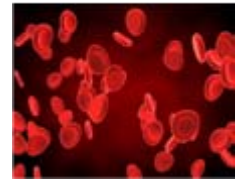


The GP Update Handbook



Spring 2010

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Neuropathic pain	
NICE guidance on neuropathic pain (both diabetic and non-diabetic) is covered in the section on managing diabetic foot complications in the diabetes chapter.	
Only available online (www.gp-handbook.co.uk):	
Bell's Palsy	
Carpal Tunnel Syndrome	

Neurology

Vertigo

BMJ 2009;339:b3493

'I'm feeling all dizzy doctor'...

I am sure this is a phrase that sinks many a GPs heart. It is certainly one that perplexes my registrars each year! My usual follow on question is 'People mean lots of different things when they say the word dizzy. Can you describe exactly what you mean?' On a bad day, this is invariably followed by, 'You know doctor, dizzy, all dizzy'! So this review in the BMJ is useful!

Let's first be clear – here I am talking about rotatory vertigo: the illusion of movement – often described by patients as 'the world spinning'. This comes from the labyrinth or brainstem. I am not talking about pre-syncopal postural hypotension, that sensation you get if you leap out of bed too fast in the morning! The problem is that in the elderly the two often overlap.

Important features in the history

When taking a history from a patient with vertigo ask about:

- Onset and duration
- Is it positional?
- Is it sustained or does it get better if you keep still for any length of time?
- Hearing loss or tinnitus
- Headaches.

New onset headache with vertigo is considered to be a red flag, especially if the headache is occipital (?stroke or tumour). The BMJ review recommends most people with new onset headache and vertigo need admission unless there is a good history suggesting this is migrainous.

Examination

- **Eyes:** Check eye movements are full
Check for Horner's syndrome (miosis (constricted pupil) with partial ptosis)
Examine the eyes for nystagmus and do the Hallpike's manoeuvre (as discussed in the section on BPPV which follows this review of vertigo).
Remember that in BPPV nystagmus is not sustained.
Vertical nystagmus is a red flag (suggest brainstem or cerebellar disease).
- **Ears:** Check tympanic membranes, check for deafness.
 - Sudden unilateral hearing loss with vertigo suggests acute ischemia of labyrinth/brainstem or acoustic neuroma or a perilyphatic fistula (very rare – occurs after straining/trauma)
 - Ménière's classically presents with vertigo, hearing loss and tinnitus.
- **Face:** exclude facial weakness, dysphagia or dysphonia (central cause?).
- **Focussed neurological examination to exclude central causes:**
 - Check cranial nerves
 - Check for long tract symptoms (weakness or numbness of limbs)
 - If unable to walk, refer, as cause is likely to be serious. Subjective unsteadiness is common and acceptable.

Remember most central causes will have other features: acoustic neuromas (very rare) are an exception though! Therefore an MRI is rarely helpful in isolated vertigo.

Central causes

If you suspect a central cause (other than migraine) then refer urgently.

- **Migraine (common).**

Vestibular migraines (= migraines with rotational vertigo) are relatively common. Often go undiagnosed, because headache may be mild. Vertigo may occur before, during or after aura or headache and may take several days to go. However if this a first episode, with no past history, remember that people with brain stem strokes often present with vertigo & headache.

- **Stroke** (cerebellar or lateral medullary).
- **Cerebro-pontine tumour** (rare).
- **Brainstem demyelination** (rare).
- **Acoustic neuroma** (very rare).

Peripheral causes

- **Benign paroxysmal positional vertigo** (common – 40% of cases): see the next section for a fuller discussion of BPPV.
- **Acute vestibular neuritis (labyrinthitis)** (common – 40% of cases).
Common, often younger or middle aged.
Sustained non-positional vertigo
Unidirectional nystagmus (usually horizontal)
Nausea and vomiting are common
No hearing loss
No tinnitus
No neurological symptoms or signs.

Cause unclear. Sometimes preceded by a viral URTI infection.

Severity peaks over a few days and then starts to settle over the next few days.

The head thrust test may be helpful to confirm acute vestibular neuritis:

Ask patient to fix their eyes on your nose. Turn their head rapidly to one side (you only need to move it by 5-10°, but speed is important). In normal individuals they will remain looking at your nose. If the vestibular-ocular reflex fails (as it does in acute vestibular neuritis) then the patient's eyes will move as their head moves then jump back to fix on your nose.

