CHAPTER 6

Postconcussive Headache

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INTRODUCTION

Polytrauma, including traumatic brain injury, has been associated with significant pain sequelae.[1] Postconcussive headache (PCH) has historically been viewed as a singular headache disorder with some quoting an incidence of headache occurring in nearly 90% of concussive brain injuries (CBIs) with a fairly alarming rate of chronicity. Whether PCH chronicity is a reflection of our lack of understanding of the condition and/or a consequence of suboptimal diagnoses remains to be elucidated. The idea that PCH is a singular headache disorder is now known to be an oversimplification of a much more complex and variable pathoetiology that is often mixed with diverse biopsychosocial factors that may influence symptom presentation, pain adaptation versus promulgation, and prognosis. Other factors such as base rate misattribution (e.g., migraine incidence in the military), sociocultural issues, nocebo effects, and/or compensation/litigation must also be considered.[2–4] Increased understanding regarding the array of post-traumatic headache (PTHA) pain generators following CBI has further emphasized the complexity of PCH and the interrelationships of vascular, autonomic, peripheral nerve, muscular, osseous/joint, and other contributors to such headaches.[3–5]

Risk factors for development of specific headache phenotypes may differ in different types of CBI mechanisms, including assaults, vehicular, sports, whiplash, and blast injury, but no methodologically sound data are available yet to answer this question. Theoretically, each of the aforementioned mechanisms of injury may put a patient at greater risk for certain types of post-traumatic head pain generators than others. Additionally, the acute, subacute, and chronic variants of PCH have not been studied in a manner to determine differences in incidence, prevalence, etiology, diagnostic accuracy, treatment efficacy and prognosis. Similarly, risk factors for PCH and vulnerabilities for developing so-called "chronic" PCH remain debated in part due to variable study methodology. Some literature suggests that preinjury migraine is associated with protracted recovery from sports-related CBI, greater levels of postconcussive symptom complaints, as well as more impairment on subacute neuropsychological testing (particularly in females), although controversy remains.[6,7] The bulk of the literature does note a positive association between acute headache symptom burden and protracted recovery from CBI.[6,7]

The effect genetic loading risk factors, preexisting neck pain, and/or headache associated with whiplash injury, impact preparedness, dizziness, sex, anatomic variables between males and females, cultural norms/perspectives, affective disorders, and characterological traits have on influencing PCH incidence/reporting, severity, and duration are still debated. There is some literature, however, that suggests that such factors may impact outcome. PCH can adversely impact neuropsychological testing performance, alter sleep quality and behavior (e.g., irritability and decreased frustration tolerance), as well as lead to secondary symptoms of depression and anxiety and has also been associated with poorer outcomes from CBI.[3,4]

Terminology remains in limbo and there is no international consensus on how best to "label" PCHs; however, consideration should be given to avoiding nonhelpful generic labels such as PCH or PTHA and not providing further diagnostic information. Instead, practitioners need to stipulate the presumed pain generators (e.g., cervicogenic, migraine, tension, neuralgic, etc.), which may at times be mixed in order to adequately direct treatment decisions.[8] Use of such general terms may also cause practitioners and others to misattribute the headache disorder to traumatic brain injury when, in fact, its cause may be extracerebral as in the case of impact injury, post-traumatic neuralgic or neuritic pain, referred myofascial and cervicogenic headache, among other possibilities. The phrase

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"chronic PCH" should also be avoided since a chronicity label may only have been given due to an incorrect diagnosis and/or treatment, which results in persistence of the underlying pain disorder. Such labels can only serve to negatively bias evaluating clinician perspectives. It is therefore preferential to document the headache onset date and duration of symptoms as well as the specific presumed pain generators.

CLASSIFICATION CHALLENGES

Use of the current IHS (International Headache Society) International Classification of Headache Disorders (ICHD-3 and 3B) systems has its limitations with regard to how PCHs and PTHAs in general are classified. There is a lack of differentiation of the various causes of PCH/PTHA and all PTHAs are categorized under section 5 of ICHD-3 and are divided into three broad categories relative to headache being due to trauma to the head, neck, or secondary to craniotomy. The specific categories include acute as well as persistent headache attributed to traumatic injury to the head, acute as well as persistent headache attributed to whiplash, and acute as well as persistent headache attributed to craniotomy. Acute PTHA is defined as headache that resolves within 3 months from the date of injury, whereas persistent PTHA refers to headache lasting greater than 3 months (although this is somewhat distinct from typical "chronic pain" definitions that use a timeframe of greater than 6 months before chronicity is established). There is additional division of the classification system based on the severity of TBI, which is dualistic and divides TBI into two broad categories of mild TBI and moderate/severe TBI. Onset of headache must be within 7 days of injury (although to our knowledge this is empirical and not evidence-based), regaining consciousness or discontinuation of medications that might impair the ability to sense or report headache following the injury in question.[9] The 7-day onset criterion has been shown to underestimate incidence of this disorder; specifically, PCH may have its onset after 1 week post injury.[10] The terminology used in ICHD is confusing as brain injury and head injury appear to be used interchangeably and should not be since the two phenomena can occur separately or conjointly. There is no mention of the myriad other causes of PTHA such as migraine, tension-type headache (TTHA), neuralgic/neuritic headache, among numerous other conditions that are categorized elsewhere but may present following trauma.[11] Another problem with use of ICHD is the fact that it is only symptom based and does not consider physical examination findings or any type of diagnostic testing.[8] Based on symptom profiling, ICHD-3 has the potential to incorrectly categorize secondary headache disorders due to trauma, including overdiagnosis of migraine.[3,4] As noted by Dwyer and others, no specific headache features or treatment approaches accompany the definition, which likely limits its usefulness in both research and clinical practice.[11] Whether classification systems, such as IHS ICHD-3[9] or ICD-10,[12] are truly relevant to PCH and PTHA, more generally, remains to be seen. In the context of secondary headache disorders, such classification systems may have limited value and the potential to incorrectly or inadequately classify these headache subtypes, which in turn may lead to inappropriate treatment being rendered and as a consequence a suboptimal outcome. As a consequence of these concerns, some have advocated for a more multifaceted classification system taking into consideration the nature of the brain injury and symptom-based profiles.[13]

LIMITATIONS OF EXISTING LITERATURE

Historical studies assessing incidence of headache subtypes post-TBI have relied on neurological headache classification systems such as ICHD and ICD with little or no regard to the overlap of symptoms of PCH with PCD (i.e. autonomic symptoms like photosensitivity and sonosensitivity), physical exam correlates of underlying pain generators, and/or headache pain symptom validity measures. Additionally, the role, if any, of secondary gain incentives including litigation, worker's compensation, and social security disability in headache reporting and prognosis remains to be edified. What contribution comorbid affective issues including depression, anxiety, and PTSD have on headache presentation and persistence are also unknown. Lastly, the diagnosis of concussion may be associated with nocebo effects and negative expectancy biases as related to headache reporting and prognosis that we currently have no good methods of assessing.

