HSBO FORUM - 2025

Impact-Induced TBI (Traumatic Brain Injury) on High-Speed Boat Operators

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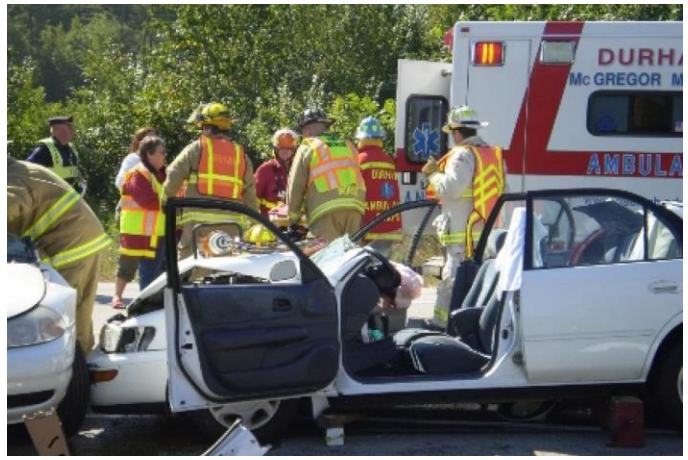
USU DOD BRAIN TISSUE REPOSITORY

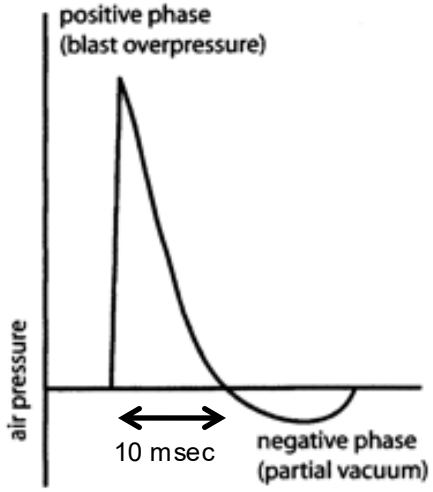
There are 2 types of TBI:

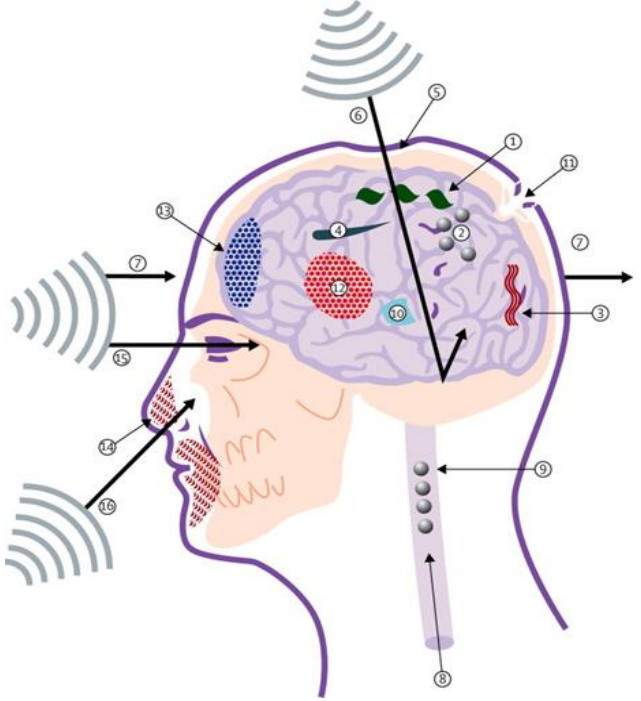
- **Impact TBI** (falls, fights, MVAs, contact sports, etc.): physical forces of impact, acceleration-deceleration, rotational forces. The pathophysiology and long term effects of impact TBI are relatively well-understood (contusion, coup-contra-coup, DAI, SDH, EDH, etc.).



- **Blast TBI** (exposure to IEDs, suicide bombs, artillery shells, breaching, etc.): physical effects of exposure to the blast wave. The pathophysiology and long term effects of blast TBI in the human are understudied and are poorly understood.

The Blast Wave





- Blast wave is a very quick (≈10 msec) pulse of high pressure that spreads in all direction at greater than the speed of sound.
- The blast wave can enter the intact skull and pass through the brain. **What effect on the structure and function of the brain does this have?**

Common Persistent Symptoms In Post-Blast TBI Subjects

Physical: headache, dizziness, blurred vision, sleep disturbance, sensitivity to light/noise, balance problems, hearing difficulties (tinnitus)/loss

Cognitive: impaired attention, concentration, recent memory, speed of processing, judgment, executive function

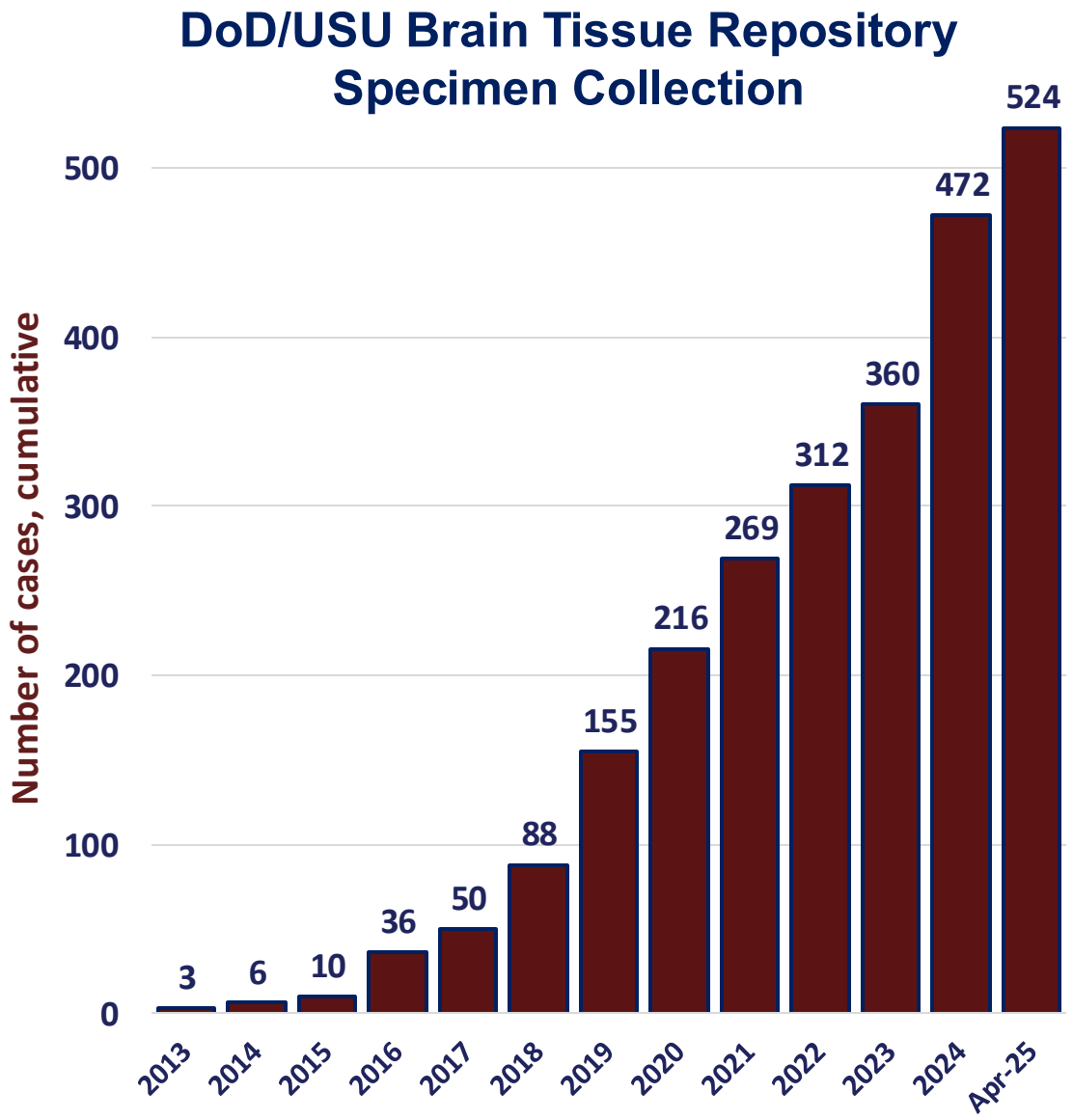
Behavioral/emotional: depression, mood swings, irritability, impulsivity, aggression, substance abuse, suicide

Can Neuroimaging Studies of Post-Blast TBI Patients Provide Answers?

