

# Post-traumatic Headaches: Subtypes and Behavioral Treatments

*Thomas Bennett*

---

Chronic, recurrent headache commonly follows head injury, and interestingly, it is seen more often in individuals who have experienced minor head trauma than in those more seriously injured. I will describe subtypes and behavioral treatments of the post-traumatic headache. One must realize, however, that headache is only one of a number of symptoms that commonly follow head injury. While it may be the symptom that results in a patient seeking medical treatment following brain or head injury, it may on the other hand be only the "tip of the iceberg".

Many patients complaining only of post-traumatic headache are found, under close neuropsychological evaluation, to be concurrently suffering from verbal and communicative disorders, deficits in information processing and reaction time, memory difficulties, problems with perception, and impaired concept formation and general reasoning ability (Alves, Colohan, O'Leary, Rimel, & Jane, 1986). Post-traumatic headaches should also be considered within the context of post-traumatic symptoms in general which are often collectively called the "post concussional syndrome". At this point it may be helpful to briefly consider the "post concussional syndrome" in general because many of the statements that I could make about posttraumatic headaches apply to other post-traumatic symptoms as well.

## **Post-Traumatic Symptoms**

The basis of post-traumatic symptoms in individuals who have sustained minor head injury (loss of consciousness of 20 minutes or less and post-traumatic amnesia of 24 hours or less) has been debated for over 200 years. The common use of

the term "post concussional syndrome" to describe the symptoms of these patients has a psychological flavor to it. It was (and still is in less informed circles) typical for physicians and psychologists to ascribe symptoms for which there was no obvious organic basis (no hematoma, no penetration of the dura, no contusion of the brain) to neurotic processes or malingering.

The modern view of post-traumatic symptoms is that they do indeed have a biological or organic basis. It is now accepted that even mild concussion usually entails some structural damage to the brain (Jennett & Teasdale, 1981). This is not to say that emotional factors do not play an important role in post-traumatic symptoms because it is generally believed that emotional factors can both exacerbate and prolong post-traumatic symptoms that are the result of biological factors. This conclusion underscores the need to employ both biological and psychological interventions in treating patients with post-traumatic symptoms.

With respect to malingering, let me point out that its incidence has been vastly overestimated. Symptoms are deliberately exaggerated in a smaller proportion of patients than was previously thought to be the case (Boll, 1982; Rimel, Giordani, Barth, Boll, & Jane, 1981). Neither insurance claims nor pending litigation is a significant factor influencing return to work or social recovery (Irving, 1972; Oddy, Humphrey & Uttley, 1978).

A number of symptoms have been identified to follow minor head injury. However, it is not accurate to describe these symptoms as a syndrome as was common practice when using the term "post concussional syndrome". While a given patient may exhibit one, two, three, or less commonly, more of these symptoms, it is rare to see a patient experience

all of them (Alves et al., 1986). Post-traumatic symptoms commonly reported after minor head injury include headaches; dizziness; impaired concentration; memory problems; sensory problems including diminished hearing, olfaction, and taste; diplopia; tinnitus; hypersensitivity to noise; insomnia; fatigue; irritability; anxiety and depression.

The mechanisms maintaining these symptoms are not always obvious, but some meaningful statements can be made. The problems with dizziness and auditory difficulties are undoubtedly related to concussion of the balance and central auditory processing functions of the inner ear. Decreased olfactory sensitivity is related to strain injury of the axons of the olfactory tract as they enter the brain. I suspect that reports of decreased taste sensitivity do not reflect a decrement in that specific modality, but rather they reflect a loss of gustatory sensitivity secondary to diminished olfactory sensitivity. Hypersensitivity to noise and hyperirritability probably at least partially reflect general diminished inhibitory or gating (filtering out) processes in the brain. Insomnia is a common sequel after head injury; in general, initiation, maintenance, and cycling mechanisms do not operate as efficiently as they should after head injury, presumably because of neural disruption resulting from rotational forces exerted on the brain stem at the time of injury.

Loss of concentration and memory problems may be reflective of diminished information processing ability in general. Gronwall and Wrightson (1974) tested information processing ability using the Paced Auditory Serial Addition Test (PASAT). They found that head injured patients who performed abnormally on this test at 35 days post-injury still complained of post-traumatic symptoms; the disappearance of these symptoms correlated nicely with restoration of normal performance on the PASAT.