In older people remember that occasionally cerebellar strokes can present with sustained nystagmus and vertigo with no other signs.

- **Ménière's disease** (rare – 10%). Classical triad: vertigo, hearing loss and tinnitus.
- **Suppurative ear disease or vestibular perilymph fistula** (very rare).

Take home messages: Vertigo
<ul style="list-style-type: none"> • First check 'dizzy' means rotational vertigo not pre-syncope. • History and focussed examination will often exclude serious causes. Only a simple neurological examination is required. • Beware of the red flags: new onset headache (especially occipital), neuro signs or symptoms, new deafness or vertical nystagmus. • Common things are common (BPPV, acute vestibular neuritis) and will often recover spontaneously. The head thrust test is helpful in acute vestibular neuritis. Migraines with vertigo often go undiagnosed – look for a past history.

Benign paroxysmal positional vertigo

DTB 2009;47:62-66

At the last set of course we ran, I asked GPs in the audience the following questions:

How many feel they can confidently do Hallpike's manoeuvre?

How many do it but are not quite sure what you are doing?

How many just don't bother to do it at all because they are not sure what they are doing?

Less than 5% of each audience felt they knew what they were doing.

A further 5% did it but were not quite sure what they were doing (me included!).

Overwhelmingly (about 90%) didn't do Hallpike's at all.

Now I pass no judgement on what is the right thing to do, but I am intrigued that so many do not do it. Read on and see what you think...

This was a really detailed review in the Drugs and Therapeutics Bulletin of a common general practice problem, but because of the conclusions in the review I will cover just the key points.

☒ Benign paroxysmal positional vertigo statistics

- Common: Lifetime prevalence 2.4%.
- Peak age 50-70 but can occur at any age.
- Twice as common in women as men.
- 30%-50% get better within 7 days.

A reminder about the pathophysiology...

Each inner ear has 3 semicircular canals (anterior, posterior and horizontal). These run at right angles to one another. Each canal has a widened, open end (utricle) and a closed end (cupula). Within the utricle is a membrane embedded with calcium carbonate crystals (otoconia). The otoconia should remain attached to the membrane and act as weights, transmitting changes in head position to the underlying sensory hairs. If otoconia become detached they can either form free floating debris in the canal (canalithiasis) or adhere to the cupula (cupulolithiasis). The important point is that the debris may continue to move once the head has stopped moving, causing a sensation of movement from the inner ear which conflicts with other sensory messages (vision, proprioception). The conflict of information gives the sensation of vertigo. Because of gravity, the posterior semicircular canal, which is the lowest, is the most likely to be affected.

Causes of benign paroxysmal positional vertigo

- In many cases no cause is found, although in 18% head trauma is identified as a cause.
- Remember 30%-50% get better within 7 days.

Clinical features

- Vertigo and nystagmus prompted by certain positions.
- Symptoms disappear rapidly if head kept still. If persistent after stopping movement then consider other causes, however rarely people have light-headedness and unsteadiness that persist between attacks.
- Hearing loss is **not** a feature. If present, BPPV is not the diagnosis.
- Nausea & vomiting occur in BPPV but also in Meniere's, migraine & vestibular neuritis (labyrinthitis).

Differential diagnosis

Red flags warrant urgent specialist assessment: Hearing loss
New onset headache
CNS signs including gait ataxia.

Other causes of vertigo &/or nystagmus include: Migraine
TIA/CVA
Acute vestibular neuritis (labyrinthitis)
Meniere's disease
Perilymphatic fistula
Acoustic neuroma
Multiple sclerosis.

Look for associated symptoms to help elucidate these causes (eg. hearing loss, tinnitus, pain, headache, photophobia).

Drugs can cause vertigo too, particularly: Some anti-epileptics (carbamazepine, phenytoin)
Antihypertensives
Cardiovascular drugs (many!).

Examination

- Look for a cholesteatoma.
- Look for vesicles (Ramsay Hunt syndrome from zoster).
- Assess cranial nerves, particularly looking for a sensorineural hearing loss.
- If vertigo or nystagmus can be reproduced by pushing on tragus and external auditory meatus of affected side then this suggests the presence of a perilymphatic fistula.

Diagnostic manoeuvre (Hallpike test)

I offer this to you if you are interested to know! The aim is to trigger the vertigo/nystagmus.

