

HEADACHE

IN PRIMARY CARE*

Key Advisers:

Dr Neil Whittaker - GP, Nelson

Dr Alistair Dunn - GP, Whangarei

Expert Reviewer:

Dr Alan Wright - Neurologist, Dunedin

Every headache presentation is unique and challenging, requiring a flexible and individualised approach to headache management.

- Most headaches are benign **primary** headaches
- A few headaches are **secondary** to underlying pathology, which may be life threatening

Primary headaches can be difficult to diagnose and manage. People, who experience severe or recurrent primary headache, can be subject to significant social, financial and disability burden.

We cannot cover all the issues associated with headache presentation in primary care; instead, our focus is on assisting clinicians to:

- Recognise presentations of secondary headaches
- Effectively diagnose primary headaches
- Manage primary headaches, in particular tension-type headache, migraine and cluster headache
- Avoid, recognise and manage medication overuse headache

DIAGNOSIS OF HEADACHE IN PRIMARY CARE

The keys to headache diagnosis in primary care are:

- Ensuring occasional presentations of secondary headache do not escape notice
- Differentiating between the causes of primary headache
- Addressing patient concerns about serious pathology

RECOGNISE SERIOUS SECONDARY HEADACHES BY BEING ALERT FOR RED FLAGS AND PERFORMING FUNDOSCOPY

Although primary care clinicians worry about missing serious secondary headaches, most people presenting with secondary headache will have alerting clinical features. These clinical features, **red flags**, are not highly specific but do alert clinicians to the need for particular care in the history, examination and investigation.

An exception to this may be slow growing intracranial tumours. For this reason fundoscopy, even though positive findings are rare, is essential for every initial headache presentation and periodically thereafter. Slow growing frontal lobe tumours are particularly liable to be silent. They may present with non-specific headache and subtle personality changes, resulting in treatment for depression. In these situations, non-response to treatment may prompt further investigation.

Red Flags in headache presentation

Red Flags in headache presentation include:

Age

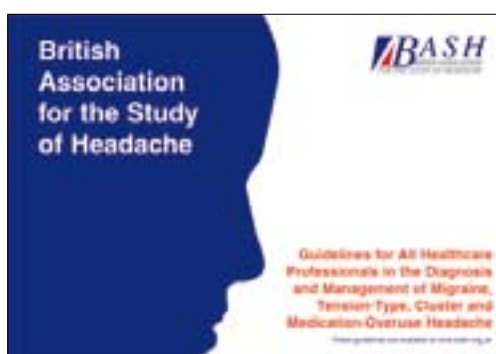
- Over 50 years at onset of new headache
- Under 10 years at onset

Characteristics

- First, worst or different from usual headache
- Progressive headache (over weeks)
- Persistent headache precipitated by Valsalva manoeuvre (cough, sneeze, bending or exertion)
- Thunderclap headache (explosive onset)

Additional features

- Atypical or prolonged aura (>1 hour)
- Aura occurring for the first time in woman on combined oral contraceptive
- New onset headache in a patient with a history of cancer or HIV
- Concurrent systemic illness
- Neurological signs
- Seizures
- Symptoms/signs of Giant Cell Arteritis (e.g. jaw claudication)



*Much of this article is adapted from: British Association for the study of headache, Guidelines for all healthcare professionals in the diagnosis and management of migraine, tension-type, cluster and medication-overuse headache. January 2007. The guideline can be downloaded from: <http://snipurl.com/1nzel>

Causes of secondary headache

The presence of red flags prompts consideration of a wide range of diagnoses. Some of these are listed below.

- **Vascular**
 - Subdural hematoma
 - Epidural hematoma
 - Subarachnoid haemorrhage
 - Venous sinus thrombosis
- **Tumour**
- **Toxins** (e.g. carbon monoxide)
- **Infectious causes**
 - Meningitis
 - Encephalitis
 - Abscess
- **Giant cell arteritis**
- **Hydrocephalus**
 - Obstructive
 - Acute
- **Metabolic disorders**

MINIMAL EXAMINATION FOR HEADACHE PRESENTATION

For all initial presentations of headache, examination includes:

- Fundoscopy
- Visual acuity
- Blood pressure measurement
- Examination of the head and neck for muscle tenderness, stiffness, range of movement and crepitation.

