Trigeminal Neuralgia

(Review article for physicians)

By Alexander Mauskop, MD and Boris Leybel, MD

Introduction

Chronic headaches afflict over 40 million Americans. Athletes are a special category of patients who can experience headaches that are specific to their sports. A survey of 178 medical students and 190 physical education students (Williams 1) showed that 35% of both groups suffered from sport- and exercise-related headaches. Among men, more physical education students suffered from headaches, probably due to high frequency of trauma-related headaches in contact sports. The widely accepted International Headache Society's (IHS) classification (1) often does not distinguish between various subtypes of sports-related headaches. This usually indicates lack of sufficient data about a particular headache type that prevents proper classification. Because IHS diagnostic categories are widely used we will use both the commonly-used term as well as IHS classification (Table 1).

The clue to correct diagnosis and effective treatment of an athlete with headaches lies in a detailed history. It is necessary to establish circumstances surrounding the onset of pain, possible precipitating factors, character of the pain (i.e. throbbing, pressure-like, sharp, stabbing, exploding, etc.), location of the pain, preceding and accompanying symptoms, such as nausea, vomiting, diarrhea, vertigo, nasal congestion, lacrimation, weakness, sensory symptoms, dysarthria, confusion, scintilating scotomata and other visual disturbances. Other factors that must be considered include history of non-sports related headaches, family history of headaches, prior and current illnesses, excessive caffeine intake, psycho-social factors and substance abuse. In addition to a thorough neurological evaluation a majority of athletes with headaches need to have an MRI scan of the brain. Rooke (43) reported that in 103 patients with exertional headaches 10 had an organic lesion that was responsible for the headaches (Table 2). Assessment of possible traumatic brain injury in a patient with post-traumatic headache is also best done by an MRI scan.

Headache syndromes

BENIGN EXERTIONAL HEADACHE

Exertional headache

Exertional and effort-induced headaches are grouped together in the IHS classification and they do have many features in common; however, there are
some clear differences. Exertional headache appears to be a response to lifting, pushing, pulling or a similar activity as well as sexual activity, coughing, sneezing or straining at stool. The Valsalva maneuver is a common denominator among all of these activities.

Paulson (27) reported five cases of weightlifter's headache where the headache was mostly occipital with neck and back pain. We also find that in a large number of our patients a strain of neck muscles and possibly ligaments is responsible for this type of headaches. This putative etiology is supported by findings of muscle spasm and tenderness in the occipital and paraspinal cervical regions. These patients do well with trigger point injections or occipital nerve blocks when pain is prolonged. They often can return to the headache-inducing activity after a period of isometric neck strengthening exercises. A typical occipital neuralgia in a football player which was relieved by a nerve block has been reported (42). Absence of a typical neuralgia does not preclude the use of occipital nerve blocks in patients with a headache and occipital tenderness. A positive clinical response to such a block does not necessarily establish the etiology of the headache as being due to an occipital nerve compression.

Exertional headaches are seen in a variety of sports. They are not necessarily in response to maximal physical effort; very often a headache occurs at submaximal activity and even at the beginning of exercise. In such cases it is important to pay attention to predisposing metabolic factors.

**Effort-induced headache**

This type of headaches is described by several authors as a response to aerobic activities such as running or swimming (Morris, 21, 28). The majority of these patients have a headache of migraine type (three or more of the following features: throbbing, unilateral, moderate or severe pain that is worsened by physical activity, accompanied by nausea, photophobia and phonophobia). A possible mechanism of effort-induced headaches is hyperventilation leading to vasoconstriction that triggers a reactive vasodilation with a resultant migraine headache. In addition to the classical contributing factors such as tyramine-rich foods, red wine, chocolate and others, athletes may have additional predisposing circumstances, such as dehydration, hypoglycemia and overheating. We have found that low serum ionized magnesium levels may be a contributing factor in some patients with migraines (10).

North(26) described a postmenopausal woman with post-exercise headache who had a headache only when she had a transdermal estrogen patch and never while on oral estrogen. The author explains this by increased absorption of estrogen from the skin during exercise and a vascular headache precipitated by estrogen "bolus".
Exertional headache can often be prevented by taking aspirin or another NSAID an hour prior to the activity. Prolonged exertional headache was reported to respond to indomethacin in 13 out of 15 patients (Diamond).

A warm-up regimen may also prevent an effort-induced migraine (Lambert). Since the headache usually begins after the exercise a prolonged cool down period may also help.

**POST-TRAUMATIC HEADACHES**

Collision and contact sports by their nature can cause head injury and post-traumatic headaches. Frequently it is a mild trauma that produces long-lasting headaches. It seems that there is an inverse correlation between the severity of the head trauma and the likelihood of a post-traumatic headaches (Kelly). In cases of subdural, epidural, intraventricular, subarachnoid or parenchymal hemorrhages severe headaches occur along with other neurological symptoms such as changes in mental status, neck stiffness, nausea, vomiting, and focal neurological findings. Such cases usually do not present diagnostic difficulties. The incidence of headaches following a concussion in football players in one study was found to be as high as 96% (Maddocks). However, prolonged post-concussion headaches appear to be less common in athletes than in the general population. In non-athletes, the unexpected nature of the head injury, emotional stress surrounding the injury and to a lesser extent litigation may all predispose to prolonged post-traumatic headaches.