Irritability, anxiety, and depression can all be contributed to by organic factors, but typically, they are more dependent on psychological processes. Coping with head injury produces feelings of loss of self-control and feelings of helplessness and hopelessness. Finding that you can no longer perform efficiently at home, school, or on the job is

very stressful, and it can aggravate other post-traumatic symptoms as well.

The nature of and basis for post-traumatic headache is more difficult to explain. There appear to be many possible causes for persisting headache after head injury. Some of these include musculoskeletal trauma to the neck, head, and jaw regions, pain in scalp scars, neuralgia of occipital or supraorbital nerves, precipitation of migraine in predisposed individuals, and occasional serious intracranial complications. Post-traumatic headaches are a significant health problem when one realizes that close to half of all patients discharged from the hospital after minor head injury report persistent headache (e.g., Alves et al., 1986).

### **Subtypes of Post-Traumatic Headaches**

The vast majority of patients who experience persistent post-traumatic headaches have no intracranial abnormalities to explain their headache pain (Dalessio, 1980). Electroencephalographic (EEG) abnormalities are not correlated with occurrence of post-traumatic headache, but the presence of scalp lacerations is positively correlated (Scherokman & Massey, 1983). At least four or five types of post-traumatic headaches have been categorized including, 1) steady pressure with cap-like distribution, 2) circumscribed superficial tenderness around the impact site, 3) episodic aching or throbbing pain which is typically unilateral, and 4) episodic, unilateral frontotemporal pain with ipsilateral mydriasis and hyperhidrosis. Recently, I have seen more and more cases where patients report unilateral or bilateral pain in the temple region or just superior to it and I will comment on this as a fifth type of post-traumatic head pain.

#### **Type 1: Steady Pressure With Cap-like Distribution**

Type 1 headaches are the most common and persistent variety of headaches that occur after head injury. Patients will often have this type of headache concurrently with one or more of the other types. As indicated, these headaches are described as a steady pressure, often with a cap-like distribution, but more commonly in a circumscribed area elsewhere than the site of the injury. There is

usually a deep tenderness present in the neck or shoulder region, and headache can often be reproduced by manual pressure on these tender areas. The intensity of the associated pain is described as being from "mild to very severe". The attacks of pain can recur for many years. Occurring intermittently, the attacks can vary from several hours to as long as ten days duration (Dalessio, 1980).

Type 1 headaches are usually made worse by effort stress, coughing, stooping or turning the head. As a result the patient may be functionally incapacitated for engaging in physical activity related to work or recreation. During periods of severe headache of this sort, patients may experience spinning sensations, dizziness, and photophobia (Dalessio, 1980). Finally, these headaches are associated with persistent and sustained muscle contraction in the head, neck, upper back, and shoulders. This can be easily demonstrated through electromyographic (EMG) studies that show excessive levels of muscle contraction in these patients. Medically, these headaches are treated with muscle relaxants, analgesics, amitriptyline, heat, and massage. They often prove to be resistant to treatments.

### **Type 2: Circumscribed Tenderness Around Impact Site**

Most patients with Type 2 headaches suffer from Type 1 headaches as well. Type 2 patients have a circumscribed, relatively superficial tenderness of the scalp at the site of the original injury which is often, but not always, associated with a visible or palpable scar (Scherokman & Massey, 1983). In most patients who experience this type of pain, there is spontaneous aching pain at the original site of impact; in some cases, headache pain only occurs when some pressure, such as a hat or a brush, is applied to the site (Dalessio, 1980). Typically, this type of posttraumatic headache pain is described as being "moderate", and usually it resolves within a year after the original injury. It appears to be related to contusion and injury to the scalp vasculature.

### **Type 3: Episodic Aching or Throbbing Pain, Usually Unilateral**

Type 3 post-traumatic headaches are described as aching, often throbbing pain, usually unilateral in

onset. They occur in attacks and are most commonly reported to occur in the temporal regions. They are also sometimes frontal, occipital, or postauricular. The attacks may be of short duration, and they may represent an intensification of symptoms for patients who also experience a background of Type 1 headache. Reported pain associated with Type 3 headaches varies from "mild to severe". The intensity of the pain is increased by effort, coughing, bending, or lying down.

