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EFNS GUIDELINES/CME ARTICLE

EFNS guideline on the treatment of tension-type headache – Report of an EFNS task force

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Background: Tension-type headache (TTH) is the most prevalent headache type and is causing a high degree of disability. Treatment of frequent TTH is often difficult.
Objectives: To give evidence-based or expert recommendations for the different treatment procedures in TTH based on a literature search and the consensus of an expert panel.
Methods: All available medical reference systems were screened for the range of clinical studies on TTH. The findings in these studies were evaluated according to the recommendations of the EFNS resulting in level A, B or C recommendations and good practice points.
Recommendations: Non-drug management should always be considered although the scientific basis is limited. Information, reassurance and identification of trigger factors may be rewarding. Electromyography (EMG) biofeedback has a documented effect in TTH, whilst cognitive-behavioural therapy and relaxation training most likely are effective. Physical therapy and acupuncture may be valuable options for patients with frequent TTH, but there is no robust scientific evidence for efficacy. Simple analgesics and non-steroidal anti-inflammatory drugs are recommended for the treatment of episodic TTH. Combination analgesics containing caffeine are drugs of second choice. Triptans, muscle relaxants and opioids should not be used. It is crucial to avoid frequent and excessive use of analgesics to prevent the development of medication-overuse headache. The tricyclic antidepressant amitriptyline is drug of first choice for the prophylactic treatment of chronic TTH. Mirtazapine and venlafaxine are drugs of second choice. The efficacy of the prophylactic drugs is often limited, and treatment may be hampered by side effects.

Objectives

These guidelines aim to give evidence-based recommendations for the acute and prophylactic drug treatment of TTH. In addition, the guidelines aim to provide a short overview on non-drug treatment of TTH based on the best performed controlled trials, reviews and meta-analyses, whilst the vast amount of uncontrolled reports of non-drug treatment will not be considered. A brief clinical description of the headache disorders is included. The definitions follow the diagnostic criteria of the International Headache Society (IHS) [1].

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This is a Continuing Medical Education article, and can be found with corresponding questions on the Internet at http://www.efns.org/EFNS Continuing-Medical-Education-online.301.0.html. Certificates for correctly answering the questions will be issued by the EFNS.

Background

Tension-type headache is classified into three subtypes according to headache frequency: infrequent episodic TTH (< 1 day of headache per month), frequent episodic TTH (1–14 days of headache per month) and chronic TTH (≥15 days per month) [1] (Table 1). This division may seem artificial but has proved to be highly relevant for several reasons. First impact on quality of life differs considerably between the subtypes. A person

Table 1 Diagnostic criteria of tension-type headache of the IHS classification [1]

2.1 Infrequent episodic tension-type headache
A. At least 10 episodes occurring on <1 day per month on average (<12 days per year) and fulfilling criteria B–D
B. Headache lasting from 30 min to 7 days
C. Headache has at least two of the following characteristics:
1. Bilateral location
2. Pressing/tightening (non-pulsating) quality
3. Mild or moderate pain intensity
4. Not aggravated by routine physical activity such as walking or climbing stairs
D. Both of the following:
1. No nausea or vomiting (anorexia may occur)
2. No more than one of photophobia or phonophobia
E. Not attributed to another disorder
2.2 Frequent episodic tension-type headache
As 2.1 except for:
A. At least 10 episodes occurring on ≥ 1 but <15 days per month for at least 3 months (≥ 12 and <180 days per year) and fulfilling criteria B-D
2.3 Chronic tension-type headache
As 2.1 except for:
A. Headache occurring on ≥ 15 days per month on average for >3 months (≥ 180 days per year) and fulfilling criteria B–D
B. Headache lasts hours or may be continuous
D. Both of the following:
1. No more than one of photophobia, phonophobia or mild nausea
2. Neither moderate or severe nausea or vomiting

having headache every day from the time of waking, persisting until bedtime, month in and month out, is disabled. At the other extreme, a mild headache once every other month has very little impact on health or functional ability and needs little if any medical attention. Second, the pathophysiological mechanisms may differ significantly between the subtypes; peripheral mechanisms are probably more important in episodic TTH, whereas central pain mechanisms are pivotal in chronic TTH [2]. Third, treatment differs between the subtypes, with symptomatic and prophylactic treatments being more appropriate for episodic and chronic TTH, respectively. Therefore, a precise diagnosis is mandatory and should be established by means of a headache diary [3] completed for at least 4 weeks.