- Position the patient on the couch so that when lying down their head hangs over the back of the couch.
- Ask the patient to keep their eyes open and fixed on your forehead.
- From sitting, the patients head is turned 30-45 degrees in one direction and then they are rapidly laid down so that their head hangs below the level of the couch (about 30 degrees below the couch). Hold for up to 1minute. Look for nystagmus which begins 5-20seconds after reaching this position. Ask about vertigo too. Repeat with head turned to opposite side.
- On repeating the manoeuvre the nystagmus/vertigo should become less obvious (fatigability).

Which canal is affected?

If you do Hallpike's you can work out, from the type of nystagmus, which semicircular canal is affected, but as the DTB concludes that everyone should be offered the same treatment, this seems a rather academic exercise. However, for the sake of completeness, I'll include it:

- **Posterior canal debris usually causes rotatory up-beating (geotropic) nystagmus.**
Now bear with me while I explain this, the other sorts of nystagmus are easier... basically the top pole of the eyes rotates towards the undermost, affected ear whilst there is also some vertical nystagmus (eyes moving to forehead). Remember that the posterior canal is most often affected because of its position and the effects of gravity.
- **Horizontal canal debris will result in pure horizontal nystagmus.**
- **Anterior canal debris usually causes down-beating nystagmus** (but this may also be triggered by a central cause).
- **Beware of sustained or non-fatiguing nystagmus as this is unlikely to be due to BPPV (indicates CNS cause).**

Treatment

Benign paroxysmal positional vertigo (BPPV) treatment programmes tend to be based on the idea that the loose particles can be manoeuvred out of the way. The evidence base for many of these treatments comes from small, sometimes non-randomised trials, and is therefore not very strong! Added to that, the DTB suggests everyone should be offered the same treatment anyway, so I will describe only that treatment! If you have a special interest in this you might want to read the whole of the DTB article.

- **The DTB concludes Epley's manoeuvre should be offered to those with BPPV, and they could try Brandt-Daroff at home if they so wished, acknowledging the weak evidence base (see below for how to do these exercises).**
- **Drug therapy, particularly vestibular sedatives, are not encouraged.** There is no evidence to suggest that any drugs help those with BPPV. In fact there is some evidence that vestibular sedatives actually may slow recovery if used long term as they impair the feedback that allows patients to develop compensation, which is part of the recovery.

Epley Manoeuvre

Five basic movements that aim to clear debris from the posterior canal. Watch it on video to see how to do it (see useful websites, below).

Modified Epley manoeuvre has now been developed which patients can do at home without a health professional. The modification comes from the pillow which raises the shoulders and allows the head to rest on the bed (not dangling over the edge), but still below the horizontal plane.

Does it work?

- **Conventional Epley has good evidence of benefit – NNT of 2-4, with benefit within 1 day to 4 weeks.**
- Restricting activities post-procedure (sometimes still recommended) does not improve efficacy and is not required.
- Modified Epley was shown in one trial to be more effective than Brandt-Daroff, but a systematic review suggested insufficient evidence to know whether modified Epley was more beneficial than any other procedure, including Brandt-Daroff.

Brandt-Daroff exercises (self-treatment)

From sitting, the patient rapidly moves to the position that triggers symptoms (usually nose up, with lateral aspect of occiput resting on bed – so not dissimilar to Hallpike, but without dangling off the edge of the bed). Hold the position for 30s or until vertigo subsides.

Sit up again for 30s.

Then lie down again, but this time turning the head in the opposite direction for 30s, then sit up again.

It is recommended that the patient does these every 3 hours during the day until they have 2 vertigo free days.

Does it work?

Evidence for efficacy comes from a study of 124 people randomised to Brandt-Daroff exercises v sham movements and those doing the Brandt-Daroff manoeuvres had less vertigo over a 60 day period.

Surgical treatment

This is a last resort only because permanent sensorineural deafness is not uncommon.

So what does all this mean in practice?

- The DTB concludes Epley should be offered to those with BPPV, and they could try Brandt-Daroff at home if they so wished, acknowledging the weak evidence base.
- Drug therapy, particularly vestibular sedatives, are not encouraged.
- Surgery is reserved as a last resort.