The literature is highly variable in study methodologies, criteria for "concussion" diagnosis, and delineation of involvement of potential secondary gain incentives that may serve to perpetuate headache complaints. For example, the study by Lane et al. found a headache persistence rate at 24 months of 70% with 90% of patients meeting criteria for migraine or probable migraine.[14] Stacey, Lucas, and Dikmen et al. assessed PTHA (very few with mTBI) natural history over 5 years and used ICHD-2 criteria and found a high rate of frequent headache at 60 months (36%)

with migraine accounting for just under 60% of headache subtypes and with close to 30% of headaches being "unclassifiable".[15] Yet, other studies show significantly lower rates of headache complaint persistence and much lower rates for migraine-type headaches.[16–18] These disparities reinforce the calls for further research on PCH using randomized controlled study protocols and broadening the scope of the assessments.

It should also be noted that cervical whiplash injury has been found to be associated with various autonomic comorbidities that may mimic postconcussive symptomatology, which can further complicate headache classification. Given the complexities of these injuries, it would be naïve to assume that headache classification is as simple as finding one thing that is causing the headache. In most cases, there are multiple pain generators and/or perpetuators that require holistic assessment and treatment. The neck should be considered a primary target for examination in the context of PCH assessment and yet, has historically received little recognition or attention.

Another significant area of concern is the nomenclature inconsistencies across studies as related to use of brain, head, and neck injury labels and criteria for the same. There is often inadequate information regarding whether subjects truly met criteria to be diagnosed with concussion, as well as a lack of documentation on comorbid injuries to the cranium, cranial adnexal structures, and/or neck. Another common oversight is the focus on CBI as the most likely explanation for the PCH. Headache onset after concussion does not mean that the concussion itself was necessarily the cause of the headache. Clinicians should understand that aside from concussion, cranial impact injuries, cranial adnexal trauma, e.g., TMJ, and cervical acceleration deceleration (whiplash) injuries, among other conditions often cause PCH and/or contribute to it. There is also likely a reporting bias relative to preexisting headache disorders as well as genetic loading risk factors, both of which likely get underreported either due to recall bias or lack of knowledge.[19] Lastly, there has been inadequate study of the potential role that prescription drug–induced headaches, and more importantly, medication overuse headaches (MOHs) play in the PCH population.

PCH PATHOETIOLOGY

Anatomic sources of head pain that may be relevant in the assessment of a patient presenting with PCH include the dura, venous sinuses, and cranial cavities, including sinuses, eye sockets, etc. The skin, nerves, muscles, and periosteum of the cranium are all pain sensitive. Cervical/cranial joint capsules (including the temporomandibular joint), cervical facets/zygapophyseal joints, peripheral nerves (supraorbital, trochlear, greater occipital, lesser occipital, as well as third occipital nerves), and the cervical sympathetic plexus may all be pain generators that produce local or referred head pain.[20,21]

Given CBI injury mechanisms, it is possible to incur various types of trauma to the aforementioned structures including but not limited to impact injuries (both long and short impulse), stretch injuries of various tissues (both rotational and linear), penetrating injuries, shearing/tearing injuries, and compression, as well as herniation-related forces. One of the most common, yet often overlooked, sources of head pain following CBI is referred cervical pain, which is typically associated with acceleration-deceleration insults or whiplash-associated disorders. Postwhiplash sequelae may include referred myofascial pain emanating from any of the four layers of the posterior cervical, as well as anterolateral cervical musculature; traumatic neuralgias (as noted above), vertebral osseous somatic dysfunction, disc herniation and/or rupture, ligamentous injury and/or facet joint trauma (with potential for osteoarthropathy and/or traumatic injury to the medial branches of the dorsal ramus). Risk factors for persistent postwhiplash symptoms including headache should be identified and addressed as apropos.[22] These injuries can be seen in the absence of CBI or as a comorbid feature and should always be considered in the context of identifying the specific pain generators contributing to the PCH even in the absence of subjective complaints of cervicalgia.

The neurobiology of PCH following a single traumatic insult may involve cerebral/intracranial, cranial as well as cranial adnexal, and cervical structures involved with encoding and processing of craniocervical noxious stimuli. Pain receptors may be activated by tissue injury and neuroinflammation and mediated by bradykinin, serotonin, substance P, histamine, leukotrienes, cytokines, and prostaglandins.[23,24] Additionally, it seems clear that there are likely both peripheral and central mediators of PCH. Acutely, it is more likely that peripheral mechanisms account for pain mediation rather than central or supraspinal causes. Cranial mechanical hyperalgesia likely related to peripheral structure injury to blood vessels, nerve fibers, and osseous structures, as well as the inflammatory cascade, likely serve as sources of peripheral pain sensitization and PCH. The aforementioned

would suggest that earlier more aggressive treatments during the acute phase could prevent secondary chronification through central sensitization mechanisms. Some have speculated that central mechanisms are involved in persistent allodynia related to somatosensory cortex injury. Mechanisms of central sensitization are still being explored; however, it appears that abnormal neuronal excitability may lead to altered processing of sensory stimuli causing cortical spreading depression and trigeminal activation. There are numerous pathways in the neuromatrix that when damaged may lead to centrally mediated pain. Central sensitization contributes to both acute allodynia and headache persistence. Sensitization, whether peripheral and/or central, is not just relevant to post-traumatic migraine but may be seen in cervical whiplash injury, traumatic temporomandibular disorder, among other conditions. Repetitive concussions may promote trigeminal sensitivity and microglial proliferation, astrocytosis and neuropeptide release in the trigeminovascular system further exacerbating the underlying headache disorder.[25]

Nitric oxide synthase and calcitonin gene—related peptide have been implicated as at least potentially contributory to mediating allodynia in migraineurs following concussion, which has been linked with trigeminal neuroplastic changes.[26] Others have speculated that the pathophysiology of PTHA is shared with the pathophysiology of brain injury itself relative to inflammatory responses. These inflammatory responses could persist beyond timeframes for actual beneficial physiological effect leading to secondary injuries due to alterations in neuronal excitability, axonal integrity, central processing, as well as other changes.[27]

A key anatomic entity that must be appreciated in the pathobiology of PCH is the convergence of upper cervical and trigeminal nociceptors through the trigeminal nucleus caudalis (TNC). Nociceptive input from a variety of sources in the neck can activate the TNC via occipital and cervical afferents (whether from muscles or joints). This phenomenon may account for improvements seen in postconcussive migraine and tension headache when cervical nociceptive afferent inputs, if present, are appropriately identified and treated, leading to decreased or negated nociceptive afferent input into the TNC. Such treatment can also help modulate peripherally sensitized trigeminal branches such as the supraorbital nerves.[4]

Lastly, the disparate findings on the correlation between injury severity parameters and type, frequency, severity, and duration of PTHA seemingly begs the question of whether other non-CBI-related pain generators and/or mechanisms are involved in both the origin of PCH and its promulgation.

