Quality of Life With Posttraumatic and Naturally Occurring Headaches and Implications for Psychological Treatment

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Head Injury and Post-Concussional Symptoms

The terms ‘head injury’ and ‘brain injury’ are used in the literature almost synonymously. The author uses the term ‘head injury’ for the purpose of this paper as it is commonly used in the literature and because injuries to the head include brain injuries.

Head injuries due to accidents are a major health problem. The annual incidence of head injuries is said to be around 150 per 100,000 in the general population (British Society of Rehabilitation Medicine, 1998; Evans, 1992; Masson, Maurette, Salmi et al., 1996; McClelland, 1996). Santalucia and Feldmann (2000) reported the incidence to be between 175 to 367 per 100,000 population. Motor traffic accidents are the most frequent causes for head injury (42%). Further causes are falls (23%), assaults (14%) and sports injuries (6%) (Packard, 1999).

Head injury often results in ‘a persisting constellation of subjective disabilities and complaints that have been termed the post-concussional syndrome’ (PCS) (Youngjohn, 1997). The aetiology of PCS has long been a controversy. On the one hand, symptoms are attributed to neurological dysfunctions and structural impairments (Radanov, 2001; Watson, Fenton, McClelland, Lumsden, Headley, Rutherford, 1995). On the other hand, it is understood that psychological factors play a larger role and have some interaction with physiological factors (Lishman, 1998 & 1988; Smith-Seemiller, Fow, Kant, Franzen, 2003). Between 51% and 86% of mild head injury patients suffer PCS (Powell, Collin, Sutton, 1996). The core symptoms are somatic (headache, vertigo, insomnia, visual disturbances, sensitivity to light and noise), affective (anxiety, irritability, depression) and cognitive (memory, attention, speed of information processing) (Smith-Seemiller et al., 2003). PCS persists for longer than three months in more than 60% of the cases, which means that patients experience long term disabilities (Masson et al., 1996; Powell et al., 1996; Rimel, Giordiani, Bacth et al., 1981).

Characteristics of Posttraumatic Headaches

Posttraumatic headache is best understood within the context of the PCS. Its characterization is distinct from concussion. However, it contributes to the complexity of the PCS. Posttraumatic headache has, like the PCS, a multifaceted aetiology caused by organic impairment and influenced by psychosocial attributes (Martelli, Grayson, Zasler, 1999). Headache appears to be the most common symptom after mild head injury (Goldstein, 1991; Packard, 1999; Radanov, Di Stefano, Augustiny, 2001). This suggests that the rehabilitation of posttraumatic headache can facilitate the recovery from PCS. The incidence of posttraumatic headache is as high as 90% so it is nearly a universal symptom after mild head injury (Saper, 2000). The incidence of posttraumatic headache lasting longer than 6 months is as high as 44%. Cases lasting around 4 years are estimated to be about 20% (Martelli et al., 1999). It appears that the milder the injury the more frequently posttraumatic headache is noted as a symptom (Alves, Colohan, O’Leary et al., 1986; Bennett, 1988; Yagamuchi, 1992). Also Couch and Bearss (2001) showed an inverse relation between the extent of a head injury and the occurrence of chronic daily headaches. Eighty percent of patients with minimal head injury suffered chronic daily headache, whereas only 27% of patients with moderate/severe head injury had chronic daily headache.

Posttraumatic headache is classified by the International Headache Society (1988) into acute and chronic posttraumatic headache. The criteria for acute posttraumatic headache require that the headache begins 14 days after the trauma and disappears within eight weeks after the trauma. The criteria for chronic posttraumatic headache require the headache to last longer than eight weeks following the trauma. Additionally, to be diagnosed as posttraumatic headache, it has to differ from preexisting headache. The criteria of the International Headache Society allow coding for more than one type of headache in a patient.

The diagnosis and treatment of the posttraumatic headache can be difficult as there may be subjective symptoms with minimal evidence of organic abnormality as well as genetic, biological and psychological predisposing factors (Packard, 1999; Packard & Ham, 1994). Recent developments in technology might provide means of assessing mild head injury (such as PET, SPECT, MIRJ, evoked potential studies), thus neurophysiological abnormalities can be detected. The biochemical mechanisms of mild head injury and migraine appear to have similarities, which points to a neurochemical/organic underpinning of the condition (Packard, Ham, 1997). Selective vulnerability to posttraumatic headache is described by Gennarelli (1993). A genetic predisposition may not only pose a risk, but may worsen the outcome. Women are reported to suffer posttraumatic headache more frequently that men (Jensen ,Nielsen, 1990) and age has a negative effect on recovery (Bohnen, Twijnstra, Jolles, 1992).