The presence of red flags or other features suggesting secondary headache indicate the need for more detailed examination. The question of whether a neurological examination should be performed, and in how much detail, is more problematic when there are no suspicious features and the history is characteristic of a primary headache.

Even when there are no red flags, a brief neurological examination, although unlikely to be positive, is a strong source of reassurance to patients and will save time in future consultations with still-worried patients. A suggested routine for a short neurological examination in these circumstances is available on a brief video on our web site, www.bpac.org.nz **keyword: 'Neuroexam'**

DIAGNOSIS OF PRIMARY HEADACHE

Primary headache is usually caused by tension-type headache, migraine, with or without aura, or cluster headache. Mixed headache types do occur, for example many people experience both migraine and tension-type headaches. Differentiation between the primary headaches is important because there are effective interventions available for each of them.

Headache diaries are useful diagnostic tools, which help the diagnosis of headaches and identification of any predisposing or precipitating factors.

Tension-type headache is the commonest form of primary headache

Most people will have at least one episode of tension-type headache during their lifetime. It is the commonest form of primary headache. The headache is usually described as tightness or pressure, like a tight band, around the head and often spreads to, or appears to arise from, the neck.

Tension-type headache is usually episodic, of low frequency and short duration but chronic tension-type headache can occur on more days than it is absent. Photophobia or exacerbation by movement can occur but these are usually less prominent features than in migraine.

Tension-type headaches are associated with stress and functional or musculoskeletal problems of the neck and often these occur together. Muscles of the head or neck are often tight and tender.

It is often useful to explain to patients that the pain is related to tension in the muscles of the head and neck and is often made worse by stress. This helps exploration of stressors without the patient feeling the clinician thinks 'it is all in my mind'.

Features of Migraine

Adults with migraine usually have a family history of migraine and experience recurrent episodes of moderate or severe headaches (which may be unilateral and/or pulsating) lasting for several hours or up to 3 days. These are typically associated with gastrointestinal symptoms, limitation of activity and avoidance of light and noise. There is often a preceding aura. People with migraine are free from symptoms between attacks.

When considering a differential diagnosis between migraine and tension headache, **the following features are common in migraine but not usually seen in tension headache.**

- Aura
- Unilateral headache
- Hypersensitivity, such as to light and noise
- Gastrointestinal symptoms, such as nausea or vomiting

The diagnostic criteria for migraine are reproduced in Table 1. These may be useful in the diagnosis of headache when there is some doubt about the diagnosis, particularly when there is no aura. When migraine is accompanied by aura the diagnosis is easier and only two episodes are required to make the diagnosis.

Table 1: Diagnostic criteria for migraine without aura

A	At least 5 attacks fulfilling criteria B–D
B	Headache attacks lasting 4–72 hours* (untreated or unsuccessfully treated)
C	Headache has at least two of the following characteristics: 1. Unilateral location* 2. Pulsating quality (i.e. varying with the heartbeat) 3. Moderate or severe pain intensity 4. Aggravation by or causing avoidance of routine physical activity (e.g. walking or climbing stairs)
D	During headache at least one of the following: 1. Nausea and/or vomiting* 2. Photophobia and phonophobia
E	Not attributed to another disorder (history and examination do not suggest a secondary headache disorder or, if they do, it is ruled out by appropriate investigations or headache attacks do not occur for the first time in close temporal relation to the other disorder).

*In children, attacks may be shorter-lasting, headache is more commonly bilateral, and gastrointestinal disturbance is more prominent.

One third of people with migraine have preceding aura

Approximately one third of people who get migraine, experience preceding aura. Usually auras last for between 5 to 60 minutes before the onset of migraine headache and settle as headache commences. The most frequently reported auras are visual disturbance, such as flickering or jagged lines or blind spots. Visual blurring or spots before the eyes are non-specific symptoms and do not represent aura. Other transient focal neurological symptoms, such as unilateral paraesthesia of a hand, arm or the face, and dysphasia, can also occur as aura in migraine.