PCH CLINICAL PRESENTATIONS—AN OVERVIEW

Headache and neck pain are the most common physical complaints following CBI and are experienced early after injury in a very large percentage of patients and, in a smaller percentage, longer term (however, the latter literature is not as strong methodologically). The major types of headaches seen following trauma include musculoskeletal headache (including direct cranial trauma, cervicogenic headache, and TMJ disorders), neuromatous and neuralgic (nerve) headache, TTHA, migraine, as well as less common causes such as dysautonomic headaches, seizures, facial and/or skull fractures, cluster headaches, paroxysmal hemicrania, post-traumatic sinus infections, drug induced headaches, MOHs, and the surgical conditions previously mentioned. There remain controversies regarding the occurrence and causal relationship of trigeminal autonomic cephalgias such as cluster and paroxysmal hemicranias with concussion. The overlap in headache subtypes as related to cervicogenic, referred myofascial, migraine, and tension headache cannot be overemphasized in the context of both assessment and treatment implications.[28]

The majority of headaches following CBI are most likely benign and do not typically require surgical treatment; although, there are, on occasion, complications that occur after both CBI and more severe injury that may cause headache and require surgical intervention. Subdural and epidural hematomas, carotid cavernous fistulas, traumatic carotid or vertebral artery dissection, cavernous sinus thrombosis, as well as post-traumatic intracranial pressure (ICP) abnormalities (high vs. low ICP), among other conditions can all be responsible for headaches and bring with them a potential need for surgical intervention. Readers are referred to more comprehensive sources for details on the aforementioned less common etiologies of PTHA.[3]

PCH is best evaluated with time taken to acquire an adequate preinjury, injury, and postinjury history and in that context, a detailed history of the presenting headache symptoms. Equally important, but often ignored, a headache physical examination (including relevant neurological and musculoskeletal elements) should be performed. As clinically indicated, other diagnostic testing, including imaging, psychoemotional, and/or pain assessments, among other examinations, may be necessary/helpful. One of the most frequent

areas of concern is whether the patient with suspected CBI requires brain/head imaging. In general, such imaging is not necessary except in cases where there are worsening headaches, medically unresponsive headaches, and/or protracted headaches after injury. The main physical findings that justify considering neuroimaging in head trauma as well as CBI include eye swelling or pupil abnormalities, diplopia or vision loss, and/or focal neurological findings.[29]

Late-onset headaches (i.e., greater than 2 months post trauma) should cue the treating clinician to think of less common injury-related conditions such as a slowly expanding extra-axial collection as a cause for the headache disorder or just as likely, a noninjury-related cause such as a space-occupying lesion (e.g., brain tumor, colloid cyst, subsequent injury), among other conditions[3]; although, late-onset headaches apportionable to the initial injury can happen even years post insult.

THE PCH HISTORY

The examination should start with taking a thorough history from the patient as related to their PCH complaint. One of the most important pieces of information to acquire in this context is to determine if the patient had any type of headache complaints preemptory to the injury in question and if so, whether the headache presentation has changed. In that context, it is also important to determine if there are genetic loading risk variables for headache such a migraine in the patient's family.[30] Important information to garner in the context of the history taking is the timing of headache onset relative to the traumatic event. Clarifying the frequency and severity of pain is also of paramount importance and ideally should include use of standardized pain rating scales and/or headache questionnaires. A nice mnemonic to assist in taking a headache history is "COLDER"…character of pain, onset, location, duration, exacerbation, and relief. Understanding how the headache has evolved post injury is also important as is acquisition of a treatment history relative to what specific treatments were prescribed and what the patient's response was to the same. As it relates to prior drug treatment, it is important to note that the clinician should edify both the dose of the medicine as well as the duration that the medicine was given to assure that an adequate trial was provided. As far as nonpharmacological treatments are concerned, the clinician should establish that there were adequate interventions utilized to address the underlying pain generators as might be the case with osteopathic, chiropractic, physical therapy, and/or psychological management. The clinician should get as much information as possible about injury mechanisms to understand risk factors for particular PCH pain generators such as might be associated with brain injury itself, cranial impact injuries, and/or cervical whiplash. Lastly, clarification of the functional consequences of PCH is important and may include limitations in physical activity, including exercise and sexual activity, work or school pursuits, sleep quality, and mood alterations, among other factors to explore. Use of headache questionnaires to assess disability from same such as MIDAS or HIT-7, headache diaries, and tracking tools/applications for smartphones (e.g., Curelator, Headache Diary Lite Pro, iHeadache, Migraine Buddy, MigrainePal and My Migraine Triggers), although none have been validated for PCH, may further complement the information garnered during history taking.[4] Given the existing literature on the accuracy, or limits thereof, of histories provided by people with brain injury, it is important to interview corroboratory sources as well due to potential limitations in patient insight and memory.

THE PCH PHYSICAL EXAMINATION

The hands-on physical examination, which is often ignored in PTHA assessment, should take into consideration central and peripheral neurological clinical findings, as well as musculoskeletal clinical findings. The neurological evaluation should entail an elemental neurological examination of all 12 cranial nerves, funduscopic examination (to rule out papilledema), deep tendon reflexes including pathological reflex testing, sensory examination including visual field confrontational testing, motor examination and cerebellar assessment (which should also encompass measures of postural stability), assessment for meningismus, and mental status evaluation. Appropriate cognitive screening should be performed as clinically indicated. The peripheral neurological examination should include assessment for neuralgic and/or neuritic headache pain generators such as supraorbital neuralgia, temporoauricular neuralgia, and occipital neuralgia. The musculoskeletal examination should include palpatory assessment of the face, temporomandibular joints, head and craniocervical junction, cervicothoracic spine, and upper thorax at a minimum to assess for pathologic findings.

The musculoskeletal evaluation of the patient with PCH should be holistic in nature as the entire body should be screened, not just the cervical spine. Sitting and standing postures should be noted, preferably

without the patient's awareness, so that the clinician can obtain a realistic view of postural habits. Details such as forward head, increased kyphosis in the thoracic spine, and increased or flattening of the lordotic curvature in the lumbar and cervical spines are important to note as well as scapular positioning. A closer inspection for body asymmetries (e.g., head tilt, shoulder droop, rotated or tilted pelvis, and leg length discrepancy) are also important to note as these asymmetries may be contributing to ongoing cervical dysfunction and pain complaints. Jaw range of motion and tracking is an often overlooked but important assessment as TMJ issues commonly refer pain into the head and have been associated with both facial trauma and cervical whiplash injuries.[3,4] The temporomandibular joints should also be auscultated, as clinically indicated, for abnormal articular sounds.

