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POST TRAUMATIC HEADACHES

Post Traumatic Headache

- “He thrust unto him with his horse , and smote him on high on the helm, a great stroke, and astonied him sore” (Malory Morte D’Arthur)
- Little mention of headaches in ancient epics –plenty” falling into swounds”

Post Traumatic Headache

1861 Waller Lewis- headaches, sleep disturbances, tinnitus, spinal pain and tenderness in absence of signs.

Erichsen (1866) “On railway spine and other injuries of the nervous system”

Gowers (1886) persistent pain caused by a state of sensitization of the nerves which persists despite “the sovereign balm” of compensation.

Post Traumatic Headache

Charcot (1889) manifestations of hysteria,"psychonervous commotion"

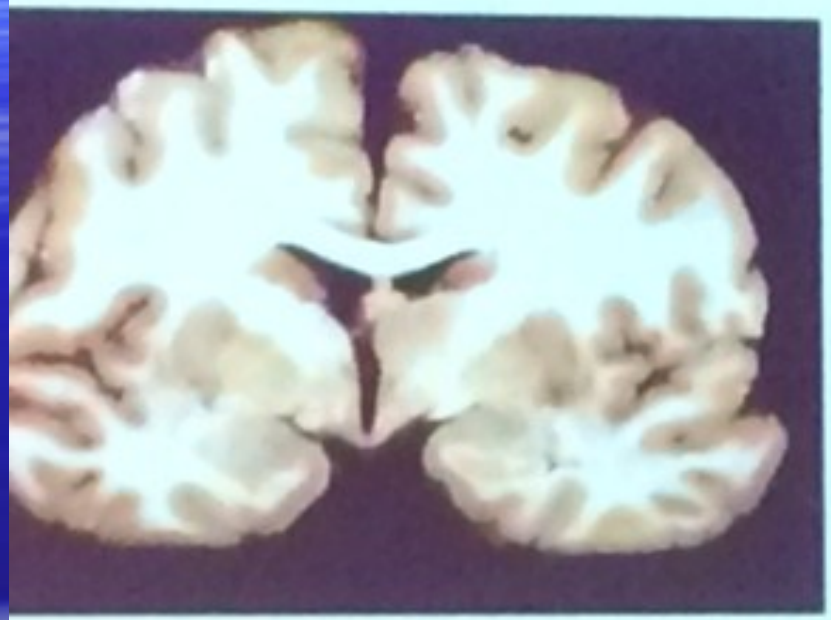
Oppenheim (1901) "traumatic neuroses" the combination of psychic and physical induced "molecular alterations"

Mitchell (1866) "and felt no more in heart and brain the weary weight of sin and pain"- described neuropathic pain

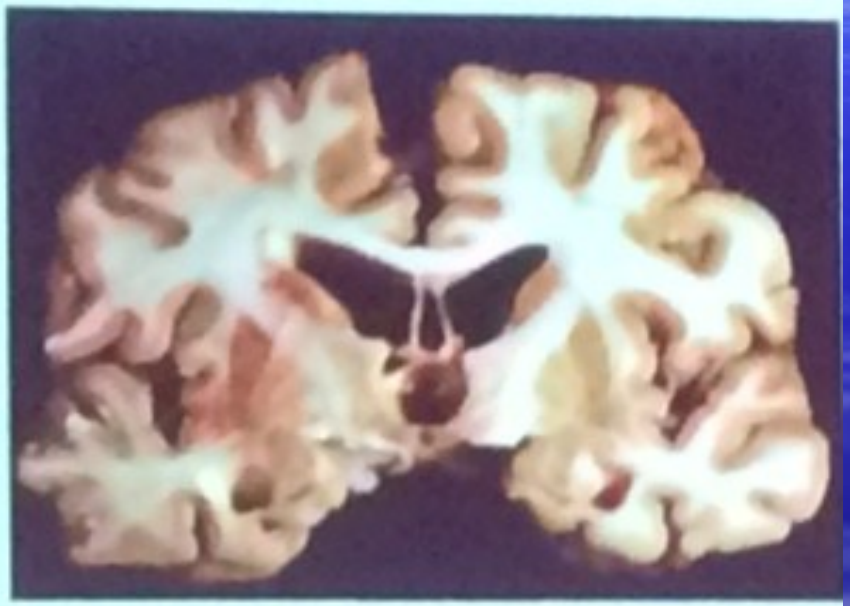
Freud (1905) the train is a metaphor for sex

