BASH MANAGEMENT GUIDELINES

GUIDELINES FOR ALL DOCTORS IN THE DIAGNOSIS AND MANAGEMENT OF MIGRAINE AND TENSION-TYPE HEADACHE

BRITISH ASSOCIATION FOR THE STUDY OF HEADACHE

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1.0 INTRODUCTION

Headache affects nearly everyone at least occasionally. It is estimated to become a problem at some time in the lives of about 40% of people in the UK. It is one of the most frequent causes of consultation in both general practice and neurological clinics. In its various forms, headache represents an immense socioeconomic burden.

Migraine occurs in 12-15% of the UK population, in women more than men in a ratio of 3:1[1]. An estimated 187,000 attacks are experienced *every day*, with three quarters of those affected reporting disability at least sometimes[1]. Whilst migraine occurs in children (in whom the diagnosis is often missed) and in the elderly, it is most troublesome during the productive years (late teens to 50's). As a result, almost 90,000 people are absent from work or school because of migraine every working day[1]. The cost to the economy through migraine alone of lost work time and impaired working effectiveness may be £1.5 billion per annum.

Despite these statistics there is evidence that migraine is under-diagnosed and under-treated in the UK as is the case throughout Europe and in the USA.

Tension-type headache affects up to 80% of people from time to time[2], many of whom refer to it as "normal" or "ordinary" headache. Consequently, it is mostly treated without reference to physicians, using over-the-counter (OTC) medications and generally effectively. Nevertheless, its high prevalence relative to that of migraine results in what may be a similar economic burden through lost work or reduced working effectiveness. In a small minority of people, tension-type headache is frequent, and some 2-3% of adults have the chronic subtype[3] distinguished by occurrence on more than 15 days per month. These people have high morbidity, may be substantially disabled and many are chronically off work.

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2.0 SCOPE AND PURPOSE OF THESE GUIDELINES

These guidelines are intended for all doctors who manage headache. Whether in general practice or specialist clinics, the *approach* to management is the same.

The purpose of these guidelines is to suggest strategies of management of migraine and tension-type headache which have been found by specialists to work well. Headache management requires a flexible and individualised approach, and there may be circumstances in which these suggestions cannot easily be applied or are inappropriate.

It *is* recommended that healthcare commissioners incorporate these guidelines into any agreement for provision of services.

Where evidence exists, these guidelines are based on it. Unfortunately, the formal evidence for much of them is insecure and there is reliance on expert opinion based on clinical experience.

2.1 Writing and approval process

The members of the writing group are headache specialists. Each edition of these guidelines, and major revisions thereof, are distributed in draft for consultation to all members of the British Association for the Study of Headache (BASH), amongst whom are general practitioners with an interest in headache, and to all neurologist members of the Association of British Neurologists. Final approval for publication is by Council of BASH.

2.2 Currency of this edition

These guidelines are current *until the end of October, 2000*. They are updated as developments occur or on production of new and relevant evidence.

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3.0 HEADACHE CLASSIFICATION

Various schemes preceded the proposals of the International Headache Society (IHS)[4], now widely adopted (though under review). In the IHS classification, which includes operational diagnostic criteria, headache disorders are classified under 13 headings (table I). The first four of these cover the primary headache conditions.

Table I
IHS classification* of headache disorders, cranial neuralgias and facial pain[6]

Primary	1. Migraine, *including:* 1.1 Migraine without aura 1.2 Migraine with aura
	2. Tension-type headache, *including*: 2.1 Episodic tension-type headache 2.2 Chronic tension-type headache
	3. Cluster headache and chronic paroxysmal hemicrania
	4. Miscellaneous headaches unassociated with structural lesion
Secondary	5. Headache associated with head trauma, *including:* 5.1 Acute post-traumatic headache 5.2 Chronic post-traumatic headache
	6. Headache associated with vascular disorders, *including:* 6.3 Subarachnoid haemorrhage
	7. Headache associated with non-vascular intracranial disorders, *including:*

	7.1.1 Benign intracranial hypertension 7.3 Intracranial infection 7.6 Intracranial neoplasm
	8. Headache associated with substances or their withdrawal, *including:* 8.1.4 Acute alcohol induced headache 8.2.1 Chronic ergotamine induced headache 8.2.2 Chronic analgesics abuse headache 8.3.1 Alcohol withdrawal headache (hangover)
	9. Headache associated with non-cephalic infection
	10. Headache associated with metabolic disorder
	11. Headache or facial pain associated with disorder of cranium, neck, eyes, ears, nose, sinuses, teeth, mouth or other facial or cranial structures, *including:* 11.2.1 Cervical spine 11.3.1 Acute glaucoma 11.5.1 Acute sinus headache
	12. Cranial neuralgias, nerve trunk pain and deafferentation pain, *including:* 12.1.4.1 Herpes zoster 12.2 Trigeminal neuralgia
	13. Headache not classifiable

**This table is a simplification of the IHS classification*

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4.0 DIAGNOSIS OF HEADACHE

4.1 Taking a history

There are no diagnostic tests for migraine or tension-type headache, and the history is all-important. A headache history requires time to elicit, and not finding the time to take it fully is the probable cause of most misdiagnosis. A simple and helpful ploy when the patient first presents in a busy clinic is to request the keeping of a diary over a few weeks. The pattern of attacks is a very helpful pointer to the right diagnosis, and review can be arranged at a time

less rushed. First, of course, it must be ascertained that a condition requiring more urgent intervention is not present (see 5.0).

In children, migraine and tension-type headache may be less clearly distinct[5].

Different headache types are not mutually exclusive. Patients are often aware of more than one headache type, and a separate history should be taken for each. The crucial elements of a headache history are set out in table II.

Table II. An approach to the headache history	
1. How many different headaches types does the patient experience? Separate histories are necessary for each. It is reasonable to concentrate on the most bothersome to the patient but others should always attract some enquiry in case they are clinically important.	
2. Time questions	a. Why consulting now? b. How recent in onset? c. How frequent, and what temporal pattern (especially distinguishing between episodic and daily or unremitting)? d. How long lasting?
3. Character questions	a. Intensity of pain b. Nature and quality of pain c. Site and spread of pain d. Associated symptoms
4. Cause questions	a. Predisposing and/or trigger factors b. Aggravating and/or relieving factors c. Family history of similar headache
5. Response questions	a. What does the patient do during the headache? b. How much is activity (function) limited or prevented? c. What medication has been and is used, and in what manner?
6. State of health between attacks	a. Completely well, or residual or persisting symptoms? b. Concerns, anxieties, fears about recurrent attacks, and/or their cause

4.2 Migraine

Patients with migraine typically give an account of recurrent episodic moderate or severe headaches (which may be unilateral and/or throbbing) lasting part of a day or up to 3 days, associated with gastrointestinal (and sometimes visual) symptoms, during which they limit activity and prefer dark and quiet. They are free from symptoms between attacks.

Diagnostic criteria for ***migraine without aura*** (common migraine), proposed by IHS (table III), are now widely employed. It is easy to regard these as a check list, sufficient if ticked by a nurse or even the patient, but they require clinical interpretation. One of their weaknesses is that, because they supposedly define attacks, not patients, they do not describe the all-important *patterns* of occurrence of attacks. Nevertheless, if used as they are meant to be, supplementary to normal enquiry practice, these criteria are useful for distinguishing between migraine without aura and its principal differential diagnosis, tension-type headache.