The recommendations in this article are based on the scientific evidence from clinical trials and on the expert consensus by the respective task force of the EFNS. The legal aspects of drug prescription and drug availability in the different European countries will not be considered. The definitions of the recommendation levels follow the EFNS criteria [4]. Briefly, a level A rating (established as effective, ineffective or harmful) requires at least one convincing class I study or at least two consistent convincing class II studies. A level B rating (probably effective, ineffective or harmful) requires at least one convincing class II study or overwhelming class III evidence, whilst a level C rating (possibly effective, ineffective or harmful) requires at least two convincing class III studies [4].

In general, non-pharmacological management should always be considered in TTH [5]. When it comes to pharmacological management, the general rule is that patients with episodic TTH [1] are treated with symptomatic (acute) drugs, whilst prophylactic drugs should be considered in patients with very frequent episodic TTH and in patients with chronic TTH [1]. Analgesics are often ineffective in patients with chronic TTH. Furthermore, their frequent use produces risk of toxicity (e.g. kidney and liver problems) as well as of medication-overuse headache [6].

Search strategy

A literature search was performed using the reference databases MedLine, Science Citation Index and the Cochrane Library; the key word used were 'tension-type headache' (last search October 2009). In addition, a review book [7] and treatment recommendations from the British Association for the Study of Headache [8] were considered. Trials published in English and conducted amongst adult patients (aged 18 and older) with reasonable criteria designed to distinguish TTH from migraine were considered. For drug treatments, randomized placebo-controlled trials and trials comparing different treatments were considered. For non-drug treatments, controlled trials were considered.

Method for reaching consensus

All authors performed an independent literature search. The first draft of the manuscript was written by the chairman of the task force. All other members of the task force read the first draft and discussed changes by email. Three more drafts were then written by the chairman and each time discussed by email. All recommendations had to be agreed to by all members of the task force unanimously. The background of the research strategy and of reaching consensus and the definitions of the recommendation levels used in this article have been described in the EFNS recommendations [4].

Epidemiology

The lifetime prevalence of TTH was as high as 78% in a population-based study in Denmark, but the majority had episodic infrequent TTH (1 day a month or less) without specific need of medical attention [9]. Nevertheless, 24–37% had TTH several times a month, 10% had it weekly and 2–3% of the population had chronic TTH usually lasting for the greater part of a lifetime [9,10].

The female:male ratio of TTH is 5:4 indicating that, unlike migraine, women are only slightly more affected than men [11,12]. The average age of onset of TTH is higher than that in migraine, namely 25–30 years in cross-sectional epidemiological studies [10]. The prevalence peaks between the age of 30 to 39 and decreases slightly with age. Poor self-rated health, inability to relax after work and sleeping few hours per night have been reported as risk factors for developing TTH [13].

A recent review of the global prevalence and burden of headaches [11] showed that the disability of TTH as a burden of society was greater than that of migraine, which indicates that the overall cost of TTH is greater than that of migraine. Two Danish studies have shown that the number of workdays missed in the population was three times higher for TTH than for migraine [10,14], and a US study has also found that absenteeism because of TTH is considerable [15]. The burden is particularly high for the minority who have substantial and complicating co-morbidities [16].

