

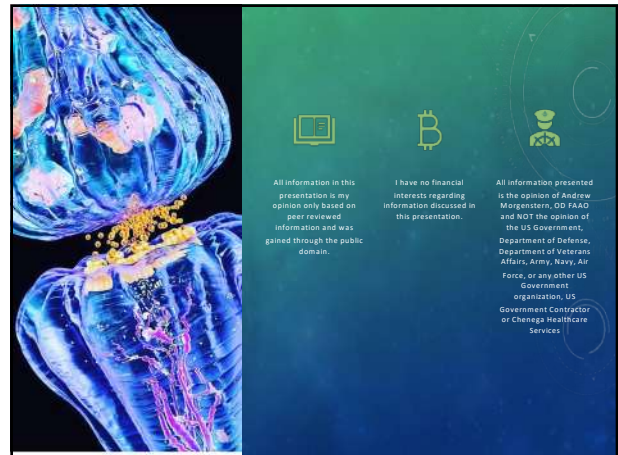
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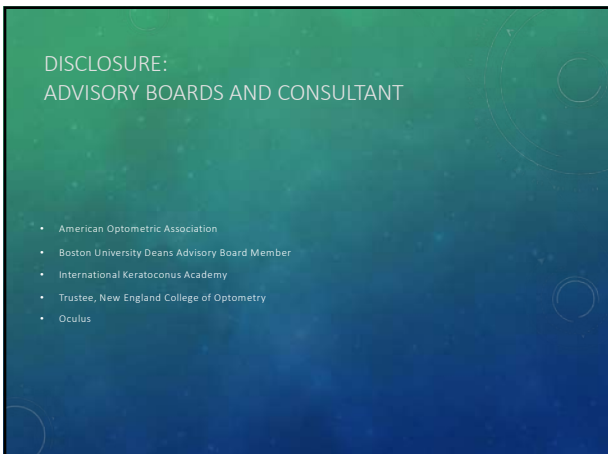
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NFL says regular-season concussions increased 18% in 2022

Kevin Seifert
ESPN Staff Writer
Feb 1, 2023


The NFL acknowledged Friday that concussions rose significantly during the 2022 regular season, a sobering outcome that aligned with a season-long public conversation about head injuries.

According to data released by the league, there were 149 concussions suffered over 271 games this season. That's an 18% jump from 2021 (126) and 14% higher than the three-year average (130) between 2018 and 2020.

NFL chief medical officer Dr. Allen Sills on Friday attributed the rise to a number of factors, including a protocol change that he said "broadened and strengthened" the definition of a concussion following a series of injuries suffered by Miami Dolphins quarterback Tua Tagovailoa.

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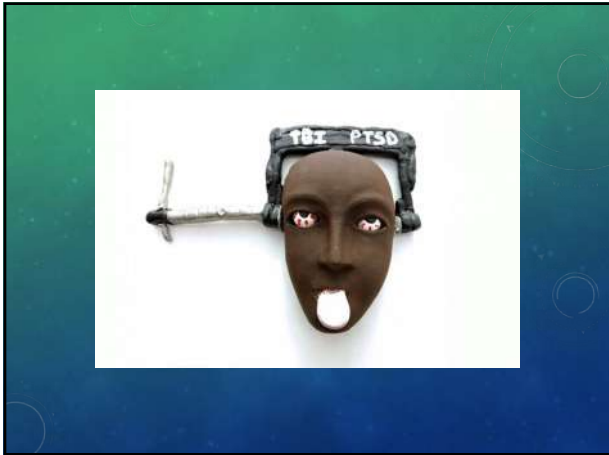
PURITY OF INFORMATION



"Don't believe everything you read on the Internet just because there's a picture with a quote next to it."

—Abraham Lincoln

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THE AOA AND OTHER ORGANIZATIONS HAVE DEVELOPED WONDERFUL GUIDES FOR YOU!


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Brain Injury Committee:

Chair *Christyna Rakoczy, O.D.*
 Past Chair *Kara Gagnon, O.D.*
 VRS Chair *Maria Santullo Richman, O.D.*
Allen Cohen, O.D.
Brenda Hainke Montecalvo, O.D.
Mitchell Scheiman, O.D.

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Mitchell Scheiman, O.D.



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AOA TBI TASK FORCE

- Trustee *Lori Latowski Grover, OD*
- *Keith Smithson, OD*
- *Carl Garbus, OD*
- *Amanda Nanasy, OD*
- *Eric Ikeda, OD*
- *DeAnn Fitz, OD*
- *Maria Richman, OD*
- *Michael Dueñas, OD*
- *Andrew Morgenstern, OD*



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AOA TBI TASK FORCE

“Doctors of optometry play a key role throughout the continuum of TBI care, from prevention and primary care to tertiary rehabilitative care,” she adds. “Advocacy to minimize the negative impacts of TBI, from clinical care to policy, must include optometrists who, as physicians, coordinate and collaborate care in conjunction with other doctors and health professionals to manage and treat TBI and its sequelae.” Trustee Lori L. Grover, OD



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CLINICAL RECOMMENDATION FOR THE EYE CARE PROVIDER
EYE AND VISION CARE FOLLOWING BLAST EXPOSURE AND/OR POSSIBLE TRAUMATIC BRAIN INJURY

Introduction and Background
 The use of military munitions in a combat setting is often associated with the potential for blast exposure to the head and neck. Blast waves, which are the result of the rapid expansion of air, can cause damage to the head and neck. Blast waves can also cause damage to the eyes. The purpose of this clinical recommendation is to provide eye care providers with information on the signs and symptoms of eye injury following blast exposure and to provide recommendations for the management of eye injury following blast exposure.

Clinical Recommendations
 This clinical recommendation is designed to help guide the management of eye injury following blast exposure. The recommendations are based on the current evidence and are intended to be used as a guide. The recommendations are based on the current evidence and are intended to be used as a guide. The recommendations are based on the current evidence and are intended to be used as a guide.

Summary of Argument of Case
 The purpose of this clinical recommendation is to provide eye care providers with information on the signs and symptoms of eye injury following blast exposure and to provide recommendations for the management of eye injury following blast exposure. The purpose of this clinical recommendation is to provide eye care providers with information on the signs and symptoms of eye injury following blast exposure and to provide recommendations for the management of eye injury following blast exposure.

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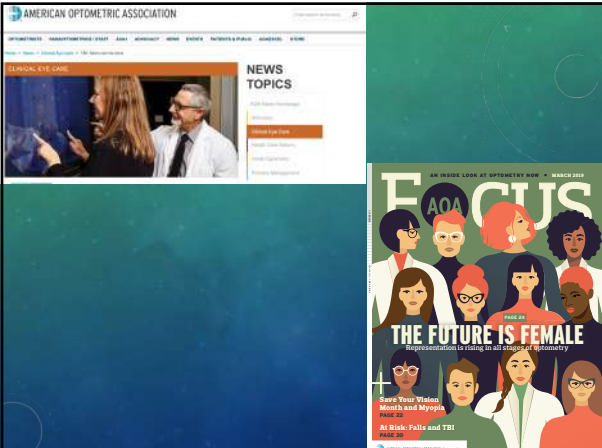
JAMA Pediatrics | Special Communication

Centers for Disease Control and Prevention Guideline on the Diagnosis and Management of Mild Traumatic Brain Injury Among Children

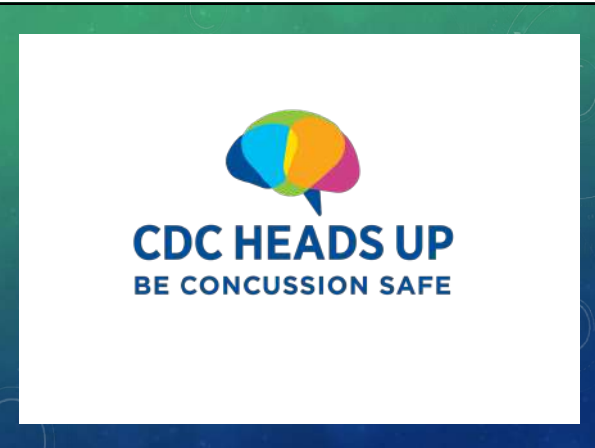
CONCLUSIONS AND RELEVANCE This guideline identifies the best practices for mTBI based on the current evidence; updates should be made as the body of evidence grows. In addition to the development of the guideline, CDC has created user-friendly guideline implementation materials that are concise and actionable. Evaluation of the guideline and implementation materials is crucial in understanding the influence of the recommendations.

JAMA Pediatr. 2018;172(11):e182853. doi:10.1001/jamapediatrics.2018.2853
 Published online September 4, 2018. Corrected on November 5, 2018.

