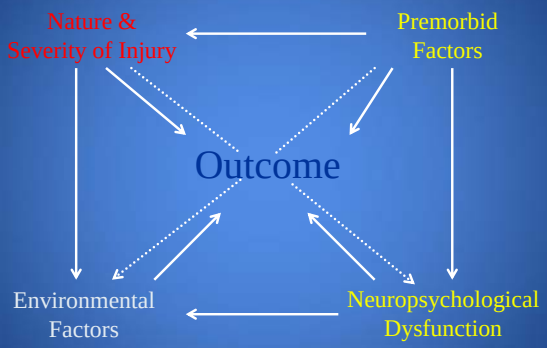

Neuropathology of TBI

While TBI is characterized by substantial heterogeneity in its pathophysiology, it can be viewed as generally resulting in frontal-temporal damage superimposed on more diffuse pathology

- Orbitofrontal and anterior temporal regions are particularly susceptible to damage.
- Research has demonstrated this pattern to be independent of site of impact.

What is Traumatic Brain Injury (TBI)?

Traumatic brain injury is damage to the brain caused by externally acting physical forces.

- Injury is acquired (not developmental or congenital), and happens suddenly
- Usually associated with some period of altered or loss of consciousness
- Types of TBI
  - Focal
  - Diffuse

Stages of Brain Injury

Typically occurs in two stages:

- ***Primary injury***: damage that occurs at the time of impact
- ***Secondary injury***: damage caused by the physiological processes which are activated by the primary injury.
  - Compromise of cerebral circulation from compression of brain due to brain swelling, hematomas, ↑ intracranial pressure, herniation
  - Cellular: release of cytotoxic substances, change in neurotransmitter levels

Mechanisms

- Acceleration/Deceleration
- Blow/Blunt trauma
- Penetrating
- Static

Severity of TBI

Severity Grades of TBI		
Mild (Grade 1)	**Moderate (Grade 2)**	**Severe (Grade 3 & 4)**
Altered or LOC < 30 min with normal CT &/or MRI	LOC < 6 hours with abnormal CT &/or MRI	LOC > 6 hours with abnormal CT &/or MRI
GCS 13-15	GCS 9-12	GCS < 9
PTA < 24 hours	PTA < 7 days	PTA > 7 days

Key Word: "Heterogeneity"

TBI is not only characterized by substantial variability in pathophysiology, but in the problems survivors experience and in the ultimate outcomes they attain.

Points to Remember

- There is no typical pattern of deficits associated with TBI because of the complexity and individual variation of the brain and variations in mechanisms of injury and the results of the trauma
- Specific impairments in function may be seen in behaviors that are strongly localized to or heavily dependent on particular areas of the brain
- Impairments in executive abilities and motor abilities are frequently seen with damage to the frontal lobes
- Consolidating new information into memory may be impaired with damage to the temporal lobes, especially damage to the hippocampal area
- Difficulties in language comprehension or comprehension of visual stimuli are seen with lesions to the parietal lobes
- Damage to the occipital lobes may produce deficits in visual abilities such as cuts in the visual field or cortical blindness
- Diffuse axonal injury is associated with slowed thinking and difficulty accomplishing tasks

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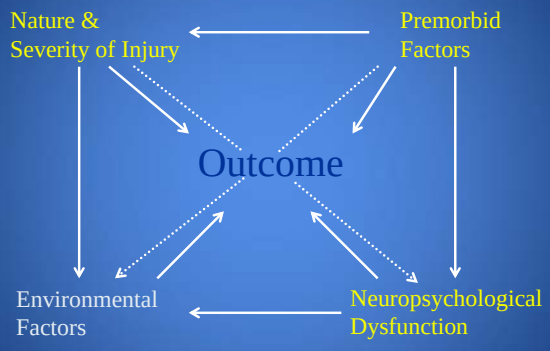

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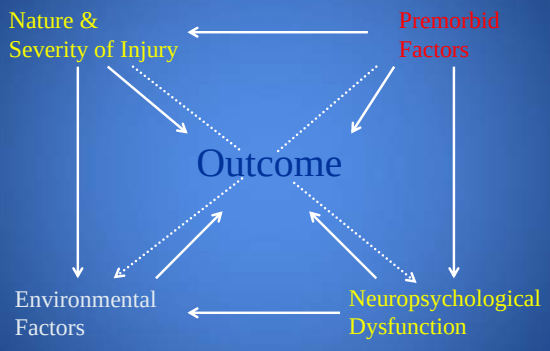

Premorbid Risk Factors

- After one TBI, there is a 3-fold increase in the probability for a second injury
- After a second TBI, the risk for a third TBI is 8-times greater
- Males are 2-times as likely as females to sustain TBI
  - differences in risk exposure and lifestyle
  - males aged 14 to 24 years are at highest risk

Premorbid Risk Factors (cont.)

Predominant risk factor.....?

Alcohol

Premorbid Risk Factors (cont.)

- > 50% of persons with TBI were intoxicated at the time of injury.
- One study (N = 14,920) of consecutive patients with diagnosis of traumatic injury seen in a large, metropolitan hospital's E.R.
  - head injury was a more common finding among intoxicated (64%) than sober (18%) patients,
  - alcohol increased the probability of TBI in every major cause of trauma except self-inflicted.

Premorbid Risk Factors (cont.)

- Hx of alcohol dependence has been found in a large portion of TBI patients, ranging from 25% to 68%.
- Literature indicates that chronic alcohol abuse potentiates the mechanical and neurochemical process involved in TBI.

Premorbid Risk Factors (cont.)