Chronic Traumatic Encephalopathy - CTE

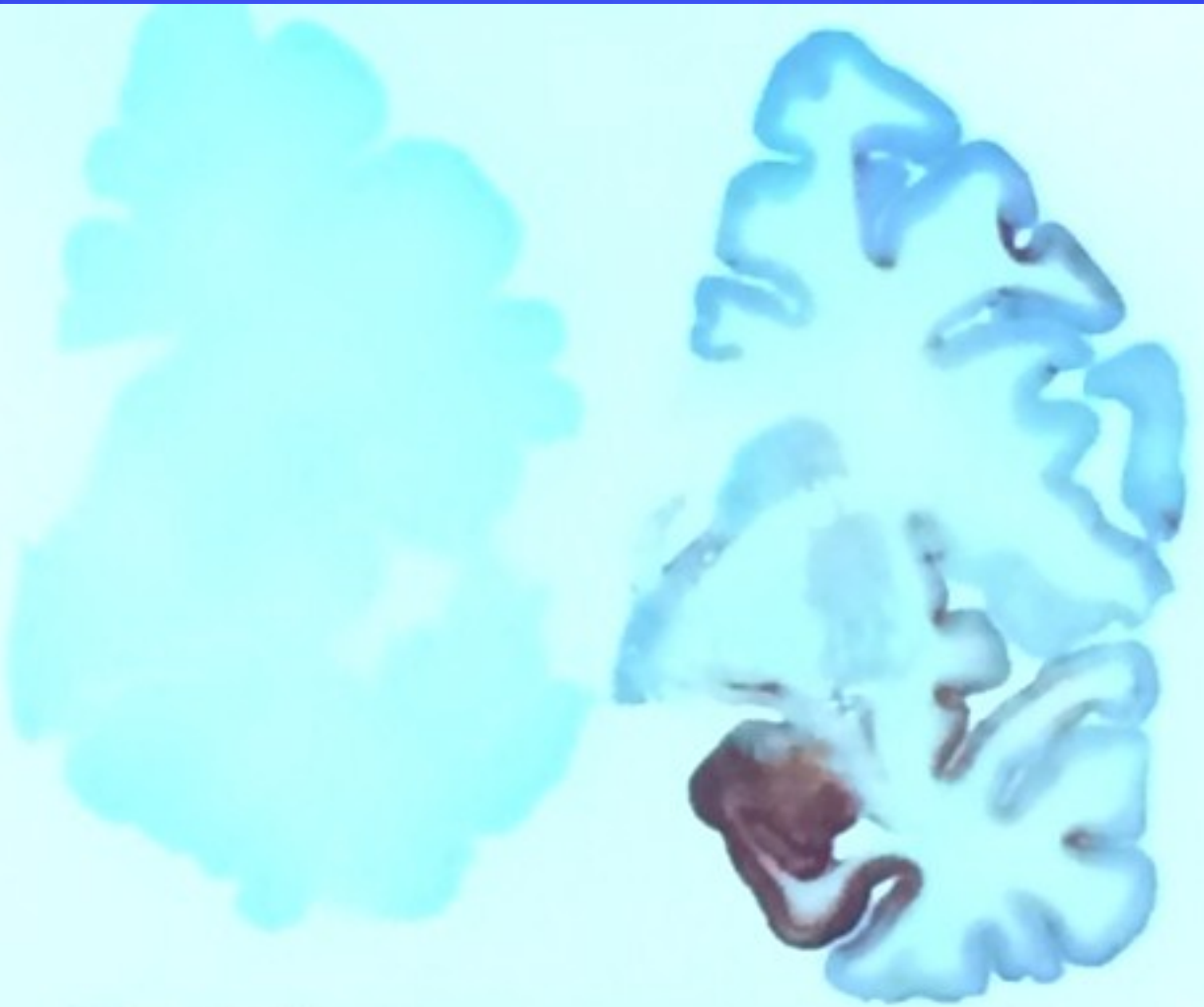
Normal



Advanced CTE



Courtesy Ann McKee, MD, Boston University Alzheimer's Disease Center



Courtesy Ann McKee, MD, Boston University Alzheimer's Disease Center

Post Traumatic Headache

Henry Miller (1961)

No one returns to work before settlement

Never occurs if no hope for compensation

Never follows severe head injury

Never occurs in professionals

Patients symptom free after compensation

Never occurs in sports injuries

Post Traumatic Headache

Reginald Kelly (1975, 1981)

76% returned to work pre settlement

79% were professionals

46% severe injury

26 of 151 back at work at time of settlement

Post Traumatic Headaches

- ICHD criteria- headache occurring within 7 days of injury-but?
- Acute or chronic with the same phenotypes as primary headaches
- The head has a limited number of ways it can express itself to us to gain our attention

Post Traumatic Headache

- Tension Type Headache
- Migraine w/o aura
- Cluster Headache
- Other Trigeminal Autonomic Syndromes

Migraine Without Aura

- 5 recurrent headaches
- Lasts 4 to 72 hours
- With 2/4
  - Unilateral
  - Pulsating (throbbing) quality
  - Worsening of the headache with movement
  - Moderate to severe
- Accompanied of 1/2
  - Nausea or vomiting,
  - Aversion to light, sound and/or osmophobia
- Not attributed to another disorder

Post Traumatic Headache

- **Acute PTH associated with moderate or severe head trauma(5.1.1**
- **A** Headache , no typical characteristics
- **B** Head trauma: loc >30 mins, GCS <13, PTA>48 hours, imaging of a brain lesion
- **C** Headache develops within 7 days post trauma or after regaining consciousness
- **D** One or other of the following; 1) Headache resolves within 3 months,2) Headache persists but 3 months have not passed

Post Traumatic Headache

Acute PTH attributed to mild head injury (5.1.1.2)

- **A** Headache , no particular characteristics
- **B** Headache with all of the following: 1)Either no LOC or LOC <30 mins duration, 2) GCS 13, 3) Symptoms and /or signs of concussion.
- C Headache develops within 7 days after head trauma
- D One or other of the following 1)Headache resolves within 3 months of the trauma, 2) 3 months have not yet passed.

Chronic Post Traumatic Headache

- Same criteria as for acute pth but persist for more than 3 months after trauma
- Can simulate any primary headache
- Mechanisms poorly understood.