Clinical aspects

TTH is characterized by a bilateral, pressing tightening pain of mild to moderate intensity, occurring either in short episodes of variable duration (episodic forms) or continuously (chronic form). The headache is not associated with the typical migraine features as vomiting, severe photophobia and phonophobia. In the chronic form, only one of the latter two accompanying symptoms or mild nausea is accepted [1] (Table 1). Because of lack of accompanying symptoms and the relatively milder pain intensity, patients are rarely severely incapacitated by their pain. TTH is the most featureless of the primary headaches, and because many secondary headaches may mimic TTH, a diagnosis of TTH requires exclusion of other organic disorders.

Diagnosis

The diagnosis of TTH is based on the typical patient's history and a normal neurological examination. A correct diagnosis should be assured by means of a headache diary [3] recorded over at least 4 weeks. The diagnostic problem most often encountered is to discriminate between TTH and mild migraine. If the headache is strictly unilateral, the debated entity cervicogenic headache should be considered [17]. The diary may also reveal triggers and medication overuse, and it will establish the baseline against which to measure the efficacy of treatments. Identification of a high intake of analgesics is essential because medication overuse requires specific treatment [6]. Paraclinical investigations, in particular brain imaging, is necessary if secondary headache is suspected (e.g. the headache characteristics are untypical), if the course of headache attacks changes or if persistent neurological or psychopathological abnormalities are present. Significant co-morbidity, e.g. anxiety or depression, should be identified and treated concomitantly. Poor compliance with prophylactic treatment may be a problem in chronic TTH as it is in migraine [18]. It should be explained to the patient that frequent TTH only seldom can be cured, but that a meaningful improvement often can be obtained with the combination of drug and non-drug treatments.

Acute drug treatment of TTH

Acute drug therapy refers to the treatment of individual attacks of headache in patients with episodic and chronic TTH. Most headaches in patients with episodic TTH are mild to moderate, and the patients often can self-manage using simple analgesics (paracetamol or aspirin) or non-steroidal anti-inflammatory drugs (NSAIDs). The efficacy of the simple analgesics tends to decrease with increasing frequency of the headaches. In patients with chronic TTH, the headaches are often associated with stress, anxiety and depression, and simple analgesics are usually ineffective and should be used with caution because of the risk of medication-overuse headache at a regular intake of simple analgesics above 14 days a month or triptans or combination analgesics above 9 days a month [19]. Other interventions such as non-drug treatments and prophylactic pharmacotherapy should be considered.

The effect of acute drugs in TTH has been examined in many studies, and these have used many different methods for the measurement of efficacy. The guidelines for drug trials in TTH from the International Headache Society recommend pain-free after 2 h as the primary efficacy measure [20]. This has been used in some studies whilst many studies have used other efficacy measures such as pain intensity difference, time to meaningful relief. This makes comparison of results between studies difficult.

Simple analgesics and NSAIDs

Paracetamol 1000 mg was significantly more effective than placebo in most [21–27] but not all [28,29] trials, whilst three trials found no significant effect of paracetamol 500 mg to 650 mg compared with placebo [21,28,30].

Aspirin has consistently been reported more effective than placebo in doses of 1000 mg [21,31,32], 500 mg to 650 mg [21,32–34] and 250 mg [32]. One study found no difference in efficacy between solid and effervescent aspirin [34].

Ibuprofen 800 mg [33], 400 mg [24,25,33,35,36] and 200 mg [37] are more effective than placebo, as are

ketoprofen 50 mg [28,37], 25 mg [27,29,37] and 12.5 mg [29]. One study could not demonstrate a significant effect of ketoprofen 25 mg possibly because of a low number of patients [28]. Diclofenac 25 mg and 12.5 mg have been reported effective [35], whilst there are no trials of the higher doses of 50–100 mg proved effective in migraine. Naproxen 375 mg [26] and 550 mg [30,38] and metamizole 500 and 1000 mg [31] have also been demonstrated effective. The latter drug is not available in many countries, because it carries a minimal (if at all) risk of causing agranulocytosis. Treatment with intramuscular injection of ketorolac 60 mg in an emergency department has been reported effective [39].