Visual or other transient focal neurological signs presenting for the first time in older people always raise the possibility of Transient Ischaemic Attacks (TIAs). Prolonged aura in all age groups, especially continuing after resolution of headache and aura which involve muscular weakness, are indications for specialist investigation to exclude other causes.

Headache in migraine is not always unilateral

Although migraine headache is often unilateral it is not always so and the diagnosis of migraine should not be abandoned when headache is bilateral. The headache of tension-type headache is usually bilateral, but may be unilateral.

Migraine is usually accompanied by hypersensitivity

Hypersensitivity to stimuli, which are not normally noxious, is a common feature of migraine. Photophobia and phonophobia are the most frequently reported but hypersensitivity to touch (allodynia), smell (osmophobia), movement and pulsation of the arteries are also often experienced.

Hypersensitivity in migraine appears to be related to the central sensitisation and resulting peripheral sensitisation that occur in migraine.

Gastrointestinal upsets often prominent in migraine

Nausea and vomiting in migraine may be related to vestibular hypersensitivity and can be a prominent disabling feature of migraine episodes. Although anorexia and mild nausea may occur in tension-type headache, it is not usually a major feature.

Visual or other transient focal neurological signs presenting for the first time in older people always raise the possibility of Transient Ischaemic Attacks (TIAs).

Features of Cluster Headache

Cluster headache, unlike migraine, affects mostly young men (male:female = 6:1). Typically, the headaches occur in bouts for 6 to 12 weeks, once every year or two. The pain is severe, unilateral and disabling. During bouts, headache usually occurs daily, at a similar time each day.

Associated autonomic features include ipsilateral conjunctival injection, lacrimation, rhinorrhea, nasal congestion and ptosis. These do not always occur but the presence of one or two of these together with a typical cluster headache pattern clinch the diagnosis.

MANAGEMENT OF TENSION-TYPE HEADACHE

Management of tension-type headache includes general exercise, stress reduction, treatment of any underlying musculoskeletal problems and analgesia. Episodic use of aspirin or ibuprofen is usually sufficient. Paracetamol appears less effective. Complementary therapies such as yoga, meditation and acupuncture may help some people.

Although treatment sounds easy, in practice, implementation may be complicated. Patients may be expecting high-tech investigations to rule out serious pathology, physiotherapy and counselling may be unaffordable and often the stressors associated with the headaches are not amenable to change. This can result in over-reliance on medication.

Chronic use of medication for pain relief carries high risk of medication overuse headache. Analgesia use, should therefore, preferably be limited to no more than two days per week. Opiates, such as codeine, carry particularly high risk of medication overuse headache.

A three-week course of an NSAID, such as naproxen, may break the cycle of continuing pain and cover the early management of predisposing and precipitating factors, such as musculoskeletal problems and stress.

If this fails, the prophylactic medication of choice is amitriptyline; starting very low (5–10 mg at night) and increasing slowly every three weeks until symptoms are controlled, up to 75–150 mg at night. As in other chronic pain syndromes, the effectiveness of amitriptyline does not depend on its antidepressant activity. If amitriptyline is not well tolerated, nortriptyline has fewer side effects and may be an effective alternative.

A randomised controlled trial of botulinum toxin for chronic tension-type headache showed it to be ineffective.

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MIGRAINE MANAGEMENT REQUIRES A SYSTEMATIC APPROACH

Migraine management can be complicated and requires a systematic approach to:

1. Management of predisposing factors
2. Trigger identification and avoidance
3. Acute pain relief
4. Prophylaxis

MANAGING PREDISPOSING FACTORS IN MIGRAINE

Several factors are known to predispose people to migraine. These include stress, depression, anxiety, head or neck trauma and hormonal changes such as around menstruation or menopause. Management of these factors can have a significant impact on migraine frequency and severity. Keeping a diary will help to identify any predisposing and triggering factors.

