One in 10 people have migraine. The patient’s history is the essential diagnostic tool. Treatment options include acute rescue, lifestyle strategies, alternative remedies, and prophylactic drugs. Most patients are managed in primary care; many never consult a doctor. The triptans have improved acute treatment, and renewed scientific interest in migraine. Overuse of acute rescue medication can lead to chronic daily headache.

**PATHOPHYSIOLOGY**

Spontaneous overactivity and abnormal amplification in pain and other, predominantly sensory, pathways in the brainstem, leads to migraine. Current opinion favours a primarily neural cause, involving feedback loops through innervation of cranial arteries in the trigeminovascular system. A relative deficiency of 5-hydroxytryptamine (5-HT) may be near the root cause, and is linked to the action of most drug treatments. Ongoing research is studying the relevance of calcium channel abnormalities, and peptides such as calcitonin gene related peptide, which may be closer than 5-HT to the underlying cause, offering hope for improved treatment in the future. Migraine is usually polygenic. Uncommon migraine variants, such as familial hemiplegic migraine and CADASIL, are single gene disorders. These are neurodegenerative, not primarily headache conditions.

**DIFFERENTIAL DIAGNOSIS**

Migraine is typically manifest by episodic disabling headache, though it is more than just head pain. Differential diagnosis is from tension type headache (TTH), with which migraine is co-morbid (this differential is also discussed from the other perspective elsewhere in this supplement). Migraine lasts hours or days, and is absent more often than it is present; the average attack frequency is once a month. TTH is often chronic and it is present more often than it is absent. Migraine should be distinguished from cluster headache, which is relatively rare and causes recurrent unilateral headache with autonomic dysfunction. The third, common though often challenging differential diagnosis is medication overuse headache (MOH). This typically complicates migraine which is then transformed into a chronic daily headache similar to chronic TTH often with some migrainous features.

**DIAGNOSIS**

The International Headache Society criteria’ are very helpful in the diagnosis of migraine. An abbreviated version is shown in table 1. These criteria can be too restrictive and therefore may be interpreted flexibly by experienced clinicians. There are two main types of migraine: migraine without aura (MO), and migraine with aura (MA). Many people have both; MO is at least three times as common as MA. Note that family history, trigger factors, and treatment response have no additional diagnostic value.

Migraine without aura

Formerly called common migraine, the diagnosis of MO is suggested by a history of episodic disabling headache lasting between a few hours and a few days, accompanied by gastrointestinal symptoms or by heightened special senses. The International Headache Society criteria may be fulfilled when pain is mild or generalised: it does not have to be severe or unilateral. It is unusual to be able to distract oneself from MO with exercise or hard work, in contrast to TTH. The frequency and periodicity of migraine is important: migraine-like headache more than twice every week is unlikely to be MO alone, but it may be MO complicated by MOH and/or TTH. This is common in patients referred with “intractable migraine” or “status migrainosus”.

Migraine with aura

In MA, formerly called focal or classical migraine, the aura evolves over time, usually many minutes; one aspect of the aura improves while another is deteriorating. Visual aura usually leads to easy diagnosis. Auras affecting sensation, movement, cognition, vestibular function, or consciousness may be difficult to distinguish from thromboembolism, or from epilepsy (especially occipital seizures). People presenting with recent onset MA often give a longer history of MO, mistakenly
of attack? People with migraine and TTH will usually volunteer then migraine is unlikely. Can they identify more than one type a patient does during an attack. If they can carry on as normal a patient who retreats to the dark and quiet. Enquire what the queasiness. Light and noise sensitivity may not be described by symptoms: many who deny nausea will readily acknowledge pain: a diary can be very helpful. Ask carefully about other Establish the duration, frequency and periods of freedom from the history. This rarely takes more than a couple of minutes.