To date, routine neuroimaging studies have not provided a consistent signal alteration to indicate the presence of pathological lesions in the brains of post-blast TBI patients with significant persistent symptomatology.

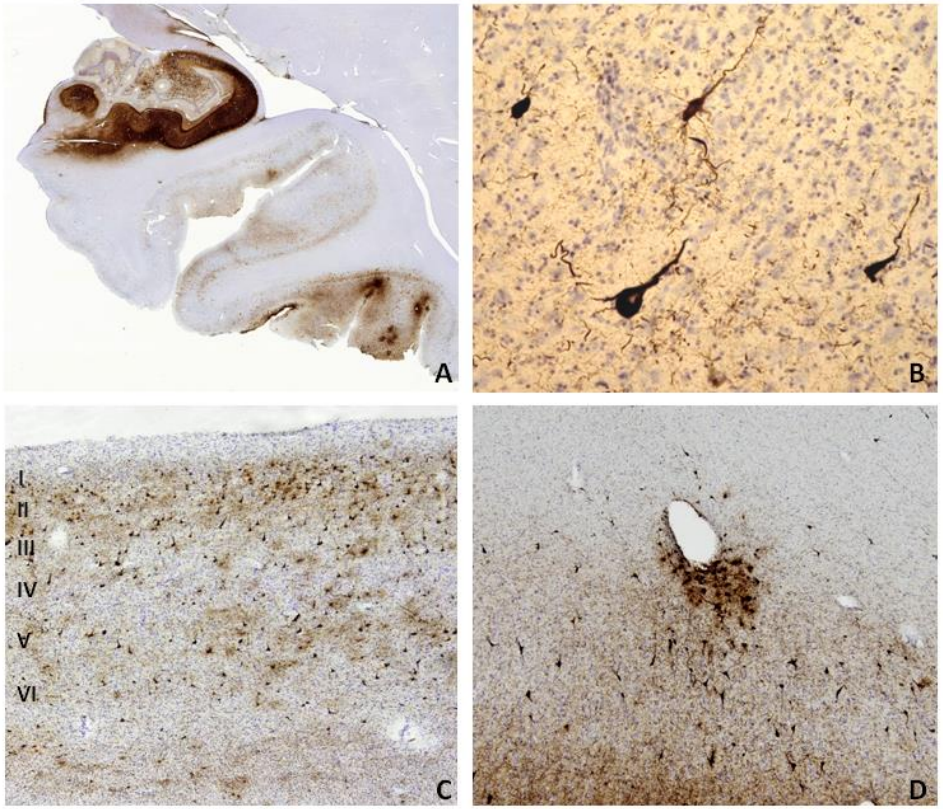
The DoD/USU Brain Tissue Repository

- 98% of brains from those who have served in the military: Active duty and retired; All branches represented. All consented for use in research, IRB approved
- 10% Special Forces Operators (heavy combat/blast exposure)
- **3 specimens from SWCC operators**
- 27% with contact sports history
- 19% with significant civilian impact-type TBI, unrelated to sports
- 23% with known/reported blast exposure
- COD - 23% suicide (natural: 58%; accident: 17%)
- 41% with reported history of alcohol and/or substance abuse



Chronic Traumatic Encephalopathy (CTE); Punch Drunk Syndrome; Dementia Pugilistica

- CTE is a chronic, progressive neurodegenerative disease involving accumulation of pathologic *tau* protein in the brain in a recognizable and specific pattern.
- CTE is almost exclusively seen in patients with a history of repeated **impact** TBIs, especially following participation in contact sports (NFL football, ice hockey, etc.).
- The clinical features of CTE are very similar to what is seen in symptomatic blast exposed Service Members.



from Shively, et al. *Arch. Neurol*. 2012

Could CTE explain the persistent symptomatology seen in blast-exposed Service Members or are other forms of brain pathology present?

We set out to determine prevalence of Chronic Traumatic Encephalopathy (CTE) in a large military series: the brains of 225 deceased service members consecutively donated to the DoD/USU Brain Tissue Repository.

David Priemer, MD

- All specimens were extensively sampled and comprehensively examined neuropathologically for evidence of CTE (blind to clinical information).
- All specimens were derived from individuals who had served in the military, without consideration of combat exposure, symptomatology, participation in contact sports, etc.
- A detailed semi-structured interview of the next-of-kin was used to determine prior history of participation in contact sports and any other significant civilian TBI incidents.
- Many of the cases in this cohort experienced extensive blast exposure and exhibited prominent persistent behavioral/neurologic symptomatology often referred to as the “invisible wound.”
- Blind broken and then compared the clinical features and exposure histories of the CTE-positive vs CTE-negative cases.

Pathognomonic CTE Lesion:

p-tau aggregates in neurons, with or without thorn-shaped astrocytes, at the depth of a cortical sulcus around a small blood vessel, deep in the parenchyma, and not restricted to the subpial and superficial region of the sulcus.

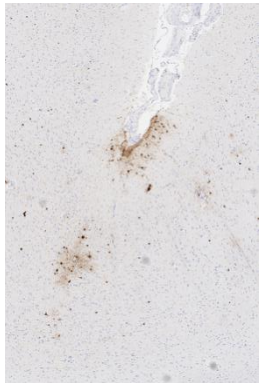
J Neuropathol Exp Neurol
Vol. 80, No. 3, March 2021, pp. 210–219
doi: 10.1093/jnen/nlab001

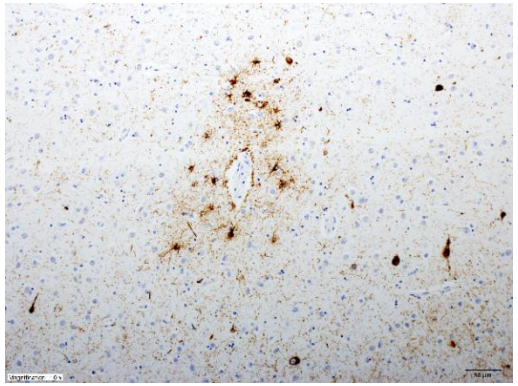
OXFORD

ORIGINAL ARTICLE

The Second NINDS/NIBIB Consensus Meeting to Define Neuropathological Criteria for the Diagnosis of Chronic Traumatic Encephalopathy

Kevin F. Bieniek, PhD, Nigel J. Cairns, PhD, FRCPath, John F. Crary, MD, PhD, Dennis W. Dickson, MD, Rebecca D. Folkerth, MD, C. Dirk Keene, MD, PhD, Irene Litvan, MD, Daniel P. Perl, MD, Thor D. Stein, MD, PhD, Jean-Paul Vonsattel, MD, William Stewart, PhD, FRCPath, Kristen Dams-O'Connor, PhD, Wayne A. Gordon, PhD, Yorghos Tripodis, PhD, Victor E. Alvarez, MD, Jesse Mez, MD, Michael L. Alosco, PhD, Ann C. McKee, MD, and the TBI/CTE Research Group





NOTE: These criteria recommend examining 5 cortex-containing samples. In our study we examined an average of 13 cerebral cortical samples. The identification of a **single CTE pathognomonic lesion** in a brain specimen is considered diagnostic for the disease, as was for this study.

Results: Prevalence of Neuropathologically Confirmed CTE in DoD/USU BTR Specimen Collection

Age at Death	Number of Specimens Evaluated	Cases Negative for CTE Diagnosis	Cases Meeting Consensus Diagnosis Criteria for CTE (age-related prevalence)	Number of CTE+ lesions found in brains with CTE* * Mean number of cerebral cortical sections examined = 12.7
18-30	32	32	0 (0.0%)	
31-40	35	34	1 (2.9%)	1
41-50	48	45	3 (6.7%)	1/4/4
51-60	69	65	4 (5.8%)	1/1/1/5
61-70	34	34	0 (0.0%)	1
>70	7	5	2 (28.6%)	‡
TOTAL	**225**	**215**	**10 (4.4%)**	**5 with 1 lesion; 2 with 4 and 1 with 5 lesions ‡ With AD changes (can not quantify)**

Characteristics of Neuropathologically Confirmed Cases of CTE among 225 DoD/USU Case Series

Age at Death	Cases Meeting Consensus Diagnostic Criteria for CTE (age-related prevalence)	Participation in Contact Sports	History of Blast TBI	History of Civilian Impact TBI with LOC and/or severe TBI
18-30	0 (0.0%)	0/0	0/0	0/0
31-40	1 (2.9%)	1/1	0/1	1/1
41-50	3 (6.7%)	3/3	2/3	3/3
51-60	4 (5.8%)	4/4	1/4	2/4
61-70	0 (0.0%)	0/0	0/0	0/0
>70	2 (28.6%)	2/2	0/2	1/2
TOTAL	**10 (4.4%)**	**10/10**	**3/10**	**7/10**

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 JUNE 9, 2022 VOL. 386 NO. 23

Chronic Traumatic Encephalopathy in the Brains of Military Personnel

David S. Priemer, M.D., Diego Iacono, M.D., Ph.D., C. Harker Rhodes, M.D., Ph.D., Cara H. Olsen, Dr.P.H., and Daniel P. Perl, M.D.