Optimal dose

There are only few studies investigating the ideal dose for drugs used for the acute treatment of TTH. One study demonstrated a significant dose–response relationship of aspirin with 1000 mg being superior to 500 and 500 mg being superior to 250 mg [32]. Ketoprofen 25 mg tended to be more effective than 12.5 mg [29], whilst another study found very similar effects of ketoprofen 25 and 50 mg [37]. Paracetamol 1000 mg seems to be superior to 500 mg, as only the former dose has been demonstrated effective. In lack of evidence, the most effective dose of a drug well tolerated by a patient should be chosen. Suggested doses are presented in Table 2.

Comparison of simple analgesics

Five studies reported NSAIDs to be significantly more effective than paracetamol [24,25,28–30], whilst three studies could not demonstrate a difference [21,26,27]. Five studies have compared efficacy of different NSAIDs, and it has not be possible to clearly demonstrate the superiority of any particular drug [31,33,35,37,40].

Adverse events

A thorough review of the acute drug treatment of TTH could not detect any difference in adverse events between paracetamol and NSAIDs or between these drugs and placebo [41]. However, it is well known that NSAIDs have more gastro-intestinal side effects than paracetamol, whilst the use of large amounts of paracetamol may cause liver injury. Amongst the NSAIDs, ibuprofen seems to have the most favourable side effect profile [41].

Combination analgesics

The efficacy of simple analgesics and NSAIDs is increased by combination with caffeine 64–200 mg [22,23,42–45]. There are no comparative studies examining the efficacy of combination with codeine. It is clinically well known that caffeine withdrawal can cause headache and chronic daily headache has been reported to be associated with use of over-the-counter caffeine combination products [46]. Therefore, it is probable that combinations of simple analgesics or NSAIDs with caffeine are more likely to induce MOH than simple analgesics or NSAIDs alone. Until otherwise proven, we therefore recommend that simple analgesics or NSAIDs are drugs of first choice and that combinations of one of these drugs with caffeine are drugs of second choice for the acute treatment of TTH. Combinations of simple analgesics with codeine or barbiturates should not be used, because use of the latter drugs increases the risk of developing medication-overuse headache [46].

Triptans, muscle relaxants and opioids

Triptans have been reported effective for the treatment of interval headaches [47], which were most likely mild migraines [48], in patients with migraine. Triptans most likely do not have a clinically relevant effect in patients with TTH [49,50] and cannot be recommended. Muscle relaxants have not been demonstrated effective in episodic TTH [51]. Use of opioids increases the risk of developing medication-overuse headache [46]. Opioids are not recommended for the treatment of TTH.

Table 2 Recommended drugs for acute therapy of tension-type headache

Substance	Dose	Level of recommendation	Comment
Ibuprofen	200–800 mg	A	Gastrointestinal side effects, risk of bleeding
Ketoprofen	25 mg	A	Side effects as for ibuprofen
Aspirin	500–1000 mg	A	Side effects as for ibuprofen
Naproxen	375–550 mg	A	Side effects as for ibuprofen
Diclofenac	12.5–100 mg	A	Side effects as for ibuprofen, only doses of 12.5–25 mg tested in TTH
Paracetamol	1000 mg (oral)	A	Less risk of gastrointestinal side effects compared with NSAIDs
Caffeine comb.	65–200 mg	B	See below[a]

The level of recommendation considers side effects and consistency of the studies. There is sparse evidence for optimal doses. The most effective dose of a drug well tolerated by a patient should be chosen; NSAID, non-steroidal anti-inflammatory drugs; TTH, tension-type headache;
[a]Combination with caffeine 65–200 mg increases the efficacy of ibuprofen [43] and paracetamol [23,42], but possibly also the risk for developing medication-overuse headache [46,53]. Level of recommendation of combination drugs containing caffeine is therefore B.