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CDC DATA ON TBI

Coordination is Key




Support better monitoring of TBI through collaboration and communication.

www.cdc.gov

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An estimated, 283,000 children seek care in U.S. emergency departments each year for a sports- or recreation-related TBI. TBIs sustained in contact sports account for approximately 45% of these visits. Football, bicycling, basketball, playground activities, and soccer account for the highest number of emergency department visits.

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HEALING OUR SOLDIERS
Uncovering the secrets of Traumatic Brain Injury

Exploring the Hidden Culture in Hawaii The Small Strange Lives of Miss

NEARLY 383,947 CASES OF TBI WERE REPORTED IN THE U.S. MILITARY FROM 2000 TO 2018 AND IT HAS BEEN REPORTED THAT AS MANY AS 75% OF THESE TBI PATIENTS REPORTED HAVING VISION CHANGES

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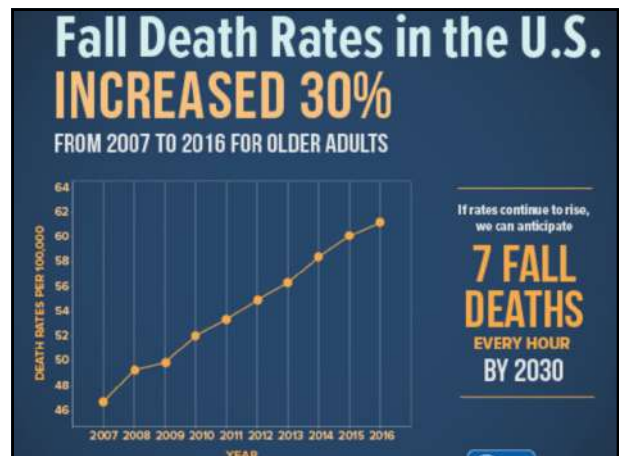
FALLS AND TBI IN THE ELDERLY

22

FALLS ARE THE MOST COMMON CAUSE OF TBI



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MMWR | STOP HIGH SCHOOL STUDENT CONCUSSIONS

STUDENTS ARE AT RISK FOR CONCUSSIONS

- 15% REPORTED AT LEAST ONE CONCUSSION IN PREVIOUS YEARS
- 6% REPORTED MORE THAN ONE CONCUSSION

SOME STUDENTS ARE AT HIGHER RISK

MALES

STUDENTS WHO PLAY ON SPORTS TEAMS

CREATE A CULTURE OF CONCUSSION SAFETY

- LEARN ABOUT CONCUSSION SYMPTOMS
- REPORT SUSPECTED CONCUSSIONS
- SEE A HEALTHCARE PROVIDER

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OBJECTIVES

- Statistics on mTBI (Concussion) and TBI
- Diffuse Axonal Injury (DAI)
- Chronic Traumatic Encephalopathy (CTE)
- Rehabilitative Options
- Impact of Vision Deficits on Return to Play/Work
- Military and mTBI/TBI
- NFL and NFL Concussion Protocol

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AMERICAN OPTOMETRIC ASSOCIATION

Evidence-Based Clinical Practice Guidelines

Comprehensive Pediatric Eye and Vision Examination

AMERICAN OPTOMETRIC ASSOCIATION

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AMERICAN OPTOMETRIC ASSOCIATION

BRAIN INJURY ELECTRONIC RESOURCE MANUAL (BIERM) PART A AND B

BRAIN INJURY ELECTRONIC RESOURCE MANUAL

10/2006 2nd Edition

The BIERM serves as a comprehensive resource to aid optometrists in evaluating patients with brain injury.

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TBI AND OPTOMETRY

Traumatic Brain Injury

What is the Optometrist's Role?

Rehabilitation of TBI patients is faster and more complete when optometrists are part of the health care team. By Col. Francis L. McVeigh, O.D., M.S.

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OBJECTIVES

- Statistics on mTBI (Concussion) and TBI
- Diffuse Axonal Injury (DAI)
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THREE MOST COMMON VISUAL DYSFUNCTIONS

- Accommodation
- Near Point of Convergence
- Saccades and Pursuits


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IMPORTANT TESTING

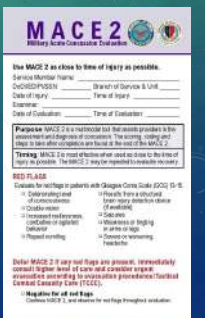
Vestibular/Ocular-Motor Screening

VOMS

For Concussion Instructions



DHA

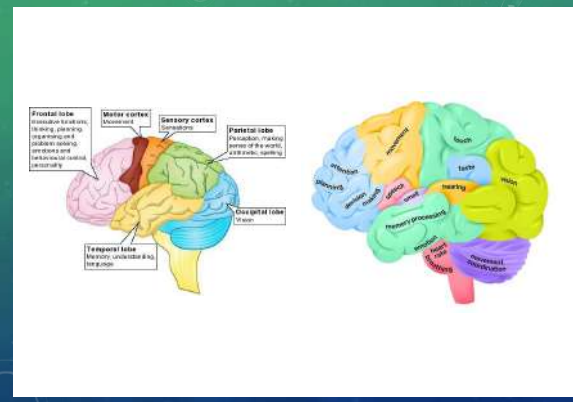


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LOBES OF THE BRAIN

- FRONTAL
- PARIETAL
- TEMPORAL
- OCCIPITAL
- CEREBELLUM
- BRAIN STEM

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WHAT IS A TBI?

According to the Centers for Disease Control (CDC)

“A TBI is caused by a bump, blow, or jolt to the head or a penetrating head injury that disrupts the normal function of the brain. Not all blows or jolts to the head result in a TBI. The severity of a TBI may range from “mild” (i.e., a brief change in mental status or consciousness) to “severe” (i.e., an extended period of unconsciousness or memory loss after the injury). Most TBIs that occur each year are mild, commonly called concussions.

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TYPES OF TBI

Mild	Moderate	Severe
<ul style="list-style-type: none"> • Primary damage / injury mechanism: predominantly blast, non-penetrating • Loss/alteration of consciousness: <30 minutes • Amnesia: <24 hours • GCS: 13-15 • Imaging: negative • Comorbidity: Post Traumatic Stress Disorder, overlapping symptoms • Outcome: Transient neuropsychiatric deficits, mostly full-recovery, long-term neuropsychiatric especially after repeated injuries are frequent 	<ul style="list-style-type: none"> • Primary damage / injury mechanism: frequently mixed, blast + acceleration/deceleration, typically non-penetrating • Loss/alteration of consciousness: >30 minutes, <24 hours • Amnesia: >24 hours, <7 days • GCS: 9-12 • Imaging: transient changes • Comorbidity: PTSD, other injuries • Outcome: mild-to-moderate, typically chronic, neurological and neuropsychiatric abnormalities 	<ul style="list-style-type: none"> • Primary damage / injury mechanism: complex, blast + acceleration/deceleration + penetration • Loss/alteration of consciousness: >24 hours • Amnesia: >7 days • GCS: <9 • Imaging: positive, lasting abnormalities • Comorbidity: Polytrauma, such as multiple-organ injuries • Outcome: death, significant, neurological and neuropsychiatric deficits, severe, chronic physical and neuropsychiatric disabilities

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BRAIN INJURY TYPES AND TERMS

- Concussion (mTBI)
- Diffuse Axonal Injury (DAI)
- Chronic Traumatic Encephalopathy (CTE)

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OTHER TYPES OF BRAIN INJURIES (TRAUMATIC AND ACQUIRED)

- Aneurysm
- Anoxic Brain Injury
- Closed Head Injury
- Brain Contusions
- Hypoxic-Ischemic Injury
- Hematoma
- Infarction
- Intra-Cranial Pressure
- Open Head Injury
- Organic Brain Injury
- Subarachnoid Hemorrhage

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CONCUSSION

- Another name for Mild Traumatic Brain Injury (mTBI)
- Most common form of TBI
- Effects are usually temporary but can include headaches and problems with concentration, memory, balance and coordination
- Can easily affect vision

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WHAT IS DIFFUSE AXONAL INJURY (DAI)?

- Occurs in about half of all severe head traumas
- Can also occur in moderate and mild brain injury
- Typically diffuse and not focal
- Severe DAI is one of the leading causes of death in people with traumatic brain injury
- Neuro-Imaging???