Relevant Conclusions of our NEJM Study:

- While CTE may be a serious concern following repeated impact TBI, such as is seen in former elite contact sport participants, the disorder is actually uncommon among those who have served in the military and was exclusively seen in association with a history of also playing contact sports.
- When diagnosed among military Service Members, the degree of pathologic involvement of the brain by CTE-related lesions tended to be rather minimal.

Summary and Conclusions – Updated Series (427 Military Cases)

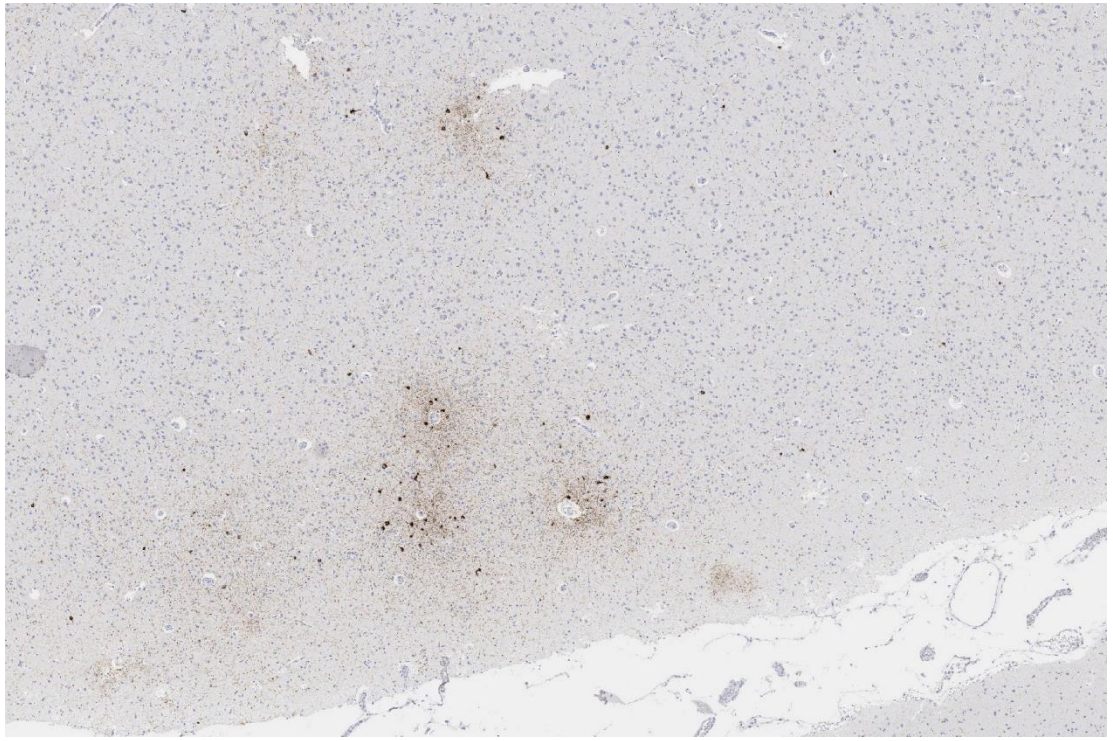
- **CTE remains uncommon in the expanded military brain series from the DoD/USU Brain Tissue Repository, despite high rates of blast and other combat-related exposures.**

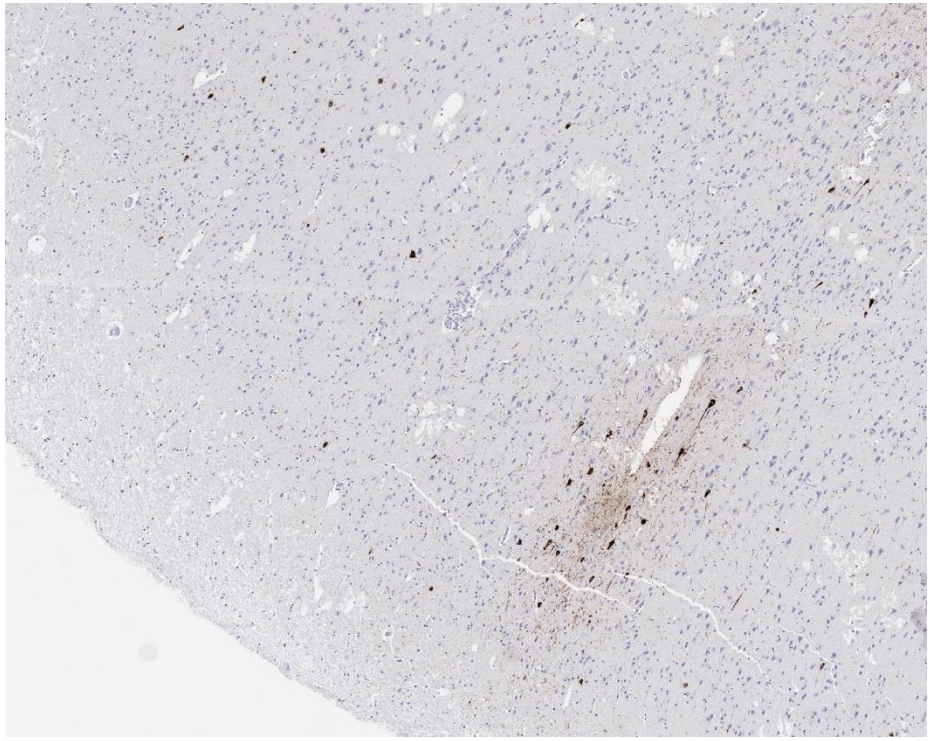
- When identified, **CTE overwhelmingly occurred in the context of a past history of contact sports participation**, and blast exposure did not correlate to CTE pathology
  - The DoD/USU Brain Tissue Repository has yet to demonstrate an association with blast exposure and CTE development

- **When seen, the extent of CTE pathology was most often only mild or minimally diagnostic**, and thus of questionable clinical significance.

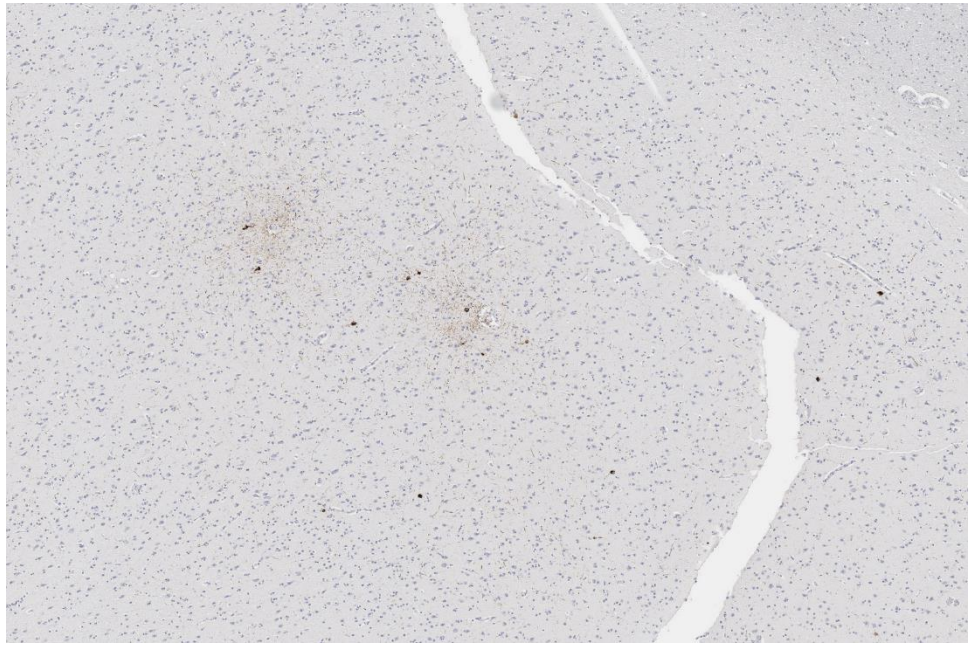
We know what this produces in the brain (American Football)