Conclusions

Simple analgesics and NSAIDs are the mainstays in the acute therapy of TTH (Table 2). Paracetamol 1000 mg is probably less effective than the NSAIDs but has a better gastric side effect profile [52]. Ibuprofen 400 mg may be recommended as drug of choice amongst the NSAIDs because of a favourable gastrointestinal side effect profile compared with other NSAIDs [52]. Combination analgesics containing caffeine are more effective than simple analgesics or NSAIDs alone but are regarded by some experts [53] to more likely induce medication-overuse headache. Physicians should be aware of the risk of developing medication-overuse headache as a result of frequent and excessive use of all types of analgesics in acute therapy [6]. Triptans, muscle relaxants and opioids do not play a role in the treatment of TTH.

Although simple analgesics and NSAIDs are effective in episodic TTH, the degree of efficacy has to be put in perspective. For example, the proportion of patients that were pain-free 2 h after treatment with paracetamol 1000 mg, naproxen 375 mg and placebo were 37%, 32% and 26%, respectively [26]. The corresponding rates for paracetamol 1000 mg, ketoprofen 25 mg and placebo were 22%, 28% and 16% in another study with 61%, 70% and 36% of subjects reporting worthwhile effect, respectively [27]. Thus, efficacy is modest, and there is clearly room for better acute treatment of episodic TTH.

Recommendations

Simple analgesics and non-steroidal anti-inflammatory drugs are recommended for the treatment of episodic TTH. Combination analgesics containing caffeine are drugs of second choice. It is crucial to avoid frequent and excessive use of analgesics to prevent the development of medication-overuse headache.

Prophylactic drug treatment of TTH

Prophylactic pharmacotherapy should be considered in patients with chronic TTH, and it can be considered in patients with very frequent episodic TTH. Co-morbid disorders, e.g. overweight or depression, should be taken into account. For many years, the tricyclic antidepressant amitriptyline has been used. More lately other antidepressants, NSAIDs, muscle relaxants, anticonvulsants and botulinum toxin have been tested in chronic TTH. The effect of prophylactic drugs in TTH has been examined in surprisingly few placebo-controlled studies, which have used different methods for the measurement of efficacy. The guidelines for drug trials in TTH from the International Headache Society recommend days with TTH or area-under-the-headache curve (AUC) to be used as primary efficacy measure [20]. These parameters have been used in some studies, whilst other studies have used other efficacy measures such as pain reduction from baseline, headache intensity. This makes comparison of results between studies difficult.

Amitriptyline

Lance and Curran [54] reported amitriptyline 10–25 mg three times daily to be effective, whilst Diamond and Baltes [55] found amitriptyline 10 mg/day but not 60 mg/day to be effective. Amitriptyline 75 mg/day was reported to reduce headache duration in the last week of a 6-week study [56], whilst no difference in effect size between amitriptyline 50–75 mg/day or amitriptylinoxide 60–90 mg/day and placebo was found in one study [57]. However, also the frequencies of side effects were similar on amitriptyline and placebo in the latter study. The inability to detect the well-known side effects of amitriptyline suggests insensitivity of the trial for reasons which remain obscure. Bendtsen *et al.* [58] found that amitriptyline 75 mg daily reduced the area-under-the-headache curve (calculated as headache duration times headache intensity) by 30% compared with placebo, which was highly significant. Holroyd and colleagues [59] treated patients with antidepressants (83% took amitriptyline median dose 75 mg daily and 17% took nortriptyline median dose 50 mg daily) and compared this with stress management therapy and with a combination of stress management and antidepressant treatment. After 6 months, all three treatments reduced headache index with approximately 30% more than placebo, which was highly significant.