Table 1 Abbreviated International Headache Society criteria for the common primary headaches

<table>
<thead>
<tr>
<th>Migraine without aura</th>
<th>Migraine with aura</th>
</tr>
</thead>
<tbody>
<tr>
<td>Duration 4 hours to 3 days</td>
<td>At least 3 of the following:</td>
</tr>
<tr>
<td>Nausea/vomiting and/or light and noise sensitivity</td>
<td>Reversible focal brainstem or cortical dysfunction</td>
</tr>
<tr>
<td>Two of the following:</td>
<td>Aura develops over &gt;4 minutes, or 2 auras in succession</td>
</tr>
<tr>
<td>Unilateral pain</td>
<td>Each aura &lt;60 minutes</td>
</tr>
<tr>
<td>Moderate or severe intensity pain</td>
<td>Headache &lt;60 minutes following aura</td>
</tr>
<tr>
<td>Aggravation by simple physical activity</td>
<td>Pulsating pain</td>
</tr>
</tbody>
</table>

Episodic tension-type headache
- Duration 30 minutes to 7 days
- At least 2 of the following:
  - Mild or moderate intensity pain
  - Bilateral pain
  - No aggravation by simple physical activity
  - Pressing or tight (non-pulsating) pain
  - No nausea/vomiting, may have light or noise sensitivity (not both)

Chronic tension-type headache
- >15 days pain per month, for >6 months
- At least 2 of the following:
  - Mild or moderate intensity pain
  - Bilateral pain
  - No aggravation by simple physical activity
  - Pressing or tight (non-pulsating) pain
  - No vomiting, one only of nausea, light sensitivity, noise sensitivity

All of the above diagnostic criteria require the exclusion of secondary (that is, pathologically verifiable) causes for pain. This is normally achieved on clinical grounds.

Table 2 Suggested brief central nervous system examination for routine, outpatient headache practice

| Romberg’s sign |
| Tandem gait |
| Drift of outstretched hands |
| Finger–nose test |
| Finger dexterity |
| Binocular visual fields, to confrontation |
| Eye movements |
| Facial weakness |
| Papillary responses and Horner’s syndrome |
| Tendon reflexes and plantar responses |
| Fundoscopy |

diagnosed as “bilious attacks”, “sinusitis”, or “normal headaches”. Visual symptoms are positive (seeing things which are not there), homonymous and binocular, though some patients insist that visual aura is monocular, raising the possibility of retinal origin. Aura typically precedes migraine headache, though can occur at any time in relation to pain. Aura is not always contralateral to pain. Migraine aura without headache is common, especially in middle age: a flurry of MA episodes without headache often triggers referral fearing transient ischaemic attacks (TIA). Thromboembolism may be accompanied by headache (especially with vertebral or carotid dissection, in which pain usually precedes impairment), but is distinguished from MA by abrupt, non-evolving associated impairment, confined to a single vascular territory. MA is much more common than TIA at all ages: around 40 years of age, MA is about 2500 times more prevalent than TIA, and is still about 15 times more common at 70.

HISTORY
The patient’s spontaneous account is the most important part of the history. This rarely takes more than a couple of minutes. Establish the duration, frequency and periods of freedom from pain: a diary can be very helpful. Ask carefully about other symptoms: many who deny nausea will readily acknowledge quiescence. Light and noise sensitivity may not be described by a patient who retreats to the dark and quiet. Enquire what the patient does during an attack. If they can carry on as normal then migraine is unlikely. Can they identify more than one type of attack? People with migraine and TTH will usually volunteer the migraine history, but may say that their daily muzzy head is the more debilitating symptom.

Document previous and current treatment strategies: do not be stumped by “I’ve tried everything and nothing works”. Note drugs, doses, and duration: many take ineffective or weak agents, at insufficient dose, at inappropriate times. Overuse of acute medication is often the reason why prophylaxis is ineffective. This is revealed only by cunning enquiry. Those on repeat daily doses of acute rescue drugs often disclose: “I only use it when the pain is bad” or “I keep it to a minimum”. Efficacy is characteristically reported as “it takes the edge off it”. Establish frequency and quantity of prescription or purchase (for example, “200 Co-proxamol a month”; “12 extra-strong painkillers twice a week”). Stash lists may be revealing: those who are biochemically or emotionally dependent store analgesics in their handbag or briefcase, the bedroom, bathroom, kitchen, office or car.