The most improvement in posttraumatic headache is expected during the first six months following trauma. Beyond that time, there is a greater tendency for the symptoms to become chronic. Packard and Ham (1994) report a persistence of posttraumatic

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headache for more than three years in 15 - 30% of cases.

Types of Posttraumatic Headaches

Individuals with posttraumatic headache may develop one or several types of headache (Haas, 1996). The most common types - tension headache, migraine, cluster headache and mixed posttraumatic headache - are similar to their non-traumatic counterparts.

Tension type posttraumatic headache:

This is a common headache type following head injury. Tension headache may be chronic or episodic. Combinations of tension type and vascular headache are possible. Radanov et al., (2001) argue that pre-traumatic headache provides a risk factor for posttraumatic headache on the basis of pre-existing central sensitization, triggering muscle contraction at the time of injury, leading to posttraumatic tension type headache.

Posttraumatic migraine

The clinical features of posttraumatic migraine are almost identical to those of non-traumatic, with or without aura. They have the same characteristics as the naturally occurring migraine attacks with throbbing, lateralized headache accompanied by nausea, vomiting, photophobia and response to classic migraine medication (Goldstein, 1991). It is postulated that a genetic predisposition to migraine is triggered following a head trauma (Weiss, Stern, Goldberg, 1991). Consequently, posttraumatic migraine would be normal migraine occurring after trauma (Packard, Ham, 1994). Pre-existing migraine can be exacerbated following head trauma.

There seem to be neurochemical mechanisms in migraine which overlap with changes that occur in mild head injuries. These similarities may play an aetiological role in some headache problems that occur after trauma (Packard, Ham, 1997).

Cluster-like posttraumatic headache

This is a more unusual headache syndrome (Reik, 1987; Packard & Ham, 1996; Turkewitz, Wirth, Dawson, Casaly, 1992). The onset of the headache is usually very fast with a relatively short duration. Cluster-like posttraumatic headaches may present without the period of remission as in the episodic variety (Duckro, Greenberg, Schultz, Burton, Tait, Deshields, Richardson, 1992).

Other types of posttraumatic headache

The literature (Goldstein, 1991; Packard, 1999) describes further types of headache following trauma of the head and neck. These include temporomandibular joint syndrome, pericarotid syndrome, dysautonomic headache, whiplash injury, muscle contraction headache, occipital neuralgia, cervicogenetic headache and analgesic rebound headache. These types of headache are rare and may not be the sole cause of posttraumatic headache, but may contribute to the syndrome.

Posttraumatic headache may also occur when the soft tissues of the head are injured or when there is scar formation. The site of the injury is often sensitive to finger pressure. Patients may have different types of headaches at different times or a variety of symptoms together that are characteristic of more than one type of headache (Speed, 1986).

Haas (1996) argued that posttraumatic and naturally occurring headache were very similar. He found tension type headache and migraine without aura to be the most frequent types of posttraumatic headache. They could be placed in the naturally occurring headache categories of either chronic tension type headache or migraine without aura. Other studies confirm this similarity (De Benedittis, De Santis, 1983; Duckro et al., 1992; Duckro, Chibnall, Greenberg, 1995; Weiss et al., 1991). Another study reports similarities of posttraumatic headache with other headache types, e.g., migraine with aura, cluster headache, episodic tension-type headache (Benoliel, Eliav, Elishoov, Sharav, 1994). Dawn (2003) in his comparative study found that chronic posttraumatic headache was characterized by higher headache frequency and disability than non-traumatic headache.

Pathophysiology and Neurochemical Mechanisms of Posttraumatic Headaches

The pathophysiology of headache following trauma to the head and neck is explained by damage to: bones, blood vessels; the upper cervical spine; neurological pathways in the spinal cord and brain stem and nerve fibres. Further explanation includes fiber degeneration, soft brain tissue injuries and changes in the neurochemical metabolism. Cerebral blood flow changes were reported months, even years, after the injury, which may be associated with chronic posttraumatic symptoms (Gilkey, Ramadan, Aurora, Welch, 1997).

