# **CHAPTER I**

# Mental Health Issues

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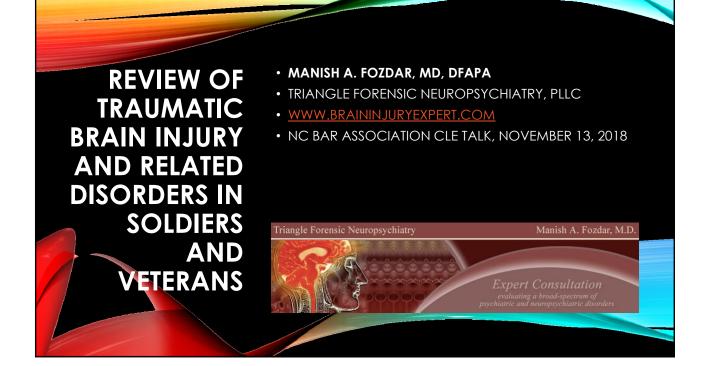


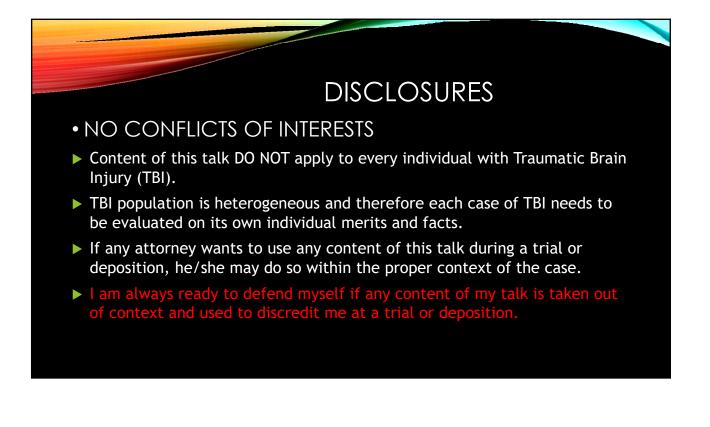
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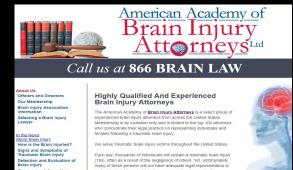
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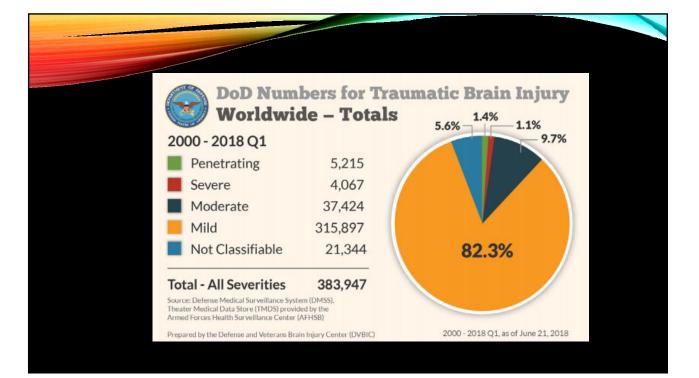


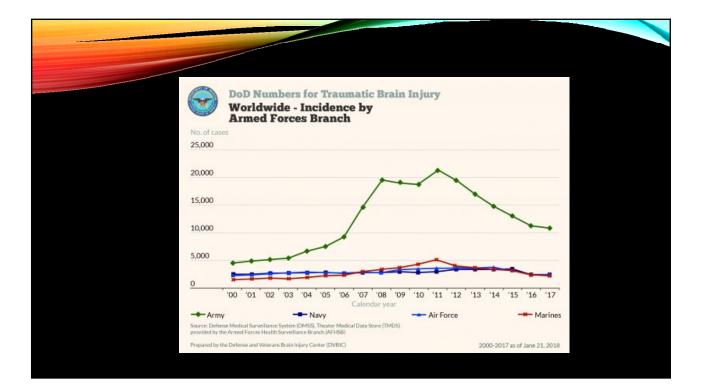
# YOUR THIRST FOR KNOWLEDGE!