Table III
IHS diagnostic criteria for migraine without aura*

An idiopathic, recurring headache disorder with:

A	At least 5 attacks fulfilling B-D
B	Headache attacks lasting 4-72 hours
C	Headache having at least two of the following characteristics: 1. Unilateral location 2. Pulsating quality 3. Moderate or severe intensity 4. Aggravation by routine physical activity
D	During headache at least one of the following: 1. Nausea and/or vomiting 2. Photophobia *and* phonophobia
E	At least one of the following: 1. History and examination do not suggest any condition to which the headache may be secondary 2. History and/or examination do suggest such a condition, but investigation has excluded it 3. Such a condition exists, but migraine did not begin in temporal relation to it

bilateral, and gastrointestinal disturbance is more prominent.

Migraine with aura (classical migraine) is diagnosed relatively easily. The occurrence of aura preceding episodic headache clinches it, but beware of patients who bring "visual disturbance" into their accounts because of what they have read about migraine. Visual blurring and "spots" are *not* diagnostic. Transient hemianopic disturbances prior to headache, lasting 10-30 minutes (occasionally up to 1 hour), or a spreading scintillating scotoma (patients may draw a jagged crescent if asked), and other reversible focal neurological disturbances such as unilateral paraesthesiae of hand, arm or face (the leg is rarely affected), are symptoms of migraine aura. They occur in *some* attacks experienced by the roughly one third of migraine sufferers who have migraine with aura.

Rarely, migraine aura is prolonged and may persist after resolution of the headache. Even more rarely, a permanent deficit results. A very small number of families carry the dominant gene of ***familial hemiplegic migraine***. All of these cases should be referred to specialists for exclusion of other disease.

In some, particularly older patients, typical migrainous aura (usually visual) occurs without any further development of a migraine attack (***acephalalgic migraine***). Because transient ischaemic attack is in the differential diagnosis these cases should be referred to specialists.

Patients may, at different times, have attacks of migraine with and migraine without aura. They may, over a lifetime, change from a predominance of one subtype to the other.

"Diagnosis" by treatment

It is tempting to use the specific and effective anti-migraine drugs now available as a diagnostic test for migraine, a condition where an empirical approach to management ("Try this and see how it works") is not always unreasonable. However, sumatriptan and drugs of its class are at best effective in three quarters of attacks, so as a diagnostic test they have rather low sensitivity. Furthermore, tension-type headache can sometimes respond to triptans, so they are not totally specific. This approach is likely to mislead.

4.3 Tension-type headache (TTH)

More than one headache type goes under this name, which replaces the terms "tension headache" and "muscle contraction headache". TTH may be stress-related or associated with functional or structural cervical or cranial musculoskeletal abnormality, and these aetiological factors are not mutually exclusive. Patients may admit or deny stress. Clinically, there are cases where stress is obvious and likely to be aetiologically implicated (often in headache that becomes worse during the day) and others where it is not apparent. Equally there are cases with musculoskeletal involvement evident in the history (or on examination) and others where this is not a factor.

Episodic tension-type headache occurs in attack-like episodes, as does migraine, with variable and often very low frequency and mostly short-lasting (no more than a few hours). Headache can be unilateral but is more often generalised, typically described as pressure or tightness, like a vice or tight band around the head and commonly spreading into or arising from the neck. It is rarely significantly disabling, and lacks the specific features and associated symptom complex of migraine (although mild nausea, photophobia and

exacerbation by movement are common to many headaches).

What causes people with TTH to consult doctors is that it is becoming frequent, in which case it may no longer be responding to painkillers. ***Chronic tension-type headache*** occurs by definition on >15 days a month, and may be daily. This condition *is* disabling.

Both migraine and TTH are aggravated by stress and, in practice, there are occasions when the distinction is not easily made. Especially in a patient with frequent headache (more than once a week) and difficulty in distinguishing between migraine and TTH, there may be a mixture of the two (so-called ***mixed headache***). Unless both conditions are recognised, management is unlikely to be successful.

4.4 Differential diagnosis

Headache in almost any site, but often posterior, may arise from functional or structural derangement of the neck (***cervicogenic headache***), precipitated or aggravated by particular neck movements or positioning and associated with altered neck posture, movement, muscle tone, contour and/or muscle tenderness.

Headache, whether episodic or chronic, should *not* be attributed to ***sinus disease*** in the absence of other symptoms suggestive of it. Chronic sinusitis is *not* a validated cause of headache unless there is an acute exacerbation[7]. ***Errors of refraction*** are widely *overestimated* as a cause of headache which, if it does occur, is mild, frontal and in the eyes themselves, and absent on waking[8]. Headache should *not* be considered secondary to conditions affecting the ***ears, temporomandibular joints*** or ***teeth*** unless other symptoms are indicative of these.

4.4.1 Chronic daily headache

Chronic daily headache (CDH) is a *descriptive*, *not diagnostic*, term for headache occurring over weeks or longer on more days than not, or for more than 50% of the time. It has been estimated to be present in up to 4% of the entire population and accounts for up to 40% of referrals to special headache clinics. It costs the UK economy in lost working time up to £1 billion per year, yet it is very poorly characterised.

Headaches occurring every day are not migraine (or, at least, most of them are not migraine). Nosologically, CDH includes chronic tension-type headache as well as several other distinct entities, some of which are secondary headaches. Amongst these are headache with depression and cervicogenic headache (see above). However, there is growing evidence that a common cause of CDH is over-frequent consumption of medication used to treat acute headache.

4.4.2 Medication overuse headache (MOH)

This term has displaced the pejorative alternatives of drug, analgesic or medication *abuse* or *misuse* headache. It is estimated that 1 in 50 people suffer from MOH.

Headache secondary to overuse of medication intended for the treatment of headache was first noted with phenacetin. It became more apparent in patients overusing ergotamine prescribed, rightly or wrongly, for migraine. Ergot is very slowly eliminated from the body,

so is readily accumulated if taken 3 times a week or more frequently, and produces what ought to be a readily recognised withdrawal syndrome of sick headache. The patient, however, might reasonably mistake this for recurrent migraine. Continued repeated use typically leads to ever shortening periods between headache recurrence and medication intake until both are daily. The patient claims (rightly) that only further doses of ergotamine bring relief.

Fortunately chronic ergotamine intoxication, a potentially serious condition, is less seen nowadays. But it is becoming increasingly clear that a related syndrome occurs with the 5-$HT_{1B/1D}$ agonists (triptans), all of which are known to be associated with headache recurrence by a mechanism not yet clear. The high cost of these drugs has stood in the way of their over-frequent use but, nonetheless, cases are occurring.

What is recognised more and more is that MOH results also, and more commonly, from chronic overuse of analgesics to treat headache. Aspirin and paracetamol (particularly in combination with codeine) and probably NSAIDs are all causally associated with this condition. Whilst the mechanism is unclear, it is different from that of ergotamine intoxication and probably involves changes in neural pain pathways. Consequently, it may take a long time (weeks to months) for the headache to resolve after withdrawal.

MOH must *not* be diagnosed as "refractory migraine" and prophylactic medication added, which can only aggravate the situation. Any patient complaining of CDH should give a detailed account of medication use (including, and particularly, OTC medications). If they cannot, or are suspected of having unreliable recall, they should keep a prospective diary over a few weeks. Some patients dissemble, and need an understanding approach if a practice of which they may be ashamed is to be brought into the open.

It is very difficult to diagnose any other headache in the presence of MOH. This condition must be detected and managed, lest there be some other condition lurking beneath.

4.4.3 Cluster headache (CH) and related conditions

There is another group of disorders where daily occurrence of headache (often several attacks daily) is usual. The most common is cluster headache (formerly known as migrainous neuralgia).

CH affects mostly men (male to female ratio about 6:1) in their 20s or older (very rarely children) and very often smokers. The condition has its name because, typically (although there is a less common chronic subtype), headaches occur in bouts, once a year or two years for 6-12 weeks, often at the same time each year.

