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Posttraumatic Headache: A Review

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Abstract There has been intense controversy about postconcussion syndrome (PCS) since Erichsen's publication in 1866 on railway brain and spine. Headache as a result of trauma is one of the most common secondary headache types. Posttraumatic headache (PTH) remains a very controversial disorder, particularly with relation to chronic PTH following mild closed-head injury. PTH is one of several symptoms of PCS, and therefore may be accompanied by additional cognitive, behavioral, and somatic problems. PTH also is an important public health issue due to its associated disability and often refractory clinical course. While current awareness of PTH has become more prominent due to increased scrutiny given to both combat-related and sports-related head injuries, directed treatment remains a difficult challenge for physicians. Because of the frequently associated medicolegal aspects, PTH is one of physicians' least favorite types to treat. The article reviews both PCS and PTH.

Keywords Posttraumatic headache · Traumatic brain injury (TBI) · Postconcussion syndrome · Combat-related headache · Sports-related headache

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Introduction

Posttraumatic headache (PTH) remains among the most controversial headache topics due to its propensity for chronicity and often associated medicolegal aspects. PTH can occur after mild, moderate, or severe traumatic brain injury (TBI), usually resolving within the first 3 months, although a minority develop chronic headaches. Paradoxically, PTH is more common after a mild TBI than more severe injuries [1••]. In a recent controlled prospective study on the prevalence of PTH following mild TBI, Faux and Sheedy [2] found 15% of those with minor head injury reported persistent PTH at 3 months, compared to 2% of controls.

While awareness among the public and physicians has recently increased due to combat-related head injuries occurring in US soldiers and head injuries in professional football players, treatment is challenging. This article reviews the history, epidemiology, types, pathophysiology, treatment, and prognosis of PTH.

History

Postconcussion syndrome (PCS) has been controversial for over 140 years. In 1879, Rigler [3] raised the important issue of compensation neurosis when he described the increased incidence of posttraumatic invalidism after a system for financial compensation was established for accidental injuries on the Prussian railways. PCS remained controversial throughout the 20th century as well. In 1961, Miller [4] summarized the viewpoint of those who believe that PCS is really a compensation neurosis: "The most consistent clinical feature is the subject's unshakable conviction of unfitness for work."

Epidemiology

There are approximately 1.4 million reported incidents of TBI in the United States every year [5] with mild injuries accounting for 70% to 90% [6]. However, many cases go unreported. Headaches are variably estimated as occurring in 25% to 78% of persons following mild TBI [7, 8•]. For an industrialized country such as the United States, estimates of the relative causes of TBI are as follows: motor vehicle accidents (45%), falls (30%), occupational accidents (10%), recreational accidents (10%), and assaults (5%) [9]. Headache is a cardinal feature and the most common symptom of PCS, a symptom complex that includes dizziness, fatigue, irritability, anxiety, insomnia, losses of concentration and memory, and noise sensitivity [7, 8•]. Loss of consciousness does not have to occur for PCS to develop. Whiplash-type injuries in motor vehicle accidents also result in PTH. No reporting system exists, and thus, the actual number of whiplash injuries per year is unknown.

Overall, PTH accounts for approximately 4% of all symptomatic headaches. According to the International Headache Society criteria, the onset of headache should be less than 7 days after an injury to be technically classified as posttraumatic in nature [10].

Types of Posttraumatic Headache

Tension-type Headache

Tension-type headaches (TTH) account for 85% of reported PTHs. They can occur in a variety of distributions, including a generalized, nuchal-occipital, bifrontal, bitemporal, cap-like, or headband location. The headache, which may be constant or intermittent with variable duration, is usually described as a pressure, tightness, or dull aching. It may be present on a daily basis.

Occipital Neuralgia

The term occipital neuralgia is a misnomer in some ways because the pain is not necessarily from the occipital nerve and does not usually have a neuralgic quality. Occipital neuralgia is a common type of PTH, but frequently is seen unrelated to injury. The pain may have an aching, pressure, stabbing, or throbbing quality and may be located in a nuchal-occipital, parietal, temporal, frontal, periorbital, or retro-orbital distribution (greater form) or lateral/around the ear (lesser form). Occasionally, a true neuralgia may be present, with paroxysmal shooting-type pain. The headache may last for minutes to days and can be unilateral or bilateral.