Another type of trauma-related headache is an acute migraine induced by a head injury (Mathews, Bennett, Kalanak, Kinsella). In some patients a single trauma causes recurrent migraines (Russell, Ashworth). It appears that migraines without aura induced by an initial trauma are different from non-traumatic migraines because of a different risk for migraines among first degree relatives (Russell). Cluster-like headaches have been reported to occur after a head injury (Reik, Bracker).

Beta blockers are effective for migraine and cluster headaches. However, because they may impair an athlete's performance by limiting the heart rate increase necessary for strenuous activities, calcium channel blockers such as verapamil are preferred for migraine and cluster headache prophylaxis. Abortive treatment of a migraine or cluster attack begins with NSAIDs. NSAIDs are often ineffective and combination drugs that contain butalbital and caffeine (Fiorinal, Fioricet, Esgic) or ergotamine and caffeine (Wigraine tablets, Cafergot supp.) can be used. The amount of these drugs should be limited to no more than 15-20 tablets a month. Excessive caffeine intake, including dietary caffeine and caffeine in OTC drugs (Anacin, Excedrin) can cause refractory rebound headaches.

In some athletes post-traumatic headache fits the criteria for a tension-type
headache (non-pulsatile, not severe, non-pulsatile, bilateral and not associated with nausea, photophobia or phonophobia). Management of prolonged headaches of this type is similar to that in non-traumatic cases. Tricyclic antidepressants are effective, although they can produce somnolence and dry mouth which can interfere with athletic performance. Newer SSRI (selective serotonin re-uptake inhibitors) type antidepressants can be effective with fewer side effects, although controlled studies in headache patients have not been done.

Neck injury frequently accompanies head trauma and the neck itself can be a source of severe headaches. Cervical roots C2-C3 can produce a shooting, neuralgic type headache in the occipital area similar to a headache produced by the greater occipital nerve entrapment.

Headache due to traumatic carotid artery dissection is described mostly in contact sports (Li), but one case in a scuba diver has been reported (Nelson). This is an uncommon condition that can easily go undiagnosed and therefore constant awareness of this possibility is necessary for early detection. By the time neurological signs appear an infarction may have occurred. Doppler ultrasound studies, MRA or a conventional angiography can establish the diagnosis. Prompt treatment with anticoagulation and sometimes surgery can prevent infarction and permanent neurological damage.

In the evaluation of an athlete with a posttraumatic headache it is critical not to miss any underlying organic pathology. Neurological examinations should include a mental status examination, appropriate neuropsychological tests, and an MRI scan of the head and if indicated the neck area. An athlete should be allowed to return to competitive sports not earlier than one week after the resolution of the headache and any accompanying neurological abnormalities. The one week period is intended to avoid the most catastrophic and often unpredictable second impact syndrome. This syndrome occurs when an additional blow to already existing minor trauma may precipitate brain swelling and a catastrophic increase in intracranial pressure with brainstem herniation and death within minutes.

**SPORT DEVICE TRIGGERED HEADACHE**

Headache triggered by constricting equipment was described in swimmers due to pressure of their goggles and in hockey players due to their helmets. This type of headache is usually dull, located in temporal and occipital areas, but sometimes it can have features of a migraine headache with all the typical manifestations including an aura. Loosening goggles can also stop a headache. Muscle contraction headache has been described triggered by the gripping of a mouthpiece, causing strain of facial and cervical muscles.

**ENVIRONMENTALLY TRIGGERED HEADACHE**
Jokl described a migraine-like headache in runners at the 1968 Olympics in Mexico City (7000 feet above sea level), and absence of headache in these well trained athletes at the sea level. Altitude-triggered headache typically begins at 3000 meters above sea level. The mildest mountain sickness syndrome also includes nausea, vomiting, and fatigue. The headache is throbbing in character, increases with any physical activity, occurs after 6-96 hours of exposure and can resolve with acclimatization but may require treatment. Above 5000 meters, patients can develop acute cerebral edema and may need prompt medical intervention with immediate, rapid descent, oxygen, and steroids. A single report suggests the efficacy of sumatriptan (Bartsch), while a randomized, double-blind trial showed that ibuprofen, but not sumatriptan is effective for high-altitude headache (Burtscher). Acetazolamide can be used prophylactically 3 days prior to ascent, and at least 1 week of acclimatization at 3000-5000 meters to prevent serious complications. Low oxygen tension usually triggers migraines in these athletes. High-altitude headache is seen in mountain climbers and skiers. Dysbaric air embolism and decompression sickness can also present with a headache. Dysbaric air embolism occurs usually within minutes, not only with a headache, but with focal neurological signs as well. Neurological injury from decompression often involves the spinal cord. Early diagnosis is crucial because treatment of dysbaric air embolism and decompression sickness is not the same. Divers also can develop vascular headaches due to accumulation of CO2 and headaches induced by a cold stimulus.

Bibliography