Take home messages: Benign Paroxysmal Positional Vertigo

- Diagnosis is based on history, examination and particularly the Hallpike test.
- Be sure to exclude more sinister causes, checking particularly for any hearing loss, tinnitus, pain, headache or photophobia. Consider drugs as a cause.
- Consider manoeuvres to improve symptoms, but note poor evidence base. Epley manoeuvre is recommended and Brandt-Daroff home treatment programme can be tried, although evidence is limited.
- Don't use vestibular sedatives: they may make things worse.

CPD Ideas

Are you confident in your diagnosis of BPPV and the use of the Hallpike test? You could practice with a colleague if you are not sure.

If you don't know who offers Epley manoeuvres locally you could find out.

Are you using vestibular sedatives? Should you stop?

Useful websites**For professionals:**

Watch how to do Epley manoeuvre on line: <http://emedicine.medscape.com/article/791414-media>

For patients:

A patient information leaflet on Epley manoeuvre:

<http://www.gloshospita;s/nhs.uk/ppi/leaflets/pdf/GHPI0820.pdf>

A sequence of pictures showing patients how to do the Bandt-Daroff manoeuvres:

<http://www.tchain.com/otoneurology/disorders/bppv/brandt/first.html>

Headaches

'It's his head, doctor'. How often do we hear that? However this time I was not in my clean and well equipped NHS surgery. The child looked 7 or 8. He had walked to the clinic, escaping from war torn Rwanda a few miles down the road. He had a wound on the back of his head covered with a rather grubby rag and was eyeing me suspiciously. I removed the rag and inspected the wound. The machete had gone deep, but that was not unusual. To my utter horror however, what I saw at the bottom of the wound was not scalp or bone, but brain. I hastily replaced the skin, tissues and rag. Amazingly the child survived with skin suturing and antibiotics. He was adopted by a local family and now thrives! But the phrase 'It's my head, doctor' still makes my heart race and my stomach queasy!

SIGN guidelines on headaches

SIGN 107, 2008

This Scottish Intercollegiate Guidelines Network helpfully reviews the diagnosis and management of headache in primary and secondary care. I'll focus on those parts relevant to us in general practice.

Definitions

The SIGN guidelines define headaches as follows:

- **Primary headache disorders: not associated with underlying pathology**
eg. migraine, tension headaches, cluster headaches.
- **Secondary headache disorders: headache attributed to underlying pathology**
eg. head pain from infection, neoplasms, drug induced.
- **Chronic headache: headache for >15d/month for > 3 months.**

Red flags

SIGN suggest that in patients with a headache, the following should prompt further investigation:

- New onset or change in headache in patients >50y.
- Thunderclap headaches (high intensity headache of abrupt onset: seconds-5mins, **Refer immediately** for further investigation. Commonest cause is sub arachnoid haemorrhage but other forms of intracerebral bleeding, cerebral venous sinus thrombosis, arterial dissection and pituitary apoplexy can all present in this way.)
- Focal neurological symptoms (eg. limb weakness, aura <5mins or >1hr).
- Non-focal neurological symptoms (eg. cognitive disturbance).
- Change in headache frequency, characteristics or associated symptoms.
- Abnormal findings on neurological examination.
- Headache that changes with posture.
- Headache waking the patient (although migraine is the most common reason for a headache on waking).
- Precipitated by physical exertion or valsalva manoeuvre (coughing, laughing, straining).
- Patients with risk factors for cerebral venous sinus thrombosis (eg. hypercoagulable states, pregnancy).
- Jaw claudication/visual disturbance.
- Neck stiffness.
- Fever.
- New onset headache in a patient with cancer/HIV.

History

- The SIGN guidelines warn us that **an inadequate history is probably the most common cause for mis-diagnosis of headache!**
- **Because of this warning I have outlined the characteristics of the common sorts of primary headaches in a table in the appendix. Apologies if this seems like teaching grandmothers to suck eggs!**
- **Headache diaries are recommended** (see the example in the appendix).

Key features to cover in the history are:

How many different sorts of headaches does the patient get? Take a detailed history for each sort.

Time

- How recent onset?
- Duration, frequency, temporal pattern (episodic, daily or unremitting)?
- Why consulting now?

Character of pain

- Intensity, nature and quality of pain, site and spread of pain, associated symptoms?
- Causes?
- Predisposing and/or triggering factors? Aggravating/relieving factors?
- Family history of similar sorts of headache?

Responses

- What does the patient do during an attack?
- How much is activity/function limited or prevented?
- What medication has been used?