Cervical spine range of motion can provide valuable insights into dysfunction (e.g., limited right rotation and side-bending can indicate a right cervical facet dysfunction and rotation less than 45° can indicate dysfunction at C2).[31] Auscultation for bruits should be done as appropriate over the carotids, closed eyes, temporal arteries, and mastoids for assessment of arteriovenous fistulas. Palpatory examination should include the face, head (including TMJ and masticatory muscles), shoulder girdles, and neck musculature. This must be done in a controlled, layer-by-layer fashion to truly localize pathology with an eye to identifying activated trigger points and referred pain patterns. Common special tests that should be included in the musculoskeletal examination are the cervical flexion rotation test, to assess for C2 dysfunction; Spurling's test, to assess for cervical facet dysfunction; alar ligament stress test, anterior shear test, Sharp Purser test, and the tectorial membrane test to assess for upper cervical instability. When assessing facets, the examination focus should be on the first three cervical levels as these levels can directly refer pain into the head through nociceptive inputs into the trigeminocervical nucleus; although there remains some debate about whether referral of pain can occur below the C3 level. A systematic review of physical examination tests for screening for cervicogenic headache found that the cervical flexion-rotation test exhibited the highest reliability and strongest diagnostic accuracy.[32] Referred cervical myofascial pain as a consequence of cervical whiplash is a particularly common cause of PCH. This fact should emphasize the importance of a good musculoskeletal examination in any patient presenting with PCH including, of course, the neck. One can often find activated trigger points in the suboccipital musculature, sternocleidomastoid, and/or upper trapezius muscles as common sources of referred pain into the head in such patients.

Neuralgic and neuritic pain generators associated with surgical trauma, direct scalp contusional injury, and craniocervical acceleration/deceleration forces are often overlooked. Clinicians should understand the anatomy of the peripheral nerves innervating the face and scalp including but not limited to the occipital nerves as well as their ability to be treated through interventional pain management techniques. The interconnectivity of upper cervical roots and brainstem trigeminal centers through the trigeminocervical complex is critical to keep in mind as previously noted and to understand in the context of PCH assessment when there is comorbid cervical whiplash injury.[33]

Migraine and tension headache have also been shown to not uncommonly be associated with abnormal musculoskeletal examinations of the head and neck. Given this fact, clinicians should always assess these structures even if the diagnosis of PCH falls in the migraine tension spectrum.[34] It is also important to note that patients may have had neck injuries and be unaware of that fact and even deny pain complaints referable to the same but still have pathological findings on neck examination. In patients with new-onset headache, examiners must also ascertain that they are not tender over the temporal arteries or demonstrating signs of nuchal rigidity, the latter which may be associated with a pathologic meningeal process such as meningitis.

PCH TREATMENT PRINCIPLES—AN OVERVIEW

Treatment should be instituted in a holistic fashion as early as possible with the goals of maximizing the benefit/risk ratio of any particular intervention, prescribing treatment that can be optimally complied with and educating the patient and family regarding the condition, its treatment, and prognosis in a coordinated attempt to minimize risk for longer term PCH complaints and disability as well as improve quality of life and pain adaptation.[35] Ideally, treatment should be interdisciplinary and multipronged as clinically warranted with the most essential team members being the physician, physical therapist, and psychologist.

The pharmacological management of PCH is replete with challenges due to the lack of an adequate body of evidence-based medicine examining the efficacy of pharmacotherapeutic agents in this population.[36] The general practice trend is to approach these headache

disorders as they would be treated in primary headache disorders.[37] There are no FDA-approved drug treatments specific to pediatric or adult PCH or for that matter PTHA more generally. General rules of pharmacological prescription in persons after concussion are to start low, go slow, minimize polypharmacy, choose agents that will likely be effective given the specific condition being treated (as such may be available), reassess need for medication over time, monitor use/abuse as clinically indicated, and attempt to choose medications that can be taken once to twice a day at most to optimize compliance.[38]

Treatment approaches should emphasize conservative measures first as possible as interventional treatments lack methodologically sound evidence.[39] Such interventions may include physical modalities, lifestyle changes, postural education, and behavioral therapy among other treatments.[40] Knowledge regarding psychological assessment and behavioral therapies in these types of pain patients is essential to holistic management as is clinical acumen on methods to ascertain pain reporting response biases and pain catastrophizing.[41–43] Interventional procedures such as facet, peripheral nerve, as well as sphenopalatine ganglion blocks should be considered as clinically appropriate.[44] With the advent of a number of different types of neuromodulation treatments, clinicians have more treatment choices particularly for post-traumatic migraine and certain neuralgias although these interventions have yet to be studied in this patient group.

Pain medications that are not specific for the particular headache subtype should usually be avoided, particularly such agents as opiates and barbiturates, which may cause a variety of long-term adverse effects including, but not limited to, MOH, adverse endocrine effects, drug tolerance over time, impairment issues that may affect safety for driving and/or equipment use, and addiction, among other risks.[4,37] There is seldom an indication for prescribing opiates in this group of patients, particularly for longer term use. Additionally, not all pain is opiate responsive, and clinicians should therefore use caution when prescribing such agents. Patients should only be placed on chronic opioid treatment if they demonstrate true failure to respond to a variety of other pharmacotherapeutic agents with less potential short- and long-term risks, have opiate responsive pain, and are not candidates for neuromodulation, interventional pain management procedures, and/or surgery. Pediatric patients, those with significant Axis II issues, or those with a history of chronic substance abuse should generally not be considered candidates for opioid therapy.

Once central sensitization is suspected to have taken place, the prognosis becomes more guarded for complete pain resolution; however, clinicians must be familiar with methodologies for modulating this type of complication in the context of PCH. A multipronged approach has been shown to have the best results by focusing on specific targets for desensitization including both bottom-up and top-down strategies such as oral medications, topical analgesic therapies, as well as metabolic and neurotrophic factors all with the goal of decreasing hyperexcitability in the central nervous system.[45]

PCH: COMMON PRESENTATIONS AND RECOMMENDED TREATMENT APPROACHES

The following will summarize the clinical presentation and treatment of the most common PCH variants. Please see Table 6.1 for an overview of headache subtypes, typical symptom presentation, examination findings, and treatment options.

Migraine

Clinical presentation

As per ICHD-3, migraine is defined based on clinical history. The patient must have at least five headache attacks that lasted for a duration of 4–72 h, with the headache having at least two of the following characteristics: unilateral (nonside alternating), location, pulsatile quality, pain intensity that is moderate or severe, and pain aggravated by activity or pain that limits activity.[9] It should be noted, however, that more than 50% of people who suffer from migraines report nonthrobbing pain at some time during the attack. During the headache there must be at least one of the following reported: nausea or emesis, and/or photophobia/phonophobia (also referred to his photosensitivity and sonosensitivity). There must not be any other explanation for the headache to classify it as migraine. Of note, migraine attacks commonly occur during waking hours but less commonly may awaken a person from sleep.