The headache may be due to entrapment of the greater occipital nerve in the aponeurosis of the superior trapezius or semispinalis capitis muscle, or may be a referred pain without nerve compression from trigger points in these or other suboccipital muscles. Digital pressure over the greater occipital nerve at the midsuperior nuchal line reproduces the headache. However, pain referred from cervical and posterior fossa pathology can present similarly [11].

Migraine Headache

Recurring attacks of migraine (with and without aura) can result from mild head injury. After minor head trauma, patients of all ages can develop a variety of transient neurological sequelae that are not always associated with headache and are perhaps due to vasospasm. Five main clinical types are recognized: hemiparesis; somnolence, irritability, and vomiting; a confusional state; transient blindness, often precipitated by occipital impacts; and brainstem signs [12].

Cluster Headache

Posttraumatic cluster headache is an extremely rare entity (lifetime prevalence, 1%) and is infrequently reported in the medical literature. Cluster headache (CH) is a primary headache disorder classified as one of the trigeminal autonomic cephalgias [13]. Acute attacks involve the trigeminovascular system with associated unilateral excruciating pain. These events typically include the autonomic symptoms of lacrimation, ptosis, conjunctival injection, nasal stuffiness, and rhinorrhea. It is suggested that the "Type A," risk-taking lifestyle of patients with CH may predispose them to bodily harm and subsequent TBI [14, 15].

Low Cerebrospinal Fluid Pressure Headache

Trauma has been recognized as a cause of intracranial hypotension due to a cerebrospinal fluid (CSF) leak [16]. Such trauma is often quite trivial in nature, including coughing, minor falls, lifting, or pushing. In more pronounced trauma, the likely mechanism is due to significant impact on the spinal axis at the time of injury, resulting in a transient rise in CSF pressure with subsequent dural root sleeve tear [17]. Blunt trauma may also result in a cribriform plate fracture, predisposing to a CSF leak.

Supraorbital and Infraorbital Neuralgia

Injury of the supraorbital branch of the first trigeminal division as it passes through the supraorbital foramen just inferior to the medial eyebrow can cause supraorbital neuralgia. Similarly, infraorbital neuralgia can result from trauma to the inferior orbit. Shooting, tingling, aching, or burning pain along with decreased or altered sensation and sometimes decreased sweating in the appropriate nerve

distribution may be present. The pain can be paroxysmal or fairly constant.

Whiplash and Cervicogenic Headache

Neck injuries commonly accompany head trauma and can produce headaches. The specific mechanism involves the merging of trigeminal and cervical afferents in the trigeminocervical nucleus [18, 19]. Typical pain includes throbbing and/or pressure-like pain originating in the occipital region, migrating anteriorly to involve the temporoparietal areas in a unilateral distribution. These headaches can often display migrainous qualities as well. Posttraumatic cervicogenic headaches are also commonly associated with whiplash injuries.

Whiplash is a sudden acceleration then deceleration of the neck resulting in pain at the time of trauma. The triad of neck pain, restriction/neck mobility, and headache are the major constituents of whiplash syndrome. There is a general female predominance in whiplash cases, with approximately two-thirds being female [10, 19, 20]. A previous primary headache disorder is known to be a major risk factor for headache both in the acute and chronic stages following a head or neck injury.

Other Types

Subdural and epidural hematomas both may be associated with headache due to the associated space-occupying abnormalities. The patient is frequently a child or young adult who sustains an apparently trivial injury, often without loss of consciousness. A persistent headache then develops, often associated with nausea, vomiting, and memory impairment, that might seem consistent with a PCS. After the passage of days to weeks, focal findings develop. Hemorrhagic cortical contusions may also cause headache due to subarachnoid hemorrhage.