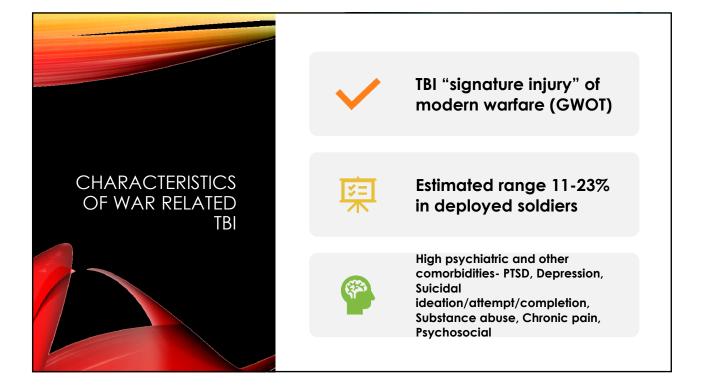
- Google search as of 9/28/2013 on 'Brain Injury Lawyers' produced 4.9 million results
   in 0.30 seconds!!!
- Google search as of 11/09/2018 on 'Brain Injury Lawyers' produced 22,100,000 results in 0.58 second!!

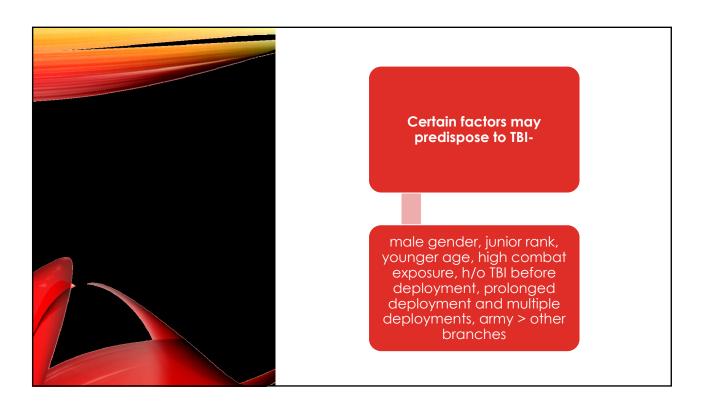














### • Most common mechanisms of TBI:

- Blast- 33.1%
- Object hitting the head- 31.7%
- Fall- 13.5%

	Single head injury	Multiple head injuries		
Outcome	n (%)	n (%)	$\chi^2$	P value
PTSD	26 (28.0)	62 (62.3)	22.5661	<0.0001
Depression	41 (44.8)	61 (61.7)	5.4111	0.0200
Suicidal ideation	16 (17.3)	31 (31.4)	5.1371	0.0234
Violence	16 (16.9)	20 (19.8)	0.2674	0.6051
Back pain	50 (54.0)	74 (74.8)	9.0900	0.0026
Headache	55 (59.8)	63 (63.8)	0.3254	0.5684
Any pain	52 (56.5)	74 (75.4)	7.5518	0.0060

# A NEW CONTRACTOR

# **HHS Public Access**

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Traumatic Brain Injury in Iraq and Afghanistan Veterans: New Results from a National Random Sample Study



#### • Primary Blast Injury

- Direct effect of the blast wave
- Due to dynamic pressure changes
- Occurs in air filled organs- lungs, ears, kidney
- May cause concussion