Treatment

There are three basic approaches to pharmacotherapeutic management of post-traumatic migraine. These include prophylaxis, abortive therapy, and symptomatic therapy. Successful treatment of migraines may be paradoxically achieved by reducing medication use. In medication overuse, headache worsening typically occurs because of the overuse of abortive pain medications.

TABLE 6.1
Postconcussive Headache Subtypes, Symptoms, Examination, and Treatment.

Headache Pain Generator	Presenting Symptoms	Physical Examination Caveats	Treatment Recommendations
Migraine	Unilateral location, pulsatile/throbbing pain character, pain intensity that is moderate to severe and aggravated by activity. Classically associated with nausea or emesis and or photo- and/or phonosensitivity. Typically occurs during waking hours.	Potential for associated musculoskeletal examination abnormalities of the head and neck which may compound and/or perpetuate trigeminovascular instability.	Consideration of prescription pharmacotherapeutic interventions involving symptomatic, abortive, and/or prophylactic medications including medications such as botulinum toxin. Can also consider use of naturopathic agents and over-the-counter medications including agents with caffeine. Interventional procedures including sphenopalatine ganglion blocks, occipital nerve blocks, and neuromodulation should all be considered as clinically appropriate/lifestyle changes and behavioral interventions including cognitive behavioral therapy (CBT) with stress management training, biofeedback, and relaxation training among other interventions should also be utilized as apropos. Physical therapy referral may be appropriate to treat concurrent abnormal musculoskeletal findings.
Tension-type headache (TTHA)	Pain is usually bilateral and occipitofrontal in location with a "tight band" feeling that may have a throbbing quality and is usually gradual in onset with duration being highly variable with a more constant quality but generally with mild to moderate pain intensity. Typically not aggravated by physical activity. May be associated with photo- and/or phonosensitivity. Typically made worse by lack of sleep and stress.	Potential for associated musculoskeletal examination abnormalities of the head and neck.	Consideration of prescription pharmacotherapeutic interventions for abortive as well as prophylactic therapy. Avoid use of muscle relaxants. Consider nonpharmacologic treatments including CBT, biofeedback, relaxation therapy, massage, stress inoculation treatment, and exercise among other interventions. Physical therapy referral as appropriate to treat concurrent abnormal musculoskeletal findings.
Cervicogenic	Headaches tend to be unilateral and nonalternating as far as laterality although they can on occasion be bilateral. Pain described typically is nonthrobbing, nonlancinating with moderate to severe pain intensity with episodes of varying duration. May see a variety of comorbid autonomic symptoms associated with these types of headaches (i.e., blurry vision, dizziness).	Assess for upper cervical vertebral somatic dysfunction, cervical instability (i.e., ligamentous), facet-mediated pain, neuralgic pain generators such as occipital neuralgia and/or activated myofascial trigger points in the neck and or upper shoulder girdles that may refer into the head.	Physical modalities including manual therapies and physical therapy interventions with consideration of focused exercise therapies and in some cases interventional pain management procedures. Rarely surgery.

Neuralgia/Neuritic	Variable presentations based upon nature of nerve dysfunction from dysesthetic more diffuse pain symptoms to very focal point tenderness to shooting or lancinating-type pain with referral. Location will be variable depending upon the nerve affected. Duration can be episodic or constant.	Assess for dysesthetic areas on the scalp, positive Tinel's signs over major nerves (i.e., supraorbital, greater occipital) or their branches, nerve tenderness, as well as referred pain.	Treatment consideration should include enteral, topical, and injected medication therapies. Additionally, cryoneurolysis, radiofrequency ablation, and neuroablation can be considered, although newer techniques involving neuromodulation may provide more conservative yet effective management.
Medication overuse headache	Typically presents as a holocephalic, throbbing headache which can be persistent. They tend to occur daily to nearly every day and may awaken a person in the early morning. Tend to improve with pain relief medication and will return as medication wears off. May be associated with nausea, restlessness, behavioral changes, and cognitive difficulties.	Likely to find parallel physical examination abnormalities as seen with migraineurs or patients with TTHA.	Discontinuation of offending medication with replacement with alternate treatment for the suspected background headache disorder which should be initiated either during or immediately following withdrawal. Nonpharmacologic therapies such as biofeedback and targeted physical therapy may also be indicated. Support groups and behavioral techniques have also been found to enhance success of treatment in some patients.

Migraine prophylactic therapy should be aimed at improving quality of life, decreasing abortive drug therapy usage, as well as complications, and reducing attack frequency, severity, and/or duration. There are generally considered to be three broad classes of medications currently endorsed for migraine prophylaxis. These include antiepileptic drugs (AEDs), antihypertensives, and antidepressants.[46] Botulinum toxin A has also been FDA approved for use in chronic migraine management and is typically reserved for patients who have failed three preventive medications and are experiencing chronic migraine per aforementioned criteria.

The most recent guidelines for the prevention of episodic migraines by the American Headache Society/American Academy of Neurology, the Canadian Headache Society, and the European Federation of Neurological Societies were published in 2012. The medications with the highest level of evidence for migraine prevention were sodium valproate, butterbur, topiramate, propranolol, timolol, and metoprolol.[47] Although the aforementioned guidelines included only studies published up to 2009, more recent studies have confirmed most of the findings of previous systematic reviews.

Several "migraine-specific" prophylactic drugs have recently been developed, including CGRP receptor antagonists (abortive use) and monoclonal antibodies targeting CGRP (prophylactic use). These medications target known migraine pathophysiological mechanisms. Several oral CGRP receptor antagonists are now on the market and have shown promising efficacy in treating migraine with superiority to placebo and comparability to triptans. Some trials have been discontinued because of the concerns of hepatotoxicity after taking the drug for multiple consecutive days. Four monoclonal antibodies targeting CGRP or the CGRP receptor have been tested in humans for the prevention of migraine: galcanezumab, eptinezumab, erenumab, and fremanezumab. Another major breakthrough in the treatment of migraine has been the development of 5-hydroxytryptamine 1F (5-HT1F) receptor agonists, such as lasmiditan, which are similar to triptans but without their vascular side effects. This drug class binds more specifically to the serotonin 1F receptor than do triptans, which are less specific and bind to other vasoconstricting subtypes of serotonin receptors. Still in phase III trials, this drug class may be valuable for an aging population that is no longer eligible for triptan therapy because of cardiovascular and cerebrovascular risk factors.[46]

Naturopathic agents such as feverfew and butterbar can be considered as prophylactic antimigraine alternatives with the latter agent being available commercially as Petadolex, which is a patented, standardized CO_2 rhizome root extraction. Although butterbar has been used for many years, it has recently been shown to have potential for serious hepatotoxicity. Other agents including magnesium, riboflavin, and coenzyme Q10 have been touted as also being beneficial with empirical data being best for magnesium supplementation. Further studies are required to address persistent questions about efficacy, optimal dosing ranges, and parameters for choosing one drug over another in a particular patient.