New-onset orgasmic cephalgia can follow mild head trauma within 3 to 4 weeks. A novel form of trigeminal-based pain termed posttraumatic external nasal pain syndrome was recently described by Rozen [21]. Other less common forms of PTH reported include trigeminal neuralgia; dysautonomic cephalgia; hemicrania continua; paroxysmal hemicrania; short-lasting unilateral neuralgiform attacks with conjunctival injection and tearing syndrome; short-lasting unilateral neuralgiform headache attacks with cranial autonomic features; and temporomandibular joint injury [22].

Pathophysiology of Posttraumatic Headache

The pathophysiology of mild TBI and associated PTH is thought to be partly neurometabolic in origin. Release of excitatory neurotransmitters such as aspartate, glutamate, and acetylcholine may serve as a potential substrate for mild TBI. For most individuals, mild TBI is not associated with macroscopic abnormalities on neuroimaging. However, some studies, including MRI, single-photon emission computerized tomography, and positron emission tomography, can demonstrate structural and functional deficits [23]. Recent studies using diffusion tensor imaging (DTI) are proving to be more sensitive than conventional imaging methods in detecting subtle but clinically meaningful changes following mild TBI. Specific pathways of interest include the hippocampal fornix, inferior fronto-occipital, inferior longitudinal fasciculus, corpus callosum, corticospinal tracts, and the uncinate fasciculus [24••]. DTI was used by Kraus et al. [25•] to confirm that greater white matter pathology predicted greater cognitive deficits, suggesting DTI as an objective means for determining the relationship of cognitive deficits to TBI. MacKenzie et al. [26] found whole-brain atrophy occurring even after mild TBI, with the amount of atrophy being most pronounced in those injuries producing loss of consciousness. DTI studies have also discovered regions of associated axonal swelling in patients with mild TBI [27]. It is now recognized that axons are not sheared at the time of injury in mild TBI, but instead undergo a series of changes that may result in a secondary axotomy within 24 h [27]. Overall, the etiology of PTH is likely multifactorial and remains poorly understood.

Controversy Surrounding PCS and PTH

Some symptoms of PCS will be experienced by 30% to 80% of patients with mild to moderate TBI [28]. PCS has been considered controversial almost immediately following its introduction 144 years ago. A subdivision into early PCS and late/persistent PCS (symptoms and signs persisting for more than 6 months) can be helpful in identifying potential confounding factors. About 20% of patients will develop the persistent type. In the late group, psychological pathology and compensation issues may contribute to persisting symptoms. For the minority of patients with persistent postconcussion symptoms, a variety of explanations have been advanced, including the following: psychogenic disorders; psychosocial problems; base rate misattribution; chronic pain; compensation and litigation; expectation of chronic symptoms; and malingering [29].

PTH in United States Soldiers

Since the start of military operations to date, more than 1.8 million United States combat personnel have been deployed

to Afghanistan and Iraq. Blasts or explosions account for about 75% of mild TBIs. A history of mild head trauma, usually caused by exposure to blasts, is found in nearly half of returning US soldiers seeking specialized care for headaches. In many cases, the head injury was temporally associated with either the onset of headaches or the worsening of preexisting headaches, implicating trauma as a precipitating or exacerbating factor [30].

There are only a few studies of the types, durations, risk factors, and overall prevalence of PTHs in US soldiers injured in Iraq and Afghanistan. Helseth and Erickson [31] reported migraine as an underdiagnosed and undertreated entity in US Army trainees, often adversely affecting training due to illness and injury-related lost work days. Migraine is considered a significant medical problem in service personnel due to the strenuous work environment and exposure to known stressors. Triggers for migraines can include sleep deprivation, physical exertion, change in environmental conditions, emotional stress, and dehydration. A 2008 study by Theeler et al. [32] found a high prevalence of migraine in US soldiers deployed to combat without physical trauma, with 17.4% of males and 34.9% of females reporting a headache consistent with migraine during the prior year, much greater than a civilian population [32]. In contrast to civilian injuries, migraines account for perhaps 75% of PTH in soldiers [32, 33••]. PTH is also more common in those with a history of headache. Such observations indicate further research is needed to better understand the propensity for clinical migraine expression in combat personnel experiencing PTH versus TTH in their civilian counterparts.