State of health between attacks

- Completely well or residual/persisting symptoms?
- Concerns/anxieties/fears about headaches and their causes?

Examination

- **The SIGN guidelines recommend a neurological examination, fundoscopy and BP** (and obviously other clinical examination as indicated).
- **Assess neck movements in all patients** to exclude neck pain as a cause/contributory factors. Assess posture, range of movements, muscle tone and tenderness.

Management

I'll discuss the management of the different sorts of headaches one by one.

Tension Headaches

- **No investigations needed if classic history, absence of red flags and normal neurological examination.**
- **Aspirin and paracetamol recommended for acute treatment.**
- **Treat chronic tension headaches with tricyclics** (preferably amitriptyline 25-150mg).

Migraines

- **SIGN guidelines say neuroimaging not recommended if:**
 - Clear history of migraine (see table in the appendix)
 - No red flags for potential secondary headache
 - Normal neurological examination.

Acute treatment

- **Treatments (especially triptans) need to be taken as soon as possible to maximise efficacy** (*'Carry one with you in your wallet/purse...'*)
- **First line: Aspirin (900mg) or ibuprofen (400mg) for all severities of migraine.**
- **Add in paracetamol (1g) for mild-moderate headaches.**
- **If previous attacks not controlled with oral analgesia add in oral triptans.**

Recommended triptans are:

Almotriptan (12.5mg)
Eletriptan (40-80mg)
Rizatriptan (10mg)

These triptans are recommended because they are as good/better than sumatriptan 100mg in terms of initial pain relief and are well tolerated. **Almotriptan is the cheapest.**

- If no response from one triptan, it is worth trying a different triptan.
- For prolonged attacks which recede and then return try sumatriptan 50-100mg (with naproxen 500mg).
- **If nausea/vomiting a problem use oral/rectal antiemetics** (eg. metoclopramide).
- Do not routinely use opioids (because of risk of medication overuse headaches).
- Discuss medication overuse headaches with patients when initiating therapy for migraines.

Prophylaxis of migraine

- Trial data suggests prophylaxis reduces severity & frequency of attacks by half.
- SIGN do not indicate at what stage prophylaxis should start, although most would be guided by frequency and severity, and functional impairment.
- **Beta-blockers should be used first line as prophylaxis** (propranolol 80-240mg/day).
- **Alternatives are:**
 - **Antidepressants**
Amitriptyline is used in doses of 25-150mg od and venlafaxine at 75-150mg.
 - **Antiepileptics**
Topiramate 50-200mg/day (reduces frequency & severity).
Valproate 0.8-1.5g/day (reduces frequency & severity).
Gabapentin 1.2-2.4g/day (reduces frequency but not severity).
- **Non-pharmacological prophylaxis**
 - Stress management may reduce frequency and severity of migraines.
 - Acupuncture can work as prophylaxis.

What is the link between migraine and stroke?

BMJ 2009;339:b3914

A systematic review and meta-analysis confirms the links:

- **Those with migraine with aura have a 2 fold increased risk of ischaemic stroke.**
- Those without aura have no increased risk.
- Those with migraine have no increased risk of other cardiovascular diseases such as MI, suggesting the risk is due to something occurring in the brain (vascular spasm?) rather than atheroma (although the study may be underpowered to detect this).

Migraine in pregnancy

A BMJ was useful because it offered advice on what happens to migraine in pregnancy. This is something my patients often ask about (BMJ 2008;336:1502-4).

- **Pregnancy usually reduces the frequency and severity of attacks, especially if women get migraines without aura.**
- **Having said that migraines may be worse in the first trimester and reduce significantly thereafter – many women have no migraines at all in the third trimester.**
- **Nausea associated with migraines may get worse during pregnancy.**
- **Headache is common in all women post partum (1/3rd get a headache on day 3-6, even in the absence of a history of migraines).**

In pregnancy SIGN recommend:

- Try to avoid medication for headache, especially in the first trimester.
- Paracetamol is safe and should be used for migraines and tension type headaches.
- If paracetamol insufficient use aspirin (300mg) or ibuprofen (400mg) in first and second trimester. Regular/high dose ibuprofen in later pregnancy associated with increased risk of foetal complications. Aspirin is contraindicated in the third trimester.

Do migraines in pregnancy increase risk of stroke?