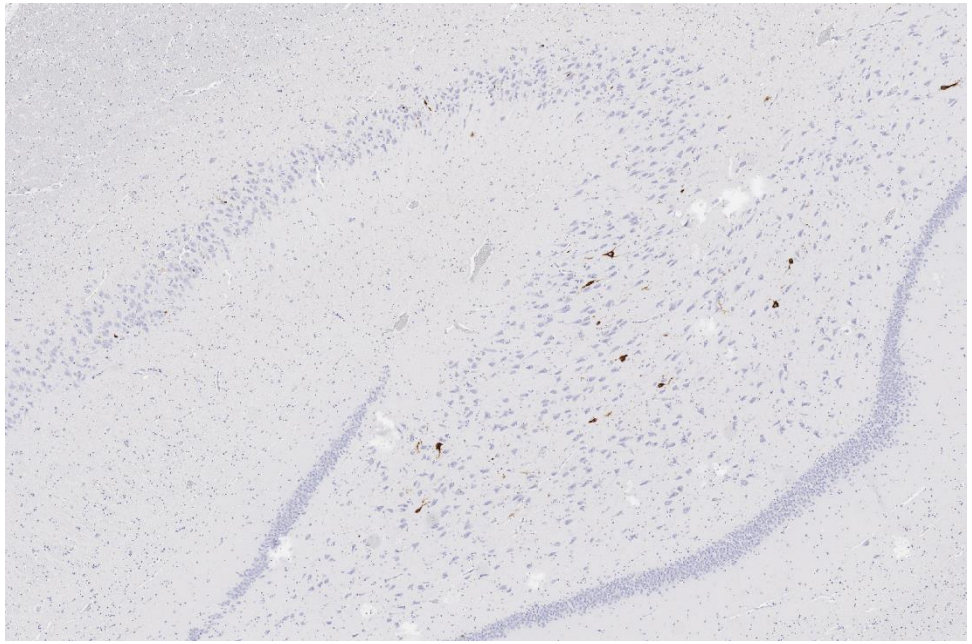
But what about the long-term effects on the brain of this activity?





A “typical” case of CTE in a former NFL football Player (age-matched to the next case you will see

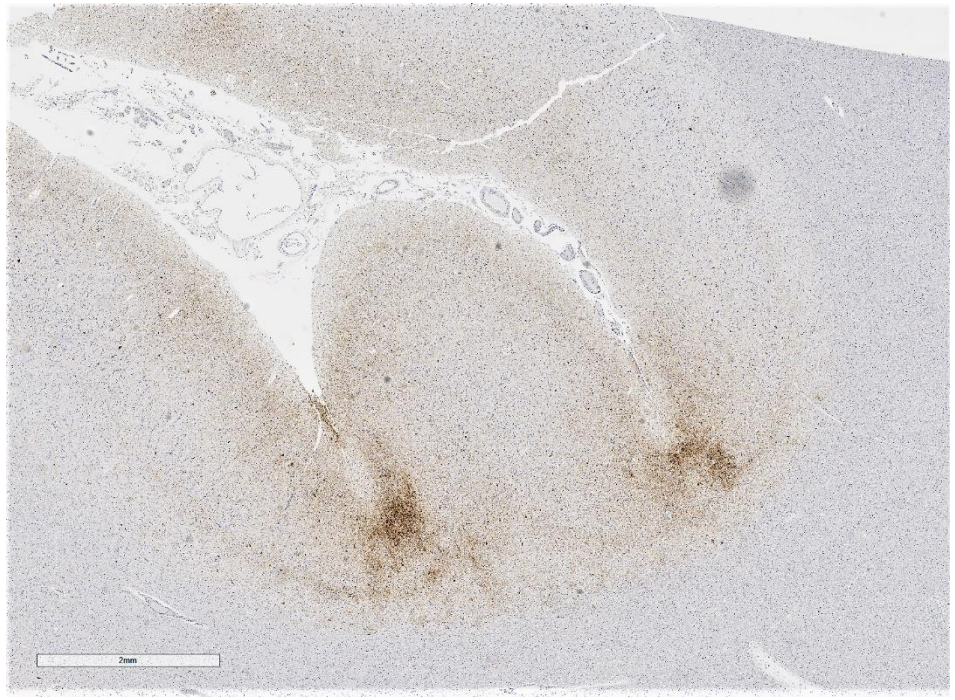


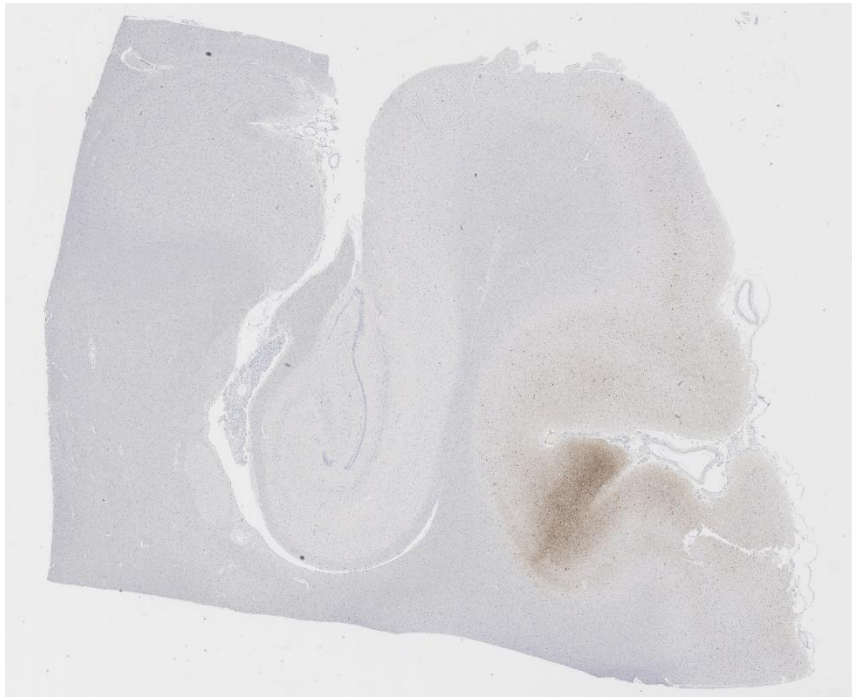


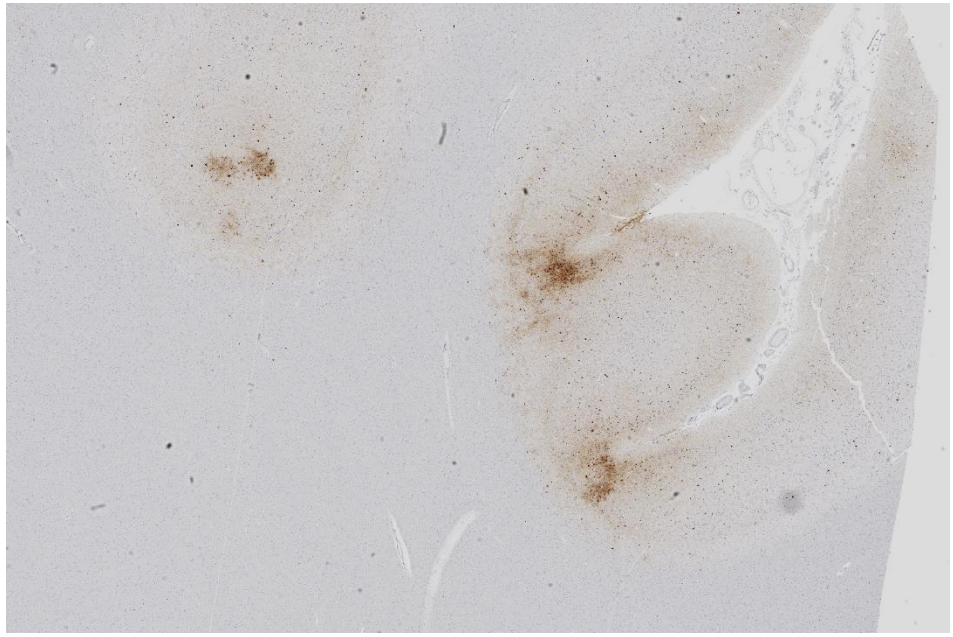
pTau stain(brown) showing CTE in a Hall-of-Fame former NFL player who died of suicide in his 40’s (no military history)