Neurochemical alterations following mild traumatic head injury involve a cascade of destructive events that begin with neuronal depolarisation and potentially lead to cell impairment.
and destruction. The neurochemical changes include neuronal depolarization, excessive release of excitatory neurotransmitters and amino acids, serotonergic dysfunction, alterations in endogenous opiates, reductions in cerebral metabolic activity, loss of calcium homeostasis and changes in magnesium levels. These primary neurochemical disturbances are found to lead ultimately to neurological deficits. These neurochemical changes also underlie migraine headaches and are observed after mild and, to a lesser degree, after more severe head injuries (Packard, Ham, 1996). In both, mild head injury and migraine seem to be dysfunctional systems which might activate a “common headache pathway” (Packard, Ham, 1996). From the literature reviews there appear to be no objective findings of a direct link between the physiological and neurochemical changes observed after mild head injury and posttraumatic headache. However, their similarities make a link between both conditions likely.

**Psychological Factors Contributing to Posttraumatic Headaches**

Pain is the place where the mind and the body meet. Following a head injury, patients try to make sense of their experience. The sensation of pain and the suffering as a consequence, may also have a personal and symbolic meaning.

Psychosocial disturbances following traumatic brain injury include Posttraumatic Stress Disorder, generalized anxiety, depression, behavioural disorders, social/family tension, occupational/financial problems and cognitive impairments (Lishman, 1998; Williams, Evans, 2003). Ham, Andrasik, Packard and Bundrick (1994) compared chronic posttraumatic headache patients to another chronic pain and a non-pain group. The results indicated that individuals with posttraumatic headache exhibited more psychopathology than individuals with idiopathic headache or the control subjects.

Fields (1997) showed that psychological factors can be very powerful in increasing or decreasing pain. Psychological processes can influence the circuit that controls nociceptive neurons in the dorsal horn. Attention and expectation can alone produce pain sensations (even in the absence of noxious stimuli). Neurons responsible for pain transmission are modulated by somatosensory, cortical and limbic structures via the periaqueductal grey matter and brainstem regions. These pathways can be activated by psychological factors having control over pain transmitting neurons. Even in the absence of damage due to head trauma, physiological mechanisms appear to convert psychological distress into painful sensations. Psychological mechanisms do not only contribute to the perception of acute pain, but also influence the development and maintenance of posttraumatic headache.

Stress following injury is common. Often the head injury opposes a life-threatening event which leads to a disruption of the personal, family and occupational life of the survivor. Cognitive difficulties have an impact upon the ability of the individual to adjust to changed circumstance and the loss/decrease of quality of life.

Personality factors (e.g., coping styles, perception of self/others/world) usually do not in themselves cause a headache, but they can be involved in symptom development, response to injury and headache management. Individuals with good coping abilities would tend to adjust better to mild head injury and treatment than anxious, depressed or insecure individuals (Packard, Ham, 1994; Packard, 1999). In an earlier study, De Benedittis and De Santis (1983) compared psychopathological profiles of mild traumatic brain injury patients with those who did not develop headaches. They reported that the non-headache group scored in the normal range on the Minnesota Multiphasic Personality Inventory (MMPI) profile. Patients with mild to moderate post-traumatic headache scored only slightly abnormal on the personality inventory whereas patients with severe posttraumatic headaches had abnormal results in the ‘neurotic triad’ (hypocondriasis, depression and hysteria) and emotional and social discomfort.

In summary, psychological mechanisms need to be taken into account in medical and psychological headache treatment. Pain perception and fear of pain may limit a person’s ability to function and may lead to the adoption of unhelpful coping styles. These can include avoidance of activity and medication overuse, leading to medication-induced chronic headache (Warner, Fenichel, 1996).

**The Impact of Legal Matters on Posttraumatic Headaches**

Posttraumatic headaches following an accident often result in legal disputes. It is argued that symptoms following head injury were over-reported in cases of ongoing/outstanding compensation claims (Lees-Haley, Brown, 1993; Fox, Lees-Haley, Earnest, Dolezal-Wood, 1995). Binder and Rohling (1996) have investigated the effects of litigation on neuropsychological complaints and test performance. Their results demonstrate consistent, significant effects leading to decreased test performance or increased symptom reporting across all studies that were examined.

However, other studies have challenged the ‘accident neurosis’ hypothesis on the basis of empirical research (Alexander, 1995). Packard (1992) reported that the participants in his study continued to suffer with headaches after settlement. He is also of the opinion that litigation does not prolong the duration of the illness. Young and Packard (1997) support the argument that symptom chronicity is not determined by litigation or that settlement terminates the symptom. Radanov et al., (2001) argue that psychosocial problems that follow brain injury emerge secondarily and reject the relevance of posttraumatic headache due to compensation neurosis.