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DIFFUSE AXONAL INJURY

- White Matter & Junctional Injury
- Results from the brain moving back and forth in the skull as a result of acceleration or deceleration (coup & contre-coup)
- Shearing injury occurs as tissue slides over tissue
- Responsible for unconsciousness, as well as the vegetative state that occurs after a severe head injury

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NORMAL NEURAL CELL AND AXON

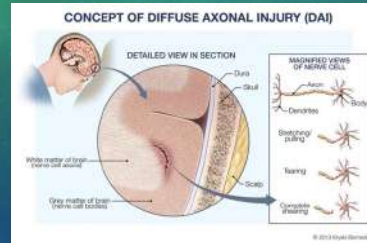
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SIZE AND DIAMETER OF AN AXON



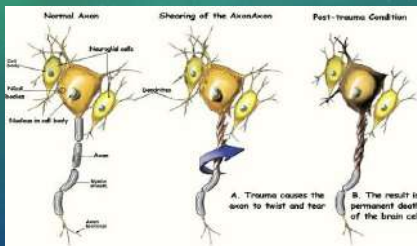
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DIFFUSE AXONAL INJURY GREY/WHITE MATTER JUNCTION



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DIFFUSE AXONAL INJURY



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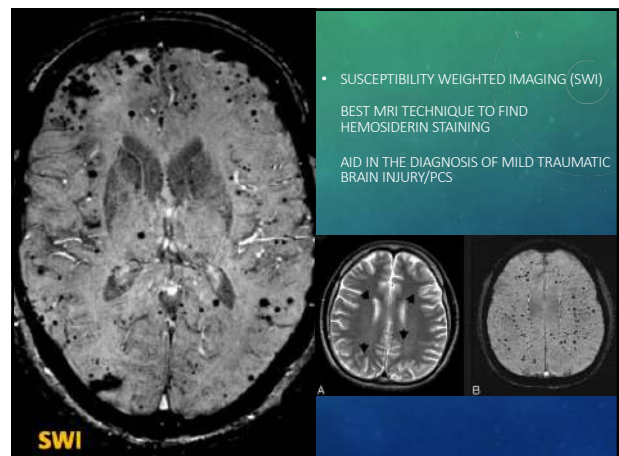
SUSCEPTIBILITY WEIGHTED IMAGING

- MRI sequence which is particularly sensitive to compounds which distort the local magnetic field and as such make it useful in detecting blood products, calcium, etc.
- Can detect subtle findings such as venous damage, and cerebral microbleeds when other scanning modalities cannot.

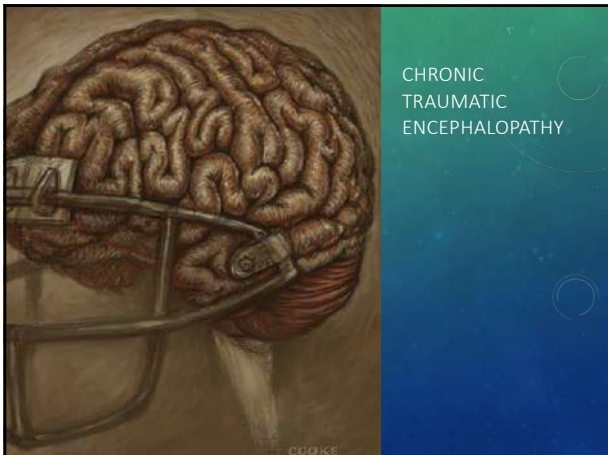
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CHRONIC
TRAUMATIC
ENCEPHALOPATHY

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WHAT IS CHRONIC TRAUMATIC ENCEPHALOPATHY?

Boston University Center for the Study of Traumatic Encephalopathy

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THREE MOST COMMON VISUAL DYSFUNCTIONS

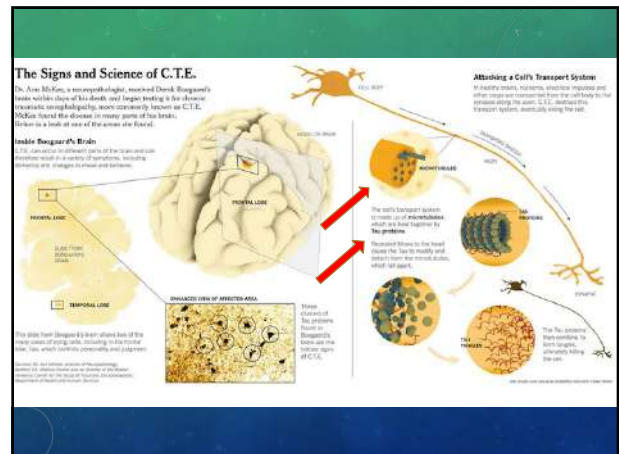
- Accommodation
- Near Point of Convergence
- Saccades and Pursuits

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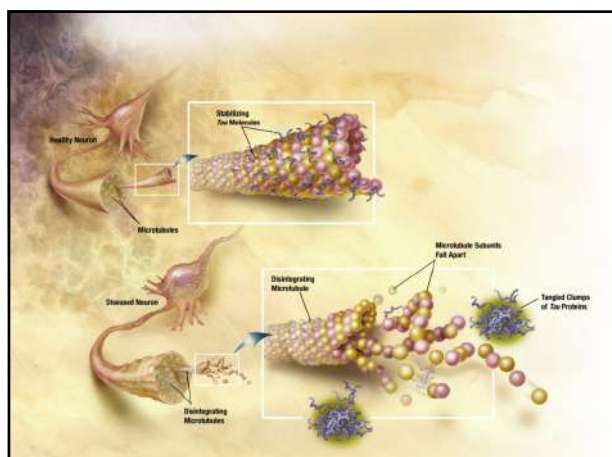
CHRONIC TRAUMATIC ENCEPHALOPATHY (CTE)

- Chronic Traumatic Encephalopathy (CTE) is a progressive degenerative disease of the brain found in athletes (and others) with a history of repetitive brain trauma, including symptomatic concussions as well as asymptomatic sub-concussive hits to the head.

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HISTORY OF CHRONIC TRAUMATIC ENCEPHALOPATHY

- CTE was originally reported in 1928 by a pathologist, who described the clinical aspects of a progressive neurological deterioration ('punch drunk') that occurred after repetitive brain trauma in boxers.
- Originally termed dementia pugilistica' (*pugilistica comes from the Latin root pugil, for boxer*)

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CHRONIC TRAUMATIC ENCEPHALOPATHY (CTE)

- The trauma triggers progressive degeneration of the brain tissue, including the build-up of an abnormal protein called **TAU**.
- Changes in the brain can begin months, years, or even decades after the last brain trauma or end of active athletic involvement.
- The brain degeneration is associated with memory loss, confusion, impaired judgment, impulse control problems, aggression, depression, and, eventually, progressive dementia.

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JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION ORIGINAL INVESTIGATION JULY 25, 2017

JAMA | Original Investigation

Clinicopathological Evaluation of Chronic Traumatic Encephalopathy in Players of American Football

Jesse Mez, MD, MS, Daniel H. Daneshvar, MD, PhD, Patrick T. Kiernan, BA, Bobak Abdolmohammadi, BA, Victor E. Alvarez, MD, Bertrand R. Huber, MD, PhD, Michael L. Alisco, PhD, Todd M. Solomon, PhD, Christopher J. Nowinski, PhD, Lisa McHale, EdS, Kerry A. Cormier, BA, Caroline A. Kubilus, Brett M. Martin, MS, Lauren Murphy, MBA, Christine M. Baugh, MPH, Phillip H. Montenegro, BA, Christine E. Chaisson, MPH, Yorghos Tripodis, PhD, Neil W. Kowall, MD, Jennifer Weuve, MPH, ScD, Michael D. McClean, ScD, Robert C. Cantu, MD, Lee E. Goldstein, MD, PhD, Douglas I. Katz, MD, Robert A. Stern, PhD, Thor D. Stein, MD, PhD, Ann C. McKee, MD

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RESULTS Among 202 deceased former football players (median age at death, 66 years [interquartile range, 47-76 years]), CTE was neuropathologically diagnosed in 177 players (87%; median age at death, 67 years [interquartile range, 52-77 years]; mean years of football participation, 15.1 [SD, 5.2]), including 0 of 2 pre-high school, 3 of 14 high school (21%), 48 of 53 college (91%), 9 of 14 semiprofessional (64%), 7 of 8 Canadian Football League (88%), and 110 of 111 National Football League (99%) players. Neuropathological severity of CTE was distributed across the highest level of play, with all 3 former high school players having mild pathology and the majority of former college (27 [56%]), semiprofessional (5 [56%]), and professional (101 [86%]) players having severe pathology. Among 27 participants with mild CTE pathology, 26 (96%) had behavioral or mood symptoms or both, 23 (85%) had cognitive symptoms, and 9 (33%) had signs of dementia. Among 84 participants with severe CTE pathology, 75 (89%) had behavioral or mood symptoms or both, 80 (95%) had cognitive symptoms, and 71 (85%) had signs of dementia.

CONCLUSIONS AND RELEVANCE In a convenience sample of deceased football players who donated their brains for research, a high proportion had neuropathological evidence of CTE, suggesting that CTE may be related to prior participation in football.