### • Secondary Blast Injury

\*Object strikes the body

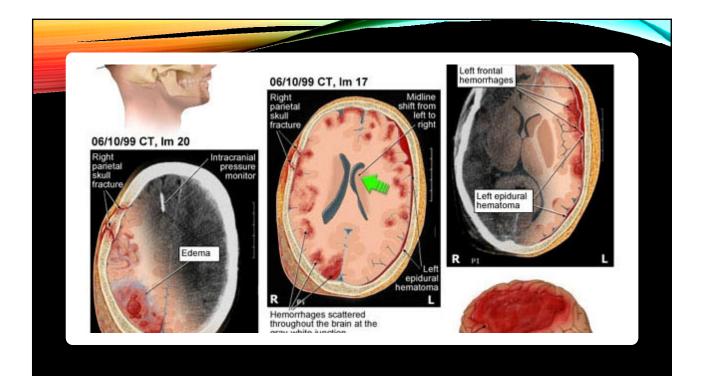
### • Tertiary Blast Injury

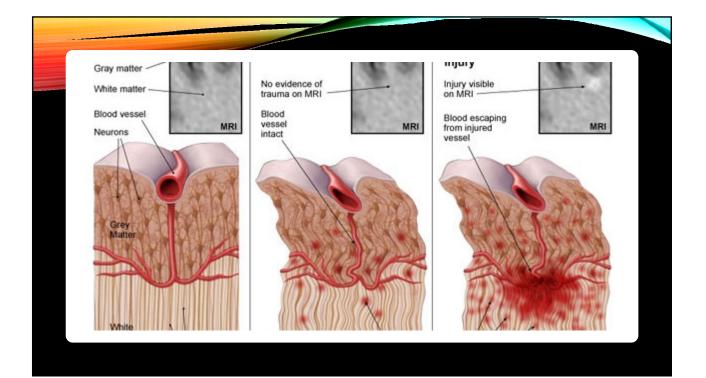
Individual strikes the object

Quaternary Blast Injury

Other effects- burns, crush injuries, infections, delayed collapse

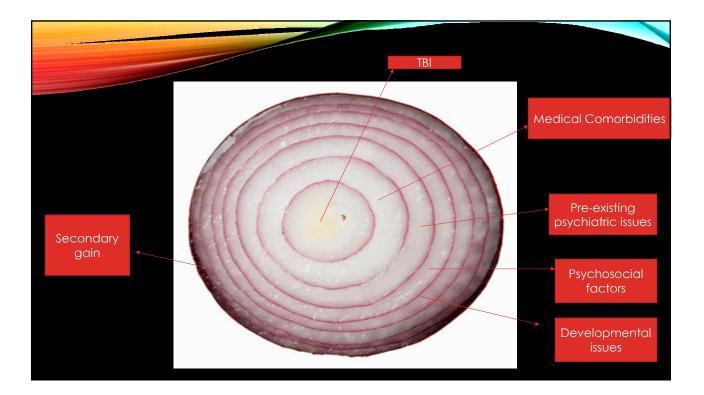
PRIMARY EFFECTS	SECONDARY EFFECTS
Contusions	* Hematomas- epidural
<ul> <li>Diffuse Axonal Injury</li> </ul>	subdural
	intracerebral
	* Cerebral Edema
	* Hydrocephalus
	* Intracranial Hypertension
	* Infection
	* Neurotoxicity
	- Calcium Influx , Excitotoxicity (NMDA)
	Lipid Peroxidation, Phospholipase
	activation
Neurochemical Changes after	er TBI
<ul> <li>Mixed data</li> </ul>	
<ul> <li>Alterations in levels of NE, 5</li> </ul>	5HT, DA, Ach, Aspartate, Glutamate, Glycine, GABA

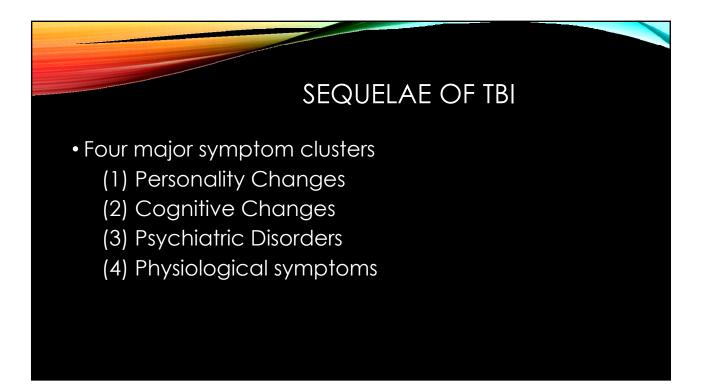












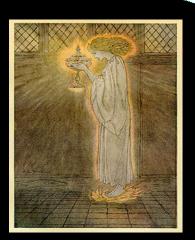
### NEUROPSYCHIATRIC ASSESSMENT OF ALLEGED TBI CASE.

- History Taking
- History Taking
- History Taking
- Most patients with TBI don't volunteer a lot of information.
- Obtain collateral information from family, friends, coworkers, medical records, accident reports, injury reports, video surveillance......
- Thorough neuropsychiatric evaluation including neurological exam.
- Brain Imaging- CT, MRI, SPECT, PET, MRS, DTI
- EP Studies- EEG, Computed EEG, BEAM
- Labs
- Neuropsychological Testing



# • Lewis (1942) referred to it as- that common dubious psychopathic condition, the bugbear of the clear-minded doctor and lawyer.