The pain of CH is intense (probably as severe as that of renal colic) and strictly unilateral. Although most often focussed in one or other eye, it can spread over a larger area of the head which sometimes misleads the diagnosis. There may, also, be a continuous background headache. The other features should leave no diagnostic doubt, although unusual patterns do occur, especially in women. Typically CH occurs daily, at a similar time each day, and usually but far from always at night, 1-2 hours after falling asleep. The wakened patient, unable to stay in bed, agitatedly paces the room, even going outdoors. He may beat his head on the wall or floor until the pain diminishes, usually after 30-60 minutes. The associated autonomic features of ipsilateral conjunctival injection and lacrimation, rhinorrhoea or nasal

blockage, and ptosis as the most obvious feature of a partial Horner's syndrome, may not all be present but almost invariably at least one or two secure the diagnosis. (There are other rare causes of painful Horner's syndrome; referral to specialists is appropriate where doubt occurs.)

4.4.4 Undiagnosed headache

A small minority of headaches do not meet recognised criteria and cannot reliably be diagnosed.

4.5 Physical examination of headache patients

All of the headaches so far discussed are diagnosed solely on history, with signs present in cluster headache patients if seen during attacks (occasionally, ptosis may persist between). The purpose of physical examination is sometimes debated but, for reasons given below, the optic fundi should always be examined during the diagnostic consultation. Blood pressure measurement is recommended. *In children*, some paediatricians recommend that head circumference is measured at the diagnostic visit, and plotted on a centile chart.

Examination of the head and neck for muscle tenderness (generalised, and tender "nodules"), stiffness, limitation in range of movement and crepitation is often revealing, especially in TTH. Positive findings may suggest a need for physical forms of treatment but not necessarily headache causation. It is uncertain whether routine examination of the jaw and bite contribute to headache diagnosis but may reveal incidental abnormalities.

In most people with migraine and in some with TTH, especially the chronic form, *reassurance* is very much part of successful management. The physical examination adds to the perceived value of reassurance and, within limits, the more thorough the examination the better. The time spent will likely be saved several times over, obviating many future consultations called for by a still worried patient.

In patients seen in specialist clinics, fewer than 1% have had headaches secondary to an intracranial lesion and, in published series, all had physical signs of it. This reinforces the importance of physical examination in diagnosing serious causes of headache such as tumour (see 5.0), although the history would probably be revealing in these cases.

4.6 Investigation of headache patients

Investigations, including cranial CT, do not contribute to the diagnosis of migraine or of TTH. They are indicated *only* when history or examination suggest headache may be secondary to some other condition. Cervical spine x-rays are indicated when neck signs suggest origin from the neck, although they may not reveal a treatable condition.

Eye tests by an ophthalmic optician are unlikely to contribute to headache diagnosis, although many patients believe they will.

4.7 Conclusion

The great frequency with which complaints of headache are encountered in clinical practice coupled with a very low relative incidence of serious causes (see 5.0) makes it difficult to

maintain an appropriate level of suspicion. If headache is approached with a standard operating procedure that supplements history with fundoscopic examination, brief but comprehensive neurological examination (which repays the time spent through its therapeutic value) and the use of diaries to record headaches, associated symptoms and medication use, and an awareness of the few important serious causes, errors should be avoided.

The greatest clinical difficulty, usually, is in distinguishing between migraine and TTH, which may coexist. The real concern, on the other hand, is that so much headache is iatrogenic. Many misused drugs are bought OTC. Failure to discover this in the history results in inappropriate treatment.

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5.0 SERIOUS CAUSES OF HEADACHE

Non-specialists may worry that these are in the differential diagnosis of migraine. Whereas *new or recently changed headache calls for especially careful assessment*, the reality is that intracranial lesions (tumours, subarachnoid haemorrhage, meningitis) give rise to histories that should bring them to mind. Physical signs should then be elicited leading to appropriate investigation or referral.

Rarely do ***intracranial tumours*** produce headache until quite large. Raised intracranial pressure is apparent in the history. Epilepsy is a cardinal symptom of intracerebral space occupying lesions, and loss of consciousness should be viewed very seriously. In all likelihood, focal neurological signs are present. Problems are more likely to occur with slowly growing tumours, especially those in neurologically "silent" areas of the frontal lobes. Subtle personality change may result in treatment for depression, with headache attributed to it. Investigation may be prompted eventually by non-response to treatment, but otherwise some of these can be very difficult to pick up, whilst their infrequency does not justify routine brain scanning. *Fundoscopic examination is mandatory* at first presentation with headache, and it is always worthwhile to repeat it during follow-up.

The signs of fever and neck stiffness usually accompanying ***meningitis***, in an obviously ill patient, demand urgent referral to specialist care. Headache may be generalised or frontal, perhaps radiating to the neck, and accompanied later by nausea and disturbed consciousness.

The clinical diagnosis of ***subarachnoid haemorrhage*** (SAH) is often straightforward, although the headache is not always of sudden onset, and neck stiffness may take some hours to develop. The headache of SAH is often described as the worst ever, but some patients are inclined to use such descriptive terms of migraine, rather devaluing them as diagnostic indicators. Even "explosive" features can occur with migraine (so-called "thunderclap headache"). Nevertheless, unless there is a clear history of uncomplicated headaches from which the present one is not particularly different, these characteristics indicate the need for investigation by brain imaging, then CSF examination. The serious consequences of missing SAH call for a low threshold of suspicion. In the elderly particularly, classical symptoms and signs may be absent.

New headache in *any patient over 50 years of age* should raise the suspicion of ***temporal arteritis*** (TA). Headache is the most constant feature of TA, often with marked scalp

tenderness, these two symptoms causing the patient to seek a consultation. The headache is persistent but often worse at night and can be severe, in a patient who does not feel entirely well. Jaw claudication is so suggestive that, in its presence, the diagnosis is TA until proved otherwise. Whilst the temporal artery may be inflamed, and tender, tortuous and thickened to palpation, this is an unreliable sign. Unfortunately, the ESR can equally be an unreliable investigation, since it may be normal, or may be raised in the elderly for other reasons. Temporal artery biopsy is usually necessary to secure the diagnosis. The dilemma is that treatment may be long-term and toxic (steroids need to be given in high dosage), and is not to be undertaken without very good reason and not as a diagnostic test.

Non-specific headache can be a symptom of ***primary angle-closure glaucoma*** (PACG). This is rare before middle age, when its prevalence is close to 1:1,000. Family history, female gender and hypermetropia are recognised risk factors[9]. PACG may present dramatically with acute ocular hypertension, a unilateral painful red eye with the pupil mid-dilated and fixed, associated nausea and vomiting and, essentially, impaired vision. In other cases, headache or eye pain may be episodic and mild, with the diagnosis of PACG suggested if the patient reports coloured haloes around lights[10]. The diagnosis of PACG is confirmed by skilled slit-lamp examination and gonioscopy. Glaucoma should not to be missed and should prompt immediate referral.

A rare cause of headache, usually in obese young women, is ***idiopathic intracranial hypertension*** (formerly termed benign intracranial hypertension or pseudotumor cerebri). This may not readily be diagnosed on history alone, though this may suggest raised intracranial pressure. The condition is not common but it should always be in the physician's mind. The physical sign of papilloedema indicates the diagnosis in adults, but is not seen invariably in children with the condition. It is confirmed by CSF pressure measurement.

In 1996/97, 21 people died in UK and a further 90 suffered ill-health from ***carbon monoxide (C0) poisoning***[11]. The condition is therefore uncommon but potentially fatal, and avoidable. The symptoms of subacute CO poisoning include headaches, nausea, vomiting, giddiness, muscular weakness, dimness of vision and double vision. Measurement of blood carboxyhaemoglobin concentration shortly after exposure confirms the diagnosis. In suspected cases with recurrent illness, domestic gas appliances should be checked. Gas flames should burn blue, not yellow or orange.