IDENTIFICATION AND AVOIDANCE OF TRIGGER FACTORS IN MIGRAINE

Unfortunately, most migraine episodes have no obvious trigger, but if triggers can be identified, avoidance is often very effective. Frequently reported triggers include:

- Relaxation after stress
- Change in habit, such as a missed meal, late night or travel
- Bright lights and loud noise
- Dietary triggers, such as certain alcoholic drinks, some cheeses
- Unaccustomed strenuous exercise

MANAGEMENT OF ACUTE MIGRAINE HEADACHE

A systematic, three-tiered approach to the management of acute migraine headache is useful. Additional measures for emergency treatment at home and treatment of a relapse may be needed.

Using a systematic approach ensures each treatment modality is given a reasonable trial of effectiveness and highlights which treatments are effective for particular patients. BASH suggests that failure of treatment on one tier on three occasions should be the criterion for moving onto the next tier.

These tiers should all preferably be combined with rest and sleep; a stat dose of temazepam may be useful to achieve this

- **Tier one¹:** analgesic +/- antiemetic
- **Tier two:** specific anti-migraine drugs
- **Tier three:** combination therapies
- **Emergency treatment:** intramuscular NSAID and antiemetic
- **Relapse:** repeat symptomatic analgesics from step one and two and consider repeat of triptan

Footnotes

1. Tier one incorporates stages one and two of the BASH recommendations, which split oral and rectal analgesia +/- antiemetic into separate stages.
2. This is naproxen 250 mg plus naproxen 500 mg, or naproxen sodium 275 mg plus naproxen sodium 550 mg.
3. Cataflam or voltaren rapid. These appear to be more rapidly absorbed than diclofenac sodium.
4. EBM Reviews - Cochrane Central Register of Controlled Trials Di Monda V, Nicolodi M, Aloisio A, et al Efficacy of a fixed combination of indomethacin, prochlorperazine, and caffeine versus sumatriptan in acute treatment of multiple migraine attacks: a multicenter, randomized, crossover trial. *Headache*. 2003; 43(8):835-44

Tier one: analgesic +/- antiemetic

Step one consists of analgesia with aspirin or other NSAID, with the best evidence for ibuprofen and naproxen. These are usually given orally with **standard release** preparations at **higher doses**, taken **early in the attack** to avoid delayed absorption due to gastric stasis. **Delayed release preparations are not suitable.**

Recommended doses for adults are:

Aspirin: 600–900 mg, up to four doses in 24 hours

Ibuprofen: 400–600 mg, up to four doses in 24 hours

Naproxen²: 750–825 mg, with further 250–275 mg up to twice in 24 hours

Diclofenac-potassium²: 50–100 mg up to a total of 200 mg in 24 hours

General contraindications to NSAIDs must always be kept in mind but there is little evidence for paracetamol use on its own in migraine. In practice, paracetamol does appear to be useful, especially when combined with metoclopramide.

Metoclopramide promotes gastric emptying. Even when nausea and vomiting are not present, this is likely to improve absorption of analgesics and there is some evidence that metoclopramide on its own gives relief in migraine.

When nausea or vomiting render oral administration problematic, rectal preparations of analgesics and anti-emetics may be more suitable. Diclofenac suppositories, 100 mg, used up to twice in 24 hours are recommended by BASH.

Anti-emetic suppositories are useful if nausea and vomiting is a problem. Prochlorperazine, 25 mg, is available as a suppository in New Zealand.

There has been a recent resurgence of interest in the use of preparations containing fixed drug combinations. In a randomised controlled trial, a fixed combination suppository of indomethacin, prochlorperazine and caffeine, was as effective as sumatriptan.

Opiates and opioids should, in general, be avoided during acute migraine. They provide little additional benefit, have potential for addiction and, as discussed on page 22, can be associated with medication overuse headache. Any history of alcohol or drug abuse or dependency is a strong warning that problems are likely.

Tier two: specific anti-migraine drugs

The triptans are serotonin agonists used in acute migraine management. Sumatriptan is the only funded triptan in New Zealand.

