Management of Headache Following Concussion/Mild Traumatic Brain Injury

Presenter

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Disclosures

- I have no relevant financial or non-financial relationships to disclose relating to the content of this activity.
- The views expressed in this presentation are those of the author and do not necessarily reflect the official policy or position of the Department of Defense, nor the U.S. Government.
- Commercial support was not received for this activity.

Learning Objectives

1. Describe the occurrence of post-traumatic headache (PTH) following a concussion/mild traumatic brain injury (mTBI)
2. Discuss current thoughts on the pathophysiology of PTHs
3. Distinguish between the common PTH types
4. Apply methods to assess, diagnose and manage common PTH types
5. Understand non-pharmacologic and pharmacologic treatments
6. Brief review of literature on current treatment of PTH

PLOS ONE

OPEN ACCESS

Citation: Remigio-Baker RA, Kiser S, Ferdosi H, Gregory E, Engel S, Sebesta S, et al. (2020) Current patterns of primary care provider practices for the treatment of post-traumatic headache in active duty military settings. PLoS ONE 15(7): e0236762. https://doi.org/10.1371/journal.pone.0236762

Editor: Firas H. Kobeissy, University of Florida, UNITED STATES

Received: May 11, 2020

Accepted: July 11, 2020

Published: July 24, 2020

RESEARCH ARTICLE

Current patterns of primary care provider practices for the treatment of post-traumatic headache in active duty military settings

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Qualitative study, interviewing 65 (MDs, PAs and NPs) primary care providers (PCP) at multiple military bases.

Implementation of PCP education and training on PTHs needed.

INFORMATION FOR HEALTH CARE PROFESSIONALS

AMERICAN HEADACHE SOCIETY
www.AmericanHeadacheSociety.org

Concussion and Post-Traumatic Headache

Alan G. Finkel, MD, FAHS
Carolina Headache Institute, Chapel Hill, NC

INTRODUCTION

Recent attention to the consequences of head injury has alerted the public and health professionals to the need for better understanding of concussion. Professional and youth sports, and military Traumatic Brain Injury (TBI) have provided testing grounds for better evaluations and treatments. Headache is amongst the most prominent of the symptoms that may linger after mild TBI. Post Traumatic Headache (PTH) may be the most common and poorly understood of all the lingering symptoms.

SCOPE OF THE PROBLEM

The Centers for Disease Control (CDC) reports that TBI results in nearly 1.4 million emergency room visits, 275,000 hospitalizations and 52,000 deaths per year. Mild TBI accounts for most of

Post-traumatic Headache (PTH)

- PTH may occur from injury to the head and also the neck or face
- The headache started in close proximity to the injury, awareness of the injury or ability to feel the headache
- PTH is classified as acute (<3 months) or persistent (>3 months) based upon duration of headache

International Classification of Headache Disorders (ICHD), third edition:

Headache attributed to traumatic injury to the head

Acute post-traumatic headache attributed to mild traumatic injury to the head

Diagnostic criteria:

For you bookworms who like the exact diagnostic criteria, I give you this gift

- A. Any headache fulfilling criteria C and D
- B. Injury to the head fulfilling both of the following:
  - 1. Associated with none of the following:
    - a) Loss of consciousness for > 30 minutes
    - b) Glasgow Coma Scale (GCS) score < 13
    - c) Post-traumatic amnesia lasting > 24 hours
    - d) Altered level of awareness for > 24 hours
    - e) Imaging evidence of traumatic head injury such as intracranial hemorrhage and/or brain contusion
  - 2. Associated immediately following the head injury with one or more of the following symptoms and/or signs:
    - a) Transient confusion, disorientation or impaired consciousness
    - b) Loss of memory for events immediately before or after the head injury
    - c) Two or more other symptoms suggestive of mild traumatic brain injury: nausea, vomiting, visual disturbances, dizziness and/or vertigo, impaired memory and/or concentration.
- C. Headache is reported to have developed within 7 days after one of the following:
  - 1. The injury to the head
  - 2. Regaining consciousness following the injury to the head
  - 3. Discontinuation of medication(s) that impair ability to sense or report headache following the injury to the head
- D. Either of the following:
  - 1. Headache has resolved within 3 months after the injury to the head
  - 2. Headache has not yet resolved but 3 months have not yet passed since the injury to the head
- E. Not better accounted for by another ICHD-3 diagnosis

5.2.2 Persistent headache attributed to mild traumatic injury to the head

Diagnostic criteria: as for above except for D.

D. Headache persists for > 3 months after the injury to the head

Why are Post Traumatic Headaches different?

- 69 million people every year experience a mTBI with 37-69% of those reporting PTH.[1-4]
- The one-year incidence of new or worsening PTHs in patients with mTBI is over 58%.[5]
- Patients with associated PTHs develop many other co-morbid conditions such as symptoms of depression, anxiety and sleep disturbances
- PTHs are a common secondary headache associated with considerable disability and reduced quality of life

Why are Post Traumatic Headaches different?

- 40 % of patients that develop PTHs develop persistent/chronic PTHs later
- 35 % of patients that sustain an mTBI with PTHs will not returned to full work within 3 months
- There is still much we do not understand about PTHs, there are few evidenced based treatment options.

Pathophysiology

Bottom Line Up Front

These can promote development of PTH:

- Immediate effects of brain contusion
- Cerebral blood vessel damage
- Axonal shearing
- Secondary cascade of metabolic and cellular excitotoxic and inflammatory changes

However, the precise relationship is not well understood

Possible mechanisms underlying the pathophysiology of PTH.[6]

Impaired descending pain modulation and afferent activation.
Structural and functional remodeling in cortical and subcortical regions could impair pain-modulating systems
Nociceptive input from trigeminal and upper cervical afferents might also converge on the trigeminocervical complex.

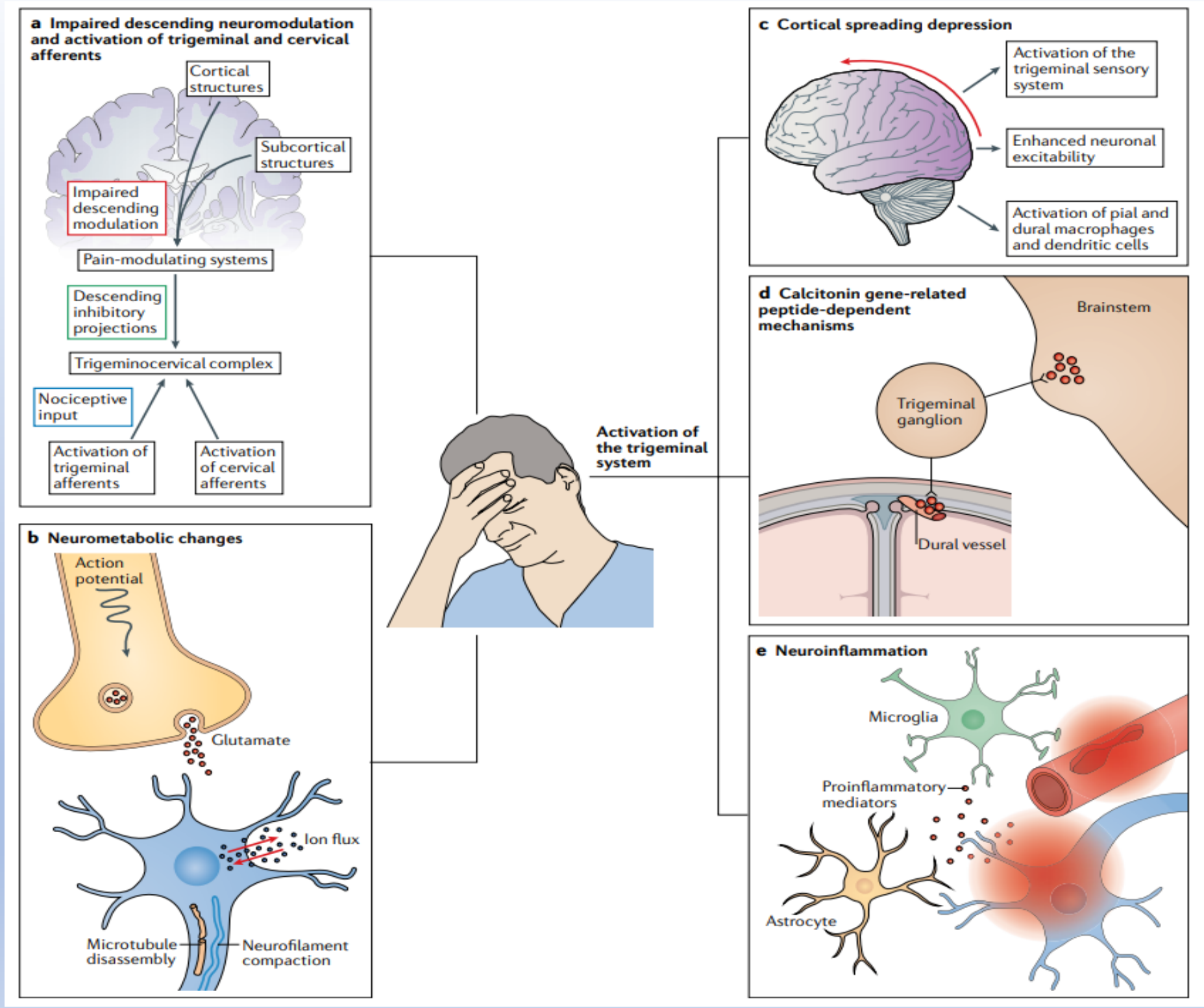
Neurometabolic changes.
Neuronal injury leading to unregulated flux of ions, and restoration of ion homeostasis causes energy depletion and oxidative stress.
Mechanical disruption triggers neurofilament collapse and microtubule disassembly, causing axonal damage.