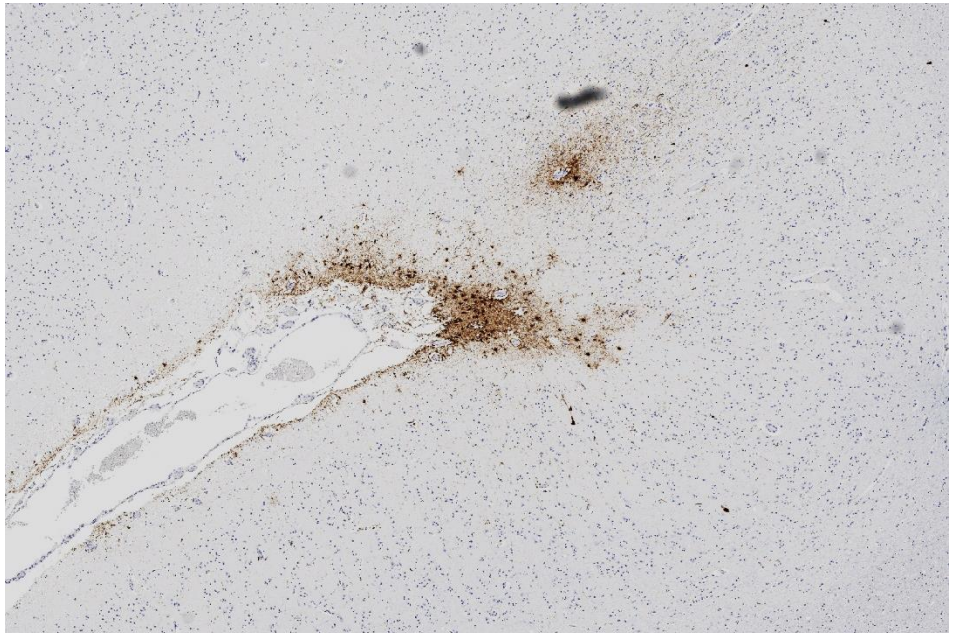
INDEX CASE:

- Retired SWCC driver in his mid-40's with at least 4 deployments during a 12-year career
- Contact sport history - played baseball in high school and "semi-pro"
- Had fired large weapons and reported several blast exposures
- After last deployment diagnosed with severe PTSD, alcohol abuse
- Severe headaches, 10 years; chronic sleep impairment
- In year prior to death, "Not thinking well." Difficulty putting sentences together when writing. Rapid mental decline.
- 7 month prior to death, hallucinations, paranoia (thought people were bugging his car, phone and house, thought he was being tracked)
- Said goodbyes to family
- Died by suicide with self-inflicted gunshot wound to the chest