EXAMINATION
The main goal of examination is to consider structural brain disease. It also provides an opportunity to screen for co-morbid disease such as hypertension and depression (neither of which commonly cause headaches), and to reassure the patient, their family, and the referring doctor.

The “full neurological examination” is not possible in the time available in primary or routine secondary care, nor is it often necessary. Table 2 lists the author’s personal screen, which has been criticised both for its brevity and its length. Papilloedema and ataxia are probably the two most important physical signs.

INVESTIGATION
Almost everyone with migraine needs no investigation. The goal of investigating is to exclude other causes of migraine-like symptoms, not to confirm migraine—which tests can never do.

Abbreviations

- 5-HT: 5-hydroxytryptamine
- MA: migraine with aura
- MO: migraine without aura
- MOH: medication overuse headache
- NNT: number needed to treat
- NSAIDs: non-steroidal anti-inflammatory drugs
- OCP: oestrogen-containing contraceptive pill
- TIA: transient ischaemic attack
- TTH: tension type headache
Imaging

A “brain scan” is often requested by the patient or the non-specialist. Imaging may reassure and, conversely, may generate concern. Arguably, every healthy head should be imaged. This would result in an impossible workload: 18% of women and 6% of men have migraine; 3% have chronic daily headache. If imaged once with normal results, when should imaging be repeated? The author’s experience is that about one third of UK headache patients in secondary or tertiary care are imaged, two thirds of which occurs before neurological referral. In practice it can be difficult and unhelpful to resist demand for imaging in a patient centred health care system. Magnetic resonance imaging is always preferable to computed tomography because of better resolution and lack of exposure to ionising radiation, except for emergency presentation with possible cerebral haemorrhage. Table 3 provides some suggested imaging strategies.

Other tests

Older people with new headache should have their erythrocyte sedimentation rate assessed to address the possibility of giant cell (temporal) arteritis, which is uncommon in headache practice. A chest x ray should be considered in smokers, or in those who may have metastatic cancer.

TREATMENT

Rest is important for almost all migraine attacks; sleep, if possible, can abort migraine. Some people with infrequent or relatively mild attacks may prefer simply to rest while the migraine attack settles naturally, rather than take medication. It is important to differentiate between acute rescue, and prevention using lifestyle or drugs. Ask your patient which of these they would like to discuss first. Guidelines have been published in the UK and the USA.

Acute rescue

Analgesics and antiemetics

Analgesics and antiemetics are effective for many migraines. Impaired absorption from gastric stasis during migraine is helped by the addition of a prokinetic antiemetic (even if nausea or vomiting are not prominent), and by the use of a large dose of aspirin, 900–1200 mg, dissolved before ingestion, and taken as soon as a migraine appears to commence. Some patients prefer a non-steroidal anti-inflammatory drug (NSAID), or paracetamol 1000 mg. Fixed drug combinations of aspirin or paracetamol, with an antiemetic, are expensive. Domperidone has fewer side effects than other antiemetics, and is available over the counter. Rectal antiemetics or NSAIDs are worth considering.

Opiates should not be used because they worsen gastrointestinal symptoms, and have abuse potential especially in the genesis of MOH.

Triptans

Five triptans are currently licensed in the UK. They are expensive (table 4) and are only slightly more effective than simple analgesia with an antiemetic, on the number needed to treat (NNT) basis. These data conceal substantial inter- and intra-patient variation. Some people respond very well to triptans, and poorly to other agents; response varies from attack to attack. Triptans should be used only to treat migraine pain of at least moderate severity; they should not be used during prodrome, or during aura in anticipation of headache. Three meta-analyses of triptans allow comparison of efficacy and tolerability. Rizatriptan is consistently the most effective, though almotriptan has a good balance between tolerability and efficacy. Sumatriptan (Imigran) was the first to market in the UK and is familiar to most doctors. Sumatriptan is the only injectable triptan, offering the most rapid, effective, and expensive treatment; the nasal spray is relatively rarely used. Naratriptan (Naramig) is the least potent, but is well tolerated. Zolmitriptan (Zomig) has potentially the lowest NNT (at the extreme of the 95% confidence interval), using its top dose of 5 mg. Rizatriptan (Maxali) has the lowest mean NNT, and the lowest cost per effectively treated migraine. Almotriptan (Almogran) costs less than the other triptans, with good sustained freedom from pain, and tolerability equivalent to naratriptan.