Cortical spreading depression (CSD). CSD events might occur after TBI, leading to activation of the trigeminal sensory system, enhanced neuronal excitability and activation of pial and dural macrophages and dendritic cells.

Calcitonin gene-related peptide (CGRP)-dependent mechanisms. CGRP is involved in migraine, and animal data suggest that CGRP-dependent pain mechanisms are involved in headache generation after TBI. **

Neuroinflammation. Activation of glial cells following TBI results in the production and release of pro-inflammatory mediators, which then cause neuronal damage, vascular damage and leakage from the blood–brain barrier.

Pathophysiology

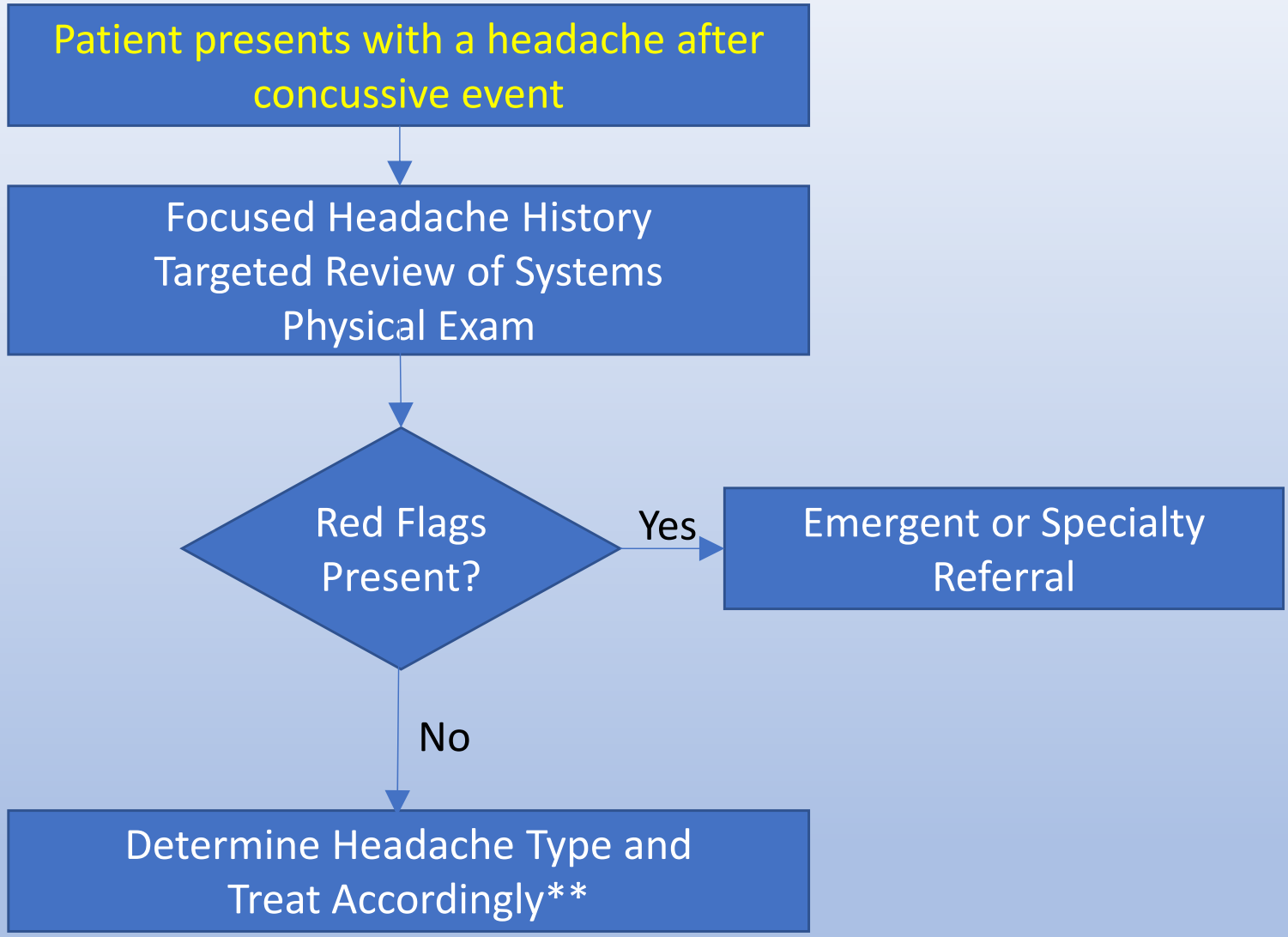


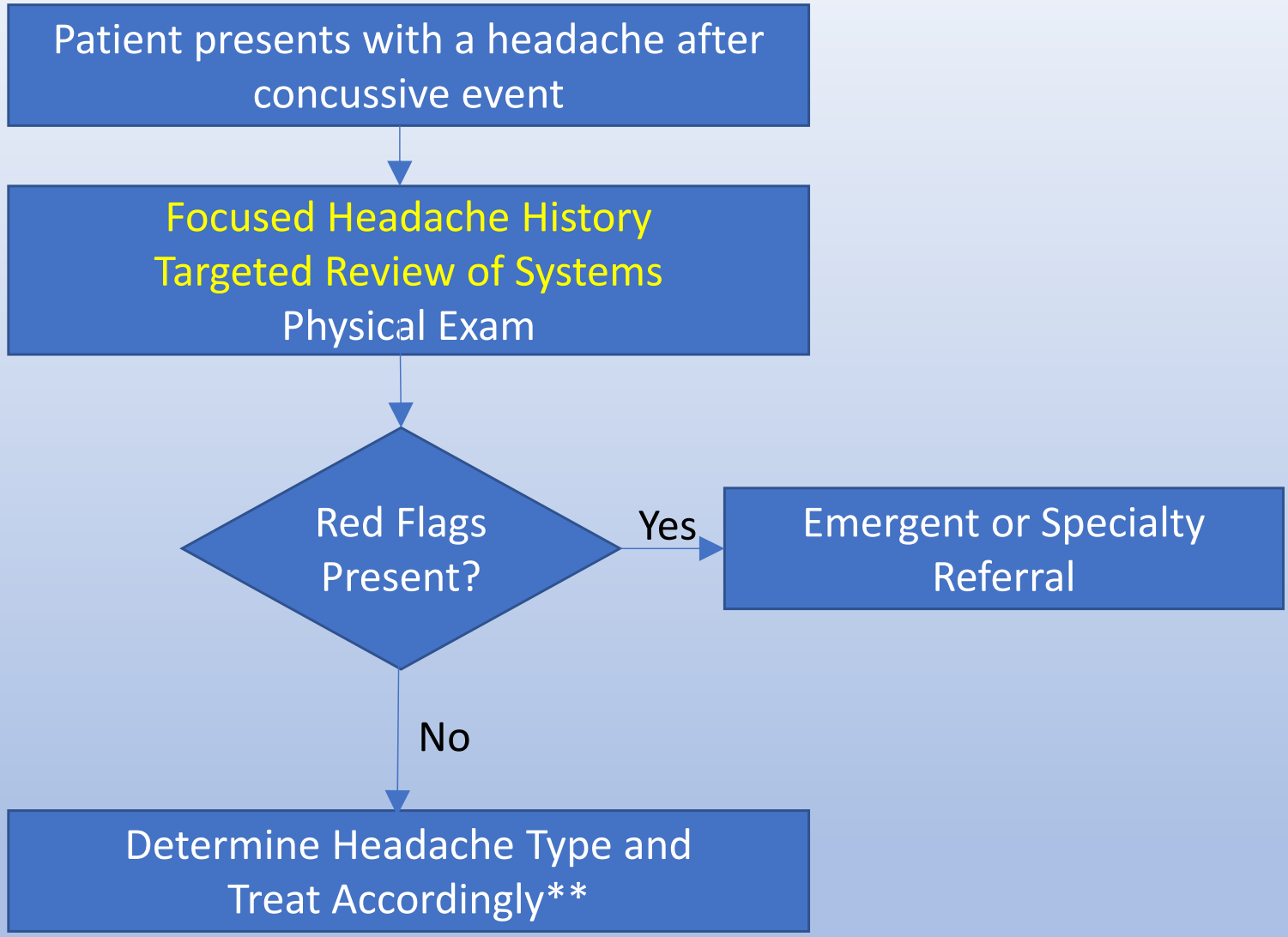
Post-traumatic Headache Risk Factors

- The most common risk factors for the development of PTH include:
  - Age $\leq$ 60[1,7]
  - Premorbid history of headache[7,8] or TBI
  - Female[2,8]
  - Presence of comorbid psychiatric disorders[2]
  - Mild TBI (versus moderate or severe)[5]

Post-traumatic Headache Risk Factors (cont.)