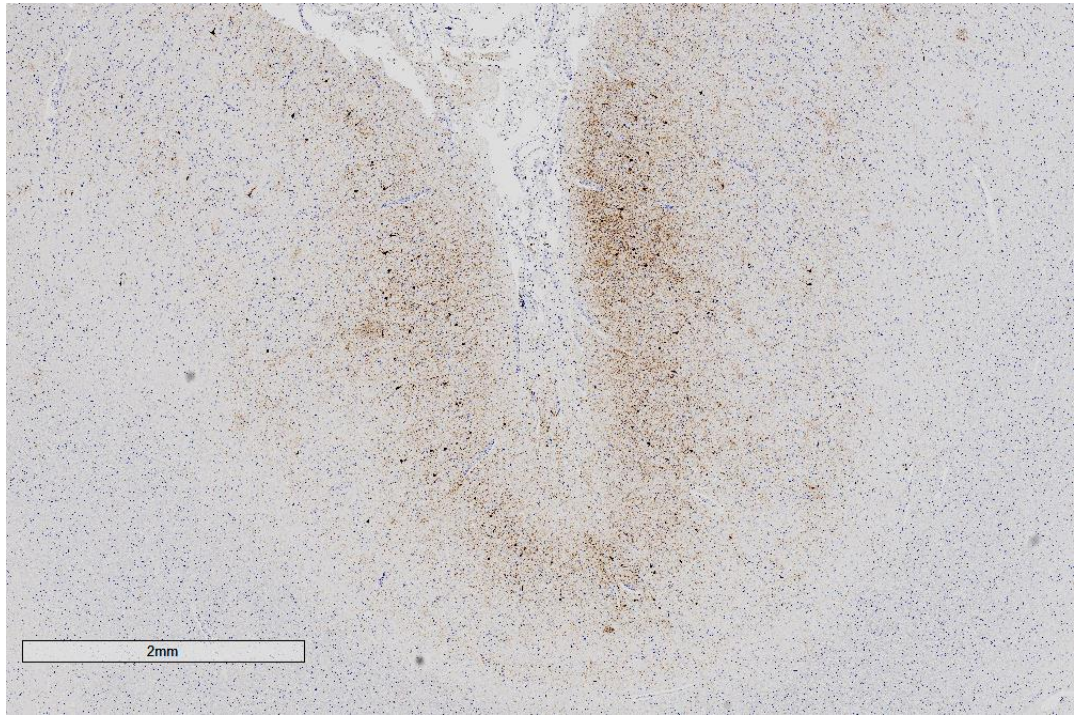


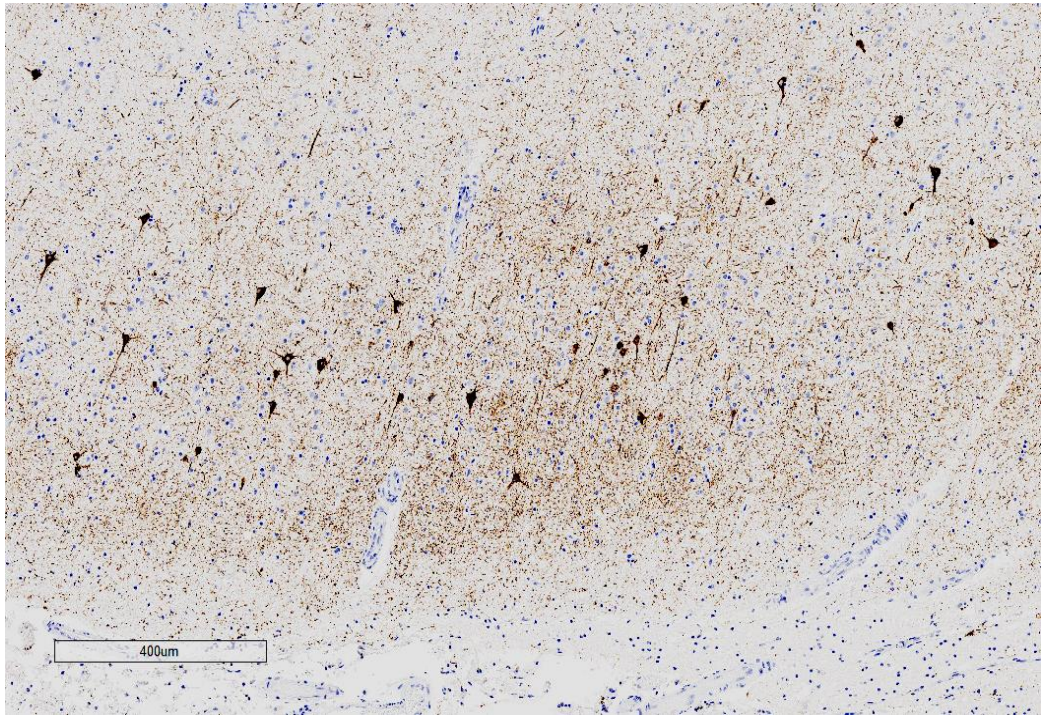


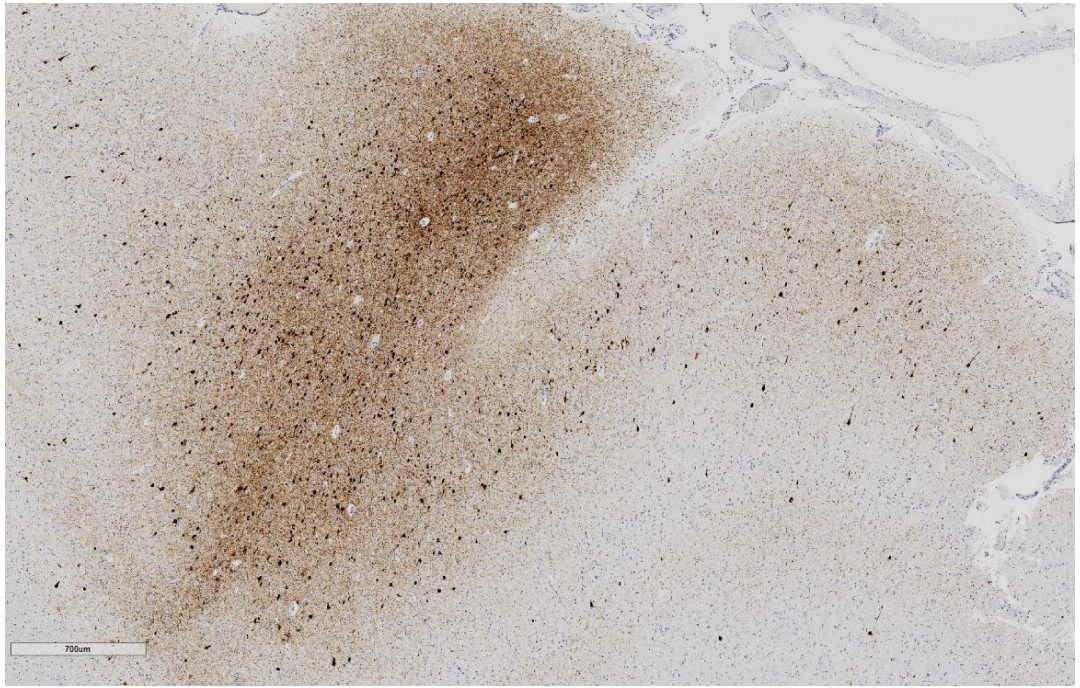


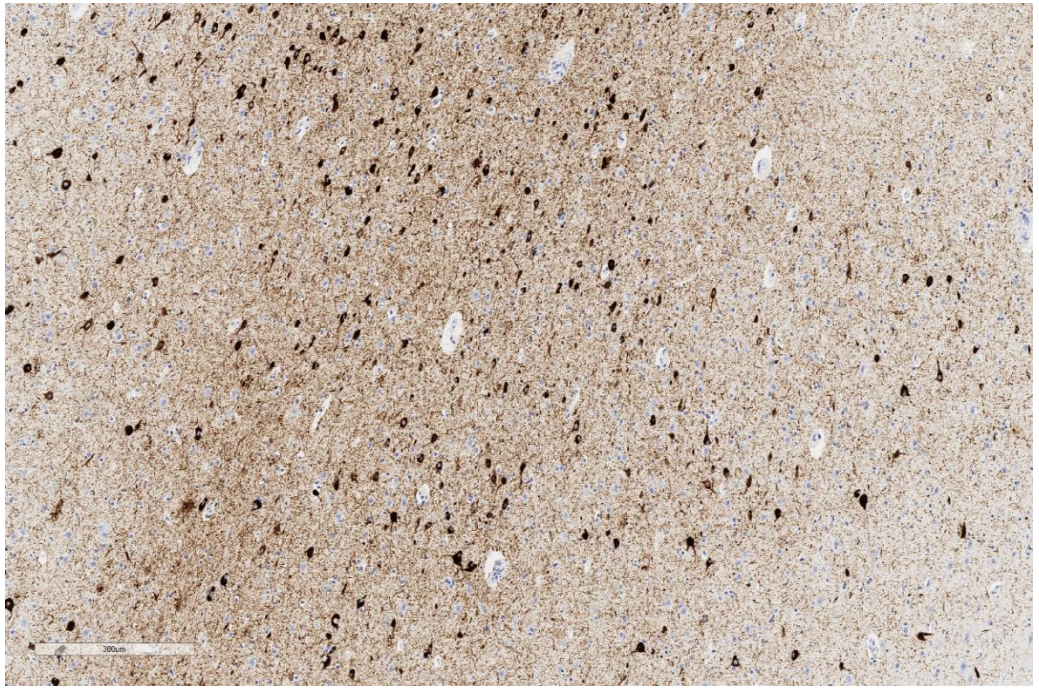
No Photographs Please

Multiple prominent *pTau* positive lesions (neurons and astrocytes) predominating in the depths of sulci of the neocortex. Brown stain = *pTau*. **Diagnostic of severe CTE**