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6.0 MANAGEMENT OF MIGRAINE

6.1 Objectives of management

Cure is not a realistic aim and patients need to understand this. On the other hand, there is evidence that many migraine sufferers have unduly low expectations of what is achievable through optimum management. In the past, physicians' attitudes have reinforced this. The shared objective should be control of symptoms so that the effect of the illness on a patient's life and lifestyle is the least it can be.

6.2 Basic principles

To this end, patients should work through the treatment options in a rational order, and

continue to do so *until it is certain they have found what suits them best*. In applying the following guidelines, ***follow-up*** should ensure optimum treatment has been established. Denial of best available treatment is difficult to justify for patients generally and, therefore, for individual patients. Unnecessary pain and disability are the result. In addition, increasingly it is being demonstrated that *under-treatment is not cost-effective*: sufferers' and their carers' lost time is expensive, as are repeated consultations in the search for better therapy.

It should be remembered that ***needs may change***. Migraine typically varies with time, and concomitant illness including other headaches may develop.

Children often respond to conservative management, which should therefore be the initial approach. Reassurance of parents is an important aspect of treating children. Otherwise, most can be managed as adults, with allowance for different symptom presentation and perhaps different dose-requirements and contraindications.

Children with troublesome migraine not responding to trigger avoidance and simple analgesics taken early with or without antiemetics *should be seen by a paediatrician with an interest in headache* since their management is family-based.

In ***adults***, there are four elements to good migraine management:

- correct and timely diagnosis;
- explanation and reassurance;
- predisposer/trigger identification and avoidance;
- intervention (drug or non-drug).

Diagnosis has been covered already. ***Explanation*** keeps patients' expectations realistic, and fosters appropriate use of therapy. ***Reassurance*** following diagnosis and explanation is all some patients need. In any event the effect of reassurance is added to that of any therapeutic intervention.

6.3 Predisposing and trigger factors

Predisposing factors should be distinguished from precipitating or trigger factors (see 6.3.2). Certain predisposing factors are well recognised. They are not always avoidable but may be treatable (see table).

6.3.1 Predisposing factor	***Management summary***
Stress	Lifestyle change; stress reduction/coping strategies (see 6.6.2)
Depression/anxiety	Specific therapy
Menstruation	See 6.5.6
Menopause	Hormone replacement therapy (see 6.5.9)
Head or neck trauma	Physiotherapy (see 6.6.1)

6.3.2 Trigger factors

Trigger factors are important in occasional patients but generally are less so than commonly supposed. Dietary sensitivities affect, at most, 20% of migraine sufferers. Many attacks have no obvious trigger and, again, those that are identified are not always avoidable (see table).

6.3.2 Trigger factor	***Management summary***
Relaxation after stress, especially at weekends or on holiday	Stress avoidance; lifestyle change (see 6.6.2)
Other change in habit: missing meals; missing sleep; lying in late; long distance travel	Avoidance if possible; avoidance of other cumulative triggers
Bright lights and loud noise (both perhaps stress-inducing)	Avoidance
Dietary: certain alcoholic drinks; some cheeses; citrus fruits; possibly chocolate	Avoidance if indicated (see 6.3.3)
Strenuous unaccustomed exercise	Keeping fit / avoidance
Menstruation	See 6.5.6

Diaries (see 6.3.3) may be useful in detecting triggers but the process is complicated as triggers appear to be cumulative, jointly contributing to a "threshold" above which attacks are initiated. Too much effort in seeking triggers causes introspection and may be counter-productive. Enforced lifestyle change is inappropriate management if it adversely affects quality of life by more than is offset by improvement in migraine. Simple advice to patients is to minimise potential triggers: at stressful times eat regularly, for example.

Anxiety and emotion. Most migraineurs cope well with stresses but many have attacks when they relax (*eg, weekend headache*). Stress may induce other triggers such as missed meals, poor sleep and muscle tension. Although stress may be unavoidable, its existence may make it more important to avoid other triggers.

Missing breakfast may trigger attacks late morning, ***missed lunch*** may induce or provoke attacks in the afternoon. When attacks are present on waking it is worthwhile considering the time of the ***evening meal*** — often, in these circumstances, eaten early.

A ***food*** is a trigger when: a) migraine onset occurs within *6 hours of intake*; b) the effect is reasonably *reproducible*; c) withdrawal leads to *improvement*. Most migraineurs can eat whatever they like as long as they keep up with their energy demands. A few susceptible individuals note a definite relationship between the consumption of certain foods, particularly alcohol, and the onset of migraine. The foods may not always trigger an attack but tip the balance when the person is vulnerable. Dietary triggers, if real, become obvious to patients and are usefully avoided. A suspected food should be excluded for a few weeks. When many foods are suspect, supervision by a dietician is advisable as elimination diets can result in malnutrition. Excluded foods should be reintroduced if there is no significant improvement. There is no case for blanket avoidance of cheese, chocolate or other foods, nor for other dietary manipulation.

Cravings for sweet or savoury foods are probably ***prodromal symptoms*** heralding the headache, not triggers.

Food allergy (*ie*, an immunological process) has ***no part*** in the causation of migraine.

Too much and too little sleep can both play a role. Sleepless nights result in over-tiredness which triggers migraine. Conversely, sleeping in for even half an hour longer than usual, often at the weekend, can trigger migraine. In both cases, the *cause* of the altered sleep pattern (stress, relaxation) may be the true trigger.

Hormonal changes. Migraine is three times more common in women than in men. Attacks in most women start around puberty and continue until the menopause, with respites during pregnancy. Many women are far more susceptible to migraine at the time of their periods and a small percentage have attacks exclusively at or near (± 48 hr) onset of menstruation (***menstrual migraine***). Women with obvious hormonal triggers may benefit from specific intervention (see 6.5.6).

Strenuous exercise can precipitate an attack in a person unaccustomed to it. This puts many people off exercise when in fact regular exercise may help prevent migraine attacks. This is because it improves blood sugar balance, helps breathing, stimulates the body to release its own natural pain killers and promotes a general sense of well-being.

6.3.3 The trigger diary

When migraine attacks are frequent, a trigger diary may be useful in addition to the attack diary. Patients can be given a list of common triggers and record those present each day whether they have a migraine attack or not. The daily trigger diary and attack diary are best reviewed after at least five attacks. The information in each is compared for coincidence of (multiple) triggers with attacks.

6.4 Drug intervention (acute)

The evidence-base for many acute anti-migraine drugs is poor. For aspirin/metoclopramide combination the evidence is better[12] and for the triptans it is good but not perfect (much is unpublished in peer-reviewed manuscripts). Whilst, logically, drug treatment should be selected for each patient according to his or her need and expected response to it ("stratified management"), little basis other than guesswork presently exists for achieving this. In particular, the superiority of triptans over other treatments can be questioned[13].

Consequently, there is a ***treatment ladder***, and *all patients should start on the first step of this ladder* ("stepped management"). Stepped management is *not* contrary to the principle of individualised care: on the contrary, it is a reliable strategy for achieving it based on evidence manifestly applicable to the individual patient. Speed is sacrificed only if a better alternative exists, for which a search continues. It is suggested, but not an invariable rule, that ***failure on three occasions*** should be the minimum criterion for progressing from each step to the next. Statistically, three consecutive failures is still compatible with an 80% success rate but, in practice, few patients will persist.

People who recognise attacks of more than one sort, or of differing severity, may apply different steps for each accordingly.

