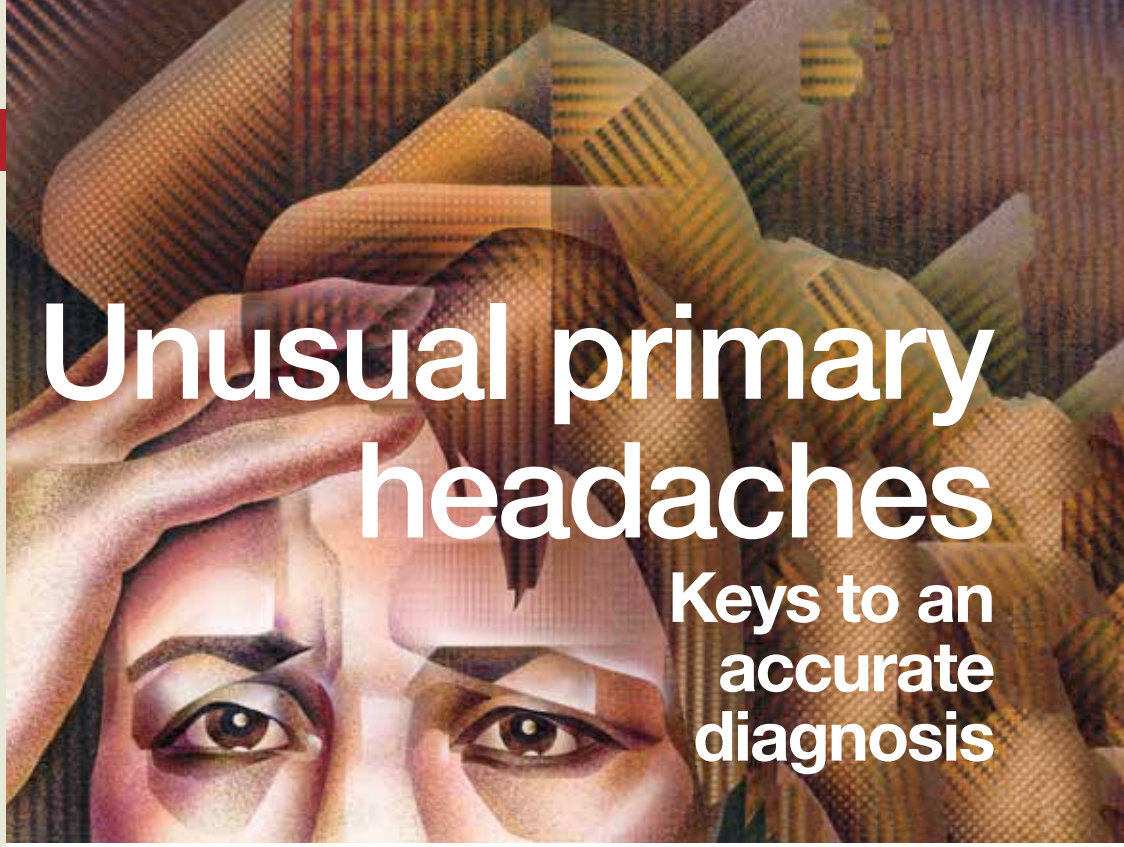
MedicineToday 2013; 14(10): 38-44

PEER REVIEWED FEATURE
POINTS: 2 CPD/2 PDP



Unusual primary headaches

Keys to an accurate diagnosis

Key points

- Primary headache disorders other than migraine and tension-type headache are less well known; however, not all are rare and they will be encountered occasionally in general practice.
- The trigeminal autonomic cephalgias (TACs) are short-lasting headaches with prominent autonomic features and should be differentiated from short-lasting headaches without autonomic features.
- Photo- and phonophobia are usually unilateral in TACs, but are bilateral in migraine, even when the pain is lateralised.
- Autonomic symptoms tend to be lateralised to the side of the pain, prominent, and consistent between attacks in TACs; in migraine, they are generally mild, bilateral and do not parallel attack severity.
- The diagnosis of other recurrent primary headache disorders is often suggested by the specific trigger factors (e.g. sexual activity, exertion, or coughing) or specific features of the pain (e.g. local, brief stabbing pain, or focal coin-shaped area of pain).