Hoge et al. [34] report that mild TBI occurring among deployed soldiers is also strongly associated with posttraumatic stress disorder (PTSD) and physical health problems 3 to 4 months after soldiers return home. Individuals with mild TBI and PTSD require treatment of both conditions for optimal clinical outcome. A definitive step in providing effective care for TBI is identifying individuals who may benefit from treatment. During wartime this is extremely difficult, given the strained medical system in the field. As a result, less obvious milder injuries such as closed-head injury without loss of consciousness may be overlooked or only receive limited attention. Unfortunately, this also happens among injured military personnel returning from deployment in Iraq and Afghanistan. Clinical screenings conducted at Walter Reed Army Medical Center by the Defense and Veterans Brain Injury Center identified a substantial number of patients with previously undiagnosed TBI [35•]. Many of those patients were being medically evaluated for other reasons. It was not until specialized screening was undertaken that their TBI was identified. The Department of Veterans Affairs has recently recognized that resources are inadequate to deal with the number of patients returning from combat with sequelae of TBI [36]. Fortunately, families and communities continue to become more cognizant of the special needs of these returning veterans. Given the great number of associated PTHs and the likelihood of chronicity in some, growing numbers will be seen by civilian physicians in years to come as soldiers are discharged and enter civilian life seeking care outside of veteran's facilities.

PTH in Sports

Mild TBI and its associated sequelae have been long associated with competitive athletics. Approximately 300,000 of the 1.5 million head injuries reported each year in the United States are thought to be sports related, 9% of which require hospitalization [37]. The far majority of these events occur in athletes in high school or younger. A substantial minority of amateur and professional athletes who sustain a concussion report no residual symptoms 24 h postinjury. McCrea et al. [38] found 90% of National Collegiate Athletic Association (NCAA) football players who have a concussion to have a complete clinical recovery within 7 days [38]. In a recent large-scale prospective study of high-school football players, Collins et al. [39] found approximately 90% of athletes who have a concussion to have definite clinical recovery within 1 month. Despite this likelihood of brief symptom duration and short time period to recovery, a minority progress to an unrelenting chronic process.

The NCAA Football Rules Committee in February 2010 announced support of more stringent return-to-play standards for players exhibiting in-game concussive symptoms. Concussion management in the National Football League (NFL) has also received intense public scrutiny of late due to previous criticisms regarding their handling of acute head injuries. On December 2, 2009, the same day that head injuries to stars Ben Roethlisberger, Kurt Warner, and Brian Westbrook dominated the sports news, the NFL announced its most stringent guidelines to date. Symptoms requiring immediate removal from a NFL game now include amnesia, poor balance, and an abnormal neurological exam, regardless of whether those symptoms resolve during the course of the game [40]. Interestingly, under the current revised guidelines a player can return to the field despite the complaint of headache, as long as it is not deemed "persistent." NFL commissioner Roger Goodell maintains that some level of discretion must be left to team physicians, allowing for their independent interpretation of symptoms. This topic of return-to-play restrictions has also received recent attention on a legislative level. Many states are considering measures that would toughen restrictions on young athletes returning to play after head injuries.

Washington led the way last year, passing what is considered the nation's strongest return-to-play statute. Athletes under 18-years-old showing concussion symptoms cannot take the field again without a licensed health care provider's written approval. Several other states have similar bills pending.

Despite this recent attention given to concussion-related symptoms, no credible studies exist regarding the prevalence of PTH in the NFL. Headache is consistently the most common symptom following concussion and occurs in approximately 85% of athletes with sports-related concussion [41]. Very few studies have examined the effects of headache (PTH or otherwise) regarding quality of life in the athletic population. As a result of the intensity of activity and the occurrence of head trauma, athletes are certainly exposed to significant headache risk factors. Sallis and Jones [42] investigated this topic in a sample of both college and high school football players in the United States. A previous headache related to hitting during a football game was reported by 85%. A headache during the specific game questioned was reported by 21% of respondents. Only 19% of those experiencing headache informed appropriate medical personnel, and only 6% of those were removed from play. Ingebrigtsen et al. [43] found approximately 40% of athletes who experience a concussion developed other postconcussive symptoms such as drowsiness, dizziness, irritability, and sleep disorders [43]. Similar to military personnel, PTH experienced in athletes is often a migrainous phenotype. Individuals reporting headache immediately following sports-related injury have also been shown to have greater neurocognitive deficits [44]. However, adherence to an evidence-based treatment paradigm utilizing standardized computer-based neurocognitive testing allows most injured athletes to safely resume competition within 1 to 2 weeks.