As a general rule, all acute drug therapy should be combined with ***rest and sleep*** (promoted if necessary with ***temazepam*** or ***zolpidem***). However, the central objective of treatment for some patients is to be able to carry on with their activities and, for these, this recommendation is inappropriate.

6.4.1 Step one

1a) ***Simple oral analgesia (aspirin 900mg*** or ***paracetamol 1000mg)*** or ***ibuprofen 400mg***, preferably in ***soluble*** formulation, or over-the-counter migraine preparations including ***Midrid*** (paracetamol 325mg plus isometheptene 65mg per tablet), initial dose 2 tablets, but *not* codeine or dihydrocodeine (see 6.4.11).

All of these are best taken ***early*** in the attack when absorption may be least inhibited by gastric stasis.

1b) ***Simple oral analgesia*** as above, or prescription-only non-steroidal anti-inflammatory drugs - ***tolfenamic acid rapid release 200mg*** (repeated if necessary after 1-2 hours), ***naproxen 500mg*** or ***diclofenac 50-75mg*** in *non-delayed release formulations* - orally combined with ***metoclopramide 10mg*** or ***domperidone 20mg*** (these antiemetics promote gastric emptying). ***Paramax sachets*** (paracetamol 500mg plus metoclopramide 5mg per sachet) are a convenient preparation whilst ***MigraMax*** (lysine acetylsalicylate 1620 mg [equivalent to aspirin 900mg] plus metoclopramide 10mg per sachet) is newly marketed in the UK; there is no other way at present to give metoclopramide in a soluble oral formulation. ***Paramax tablets*** and ***Domperamol*** (paracetamol 500mg plus domperidone 10mg per tablet) are not soluble.

***Contraindications to step one*:**

In adults there are none, unless it has clearly failed before. There may be specific contraindications to aspirin. In children under 12 years aspirin should be avoided and metoclopramide is not recommended for children or adolescents.

6.4.2 Step two

Use parenteral routes: ***diclofenac suppositories 100mg*** for pain plus ***domperidone suppositories 30mg*** (if needed) for nausea/vomiting.

***Contraindications to step two*:**

Peptic ulcer (misoprostol 800µ g or omeprazole 20-40 mg daily may give limited gastroduodenal protection[14]) or lower bowel disease. The occurrence of diarrhoea during acute migraine may prevent effective use. Some patients will not accept suppositories.

6.4.3 Step three

Specific anti-migraine drugs (triptans). The marketed drugs of this class differ in ways that might rationally suggest one rather than another for a particular patient. However, there are unpredictable individual variations in response to different triptans. Many patients reasonably wish to try each and judge for themselves, and this should be encouraged.

Unlike symptomatic therapy, *triptans should not be taken too early*. They appear to be ineffective if administered before the headache has developed.

Where triptans are taken orally, concomitant administration of metoclopramide or domperidone is suggested on *theoretical* grounds: there is no formal evidence to support their use.

All triptans are associated with return of symptoms within 24 hours in 20-40% of patients who have initially responded (*recurrence*). This is a troublesome limitation.

Sumatriptan was launched in 1991. In 9 years' clinical experience, usage exceeds 250 million doses world-wide. The ***50mg oral*** formulation is appropriate for first use of a triptan, or for patients troubled by drowsiness or other central nervous system side-effects of zolmitriptan. Sumatriptan ***100mg orally*** or ***20mg nasal spray*** (according to each patient's preference) are available if a more potent treatment is required. If a *rapid response is important above all*, or if *vomiting precludes oral therapy*, ***6mg subcutaneously*** is recommended (not nasal spray, since its bioavailability depends in part on ingestion).

Zolmitriptan 2.5mg orally is equally appropriate for first use of a triptan. It has better and more predictable absorption, and is less expensive, than oral sumatriptan. It has a wider therapeutic margin than naratriptan or sumatriptan and a second dose may be taken after two hours if needed. If this is usually the case, a first dose of ***5mg*** is allowable. Asthenia and somnolence tend to limit the dose. Zolmitriptan is licensed for and may be useful in children aged 12 years or over, whereas other triptans appear to be ineffective in adolescents.

Rizatriptan 10mg orally and ***10mg MELT*** (rapidly dispersible wafer placed on the tongue) have been associated with rapid onset of effect in clinical trials, and recent comparative studies indicate superior efficacy to all other triptans currently available. Each formulation costs the same, marginally more than zolmitriptan or naratriptan. Increasing clinical experience of their use suggests rizatriptan is an alternative to sumatriptan 100mg when a more potent treatment is required. *Metabolism is affected by propranolol* and patients on this drug should take ***5mg orally***.

Naratriptan 2.5mg orally is well tolerated but its low potency and slow onset of effect limit its use in patients seeking a rapid response. It is equal in cost to zolmitriptan 2.5mg. It is recommended when side-effects to sumatriptan 50mg or zolmitriptan 2.5mg are troublesome. The evidence for less *recurrence* is not entirely convincing but, where recurrence of symptoms after initial efficacy is a particular problem (see 6.4.8), this drug may nonetheless be worth trying.

***Contraindications to step three*:**

a) Uncontrolled hypertension.

b) Risk factors for coronary heart disease or cerebrovascular disease: past history; signs; strong family history (the significance of this is age-related); advanced age. In case of uncertainty, cardiological referral and exercise ECG are recommended.

c) Children under 12 years:

no experience has been reported and neither safety nor efficacy are established.

***If step three fails*:** Review diagnosis. Review compliance and manner of use. Steps four and five *may* be worth trying. Consider prophylaxis (see 6.5).

6.4.4 Step four

Dihydroergotamine (DHE) nasal spray 1mg (and optionally 0.5-1mg after 15 min) or ***ergotamine 1-2mg*** preferably as suppository. The dose per suppository is 2mg and a half suppository is adequate for some people. These drugs may be tried if recurrence is a particular problem (see 6.4.8), but ***toxicity and misuse potential*** are impediments to their use, particularly in the case of ergotamine. DHE is costly compared with ergotamine and the spray device has to be assembled immediately before use and then expires after 8 hours.

***Contraindications to step four*:**

As for step three, with *greater caution* appropriate. DHE and ergotamine are *not options if triptans are contraindicated*.

DHE and ergotamine should not be taken *concomitantly* with any triptan, but are probably safe after 12 hours (see 6.4.7). They should *not* be used by patients on beta-blockers, which impair nutrient flow to the skin: cases of digital gangrene have been reported. They are not advised for children.

6.4.5 Step five

There is no formal evidence for combinations, but ***steps one + three*** may be worth trying followed by ***steps two + three***. Though not common practice, ***self-injected diclofenac 75mg*** may be tried. It is difficult: the intramuscular volume is 3ml, requiring two injection sites.

6.4.6 Emergency treatment of patients at home

This usually falls to general practitioners. If an effective therapy has not been established previously, the options are: ***intramuscular diclofenac 75mg*** (not pethidine: see 6.4.12) and/or ***intramuscular chlorpromazine 25-50mg*** (potent antiemetic and sedative). Early follow-up is recommended.

6.4.7 Treatment of recurrence within the same attack after initial efficacy

Symptomatic medications (steps one and two) may be repeated within their dosage limitations.

In the case of *triptans*, there is evidence of efficacy of a second dose for recurrence but no evidence that it is the most appropriate treatment. There is informal evidence that *repeated dosing with these drugs quickly gives rise to repeated rebound, perhaps over several days*. Instead ***diclofenac*** may be tried, perhaps pre-emptively where recurrence is usual and expected. Further experience is needed with ***tolfenamic acid***. ***DHE*** or ***ergotamine*** may be useful for this purpose but safety and efficacy have not been formally established; they should not be used within 12 hours after any triptan.