- Research suggests other risk factors include:[9]
  - Patient's expectation of developing a headache after head injury
  - Sleep disturbances
  - Mood disturbances
  - Psychosocial stressors


Patient presents with a headache after concussive event
Focused Headache History
Targeted Review of Systems
Physical Exam
Red Flags Present?
Yes
Emergent or Specialty Referral
No
Determine Headache Type and Treat Accordingly**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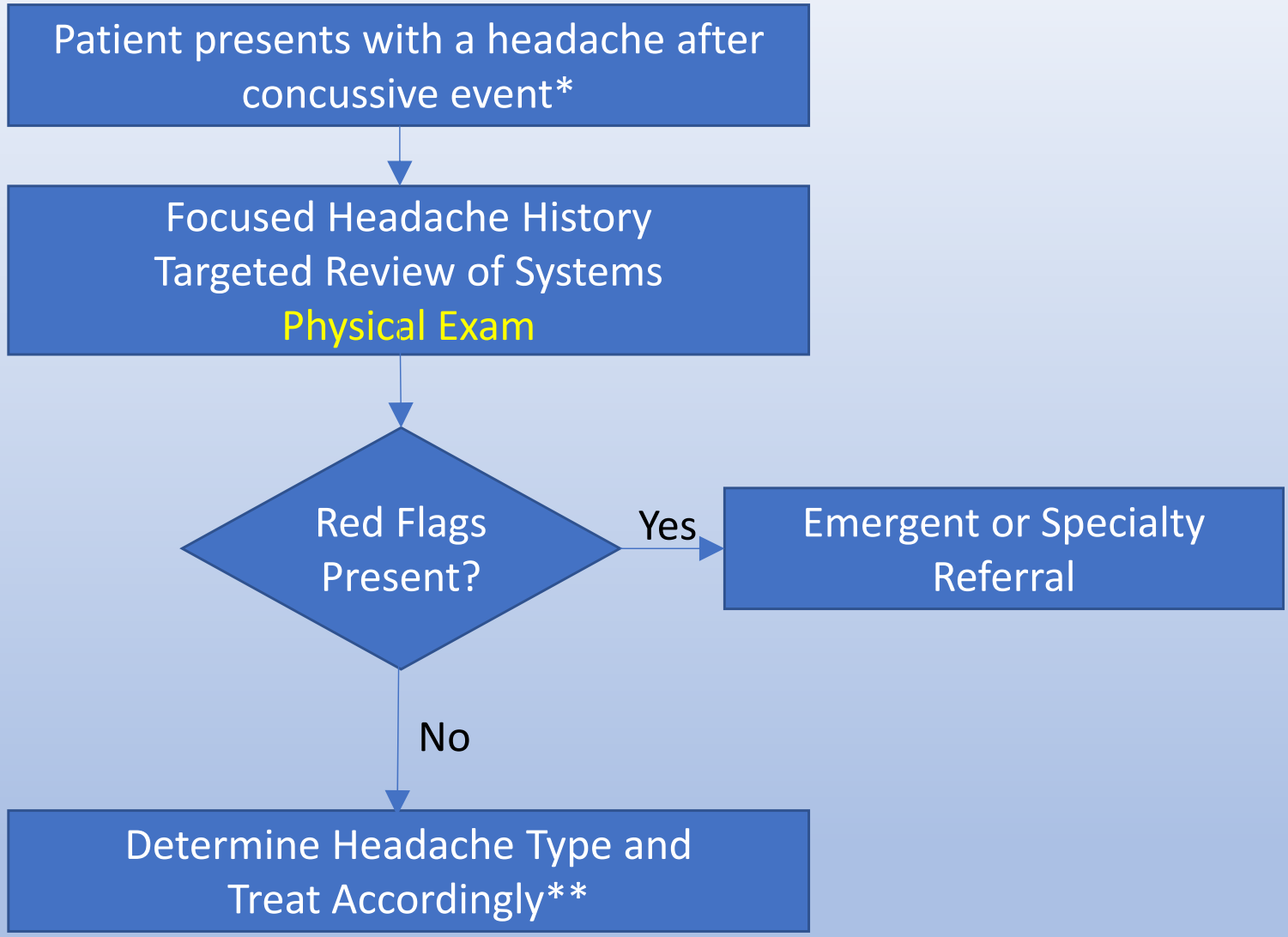
Focused Headache History

Assessment area	Examples of questions and information to collect
Symptoms	• Persistent pain in head or neck after a concussion (Use of 0-10 scale is recommended, 1= barely present, 5= pain beginning to interfere with activity, and 10= worst imaginable pain)
Location	• Right or left sided • Bilateral vs. unilateral • Face • Stays in one place or moves around (radiates) • Back or on top • Forehead • Neck
Description of pain	• Throbbing/pulsating • Pressing/squeezing • Stabbing, sharp, or dull/nagging • Pain with chewing or opening mouth • Head, face or neck tenderness • Decreased jaw movement
Frequency and duration	• Episodic or continuous • Seconds, minutes, hours, days or constant
Associated physical symptoms	• Vision changes (blindness, blurry vision, double vision, eyelid droop, tearing, eye redness or puffiness) • Light, noise and odor sensitivity, nose blockage/discharge • Nausea, loss of appetite, hunger, bowel changes • Premonitory symptoms (fatigue, difficulty concentrating) • Neck stiffness or pain • Yawning • Pallor • Auras (visual, sensory or dysphasic speech disturbances) • Numbness or tingling around lips, arms or legs

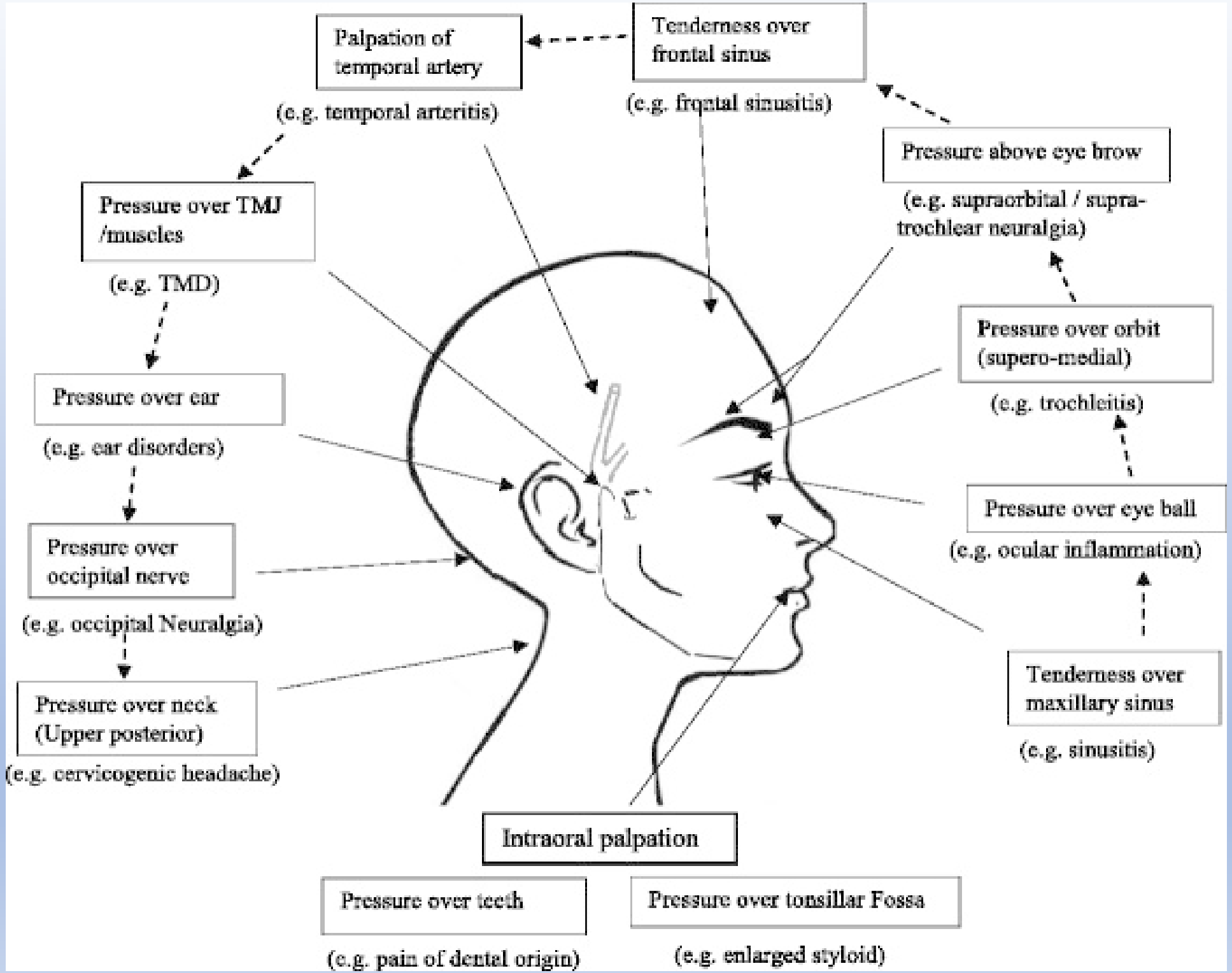
Focused Headache History (cont.)