Repetitive brain trauma is now well-associated with progressive neurological deterioration. This entity, now known as chronic traumatic encephalopathy (CTE), is a progressive neurodegenerative disorder clinically associated with memory disturbances, behavioral and personality changes, parkinsonism, and speech and gait abnormalities. Pathologically it is characterized by cerebral and medial temporal lobe atrophy, ventriculomegaly, and extensive tau protein deposition. The neurofibrillary tangles and neuritic threads visualized originate in the neocortex and progress to the hippocampus [45]. The clear environmental etiology of CTE allows for a definitive neuropathological distinction from other neurodegenerative disorders.

Popular media attention has highlighted the behavioral disturbances of CTE, including the case of former professional wrestler Christopher Benoit. Prior to hanging himself in 2007, Benoit is believed to have asphyxiated his wife and son. Subsequent autopsy revealed significant accumulation of tau protein, consistent with the diagnosis of CTE [46]. Autopsies performed on former NFL players with multiple recorded concussions revealed similar findings. These players were reported postmortem by family to have significant behavioral and psychiatric complaints postretirement. In 2005, Guskiewicz et al. [47] surveyed 3683 retired professional football players belonging to the NFL Retired Players Association. They found retired NFL players to have a 37% higher risk of Alzheimer's than other US males of the same age. At least one concussion during playing career was reported by 60.8% of players, while 24% reported three or more. Of those having at least one concussion, 17.6% reported a permanent effect on their thinking or memory skills [47].

Currently, there are no reliable biomarkers for the diagnosis of CTE. Further studies are underway to identify and define the neuropathological cascades of CTE in football players suffering repetitive mild TBI. This clinical data could help to form the basis for prophylactic and therapeutic points of intervention. Given the incidence of concussion and subsequent PCS in current and former professional football players, the authors would encourage investigation into the possible predictive nature of PTH and the development of CTE in this patient population.

Treatment

A multidisciplinary approach is stressed in the treatment of PTH due to the multitude of associated symptoms. There is a paucity of medical literature on headache management specific to PTH. Before the implementation of any treatment plan, the physician must perform a comprehensive and systematic evaluation. Prophylactic therapies for a migraine or tension-type presentation include the standard approach with β-blockers, antidepressants, or antiepileptic drugs. To date, only three studies, all done without controls, have been performed investigating the medical prophylaxis of PTH. Each of these studies, which involved either a singular or dual approach consisting of propranolol, amitriptyline, or valproate [48•], showed statistically significant efficacy. For acute attacks and exacerbations, NSAIDs, simple analgesics, and triptans are usual first-line options depending upon the headache type. Physicians should be aware of potential medication-overuse headaches with the frequent use of over-the-counter medications containing caffeine. Medication-overuse headache may also result from the overuse of ergotamines, triptans, and possibly NSAIDs. Prescription drugs containing narcotics, butalbital, or benzodiazepines generally are used sparingly and with caution due to the risk for abuse, habituation, and medication-overuse headaches.

Trigger point injections and occipital nerve blocks may be effective. Loder and Biondi [49] reported botulinum

toxin injections as efficacious for chronic PTH. Non-pharmacologic treatments include the following: physical therapy and manipulation; biofeedback and relaxation therapy; transcutaneous nerve stimulators; and cognitive and behavioral therapies. Prospective studies of existing treatments and new approaches are clearly needed for more effective management.

The complaints of many patients with persistent PTH and other legitimate postconcussion symptoms may be inappropriately minimized by health care professionals, family members, friends, and employers. They may have sustained a mild head injury with minimal if any external trauma and seem fine.