Other antidepressants

The tricyclic antidepressant clomipramine 75–150 mg daily [60] and the tetracyclic antidepressants maprotiline 75 mg daily [61] and mianserin 30–60 mg daily [60] have been reported more effective than placebo. Interestingly, some of the newer more selective antidepressants with action on serotonin and noradrenaline seem to be as effective as amitriptyline with the advantage that they are tolerated in doses needed for the treatment of a concomitant depression. Thus, the noradrenergic and specific serotonergic antidepressant mirtazapine 30 mg/day reduced headache index by 34% more than placebo in difficult to treat patients without depression including patients who had not responded to amitriptyline [62]. The efficacy of mirtazapine was comparable to that of amitriptyline reported by the same group [58]. A systematic review concluded that the two treatments may be equally effective for the treatment of chronic TTH [63]. The serotonin and noradrenaline reuptake inhibitor venlafaxine 150 mg/day [64] reduced headache days

from 15 to 12 per month in a mixed group of patients with either frequent episodic or chronic TTH. Low-dose mirtazapine 4.5 mg/day alone or in combination with ibuprofen 400 mg/day was not effective in chronic TTH. The selective serotonin reuptake inhibitors (SSRIs), citalopram [58] and sertraline [65], have not been found more effective than placebo. SSRI's have been compared with other antidepressants in six studies. These studies were reviewed in a Cochrane analysis that concluded that SSRI's are less efficacious than tricyclic antidepressants for the treatment of chronic TTH [66].

Miscellaneous agents

There have been conflicting results for treatment with the muscle relaxant tizanidine [61,67], whilst the NMDA-antagonist memantine was not effective [68]. Botulinum toxin has been extensively studied [69–79]. It was concluded in a systematic review that botulinum toxin is likely to be ineffective or harmful for the treatment of chronic TTH [63]. The prophylactic effect of daily intake of simple analgesics has not been studied in trials that had this as the primary efficacy parameter, but explanatory analyses indicated that ibuprofen 400 mg/day was not effective in one study [80]. On the contrary, ibuprofen increased headache compared with placebo indicating a possible early onset of medication-overuse headache [80]. Topiramate [81] and buspirone [82] have been reported effective in open-label studies.

Conclusions

Amitriptyline has a clinically relevant prophylactic effect in patients with chronic TTH and should be drug of first choice (Table 3). Mirtazapine or venlafaxine are probably effective, whilst the older tricyclic and tetracyclic antidepressants, clomipramine, maprotiline and mianserin, may be effective. A recent systematic review [63] concluded that amitriptyline and mirtazapine are the only forms of treatment that can be considered proven beneficial for the treatment of chronic TTH. However, the last search was performed in 2007 before publication of the study on venlafaxine [64].

Amitriptyline should be started at low dosages (10–25 mg/day) and titrated by 10–25 mg weekly until the patient has either good therapeutic effect or side effects are encountered. It is important that patients are informed that this is an antidepressant agent but has an independent action on pain. The maintenance dose is usually 30–75 mg daily administered 1–2 h before bedtime to help to circumvent any sedative adverse effects. The effect is not related to the presence of depression [58]. A significant effect of amitriptyline may be observed already in the first week on the therapeutic dose [58]. Therefore, it is advisable to change to other prophylactic therapy, if the patient does not respond after 4 weeks on maintenance dose. The side effects of amitriptyline include dry mouth, drowsiness, dizziness, obstipation and weight gain. Mirtazapine, of which the major side effects are drowsiness and weight gain, or venlafaxine, of which the major side effects are vomiting, nausea, dizziness and loss of libido, should be considered if amitriptyline is not effective or not tolerated. Discontinuation should be attempted every 6–12 months. The physician should keep in mind that the efficacy of preventive drug therapy in TTH is often modest and that the efficacy should outweigh the side effects.

Table 3 Recommended drugs for prophylactic therapy of tension-type headache

Substance	Daily dose	Level of recommendation
Drug of first choice		
Amitriptyline	30–75 mg	A
Drugs of second choice		
Mirtazapine	30 mg	B
Venlafaxine	150 mg	B
Drugs of third choice		
Clomipramine	75–150 mg	B
Maprotiline	75 mg	B
Mianserin	30–60 mg	B

The level of recommendation considers side effects and number and quality of the studies.