This study received a lot of publicity and it was reported in the media that ‘migraine during pregnancy gives you 30x the population risk of stroke’. However the study was pretty flawed – they looked for women admitted in the peripartum period with a code for migraine in their discharge summaries. They then found that these women had an increased risk of also having a diagnostic code for stroke or MI (odds ratios 30 and 4.9 respectively). However they made no attempt to validate codes used (ie. to check whether women with a code of migraine actually had migraines), and many women without this code may well also have suffered from migraines but if they didn’t have one during admission this is unlikely to be recorded. In addition no adjustment was made for vascular risk factors (smoking, hypertension, diabetes etc.) (BMJ 2009;338:b664).

- **This means the data is highly polluted by confounding!**
- **The data cannot be extrapolated back to a general practice population; pregnant women coming to us with migraines are not therefore at a 30x increased risk of stroke.**

Now back to the SIGN guidelines...

Cluster Headache

Cluster headaches are the commonest form of **trigeminal autonomic cephalgias (TACs)**.

What are TACs?

- Unilateral pain in the distribution of the trigeminal nerve.
- Cluster headaches are the commonest type, but still rare (1 in a 1 000). Next commonest form is paroxysmal hemicrania (1 in 50 000!) which are shorter (2-45mins) but more frequent (up to 40/day).

Diagnosis of cluster headaches/TACs

- Pain is **strictly unilateral** in cluster headaches.
- **Ipsilateral** autonomic features should be present, for example:
 - Conjunctival injection and/or lacrimation
 - Miosis and/or ptosis
 - Eye lid oedema
 - Nasal congestion and/or rhinorrhoea
 - Forehead/facial sweating.
- Often restless/agitated.

Treatment of cluster headaches/TACs

- **If you suspect cluster headaches or TACs, refer.**
- Subcutaneous sumatriptan (6mg) is first line for acute treatment.
- Nasal sumatriptan or zolmitriptan if subcut sumatriptan not effective/tolerated.
- For prophylaxis verapamil 240-960mg is recommended.
- Oxygen is recommended but note the recommendation is based on a trial of 19 men done 20 yrs ago! There is more on this on the OUCH! website (see useful websites).
- **What about melatonin for cluster headaches?** For those with chronic cluster headaches 10mg of melatonin at bedtime may help as prophylaxis but this is based on one small study (Wessex Drug and Medicines Information Centres, via Clinical Knowledge Summaries on NeLM).

Secondary headaches

- Secondary headaches are headaches attributable to an underlying pathology. Causes include (and this is by no means an exhaustive list!):
 - Acute close angle glaucoma
 - Carbon monoxide poisoning
 - Temporal arteritis
 - Medication overuse (see below)
 - Raised intracranial pressure
 - Neck pain.
- **Patients with headaches and red flags should be referred for further assessment.**
- SIGN guidance reminds specialists to be aware that incidental findings are common on CT/MRI and raise ethical dilemmas and patient anxiety.

Medication overuse headache

- **Headache present 15 or more days/month which has developed or worsened while taking regular symptomatic medication.**
- **Overuse of opioids and triptans most likely cause but ALL analgesia can do this.**
- Consider whether patient has developed dependence on medication.
- **Withdrawal is the means to a cure, but warn the patient that the headaches will get worse before they get better.**
 - Abrupt withdrawal of analgesia recommended for triptans & non-opioid analgesia
 - Gradual withdrawal for opioids.
- **If after withdrawing analgesia headache persists, use prophylactics.**

I think medication overuse headaches are probably under recognised, and this helpful review in the Drugs and Therapeutics Bulletin highlighted some important points (DTB 2010;48:2-6).

The DTB reminds us that you must have a problem with headaches to get medication overuse headaches – they don't happen if you are taking analgesia for arthritis for example.

The SIGN guidance pointed out that a good history is critical to making the right diagnosis and therefore offering the correct treatment. Unfortunately the DTB review points out that medication overuse headaches often mimic other headaches so the character of the pain is less helpful – the frequency and use of analgesia should be the give away. Also note that those with medication overuse headache are more likely to have symptoms of depression, irritability, poor concentration, weakness or nausea.

Why do people get medication overuse headaches?

- It is thought the cause is due to changes in neural pain pathways. Analgesics that contain codeine or caffeine are more likely to cause problems – possibly because of their addictive properties. Regular triptan use probably causes down regulation of serotonergic pain-reducing systems therefore increasing the headaches.