**Headaches and Quality of Life**

Investigations on quality of life in headache focus on the way in which chronic or recurring pathology like headaches influence well being, welfare and health of a patient (Cavallini, Micieli, Bussone, Rossi, Napi, 1995; Dahlof, 1993). Assessing quality of life has become an important point from which to understand the complexity of health related problems. It appears that headache is not only related to the biological pathogenesis, but also to the supporting psychological variables. Sufferers complain of limitations of function and impaired quality of life (Solomon, Skobieranda, Gragg, 1993). Cavallini et al., (1995) found that the majority of patients with chronic headache experienced a reduction of their functional capacity during a headache attack which had an impact on their relationships.
Osterhaus, Townsend, Gandek and Ware (1994) found that migraine sufferers had worse quality of life than a healthy community sample. Jenkinson (1990) found that migraineurs had more problems in the areas of energy, emotional reactions, sleep, social isolation and pain. Solomon et al., (1993) found that the extent of physical limitations due to migraine was beyond expectation. They concluded that patients with chronic headaches functioned on a lower level than other patient groups with chronic painful diseases such as arthritis, back pain, diabetes and depression. These physical limitations have an impact upon welfare, another characteristic of quality of life. Osterhaus et al., (1994) found an immense loss of productivity in migraine sufferers. Their headache group had furthermore a higher percentage of patients in poor health than in the general population. Essink-Bot, van Royen, Krabbe, Bonsel, Rutten (1995) investigated the impact of migraine on quality of life and found large differences between the migraine and control group. The largest differences between the two groups found were in the areas of pain, physical role limitations, household work, social functioning, home life, vitality, energy and general health. Holroyd, Stensland, Lipchik, Hill, O’Donnell and Cordingley (2000) found that patients with chronic tension type headache were seven times more likely to suffer impairments in the areas of physical, social or role functioning measured on the Medical Outcomes Study 20-item instrument (MOS-20). Aggravating symptoms to the headache condition seem to be changes in sleep patterns (Lacroix, Barbaeree, 1990; Holroyd et al., 2000).

Monzon and Lainez (1998) investigated quality of life in migraine and chronic daily headache patients. Their findings revealed a lower level of functioning of the chronic daily headache sample in each of the health concepts than the migraine patients. These health concepts include mental health, in particular anxiety.

It is generally recognized that emotional reactions to pain, influence the impact of the pain on the individual (Fernandez, Turk, 1992; Melzack, Wall, 1983). Headache accompanied by anxiety or depression is likely to be linked with greater disability than headache of the same severity but less psychological distress. About half of the tension headache sufferers in the Holroyd et al., (2000) study qualified for anxiety and mood disorder. Although the reported psychological symptoms were only in the mild/moderate range it seems nevertheless important to fully investigate secondary symptoms in order to decide on the most appropriate treatment strategy. It appears that headache affects many important areas of a person’s life. Cavallini et al., (1995) found evidence that patients were anxious and distressed with regards to the imminence of headache episodes during the interval between headaches and planned or reduced their social life accordingly. They took anticipatory actions to reduce the pain or to eliminate triggers. Consequently, sufferers restrict their life and functioning, which impacts on their level of quality of life even during pain free periods.

A large body of research focused on the level of productivity of headache sufferers (Espir, Thomason, Blau, Kutz, 1988; Michel, Dartigues, Lindoulsi, Henry, 1997; Osterhaus, Guttermann, Plachetka, 1990 & 1992). In particular, Holroyd et al., (2000) reported that tension headache caused sufferers to stay away from work. 74% reported at least one disability day over a period of six months, but the average of disability days was seven days over the period of half a year. However, due to the constant but moderate intensity of this particular type of headache, patients with less severe pain continue with their work or other activities. Lacroix and Barbaeree (1990) reported that headache affected 65% of their participants in their occupation and 17% changed their jobs due to headaches. In an older study by Pelz and Merskey (1982), headache patients were reported to have had reduced participation in leisure activities.

Such impairment due to restrictions during acute and anticipated headache attacks put chronic sufferers in line with patients with similar functional impairment due to myocardial infarction or congestive heart failure (Steward, Greenfield, Hays, Wells, Rogers, Berry, McGlynn, Ware, 1989).