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AMONG 202 DECEASED FORMER FOOTBALL PLAYERS (MEDIAN AGE AT DEATH, 66 YEARS [INTERQUARTILE RANGE, 47-76 YEARS]),

- CTE was neuropathologically diagnosed in 177 players (87%; median age at death, 67 years [interquartile range, 52-77 years];
- Mean years of football participation, 15.1 [SD, 5.2]), including 0 of 2 pre-high school, 3 of 14 high school (21%), 48 of 53 college (91%), 9 of 14 semiprofessional (64%), 7 of 8 Canadian Football League (88%), and 110 of 111 National Football League (99%) players.
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- Among 84 participants with severe CTE pathology, 75 (89%) had behavioral or mood symptoms or both, 80 (95%) had cognitive symptoms, and 71 (85%) had signs of dementia.

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- Representative Images of Phosphorylated Tau Pathology at CTE Pathological Stages III and IVCTE indicates chronic traumatic encephalopathy; NFT, neurofibrillary tangle; ptau, phosphorylated tau. For all images, 10-µm paraffin-embedded tissue sections were immunostained with microscopic mouse monoclonal antibody for phosphorylated tau (AT8) (Pierce Endogen). Positive ptau immunostaining appears dark red, hematoxylin counterstain, calibration bar indicates 100 µm. In stage III CTE, multiple CTE lesions and diffuse neurofibrillary degeneration of the medial temporal lobe are found. In stage IV CTE, CTE lesions and NFTs are widely distributed throughout the cerebral cortex, diencephalon, and brain stem. All hemispheric tissue section images are 50-µm sections immunostained with mouse monoclonal antibody CP-13, directed against phosphoserine 202 of tau (courtesy of Peter Davies, PhD, Feinstein Institute for Medical Research, 1-200), this is considered to be an early site of tau phosphorylation in NFT formation. Positive ptau immunostaining appears dark brown. A, Former NFL player with stage III CTE. There are multiple large CTE lesions in the frontal cortex and insula; there is diffuse neurofibrillary degeneration of hippocampus and entorhinal cortex (black arrowhead). Perivascular CTE lesion: a dense collection of NFTs and large dot-like and threadlike neurites enclose several small blood vessels. B, Former NFL player with stage IV CTE. There are large, confluent CTE lesions in the frontal, temporal, and insular cortices and there is diffuse neurofibrillary degeneration of the amygdala and entorhinal cortex (black arrowhead). Perivascular CTE lesion: a large accumulation of NFTs, many of them ghost tangles, encompass several small blood vessels.

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The JAMA Network

From: Clinicopathological Evaluation of Chronic Traumatic Encephalopathy in Players of American Football

JAMA. 2017;318(4):360-370. doi:10.1001/jama.2017.8334

CTE Stage	No. of Brains	Brain Region												
		Frontal	Temporal	Parietal	Sagittal	Insula	Entorhinal	Amygdala	Hippocampus	Thalamus	SN	LC	Cerebellum	
1	11	1.1	0.6	0.2	0.4	0.3	0.0	0.4	0.1	0.3	0.5	0.6	0.9	0
2	33	1.6	1.4	1.3	1.2	1.1	1.4	1.1	0.9	0.9	1.3	1.0	2.0	0.2
3	76	2.2	2.1	1.6	2.0	2.1	2.4	2.3	2.1	1.4	2.3	1.8	2.5	0.3
4	57	2.8	2.7	1.8	2.7	2.8	2.8	2.8	2.4	2.2	2.1	2.3	2.5	0.6
Total	177	2.2	2.1	1.8	2.0	2.1	2.3	2.1	1.8	1.5	2.1	1.8	2.3	0.3

Mean phosphorylated tau pathology

0	1	2	3
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The JAMA Network

From: Clinicopathological Evaluation of Chronic Traumatic Encephalopathy in Players of American Football

JAMA. 2017;318(4):360-370. doi:10.1001/jama.2017.8334

CTE Stage	No. of Brains	Age at Death, Median (IQR), y	Neuropathological Findings, No. (%)												
			AD	DP	NP	AI	TSP-43	06	AD	LD	T102-TSP-43	T102-Tau	MND	Pern CTE, No. (%)	
1	11	66 (52-74)	3 (28)	2 (18)	1 (9)	1 (9)	2 (18)	1 (9)	0	1 (9)	0	0	0	0	0 (0)
2	33	49 (26-63)	8 (24)	8 (24)	5 (15)	7 (21)	10 (30)	1 (3)	2 (6)	1 (3)	2 (6)	1 (3)	4 (12)	21 (64)	
3	76	61 (51-78)	45 (59)	41 (54)	25 (33)	29 (38)	29 (38)	18 (23)	4 (5)	15 (20)	1 (1)	3 (4)	8 (10)	42 (55)	
4	57	70 (60-82)	31 (54)	31 (54)	41 (72)	31 (54)	41 (72)	31 (54)	31 (54)	18 (32)	18 (32)	9 (16)	2 (4)	1 (2)	27 (47)
Total	177	67 (53-78)	107 (60)	101 (57)	76 (43)	68 (39)	81 (46)	81 (46)	23 (13)	38 (21)	8 (5)	6 (3)	11 (6)	98 (55)	

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CTE Stage	No. of Brains	Age at Death, Median (IQR), y	Neuropathological Findings, No. (%)												
			AD	DP	NP	AI	TSP-43	06	AD	LD	T102-TSP-43	T102-Tau	MND	Pern CTE, No. (%)	
1	11	66 (52-74)	3 (28)	2 (18)	1 (9)	1 (9)	2 (18)	1 (9)	0	1 (9)	0	0	0	0 (0)	
2	33	49 (26-63)	8 (24)	8 (24)	5 (15)	7 (21)	10 (30)	1 (3)	2 (6)	1 (3)	2 (6)	1 (3)	4 (12)	21 (64)	
3	76	61 (51-78)	45 (59)	41 (54)	25 (33)	29 (38)	29 (38)	18 (23)	4 (5)	15 (20)	1 (1)	3 (4)	8 (10)	42 (55)	
4	57	70 (60-82)	31 (54)	31 (54)	41 (72)	31 (54)	41 (72)	31 (54)	31 (54)	18 (32)	18 (32)	9 (16)	2 (4)	1 (2)	27 (47)
Total	177	67 (53-78)	107 (60)	101 (57)	76 (43)	68 (39)	81 (46)	81 (46)	23 (13)	38 (21)	8 (5)	6 (3)	11 (6)	98 (55)	

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Neuropathologist Dr. Ann McKee has identified four stages of the degenerative disease.

CHRONIC TRAUMATIC ENCEPHALOPATHY HISTORY

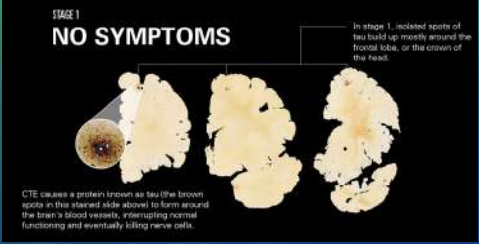


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CTE STAGE 1

**STAGE 1
NO SYMPTOMS**

In stage 1, isolated spots of tau build up, mostly around the frontal lobe, or the crown of the head.




CTE causes a protein known as tau (the brown spots in the stained slide above) to form around the brain's blood vessels, interrupting normal functioning and eventually killing nerve cells.

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CTE STAGE 2

**STAGE 2
RAGE, IMPULSIVITY, DEPRESSION**



In stage 2, symptoms begin to appear as defective tau protein affects more nerve cells in the brain's frontal lobe lobes.