Over-the-counter medications such as Advil Migraine, Excedrin Migraine, and Motrin Migraine, which are all FDA approved, should be considered first-line migraine abortives and are oftentimes quite effective if not overused. Prescription abortive medications include ergot derivatives, dihydroergotamine derivatives, and triptans, as well as combination medications such as Treximet. Dihydroergotamine-containing compounds such as DHE-45 can be given intravenously, typically with concurrent administration of metoclopramide (Reglan), for expedient abortive management of migraine. Dihydroergotamine is also available as a nasal spray, marketed as Migranal. Ergotamine formulations include oral medications such as Cafergot, as well as Ergotamine tartrate with caffeine, in addition to Ergomar sublingual tablets and Migergot suppositories. Parenteral atypical antipsychotics may also be used for abortive purposes.

Nonsteroidal anti-inflammatory drugs (NSAIDs) can be used for both prophylaxis and abortive therapy, the latter including menstrual migraine. Midrin, which is an acetaminophen-containing compound (acetaminophen-isometheptene-dichloralphenazone) has also been used for migraine headache management but typically works better for TTHAs.

Symptomatic medications are typically used for decreasing symptoms associated with nausea and emesis that may accompany migraine headaches. Traditionally, the drugs that are used for symptomatic management include prochlorperazine (Compazine) and promethazine (Phenergan), which may be given orally or via other routes such as rectal suppository. Metoclopramide (Reglan) and domperidone (Motilium) are also used as adjutants either orally or intravenously for symptomatic control and will also facilitate intestinal drug absorption.

Newer treatment interventions can also be considered in the context of migraine management, including sphenopalatine ganglion and/or greater occipital nerve blocks as well as neuromodulation modalities such as

transcutaneous magnetic stimulation (TMS) with the eNeura SpringTMS, transcutaneous direct current stimulation (tDCS) of the supraorbital nerves with Cefaly or vagal nerve stimulation with gammaCore, among other developing methodologies.[48] These modalities may be helpful in both prophylaxis and abortive treatment of migraine.

Biobehavioral interventions have also been found to be effective for migraine management and may include cognitive behavioral therapy (CBT) with stress management training, biofeedback, and root relaxation training among the most common. Ideally biobehavioral interventions should be used in conjunction with pharmacotherapeutic approaches.

Tension Headaches

Clinical presentation

By ICHD criteria, tension headaches are divided into either episodic (frequent or infrequent) or chronic and further categorized by whether or not they are associated with pericranial muscle abnormalities. These types of headaches comprise the most common primary headache disorder.[49] Episodic tension headache is usually associated with heightened motions and/or stress and tends to be of moderate intensity with a self-limiting course. Episodic tension headache tends to be responsive to over-the-counter medications although prescription medications are sometimes necessary. Chronic tension–type headache (CTTH) on the other hand recurs daily and has been shown to be correlated with abnormalities in the pericranial and cervical muscle examinations. Genetic factors seem to be more important in the pathogenesis of CTTH.[50] This type of headache is usually bilateral and occipitofrontal in location. Pain onset can have a throbbing quality and is usually more gradual than the onset seen in migraine. Duration tends to be more highly variable than other headache disorders such as migraine with a more constant quality but lower degree of severity. The headache tends to be felt as a tightening around the head ("tight hat syndrome") (although location can vary) and is typically described as nonpulsatile. TTHA is not classically aggravated by physical activity.[50] They are typically made worse by lack of sleep and stress. These types of headaches are typically not associated with nausea or vomiting although photosensitivity and sonosensitivity can be seen but must be differentiated from postconcussive impairments of the same nature as well as migrainous phenomena.[51] There is very little understood about TTHA pathoetiology in primary headache or secondary headaches such as PCH but it is likely multifactorial.

Treatment

Acute pharmacotherapy of TTHA should include NSAIDs (including acetylsalicylic acid) sometimes in conjunction with caffeine, sedatives, and/or tranquilizers. There is no scientific evidence to support the use of muscle relaxants. Prophylactic pharmacotherapy for TTHA is more diversified and without any FDA-approved drugs currently endorsed. Tricyclic antidepressants, tizanidine, botulinum toxin, and venlafaxine (the latter an SNRI) have all shown at least some benefit, with TCAs having the best evidence basis.[52] Nonpharmacological treatments for tension headache include CBT, stress inoculation treatment, biofeedback, relaxation therapy, massage, trigger point therapy, exercise, and acupuncture.[52–54] Modalities may also play a role in modulating TTHA including hot or cold packs, ultrasound, electrical stimulation, postural education, trigger point injections, occipital nerve blocks, manual medicine treatment, and stretching among other interventions.[55] Proper diet, regular exercise, and restorative sleep are crucial in any headache patient including those with tension headache.

Neuritic and Neuralgic Headache

Clinical presentation

These types of headaches may present in variable patterns depending upon the nature of the peripheral nerve injury. Small nerve fibers in the scalp may be injured in the context of penetrating injuries, surgical intervention such as craniotomies, as well as by direct impact injury resulting in scalp dysesthesia, significant tender points, and/or positive Tinel's sign over the affected region. When upper cervical distal nerve roots are involved such as greater, lesser, or third occipital nerve, the pain may be localized and noted on compression over the nerve or when more severe may radiate into the sensory distribution of the nerve. In more severe cases, there will be ipsilateral radiation into the frontotemporal scalp and occasionally retro-orbitally…both of which will be described as painful by the patient. Peripheral nerve injuries to larger nerves, whether in the face or scalp, such as the supratrochlear, supraorbital, infraorbital (face), and/or auriculotemporal nerve may present with localized as well as referred pain and may be due to peripheral nerve injury as well as central sensitization. Clinicians must be familiar with the general anatomic location of all the aforementioned nerves as well as their associated sensory distributions.