ELSPETH HUTTON MB BS, BMedSci(Hons), FRACP
RICHARD STARK MB BS, FRACP, MACLM

A patient presents to you with recurrent headaches that don't quite fit the pattern of migraine or tension-type headaches. Which disorders should you consider?

Most patients with headache have one of the primary headache disorders, with migraine and tension-type headache being the most common forms. Primary headache disorders are so common that even the less well known varieties will be seen occasionally in general practice: this paper addresses a number of these. Formal diagnostic criteria are published in the *International Classification of Headache Disorders (2nd edition)*.[1] An approach to diagnosis and investigation to exclude causes of secondary headache was published recently in *Medicine Today*.[2]

TRIGEMINAL AUTONOMIC CEPHALGIAS

The trigeminal autonomic cephalgias (TACs) fall into three groups:

- cluster headache
- short-lasting unilateral neuralgiform headache with conjunctival injection and tearing (SUNCT) and short-lasting unilateral neuralgiform headache with cranial autonomic symptoms (SUNA)
- paroxysmal hemicrania and hemicrania continua.

The TACs form a group of short-lasting headaches with prominent autonomic features that should be differentiated from short-lasting headaches without autonomic features, such as trigeminal neuralgia, hypnic headache, primary exertional headache, primary sex headache and primary stabbing headache. There appears to be a central role for hypothalamic dysfunction in the generation of TACs, with hypothalamic activation often seen on imaging studies.[3]

Autonomic features (which occur on the side of the headache) include:

- facial flushing, pallor or sweating
- redness, tearing or itchy/gritty sensation of the eye
- running or blocking of the nose

Dr Hutton is a Neurologist at The Alfred Hospital, Melbourne; and Honorary Adjunct Lecturer in the Department of Medicine and Van Cleef and Roet Centre for Nervous Diseases, Monash University. Associate Professor Stark is a Neurologist and Adjunct Clinical Associate Professor in the Neurology Department, Alfred Hospital, and the Department of Medicine, Monash University, Melbourne, Vic.

TABLE. CLINICAL FEATURES AND TREATMENT OF TRIGEMINAL AUTONOMIC CEPHALGIAS

Feature	Cluster headache	Paroxysmal hemicrania	SUNCT and SUNA
Gender predominance	More common in males	Equal	About equal
Pain type	Stabbing, boring	Throbbing, boring, stabbing	Burning, stabbing, sharp
Pain site	Orbit, temple	Orbit, temple	Periorbital
Frequency of attacks	0.5-8/day	1-40/day (usually >5/day)	3-200/day
Duration of attacks	15-180 min	2-30 min	5-240 sec
Autonomic features	Yes	Yes	Yes (especially conjunctival injection and lacrimation)
Migrainous features	Yes	Yes	Yes, in about 1/3 of cases
Alcohol trigger	Yes	No	No
Cutaneous trigger	No	No	Yes
Indomethacin effect	No	Absolute response	No
Key abortive treatments	Sumatriptan (nasal,* subcutaneous), oxygen	Nil	Intravenous lignocaine*
Key preventative treatments	Verapamil,* methysergide, lithium,* corticosteroids (short term)*	Indomethacin*	Lamotrigine,* topiramate,* gabapentin*

ABBREVIATIONS: SUNCT = short-lasting unilateral neuralgiform headache with conjunctival injection and tearing; SUNA = short-lasting unilateral neuralgiform headache with cranial autonomic symptoms.
* Off label use.

- drooping of the eyelid or change in pupillary size (miosis), or Horner's syndrome
- ear fullness
- salivation.

Patients with TCAs who have photophobia and/or phonophobia are also likely to experience these unilaterally, as opposed to bilateral sensitivity in those with migraine.[4] In addition, in contrast to the autonomic symptoms in TACs tending to be lateralised to the side of the pain, prominent, and consistent between attacks, in migraine, they are generally mild, bilateral and do not parallel attack severity.