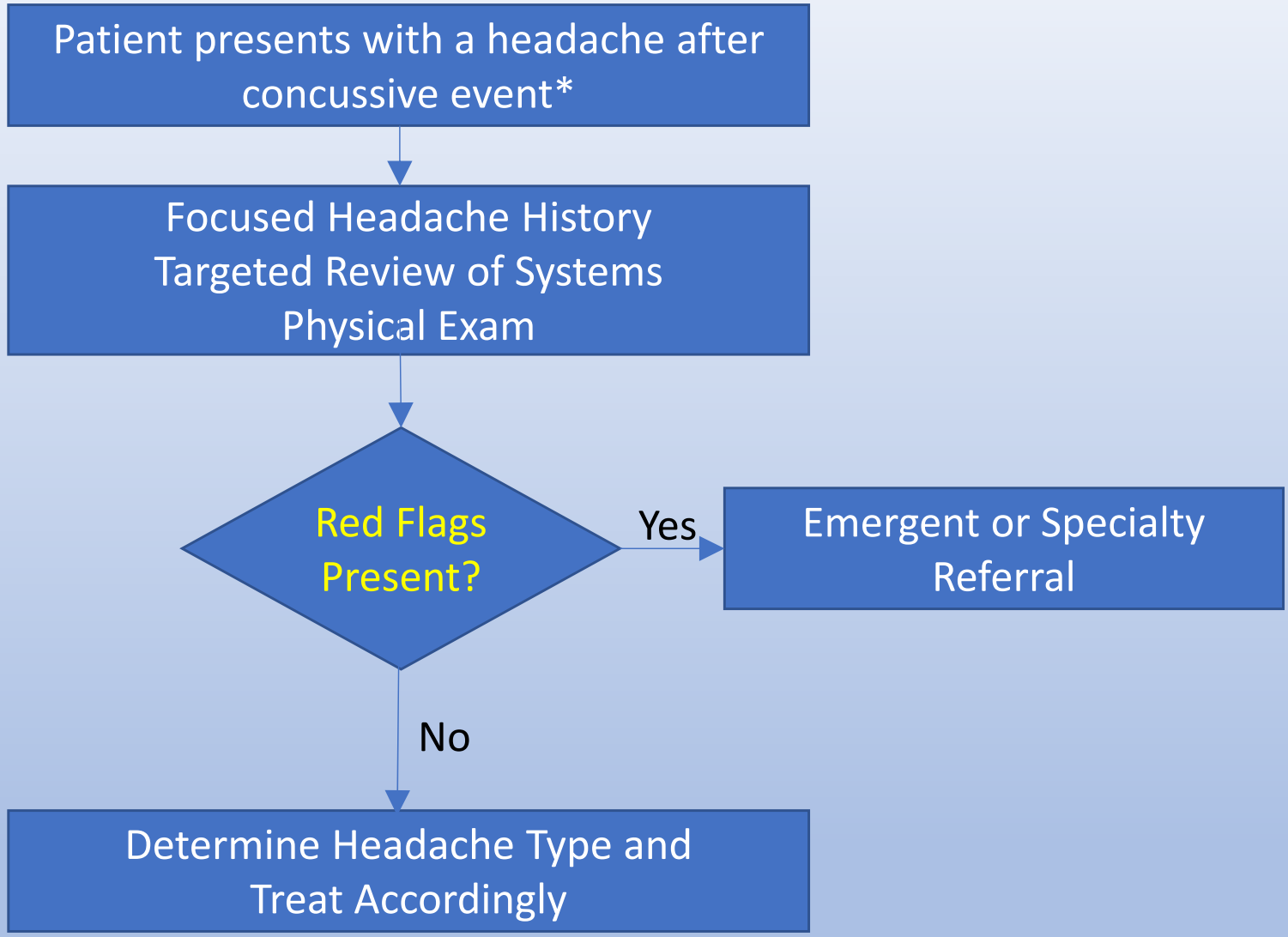
Assessment area	Examples of questions and information to collect	
Headache history	• Previous headache diagnosis • Worsening headache • Previous head trauma or TBI	• History of temporal mandibular joint (TMJ) pain • Family history
Triggers	• Sleep (too much or too little) • Physical activity • Straining or coughing • Missed meal • Food • Pregnancy • Caffeine • Muscle tension	• Emotional stress (during or after) • Bending over • Sexual activity • Change in weather • Alcohol • Menstrual cycle • Contraceptives
Social history	• Headache interferes with family, work or school • Substance use or abuse (caffeine, alcohol, tobacco), supplement use (vitamins, etc.)	
Medication history	• Previous medications used for headache prevention and rescue - Dosage, frequency and duration - Failed medications • Current medications, how often taking rescue or preventative medications	
Comorbid conditions	• Insomnia, depression, anxiety, obstructive sleep apnea	
Questionnaires	• Patient Health Questionnaire (PHQ), Neurobehavioral Symptom Inventory (NSU), Patient Global Impression of Change (PGIC), Headache Impact Test-6 (HIT)	


Patient presents with a headache after concussive event*
Focused Headache History
Targeted Review of Systems
Physical Exam
Red Flags Present?
Yes
Emergent or Specialty Referral
No
Determine Headache Type and Treat Accordingly**

Focused Headache Examination

Assessment area	Examples
Head, neck and face	• Cranial nerve examination • Neck range of motion • Palpation of head and neck for trigger points or tenderness • Evaluate for papilledema
Ears, nose and throat	• Examine the ears, nares • Palpitate the face and percuss sinuses • Temporal mandibular joint (TMJ) examination
Other neurological examination	• Reflexes • Sensory testing • Romberg testing • Pronator drift • Strength testing
Mental status	• Speech fluency • Word recall


Palpation of temporal artery
(e.g. temporal arteritis)
Tenderness over frontal sinus
(e.g. frontal sinusitis)
Pressure above eye brow
(e.g. supraorbital / supra-trochlear neuralgia)
Pressure over TMJ /muscles
(e.g. TMD)
Pressure over orbit (supero-medial)
(e.g. trochleitis)
Pressure over ear
(e.g. ear disorders)
Pressure over eye ball
(e.g. ocular inflammation)
Pressure over occipital nerve
(e.g. occipital Neuralgia)
Tenderness over maxillary sinus
(e.g. sinusitis)
Pressure over neck (Upper posterior)
(e.g. cervicogenic headache)
Intraoral palpation
Pressure over teeth
(e.g. pain of dental origin)
Pressure over tonsillar Fossa
(e.g. enlarged styloid)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Headache Red Flags and Indications for Referral

Red flags specific for headaches	
Indications for emergency referral	**Indications for specialty referral**
Concussion red flags	Presence of systemic symptoms
Thunderclap headache (sudden onset)	Associated neurological symptoms
Sudden neurological deficit	Onset after age 50*
Persistent bleeding from nose, ears or scalp	Change in pattern of headache
Cranial fracture	Valsalva precipitation
Infection resulting from a penetrating injury	Postural aggravation
Cerebrospinal fluid leakage (nose or ears)	TMJ disorder
Intracranial hemorrhage on CT	ENT disorder
Papilledema	Anticoagulant therapy*

* Patients on anticoagulant therapy or over the age of 50 have an increased risk of chronic subdural hematoma. This demographic may need imaging with or without specialty referral based on the head trauma history and provider judgment.