Mouth dispersible preparations (Maxalt melt and Zomig rapimelt) have no proven advantage over tablets; they are not absorbed through the buccal mucosa. This formulation may be intuitively patient friendly, without cost disadvantage.

The potential cardiac toxicity of triptans has caused concern, which has rarely been realised in clinical practice. Triptans, however, should not be used in those with, or at risk of, cardiac ischaemia. The problems with triptans are:

- cost
- incomplete delayed benefit
- recurrence of migraine
- tendency for a minority, perhaps as many as 10%, to overuse triptans.

There is certainly no place for near daily triptan use, though such users are not uncommon in secondary and tertiary care.

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**Table 3** Brain imaging strategies for headache patients

| First, worst, thundertonclap headache CT (emergency) |
| Exertional, cough or valsalva MRI |
| Headache + signs suggesting brain lesion MRT or CT |
| Headache + known malignancy MRI or CT with contrast |
| New headache in older person MRI or CT |
| Headache, not MO, MA, TTH, MOH or cluster MRI or CT |

CT, computed tomography; MA/MO, migraine with/without aura; MOH, medication overuse headache; MRI, magnetic resonance imaging; TTH, tension type headache.

**Table 4** Triptan costs

<table>
<thead>
<tr>
<th></th>
<th>Cost per tablet</th>
<th>NNT*</th>
<th>Cost per pain-free patient</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sumatriptan 50 mg</td>
<td>£4.70</td>
<td>4.0</td>
<td>£15.92</td>
</tr>
<tr>
<td>100 mg</td>
<td>£8.00</td>
<td>4.3</td>
<td>£34.07</td>
</tr>
<tr>
<td>Naratriptan 2.5 mg</td>
<td>£4.00</td>
<td>9.2</td>
<td>£36.75</td>
</tr>
<tr>
<td>Zolmitriptan 2.5 mg</td>
<td>£4.00</td>
<td>4.9</td>
<td>£19.49</td>
</tr>
<tr>
<td>5 mg</td>
<td>£8.00</td>
<td>3.3</td>
<td>£26.25</td>
</tr>
<tr>
<td>Rizatriptan 10 mg</td>
<td>£4.46</td>
<td>3.1</td>
<td>£13.85</td>
</tr>
<tr>
<td>Almotriptan 12.5 mg</td>
<td>£3.25</td>
<td>4.6</td>
<td>£15.00</td>
</tr>
</tbody>
</table>

*Number needed to treat to achieve one pain-free patient 2 hours post-dose.
Table 5: Prophylactic drug treatments for migraine

<table>
<thead>
<tr>
<th>Drug</th>
<th>Start dose (mg)</th>
<th>Maximum dose (mg)</th>
<th>Side effects, comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amitriptyline</td>
<td>5–10</td>
<td>200</td>
<td>Sedation and dry mouth</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Consider other tricyclics; SSRIs</td>
</tr>
<tr>
<td>Propranolol</td>
<td>20</td>
<td>320</td>
<td>Cold limbs, nightmares</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Atenolol may be better tolerated</td>
</tr>
<tr>
<td>Pizotifen</td>
<td>0.5</td>
<td>3</td>
<td>Weight gain, sedation, low efficacy</td>
</tr>
<tr>
<td>Valproate</td>
<td>500</td>
<td>1500</td>
<td>Weight gain, teratogenicity</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Consider gabapentin, topiramate</td>
</tr>
<tr>
<td>Methysergide</td>
<td>1</td>
<td>8</td>
<td>Limb pain, visceral fibrosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>6 months treatment, 1 month break</td>
</tr>
</tbody>
</table>

Many of these drugs are unlicensed in this indication. SSRIs, selective serotonin reuptake inhibitors.

headache practice. Such patients often have an established history of analgesic or ergot overuse, and triptans should be used with caution in such cases. Management is the same as for any other MOH: cure follows drug withdrawal, and is not facilitated by further prescriptions.