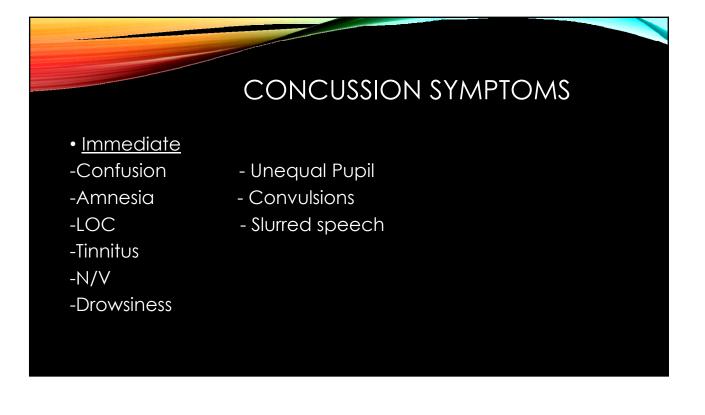
- Term concussion derived from Latin term 'concutere'- to strike together
- Holy Grail of TBI litigation and TBI scientific debate in academia

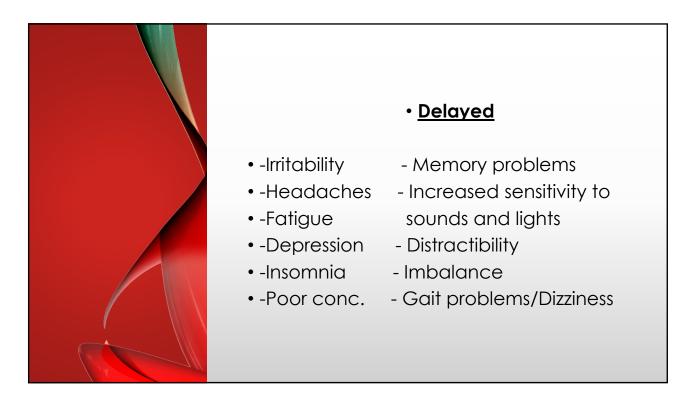


## TBI CLASSIFICATION

Mild	Moderate	Severe
Normal structural	Normal or abnormal	Normal or abnormal
imaging	structural imaging	structural imaging
LOC = 0-30 min	LOC >30 min and	LOC > 24 hrs
	< 24 hours	
AOC = a moment up to 24 hrs	AOC >24 hours. Severity based on other criteria	
PTA = 0-1 day	PTA >1 and <7 days	PTA > 7 days
GCS=13-15	GCS=9-12	GCS=3-8

Table 2. Classification of Mild TBI (Concussion)					
Grade	e Ca	intu	Colorado	AAN	
Grade		) LOC; A < 5 min	No LOC; confusion without amnesia	Transient confusion; no LOC; Concussive symptoms resolve < 15 min	
Grade		DC < 5 min; "A > 30 min	No LOC; confusion with amnesia	Transient confusion; no LOC; Concussive symptoms last > 15 min	
Grade	PT	DC > 5 min; `A > 24 urs	Any LOC	Any LOC either brief (seconds) or prolonged (minutes)	







- Person with complicated mTBI-
- Intracranial lesions such as contusion, hemorrhage etc.
- Well circumscribed posttraumatic amnesia (PTA).
- May be h/o recent concussion.

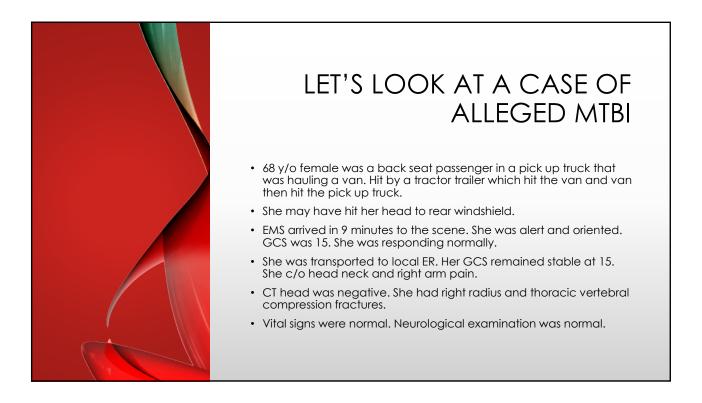
• Outcome may be similar to those with moderate TBI.