Treatment

Drug management of post-traumatic neuritic and neuralgic pain tends to emphasize focal injection

therapies and topical agents. Secondary interventions may include enteral medications such as NSAIDs, tricyclic antidepressants, SNRIs such as duloxetine or anticonvulsants such as carbamazepine, gabapentin, and pregabalin. For post-traumatic neuralgias involving larger nerves of the face, scalp, and/or craniocervical junction, local injection therapy with corticosteroids and local anesthetic remains the mainstay of treatment. Serial injections may be necessary to abate or modulate the pain generator. Greater and lesser occipital neuralgia treatment approaches may include segmental blocks at C2 and C3, cryoneurolysis, radiofrequency (RF) ablation, and neuroablation; although, newer noninvasive strategies such as neuromodulation are now showing some promise.[56] Additionally, RF lesioning as well as surgical interventions do not necessarily guarantee resolution as there is a relatively high recurrence rate. Interestingly, blocks of afferent cervical nociceptive inputs have been shown to modulate trigeminal nerve pain as well as neurogenic inflammation[28]

Diffuse neuritic pain associated with scalp injuries such as postcraniotomy pain or local blunt trauma should be addressed with compounded topical formulations. Such topical agents, typically applied as an ointment or gel, generally need to be applied 3 to 4 times per day to be optimally effective. Topical agents for neuropathic pain may include TCAs, local anesthetics, NSAIDs, AEDS such as gabapentin, clonidine, and/or ketamine hydrochloride, among other agents. There are often challenges getting insurance coverage for compounded topical pain medications. For information on local and/or regional compounding pharmacies see https://www.achc.org/pcab-accredited-providers.html.

Myofascial Pain Related Headaches

Clinical presentation

Myofascial pain typically presents with pain in the neck and upper back/shoulders with potential to refer into the base of the skull in the head in general. Myofascial pain is associated with development of painful active trigger points (as opposed to latent trigger points which do not replicate the patient's pain complaint pattern). A myofascial trigger point (sometimes just called a trigger point) is a tight knot located within a taut muscular band. The knot or nodule can be distinctly felt underneath the skin and is tender when pressed. In addition, when pressure is applied to the knot, the taut muscular band which holds the knot contracts. This creates a twitching of the muscle that can be felt or visually observed, the so-called "twitch response."[57,58]

When a trigger point is located in the neck, shoulder, and/or head muscles, it can cause referred or spreading pain that can be perceived as headache. The headache may be localized or more diffuse depending on the number and location of the aforementioned trigger points. The patient may be misdiagnosed as having tension headache with bilateral referred cervical myofascial pain unless an appropriate musculoskeletal examination is performed and even then, the conditions can be comorbid and in such cases, both need to be addressed from a treatment standpoint. It should be further noted that such myofascial trigger points can be seen in association with headache due to whiplash, migraine, and tension headache.[57,58]

Treatment

Treatment should focus on an understanding of myofascial pain disorder triggers, as well as perpetuating factos. Myofascial therapies are the mainstay of such treatment and should include specific stretching exercises, deep soft tissue work, and myofascial techniques such as trigger point acupressure, and dry needling, among other techniques as clinically indicated.[57,58] Postural reeducation can be a very important component of holistic MPD treatment since incorrect posture (i.e., forward head position or upper crossed syndrome) may perpetuate referred headache pain. Manual techniques to restore proper mobility and alignment, muscle strengthening, and individualized exercise programs should all be considered in the context of such treatment.[58]

Pharmacologic interventions for myofascial pain, although traditionally not first-line treatment, include NSAIDs, tricyclic antidepressants, and possibly, muscle relaxants. Muscle relaxants have no proven efficacy, although some resemble TCAs in their structure and clinical effect and may contribute to treatment efficacy. Antispasticity drugs are seldom used for myofascial pain, although tizanidine may have some theoretical benefit over other traditional antispasticity agents due to its antinociceptive properties garnered through its effect on blocking of substance P. For chronic intractable myofascial pain, some have advocated for use of botulinum toxins although there are inconclusive data to support this use in the head and neck regions.

Cervicogenic Headache

Clinical presentation

The clinical presentation may be confounding due to the overlap of symptoms with other possible primary as well as secondary headache disorders, including tension headache, migraine, and greater occipital neuralgia headache. In part, this confusion occurs because even the aforementioned headaches tend to be associated

with complaints of neck pain and/or with pathological cervical physical examination findings in the absence of cervicalgia. Headaches tend to be unilateral and not alternating side to side although they can be bilateral.[59–61] Headaches are typically nonthrobbing, nonlancinating, and associated with moderate to severe pain with episodes of varying duration. To further complicate things, cervicogenic headaches have also been noted to be associated with sono- and photosensitivity, nausea, dizziness, unilateral blurred vision, among other symptoms.[62] It is critical to differentiate cervicogenic headaches from other headache etiologies[63] and recognize that afferent nociceptive input into the trigeminocervical complex can aggravate or perpetuate migraine as well as tension headache.

Treatment

Conservative treatment options should be tried first, including physical therapy and manual therapies (manipulation with or without mobilization), whether chiropractic, osteopathic, or craniosacral.[54,64,65] Low load endurance craniocervical and cervicoscapular exercises have also been shown to have a role in amelioration.[54] Exercise including postural and strengthening can play a key role in ameliorating/modulating such headaches.[66] In more intractable cases, interventional pain management procedures such as anesthetic blocks of the upper cervical joints can be helpful in both assessment and treatment.[67]

Medication Overuse Headache

Clinical presentation

MOH is defined as a headache occurring at least 15 days per month in patients with preexisting headache disorders and with concurrent use of either simple analgesic agents such as ibuprofen or naproxen at least 15 days per month or other analgesic agents such as triptans or ergotamines 10 days per month for at least 3 months.[68] MOH may be seen from excessive use of a variety of analgesic and/or abortive headache agents, including ergotamines, opiates, caffeine, triptans, and/or barbiturates.[69] Overuse of these medications may lead to development of increased headaches and even CDH. Patients may become dependent on these symptomatic headache medications. The headache tends to present as holocephalic and throbbing.[70] Headache medication overuse may also make headaches refractory to prophylactic headache medication and the affected person more sensitive to headache triggers. Unfortunately, most patients are unaware of MOH, so providers should be diligent in evaluating the frequency of medication use and educating patients as well as their family about the risks of MOH if medications are not taken as prescribed.[68]

Treatment

Although withdrawal and reduction of abortive medications is considered to be the treatment of choice, there is no standard practice recommended as related to withdrawal at this time. Drug withdrawal, particularly when abrupt, normally results in worsening of headache. Alternatively, if MOH is a concern, the offending medication should be slowly weaned with concurrent alternative preventative headache management options prescribed with evidence based on randomized studies best for topiramate and onabotulinumtoxin A.[50,68] What role some of the newer generation migraine medications may have is yet to be determined. Detoxification can be done as an outpatient, in a day hospital, or in an inpatient setting depending on the severity of the MOH and the comorbidities of the specific case.

PCH NATURAL HISTORY, PROGNOSIS, AND OUTCOME

There are inadequate evidence-based studies to stipulate the natural history, prognostic factors, and long-term outcomes of PCH, in part, because PCH is not one single pathophysiological disorder but rather a symptom descriptor that may involve multiple pain generators/causes including psychogenic ones. The relative lack of prospective, controlled, and blinded studies that adequately consider variables associated with headache cause, treatment, and secondary complications only further challenges our ability to understand its natural history and provide opinions regarding prognosis. There are also multiple methodological challenges in studying an impairment that is predominantly based on subjective patient report including issues of misattribution bias (on the part of the patient as well as the assessing clinician), patient recall bias, nocebo effects of the diagnosis, cultural influences on pain perception and reporting, and potential response bias relative to symptom amplification as well as minimization (depending on incentives…which may be subconscious, conscious, or some combination of both) regarding pain reporting and/or associated pain–related disability, among other issues.