Post Traumatic Headache

- **Symptoms:** Headache plus nausea, dizziness, vomiting, orthostatic and thermal dysregulation, neurasthenic depression, cognitive defects, irritability, photophonophobia.
- TTH: dull,pressing
- Cervicogenic: dragging, triggerable
- Migrainelike: pulsating
- Cluster-type: stabbing, pulsating, dragging

Post Traumatic Headache

Causes

MVA	42%
Falls	23%
Assaults	14%
Sports	6%

Post Traumatic Headache

- **Postcraniotomy Headache**
- **Acute**
- **Chronic**

  More often after infratentorial procedures, prevented by osteoplastic procedures, not using fibrin, avoiding extensive drilling of IAC, duraplasty

Post Traumatic Headache

Case History

A 34 year old labour lawyer,accident Jan 23rd 1998. Hit head in occipital area. Pain started there and then generalised. Sleepy. No energy or concentration, right arm tremulous, irritable, some loss of smell. O/E mild nystagmus to left, tender back of neck and numb R occipital nerve distribution.

Rx Amitriptyline and Naprosyn

25th May 1999- rear ended– daily headache

Botox every 3 months to present- good response

Post Traumatic Headache

Whiplash Associated Disorder

Grade 0: no complaints no signs

Grade 1: neck pain, stiffness, or tenderness

Grade 2: neck complaints + musculoskeletal signs, reduced neck mobility and/ or tender muscles

Grade 3: as above + neurological signs, reflexes, weakness, sensory deficits

Grade 4: fracture or dislocation

Post Traumatic Headache

Headache in WAD

Headache in 40-80% of acute Whiplash (Di Stefano 1995)

34% persistent neck pain within year of injury, 14% neck pain for more than 6 months (Bovin et al 1994)

3 years after injury headache in 82%, 46% occipital, 34% generalized, constant in 50%(Balla et al 1987)

Post Traumatic Headache

Pathophysiology

Peripheral damage to scalp, neck and jaw

Central disturbances

Secondary sensitization

Blood flow disturbances

Chronic Daily Headache

- Definition
- Classification
- Pathophysiology
- Relationship between Classification and pathophysiology

Headache Disorders Causing Chronic Headache

Primary Headache

-Chronic Migraine

-Chronic TTH

-Hemicrania Continua

-New Daily persistent Headache

Secondary headache

-Chronic Headache…head injury

-Chronic headache…whiplash

-Med Overuse

-Cervicogenic headache

-other secondary headache

Pathophysiology of Migraine

- Central sensitization of pain systems after repeated bouts of pain
- Damage to CNS pain modulating systems
- CNS changes secondary to med overuse
- Abnormal focal neuronal activity (CNS pain generators)
- Persistent activity in a peripheral pain generator

Pathophysiology of Migraine

Implementing Pathophysiology Into Treatment

- Focus had been on acute therapy to manage individual migraine episodes
- New advances in pathophysiology have transformed the concept of what migraine is
  - Migraine is **a CNS** disorder
  - Genetic predisposition
- This has paved the way for improved treatment
  - Treatment of migraine as a disorder
  - Emphasis on preventive + acute

Pathophysiology of Migraine

The Genetic Basis

- P/Q type Ca^{++} channel
  - Presynaptic
  - Voltage gated
  - Occipital cortex
  - Trigeminal nucleus caudalis
  - Linkage to chromosome 19
- Na-K ATP Pump
  - Linkage to chromosome 1