Differentiation between the subtypes of TAC is usually possible by determining the cycle pattern and the length and frequency of the attacks. The Table illustrates the key features differentiating the types of TAC. Other potentially catastrophic causes of sudden headache should always be considered, such as aneurysmal disease and subarachnoid haemorrhage, arterial dissection and acute angle closure glaucoma (which may present with headache, tearing and redness of the eye, as well as agitation).

Pituitary gland pathology may accompany TACs, with up to 10% of patients with pituitary tumours and headache presenting with a TAC phenotype.[5] MRI with pituitary views and pituitary function testing are thus appropriate in the workup of patients with an initial TAC presentation.

Cluster headache

Cluster headache affects around 0.1% of the population,[6] with a male predominance, a higher frequency in cigarette smokers, and peak onset in the second and third decade of life.[7] The headaches are severe and boring in quality, lasting 15 to 180 minutes and typically orbital, supraorbital or temporal in location. The pain is typically described as the most severe the patient has ever experienced, rivalling childbirth. Marked agitation and restlessness are

usually associated with cluster headache, in contrast with the avoidance of movement seen during attacks of migraine.[7] Patients are usually asymptomatic between attacks.

Cluster headaches tend to occur in bouts, with the attacks within a bout linked to the circadian rhythm, occurring at the same time each day. Also, bouts exhibit a circannual periodicity; they are often more frequent in spring or autumn, around the time that the clocks change for daylight saving, and typically last about eight to 10 weeks.

Alcohol, nitrates, exercise and warmth often trigger attacks. The attacks may be associated with 'migrainous' features, such as photophobia, phonophobia, nausea and vomiting. However, the other clinical features and temporal pattern are so distinctive that diagnostic confusion between cluster headache and migraine should be rare.

Cluster headaches are classified according to the duration of the bout. Episodic forms occur in bouts lasting seven days to one year, with bouts separated by at least a one-month pain-free period, while a chronic cluster lasts for one year or longer, without remissions, or with less than one-month remission.[8]

As headache resembling cluster headache may occur secondary to a number of structural pathologies, good quality imaging with MRI focused especially on the pituitary and cavernous sinus region is prudent in all cases.

Management of cluster headaches involves both preventative and acute treatment measures. Agents to abort the acute attack include:

- 100% oxygen administered at 12 to 15 L/min via a non-rebreather mask for 15 to 20 minutes
- subcutaneous sumatriptan 6 mg
- intranasal sumatriptan 20 mg (off label use).

Oral triptans are not usually helpful because by the time they become effective the relatively short duration cluster headache is already subsiding spontaneously.

Preventative treatments are useful in patients with chronic cluster headache and may shorten the duration of the active period in those with the episodic form. Options for prevention depend on the length of the bout. Longer bouts require safe and effective agents that can be administered long term, such as verapamil, lithium and topiramate (all off label uses), whereas shorter bouts may be managed by methysergide or by prednisolone (off label use), which act quickly but should not be used for long periods.

Verapamil often needs to be used at higher than usual doses, reached by gradually increasing the dose: up to 960 mg/day in split doses of the immediate release form has been used. There is a risk of conduction block occurring with higher doses, so two-weekly monitoring with ECG after each dose increase to detect any emerging conduction block (lengthening of the PR interval) should be performed before the next increase. Six-monthly ECG can be performed when a stable dose is reached.[9]

A useful strategy for patients with episodic cluster headache is to use prednisolone (starting at 75 mg/day and weaning rapidly to zero over two to three weeks) as a means of gaining rapid control of the bout, and starting verapamil at low dose (for example, 40 mg thrice daily, increasing every week or so) at the same time. The verapamil thus starts to have an impact as the prednisolone is weaned.

Infiltration of the greater occipital nerve with corticosteroids and local anaesthetic is helpful for some patients.

Surgical options may be considered in cases that are refractory to medical treatment, and include occipital nerve stimulators[10] and posterior hypothalamic neurostimulation.[11]

SUNCT/SUNA

SUNCT and SUNA are rare TACs characterised by short-duration attacks of unilateral head pain with associated autonomic features, often triggered by cutaneous stimuli. SUNCT is defined by the presence of significant tearing and conjunctival injection, and SUNA by their absence; however, these conditions are clearly part of the same spectrum.