Any study of chronic PCH must also address the inherent comorbidities of the psychological and medical effects of chronic pain (and the associated stress) on not only the patient's reporting of their pain but also on a myriad of other aspects of function, including cognition, behavior, and sleep. Studies to date have not

integrated data from focused headache physical assessments that take into consideration neurological and musculoskeletal findings nor have they included measures of response bias, pain validity reporting, or analysis of the potential influence of secondary gain factors in headache reporting. Additionally, studies have not linked specific examination findings with current headache classification systems (the latter of which have been criticized relative to their lack of applicability and relevance to this particular population). Importantly, historical studies have not attempted to assess the accuracy of the PCH-related diagnoses nor their historical response to treatment. Without knowing what the specific PCH pain generators are in a population and/or how they were treated, we cannot make determinations of the true incidence of "chronic" PCH. Any such conclusions would furthermore be based on the assumption or confirmation that the headache condition(s) was/were appropriately diagnosed and treated. That being said, there is no literature to the authors knowledge regarding the natural history of untreated PCH.

Based on the available studies, headache tends to improve in the months following trauma, whether to the brain, head, or neck. How much improvement is seen will be dependent on injury severity, preinjury physical and psychoemotional status, presence of complicating factors such as nocebo effects, secondary gain incentives, resilience, among other factors, as well as appropriate diagnosis of the underlying pain generators and subsequent optimal treatment for same. Appropriate and timely treatment shortens the period of impairment, associated disability, lost work hours, as well as pain and suffering. There is very limited evidence regarding the impact of ongoing litigation on the persistence of headache complaints but that which exists suggests that patients still continue to report significant symptoms even after litigation has ended.[71] Further research to confirm prior findings is strongly recommended.

A small number of patients will develop intractable and sometimes severe PCH; however, this group of patients has been poorly studied and the influence of other factors including inappropriate headache categorization and treatment, MOH, secondary gain incentives, cultural factors, preinjury genetic loading risk variables, as well as psychogenic factors in such patients remains unclear. The first prospective controlled study examining PCH persistence found that approximately 15% of patients continued to complain of headache at 3 months post injury[72]; however, accuracy of diagnosis and appropriateness of treatment were not confirmed. Kjeldgaard et al. studied "chronic PTHA" in a group with "mild head injury" and found a high correlation with unemployment and interestingly TTHA was the most common headache subtype noted with over 30% of patients having a mixed headache picture.[73] Also of note in the Kjeldgaard study was the fact that over 50% of the chronic PCH group was involved in litigation. Dumke found that headache severity was strongly correlated with a poorer prognosis for return to work; yet, based on the study it is unclear whether patients were appropriately diagnosed and treated.[74]

When properly diagnosed and treated, most patients, in our experience, are able to achieve substantive headache improvement, if not cured, particularly when PCH is addressed earlier rather than later. Good outcomes depend to a great extent on patient education and avoidance of nocebo, iatrogenic, and lexigenic effects of the injury. Additionally, work disability due to PCH, in and of itself, is very uncommon in the hands of a sophisticated practitioner with a good treatment team, unless there are significant comorbidities including mental health issues and/or secondary gain incentives.

PCH prognosis must be based on an exact understanding of headache etiology (based on history and focused examination and questionably headache classification systems), overlay as relevant of psychogenic factors (including patient preinjury characterological issues) and secondary gain incentives, response to appropriate historical treatment, and consideration of whether the correct diagnosis and treatment for the pain generator was made from the initial assessment.[4,75]

PATIENT AND FAMILY EDUCATION

Patients, as well as significant others/caretakers, need to be educated so that they understand the relevant diagnoses, treatment plan, and prognosis. Education should also be provided regarding the importance of compliance with recommended treatments and in particular medication use as related to avoidance of MOH, potential drug side-effects, and drug interactions among other issues. Clinicians should remain accessible for questions or concerns.[4,5]

CONCLUSIONS

PCH is ultimately a symptom and not a diagnosis. This complex disorder has multiple potential causes and as a result, has multiple treatments to address the headache disorder that is associated with the underlying pain generator(s). Assessing and treating PCH is a process that

requires adequate time commitment and knowledge by the treating clinician…some will consider this "a pain" and if that is the case, then those clinicians should defer treatment to others who make it their business to assess and treat these types of patients and conditions. Pejorative and potentially self-prophesizing labels such as "chronic PCH" are often a misnomer due to the fact that the actual pain generators were never diagnosed correctly in the first place and should be avoided. There is in fact hope for those with PCH regardless of how long they have suffered from pain. The challenge is finding clinicians and treatment teams who understand the diversity of this class of disorders and have experience in holistic assessment and treatment of patients who have had concussions, cranial trauma, and/or whiplash injuries.

RESOURCES OF INTEREST FOR PATIENTS AND PRACTITIONERS

ACHC: Pharmacy Compounding Accreditation Board (PCAB): https://www.achc.org/pcab-accredited-providers.html.

American Council for Headache Education: www.achenet.org.

American Headache Society: https://americanheadachesociety.org.

American Migraine Foundation: https://americanmigrainefoundation.org.

BIAA (PTHA webinar): https://shop.biausa.org/product/BOB092216CD/20160922-post-traumatic-headache-recorded-webinar.

Brainline. Post-traumatic headache: https://www.brainline.org/article/post-traumatic-headache-after-tbi.

Chiropractic Canada. Clinical practice guideline for the management of headache disorders in adults: http://ccpor.ca/wp-content/uploads/CPG-for-the-Management-of-Headache-Disorders-in-Adults-2012.pdf.

National Headache Foundation: www.headaches.org.

Ontario Neurotrauma Foundation. Guideline for concussion/mild traumatic brain injury & persistent symptoms. Third edition (for adults over 18 years of age). Post-traumatic headache. HTTPS://BRAININJURYGUIDELINES.ORG/CONCUSSION/INDEX.PHP?ID=135&TX_ONFADULTS_ADULTDOCUMENTS%5BTHEME%5D=6&TX_ONFADULTS_ADULTDOCUMENTS%5BACTION%5D=SHOW&TX_ONFADULTS_ADULTDOCUMENTS%5BCONTROLLER%5D=THEME&CHASH=036827A60A01AE94FC9382FE19B89508.

Practical Pain Management, Migraine tracking apps for smartphones: https://www.practicalpainmanagement.com/patient/resources/pain-self-management/9-apps-tracking-your-migraine-days.

The American Council for Headache Education (provides a listing of on-line and local support groups): www.achenet.org.

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