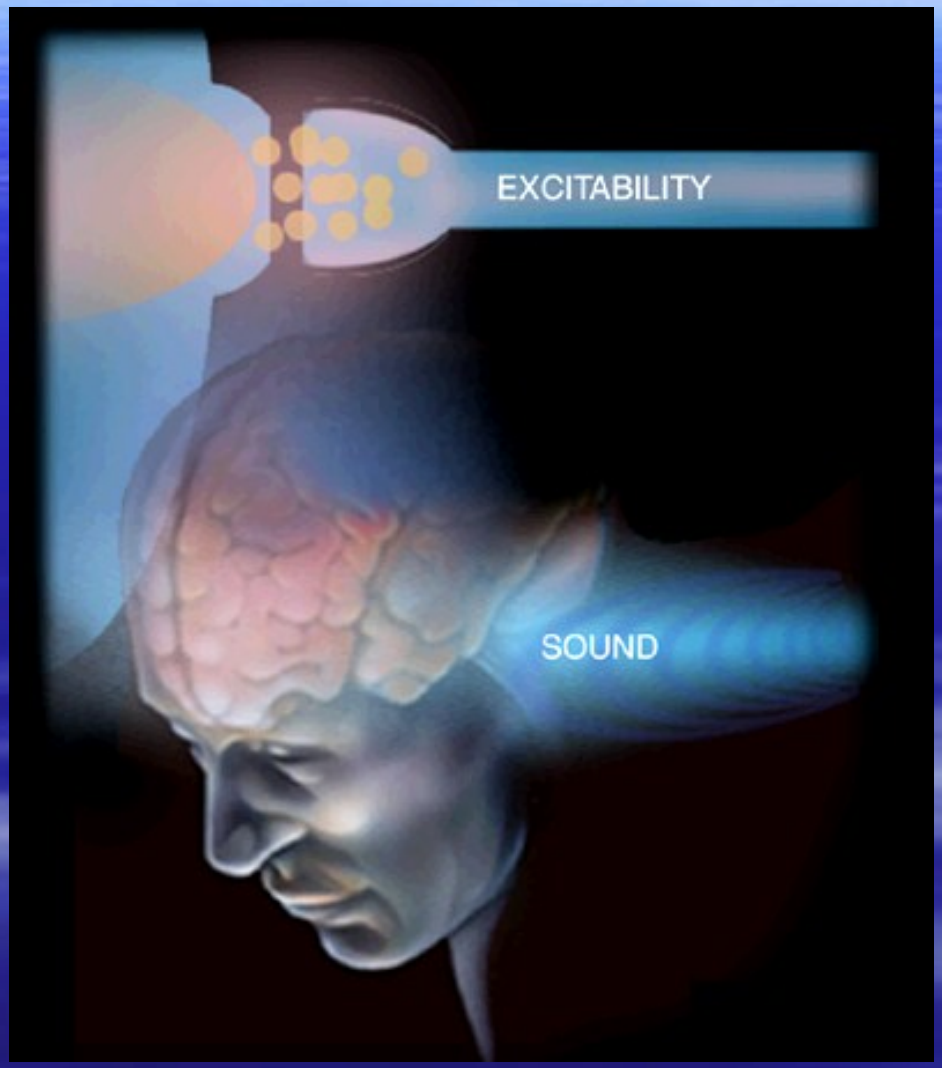


Figure courtesy of AHS Ambassadors Program.

Obhoff et al. *Cell*. 1996;87:543-552. De Fusco M et al. *Nat Genet*. 2003;33:192-196

Pathophysiology of Migraine

Hyperexcitable Cortex

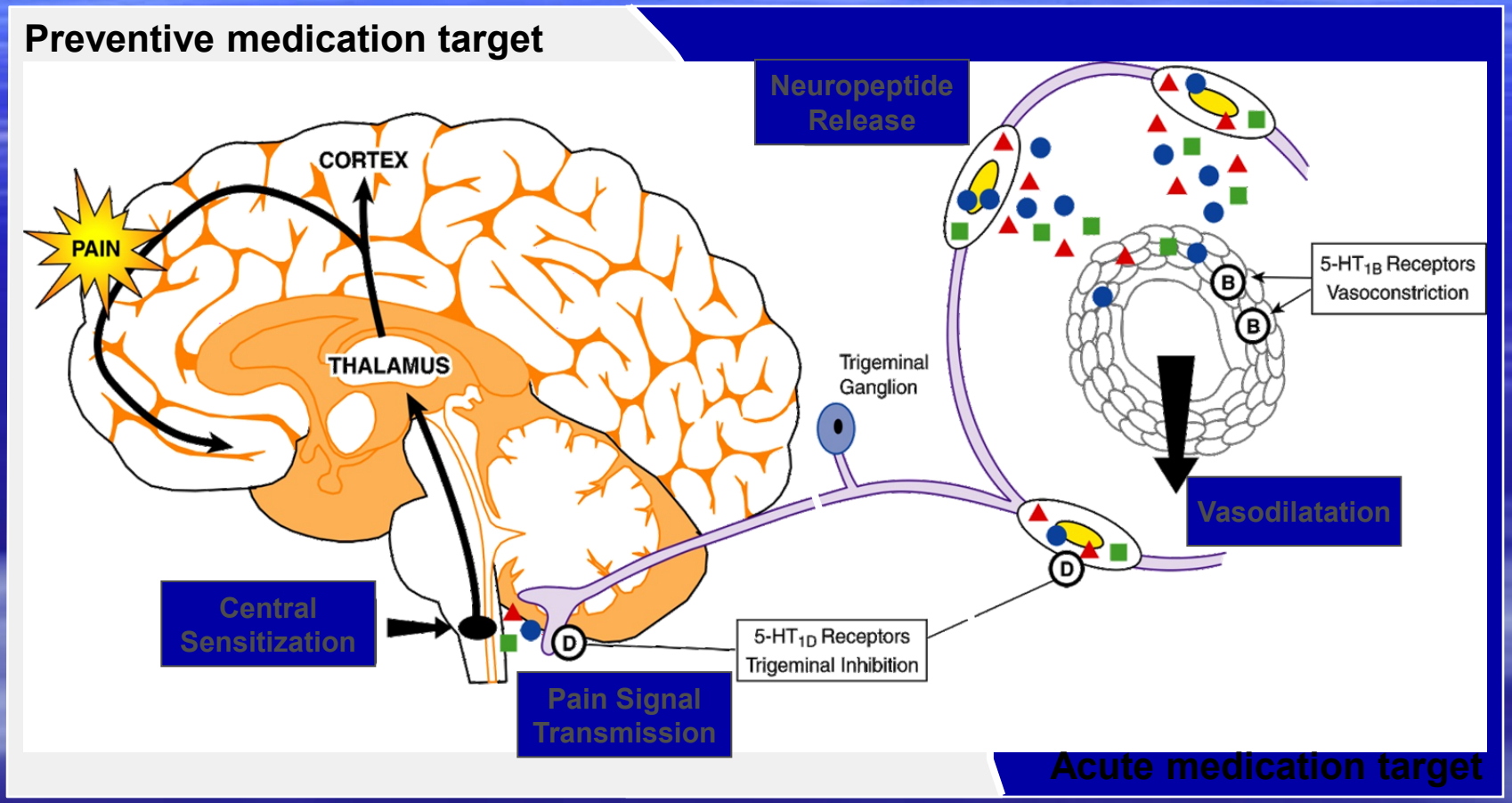
- Migraineurs have a lower threshold for occipital cortex excitation than controls
- Genetic component:
  - P/Q calcium channel, Na^+/K^+ ATPase
  - Mitochondrial defects
- Probably due to:
  - Hyperactivity of excitatory neurotransmission
    - Na^+, Ca^{++} channels, glutamate
  - Lower activity of inhibitory neurotransmission
    - GABA

GABA=gamma aminobutyric acid.