Post-traumatic headaches of the Type 3 variety, while usually unilateral in onset often become generalized. They often begin in the morning or are present upon awakening, and they may continue all day. Nausea, vomiting, and anorexia may accompany them. Dalessio (1980) reports that these headaches are not relieved by massage or head but ice bags, cold compresses, and codeine will provide relief. Ergotamine tartrate eliminated this type of pain but did not diminish Type I components resulting from excessive muscle contraction. Type 3 headaches are vascular in nature, and they are more commonly seen in patients with a migraine history, even if migraines have been rare or infrequent in the past. This type of headache disorder is related to recurrent painful distention of cranial arteries. For many patients, it represents the precipitation of a serious vascular headache (migraine) disorder in a person already at risk.

### **Type 4: Episodic, Unilateral Frontotemporal Pain With Ipsilateral Mydriasis and Hyperhidrosis**

Vijayan (1977) has described a type of headache syndrome associated with anterior neck injuries secondary to cervical whiplash (also see Khurana & Nirankari, 1986). In these patients, unilateral, frontotemporal vasodilating headaches are experienced episodically. What makes them unique is that they are accompanied by ipsilateral mydriasis (excessive dilation of the pupil) and facial hyperhidrosis (excessive sweating). When the pain subsides, the patient is left with ipsilateral ptosis (drooping of the upper eyelid) and miosis (excessive constriction of the pupil). In Vijayan's series, patients experienced between two and 12 of these headaches per month.

Type 4 headaches are related to damage to the third-order sympathetic neuron in the neck; they

reflect localized sympathetic nervous system dysfunction. Patients with these headaches were helped when treated with the beta adrenergic blocking agent, propranolol, in doses of 20-60 mg per day (Vijayan, 1977); they did not respond to ergotamine.

### **Type 5: Pain in Temple or Superior Temple Region**

A fifth type of headache syndrome, which also typically is accompanied by Type 1 headaches, is an intermittent recurrent relatively steady pain in the region of the temples or just above. The pain may be unilateral or bilateral, and when it is bilateral, it may be described as a band extending from temple to temple. It is also typically accompanied by jaw popping during chewing, leaving no doubt that it is related to temporomandibular joint (TMJ) dysfunction or injury. Many people in our society clench their teeth at night (bruxism) in response to stress. An automobile accident that results in a person striking the windshield or dashboard can easily exacerbate ongoing temporomandibular joint degeneration or significantly displace or injure a healthy joint. High resolution computerized tomography has been shown to be vastly superior to conventional radiography in detecting TMJ degeneration in post-traumatic headache patients (Tilds, Miller, & Guidice, 1986). The increasing availability of magnetic resonance imaging will enhance the detection of these difficulties further. Treatment of TMJ syndrome, whether posttraumatic or not, may require a multidisciplinary approach including orthodontic and surgical treatments, splint therapy, physical therapy, and biofeedback.

In summary, at least five distinct headache syndromes can be described in post-traumatic headache victims. With the exception of Type 4, which reflects sympathetic nerve damage, these headaches reflect musculoskeletal and vascular dysfunction that is essentially the same as that seen in chronic headache patients who have not experienced trauma. As with typical chronic headache patients, the head pain experienced by head trauma victims comes in many combinations. As indicated, patients with Types 2, 3, and 5 headaches will often complain of Type 1 headaches as well, and some patients will exhibit symptoms consistent with several subtypes concurrently. A

multimodality approach using behavioral treatment interventions will be described next. The use of behavioral treatments for patients with post-traumatic headaches has been discussed in case studies by several investigators (e.g., Daly & Wulff, 1987; Duckro, Tait, Margolis, & Silvermintz, 1985; Muse, 1986).

Behavioral Treatments of Post-Traumatic Headaches Packard (1979) asked directly, "What does the headache patient want?" and then outlined the stated needs of patients along with physician estimates of those needs. Generally speaking, physicians ranked medication higher than did the patients. The patients gave their highest rankings to the need for information about the causes of their headaches and to the need for relief from pain. The patients' responses thus indicated that education must be an integral part of any headache treatment program. Behavioral treatment of post-traumatic headaches must thus use a multifaceted approach and should include education about headaches and medications for them, physiologic therapies such as physical therapy, therapeutic massage and biofeedback, and cognitive-behavioral therapies aimed at helping the patient acquire pain management skills.