Education about mild TBI and headaches is an important part of the treatment. Physicians also can use the increasingly familiar examples from sports and well-known athletes who had to retire due to PCS such as Troy Aikman and Pat Lafontaine, which ironically lends a certain credibility to the media reports and public fascination with elite athletes. The recent attention to injuries of US soldiers has also raised awareness.

Prognosis

Most patients suffering from PTHs recover within a few months, with a small but significant minority having persistent problems. Because of the many variables in prognostic studies, the percentage of patients with symptoms after mild head injury varies greatly. The percentage of patients with headaches at 1 month varies from 31.3% to 90%, at 3 months from 47% to 78%, and at 1 year from 8.4% to 35%. Almost 25% of patients have persisting headaches at 4 years [50].

Effect of Litigation

Some studies find that litigation is the explanation for persistence of PTH. When tort compensation in Saskatchewan, Canada was changed to a no-fault system without payments for pain and suffering, the number of claims decreased by about 25%. Studies demonstrate a relationship between persistent PCS and potential financial compensation [29]. Neuropsychological testing has shown a dose-response relationship between an increasing amount of potential compensation and an increasing rate of failure on malingering indicators. This has been shown to be especially pronounced in those who have suffered only a mild TBI. On the other hand, failure of patients to recover after claims are settled does not necessarily invalidate this theory, as a financial settlement may in fact reinforce illness behavior [50].

Conclusions

PTH is the most frequent complaint of individuals suffering mild TBI and remains a poorly understood entity. The clinical presentation may reflect a coalescence of both organic and psychological injury. Treatment remains challenging in many instances due to the necessity of a multithemed approach. A vital role for the treating physician is education of the patient and family members, stressing the need for realistic goals and expectations in this often frustrating disorder. Very little quality evidence exists regarding specific PTH treatment options. There is a definite need for double-blind placebo-controlled treatment trials to define the most efficacious approach. The topic is part of an evolving field of neurological sciences attracting interest from multidisciplinary research groups worldwide. With time, this interest will allow for a better understanding of the PTH etiology and ultimately result in better treatment options for patients.

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References

Papers of particular interest, published recently, have been highlighted as:
- • Of importance
- •• Of outstanding importance

1. •• Solomon S: Post-traumatic headache: commentary: an overview. Headache 2009, 49:1112–1115. *This thorough yet concise overview of PTH discusses history, clinical features, etiology, and treatment.*
2. Faux S, Sheedy J: A prospective controlled study in the prevalence of posttraumatic headache following mild traumatic brain injury. Pain Med 2008, 9:1001–1011.
3. Evans R: Expert opinion: posttraumatic headaches among United States soldiers injured in Afghanistan and Iraq. Headache 2008, 48:1216–1225.
4. Miller H: Accident neurosis. Br Med J 1961, 1:919–925.
5. Centers for Disease Control and Prevention: Get the Stats on Traumatic Brain Injury in the United States: 2002–2006. Available at http://www.brainline.org/content/2010/03/get-the-stats-on-traumatic-brain-injury-2002-2006.html. Accessed January 2010.
6. Cassidy JD, Carroll LJ, Peloso PM, et al.: Incidence, risk factors and prevention of mild traumatic brain injury: results of the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. J Rehabil Med 2004, 36(Suppl 43):28–60.
7. Evans RW: The postconcussion syndrome and the sequelae of mild head injury. In Neurology and Trauma, edn. 2. Edited by Evans RW. New York: Oxford University Press; 2006:95–128.
8. • Evans RW: Postconcussion syndrome. Available at http://www.uptodate.com. Accessed January 2010. *This extremely thorough review discusses past and recent controversies, clinical characteristics, and the prognosis of PCS.*