Recommendations

Amitriptyline is drug of first choice for the prophylactic treatment of chronic TTH. Mirtazapine and venlafaxine are drugs of second choice.

Non-pharmacologic treatment of TTH

Information, reassurance and identification of trigger factors

Non-drug management should be considered for all patients with TTH and is widely used. However, the scientific evidence for efficacy of most treatment modalities is sparse [83–86]. The very fact that the physician takes the problem serious may have a therapeutic effect, particularly if the patient is concerned about serious disease, e.g. brain tumour, and can be reassured by thorough examination. Identification of trigger factors should be performed, as coping with triggers may be of value [87]. The most frequently reported triggers for TTH are stress (mental or physical), irregular or inappropriate meals, high intake or withdrawal of coffee and other caffeine containing

Table 4 Non-pharmacological treatments for tension of tension-type headache

Treatment	Level of recommendation
Psycho-behavioural treatments	
EMG biofeedback	A
Cognitive-behavioural therapy	C
Relaxation training	C
Physical therapy	C
Acupuncture	C

The level of recommendation considers number and quality of the studies.

drinks, dehydration, sleep disorders, too much or too little sleep, reduced or inappropriate physical exercise, psycho-behavioural problems as well as variations during the female menstrual cycle and hormonal substitution [88–90]. It has been demonstrated that stress induces more headache in patients with chronic TTH than in healthy controls probably through hyperalgesic effects on already sensitized pain pathways [91].

Information about the nature of the disease is important. It can be explained that muscle pain can lead to a disturbance of the brain's pain-modulating mechanisms [2,92,93], so that normally innocuous stimuli are perceived as painful, with secondary perpetuation of muscle pain and risk of anxiety and depression. The prognosis in the longer run was found to be favourable in a population-based 12-year epidemiological follow-up study, because approximately half of all individuals with frequent or chronic TTH had remission of their headaches [13]. It is not known whether the same is true for individuals who seek medical consultation.

Psycho-behavioural treatments

A large number of psycho-behavioural treatment strategies have been used to treat chronic TTH. EMG biofeedback, cognitive-behavioural therapy and relaxation training have been investigated the most. However, only few trials have been performed controlled with sufficient power and clear outcome measures [85]. Hypnotherapy has been reported effective [94], but there is not convincing evidence for its effect in TTH [85,95].

EMG biofeedback

The aim of EMG biofeedback is to help the patient to recognize and control muscle tension by providing continuous feedback about muscle activity. Sessions typically include an adaptation phase, baseline phase, training phase where feedback is provided and a self-control phase where the patient practices controlling muscle tension without the aid of feedback [96]. A recent review including 11 studies concluded that because of low power there is conflicting evidence to support or refute the effectiveness of EMG biofeedback compared with placebo or any other treatments [85]. However, a recent extensive and thorough meta-analysis including 53 studies concluded that biofeedback has a medium-to-large effect. The effect was found to be long lasting and enhanced by combination with relaxation therapy [97]. The majority of the studies included employed EMG biofeedback. It was not possible to draw reliable conclusions as to whether the effect differed between patients with episodic and chronic TTH.

Cognitive-behavioural therapy

The aim of cognitive-behavioural therapy is to teach the patient to identify thoughts and beliefs that generate stress and aggravate headaches. These thoughts are then challenged, and alternative adaptive coping self-instructions are considered. A variety of exercises may be used to challenge thoughts and beliefs, including experimenting with adoption of another person's view of the situation, actively generating other possible views of a situation and devising a behavioural experiment to test the validity of a particular belief [96]. One study found cognitive-behavioural therapy, treatment with tricyclic antidepressants and a combination of the two treatments better than placebo with no significant difference between treatments [59], whilst another study reported no difference between cognitive-behavioural therapy and amitriptyline [98]. Cognitive-behavioural therapy may be effective but there is no convincing evidence [63,85].