6.4.8 Patients who consistently experience recurrence

There is some evidence that this occurs more in those whose untreated attacks last longer than 24 hours.

Naratriptan may be the triptan of choice (see 6.4.3). ***Ergotamine*** has a prolonged duration of action and trials in which it has been used as a comparator suggest that it is associated with significantly less recurrence.

6.4.9 "Long-duration migraine"

Migraine lasting longer than 3 days is uncommon. Apparently long-duration attacks may be migraine with a superseding tension-type headache for which ***naproxen*** or ***diclofenac*** are preferable to specific anti-migraine drugs.

Status migrainosus is extremely rare. Multiple recurrences with repeated doses of a triptan are now a well-recognised complication, and correct management is withdrawal of the triptan. ***Diclofenac*** should be used until symptoms settle. Patients who show susceptibility to this problem should try ***ergotamine*** instead, but be very cautious in repeating the dose.

6.4.10 Slowly developing migraine

Some patients develop attacks slowly and are initially uncertain whether a headache is migrainous or not. If treatment is required at this stage, ***simple analgesics*** are recommended and may prevent further development. ***Triptans*** should not be used, if at all, until it is certain that the headache is migrainous (there is some evidence that delayed use of triptans does not significantly impair their efficacy).

6.4.11 Migraine in pregnancy and lactation

Paracetamol in moderation is safe throughout pregnancy and breastfeeding. ***Aspirin*** is safe except near to term. For nausea, ***prochlorperazine*** is unlikely to cause harm throughout pregnancy and lactation. ***Metoclopramide*** and ***domperidone*** are probably safe in second and third trimesters.

6.4.12 Drugs to avoid in acute intervention

Opiates or opiate derivatives (including morphine, pethidine, dextropropoxyphene, codeine, dihydrocodeine). These drugs increase nausea, promote systemic shut-down and have addictive potential. ***Buprenorphine*** is particularly emetic. ***Codeine*** and ***dihydrocodeine*** in combination preparations are used extensively but without evidence of added benefit; they are frequently implicated in medication overuse headache.

6.4.13 Limits to acute therapy: frequency of use

Over-frequent use of drugs for acute intervention may be one criterion for prophylaxis (see below). On a regular basis:

a) Use on ***more than two days per week*** is clearly ***inappropriate*** for migraine (though not necessarily unsafe).

b) Use on ***more than one day per week*** calls for close enquiry into how it is used, and review of the diagnosis.

6.5 Drug intervention (prophylactic)

When indicated, prophylactic therapy is used ***in addition to***, not instead of, acute therapy. The evidence-base for all prophylactic anti-migraine drugs is poor except, possibly, for valproate. The following recommendations are based on expert clinical experience.

6.5.1 Indications for prophylaxis

Prophylaxis is used to reduce the ***number*** of attacks in circumstances when acute therapy, used appropriately, gives ***inadequate symptom control***. The judge of this is usually the patient. *In children*, an index of this that might be considered is frequency of absence from school because of migraine.

Over-frequent use of acute therapy is also a criterion for migraine prophylaxis, but prophylactic drugs are inappropriate and will be ineffective for medication overuse headache. This condition must first be excluded.

There are no sound criteria for preferring one prophylactic drug to another except those of ***comorbidity*** or ***contraindications*** (including risks in pregnancy). However, there is good evidence that poor compliance is a major factor impairing efficacy of these drugs, and *once-daily dosing is preferable*.

***Duration of use*:**

Migraine is cyclical: treatment is required for periods of exacerbation. Prophylactic drugs that are ***effective*** should be continued for ***4-6 months*** then withdrawn (stopped abruptly or tapered) to establish continued need. Uninterrupted use over a year or longer is rarely appropriate. Prophylactic drugs that are apparently ***not effective*** should not be discontinued too soon or patients may be labelled non-responders prematurely (eventually, perhaps, to all drugs). There is no good guide for what should be the minimum period since efficacy may be slow to develop; ***3-4 weeks*** may be the ***minimum***, and ***3 cycles*** in the case of specific therapy for hormone-related migraine (see 6.3.5). Patients may well decide for themselves, but should be discouraged from stopping too soon unless they have unacceptable side-effects.

6.5.2 First-line prophylactic drugs

Beta-adrenergic blockers without partial agonism, if not contraindicated by asthma, heart failure, peripheral vascular disease or depression. There is reasonable clinical trials evidence of efficacy of ***propranolol***, ***metoprolol*** and ***atenolol***[15]. Cardioselectivity and hydrophilicity both improve the side-effect profile and are to be preferred. Once-daily dosing is associated with significantly better compliance. On these grounds, ***bisoprolol 5-10mg od*** may be the beta-blocker of choice although better evidence of efficacy is needed to support its routine use. ***Atenolol 25-100mg bd*** is meanwhile ***first-line***, with better side-effect profile than ***propranolol LA 80mg od-160mg bd***. The dose of each should, usually, start low in the suggested range and be increased through it in the absence of troublesome side-effects.

Sodium valproate 0.6-2.5g daily is well tolerated and there is clinical trials evidence of

efficacy[16]. It is not safe during pregnancy and therefore contraindicated when pregnancy may occur. Sodium valproate is not generally recommended for children (but see 6.5.5).

Pizotifen 1.5mg daily is sedative and should be taken at bedtime. This side-effect can sometimes be avoided by titrating upward from a starting dose of 0.5mg. Pizotifen enhances appetite, with weight gain that many sufferers (the majority of whom are women) will not accept. Clinical trials evidence of efficacy is limited. There is no evidence of greater efficacy from higher doses.

Amitriptyline 10-150mg daily, usually at night, is ***first-line prophylactic*** when (a) migraine coexists with tension-type headache (see 6.7); (b) there is associated depression; or (c) there is disturbed sleep. It may be used concomitantly with a beta-blocker as ***second-line***. Clinical trials evidence of efficacy is limited. It is wise to explain the choice of this drug to patients who do not consider themselves depressed or they may reject it. The starting dose of 10-25mg daily is increased until (a) there is efficacy; (b) there are unacceptable side-effects; or (c) 150mg is reached. Usual side-effects are sedation, dry mouth, urinary retention (in men).

Desipramine, ***nortriptyline*** and ***protriptyline*** are less sedative but there is no formal evidence of efficacy of these or other tricyclics.

6.5.3 Second-line prophylactic drugs

Methysergide 1-2mg tds is generally considered (on limited evidence) to be the most effective prophylactic but is held in reserve because of its association with retroperitoneal fibrosis. The drug seems not to have this side-effect in courses of less than 6 months (see 6.5.1).

Beta-blocker and amitriptyline together: see above. Synergistic effect is claimed for this combination without formal evidence. It is logical if there may be a depressive trait.

6.5.4 Other drugs used in prophylaxis but with limited efficacy

Calcium channel antagonists are of uncertain value. ***Flunarizine*** is not available in the UK. ***Verapamil modified release, 120-240mg bd*** is well tolerated, with headache sometimes a side-effect. Clinical trials evidence of efficacy is limited.

Selective serotonin reuptake inhibitors are second-line to tricyclics. ***Fluoxetine 20mg alter die to 40mg od*** is best studied. Clinical trials evidence of efficacy against migraine is inconclusive (against depression, its efficacy in higher doses is established).

6.5.5 Prophylaxis in children

There is little formal evidence of efficacy of prophylactic drugs in children. For the few children who need prophylaxis, ***beta-blockers*** or ***pizotifen*** (available as an elixir) may be tried. Some paediatricians use ***sodium valproate*** or ***amitriptyline*** with success.

Dosage is adjusted according to age.