Unlike symptomatic treatment, triptans should not be taken too early. They appear to be ineffective if given during aura and most effective, whilst pain is still mild or at the onset of hypersensitivity. Unfortunately, triptans are associated with return of symptoms within 48 hours in 20–50% of patients who initially respond.

Sumatriptan should not be repeated if the first dose has been ineffective but can be repeated if it was initially effective but the headache has recurred (see page 19).

Sumatriptan, 50 mg orally, is usually tried in the first instance combined with metoclopramide. If this is not effective, 100 mg orally, can be tried in future attacks. Sumatriptan can, if necessary, be given subcutaneously at a dose of 6 mg.

Contraindications to triptans include:

- Ischaemic heart disease
- Prinzmetal's angina/coronary vasospasm
- Cerebrovascular disease (CVA) or transient ischaemic attack (TIA)
- Uncontrolled hypertension
- Severe hepatic impairment
- Concurrent use or use within two weeks after discontinuation of monoamine oxidase inhibitors

Ergotamine use, for migraine, is limited by a significant risk of toxicity and drug interactions. Major side effects include: nausea, vomiting, paresthesia, and the convulsive and gangrenous effects of ergotism. Contraindications are cardiovascular and cerebrovascular diseases, Raynaud's disease, arterial hypertension, renal failure, pregnancy and breastfeeding.

Ergotamine is thought to have significantly lower relapse rates than sumatriptan and may be useful if relapse is a major problem and cannot be managed with other medications. It should not be used for at least 12 hours after sumatriptan (see page 19).

Ergotamine is available in New Zealand combined with caffeine in Cafergot. One tablet contains 1 mg of ergotamine and 100 mg of caffeine. For first time users, two tablets are taken initially with a further tablet half hourly if needed. Subsequently three tablets can be taken initially, if needed, with a further tablet half hourly. The **maximum dose** in any 24 hour period is six tablets and a maximum of ten tablets in any week.

Tier three: combination therapies

There is some evidence that a combination of naproxen and sumatriptan is superior to either drug alone and it can be worth trying this combination as Tier Three.

Emergency treatment: intramuscular NSAID and antiemetic

Emergency management of acute migraine is difficult, especially on house calls to patients not seen previously. Injections of opiates, e.g. pethidine or morphine, are best avoided. Rebound headache, potential side effects and risk of dependency generally outweigh the potential for additional pain relief.

BASH recommends for adults, when there are no contraindications, diclofenac, 75 mg, intramuscularly. However, diclofenac injections can cause serious tissue damage and it is preferable to avoid them if possible. Medsafe recommends they be given by deep intragluteal injection into the upper outer quadrant, if required.

NSAIDs by suppository are a safer alternative, and are often effective. Concurrent administration of prochlorperazine, 25 mg as a suppository is useful to control nausea and vomiting.

Chlorpromazine, 25–50 mg intramuscularly is useful as an anti-emetic and sedative in the emergency management of acute migraine.

Relapse: repeat analgesics and consider repeat of triptan

Relapse is recurrence of headache within the same episode of migraine despite initial efficacy. Management is difficult because repeated doses, especially of triptans or opiates, if they have been used, can give rise to repeated rebound over several days.

Repeat of previously used analgesics may be effective. A second dose of triptan is usually effective but does increase the risk of further rebound. A minimum of two hours is required between doses. Ergotamine may be an alternative but must be given at least 12 hours after sumatriptan.

The maximum dose of sumatriptan in any 24 hour period is:

- Oral dosage in 24 hours, 300 mg
- Sub-cutaneous dosage in 24 hours, 12 mg

Limit use of acute migraine therapy to two days per week

Regular use of acute migraine therapies for more than two days per week carries significant risk of initiating or escalating medication overuse headache and should be avoided. Regular requirement of acute migraine therapy for more than one day per week is an indication to evaluate how the medication is being used and review the diagnosis.

MIGRAINE PROPHYLAXIS

Migraine prophylaxis is indicated when symptoms cannot be adequately controlled with acute therapy. As migraine is cyclical, permanent use of prophylaxis is not usually required; it can be tapered off, after 4-6 months, to test the need for continued use.