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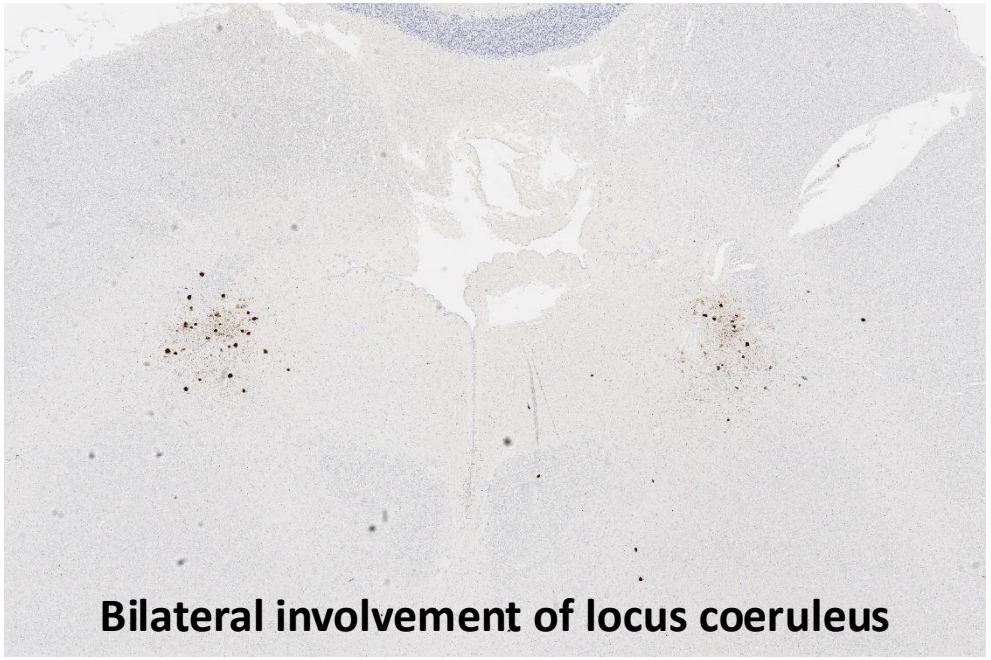
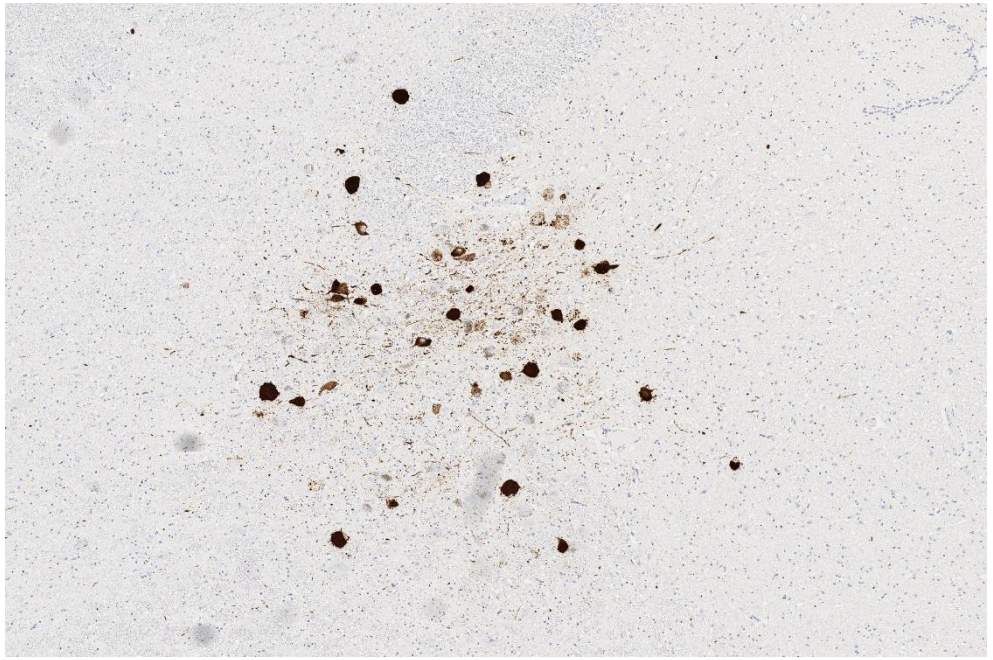
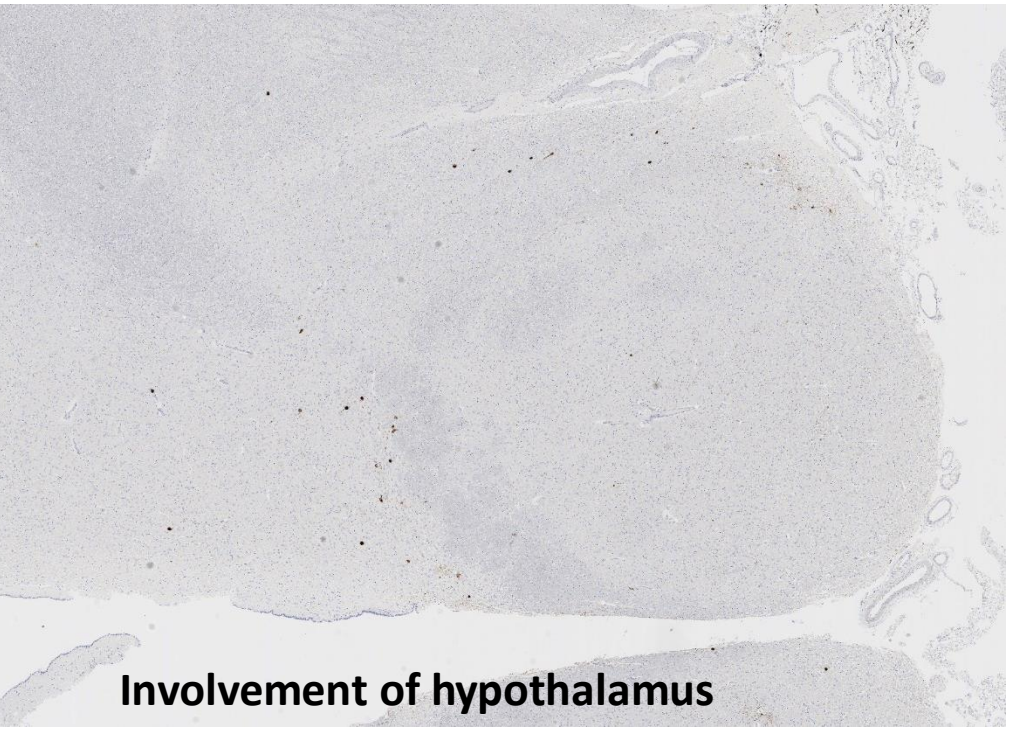
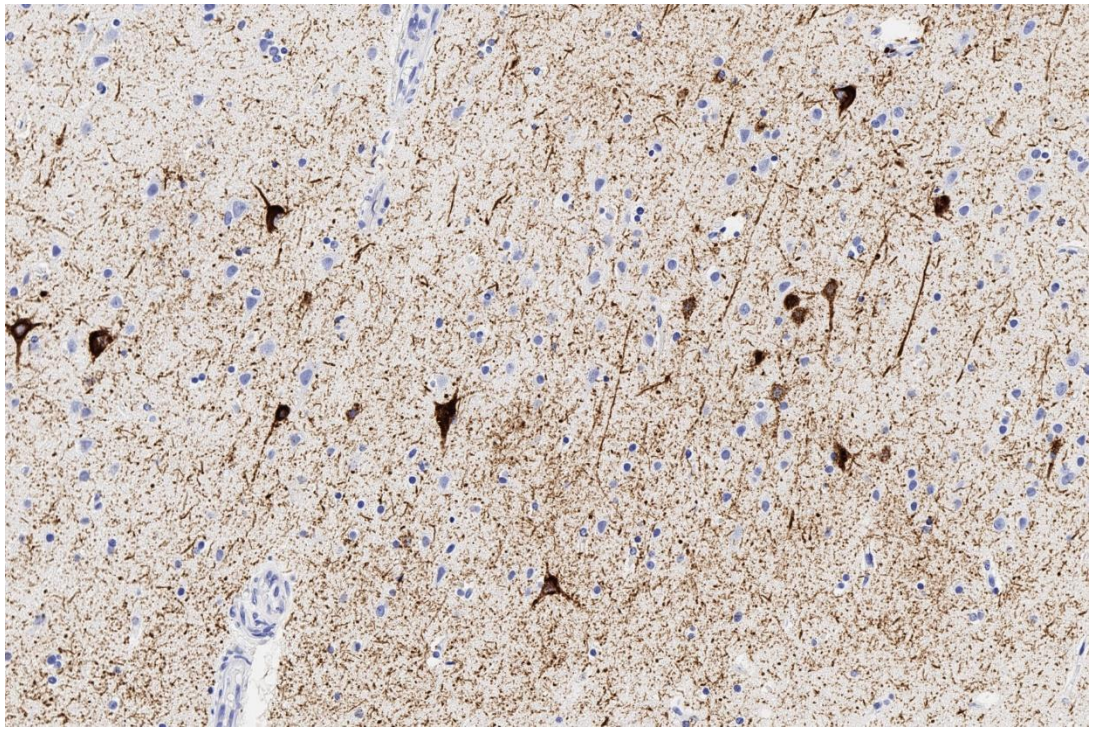


Bilateral involvement of locus coeruleus





Involvement of hypothalamus



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The subject's DNA was isolated and selectively sequenced

- Tau gene – normal
- All currently known tau-related neurodegenerative risk factor genes were tested (n=210) – all negative

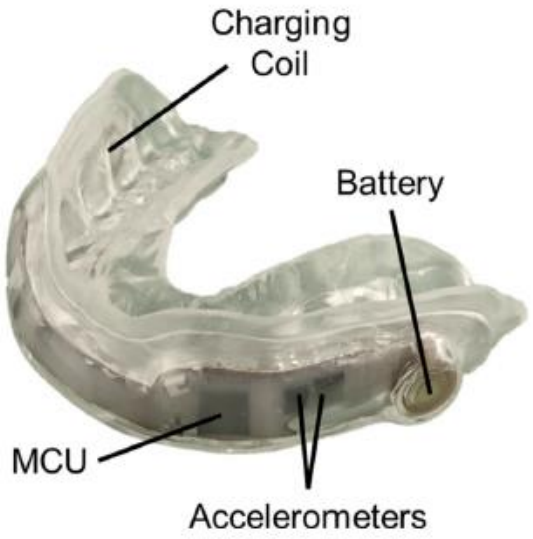
- We have now had the opportunity to examine the donated brain specimens from two additional former SWCC personnel.
- These two additional specimens did not show evidence of CTE.
- However, both were significantly younger than our index case.
- Based on experience with the examination of the brains of American football players (especially former NFL players) there is a latency between the incidents of impact TBI and the development of CTE. This latency typically is decades long.
- For the two negative cases, it is possible that not enough time had elapsed between exposure and death to develop this consequence.

How many “hits” does a fastboat operator sustain over a 12 year career?

How does this compare with the career of an elite American Football (NFL) player?

- Using mouthpiece accelerometer data employed during play and practice sessions, it has now been determined that an NFL (American football) player sustains an average of 11.5 head impacts/game that measure more than 10 g. Similar data was obtained for Division 1 collegiate football players. This data includes hits received in practice sessions (source - Gabler, LF, et al, 2025).
- Assuming a 20-year football playing career (college and professional) with an average total of 25 games/season, that equals a total **5,750 >10g** head impacts for the an elite NFL player.

Some player positions sustain differing rates of head impacts. Offensive linemen had the highest head impact rate with 13 >10g impacts/game or **6,500/career.**



A 1994 report provides data on SWCC wave impacts over a 6-day period of operation (Roesch, JR et al. Naval Surface Warfare Center report No. CSS/TR-94/38).

- They documented 8,610 boat impacts with waves (239.2 impacts/hour) with impact forces measuring from 2 to 25g and 4,492 impacts (124.9 impacts/hour) that were greater than 5g.