### **Education**

Let us forever bury the idea that post-traumatic headaches, or headaches in general, are simply psychiatric problems. Clearly, stress can produce or exacerbate headache syndromes, but headaches are best understood by considering both psychological and physiological factors. Their interaction can be understood by an explained to the patient with post-traumatic headaches.

### *The Vicious Cycle*

A major failure of the traditional organic-psychological dichotomy is to downplay the interaction among emotional factors, the autonomic nervous system, and elevated levels of muscle tension on head pain levels. Muscle tension levels are commonly elevated in the head, neck, shoulders, and upper back regions after trauma, and this is often not evaluated in diagnostic studies. The muscle contraction can cause additional pain. Similarly, it is clearly the case that emotional arousal is frequently present in these patients.

Emotional arousal increases discharge in the sympathetic nervous system. Prolonged excessive discharge in the sympathetic system will in turn exacerbate pain levels by increasing perception of the intensity of stimuli related to pain.

Unfortunately, these physical and emotional factors can create a "vicious cycle" in which pain becomes a stressor, eliciting emotional (anger, frustration) and physical (increased muscle tension) factors which then produce more pain (Zimmerman, 1981). The longer this sort of cycle persists, the more difficult it is to break. Diagramming the cycle, explaining it to the patient and then giving homework exercises to disrupt it have been very effective in helping the post-traumatic headache patient break this cycle. This type of exercise is most beneficial if the patient learns techniques to intervene at multiple points in the cycle.

When patients have learned about the physiological factors contributing to their headaches and how emotional factors can interact with physical factors to set up a "vicious cycle", they are ready to accept the fact that their pain has both biological and psychological aspects. They can then be assured that their presence in the psychologist's office is not because their physician believes their headaches are "just in their minds".

### *Medication*

I find that few patients understand the role of medication in their treatment. It is clear that medications (i.e., analgesics, those which reduce muscle spasms and overall level of muscle contraction, those that reduce emotional arousal, and antidepressants) can be useful in selected patients. The psychiatric medications may or may not produce pain relief because of their psychiatric effects. Benzodiazepines may help because of their muscle relaxant properties; antidepressants may help because they promote serotonin activity in the brain which in turn decreases pain sensations. Patients need to be informed of why they are taking certain medications. One must be careful in using antidepressants, particularly amitryptaline, in treating individuals with post-traumatic headaches. The anticholinergic side effects of these drugs can exacerbate post-traumatic symptoms related to head injury including dizziness, diminished concentration, and memory problems.

Patients also typically have a misconception about how effective their medications should be. They often believe that analgesics should eliminate the pain completely. I find that this is an example of a "fix me doc syndrome" that impedes the progress of many patients. Instead of holding the belief that "improvement is outside of their own control" (Barnat, 1986), post-traumatic headache patients need to learn that a certain amount of discomfort will often persist; it is something that must be lived with. If a medication provides no relief, then it is a failure, but medications that provide only partial relief have not necessarily failed.

### **Biofeedback**

Physiological therapies, including physical therapy, therapeutic massage, and biofeedback, can be effective treatments for post-traumatic headaches. I will only discuss the use of biofeedback therapy in this paper, but let me emphasize that we regularly refer post-traumatic headache patients to certified massage therapists and/or physical therapists as part of their treatment program. These procedures are all complimentary to one another.

Biofeedback is a procedure by which a biological signal (for example, skin temperature, muscle tension, heart rate, brain waves) is converted to an easily detected signal, such as a light or a tone, and "fed back" to a patient so that she or he can exert conscious control over that function. Thus, a person might learn to detect and subsequently lower excessive levels of muscle tension in his or her forehead, neck, shoulders, and jaw, or a person might learn to increase the recorded temperature of her or his finger tips. (Cold hands signal excessive sympathetic nervous system discharge and resulting vasoconstriction; they are a sign of stress and a common characteristic of niigraineurs.)

Biofeedback is not a cure-all, and this treatment is most effective when incorporated into a comprehensive treatment program. Nevertheless, biofeedback treatment has been used successfully for two decades to treat a variety of psychophysiological disorders including muscle contraction and vascular headaches, hypertension, gastrointestinal disorders, pain syndromes, anxiety syndromes, abnormal heartbeats, sleep disorders,

and many neurological syndromes including movement disorders and epilepsy. It is also employed in neuromuscular rehabilitation following peripheral or central nervous system damage (Bennet, 1987). I have found it to be of significant benefit for the posttraumatic headache patient, particularly when combined with stress management training.