9. Jennett B, Frankowski RF: The epidemiology of head injury. In Handbook of Clinical Neurology, vol. 13. Edited by Braakman R. New York: Elsevier, 1990:1–16.
10. Headache Classification Subcommittee of the International Headache Society: The International Classification of Headache Disorders: 2nd edition). Cephalalgia 2004, 24(Suppl 1):9–160
11. Lord SM, Barnsley L, Wallis BJ, Bogduk N: Third occipital headache: a prevalence study. J Neurol Neurosurg Psychiatry 1994, 57:1187–1190.
12. Weinstock A, Rothner AD: Trauma-triggered migraine: a cause of transient neurologic deficit following minor head injury in children. Neurology 1995, 45(Suppl 4):A347–A348.
13. Goadsby PJ: Pathophysiology of cluster headache: a trigeminal autonomic cephalgia. Lancet Neurol 2002, 1:251–257.
14. Bahra A, May A, Goadsby PJ: Cluster headache: a prospective clinical study with diagnostic implications. Neurology 2002, 58:354–361.
15. Ekbom K: Lifestyle factors in males with cluster headache. Cephalalgia 1999, 19:73–74.
16. Moriyama E, Ogawa T, Nishida A, et al.: Quantitative analysis of radioisotope cisternography in diagnosis of intracranial hypotension. J Neurosurg 2004, 101:421–426.
17. Vilming ST, Campbell JK: Low cerebrospinal fluid pressure. In The Headaches, edn. 2. Edited by Olesen J, Tfelt-Hansen P, Welch KM. Philadelphia: Lippincott Williams & Wilkins; 2000:831–839.
18. Bogduk N: The neck and headaches. Neurol Clin 2004, 22:151–171.
19. Balla J, Jansek R: Headaches arising from disorders of the cervical spine. In Headache: Problems in Diagnosis and Management. Edited by Hopkins A. Philadelphia: W. B. Saunders; 1998:241–267.
20. Radanov BP, Sturzenegger M, Di Stefano G: Long term outcome after whiplash injury. Medicine (Baltimore) 1995, 74:281–297.
21. Rozen T: Post-traumatic external nasal pain syndrome (a trigeminal based pain disorder). Headache 2009, 49:1223–1228.
22. Putzki N, Nirkko A, Diener HC: Trigeminal autonomic cephalgias: a case of post-traumatic SUNCT syndrome? Cephalalgia 2005, 25:395–397.
23. Lewine JD, Davis JT, Sloan JH, et al.: Neuromagnetic assessment of pathophysiologic brain activity induced by minor head trauma. AJNR Am J Neuroradiol 1999, 20:857–866.
24. •• Singh M, Jeong J, Hwang D, et al.: Novel diffusion tensor imaging methodology to detect and quantify injured regions and affected brain pathways in traumatic brain injury. Magn Reson Imaging 2010, 1:22–40. *This thought-provoking case-controlled study investigates the application of diffusion tensor imaging in the detection and quantification of sites of TBI.*
25. • Kraus MF, Susmaras T, Caughlin BP, et al.: White matter integrity and cognition in chronic traumatic brain injury: a diffusion tensor imaging study. Brain 2007, 130:2508–2519. *This case-controlled study investigates white matter integrity post-TBI of all severities via diffusion tensor imaging.*
26. MacKenzie JD, Siddiqi F, Babb JS, et al.: Brain atrophy in mild or moderate traumatic brain injury: a longitudinal quantitative analysis. AJNR Am J Neuroradiol 2002, 23:1509–1515.
27. Bazarian JJ, Zhong J, Blyth B, et al.: Diffusion tensor imaging detects clinically important axonal damage after mild traumatic brain injury: a pilot study. J Neurotrauma 2007, 24:1447–1459.
28. Ruff R: Two decades of advances in understanding of mild traumatic brain injury. J Head Trauma Rehabil 2005, 20:5–18.
29. Evans RW: Persistent post-traumatic headache, postconcussion syndrome, and whiplash injuries: the evidence for a non-traumatic basis with a historical review. Headache 2010, 50:716–724.
30. Ommaya AK, Ommaya AK, Dannenberg AL, Salazar AM: Causation, incidence and costs of traumatic brain injury in the U.S. Military Medical System. J Trauma 1996, 40:211–217.
31. Helseth EK, Erickson JC: The prevalence and impact of Migraines on US Military officer trainees. Headache 2008, 48:883–889.