Aurora SK et al. *Neurology*. 1998;50:1111-1114.

Pathophysiology of Migraine

Trigeminovascular Migraine Pain Pathways

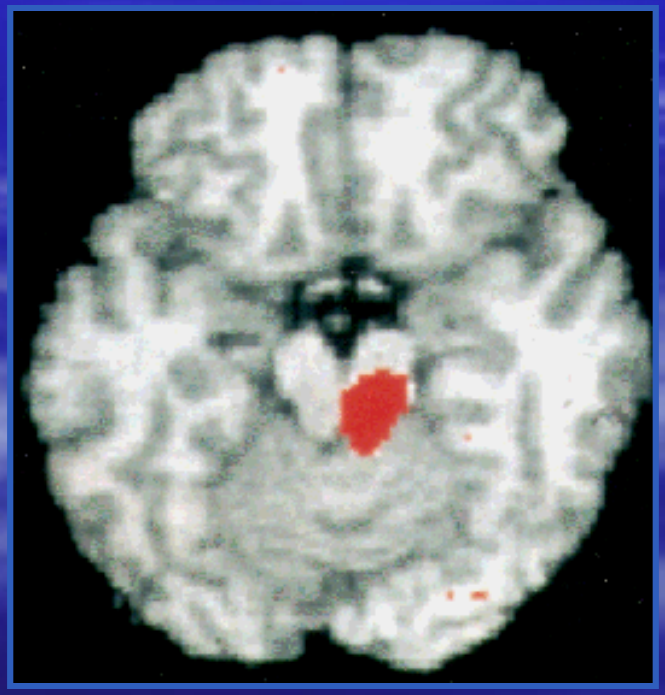


Hargreaves RJ, Shepheard SL. *Can J Neurol Sci.* 1999;26(suppl 3):S12-S1

Pathophysiology of Migraine

Brain Stem Involvement in Migraine

- Brain stem aminergic nuclei can modify trigeminal pain processing
- PET demonstrates brain stem activation in spontaneous migraine attacks
- Brain stem activation persists after successful headache treatment
- Brain stem: generator or modulator?



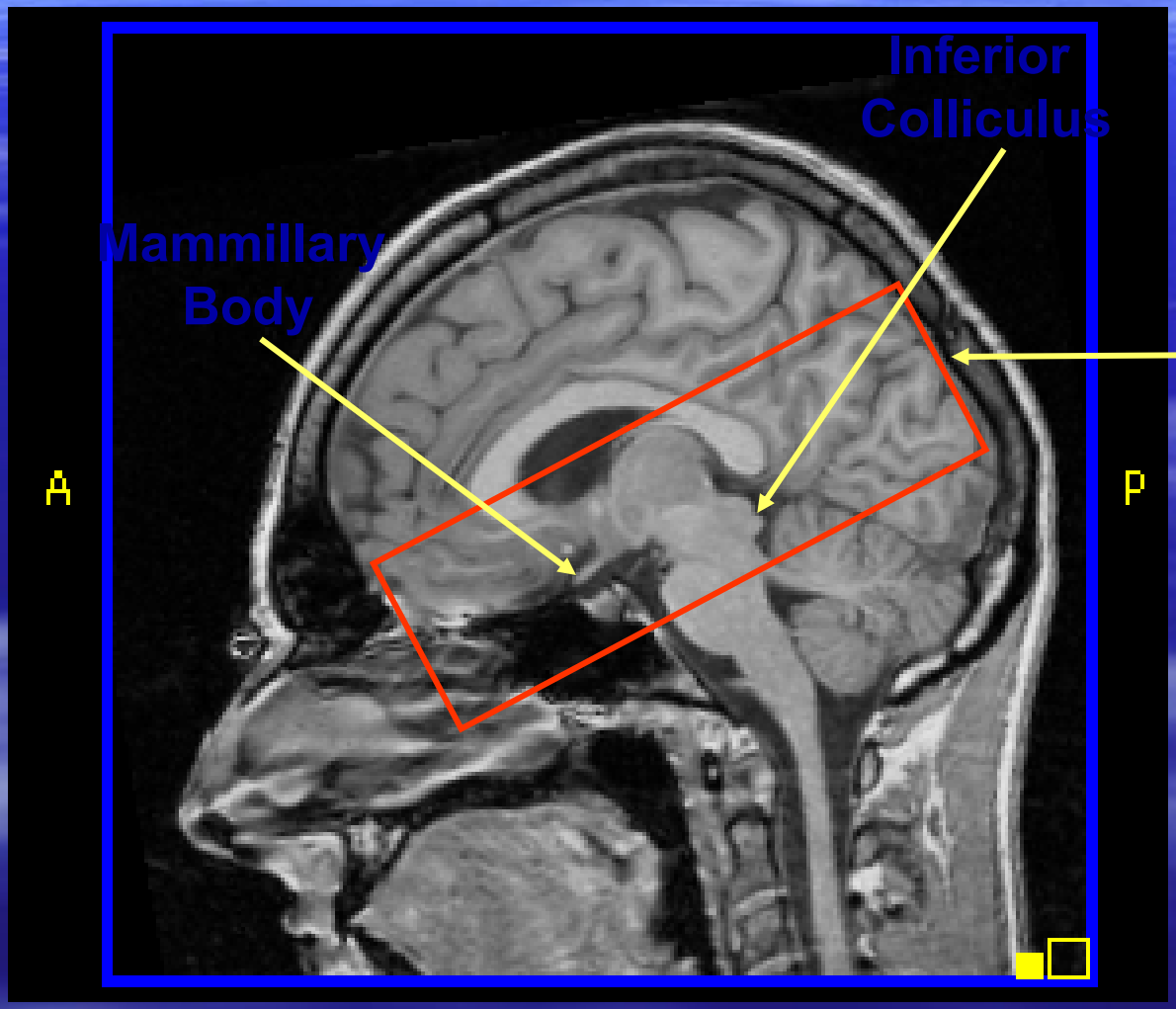
PET=positron emission tomography.