Preventing medication overuse headaches

- Monitor the use of medications for migraine and analgesia for headaches.
- Ensure patients know that using triptans for >10d a month or analgesics on >15d a month increases the risk of medication overuse headaches. If they feel they are needing more than this then ask them to come and see you to discuss optimising prophylaxis, rather than increasing reliever therapy.
- A Cochrane review showed acupuncture reduces analgesic use as much as prophylaxis in migraine. It was only a small review (241 patients) but food for thought in those who are struggling with side effects or who have previously had medication overuse headaches.
- **Treatment is to stop analgesics, as outlined above.** How quickly should the headaches improve on stopping the drugs? The DTB suggests:

For simple analgesia:	2-3 weeks
For opioids:	2-4 weeks
For triptans:	7-10 days.
- **Failure of therapy (withdrawal of drugs fails to reduce headaches) more likely if:**
 - People have been on therapy for a long time.
 - People are on drugs other than triptans (medication overuse headaches with triptans have a relatively good prognosis).
 - Underlying headache is a tension headache.
 - High levels of self-reported bodily pain or poor sleep.
- **Managing withdrawal symptoms**
 - **Nausea and vomiting** are common, antiemetics can be used.
 - **NSAIDs are recommended by some** although there have been no studies of this. Some recommend a course of 3-4 weeks either prn or regularly, whilst others suggest a reducing dose over 6w (250mg tds for 2w, bd for 2w then od for 2w).
 - Prednisolone: studies are mixed and tiny (<100 patients).
 - Triptans have also been used to cover withdrawal symptoms, but again the trials are small (50 in each arm).
 - **Remember, that once you have got them off the causative drug you need to address the underlying headache.** Use prophylactics not relievers.
 - **Once successfully off the causative drug, make sure the patient knows to avoid using it more than twice a week or they risk the medication overuse headaches recurring.**
 - **Relapse is common:**

30% by 6m
40% at 1 year
45% 4 years.

Now back to the SIGN guidelines...

Headaches during menstruation/menopause and with contraception

Menstruation

- Around menstruation migraines can be more frequent and more severe.
- Acute treatment for migraine related to menstruation: mefenamic acid or aspirin, paracetamol and caffeine. Triptans can be used if analgesia insufficient.
- Prophylaxis can be used: frovatriptan (2.5mg/day) or naratriptan (1mg bd) for 2d before onset of menstruation and for a further 4-5d.

Menopause

SIGN recommend that migraine is not a contraindication to HRT use, but that HRT may make migraines worse.

Contraception

- In migraine with aura the COCP increases risk of stroke: relative risk 9 (CI 5-15).
- The COCP in those >35y with migraine without aura also increases stroke risk.

SIGN recommend therefore that:

- **Women with migraine with aura of any age should not use the COCP.**
- **Women over 35 with migraine without aura should not use the COCP.**

Take home messages: Headaches

- **Look out for sinister features – investigate/refer promptly if identified.**
- **History is critical. Do a neurological examination, fundoscopy, blood pressure and exclude neck pathology in all patients.**
- **In migraine, early treatment is crucial to successfully abort an attack. Almotriptan is the cheapest effective triptan. Do not routinely use opioids.**
- **Beta blockers should be used first line for prophylaxis. Antidepressants and antiepileptic are alternatives.**
- **Migraines usually improve during pregnancy, but may get worse in first trimester.**
- **For tension headaches aspirin and paracetamol are recommended for acute management, with tricyclics for prophylaxis if needed.**
- **Cluster headaches are rare and require referral.**
- **Medication overuse headaches can occur with any analgesia, although they are more common with opioids and triptans.**
- **A thunderclap headache may indicate serious pathology: refer immediately.**
- **Because of the increased risk of stroke the SIGN guidelines recommend that the COCP should not be used by women of any age with migraine with aura, and women over 35 with migraine without aura.**

CPD Ideas

How well do I take a history for headaches? Does my management match up with that in the SIGN guidelines?

Useful websites

For professionals:

www.mipca.org.uk is the Migraine In Primary Care Advisers site and has some useful algorithms and guidelines.

For patients:

For migraine there are two main sites for patients the Migraine Action Association (www.migraine.org.uk) and the Migraine Trust (www.migrainetrust.org).

For cluster headaches there is a charity appropriately called OUCH! (www.ouch.org.uk).