SNOOP – **S**ystemic **N**euro **O**nset (new) **O**nset (>50) **P**rior different headache

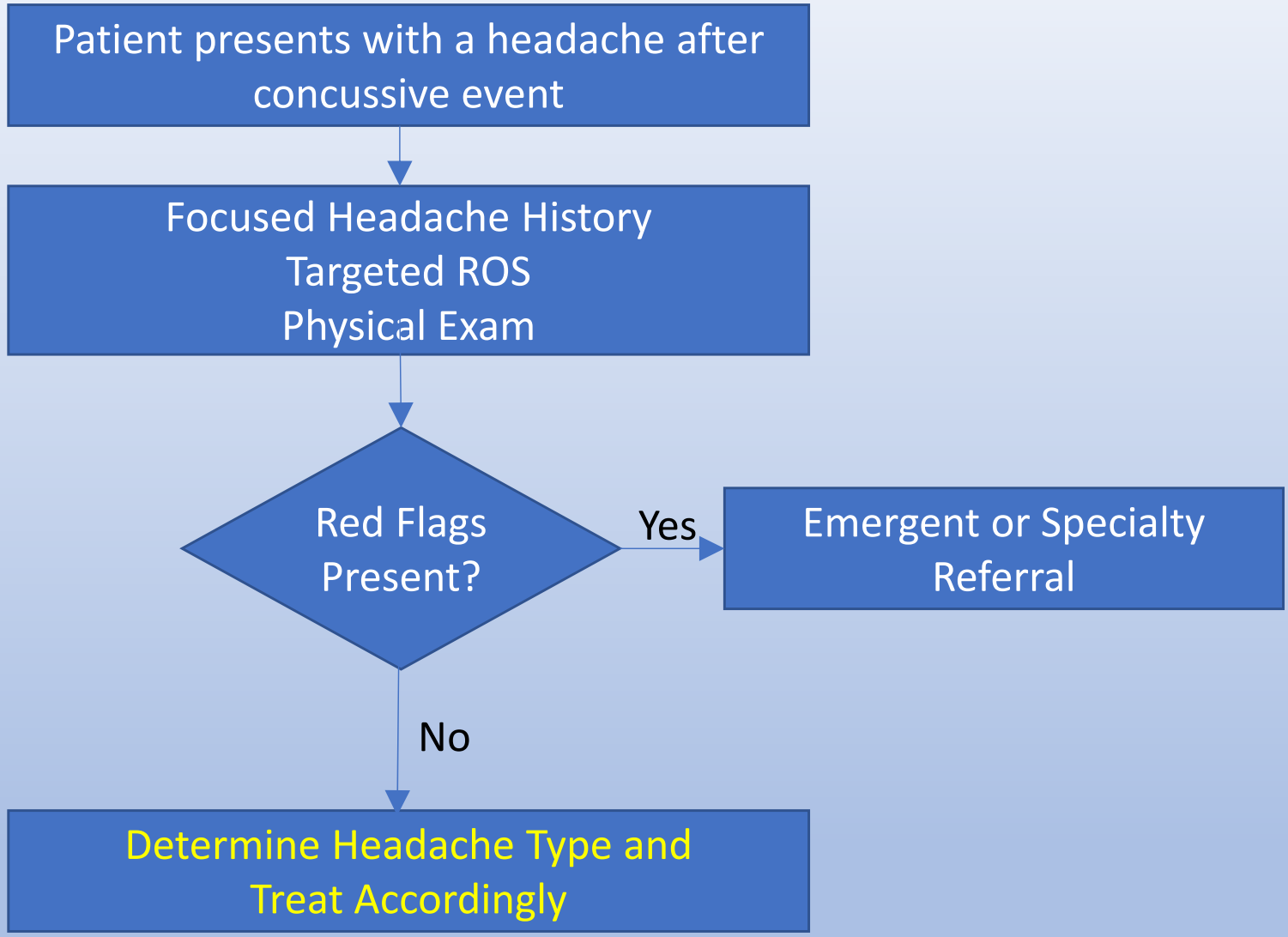
Concussion Red Flags

Indications for emergency referral
Deteriorating level of consciousness
Double vision
Increased restlessness, combative or agitated behavior
Persistent bleeding from nose, ears or scalp
Repeat vomiting
Results from a structural brain injury detection device (if available)
Seizures
Weakness or tingling in arms or legs
Severe or worsening headache

To image or not to image

- Recent significant change in pattern, frequency, or severity
- Worsening despite therapy (MRI with gadolinium contrast)
- Focal neurological signs/symptoms
- HA with exertion, cough, or sexual activity
- Orbital bruit
- Onset after age 50
- Red Flags Present

- Basic rule is CT without contrast if less than 7 days and MIR without if greater than 7 days post injury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Characteristics of Headache Types

	Migraine	Tension type	Cervicogenic	Related to neuropathic pain	Medication overuse*
Aura	Possible (15-33%)	No	No	No	No
Duration	4-72 hours	30 minutes to 7 days	Some or all of the day	Seconds, minutes, hours	Some or all of the day
Frequency	Episodic, variable	1-15 days/ month, variable	Variable	Episodic, variable	Daily > 15 days each month
Site	Unilateral	Bilateral	Usually unilateral	Unilateral	Unilateral or bilateral
Pain characteristics	Pulsating	Pressure/ tightening	Tightening and/or burning	Burning, radiating	Pressing, tightening, pulsating
Pain severity	Moderate/severe	Mild/moderate	Mild/moderate	Moderate/severe	Mild/moderate/severe
Aggravated by movement?	Yes	No	Yes with head movement	Yes	No
Nausea/ vomiting	Yes	No	No	No	No
Photophobia/ phonophobia?	Yes	No	No	No	No

*PCM should consider the possibility of medication overuse headache (MOH) when criteria in Table 5.0 are present. Optimal treatment consists of discontinuation of the offending medications, acute treatment of withdrawal symptoms and pain, and use of analgesic medication as preventative treatment only when necessary.

Migraine Headache

- Most common type of PTH, 50-60 % of PTH's.
- Many times, are severe and debilitating
- International Classification of Headache Disorders-III provides two major subtypes for migraines:
  - With aura
  - Without aura
- Treatment is the same for both subtypes.

Migraine Headache

ICD-9-CM: 346.10 (without aura)	ICD-10-CM: G43.009
ICD-9-CM: 346.00 (with aura)	ICD-10-CM: G43.109

Description:*

A. Headache attacks lasting 4-72 hours (untreated or unsuccessfully treated)
B. Headache has at least two of the following characteristics:
    1. Unilateral location
    2. Pulsating quality
    3. Moderate or severe pain intensity
    4. Aggravation by, or causing avoidance of, routine activity (e.g., walking or climbing stairs)
C. During headache at least one of the following:
    1. Nausea and/or vomiting
    2. Photophobia or phonophobia
D. May or may not be accompanied by an aura (present in 15-33 percent of patients). Most common auras are visual, other sensory, motor or speech and language

* Modified from: International Headache Society. (2013). The International Classification of Headache Disorders, 3rd edition (beta version), *Cephalalgia*, *33*(9) 629-808.

Migraine HA Non - Pharmacologic Treatment

Lifestyle changes

Environmental stimulus control

Exercise

Hydration and nutrition maintenance

Avoidance of triggers

Limit caffeine

Regular Shift Work

Relaxation training

Biofeedback

Cognitive behavioral therapy (CBT)

Migraine Headache Pharmacologic Treatment

Acute/Abortive Agents

Mild/moderate: Acetaminophen; NSAIDs[§] (>48 hours following concussion)

Severe: Triptans (e.g.,sumatriptan, rizatriptan, zolmitriptan); dihydroergotamine (DHE) nasal spray (pre-treat with antiemetic) Ketorolsac nasal spray or IM

Preventive Treatment

First Line: Tricyclic antidepressants (TCA) (e.g., amitriptyline, nortriptyline); antiepileptics (e.g., topiramate, valproate); beta blockers (e/g., metoprolol)
Second Line: Serotonin norepinephrine reuptake inhibitors (SNRI) (e.g., venlafexine); onabotulinumA[§§] (Botox); (referral recommended)

[§] Recent U.S. Food and Drug Administration Agency (FDA) warning cautions that NSAIDs can increase the risk of heart attack, heart failure, or stroke in patients with or without pre-existing heart disease, or risk factors for heart disease, even during the first few weeks of treatment, though the risk appears highest with longer use at higher doses. Detailed information on this topic is located at http://www.fda.gov/Drugs/DrugSafety/ucm451800.htm

[§§] OnabotulinumA is FDA approved for treatment of migraine headaches.