Weiller C et al. *Nat Med.* 1995;1:658-660.

Pathophysiology of Migraine

Red Nucleus and Substantia Nigra

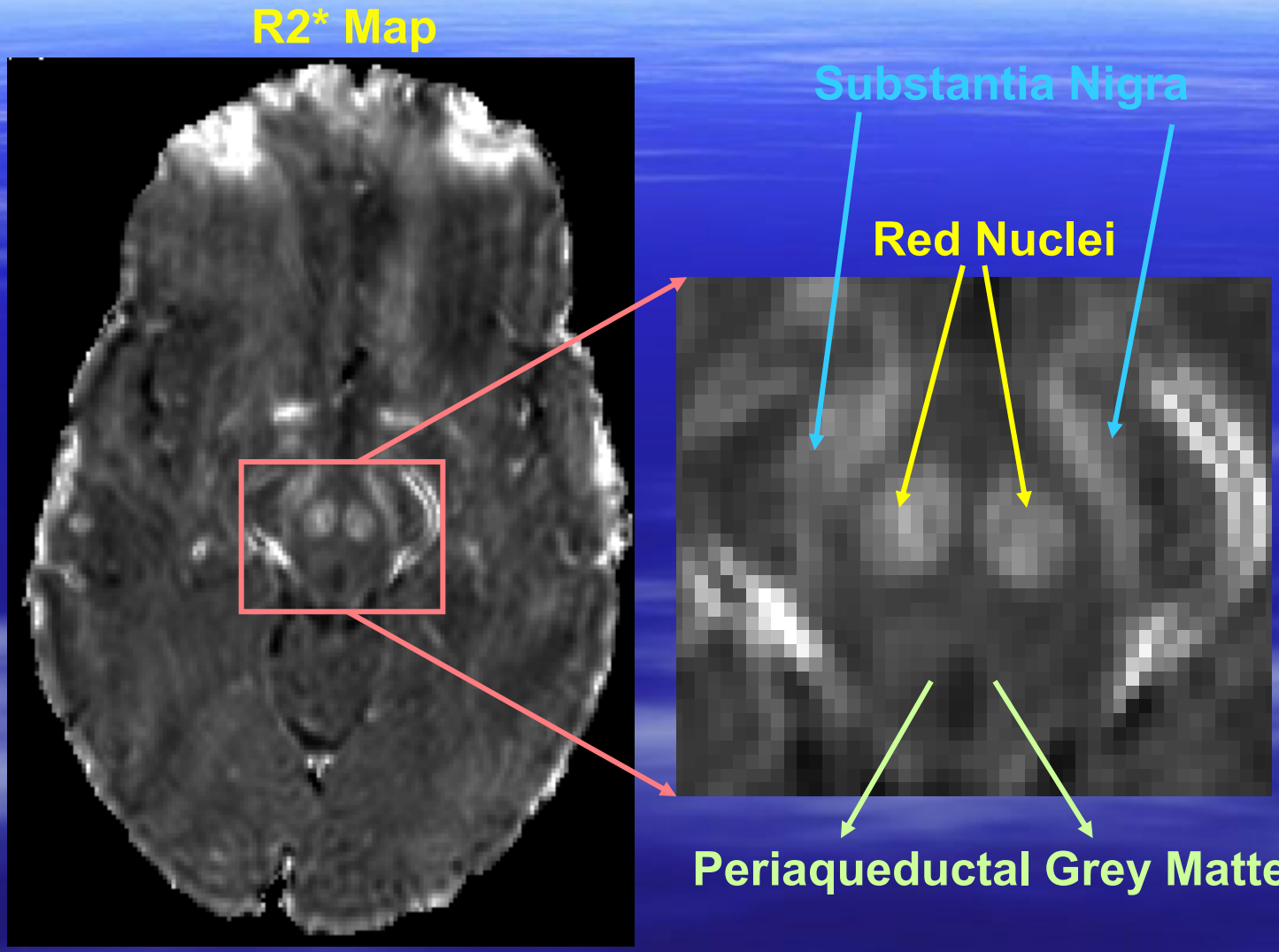
Sagittal View of Imaging Plane



Welch KMA et al. *Headache*. 2001;41:629-637.