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The JAMA Network

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JAMA. 2017;318(4):399-370. doi:10.1001/jama.2017.8334



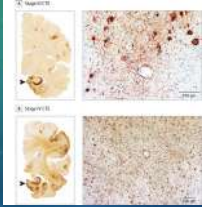
Figure Legend:
Representative images of phosphorylated tau pathology at CTE pathological stages 1 and 2. ICHTE indicates chronic traumatic encephalopathy, NFT, neurofibrillary tangle; ptau, phosphorylated tau. For all images, 10-µm paraffin-embedded tissue sections were immunostained with microscopic mouse monoclonal antibody for phosphorylated tau (AT8) (Pierce Endogen). Positive ptau immunostaining appears dark red, hematoxylin counterstain; calibration bar indicates 100 µm. Stage 1 CTE is characterized by 1 or 2 isolated perivascular epicenters of ptau NFIs and neurites (ie, CTE lesions) at the depths of the cortical sulci. In stage II, 3 or more cortical CTE lesions are found. All hemispheric tissue section images are 50-µm sections immunostained with mouse monoclonal antibody CP-13, directed against phosphoserine 202 of tau (courtesy of Peter Davies, PhD, Feinstein Institute for

76

The JAMA Network

From: Clinicopathological Evaluation of Chronic Traumatic Encephalopathy in Players of American Football

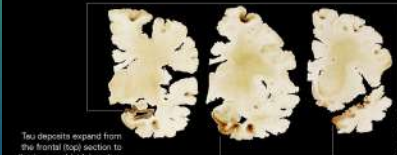
JAMA. 2017;318(4):399-370. doi:10.1001/jama.2017.8334



77

CTE STAGE 3

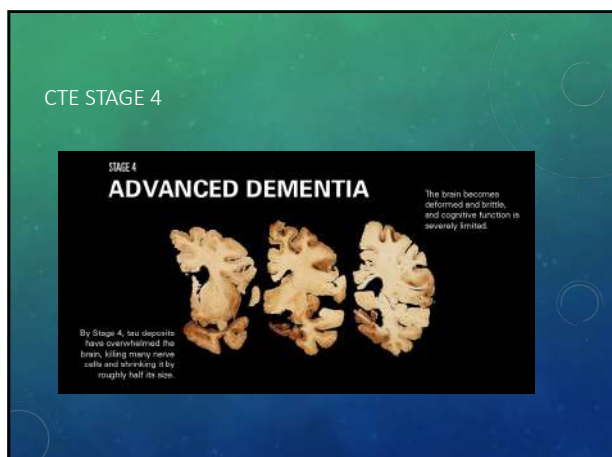
**STAGE 3
CONFUSION, MEMORY LOSS**



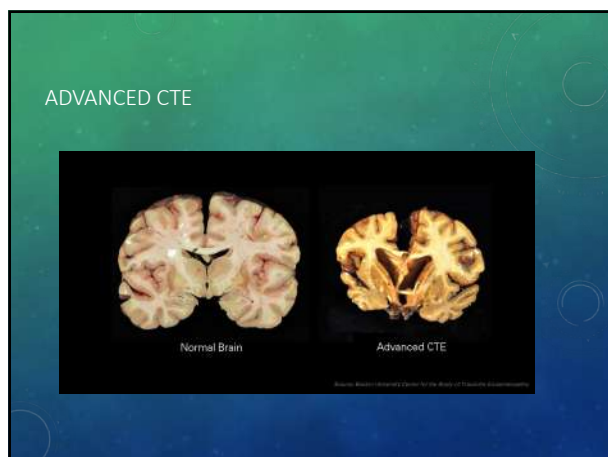
Tau deposits expand from the frontal lobe section to the temporal (side) section of the brain.

Condition begins to affect the amygdala and the hippocampus, which impairs emotion and memory.

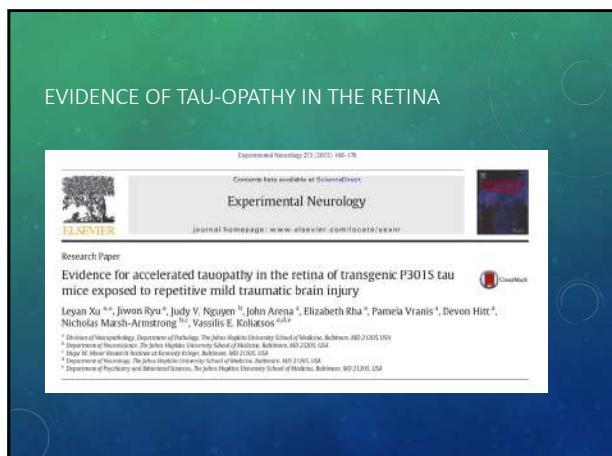
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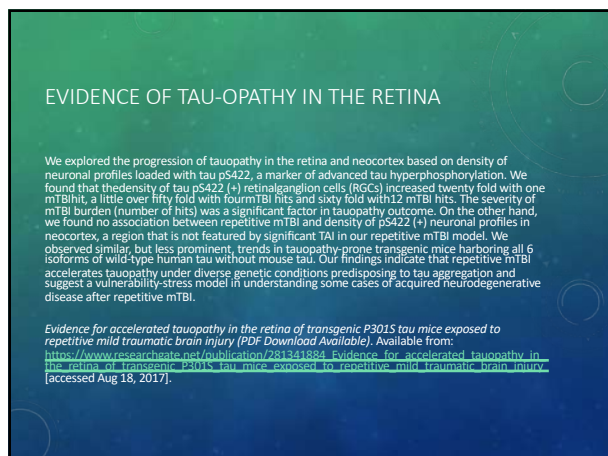
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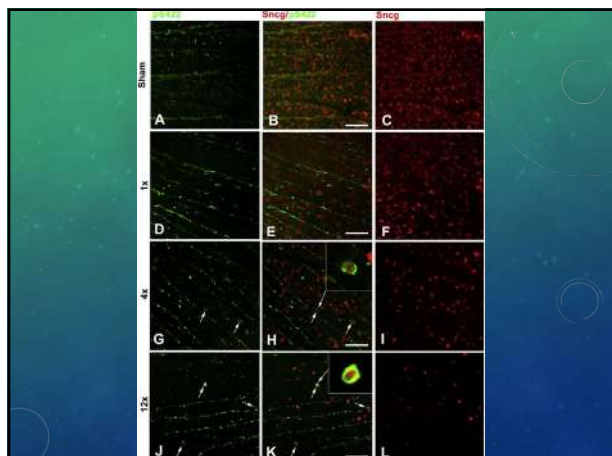
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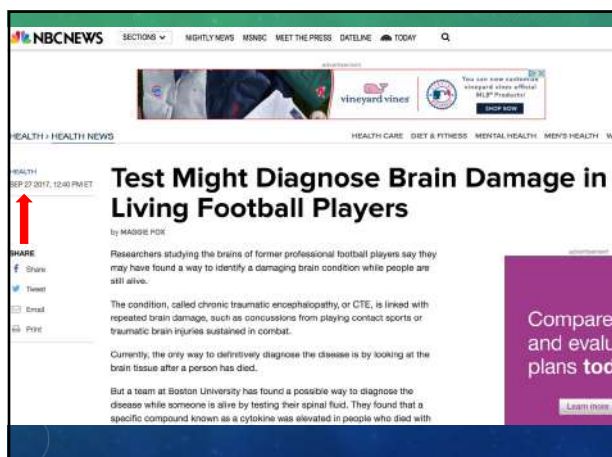
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83



84



85

WHAT IS THE INCIDENCE OF TBI?

- According to the CDC data recovered from the VA/DOD CPG on Management of Concussion/mTBI
- 1.7 Million Americans per year survive a TBI
- 230,000 are hospitalized
- 50,000 die which is ~30% of all injury related deaths

86

TOP TEN OF TBI ACCORDING TO THE CDC

Number 1

A traumatic brain injury is a blow or jolt to the head or a penetrating head injury that disrupts the function of the brain. You do not need to lose consciousness to sustain a concussion.

87

TOP TEN OF TBI

Number 2

1.7 million people sustain a TBI each year in the United States. By the numbers, every American has more than a 1:300 chance of sustaining a traumatic brain injury each year.

88

TOP TEN OF TBI

Number 3

The three groups at highest risk for traumatic brain injury are children (0-4 year olds), teenagers (15-19 year olds), and adults (65 and older). (2)

Males aged 0 to 4 years have the highest rates of TBI-related emergency department visits, hospitalizations, and deaths combined.

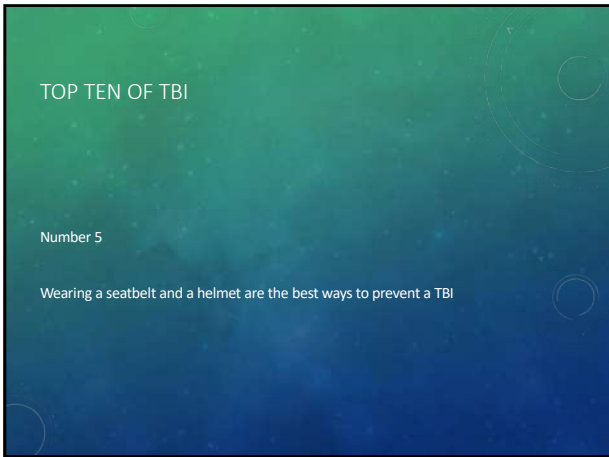
89

TOP TEN OF TBI

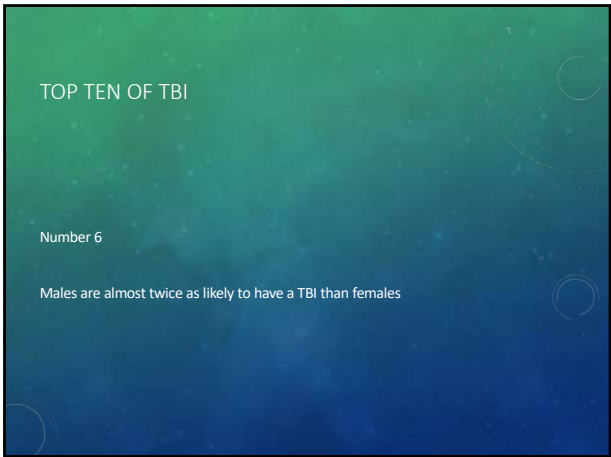
Number 4

Estimates peg the number of sports-related traumatic brain injuries as high as 3.8 million per year.