Age

- Negatively associated with recovery
- However, "the younger you are the better off you are" is not always true
  - impairment of function vs. impairment in the acquisition of function

Other Premorbid Factors

- Premorbid Intelligence
  - Positively associated with outcome
  - "Brain Reserves" Hypothesis
- Education
  - Positively associated with outcome
  - Confounded by factors such as level of intelligence which is positively associated with education

Other Premorbid Factors (cont.)

- Psychiatric Issues
  - Comorbidity with alcohol dependence
  - Personality Traits
  - Disorders of Impulse Control (ADHD, BP)
- Coping skills
  - "Hardiness"
  - Prior experience using compensatory strategies

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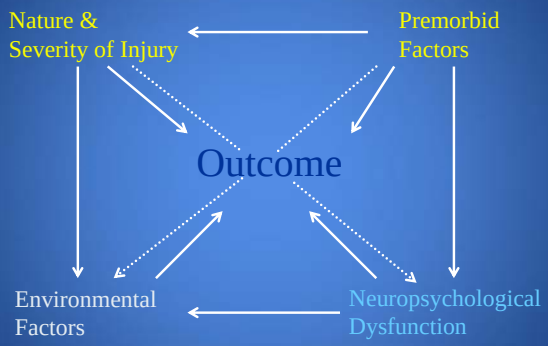

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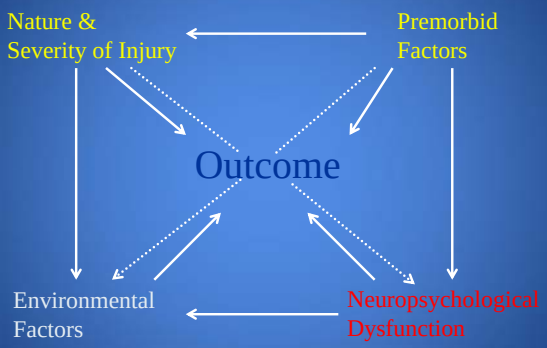

Prominent Sequelae of TBI

- Attention, Memory, and Learning
- Executive Control Functions
- Personality Changes

Memory and Learning

- Intact memory of old, previously learned information and over-learned, deeply ingrained skills
  - "Crystallized" Abilities
- Major impairments in registering, storing and retrieving **new** information
  - "Fluid" abilities

Executive Control Functions

- Reasoning, problem solving and conceptualizing
- Abstraction
- Planning and organization
- Foresight (anticipation)
- Flexibility/adaptability
- Self-awareness, self-regulation, self-control

Whole Brain Function

- The prefrontal cortex integrates and acts upon all sources of information
  - ❖external/internal
  - ❖memory stores
  - ❖visceral arousal centers
- TBI impairs the integration of the perception of the emotional relevance of a stimulus with the interpretation of the meaning of that stimulus

Personality Changes

- Emotional instability
- Impulsivity
- Disinhibition
- Restlessness
- Immaturity, Egocentrism
- Irritability
- Anxiety/Depression
- Apathy, Loss of initiative
- Decreased frustration tolerance

Common Myths/Pitfalls

- No loss of consciousness means no TBI
- Mild concussion means no impairments
- Recovery occurs in one year
- Normal neurological evaluation means no TBI
- Normal CT or MRI, means no TBI
- Normal IQ means no TBI
- Residual deficits indicate malingering
- Internal Attribution Errors: "lazy" vs. initiation deficit
- Use of inappropriate outcome criteria: "Walks and talks"

CTE: Summary

Proposition: Chronic Traumatic Encephalopathy (CTE) is a progressive tauopathy that results from repetitive, mild traumatic brain injury (TBI).

- Not a new concept:
  - "Classic" CTE (a.k.a., Dementia Pugilistica)
- "Modern" CTE
- Research on the Neuropathology
- Methodological Concerns

Summary

- High degree of media focus primarily based upon the work of two research groups
  - Robert Cantu, MD & Ann C. McKee, MD
    Center for Study of Traumatic Encephalopathy, Boston University
  - Bennet I. Omalu, MD, MPH
    - Univ Pittsburgh, U.C. Davis
    - Brain Injury Institute, West Virginia

Summary

- Increased focus of this issue has resulted in increased funding and increased personal injury litigation
- Lay and professional understanding is being shaped by highly emotional, individual cases vs. the state of our scientific knowledge
- Impacting public and private policy (e.g., NFL, NCAA, primary secondary sports)

"Classic" CTE

- Lack of well controlled studies complicate findings
  - Samples of convenience
  - Many variables not accounted for
    - Age-related changes
    - Pre-morbid psychiatric issues
    - Alcohol/Substance abuse
    - Other comorbid issues (e.g., other dementing illnesses, venereal disease)

"Modern" CTE

- Information largely from two independent research groups (McKee & Cantu et al.; Omalu et al.)
- Original publications were from autopsies of two retired NFL players (Omalu et al. 2005, 2006)
- Like DP, initial cases were from autopsies of brains donated from families concerned about players cognitive behavioral symptoms before dying and, more recently, by concerned retired athletes presenting for examination.

"Modern" CTE

- McKee et al. (2013)
  - Analysed 85 post-mortem brains with histories of "repetitive mild traumatic brain injury"
    - 21 were military veterans
    - 1 had self injurious head banging behavior
  - Found evidence of CTE in 68 (80%)

"Modern" CTE

	McKee	Omalu	Hazrati	NIH	Total	%
Num. Autopsy Cases	61	17	6	1	85	-
Pure CTE	15	2	0	0	17	20.0
CTE + Other Neuropathology	31	9	3	1	44	51.8
Neuropathology, No CTE	0	1	3	0	4	4.7
No Neuropathology	15	5	0	0	20	23.5

Gardner, Iverson, & McCrory (2014)

Learning from Extremes

- Blind but could "see"
- "I'm usually not this nice"
- Abulia and Susan B.
- No LOC, No TBI, No!
  - Medical School Bound
  - Master Sgt