Relaxation training

The goal of relaxation training is to help the patient to recognize and control tension as it arises in the course of daily activities. Relaxation training involves a range of affective, cognitive and behavioural techniques, such as breathing exercises and meditation. Relaxation training has been compared with no treatment or waiting list control [99–103] and with other interventions [104–107]. A recent review concluded that there is conflicting evidence that relaxation is better than no treatment, waiting list or placebo [85].

Conclusions

EMG biofeedback has an effect in TTH, whilst cognitive-behavioural therapy and relaxation training may have an effect in TTH, but at this moment, there is no convincing evidence to support this [63,85]. These treatments are relatively time-consuming, but unfortunately, there are no documented guidelines for which psycho-behavioural treatment(s) to choose for the individual patient. Therefore, until scientific evidence is provided, common sense must be used. Thus, it is likely that cognitive-behavioural therapy will be most bene-

ficial for the patient where psycho-behavioural problems or affective distress play a major role [96], whilst biofeedback or relaxation training may be preferable for the tense patient.

Non-invasive physical therapy

Physical therapy is widely used for the treatment of TTH and includes the improvement of posture, massage, spinal manipulation, oromandibular treatment, exercise programs, hot and cold packs, ultrasound and electrical stimulation, but the majority of these modalities have not been properly evaluated [108]. Active treatment strategies are generally recommended [108]. A recent review concluded that exercise may have a value for TTH [109]. Carlson *et al*. [110] reported better effect of physiotherapy than acupuncture. A controlled study [111] combined various techniques such as massage, relaxation and home-based exercises and found a modest effect. It was reported that adding craniocervical training to classical physiotherapy was better than physiotherapy alone [112]. A recent study found no significant long-lasting differences in efficacy amongst relaxation training, physical training and acupuncture [113]. Spinal manipulation has no effect in episodic TTH [114] and no convincing effect in chronic TTH [115,116]. Oromandibular treatment with occlusal splints is often recommended but has not yet been tested in trials of reasonable quality and cannot be recommended in general [117]. There is no firm evidence for efficacy of therapeutic touch, cranial electrotherapy or transcutaneous electrical nerve stimulation [84].

It can be concluded that there is a huge contrast between the widespread use of physical therapies and the lack of robust scientific evidence for efficacy of these therapies and that further studies of improved quality are necessary to either support or refute the effectiveness of physical modalities in TTH [84,108,118,119].

Acupuncture and nerve block

The prophylactic effect of acupuncture has been investigated in several trials in patients with frequent episodic or chronic TTH. A review [63] and a meta-analysis [120] concluded that there is no evidence for efficacy of acupuncture in TTH. Two trials reported better effect of acupuncture than basic care or waiting list but no better effect of Chinese acupuncture when compared to sham acupuncture [121,122], whilst a recent Cochrane analysis [86] concluded that there was overall a slightly better effect from acupuncture than from sham acupuncture based on the results from five trials [122–126]. Four trials compared acupuncture with physiotherapy [110,113,127], relaxation [113] or a combination of massage and relaxation [128]. Collectively, these trials suggest slightly better results for some outcomes with the latter therapies according to the recent Cochrane analysis [86]. Together, the available evidence suggests that acupuncture could be a valuable option for patients suffering from frequent TTH, but more research is needed before final conclusions can be made.

A recent study reported no effect of greater occipital nerve block in patients with chronic TTH [129].

Recommendations

Non-drug management should always be considered although the scientific basis is limited (Table 4). Information, reassurance and identification of trigger factors may be rewarding. EMG biofeedback has a documented effect in TTH, whilst cognitive-behavioural therapy and relaxation training most likely are effective, but there is no convincing evidence. Physical therapy and acupuncture may be valuable options for patients with frequent TTH, but there is no robust scientific evidence for efficacy.

Need of update

These recommendations should be updated within 5 years.

Conflicts of interest

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