As I indicated in an earlier article (Bennett, 1988), biofeedback training can have both obvious and subtle benefits for these patients. One obvious benefit would be to teach the patient with excessive levels of muscle tension to relax chronically tensed muscles and to be more sensitive to muscle tension levels. This is accomplished through electromyographic (EMG or muscle tension) training. Temperature training in biofeedback can be used to increase vasodilation (decrease sympathetic activity) and thereby decrease the likelihood of vascular headaches. We typically use a combination of these procedures, as both contribute to stress management and relaxation training. All posttraumatic headache patients can benefit from such training. EMG biofeedback is particularly applicable to patients experiencing Type 1 and Type 5 headaches, and temperature training is especially relevant for patients with Type 3 headaches.

A more subtle benefit of biofeedback is to teach the patient that he or she can have some control over an aspect of their life (for example, "I can relax my muscles and decrease the severity of my headaches"). The head injured person often has a recent history of failure and frustration, and feelings of "loss of control" or "helplessness" may predominate. By learning to have more control over something as basic as level of muscle tension or skin temperature, the patient can learn that "what I do can really makes a difference". This helps the person to overcome the "fix me doc" syndrome and places him or her in more control of their future in general. Thus, the feelings of failure, loss of control, and helplessness can be attenuated.

### **Cognitive-Behavioral Therapies**

The usefulness of cognitive-behavioral therapies in pain management and headache treatment programs is well established. A particularly effective use of these principles in the treatment of

chronic headache patients has been described by Bakal (1982), whose general program is quite helpful for patients with post-traumatic headaches. I recommend it highly. Two important features of this program are attention diversion and thought management. These methods assist the patient in decreasing the intensity of the pain and in minimizing negative thoughts and dysphoric affect that increase headache severity.

In introducing the use of attention diversion and thought management, it is important to explain to the patient that headache pain consists of both sensory and reactive components. The sensory component consists of sensations of pain that are largely determined by changes in the muscles and veins of the head. The reactive (or cognitive) component consists of the thoughts and feelings that accompany headache episodes. There are, in turn, two important aspects of the reactive component. The first is the amount of attention that is directed toward the headache. The second is our interpretation of thoughts regarding, and feelings about, the pain experience. Negative or catastrophic cognitions exacerbate pain by increasing activation of the sympathetic nervous system.

The patient being taught attention diversion learns that we normally only focus on one thing at a time, and we are free to attend to whatever we want. Thus, we can influence what we are attending to by shifting our attention from one aspect of our environment to another (internal or external). Finally, it is difficult if not impossible, to stop focusing on one's pain unless one shifts attention to something else.

With this general framework in mind, the patient is provided with a number of attention diversion strategies to learn. They are initially practiced during headache-free periods and later applied to times of significant headache discomfort. Turk (1978) provides a variety of attention-diversion techniques, including imagery production, strategies that are relatively easy to master and quite effective in interrupting and/or decreasing the intensity of a headache attack.

Thought management deals with helping the patient control negative and/or catastrophic headache-related thoughts and feelings. By now, the patient is quite aware of the role that distressing thoughts play in their headache disorder. He or she

is encouraged to understand that distressing cognitions not only increase the pain experienced, but also they interfere with the ability to cope effectively with the headache pain. These thought patterns can be identified and modified. The patient learns that the process of "negative talking", can be reversed, and training is given in how to accomplish this.

First the patient is told to be alert to those times when he or she is experiencing distressing feelings and thoughts. Second, the patient learns to use these distressing thoughts as a signal to start making positive self-statements. Third, the patient learns to actively substitute the distressing cognitions with positive, coping-oriented statements (Bakal, 1982). In order to demonstrate this process, the patient is provided with a list of positive statements (Turk, 1979) to be used during different stages of the headache episode. He or she is encouraged to develop his or her own statements as well. Again, this intervention is over-practiced during times when headaches are not present before being put into practice.