The choice of medication for prophylactic therapy for individual patients is guided by:

- Evidence of effectiveness
- Potential benefits
- Potential risks
- Ease of use
- Comorbidities

The medications most useful in primary care are shown in Table 2. In general, prophylactic therapies are started at low doses and gradually increased to avoid side effects. Once a full dose is achieved, a reasonable trial of therapy is approximately 6–8 weeks.

Table 2: Medications for migraine prophylaxis in primary care

	Evidence	Additional benefits	Risks	Dose	Comorbidities to consider
Beta blockers Good evidence base	RCTs for metoprolol, propranolol, nadolol and atenolol		Cold extremities, reduced exercise tolerance, dizziness	Metoprolol 50–100 mg BD Propranolol LA 80 mg daily to 160 mg BD	Asthma, heart failure, peripheral vascular disease, depression
Tricyclics Adequate evidence base	Evidence for effectiveness from small RCTs of amitriptyline	Helps with co-existent tension headache, other pain conditions, disturbed sleep and depression. Some evidence of synergy with beta blockers	Sedation, dry mouth, dizziness, nausea Less side effects with nortriptyline	10–150 mg at night	Concurrent use of other anti-cholinergic medications
Sodium valproate Good evidence base	RCTs		Nausea, weight gain, alopecia, spontaneous bruising, liver dysfunction	300–1000 mg BD	Contra-indicated in pregnancy

RCT = Randomised Controlled Trial

Pizotifen and clonidine have little evidence of effectiveness and are now superseded for the prophylaxis of migraine in adults. There is some evidence for the effectiveness of verapamil and the evidence for the use of fluoxetine is inconclusive.

Acupuncture is often used for migraine and trials have shown reduction in the severity and frequency of episodes. However, the quality of these trials has been questioned. There are the usual problems associated with testing complementary therapies. Medications are subject to trials before introduction, whereas complementary therapies are not usually subject to trial until they have been used for many years and positions have become entrenched. Decisions will depend on the enthusiasm of individual clinicians and patients for this modality of treatment.

MIGRAINE IN CHILDREN

In children, migraine attacks may be shorter-lasting, headache is more commonly bilateral and gastrointestinal disturbance is more prominent.

Generally, children with migraine, which cannot be controlled with simple analgesics, are best referred for specialist care. Anti-emetics are not recommended.

MANAGEMENT OF MIGRAINE DURING PREGNANCY AND BREAST-FEEDING

There are no clinical trials specifically evaluating the drug treatment of migraine during pregnancy. Fortunately, migraine frequency is usually reduced during this time. (Ever et al, 2006)

Table 3: Management of migraine during pregnancy

Paracetamol	Can be used throughout pregnancy and breast-feeding
NSAIDs	Avoid in the third trimester to avoid fetal renal damage and patent ductus. In the first and second trimester short acting NSAIDs, such as ibuprofen, are preferred
Metoclopramide	Unlikely to cause harm through pregnancy and breast-feeding
Triptans and ergotamine	Contraindicated However, women who have taken sumatriptan inadvertently in pregnancy can be reassured current evidence suggests they are at no greater risk of birth defects than the general population
Propranolol	Beta blocker with best evidence of safety during pregnancy
Amitriptyline	Lowest effective dose may be used

MANAGEMENT OF CLUSTER HEADACHE

Cluster headache is excruciatingly painful and symptomatic treatment is seldom adequate. Patients often benefit from the involvement of a specialist who has experience in the prophylactic management of cluster headache.

Sumatriptan, 6 mg subcutaneously, is the only proven highly effective treatment for acute cluster headache. Oxygen 100% for 10–20 minutes helps some people. Analgesics have no place in treating cluster headache. Ergotamine and oral triptans are not effective.

Prophylactic therapy is commenced as early as possible when a new cluster starts and alcohol should be avoided completely during cluster episodes. Verapamil, prednisone and lithium all appear to be effective prophylactic therapies for cluster headache. Cluster headache is rare and GPs are unlikely to develop experience in its management. Referral to an appropriate specialist in this area is usually the best option.