These headaches form a key differential diagnosis for trigeminal neuralgia. The key differentiating features of SUNCT/SUNA are the pain typically being in the ophthalmic division of the trigeminal nerve (rare for trigeminal neuralgia) and the autonomic features. Cutaneous triggers of pain paroxysms are often a feature of both trigeminal neuralgia and SUNCT/SUNA.[12] The pain of SUNCT/SUNA may be short-lived single stabs (lasting 5 to 240 seconds), groups of stabs or a saw-tooth pattern where the pain does not return to baseline between stabs and the attack lasts several minutes.

Patients with these headaches show no response to indomethacin (see paroxysmal hemicrania) or to the typical relievers for cluster headache. The most effective treatment is lamotrigine, with some efficacy also seen for topiramate and gabapentin (all off label uses). Ten days of intravenous lignocaine treatment may also induce a remission.[13]

Paroxysmal hemicrania

Paroxysmal hemicrania comprises short attacks of severe unilateral pain (lasting 2 to 30 minutes), typically in the ophthalmic division, with associated autonomic features recurring several times a day. There is often associated unilateral photophobia and phonophobia, as well as restlessness and agitation. The attacks usually occur during the day, and a small percentage are triggered by alcohol or a manual trigger, such as head movement, pressure on C4 or C5, the C2 root or the greater occipital nerve. There are no cutaneous triggers, such as seen in SUNCT/SUNA. Paroxysmal hemicrania may be episodic (with remissions lasting more than one month) or chronic (with no remission in one year).

Paroxysmal hemicrania exhibits a lasting response to indomethacin; however, in patients intolerant to the gastric effects of this medication, the alternatives are less clearly defined. COX-2 inhibitors,

topiramate and greater occipital nerve block with lidocaine and methylprednisolone acetate may be beneficial (all off label uses).[14]

Hemicrania continua

Hemicrania continua is a continuous side-locked headache of varying intensity. The exacerbations may be accompanied by ipsilateral autonomic symptoms, as well as migrainous features (nausea, photophobia, phonophobia). The background pain in hemicrania continua is greater than the interparoxysmal pain in the other TACs, and the exacerbations longer, which helps distinguish it from paroxysmal hemicrania.

There is a complete resolution of the headache with therapeutic doses of indomethacin, which is one of the diagnostic criteria. Similarly to paroxysmal hemicrania, topiramate, greater occipital nerve block and occipital nerve stimulators have all been reported to be beneficial.[15,16]

An oral indomethacin test may be performed using a regimen of 25 mg thrice daily for one week, increasing to 50 mg thrice daily in the second week and 75 mg thrice daily in the third. The patient should keep a headache diary before and during the test so changes in pain scores can be evaluated. Prophylactic use of a proton pump inhibitor is advisable to reduce gastrointestinal complications. If the test is positive, the dose can be gradually decreased by 25 mg thrice daily every few weeks until the minimal effective dose is established.

MISCELLANEOUS PRIMARY HEADACHES

There are a number of other headache syndromes with characteristic patterns, some occurring after specific triggers. These are not rare.

Primary stabbing headache

Primary stabbing headache has been called 'jabs and jolts' or the graphic term 'ice-pick pains' with which patients readily identify, as the pain is often described as being like a very local and very brief stab into the head.

The pain occurs spontaneously (unprovoked) and is predominantly felt in the distribution of the first division of the trigeminal nerve (orbit, temple and parietal area). Stabs last for up to a few seconds and recur with irregular frequency ranging from one to many times per day. The stabs are often confined to one area, but in some patients they may move about the head, including to the opposite side. Usually there are no accompanying symptoms such as nausea, light sensitivity or autonomic changes. Findings on examination (and imaging, if performed) are normal.

This condition is not rare; many patients do not bother to mention it. Others are concerned that it represents serious pathology and are reassured by a definite diagnosis. Only rarely do patients find it disabling and most do not require treatment. Many patients with primary stabbing headache have migraine as well,[17] in which case the pains are felt predominantly on the side most affected by these headaches.

The differential diagnoses would include brief forms of TAC such as SUNCT and paroxysmal hemicrania, but these would typically have associated autonomic symptoms.