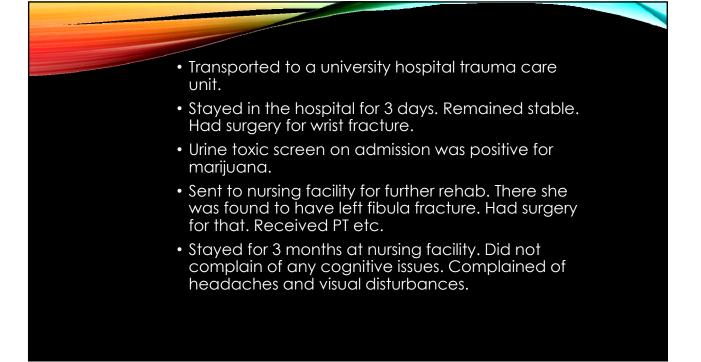


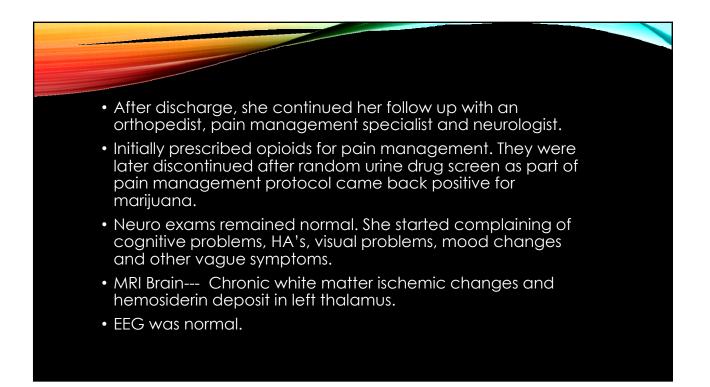
- No evidence of intracranial lesions on CT/MRI
- No evidence of well circumscribed PTA

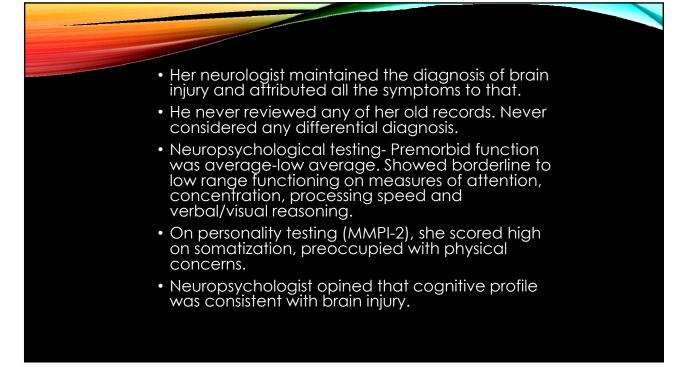


- ✓ Outcome is different. Symptoms usually resolve after 3-6 months. Anything beyond that, Persistent Post-Concussive Syndrome (PPCS), is usually due to underlying psychological factors including involvement in litigation.
- ✓ Also consider other comorbid medical issues.