**Ergots**
Ergot alkaloids may still have an occasional place in the acute management of migraine. In headache clinics, ergot users usually have ergot dependency—yet another type of MOH.

**Other acute rescue treatments**
Unlicensed options for refractory cases include high dose oxygen (100% if possible), parenteral steroids (for example, dexamethasone 4 mg), and parenteral phenothiazines (for example, chlorpromazine 5–50 mg).

**Recurrence**
Recurrence after initial improvement affects a third of migraineurs. This is a complex and poorly understood phenomenon: at the simplest level, an ineffective treatment has zero recurrence. Recurrence risk with different triptans does not simply reflect drug half life. It is arguable that triptans access their site of action only when there is migraine pain—so they terminate their own action. Some authorities try to reduce recurrence with the combination of an NSAID with triptan treatment, though this is not evidence based.

**Step care versus stratified care**
Step care means starting with simple analgesia, usually with an antiemetic, escalating in one or more steps toward triptans. With stratified care, low impact migraine is treated with simple analgesia and an antiemetic, but high impact migraine is treated first with a triptan. Stratified care results in significantly better clinical outcomes than step care,44 but at a higher price. This comparison assumes that the two strategies are equivalent. It is arguable that analgesia/antiemetic is appropriate to use very early in a migraine attack, perhaps before pain is significant—for example, during aura or prodrome, a time when triptans are known to be ineffective.

**PREVENTION OF MIGRAINE**

**Lifestyle management**
Management of lifestyle can appear to be very helpful, though evidence is largely anecdotal. Regularity of biorhythms is the key. The avoidance of relative hypoglycaemia, with a regular, fibre containing diet is probably the most helpful strategy. Dietary exclusion is rarely helpful, and should be abandoned if ineffective. Change in sleeping times at weekends and irregular shift work may usefully be avoided, as is the abrupt let-down from stress. Trigger factors often summate—for example, at times of international travel.

**Alternative therapies**
These are often acceptable to patients though are rarely evidence based. Large doses of vitamin B2, and magnesium, may be effective with several months’ latency to benefit. Manipulative treatments seem to be helpful for soft tissue pain or tenderness. Homeopathy is ineffective.41 Acupuncture probably has only an acute analgesic effect.

**Prescribed drugs**
Daily drug treatment to prevent migraine should be considered after acute treatment has been optimised, medication overuse abolished, lifestyle modification tried, and a migraine diary recorded for a month or three. Most agents offer partial benefit only, which may take 1–5 months to achieve; those most commonly used are listed in table 5. Patients must be carefully advised that the occurrence of migraine after starting prophylaxis does not mean treatment failure. Co-morbid disease may suggest initial drug choice. It is unusual to offer prophylaxis for less than two attacks a month. Treatment should be titrated first for tolerability, then for efficacy. Cycle through single drug regimens to find the best agent, with ongoing diary monitoring; occasionally, combine partially successful drugs. After six months of effective treatment, phased withdrawal should be considered.

**Hormones and migraine**
Migraine is more common in women, the sex difference beginning at puberty. Menstruation is a migraine trigger in 10% of women with migraine. This is often overestimated by the patient: true menstrual migraine can be diagnosed only after examining a few months of the headache and menstrual diary. The oestrogen-containing contraceptive pill (OCP) may lead to an improvement, but this ameliorating effect is then lost during the pill-free week. Tricycling the contraceptive pill, and using transdermal oestrogen, can be helpful.