AVOIDANCE, RECOGNITION AND MANAGEMENT OF MEDICATION OVERUSE HEADACHE

Medication overuse headache occurs most frequently from chronic overuse of analgesics, such as aspirin, NSAIDs, paracetamol and codeine, to treat headache. Frequent lower doses appear to carry greater risk than higher weekly doses. It also occurs because of rebound headache following triptan use.

Medication overuse headache may take a long time to resolve after the medication is withdrawn. Re-introduction of headache medication may resolve the headache in the short term but escalates the long-term problem.

There is no specific type of headache associated with medication overuse but patients often describe them as oppressive, often worse on waking and aggravated by physical exercise. They are not usually accompanied by nausea or vomiting.

Headaches evolve over weeks or longer, with increased frequency of the headache, often accompanied by increased analgesia use, until eventually, medication is taken in anticipation of headaches. Prophylactic medication is ineffective. Often the pattern of headaches and medication use can only be understood with the help of an accurate headache and medication diary.

Other forms of primary and secondary headache should be carefully excluded.

There are four objectives in the management of medication overuse headache:

- Withdrawal from the overused medication
- Recovery from the headache
- Re-assessment of any underlying primary headache
- Prevention of relapse

WITHDRAWAL OF OVERUSED MEDICATION

Motivation: For people who experience medication overuse headache, the outcome of withdrawal is usually good. The alternative is ever-worsening headache.

Warning: Headaches may worsen for three to seven days following withdrawal of medication. Patients need encouragement and support over this time and absence from work may be required.

Diary: Recording symptoms and medication use during medication withdrawal, allows a more objective assessment of the results of withdrawal.

Good hydration: This is thought to help.

Abrupt withdrawal: This is more successful than gradual withdrawal. When withdrawal cannot be achieved, it may be effective to offer regular naproxen 250 mg tds or 500 mg bd for three weeks to cover the withdrawal period. The aim is to prevent people responding to headache by taking medication.

RECOVERY FROM HEADACHE

The time to recovery from the headache depends on the medication type.

Triptan: 7–10 days

Simple analgesics: 2–3 weeks

Opiates: 2–4 weeks

When recovery does not follow a reported withdrawal, the headache may have other causes, or medication overuse may be continuing.

RE-ASSESSMENT OF UNDERLYING PRIMARY HEADACHE

An underlying primary headache, usually tension-type or migraine, often becomes apparent within two months. This should be managed systematically. The analgesics, which were implicated in the overuse headache, can be re-introduced after two months, if required, but care has to be taken that these are used appropriately.

PREVENTION OF RELAPSE

There is a high risk of relapse and good support will be required.

BRIEF UPDATE ON THE PATHOPHYSIOLOGY OF MIGRAINE AND ANTI-MIGRAINE DRUGS

Migraine is a group of familial disorders; individual susceptibility is conferred by genetics and exposure to triggering factors.

Migraine aura is strongly associated with a slowly spreading wave of decreased electrical activity that travels across the cortex at approximately 2–3 mm per minute. This is termed Cortical Spreading Depression (CSD). CSD is thought to also occur in migraine without aura, but is clinically silent.

An episode of CSD is followed by long-lasting suppression of neuronal activity and activation of the trigeminovascular system. Consequent release of neuropeptides produces vascular dilation and neurogenic inflammation. Headache results because of meningeal irritation and the sensitisation of nerve fibres to previously innocuous stimuli, such as the pulsing of blood vessels.

Beta blockers, valproate and amitriptyline, the first choice drugs for migraine prophylaxis in primary care, have all been demonstrated to reduce the number of CSDs in animal experiments. The mechanism by which this occurs has not yet been demonstrated, but the discovery of CSD does provide an avenue for the development of new prophylactic anti-migraine drugs.

Triptans and ergotamine, used in acute migraine, reduce headache by blocking release of the neuropeptides responsible for meningeal irritation and sensitisation of central nerve fibres.

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