Quality of life, health and welfare are affected by chronic headache conditions. Comparative studies have looked at the differences between headache diagnoses and quality of life impact (Monzon, Lainez, 1998; Solomon, Skobieranda, Gragg, 1994). Although there appears to be a lack of research, it is assumed that posttraumatic headache also has a significant impact on the individual’s life. Furthermore, being mindful of posttraumatic headache as a symptom within the post-concussional concept, it is possible that is has a distinct profile on the quality of life scales in comparison to the naturally occurring headaches.

Psychological Treatment Approaches For Chronic Headaches

The previous section described the multifaceted picture of the most common types of chronic headaches. Psychological treatments aim to change several psychological contributors to the headache.

Lipton and Steward (1999) asked patients about what they want most from the physicians who care for their headaches. Eighty six percent of patients gave the need for education the highest priority whereas only 15% of physicians deemed this to be important. This study confirms the findings of Packard (1979) in his earlier study on the needs of headache patients. Apart from the relief of pain, an intervention should enable the patient to be actively involved in a process which should also aim at improvement of quality of life, level of functioning and long-term reduction of disability (Lipton, Steward, 1995). Patients often suffer less frequent or less severe headaches and better overall management with pacing, coping, relaxation skills and education about the whole problem (Medina, 1992).

Psychological treatments such as biofeedback, relaxation, cognitive-behavioural therapy and hypnotherapy have gained widespread acceptance among professionals who specialize in the treatment of headache (Penzien, Rains, Andrasik, 2002). Chronic pain affects all aspects of the sufferer’s life and it is important that relevant health professionals address each of these areas. Multidisciplinary pain management programmes using a cognitive-behavioural approach have been demonstrated as effective (Wong, 2002).

Nearly all patients seen for behavioural treatments are taking additionally some form of prescription or over-the-counter medication. It seems that many investigations are presenting behavioural treatments without controlling for use of medicines. Behavioural treatments for chronic headaches (e.g., migraine and tension type headache) have been designed and implemented to address the complexity of biological pain, well-being, psycho-
logical functioning and environment. The effectiveness of behavioural treatments is said to be based on mechanisms such as: self-regulation of physiology, cognitive mediation, enhancement of self-efficacy and the neurochemical underpinnings.

Pennini et al., (2002) in their meta-analysis of behavioural therapies for migraine and tension-type headache concluded that these interventions lead to clinically significant reductions in headaches. They reported approximately 35-50% reductions in migraine and tension-type headache. Studies which compare medical to behavioural approaches showed that behavioural interventions can maintain their long-term improvements and are thus compatible with traditional medical approaches. Wong (2002) reported that cognitive-behavioural therapy for pain in general led to a reduction in drug consumption and thus to a reduction of the cost of treatment for pain as a whole.

On the other hand, it is important to consider the complexity of all patient variables. Therefore, a patient-tailored combination of medical and behavioural approaches might be an optimal solution for the treatment of chronic headache. Holroyd, O’Donnell, Stensland, Lipchik, Cordingley, Carlson (2001) report a reduction of tension-type headache in 64% of his participants who received a combination of stress management and antidepressant medication. Thirty-eight percent of patients in the antidepressant only control group and 35% of patients in the stress management control group experienced a reduction of headaches. Twenty-nine percent of the participants in the placebo group experienced fewer headaches at the end of the trial.

In conclusion, the management of posttraumatic headache needs to focus on both physiological changes causing pain and psychological factors which maintain or reinforce pain behaviour.

Implications for the Rehabilitation of Posttraumatic Headaches

As discussed above, there is a large body of literature on psychological treatment outcome studies for various types of headaches. However, only a few studies are specifically targeted at posttraumatic headache as a history of head injury often qualifies as an exclusion criterion (Packard, Ham, 1994 & 1996). It is believed that the complexity and severity of posttraumatic headaches exceeds that of naturally occurring headaches which is responsible for the resistance of the condition to traditional treatments (Andrasik, 1990; Martelli et al., 1999). The literature suggests that posttraumatic headache and naturally occurring headache share common pathways and they appear very similar with regards to their clinical presentation (Packard, Ham, 1997). Medicinal treatment is the same as for naturally occurring headaches, but it has been reported to have minimal effect due to unwanted side effects (Parker, 1995; Solomon, 2001). Patients’ misperceptions can result in exaggerated fears about the side effects of the medicines. Consequently, individuals do not take sufficient medication and thus chronic pain mechanisms on a neurochemical basis get established.