6.5.6 Prophylaxis for hormone-related migraine

An effect of hormones on migraine is common, and greater for migraine without aura[17]. Empirical evidence suggests oestrogen withdrawal triggers migraine in some women[18].

Menstrual migraine, defined as attacks of migraine without aura that occur regularly on day 1 of menstruation ± 2 days and at no other time, is rare[19]. Correct diagnosis of menstrual migraine is essential for successful hormonal management. The diagnosis is clinical and confirmed by diary card evidence over three months.

Depending on need for contraception, several options can be tried in whatever order seems appropriate. Prophylaxis should be tried for a *minimum of three cycles* at maximum dose before it is deemed ineffective.

A) Non-hormonal prophylaxis does not depend on regular menstruation. ***Mefenamic acid 500mg tds-qds*** can be given from the onset of menstruation until the last day of bleeding. It is recommended as *first-line in migraine occurring with menorrhagia and/or dysmenorrhoea*.

B) Hormones for menstrual migraine are *supplements*: if the women has an intact uterus and is menstruating regularly, no progestogens are necessary. ***Transdermal oestrogen 100µg*** is used from ***3 days before onset of menses for 7 days***. When this is effective but not well tolerated, ***50µg*** may be tried. Alternatively, ***oestradiol 1.5mg in 2.5g gel*** is applied daily from ***day -3 for 7 days***. The gel produces higher, more stable levels of oestrogen and may be better.

C) ***Combined oral contraceptives*** (COCs) (also see 6.5.7) and ***injectable depot progestogens*** inhibit the ovarian cycle. *Migraine in the pill-free interval* is most notable with high-progestogen contraceptives[20] and can often be resolved by changing to a more oestrogen-dominant pill. Taking the pill continuously for 9 weeks rather than 3 ("tricycling"), followed by the usual 7-day pill-free interval, results in 5 rather than 13 withdrawal bleeds per year and is an alternative approach. ***Oral progestogen-only contraception*** does *not* inhibit ovulation.

6.5.7 Migraine and hormonal contraception

Headache is a common side-effect of COCs and many women report onset of migraine after starting them. Others report improvement of pre-existing migraine[21]. There is concern that migraine and COCs are both *independent risk factors for stroke in young women*, in the latter case related to the ethinyloestradiol component. This has led to the development of opinion-based recommendations for the use of COCs in migraineurs[22], although not all experts agree.

***Relative contraindications to ethinyloestradiol COCs*:**

a) Migraine with aura (experts disagree over whether this is an absolute contraindication).

b) Migraine without aura in the presence of *one or more* additional risk factors for stroke.

c) Migraine treated with *ergot derivatives* (but not triptans).

Progestogen-only contraception is *acceptable* with any type of migraine contraindicating

synthetic oestrogens. The progestogen-only pill has a higher failure rate but ***Depo-Provera*** and the ***Mirena intrauterine system*** both have lower failure rates than COCs. Women can switch immediately from COCs to progestogen-only contraception.

6.5.8 Migraine in pregnancy and lactation

Most women with migraine improve during pregnancy. If not, prophylactics should be restricted but, when necessary, ***propranolol*** has best evidence of safety during pregnancy and lactation[23]. Women should be counselled with regard to the relative risks and benefits.

6.5.9 Migraine and hormone replacement therapy (HRT)

Hormone replacement therapy is ***not*** contraindicated: there is no evidence that risk of stroke is elevated or reduced by the use of HRT in women with migraine, with or without aura. The menopause itself commonly exacerbates migraine and symptoms can be relieved with optimised replacement therapy. Nevertheless, in practice, a number of women on HRT do find their migraine becomes worse. This is often no more than a problem of formulation or dosage.

Adequate, stable levels of oestrogen are best provided by percutaneous or transdermal delivery systems used continuously. Headache associated with cyclical progestogens may be controlled by changing the type of progestogen, using transdermal progestogens or the Mirena intrauterine system, or changing to progesterone (micronized or suppository).

After hysterectomy, oestrogen implants are an option.

6.5.10 Drugs to avoid in prophylactic intervention

Clonidine has no proven efficacy against migraine. Its use is not recommended. There is no or insufficient evidence to support the use of ***other anti-epileptic drugs***.

Oral contraceptives (see 6.5.7) may or may not exacerbate migraine and should be changed or discontinued if they do; they are contraindicated if exacerbation includes the development of focal neurological signs.

6.5.11 If prophylaxis fails

Review diagnosis. Review compliance (often poor) and concordance (often *very* poor, especially with multiple daily doses). Review other medication, especially for medication overuse. Consider combinations (no formal evidence for any). If prophylaxis still fails to have measurable benefit, *discontinue it*.

6.6 Non-drug intervention

6.6.1 Physical therapy

Improving ***physical fitness*** is believed to reduce susceptibility to migraine.

Physical therapy may be helpful where a specific indication (*eg*, neck dysfunction) exists. In other cases it may be useful as adjunctive therapy. A therapist with specific training is more

likely to achieve good results than a generalist.

Acupuncture is of uncertain benefit: better clinical trials are needed.

Dental treatment, including the fitting of splints or bite-raising appliances and other procedures to correct malocclusion, is of unproven benefit in migraine but occasional patients claim benefit. It may improve temporomandibular joint dysfunction and secondary head pain. The importance of ***bruxism*** in headache causation is undetermined.

6.6.2 Psychological therapy

Relaxation therapy, ***stress reduction*** and ***coping strategies*** are *first-line treatments* where a specific indication (*eg*, anxiety, stress) exists. In other cases they may be useful as adjunctive therapy. Their effect on migraine in these situations, particularly that of the simple device of relaxation tapes, needs formal evaluation. ***Yoga*** and ***meditation*** are said to enhance stress management and appeal to some people.

Biofeedback techniques have some support from clinical trials; being operator-dependent, they are difficult to standardise.

Hypnotherapy is of unproven value.

6.6.3 Herbals and homoeopathy

There is no basis at present for recommending either of these. The active ingredient of ***feverfew*** is sometimes claimed to be parthenolide but standardised formulations of this drug do not have proven efficacy. Other marketed preparations of feverfew are variable in what they contain. Furthermore, feverfew contains potential carcinogens; its toxicity is not well understood and its long-term effects are unknown. *It is particularly unsuitable for children*.

Homoeopathy appears to be of no value. Its basis calls for expert prescribing if it is to be used. There is ***no case*** for over-the-counter sales of homoeopathic remedies for migraine.

6.6.4 Other alternative remedies

Reflexology has no scientific basis but it may have placebo effect.

Many ***devices*** are on the market, some at considerable cost and promoted with specific but unsupportable claims of efficacy. "Testimonials" can be attributed to placebo effect and should be disregarded. Any of these that may have efficacy should be formally evaluated in clinical trials. Unless that has been done, and evidence of efficacy adduced, patients encouraged to buy them are done a disservice.

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7.0 MANAGEMENT OF TENSION-TYPE HEADACHE

7.1 Objectives of management

Episodic TTH is self-limiting, non-disabling, and rarely raises anxieties about its causation

or prognosis. Reassurance, if needed, and intermittent symptomatic treatment are often quite sufficient. Provided that patients are not at risk of escalating consumption, little more may need to be done.

Long-term remission is the objective of management of frequent episodic or chronic TTH. It is not always achievable, particularly in long-standing chronic TTH. In such cases, avoidance of aggravation by medication overuse remains important, as do recognition and appropriate treatment of contributory factors.

7.2 Basic principles

As with migraine, ***reassurance*** is important, often effective on its own, and should never be omitted.

Underlying contributory factors are of greater potential importance in TTH than in migraine. Effective treatment is likely to depend on successfully identifying these, particularly when headaches are frequent.