- A modest estimate of time spent in boating operations for a SWCC is 1500 to 1800 hours/deployment (however, lengthier cycles of deployment may result in 3000 hours, or more).
- The decedent we report here served a career with at least 4 deployments. This results in an estimated wave impacts experienced = **1,435,200 greater than 2g**.
  - And a total of **749,400*** impacts greater an 5g (*Training operations not included)

NFL total career impacts 6,500 (> 10 g)

vs.

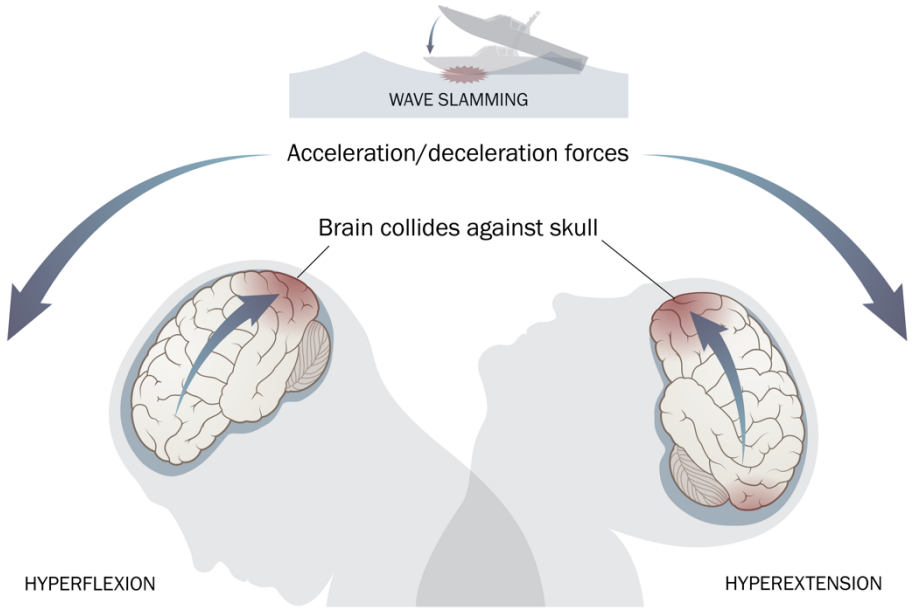
SWCC total career impacts 749,400 (>5g)

Does this case represent the “Mike Webster” of CTE in a fast boat operator? Webster was the first former professional American football (NFL) player to be documented with CTE. This observation stimulated subsequent studies leading to increasing concerns about elite contact sport participation as a risk factor for the of CTE.

Way Forward:

- These data call for further studies of fast boat operators.
- Brain functional impairment following a single outing (“post-mission hangover”, i.e. acute effects) and after a multi-year career (chronic, long-term effects). Assays of blood-borne fluid biomarkers, functional neuropsychological assessments, *tau* PET scans, etc. Comparison of newly recruited participants to long-term operators.
- Exposure dose – means are available for collecting or at least estimating such data.
- Sign up fastboat personnel as prospective brain donors (both active duty and especially retired operators). We are currently doing this in the US and can facilitate what is needed to initiate this elsewhere. If we can collect functional and exposure data on those who sign up, all the better.
- Do not selectively study symptomatic individuals – that introduces bias towards pathology.

Thank You For Your Attention



Questions Comments

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USU DOD BRAIN TISSUE REPOSITORY

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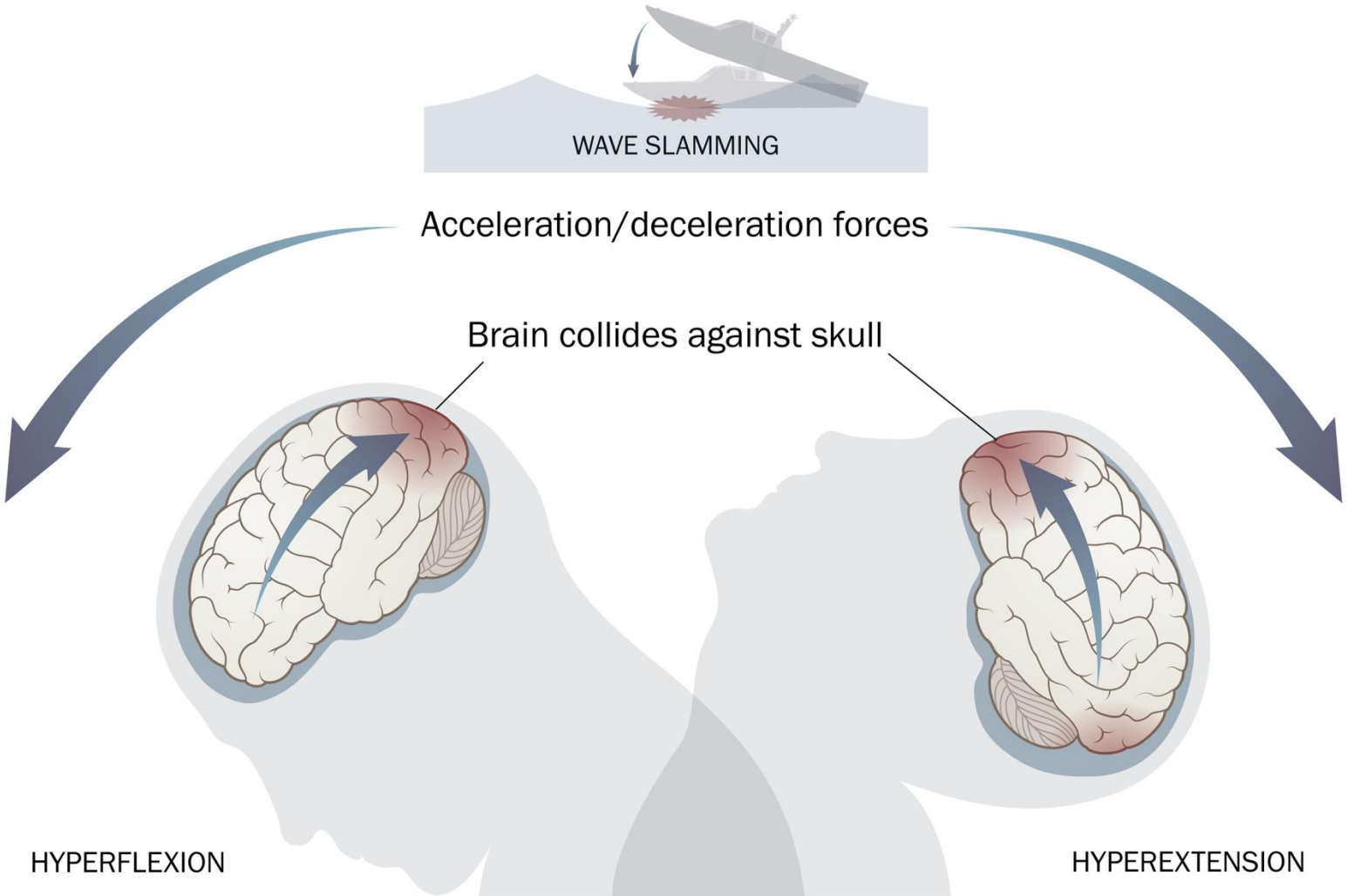
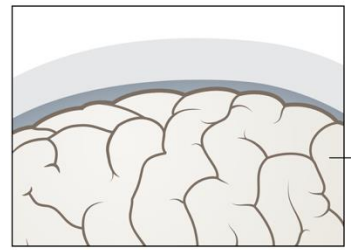
Acceleration/Deceleration Injury

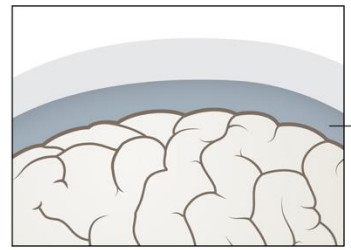


CHANGES IN BRAIN VOLUME



Maximum brain volume

17 YO



Decreased brain volume, increased movement through CSF

37 YO

Parasagittal space and cerebrospinal fluid (CSF) flow across the lifespan in healthy adults Hett, K, McKnight, CD, et al. *Fluids Barriers CNS* (2022) Mar 21;19(1):24.

The brain atrophy associated with aging provides more space between the inner table of the skull and the brain – and begins in the early 20's.