On the other hand, psychological treatment has proved to be only minimally successful (Andrasik, 1990). Cognitive impairments may hinder patients’ efforts to learn or to apply psychological strategies appropriately and motivational deficits may make it difficult to persist with the treatment. Emotional and mood variables might be imbalanced due to the disruption the injury has caused in the individual’s life. Treatments should be holistic in nature and have a strong educational component. Patients need to be given the rationale about emotional factors, the function of the autonomic nervous system and the raised tension and pain levels after head trauma. Patients need to be aware of the vicious cycle of pain, including the explanation that the stressor causes emotional (anger, frustration) and physical (muscle tension) tension and leads to inappropriate coping (avoidance, deactivation) which eventually creates more pain. In this way, patients receive essential information which encourages understanding of how all these factors contribute to their pain, as well as motivation to engage in an active behaviourally oriented treatment programme. This procedure also provides patients with reassurance that their complaints are real (Martelli et al., 1999).

Muse (1986) reported the use of Systematic Desensitization for the treatment of posttraumatic headaches and Post Traumatic Stress Disorder (PTSD). The rationale of the study was based on the assumption that the reduction of anxiety also reduced the physiological arousal levels, which then have a positive effect on posttraumatic headache. The outcome of the study confirmed that anxiety and avoidance were reduced as a result of the intervention. However, headache was less improved. Bennett (1988) argues that attention/diversion techniques are important features of a cognitive-behavioural programme for posttraumatic headache. This is based on the assumption that attention/diversion and the management of negative thoughts helps the patient to decrease the perception of the pain, minimizes negative thoughts and dysphoric affect. The sensory components of the pain can also be modified by attention/diversion and imagery. Self-monitoring of the pain is based on cognitive processes which accompany the headache episode. Therefore, management of irrational thoughts is aimed at establishing control over these and the ability to formulate helpful positive thoughts with regards to coping ability. Bennett (1988) reported three levels of results of this type of intervention. Many patients experienced a complete remission of their symptoms, others learned to self-manage their pain with a noticeable improvement in pain levels and the majority felt that at least the headache no longer interfered with their lives.

Another behavioural approach - biofeedback - has been applied to the treatment of posttraumatic headache by Ham and Packard (1996). Fifty-nine percent of patients who had received other forms of headache treatment in the past reported improvement of their pain levels. Eighty percent had moderately improved their ability to relax and cope with the pain, 93% found biofeedback helpful to some degree, 85% experienced continued relief from their headache at follow-up and 95% continued to use biofeedback in daily life. Regression analysis of their results revealed that the more chronic the headache condition was, the poorer was the expected treatment outcome. Daly and Wulff (1987) described the use of imagery and relaxation in a single case study. The patient reported significant improvement of his headaches following seven sessions of therapy. This enabled him to discontinue all medication.

In another older study McGrady, Bernal, Fine and Woerner (1983) describe a combination of biofeedback assisted relaxation and stress management in the case of posttraumatic headaches. Release of tension was especially aimed at painful areas of the head and neck which had caused the patient to compensate for their pain or to protect the painful area by adopting an unnatural
posture, thus increasing tension and exacerbating their headaches. The results demonstrated a significant decrease of muscular tension of between 48-66% and a significant decrease of headache of 41%.

**Conclusion**

Posttraumatic headache appears to have similar characteristics to naturally occurring headaches. Assumptions were made that similar psychological treatment approaches would be appropriate for posttraumatic headaches. However, the presented literature also outlined differences regarding the complexity of posttraumatic headaches as part of the PCS and its association to a significantly traumatic life event. These differences need to be investigated further in order to find determinants for treatment motivation or resistance.

The area of quality of life with posttraumatic headache is also under-researched. To achieve a deeper and more detailed understanding of functioning and emotional well being with posttraumatic headaches, it would be necessary to investigate further whether quality of life dimensions show a similar profile to those of patients with naturally occurring headaches.

Improvement of functioning in daily life and emotional well-being are central concepts for posttraumatic headache management. The application of new insights would determine the use of optimal treatment procedures. A number of studies of posttraumatic headache treatment are dated and it appears necessary to evaluate the effectiveness of new developments in psychological and medical assessment and treatment techniques, including cognitive-behavioural therapies, for this condition.

**References**