TTH may be stress-related or associated with functional or structural cervical or cranial musculoskeletal abnormality. These aetiological factors are not mutually exclusive. Clinically, there are cases where stress is obvious and likely to be aetiologically implicated and others where it is not apparent. Equally there are cases with musculoskeletal involvement evident in the history or on examination and others where this is not a factor.

The distinction between episodic and chronic TTH, based on frequency, is somewhat arbitrary. Nevertheless, it has practical importance for two reasons. One arises from the frequency of use of symptomatic medication, and at what level the potential long-term harm outweighs short-term benefit. ***Medication overuse*** must always be discovered and remedied. The second relates to likely comorbidity. ***Clinical depression*** must be diagnosed and treated appropriately. In the background of chronic TTH, either will defeat management unless recognised and adequately dealt with.

7.3 First measures

TTH is more common in sedentary people. Regular ***exercise*** is of general and potentially considerable benefit and always worth recommending.

Physiotherapy may be appropriate, and the treatment of choice, for musculoskeletal symptoms. A therapist with specific training is more likely to achieve good results than a generalist. Physiotherapy may include massage, mobilisation, manipulation and, particularly in those with sedentary lifestyles, correction of posture. Regular home exercises are often prescribed. Mobilisation and manipulation sometimes aggravate symptoms before they improve. Physiotherapy may help symptoms secondary to trauma such as whiplash injury but is less useful in degenerative disease of the neck. It is unlikely to be beneficial in stress-related illness for which ***lifestyle changes*** to reduce stress and ***relaxation therapy*** and ***cognitive training*** to develop stress-coping strategies are the mainstays of treatment. ***Yoga*** and ***meditation*** are said to enhance stress management and appeal to some people.

7.4 Drug therapy

This is of limited scope but effective nevertheless in some patients. Symptomatic treatment is appropriate for infrequent episodic TTH occurring on fewer than 2 days per week. Over-the-counter analgesics (***aspirin 600mg***, ***paracetamol 1000mg***, ***ibuprofen, 400mg***) are usually sufficient; other NSAIDs (***ketoprofen 25-50mg***, ***naproxen 250-500mg***) are sometimes indicated.

Codeine and ***dihydrocodeine*** should generally be *avoided* and there is no place for powerful analgesics. As the frequency of headaches increases, so does the risk of excessive medication use.

These treatments are *inappropriate* in chronic TTH, whether they appear to give short-term benefit or not. Nevertheless, a 3-week course of ***naproxen 250-500mg bd***, taken regularly, may break the cycle of frequently recurring or unremitting headaches and the habit of responding to pain with analgesics. If it fails, it should not be repeated.

Amitriptyline is otherwise the drug treatment of choice for frequently recurring episodic TTH or for chronic TTH. Its use in chronic pain syndromes is not dependent on its antidepressant activity. Clinical trials evidence does not establish how best to use this drug, or in what dose. Intolerance is relatively common but greatly reduced by starting at a low dose (***10-25mg at night***). Increments should be as soon as side-effects permit, perhaps of 25mg each 1-2 weeks and usually into the range ***75-150mg*** at night. Withdrawal may be attempted after improvement has been maintained for 4-6 months.

Failure of tricyclic therapy may be due to subtherapeutic dosage, insufficient duration of treatment or (commonly) non-compliance. Patients who are not informed that they are receiving medication often used as an anti-depressant, and told why, may default when they find out.

Some experts offer alternatives, *eg*, ***dothiepin***, if amitriptyline fails. ***Nortriptyline*** and ***protriptyline*** may be better tolerated but their usefulness is less certain. There is no evidence that ***SSRIs*** reduce headache in chronic TTH, though they may be indicated for underlying depression. ***Anxiolytics*** may be appropriate when specifically indicated but ***beta-blockers*** may promote depression whereas the high risk of dependence generally rules out prolonged use of ***benzodiazepines***.

7.5 If all else fails

Chronic TTH in particular is often refractory. Its association with personality factors and psychosocial dysfunction that militate against effective treatment is often suspected but not consistently demonstrated. Some of these patients end up in pain management clinics where ***cognitive therapies*** are more readily available and where non-specific therapies such as ***transcutaneous electrical nerve stimulation*** (TENS) may be offered.

The role of ***acupuncture*** is unproven but worth trying in the absence of other options. Detection of tender muscle nodules on palpation, with needling aimed at these, is said to offer a good prospect of at least limited success but evidence to support this is poor. As with physiotherapy, symptoms may at first be aggravated by acupuncture. It is sometimes claimed that early exacerbation is prognostic of later improvement.

Homoeopathy is of unknown value. Its basis calls for expert prescribing if it is to be used.

There is *no case* for over-the-counter sales of homoeopathic remedies for TTH.

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8.0 MANAGEMENT OF MIXED HEADACHE

Symptomatic medication should be restricted to no more than 2 days per week. Where migraine coexists with episodic tension-type headache and prophylaxis is considered, ***amitriptyline 10-150mg daily*** is the drug of choice (see 6.5.2 and 7.4). Some specialists are using ***sodium valproate 0.6-2.5g daily*** as an alternative.

Where migraine occurs in association with other, more troublesome headache (usually chronic tension-type headache or medication overuse headache; sometimes depressive headache), *that headache should be treated first*. Improvement in migraine often occurs concomitantly.

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9.0 COSTS OF IMPLEMENTING THESE GUIDELINES

It is predicted that fully implementing these guidelines will:

a. *improve* diagnosis, *reducing* the rate of inappropriate treatment;
b. *increase* the number of consultations per patient initially, to find the best treatment for each individual;
c. *increase* the number of patients with migraine eventually using triptans;
d. *reduce* misuse of medication, including triptans, and *reduce* iatrogenic illness;
e. *improve* the overall effectiveness of management;
f. *reduce* the need for specialist referral;
g. *reduce* the overall number of consultations eventually, as patients' symptoms and disability are better controlled;
h. *raise* expectations, especially amongst those with migraine, and lead to *more* patients consulting;
i. *reduce* the overall burden of illness, with *savings* elsewhere.

Whereas some of these outcomes will increase NHS costs, at least initially, others will reduce them. Management costs *may* rise overall, but there is no good financial argument for treating migraine suboptimally. Whilst evidence is accruing that this is not cost-effective, figures are not yet available to show the levels of savings overall that better management can achieve. Troublesome and inadequately managed TTH is also costly. Whilst not all cases can be treated effectively, there is considerable potential for making things worse by inappropriate management. Again, it is not known what savings might result from better care. It should be a priority to find out.

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10.0 AUDIT

Audit should aim to measure headache burden in the target population and its diminution over time after implementation of these guidelines. Measurements may be made in random

samples of patients large enough to represent the target population and to show change. It is not sufficient to assess outcome only in those with known headache: this will not measure success or failure in identifying and diagnosing those not complaining of or under treatment for headache, who are likely to be numerous and in whom burden may nevertheless be significant[24].

Within a primary care group, it may be appropriate to assess burden annually in random samples of 1,000 adults reselected at each audit. Of these, about 150 will have migraine, more will have tension-type headache and 20-30 will have chronic daily headache. An instrument such as MIDAS[25] may be useful. This self-administered questionnaire, which can be mailed, measures time lost from work, other chores and social activity attributable to headache over the preceding 1-3 months. Although developed for migraine, MIDAS appears to be applicable to any headache and regardless of whether any headache condition has been diagnosed. It has yet to be validated for this purpose but, as a measure of change, those people who are significantly affected by headache seem more likely to complete the assessment and those who do not can probably safely be discounted.

In addition, audit should measure direct treatment costs: consultations, referrals and prescriptions.

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