Acute/Abortive Triptan therapy

Medication	Dosage	Comments
Serotonin Receptor Agonists (Triptans)		
Sumatriptan (Imitrex) SubQ injection Oral Tablet Nasal Spray*	 6mg @ onset; may repeat in 1 hr 25, 50, or 100mg @ onset 5, 10, or 20mg @ onset	 Max 12mg/day for inj Max 200mg/day for tablet Max 40mg/day for spray
Zolmitriptan (Zomig) Oral Tablet or ODT Nasal Spray*	 2.5 - 5mg @ onset; may repeat in 2 hr 5mg @ onset; may repeat in 2 hr	Do not split ODT form Max 10mg/day Max 10mg/day
Naratriptan (Amerge)*	1 – 2.5mg @ onset; may repeat in 4 hr	Max 5mg/day or
Rizatriptan (Maxalt) Oral Tablet and ODT	5 – 10mg @ onset; may repeat in 2 hr	Max 30mg/day 15mg/day max w/Inderal
Frovatriptan (Frova)*	2.5 - 5mg @ onset; may repeat in 2 hr	Max dose 7.5mg/day
Eletriptan (Relpax)	20 – 40mg @ onset; may repeat in 2 hr	Max dose 80mg/day

Preventative medications

Medication	Dosage	Comments
Beta-Adrenergic Antagonist		
Propranolol (IR/LA)	80 to 240mg per day	IR rec Q8 – Q6 hr interval
Timolol	10mg BID	Max dose 30mg/day
Metoprolol (IR/XL)	50 – 200mg per day	
Atenolol	25-100mg per day	
Nadolol	80-160mg per day	
Anticonvulsants		
Topiramate	Starting dose 25mg Qday	Max dose 200mg
Valproic Acid/Divalproex sodium	500 – 1500mg/day in divided doses	Must monitor serum level
Gabapentin	2400mg total daily dose	
Antidepressants		
Amitriptyline	25 – 150mg QHS	
Nortriptyline	10 – 150mg QHS	
Venlafaxine (IR/ER)	75 – 225 mg daily	ER preferred formulation
Calcium Channel Blockers		
Verapamil	240 – 480mg total daily dose	Benefits seen after 8 week

Tension-Type Headache

ICD-9-CM: 339.1	ICD-10-CM: G44.209

Description:*

A. Episodes of headache, typically bilateral, pressing or tightening in quality, of mild to moderate intensity, lasting minutes to days
B. Pain does not worsen with routine physical activity and is not associated with nausea, but photophobia or phonophobia may be present
C. Occurring for 1-15 days per month.

* Modified from: International Headache Society. (2013). The International Classification of Headache Disorders, 3rd edition (beta version). *Cephalalgia, 33*(9), 629-808.

Tension-Type Headache
Assessment and Diagnosis

- Increased scalp palpation tenderness is the most significant abnormal finding in patients with tension-type headache
- Tenderness can be elicited by small rotating movements and a firm pressure over the head and neck muscles
- Tenderness is typically present between headaches

Tension-Type Headache Non-Pharmacologic Treatment*

Education on lifestyle changes	
▪Sleep hygiene ▪Exercise ▪Hydration ▪Caffeine intake ▪Physical therapy	▪Stress management ▪Acupuncture ▪Relaxation training ▪Cognitive behavioral therapy (CBT) ▪Biofeedback ▪Massage

*Nicholson, R. A., Buse, D. C., Andrasik, F., & Lipton, R. B. (2011, February). Nonpharmacologic treatments for migraine and tension-type headache: how to choose and when to use. *Current Treatment Options in Neurology*, 13(1), 28-40. Penzien, D. B., & Taylor, F. R. (2014, May). Headache toolbox. Behavioral and other nonpharmacologic treatments for headache. *Headache*, 54(5), 955-6.

Campbell, J. K., Penzien, D. B., Wall, E. M., & the U.S. Headache Consortium. (2009). Evidenced-Based Guidelines for Migraine Headache: Behavioral and Physical Treatments. Retrieved from: http://tools.aan.com/professionals/practice/pdfs/gl0089.pdf. Bell, K. R., Hoffman, J., & Watanabe, H. (2014, April). Headaches after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 95(4), 793-4.

Tension-Type Headache Pharmacologic Treatment

Acute/Abortive Agents

First line: Acetaminophen; NSAIDs[§]
Second Line: Acetaminophen/caffeine compounds

Preventive Treatment

Selective serotonin reuptake inhibitors (SSRI) (e.g., parovetine, citalopram)
Serotonin norepinephrine reuptake inhibitors (SNRI) (e.g., venlafexine)
Tricyclic antidepressants (TCA) (e.g., amitriptyline, nortriptyline);)
Tetracyclic antidepressants (e.g., mirtazapine)

§ U.S. Food and Drug Administration Agency (FDA) warning cautions that NSAIDs can increase the risk of heart attack, heart failure, or stroke in patients with or without pre-existing heart disease, or risk factors for heart disease, even during the first few weeks of treatment, though the risk appears highest with longer use at higher doses. Detailed information on this topic is located at http://www.fda.gov/Drugs/DrugSafety/ucm451800.htm

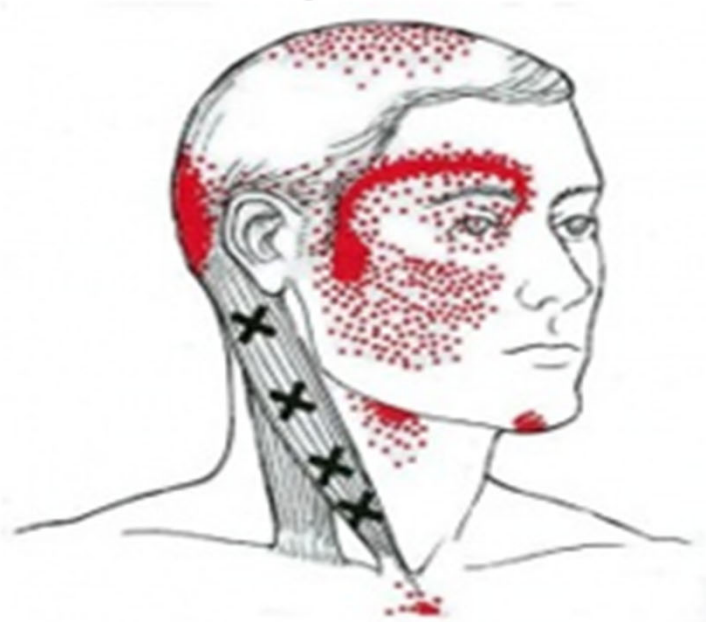
Cervicogenic Headache

ICD-9-CM: 732.2	ICD-10-CM: G44.841

Description:*

A. Headache caused by a disorder of the cervical spine or soft tissue of the neck. Usually, but not always, associated neck pain
B. Headache has developed on temporal relation the head trauma
C. Cervical range of motion is reduced
D. Headache is made significantly worse by neck movement

* Modified from: International Headache Society. (2013). The International Classification of Headache Disorders 3rd edition (beta version). *Cephalalgia, 33*(9), 629-808.

Clinical features	Cervicogenic Headache
Female:Male	50:50
Lateralisation	Unilateral without side-shift
Location	Occipital to frontoparietal and Orbital 
Frequency	Chronic, episodic
Severity	Moderate-severe
Duration	1 hour to weeks
Pain character	Non-throbbing, and non-lancinating, pain usually starts in the neck
Triggers	Neck movement, and postures, limited ROM, pressure of C0-C3
Associated symptoms	Usually absent or similar to migraine but milder, decreased ROM

Cervicogenic Headache Physical Exam

- Physical exam findings may include:
  - Reduced range of cervical motion
  - Headache pain on only one side
  - Provocation of headache by digital pressure on neck muscles
  - Posterior to anterior radiation of pain with head movement (Headache Classification Committee of the International Headache Society, 2013)

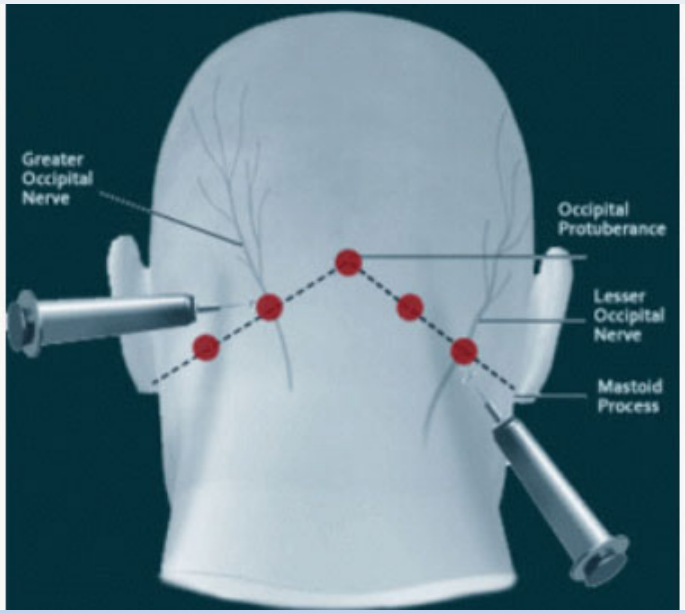
Cervicogenic Headache
Non Pharmacologic Treatment

Acupuncture

Physical Therapy focusing on the Neck

Occipital Nerve Blocks leading to

Greater Occipital Neurolysis/Neurectomy (referral)



Cervicogenic Headache Treatment

Acute/Abortive Agents

First Line: NSAIDs§
Second Line: Muscle relaxants if cervical spasms; trigger point injection (referral recommended)

Preventive Treatment

Antiepileptics (e.g., gabapentin, topiramate);

tricyclic antidepressants (TCA) (e.g., amitriptyline, nortriptyline);

serotonin norepinephrine reuptake inhibitors (SNRI) (e.g., venlafexine)