32. Theeler BJ, Mercer R, Erickson JC: Prevalence and impact of migraine among US Army soldiers deployed in support of Operation Iraqi Freedom. Headache 2008, 48:876–882.
33. •• Vargas BB: Posttraumatic headache in combat soldiers and civilians: what factors influence the expression of tension-type versus migraine headache? Curr Pain Headache Rep 2009, 13:470–473. *This thoughtful article explores the features unique to the combat environment that predispose to the expression of migraine headache post-TBI.*
34. Hoge CW, McGurk D, Thomas JL, et al.: Mild traumatic brain injury in U.S. soldiers returning from Iraq. N Engl J Med 2008, 358:453–463.
35. • Schwab KA, Ivins B, Cramer G, et al.: Screening for traumatic brain injury in troops returning from Deployment in Afghanistan and Iraq: initial investigation of the usefulness of a short screening tool for traumatic brain injury. J Head Trauma Rehabil 2007, 22:377–389. *This informative article details a preliminary assessment of a screening instrument aimed to identify individuals having suffered TBI.*
36. Department of Veterans Affairs Office of Inspector General: Follow-up Healthcare Inspection. VA's Role in Ensuring Services for Operation Enduring Freedom/Operation Iraqi Freedom Veterans after Traumatic Brain Injury Rehabilitation. Available at http://www.va.gov/oig/54/reports/VAOIG-08-01023-119.pdf. Accessed June 2008.
37. Thurman DJ, Branche CM, Sniezek JE: The epidemiology of sports-related traumatic brain injuries in the United States: recent developments. J Head Trauma Rehabil 1998, 13:1–8.
38. McCrea M, Guskiewicz KM, Marshall SW, et al.: Acute effects and recovery time following concussion in collegiate football players: the NCAA Concussion Study. JAMA 2003, 290: 2556–2563.
39. Collins MW, Lovell MR, Iverson GL, et al.: Examining concussion rates and return to play in high school football players wearing newer helmet technology: a three-year prospective cohort study. Neurosurgery 2006, 58:275–286.
40. Schwarz A: N.F.L. Issues New Guidelines on Concussions. Available at http://www.nytimes.com/2009/12/03/sports/football/03concussion.html. Accessed December 2009.
41. Guskiewicz KM, Weaver NL, Padua DA, Garrett WE Jr: Epidemiology of concussion in collegiate and high school football players. Am J Sports Med 2000, 28:643–650.
42. Sallis RE, Jones K: Prevalence of headaches in football players. Med Sci Sports Exerc 2000, 32:1820–1824.
43. Ingebrigtsen T, Waterloo K, Marup-Jensen S, et al.: Quantification of post-concussion symptoms 3 months after minor head injury in 100 consecutive patients. J Neurol 1998, 245:609–612.
44. Mihalik JP, Stump JE, Collins MW, et al.: Posttraumatic migraine characteristics in athletes following sports-related concussion. J Neurosurg 2005, 102:850–855.
45. •• McKee AC, Cantu RC, Nowinski CJ, et al.: Chronic traumatic encephalopathy in athletes: progressive tauopathy after repetitive head injury. J Neuropathol Exp Neurol 2009, 68:709–735. *This comprehensive article reviews the history, clinical features, and pathophysiology of CTE.*
46. Cajigal S: Brain Damage May Have Contributed to Former Wrestler's Violent Demise. Neurology Today 1997, 7:1, 16.
47. Guskiewicz KM, Marshall SW, Bailes J, et al.: Association between recurrent concussion and late-life cognitive impairment in retired professional football players. Neurosurgery 2005, 57:719–726.
48. • Gladstone J: From psychoneurosis to ICHD-2: an overview of the state of the art in post-traumatic headache. Headache 2009, 49:1097–1111. *This thorough review highlights the classification, epidemiology, evaluation, and management of PTH.*
49. Loder E, Biondi D: Use of botulinum toxins for chronic headaches: a focused review. Clin J Pain 2002, 18:169–176.
50. Evans EW: The Postconcussion Syndrome. In Neurology and General Medicine edn. 4. Edited by Aminoff M. Philadelphia: Elsevier; 2007:593–603.