### Conclusions

Post-traumatic headache is the most frequent complaint made by victims of minor head injury and is thus of major importance in head trauma rehabilitation. Post-traumatic headaches can be severe and persistent. As a result they can produce a major disruption in the patient's life. Their economic impact is significant in that they can prevent return to work in individuals who are otherwise recovered.

Post-traumatic headaches reflect an interaction among organic and emotional factors. For this reason, they are best treated via a multimodality approach that considers all of these factors. For many individuals, participation in such a program results in a complete remission of their symptoms. For others, the end of the program represents an improvement in their symptoms and the beginning of a long-term process of self-managing their headache pain. Most patients arrive at the point where their headaches no longer interfere with their lives. This represents a significant improvement over their prior incapacitation dependency on a variety of medications. In head injury recovery, this

is one sign that the victim can once more be in control of her or his future.

### REFERENCES

- Alves, E.M., Colohan, A.R.T., O'Leary, T.J., Rimel, R.W. & Jane, J.A. (1986). Understanding post-traumatic symptoms after minor head injury. *Journal of Head Injury Rehabilitation, 1*, 1-12.
- Bakal, D.A. (1982). The psychobiology of chronic headache. New York: Springer Publishing Company.
- Barnat, M.R. (1986). Post-traumatic headache patients II: Special problems, perceptions, and service needs. *Headache, 26*, 332-338.
- Bennett, T.L. (1988). Neuropsychological rehabilitation in the private practice setting. *Cognitive Rehabilitation, 6*(1), 1215.
- Bennett, T.L. (1987). Neuropsychological aspects of complex partial seizures: Diagnostic and treatment issues. *The International Journal of Clinical Neuropsychology, 9*, 37-45.
- Boll, T.J. (1982). Behavioral sequelae of head injury. In P.R. Cooper (Ed.) *Head Injury*. Baltimore: Williams and Wilkins.
- Dalessio, D.J. (1980). Post-traumatic headache. In D.J. Dalessio (Ed.) *Wolff's headache and other head pain*, 4th ed. New York: Oxford.
- Daly, E. & Wulff, J. (1987). Treatment of post-traumatic headache. *British Journal of Medical Psychology, 60*, 85-88. University Press, 3324-3381.
- Duckro, P.N., Tait, R., Margolis, R.B. & Silvermintz, S. (1985). Behavioral treatment of headache following occupational trauma. *Headache, 25*, 328-331.
- Gronwall, D. & Wrightson, P. (1974). Delayed recovery of intellectual function after minor head injury. *Lancet, 2*, 605-609.

- Irving, J.G. (1972). Impact of insurance coverage on convalescence and rehabilitation of head injured patients. *Connecticut Medicine*, 36, 385-391.
- Jennett, B. & Teasdale, G. (1981). *Management of head injuries*. Philadelphia, D.A. Davis.
- Khurana, R.K. & Nirankari, V.S. (1986). Bilateral sympathetic dysfunction in post-traumatic headaches. *Headache*, 26, 117-121.
- Muse, M. (1986). Stress-related, post-traumatic chronic pain syndrome: Behavioral treatment approach. *Pain*, 25, 389-394.
- Oddy, M., Humphrey, M. & Uttley, D. (1978). Subjective impairment and social recovery after closed head injury. *Journal of Neurology, Neurosurgery, and Psychiatry*, 41, 611-616.
- Packard, R. (1979). What does the headache patient want? *Headache*, 19, 370-374.
- Rimel, R.W., Giordani, B., Barth, J.T., Boll, T.J. & Jane, J.A. (1981). Disability caused by minor head injury. *Neurosurgery*, 9.
- Scherokman, B. & Massey, W. (1983). Post-traumatic headache. *Neurologic Clinics*, (2), 457-463.
- Tilds, B.N., Miller, P.R. & Guidice, M.A. (1986). The diagnostic value of high resolution computerized tomography in post traumatic head pain patients. *Headache*, 26, 117-121.
- Turk, D.C. (1978). Cognitive behavioral techniques in the management of pain. In J.P. Foreyt & D.P. Rathjen (Eds.) *Cognitive behavior therapy: Research and application*. New York: Plenum Publishing Company.
- Vijayan, N. (1977). A new post-traumatic headache syndrome: Clinical and therapeutic observations. *Headache*, 17, 19-22.
- Zimmerman, M. (1981). Physiological mechanisms of pain and pain therapy. *Triangle*, 20, 7-17.