A positive response to indomethacin in affected patients has been reported in some uncontrolled studies, while others have found partial or no response.

Cough headache

Cough headache is a headache of sudden onset, lasting from one second to 30 minutes, brought on by, and occurring only in association with, coughing, straining and/or the Valsalva manoeuvre.[18] In the typical case the headache is bilateral, very abrupt in onset and immediately at maximum severity. The severity then decreases gradually over 15 to 30 seconds, often with a pulsatile quality during this time.

This condition is, in about 40% of cases, associated with demonstrable obstruction to CSF flow at the foramen magnum; the most common such cause is Chiari malformation. This makes sense as the headache can be explained by failure of intracranial and intraspinal pressures to equilibrate immediately (as they normally do) after coughing or straining. The raised intrathoracic pressure is transmitted through the venous system to the intracranial cavity. If there is then a pressure gradient across the foramen magnum, pain will arise from traction on meninges, vessels and other pain-sensitive structures in the region. The pain wanes as pressures gradually equalise.

Cough headache obviously mandates careful imaging of the brain, and the region of the foramen magnum in particular. MRI provides excellent views of this area, but the radiologist must be alerted that this is the area of interest.

Patients in whom no abnormality is shown on imaging are classified as having 'primary cough headache', formerly called 'benign cough headache'. It is conceivable that radiologically inapparent obstructions at the foramen magnum may account for some of these cases. The authors report one case in which the symptoms were relieved after surgical exploration of the region showed fine bands of arachnoid tissue that were then cleared away.

Cough headache often responds to indomethacin, at least to some extent, but this may occur both in 'primary' cases and in those with demonstrable obstruction, so the response is not of diagnostic value.

Primary headache associated with sexual activity

Two subtypes of primary headache are associated with sexual activity ('benign sex headache'). The less common form is pre-orgasmic and usually starts as a dull bilateral ache as sexual excitement increases. The ache is felt in the head and neck and is associated with awareness of neck and/or jaw muscle contraction. The more common, and more severe, form occurs at orgasm and is explosive and instantaneous in onset. It is thus a form of 'thunderclap headache' and subarachnoid haemorrhage must be the provisional

diagnosis until definitely excluded.