§ U.S. Food and Drug Administration Agency (FDA) warning cautions that NSAIDs can increase the risk of heart attack, heart failure, or stroke in patients with or without pre-existing heart disease, or risk factors for heart disease, even during the first few weeks of treatment, though the risk appears highest with longer use at higher doses. Detailed information on this topic is located at http://www.fda.gov/Drugs/DrugSafety/ucm451800.htm

Headache Related to Neuropathic Pain

ICD-9-CM: 792.2	ICD-10-CM: 792

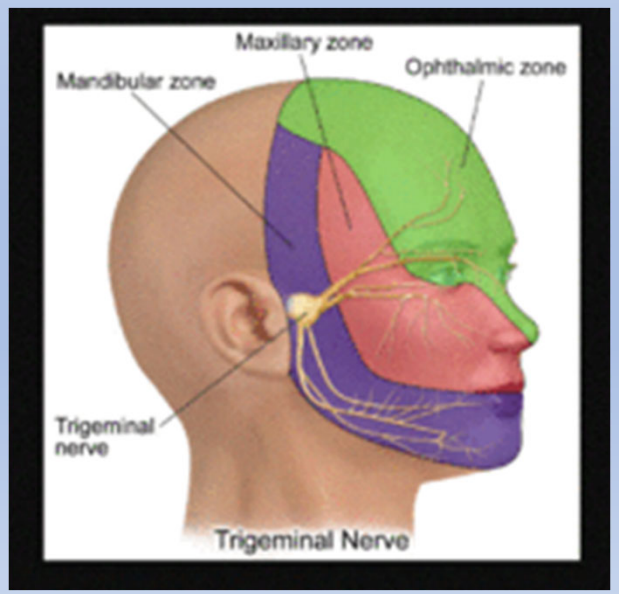
Description:*

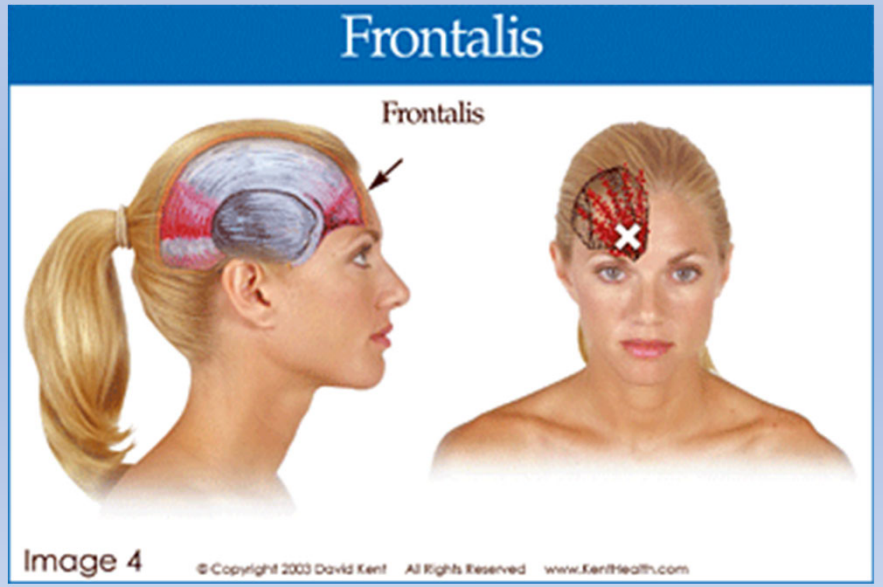
A. Pain associated with soft-tissue injury of the scalp or face
B. May have superimposed lancinating component and may also be burning, deep, and aching
C. There may be local tingling and numbness, hyperesthesia, hyperalgesia, allodynia (pain due to non-noxious stimulus) or hyperpathia (particularly unpleasant, exaggerated pain response)
D. Symptoms are long-lasting, typically persisting after resolution of the primary cause

* Modified from: International Headache Society. (2013). The International Classification of Headache Disorders, 3rd edition (beta version). *Cephalalgia, 33*(9), 629-808.

Headache Related to Neuropathic Pain Diagnosis and Assessment

- Complex chronic pain usually accompanied by soft tissue injury to the scalp or face
- Pain out of proportion to injury
- Burning, tingling type of pain
- Decreased sensation in the affected area

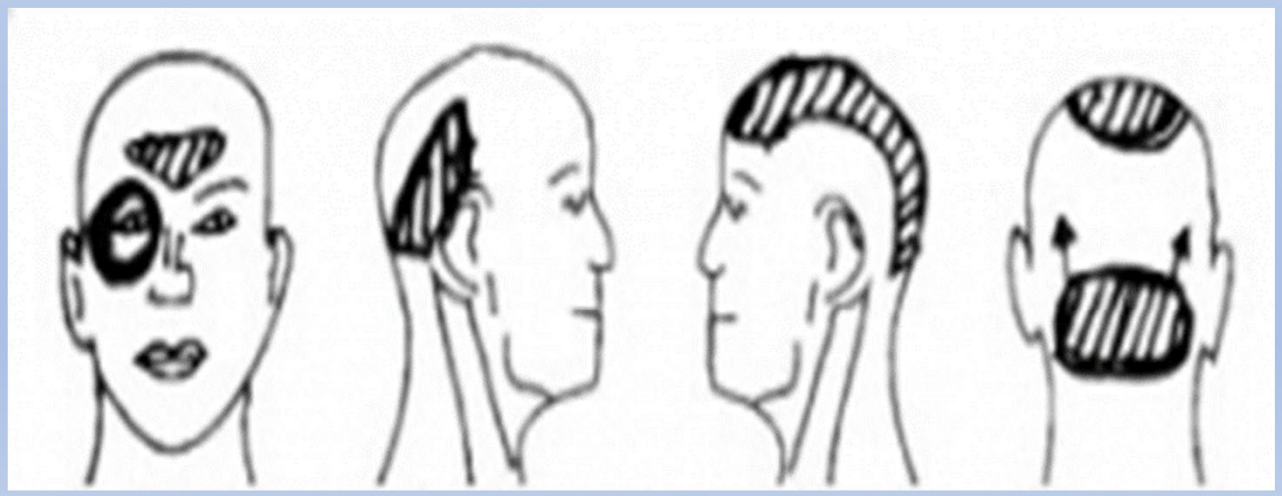




Headache Related to Neuropathic Pain Physical Exam

Findings on physical exam include:

- Signs of nerve injury detected during neurologic exam
- Pain may be elicited by palpation of face or scalp, especially over previous laceration or bruise
- May be associated with movement



Headache Related to Neuropathic Pain Treatment

Non-pharmacologic treatment

- Relaxation therapy
- Physical therapy
- Acupuncture
- Cognitive behavioral therapy (CBT)
- Massage therapy

Pharmacologic treatment§§§

Acute/Abortive Agents

First Line: Acetaminophen or NSAIDs
Second Line: Antiepileptics (e.g., gabapentin, topiramate);
tricyclic antidepressants (TCA) (e.g., amitriptyline, nortriptyline)

Preventive Treatment

Antiepileptics (e.g., gabapentin)

TCA) (e.g., amitriptyline, nortriptyline)

§§§ Attal, N., Cruccu, G., Baron, R., Haanpää, M., Hansson, P., Jensen, T.S., Nurmikko, T. & European Federation of Neurological Societies. (2010). EFNS guidelines on the pharmacological treatment of neuropathic pain: 2010 revision. *European Journal of Neurology* 17(9), 1113-e88. U.S. Food and Drug Administration Agency (FDA) warning cautions that NSAIDs can increase the risk of heart attack, heart failure, or stroke in patients with or without pre-existing heart disease, or risk factors for heart disease, even during the first few weeks of treatment, though the risk appears highest with longer use at higher doses.

General Post-traumatic Headache Treatment Recommendations

- Manage headache symptoms
- Physical and cognitive rest are important for healing and symptom resolution
- Avoid benzodiazepines[13,14], tramadol, opiates

Current PTH treatment evidence

Can we prevent chronic PTHs?

Review

ONLINE FIRST

January 19, 2021

Association of Pharmacological Interventions With Symptom Burden Reduction in Patients With Mild Traumatic Brain Injury

A Systematic Review

Charles Feinberg, BA[1]; Catherine Carr, MLIS[1]; Roger Zemek, MD[2,3]; et al

» Author Affiliations

JAMA Neurol. Published online January 19, 2021. doi:10.1001/jamaneurol.2020.5079

- Systematic review of articles published between 2000 and 2020.
- 23 studies examining 20 pharmacological interventions used to mitigate symptoms of mTBI.
- Most studies report symptom burden reduction after pharmacological intervention.