Migraine, especially MA, is possibly an independent risk factor for stroke, though confidence intervals of case–control studies are wide, making risk assessment difficult. For example, one study quotes an odds ratio of 34.4 for stroke among migraineurs who smoke and use the OCP but the 95% confidence interval is 3.3 to 361! This leads many authorities to
question the wisdom of OCPs for women with migraine. The
risk is probably very low in MO. MA is a relative contraindi-
cation to the OCP. Intradermal devices and progesterone only
contraceptives affect neither migraine nor stroke risk, so are
preferable to OCPs for women with any form of migraine.
Migraine typically improves during the second and third
trimesters of pregnancy, though can be troublesome in the
puerperium.

The climate and menopause are associated with worsening
of migraine, as often as with improvement. Transdermal, not
oral, hormone replacement therapy is often helpful, though
high doses may trigger MA.

MIGRAINE IN CHILDREN
The prevalence of migraine in childhood rises with age; under
12 years, it is more common in boys. Compared with adult
migain, attacks are shorter (as short as two hours), pain is
seldom unilateral, and aura is less common. Children do not
usually describe light or noise sensitivity though this is some-
times inferred from their behaviour; they can rarely describe
the pain as throbbing or pulsating. Gastrointestinal symptoms
are more prominent than in adults. Head pain is absent in the
migraine variants—cyclical vomiting and abdominal migraine
in which the attack is identical to MO except that pain is in the
abdomen, not the head.

Management must address the concerns of the child,
parents, and sometimes teachers as well—which may mean
three different agendas. The child's estimate of attack
frequency may be different from the parent's, and neither may
tally with frequency of days off school. A diary is invaluable
in such cases.

Acute rescue medication may be unhelpful for short
attacks; otherwise, ibuprofen is preferable to paracetamol
(aspirin is contraindicated in children under 12 years). The
child should be allowed to rest in a quiet place, and to resume
normal activities as soon as this is comfortable. It is rarely
necessary for the child to be sent home from school, though
teachers may disagree. Zolmitriptan may be used from the age
of 12 years. Sumatriptan nasal spray is safe and effective,
though unlicensed in children.

Preventative strategies should focus on lifestyle and diet,
especially ensuring regular adequate sleep, and three meals
containing fibre a day (constipation may cause headache in
children). Dietary exclusion is less disappointing in children
than adults, though this must be carefully negotiated and
foods reintroduced if migraine is unchanged.

Drug prophylaxis is occasionally necessary. Pizotifen seems
to be better tolerated and more effective in children than in
adults. β Blockers are typically the drugs of second choice.
In refractory cases, the same drugs that are used in adults may be
considered.

POST-TRAUMATIC MIGRAINE
All forms of primary headache, especially migraine, may
appear or become worse after head injury, or after more
controlled forms of trauma, such as neurosurgery. MOH is
common in these patients, who are typically offered combina-
tion analgesics as inpatients, which are continued on
discharge. Management is identical to that for patients in
whom there is no history of trauma.

In medicolegal practice, claimants state that migraine has
been triggered by minor head injury and deny any migraine
before injury. This history must be corroborated by review of
primary care notes, and of employment records as time may be
missed from work because of migraine that does not present
to the general practitioner. Where the records cast no doubt
upon the patient's history, post hoc proper hoc is an argument
whose weakness increases with time. It seems reasonable to
attribute up to a year of excess migraine to mild head injury;
before that time, a causal link is less likely.

INTRACTABLE MIGRAINE
Some patients keep coming back to clinic saying that nothing
has changed, and that their migraine is just as bad as ever.
Retake the headache history; compare current frequency and
severity with that recorded in the notes. Sometimes, the
patient has forgotten just how bad things were, and has mis-
taken incomplete cure for unchanged migraine. An ongoing
diary can be very helpful in assessing change.

The most common error is failure to diagnose MOH: probe
carefully for this history, as no amount of new prescriptions
will help these challenging patients. Has prophylaxis been
taken at maximum tolerable doses for a good few months, in
the absence of MOH? Is there co-morbid depression requiring
specific management independent of migraine? Is the
headache diagnosis correct? It is possible to mistake for
migraine the trigeminal autonomic cephalgias, such as cluster
headache or chronic paroxysmal hemicrania.