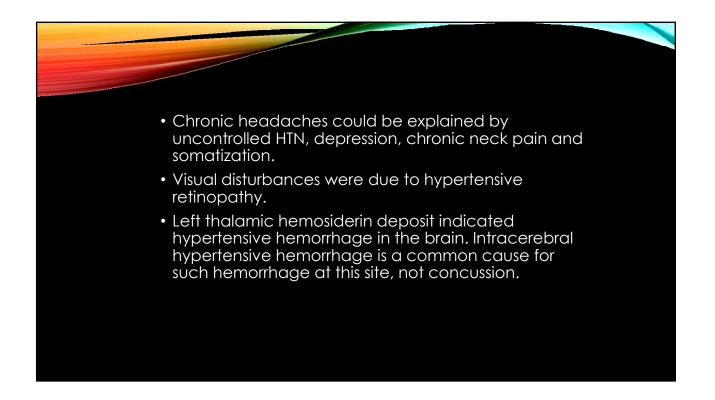




	VIEW OF PRE-INJURY IEDICAL RECORDS.
<ul> <li>Chronic history of</li></ul>	<ul> <li>Tested positive for</li></ul>
uncontrolled hypertension. <li>Refused to take medicines</li>	marijuana several times. <li>Treated extensively for</li>
to treat HTN. <li>Obesity, renal insufficiency</li>	chronic neck and back
and hyperlipidemia. <li>Chronic complaints of HA's</li>	pain. <li>Treated for anxiety and</li>
and visual disturbances. <li>Eye examination had</li>	depression. <li>Lost job in 2003, husband</li>
revealed evidence of	was imprisoned and she
hypertensive retinopathy.	had financial stressors.

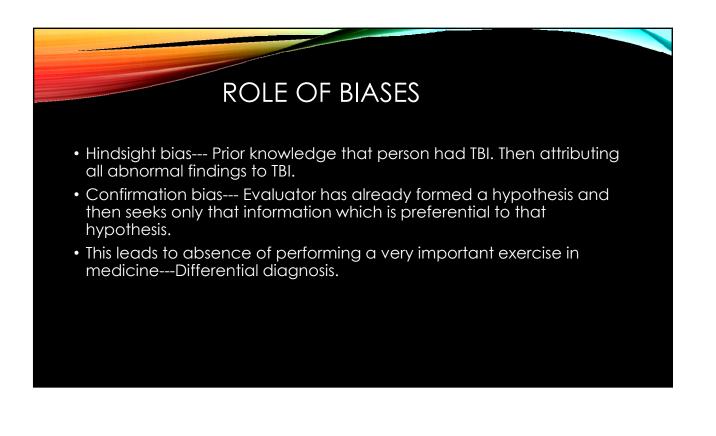
# ANALYSIS OF THE CASE.

- Questionable whether plaintiff even suffered from any mTBI based on the objective data.
- Plaintiff's experts (treating providers) did not objectively verify all the data.
- With the presumed "history" of brain injury, they attributed all her symptoms to brain injury.
- Almost all her complaints post-injury were present chronically dating back 10 years.
- Minor cognitive deficits documented on her NP testing could be assertively attributed to her cerebrovascular disease, chronic marijuana use, depression and chronic pain.



### WHAT WENT WRONG?

- Science v. Pseudoscience.
- Evasion of peer review, absence of self correction and emphasis on confirmation than refutation.
- Essence of science is bending over backwards to prove oneself wrong (Feynman).
- Faulty logic- Brain damaged people perform poorly on NP testing. Therefore, poor performance on NP testing is indicative of brain damage.
- Criteria and protocol driven practice as opposed to descriptive and analytical practice leads to over diagnosis, misdiagnosis and faulty causation analysis.



### DIFFERENTIAL DIAGNOSIS IN MTBI CASES.

#### • <u>Psychiatric disorders</u>

Depression, Bipolar disorder, Schizophrenia......

- Neurocognitive deficits are well described in these conditions.
- Verbal learning, memory, attention/concentration, processing speed, visual spatial functions, executive functioning including planning and organization.
- May persist even in remission state, especially in elderly.

■Somatization is common in certain patients.