HEADACHE
The Journal of Head and Face Pain

Research Submissions

Use of Amitriptyline in the Treatment of Headache After Traumatic Brain Injury: Lessons Learned From a Clinical Trial

Max Hurwitz DO, Sylvia Lucas MD, PhD, Kathleen R. Bell MD, Nancy Temkin PhD, Sureyya Dikmen PhD, Jeanne Hoffman PhD

First published: 13 January 2020 | https://doi.org/10.1111/head.13748 | Citations: 2

Conflict of Interest: Max Hurwitz DO: No conflicts; Sylvia Lucas MD, PhD: Advisory Board-Amgen, Biohaven, Lilly, Teva; Kathleen Bell MD: Scientific Advisory Board Helius Medical, Research Funding – Microtransponder Inc., Scientific Advisory Board – Moss Rehabilitation Research Institute, Consultant – American Academy of Physical Medicine and Rehabilitation; Nancy Temkin PhD: No conflicts; Sureyya Dikmen PhD: No conflicts; Jeanne Hoffman PhD: No conflicts.
Funding: National Institute on Disability, Independent Living, and Rehabilitation Research H133G120055/90-IF-0025.
ClinicalTrials.gov Identifier: NCT01856270.

- Fifty participants who completed 90-day assessment. 24 participants were randomly assigned to take Amitriptyline at start of study or 26 to start thirty days after enrollment. Compliance was a limiting factor.
- The early use group had significant lower headache severity 15% vs 36% with pain rating of 6 or more (0-10 scale), p=.015.

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December 04, 2018; 91 (23 Supplement 1) ABSTRACTS

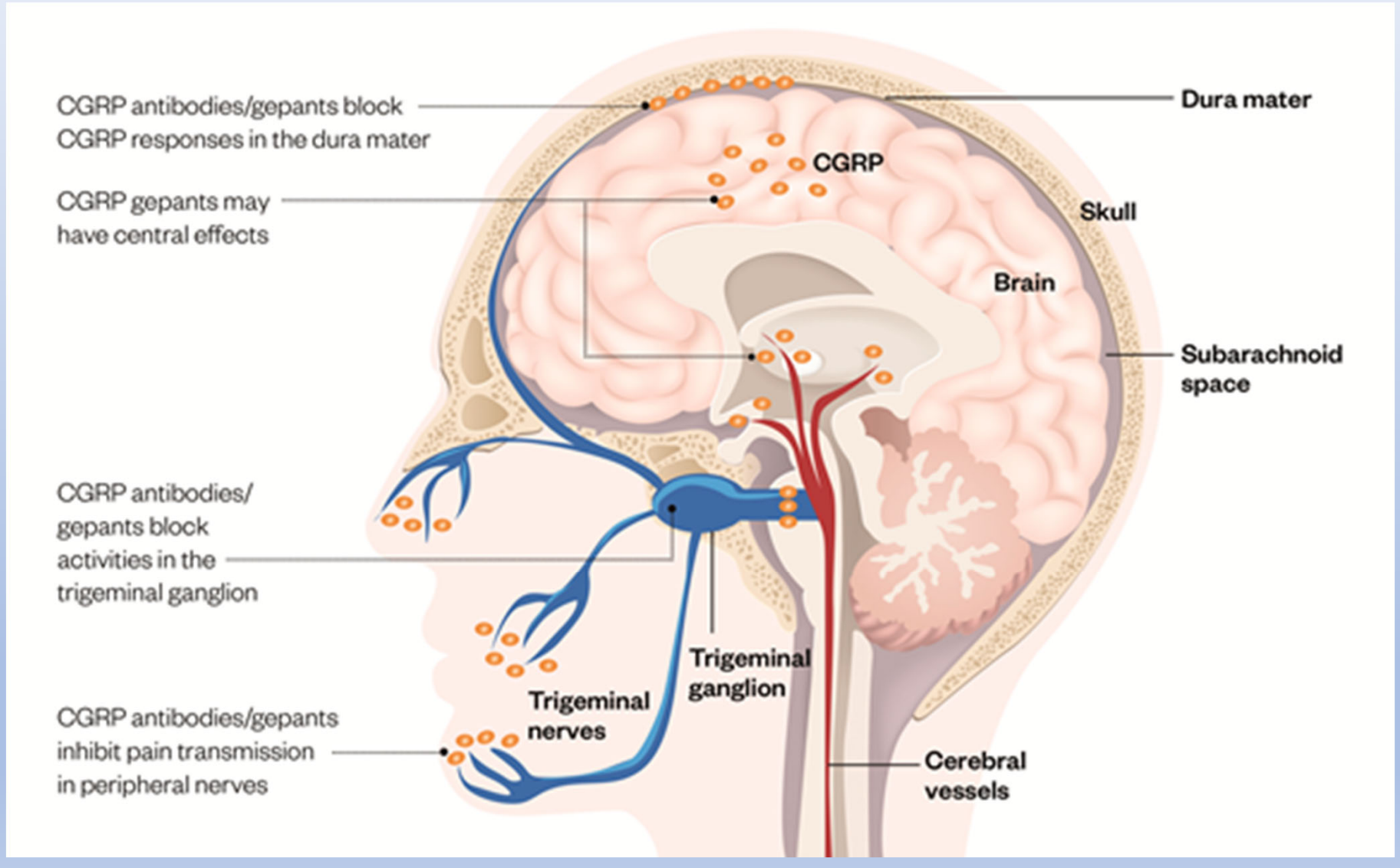
Early prophylactic treatment reduces development of chronic post traumatic headache after concussion

Crain Ian, Hoskin Justin, Al-Hasan Yazan, Zieman Glynnis, Cardenas Javier

First published December 5, 2018, DOI: https://doi.org/10.1212/01.wnl.0000550646.79759.e1

- This study had patients retrospectively enrolled from a patient population at the Barrow Concussion and Brain Injury Center (Phoenix, AZ)
- One hundred twenty patients were divided into three groups. (Untreated=36, Topamax=46, Nortriptyline=38), They were sub-divided into those that developed chronic PTH and those who didn't
- Conclusion: early intervention with non-pharmacological treatments and topiramate is associated with reduced development of cPTH

Other studies:
CGRP antagonists for Migraines



Summary

- Headache is the most common symptom after a concussion
- The four types of PTH are migraine, tension-type, cervicogenic and headache related to neuropathic pain
- Manage PTH by identifying the headache type it most closely resembles
- Early treatment can prevent long term morbidity and improve quality of life

Questions

Resources: Patient Apps

Mindfulness Coach

Concussion Coach

Breathe2Relax

- Developed by National Center for Telehealth & Technology, a Defense Centers of Excellence for Psychological Health and Traumatic Brain Injury center
- Available for free for Apple and Android devices

References

1. Lucas S, Hoffman JM, Bell KR, Dikmen S. A prospective study of prevalence and characterization of headache following mild traumatic brain injury. Cephalalgia 2014; 34:93.
2. Yilmaz T, Roks G, de Koning M, et al. Risk factors and outcomes associated with post-traumatic headache after mild traumatic brain injury. Emerg Med J 2017; 34:800.
3. Cnossen MC, van der Naalt J, Spikman JM, et al. Prediction of Persistent Post-Concussion Symptoms after Mild Traumatic Brain Injury. J Neurotrauma 2018; 35:2691.
4. Theeler BJ, Flynn FG, Erickson JC. Headaches after concussion in US soldiers returning from Iraq or Afghanistan. Headache 2010; 50:1262.
5. Kjeldgaard D, Forchhammer HB, Teasdale TW, Jensen RH. Cognitive behavioural treatment for the chronic post-traumatic headache patient: a randomized controlled trial. J Headache Pain 2014; 15:81.
6. Ashina H, Porreca F, Anderson T, et al. Post-traumatic headache: epidemiology and pathophysiological insights. Nat Rev Neurol 2019; 15:607.
7. Sawyer K, Bell KR, Ehde DM, et al. Longitudinal Study of Headache Trajectories in the Year After Mild Traumatic Brain Injury: Relation to Posttraumatic Stress Disorder Symptoms. Arch Phys Med Rehabil 2015; 96:2000.
8. Hoffman JM, Lucas S, Dikmen S, et al. Natural history of headache after traumatic brain injury. J Neurotrauma 2011; 28:1719.
9. Bryan, C. J. & Hernandez, A. M. (2011). Predictors of post-traumatic headache severity among deployed military personnel. Headache: *The Journal of Head and Face Pain*, 51(6), 945-953.
10. Cephalalgia. 2013;33:629–808. The International Classification of Headache Disorders: Third Edition-beta.
11. Ashina H, Iljazi A, Al-Khazali HM, et al. Hypersensitivity to Calcitonin Gene-Related Peptide in Post-Traumatic Headache. Ann Neurol 2020; 88:1220.
12. Friedman DI. Headaches Due to Low and High Intracranial Pressure. Continuum (Minneap Minn) 2018; 24:1066.
13. Lee, H., Lyketsos, C. & Rao, V. (2003). Pharmacological management of the psychiatric aspects of traumatic brain injury. *International Review of Psychiatry*, 15(4), 359–370.
14. Larson, E. B. & Zollman, F. S. (2010). The effect of sleep medications on cognitive recovery from traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 25(1), 61-7.