Pathophysiology of Migraine

Iron Homeostasis



Welch KMA et al. *Headache*. 2001;41:629-637.

Pathophysiology of Migraine

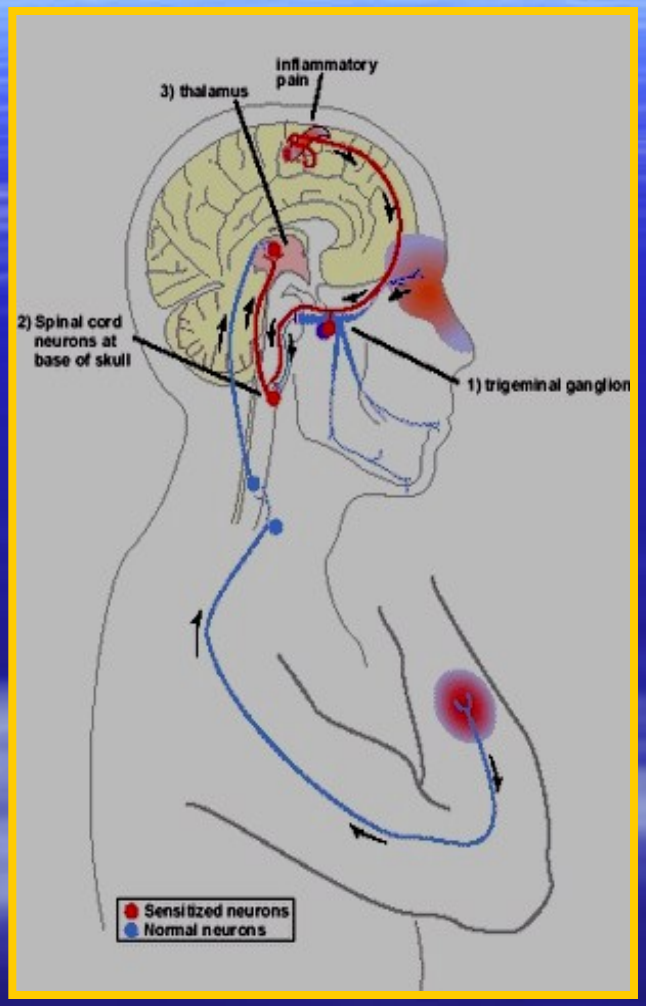
Disease Progression: Changes in PAG

- Changes observed over time in the PAG—center of the brain's powerful descending analgesic neuronal network
  - **–Iron deposition**
  - –Secondary to free-radical cell damage during migraine attacks
- Degree of PAG structural alteration depends on duration of headache history, not the age of the patient
  - **–Repeated migraine attacks, repetitive damage, decreased threshold for further migraine attacks**

Welch KMA et al. *Headache*. 2001;41:629-637.

Pathophysiology of Migraine

Central Sensitization



- Migraineurs develop increased sensitivity to stimuli due to increased nerve excitability
- 79% of migraine patients suffered from *cutaneous allodynia* during attacks due to central sensitization

Burstein R et al. *Ann Neurol*. 2000;47:614-624; Burstein R et al. *Headache*. 2002;42:390-391.

CHRONIC DAILY HEADACHE

A Primary Headache Syndrome

(Organic causes of headache are excluded)

Occurs ≥ **15 days a month**

Lasts ≥ **4 hours a day**

Post Traumatic Headache

Risk factors for chronification

Previous headache, sex (f>m 49% v 30%)

Mild injury

Abnormal position of head at impact (bobblehead model)

Low socioeconomic status, alcohol, early symptoms in ER

Litigation

Post Traumatic Headaches

- Factors for Chronification of Migraine: female sex, obesity, sleep apnea, low socio economic status, initial frequency of headache