Review the fine detail of acute rescue strategies:
> Are triptans being taken incorrectly during aura or
  prodomne, or shortly before vomiting?
> Are simple analgesics such as aspirin being used too late,
  without an antiemetic, or as whole tablets rather than being
dissolved first? Some authorities recommend dissolving
aspirin in a sweet fizzy drink.
> Have at least three attacks been treated with each of the
triptans, one by one, using the full range of doses and formu-
lations?
> Have a range of NSAID tablets suppositories and injections
been tried? An ergot preparation may be worth trying.
> Is the patient resting while waiting for acute treatment to
  work, or expecting benefit from a miracle treatment while
they try to carry on as normal?
> A combination of aspirin or NSAID, antiemetic, triptan, and
  hypnotic can be tried, though triptans should not be
  combined with each other.

A second opinion can be very helpful, if only to reassure the
patient that nothing important has been overlooked.

Patients who claim dissatisfaction with treatment may
change their mind when offered medication whose threat is
perceived as greater than that of migraine: “These tablets
(methysergide) can work well, but may be taken for six
months only, because they occasionally cause scarring around
the vital organs”; “Thank you doctor, I’ll stay as I am, but can
we think again at my next appointment?” This helps the
patient feel in control of treatment options, and therefore of
their migraine. The move from an external to an internal locus
of control may be critical to quality of life, even if experience
of pain is unchanged.

It is reasonable to allow patients to choose alternative or
complementary treatments even if there is no evidence to
support their choice; and, if these appear helpful, the neurolo-
gist should gracefully acknowledge this.

A tiny minority of migraineurs are truly resistant to
 treatment. The same consultation attitudes useful for other
incurable diseases are indicated. Listen to and record
symptoms, and accept how hard they are for the patient to
bear. It is amazing how grateful people can be for the simple
act of interested compassion without cure.
ACKNOWLEDGEMENTS
I am grateful to Dr David Symon for his helpful advice on migraine in children, and to Dr Anne MacGregor for permission to reproduce her cartoon.

Possible conflict of interest: The author has accepted hospitality from, and acted on advisory boards for, all the triptan manufacturers. He is treasurer of the British Association for the Study of Headache and a member of the International Headache Society.

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IMAGES IN NEUROLOGY
Colloid cyst of the IIIrd ventricle

Colloid cysts are relatively unusual (1.5%-neurosurgically treated tumours) benign intracranial masses located in the rostral aspect of the IIIrd ventricle. As they grow they obstruct the foramen of Munro producing hydrocephalus of the lateral ventricles. This can be acute and catastrophic because of the pedunculated nature of the tumour resulting in brain herniation and death. Both computed tomography (CT) and magnetic resonance imaging (MRI) will usually show the lesion and its effects on the ventricular system, but the multiplanar capability and higher contrast resolution of MRI will usually define its relations better.

Unless the tumour is an incidental finding, patients usually present with intractable headache and less commonly with nausea, short term memory loss, gait disturbance, blurred vision or coma. Non-specific neurological symptoms and signs are common but may be suggestive of raised intracranial pressure. The cause and origin of the cysts are still debated. The cyst size ranges from 2–50 mm and they contain colloidal material including cholesterol fats that are largely responsible for their appearances on MRI. Thin (<5 mm) axial sections on MRI or CT will show the lesion hanging from the roof of the IIIrd ventricle, while sagittal and/or coronal sections will show its relation with the foramen of Munro (fig 1). On CT the mass is hyper- or isodense with grey matter while on MR T1 weighted imaging it is usually hyperintense, reflecting the colloidal matrix and fatty content. T2 weighted sections show it as hyperintense or isointense with grey matter while FLAIR (fluid attenuated inversion recovery) sequences will show it as hypointense against a background of hypointense cerebrospinal fluid. Surgical treatment used to be carried out via a transcortical approach in an attempt to avoid damage to the fornix and resultant memory loss, but more recently stereoscopic and endoscopic approaches to the IIIrd ventricle have proved superior in many cases with less neurological and neuropsychological sequelae.

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