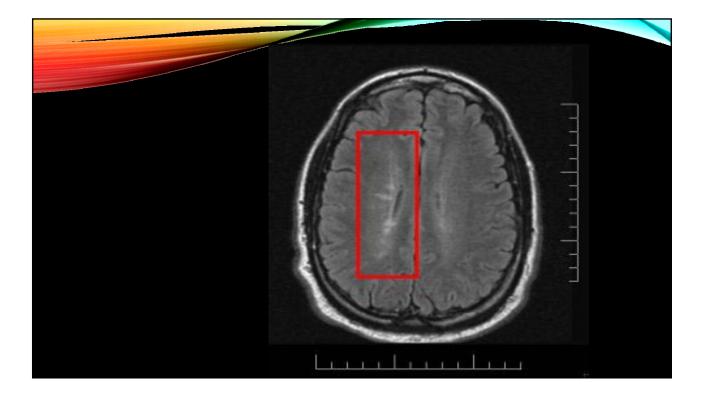


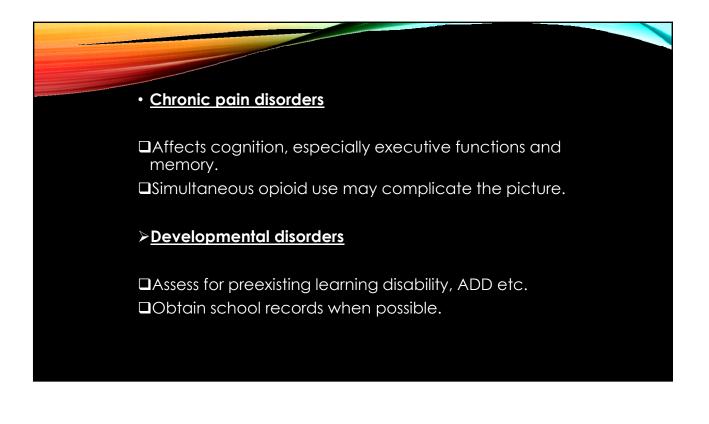
#### Medication side effects

Polypharmacy has become a norm.

- List of medications that can cause cognitive side effects is exhaustive.
- Denzodiazepines, anticholinergics, antihistamines, antiepileptics, corticosteroids, certain cardiac medications like digitalis and beta blockers, opioids, fluoroquinolone antibiotics and H<sub>2</sub> antagonists.
- Delirium and dementia can occur due to medications.



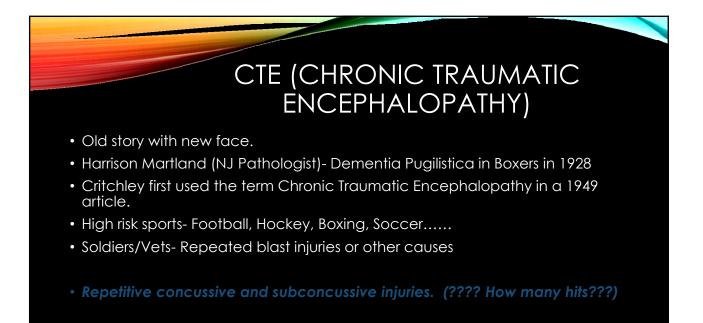




#### Malingering

Must consider this diagnosis in patients with persistent symptoms that do not improve with treatment.

□Various ways to support your diagnosis.....NP testing, Clinical observation, collateral information, inconsistency between self reported symptoms and objective evidence, attorney shenanigans!



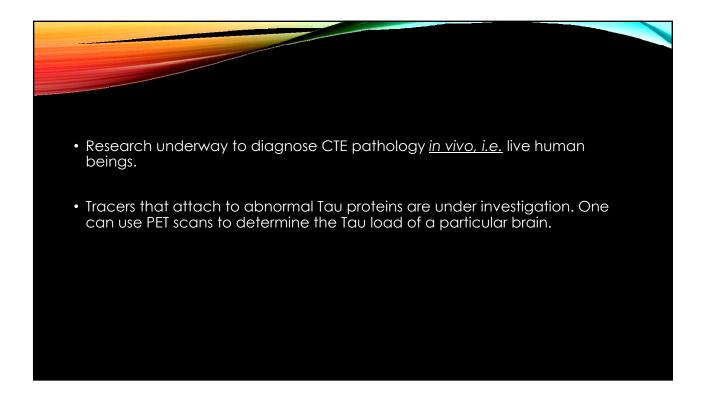
# CLINICAL PRESENTATION OF CTE

- CTE remains a NEUROPATHOLOGICAL diagnosis.
- Ann McKee and her group described clinical and neuropathological characteristics of CTE based on their analysis of 85 brains of individuals (athletes and veterans) with history of repetitive brain injuries.
- Proposed various Stages of CTE
- Proposed clinical symptom correlation with various pathological stages.



# CLINICAL SYMPTOMS

- Cognitive- attention, memory, language, visuospatial. Executive function
- Psychiatric- Depression, Suicidal ideation, Aggression, Paranoia, PTSD
- Neurologic- Parkinsonism, Movement disorder, Motor Neuron Disease



Definition Formation Forma

• <u>Chronic Traumatic Encephalopathy:</u> Psychiatric Expert Testimony: Emerging Applications. Edited by Kenneth J. Wise, MD and Clarence Watson, JD, MD. 2015. Oxford University Press