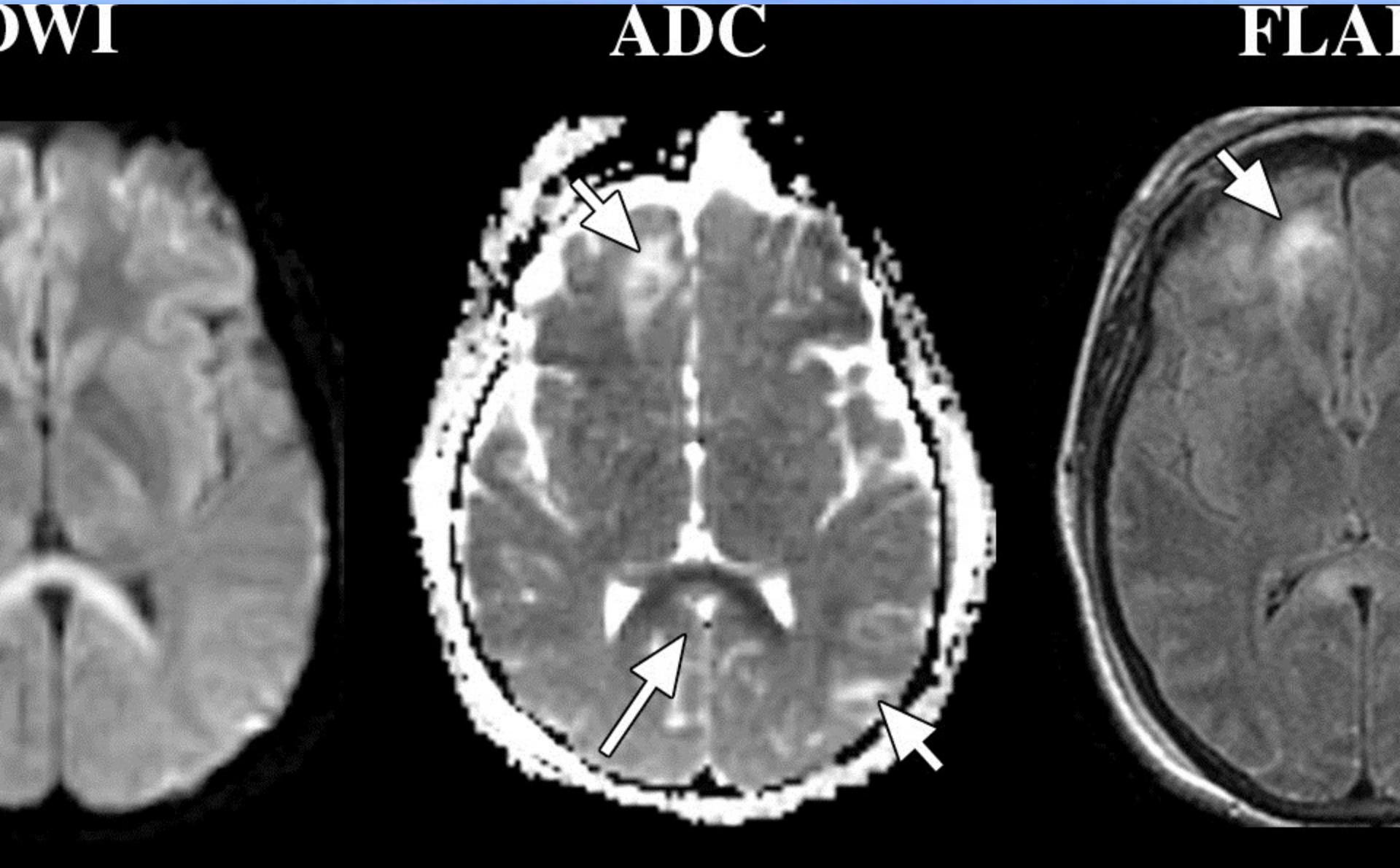
Post Traumatic Headache

Investigation

Examination:Neurological exam as well as neck and vestibular system

Imaging: rarely very useful but important to exclude coexistent features, eg. Chiari acute low CSF pressure (Miyazawa 2003)

Post Traumatic Headache



Post Traumatic Headache

Treatment

Headaches are heterogeneous so treatment strategy is aimed at the particular type of headache, ie tth or migraine symptomatically.

Does aggressive symptomatic and prophylactic treatment reduce chronification? No evidence either way.

Post Traumatic Headache

Treatment of WAD

No universally accepted guidelines

Physio, chiro, exercise etc: no evidence

Diagnostic blocks with radiofrequency neurotomy may help (Bogduk 1997)

Onabotulinum Toxin A (Freund and Scwartz 2000)

NONPHARMACOLOGIC TREATMENT FOR CHRONIC DAILY HEADACHE

Enable healthful patient behavior:

Education

Reduce medication overuse, treat rebound headache

Discontinue smoking

Regulate eating and sleeping patterns

Exercise

Biofeedback and behavioral treatment

Other psychotherapeutic interventions

Double-blind, placebo- controlled published studies in CDH

- Topiramate
  - Silvestrini et al. Cephalalgia, 2003 (n=28)
- Tizanidine
  - Saper et al. Headache, 2002 (n=136)
- Gabapentin
  - Spira et al. Neurology, 2003 (n=133)
- Fluoxetine
  - Saper et al. Headache, 1994 (n=64)
- BoNTA
  - PREEMPT (Diener et al 2010))

Post Traumatic Headaches

- **New perspectives from Iraq and Afghanistan (**Theeler et al 2012)
- Combination of ‘normal’ injury and blast injury related to IEDs and better body armour. Higher incidence of head injury.
- Reviewed 978 US soldiers who scored positive for deployment related concussion.

Post Traumatic Headache

- 20% of US servicemen with a history of concussion manifest as CDH. Incidence in population is 4%.
- When headaches develop within a week of concussive event they can be classified as PTHA according to IHCD-2 criteria.
- Chronic migraine is the predominant headache syndrome in soldiers with posttraumatic CDH

Post Traumatic Headaches

- Soldiers with CDH twice as likely to screen positive for PTSD as soldiers with episodic headaches
- 41% of soldiers with CDH screened positive for PTSD
- Migraine is the headache phenotype in 66% of soldiers with CDH

Post Traumatic Headache

Safety of Onabotulinum Toxin A for the treatment of Chronic Post Traumatic Headache in Service Members with a History of Mild Traumatic Brain Injury. (Yerry et al 2013)

Post Traumatic Headache

Conclusions

Post traumatic headache is common but is often poorly treated and overly assessed.

Try to get a clear description of the type of headache and apply IHS Classification

Treat appropriately to the Classification

Use acute and prophylactic therapy, consider Botox