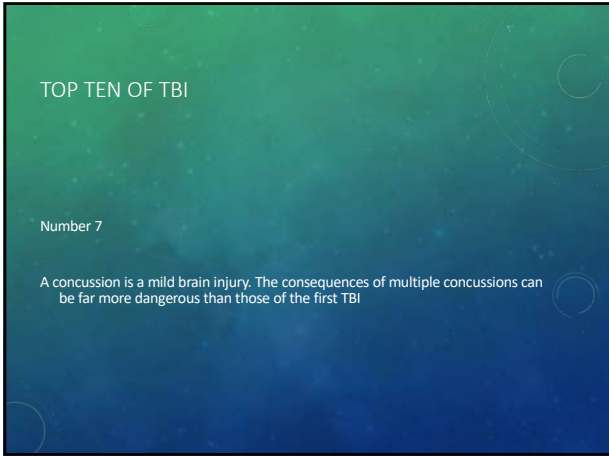
90



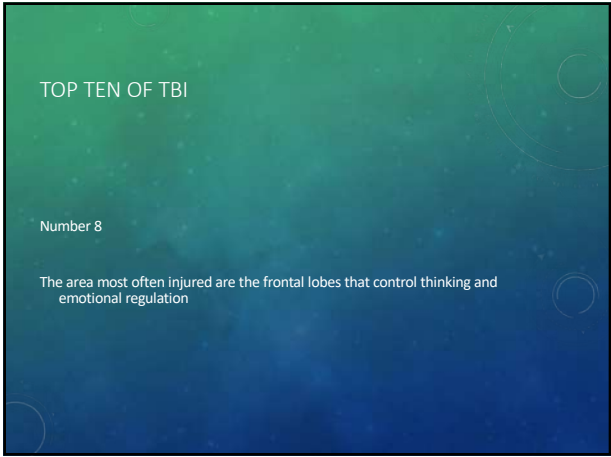
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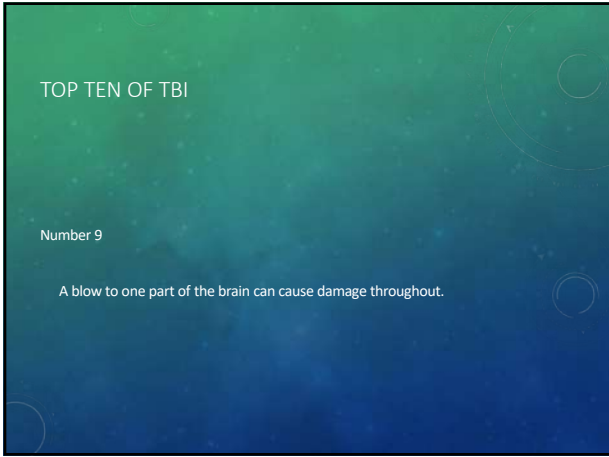
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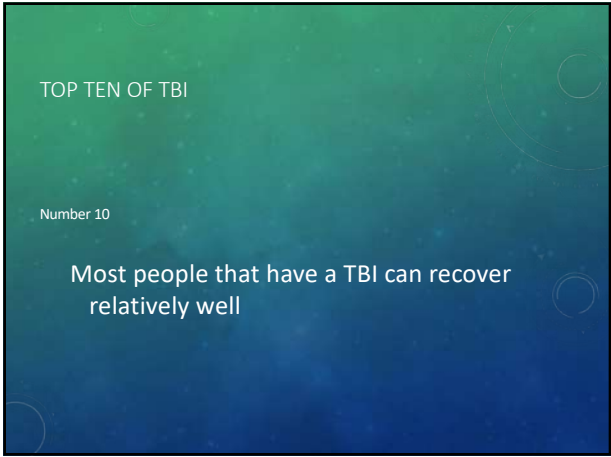
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THREE MOST COMMON VISUAL DYSFUNCTIONS

- Accommodation
- Near Point of Convergence
- Saccades and Pursuits

97

GLASGOW COMA SCALE

Psychopharmacological Treatment for Cognitive Impairment in Survivors of Traumatic Brain Injury: A Critical Review Brian W. Writer, D.O.; Jason E. Schillerstrom, M.D. *The Journal of Neuropsychiatry and Clinical Neurosciences* 2009;21:362-370.


Severity	Glasgow Coma Scale Score	Duration of Loss of Consciousness	Duration of Posttraumatic Amnesia
Mild	13-15	<30 minutes and/or	<1 hour
Moderate	9-12	1-24 hours and/or	<24 hours
Severe	8 or less	>24 hours and/or	>24 hours

98

DIAGNOSIS OF TRAUMATIC BRAIN INJURY

99

HOW MANY PEOPLE HERE FEEL COMFORTABLE MANAGING A TBI PATIENT?



100

WHAT DOES A TBI EYE EXAM LOOK LIKE?

101

WHAT TESTS DO WE PERFORM?

- Full Dilated Eye Exam?
- Cycloplegic Refraction?
- Accommodative Testing?
- Vergence Testing?
- What Else???????

102

CAN HAVE SIGNIFICANT DIFFERENCES IN DE NEAR AND DE DISTANCE VISUAL ACUITY



103

VISUAL CONSIDERATIONS IN A TBI PATIENT

- Photophobia
- Loss or decrease
 - Visual Acuity
 - Color Discrimination
 - Brightness Detection
 - Contrast Sensitivity
- Visual Field Defects
- Visual Midline Shift Syndrome

104

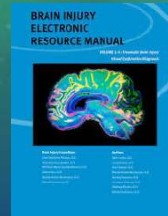
MOST COMMON VISUAL COMPLAINTS AFTER MTBI

- Oculomotor
 - Diplopia
- Binocular
 - Convergence Insufficiency
- Vision Field Losses
 - With Neglect
 - Without Neglect
- Accomodative

http://www.brainline.org/content/2010/02/vision-issues-after-brain-injury-brainline-talks-with-dr-gregory-goodrich_pageall.html

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AOA BRAIN INJURY ELECTRONIC RESOURCE MANUAL (BIERM) PART A AND B



- The BIERM serves as a comprehensive resource to aid optometrists in evaluating patients with brain injury.

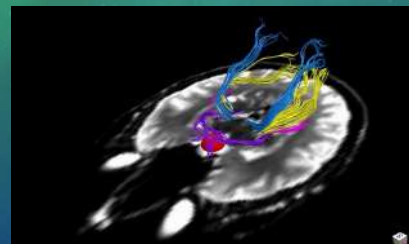
106

INJURY TO THE VISUAL PATHWAY



107

INJURY TO VISUAL PATHWAY



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EXAMINATION OF AFFERENT PATHWAYS

- Acuity
- Contrast Sensitivity
- Color
- Amsler Grid
- Confrontation Visual Fields
- Photo Stress Test
- Pupils and Near Vision Testing

109

EXAMINATION OF EFFERENT PATHWAYS

- Lids
- CN VII (Supra and Infra Nuclear)
- CN V-1
- Ocular Stability an Binocular Alignment
- Accommodation in Free Space
- Sensory Status

110

HIGH YIELD VISION TESTING

- ⊙ Distance Cover test
- ⊙ Near Cover Test
- ⊙ Versions and Pursuits
- ⊙ Accommodation
- ⊙ Saccades
- ⊙ NPC/Repeated NPC
- ⊙ Confrontation Field
 - Kinetic
 - Static Peripheral
 - Static Central
- ⊙ [J Rehabil Res Dev. 2013;50\(6\):757-68.](#)
- ⊙ [Development of a mild traumatic brain injury-specific vision screening protocol: a Delphi study.Goodrich, et al.](#)

111

VISION TESTING FOR MTBI

- History
- Qualify the mTBI with Injury History
- Sensory History
- TBI Eye Injury/Pain History
- TBI Vision History
- TBI Reading History
- [J Rehabil Res Dev. 2013;50\(6\):757-68.](#)
- [Development of a mild traumatic brain injury-specific vision screening protocol: a Delphi study.Goodrich, et al.](#)

112


TBI SPECIFIC PERCEPTUAL TESTING

- Memory
- Auditory
- Spatial
- Closure
- Motor
- Figure-Ground

113

KING-DEVICK TEST

IMHO EVERYONE NEEDS ONE FOR THEIR OFFICE



114

KING-DEVICK RESEARCH

- Important Screening tool in exam lane for student athletes

Leong DF1, Balcer LJ2, Galetta SL2, Evans G3, Gimre M3, Watt D3. The King-Devick test for sideline concussion screening in collegiate football. *J Optom.* 2015 Jan 31. pii: S1888-4296(14)00116-2. doi: 10.1016/j.optom.2014.

115


PHOTOPHOBIA

- Very Common Complaint
- Requires Assistance


116

FILTERED AND PRISM LENSES FOR DIPLOPIA AND PHOTOPHOBIA

Yellow to Amber



Fresnel Prism



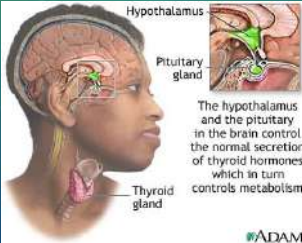
117

THE ENDOCRINE SYSTEM AND TBI

- Why would a mTBI patient develop Neuro-endocrine issues?
- Hint: What 2 major areas of the brain are in charge of hormone function

118

ENDOCRINE SYSTEM



The hypothalamus and the pituitary in the brain control the normal secretion of thyroid hormones which in turn controls metabolism

119

HORMONAL DEFICIENCIES MTBI

<p>Pituitary</p> <ul style="list-style-type: none"> • Thyroid Stimulating Hormone • Follicle Stimulating Hormone • Growth Hormone • Insulin Like Growth Factor 1 	<p>Gonadotropin</p> <ul style="list-style-type: none"> • Luteinizing Hormone • Follicle Stimulating Hormone • Testosterone • Estradiol
---	---

120

SIGNS OF NEURO-ENDOCRINE DYSFUNCTION

121

TREATMENT OF MTBI ENDOCRINE DYSFUNCTION

- http://www.dcoe.mil/content/Navigation/Documents/DCoE_TBI_NED_Training_Slides.pdf

122

TBI AND SLEEP DISORDERS

- Approximately 46% of chronic TBI patients have sleep disorders
 - Apnea 23%
 - Post traumatic hypersomnia 11%
 - Narcolepsy 6%
 - Periodic limb movement 7%
- Over 50% have insomnia complaints
- Require Nocturnal Polysomnography and the Multiple Sleep Latency Test
- [CNS Drugs, 2011, Mar;25\(3\):475-85. doi:10.2165/000000000-00000-Sleep_disorders_in_patients_with_traumatic_brain_injury_a_review_Castroiza_BJ1_Murthy_IN.](#)

123

SLEEP TESTING

www.carefusion.com

124

NOCTURNAL POLYSOMNOGRAM

- Breathing
- Brain Activity
- Heart Function
- Oxygen Levels
- Muscle Activity
- Eye Movement
- Snoring
- Quality of Sleep

125

MULTIPLE SLEEP LATENCY TEST

- Used to test for Narcolepsy
- Sleep latency is the time elapsed from the start of a daytime nap period to the first signs of sleep
- Works on the idea that the more tired you are the faster you will fall asleep
- Measures brain waves, EEG, eye movements and muscle activity

126

POST TRAUMATIC HEADACHES (PTHA)

- Most common complaint after a brain injury
 - Approximately 70%
- Different Types of PTHA
 - Tension
 - Migraine or Neuro-Vascular
 - Cervical or Cervico-Genic
 - Musculoskeletal or TMJ
 - Neuritic and/or Neuralgic Pain

© http://www.brainline.org/content/2009/03/post-traumatic-headache-after-tbi-brainline-talks-dr-nathan-zasler_pageall.html

127

TREATMENT OF PTHA

- Varies Depending on the Type and Cause
 - Manual Therapy with PT
 - Stress Management Intervention
 - Ultrasound
 - Injection Therapy
 - Nerve Blocks
 - Trigger Point Injections
 - Electrical Stimulation
 - Acupuncture
 - Medications

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ALMOST DONE



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THREE MOST COMMON VISUAL DYSFUNCTIONS

- Accommodation
- Near Point of Convergence
- Saccades and Pursuits

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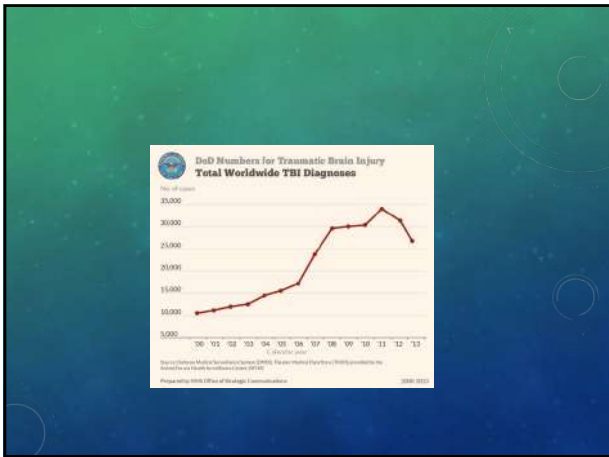
TBI AND THE MILITARY

131

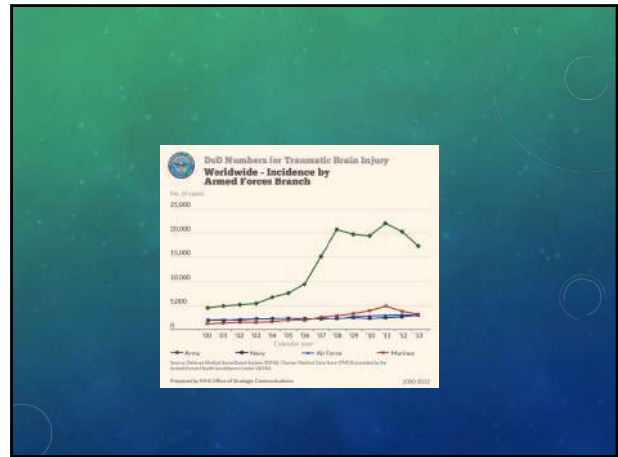
MILITARY TBI

- Between Q1 2000- Q2 2014 there were 307,283 TBI in the US Military
- The high rate of TBI and blast-related concussion events resulting from current combat operations directly impacts the health and safety of individual service members and subsequently the level of unit readiness and troop retention.
- <http://dwbic.dcoe.mil/dod-worldwide-numbers-tbi>

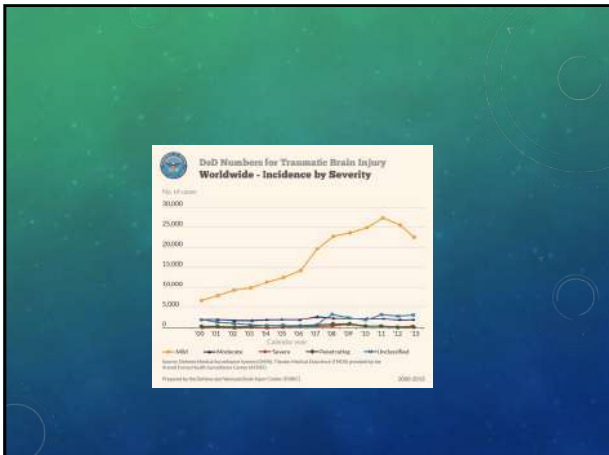
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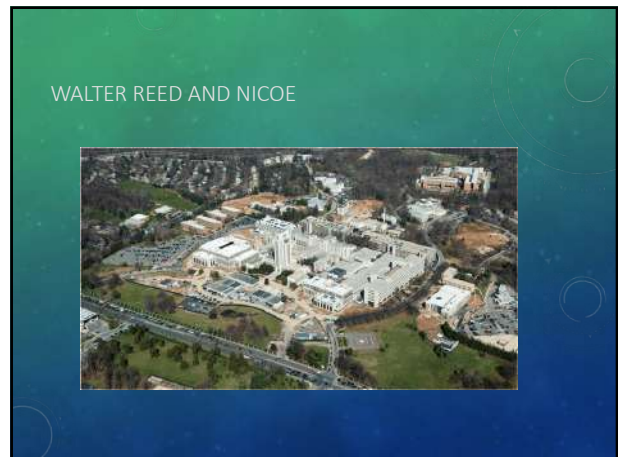
NICoE

- The mission of the NICoE is to be a leader in advancing traumatic brain injury (TBI) and psychological health (PH) treatment, research and education. This overarching mission is accomplished through three primary categories of activity
 - Research
 - Clinical
 - Education
- The NICoE provides comprehensive, interdisciplinary clinical evaluation; patient and family-focused intervention; treatment initiation; and individualized treatment planning. Patient evaluations may include a combination of traditional and complementary alternative medicine modalities, including:

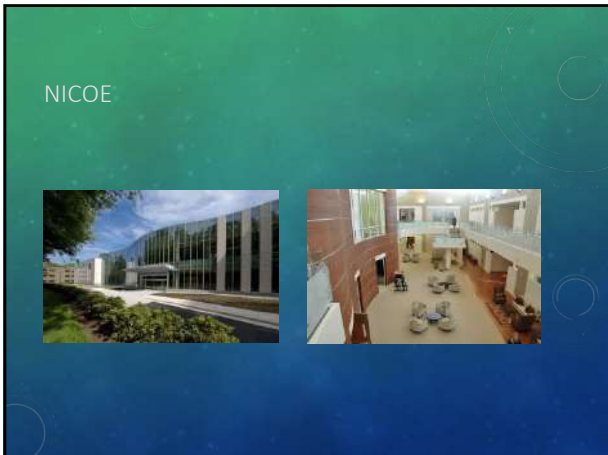
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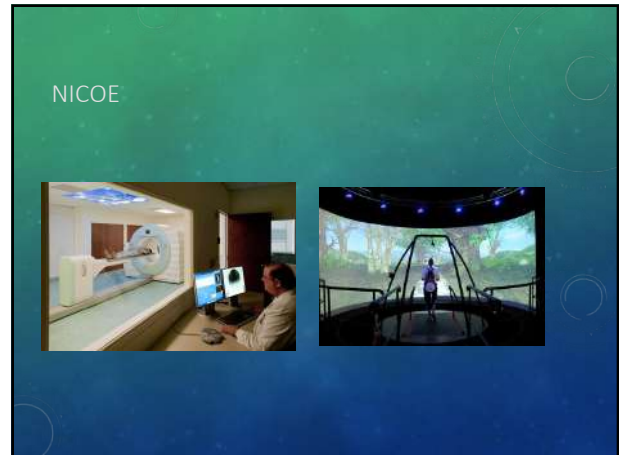
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141

- ### NICOE EVALUATION
- The Bio-Psycho-Social-Spiritual evaluation includes: Initial Intake/Triage Interview
 - Full Physical Evaluation
 - Audiology Evaluation
 - Auditory Processing Evaluation
 - Assistive Technology Evaluation
 - Complementary/Alternative Medicine (CAM) and Wellness Evaluation
 - Computer Assisted Rehabilitation Equipment (CAREN)
 - Detailed Vestibular Evaluation
 - Family Evaluation

142

- ### NICOE EVALUATION
- Fire Arms Training Simulator (FATS)
 - Magnetoencephalography/Electrophysiological studies
 - Neuroimaging
 - Neurological Evaluation
 - Neuropsychological Evaluation
 - Occupational Therapy Evaluation
 - Optometry/Vision Evaluation
 - Physical Therapy Evaluation
 - Psychiatric Evaluation
 - Research Consultation

143

- ### NICOE EVALUATION
- Sleep Evaluation
 - Speech-Language Pathology Evaluation
 - Spirituality Evaluation
 - Standard Laboratory Screening
 - Substance Use Assessment
 - Transcranial Doppler (TCD)/Visual/Perceptual Evaluation
 - Other Specialty Medical Examinations

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RETURN TO WORK POST MTBI DOD/VA CPG

- Patients sustaining a concussion/mTBI should return to normal (work/duty/school/leisure) activity post-injury as soon as possible
- A gradual resumption of activity is recommended
- If physical, cognitive, or behavioral complaints/symptoms re-emerge after returning to previous normal activity levels, a monitored progressive return to normal activity as tolerated should be recommended.

145

NFL CONCUSSION PROTOCOL



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PBS FRONTLINE CONCUSSION WATCH



http://apps.frontline.org/concussion-watch/#positions_2015

147

PBS FRONTLINE CONCUSSION WATCH NFL CONCUSSIONS BY POSITION 2015



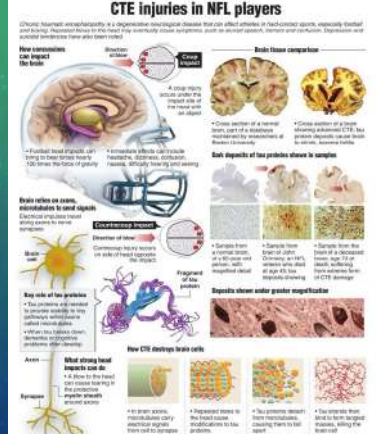
148

NFL HEAD, NECK AND SPINE COMMITTEE'S PROTOCOLS REGARDING DIAGNOSIS AND MANAGEMENT OF CONCUSSION

- Protocol Purpose: "Provide medical staffs responsible for the health care of NFL players with a process for diagnosing and managing concussion"
- Concussion Defined
 - "A complex pathophysiological process affecting the brain induced by mechanical forces" McCrory BJSM 2013
- Potential Concussion Signs and Symptoms
- NFL Sideline Concussion Assessment
- Emergency Medical Action Planning
 - Preseason Education and Assessment
 - Practice and Game Day Concussion Management
 - Return to Participation Process

149

CTE injuries in NFL players



Chronic traumatic encephalopathy is a degenerative neurologic disease that can affect athletes in high-contact sports, especially football and soccer. Research shows that the more the severely concussed athletes and in other sports, tennis and basketball. Symptoms and clinical picture may vary after diagnosis.

Brain tissue comparison: Shows normal brain tissue vs. brain tissue with CTE, highlighting the loss of neurons and the presence of neurofibrillary tangles.


Brain relies on oxygen, mitochondria to send signals: Explains the role of mitochondria in energy production and signal transmission.

Big role of tau proteins: Describes how tau proteins normally stabilize microtubules but can form neurofibrillary tangles in CTE.

How CTE changes brain cells: Illustrates the progression from normal brain cells to those with neurofibrillary tangles and eventually to cell death.

150

The relationship between the NFL and the NIH has been a contentious one from the start. In 2015, *Outside the Lines* reported that even though the gift had reportedly been unrestricted, the NFL restricted the NIH from using \$16 million of its \$30 million grant to fund a Boston University study on the degenerative brain disease chronic traumatic encephalopathy (CTE), a progressive brain disease thought to be caused by repeated subconcussive hits to the head.



Congressional Report Finds The NFL Tried To Interfere With Concussion Research

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The NFL reportedly objected to the fact that Dr. Robert Stern, the director of clinical research at the BU CTE Center, was leading the study, because he has always been very publicly critical of how the NFL has handled concussions. *Outside the Lines* found that the NFL instead steered its funds to doctors who currently or previously worked closely with the league. (The NIH ended up donating the funds for the study by itself; the NFL denied that it ever attempted to restrict the NIH funds.)

152

Back in 2012, the NFL made a heavily publicized \$30 million commitment to the National Institute of Health to fund concussion research, which the league wanted the public to see as a commitment it was taking head safety in football seriously.

But as ESPN's *Outside the Lines* reported this week, that partnership is set to expire next month even though the NFL—which is projected to reach \$14 billion in revenue this year—failed to give the NIH \$16 million of the promised funds. In other words, the league fulfilled less than half of its obligations to the NIH.

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ESPN Analyst Ed Cunningham Resigns Due to Concern over Head Injuries in Football

ESPN
#ESPN
WASH DC 2017

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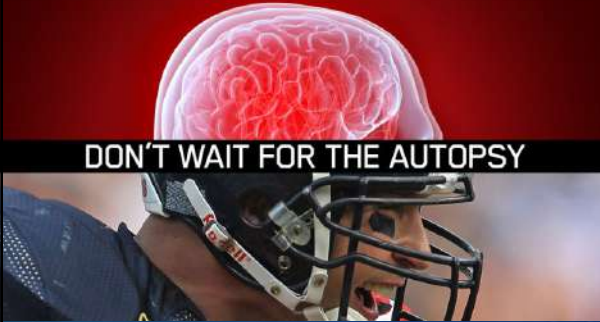
The Daily Free Press
THE INDEPENDENT STUDENT NEWSPAPER AT BOSTON UNIVERSITY
Sunday, November 5, 2017

NEURODOGS 2017
Be part of scientific discoveries as they unfold at Neuroscience 2017

NEWS ▾ SPORTS ▾ FEATURES ▾ OPINION ▾ BLOGS ▾

CAMPUS NEWS
Former NFL star Nick Buoniconti pledges brain to BU CTE Center
November 3, 2017 11:07 pm by Elise Takahama

155



DON'T WAIT FOR THE AUTOPSY

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