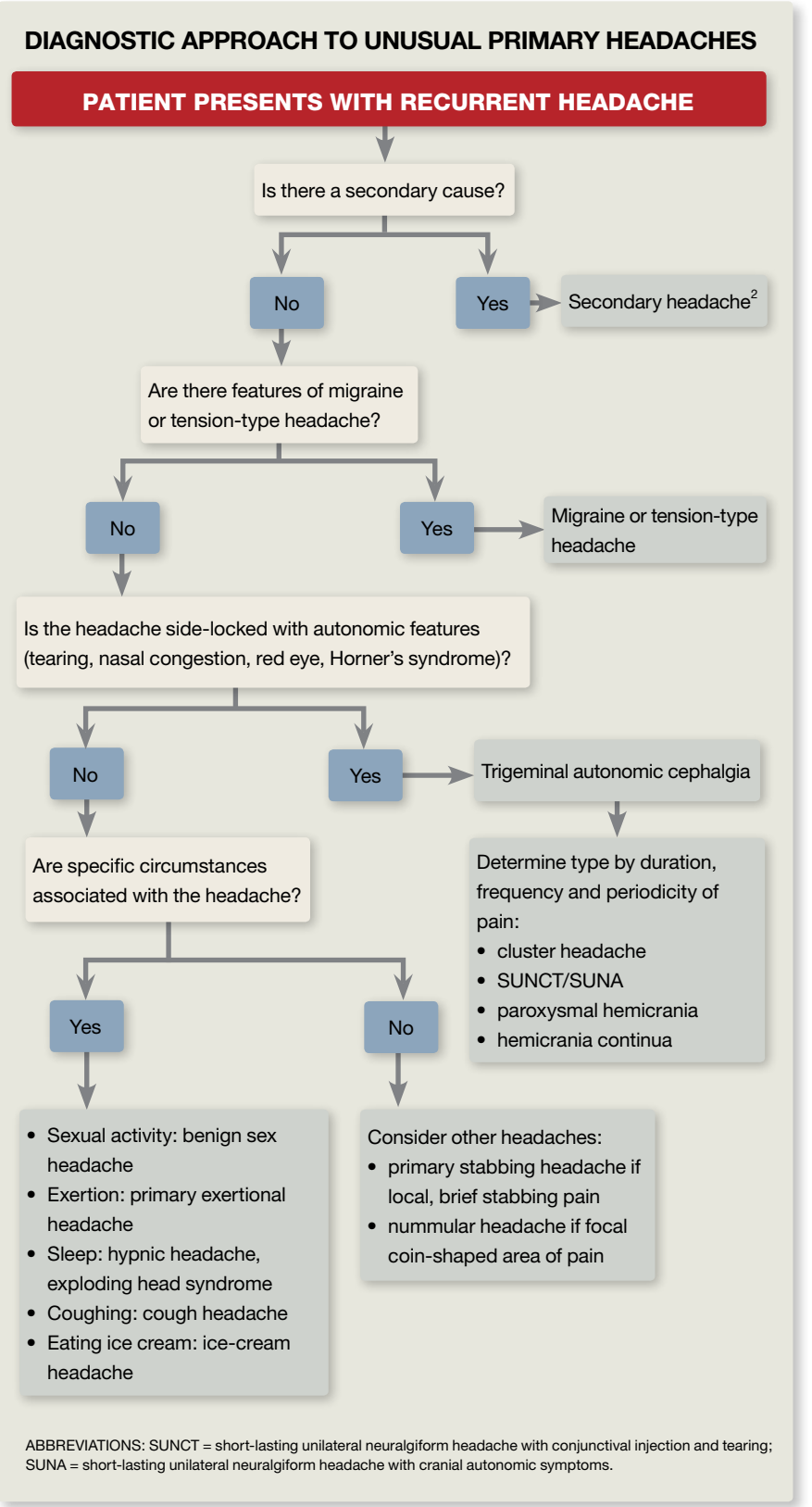
The headache of orgasmic headache is severe and patients are typically appropriately anxious about a serious cause; once this is excluded, they remain anxious about the effect recurrent headaches might have on their quality of life. The natural history of orgasmic headache is for recurrences to occur (with sexual activity or with lifting or straining) over a period of a few weeks and then for the condition to subside. During the period of susceptibility, indomethacin may provide some symptomatic relief and there is some anecdotal evidence that beta blockers (such as propranolol) or calcium channel blockers (such as verapamil) may also be of value (all off label uses).

There are many similarities between orgasmic headache and the thunderclap headache seen in reversible cerebral vasoconstriction syndrome (RCVS). RCVS may cause single or recurrent episodes (over a few weeks) of instantaneous headache and is diagnosed when computed tomography angiography, magnetic resonance angiography or other vascular imaging shows vasospasm. It may be triggered by various stimuli, often of an adrenergic nature, (such as cocaine or amphetamine use) but also by sexual activity.

It is tempting, therefore, to conclude that many cases of orgasmic headache may be a version of RCVS. In a recent study in which detailed vascular imaging was carried out in 30 patients with orgasmic headache, 60% had evidence of RCVS.[19] Furthermore, the clinical features of those without vasospasm on imaging were indistinguishable from those with vasospasm. It seems, therefore, that there is considerable overlap between the two conditions.

Primary exertional headache

Primary exertional headache, also called benign exertional headache, is probably heterogeneous. Migraine may also be provoked by exertion (or perhaps by the associated fluid depletion). Some exertional headache is explosive in onset and is triggered by sudden straining or the Valsalva manoeuvre (for example with



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weightlifting) and thus has similarities to orgasmic headache and cough headache. Other cases develop more gradually in response to more prolonged exertion.

Some degree of exertional headache is not rare. One survey found 30% of adolescents reported headache during or following exertion. The headaches were often bilateral, pulsating and brief (lasting less than an hour).[20] Migraine predisposed to exertional headache, with 47% of those with migraine reporting it compared with 21% of those without.

The pathophysiology of primary exertional headache is unknown. One group has postulated that excessive transmission of venous pressure to the intracranial space following Valsalva-like manoeuvres may contribute.[21] They found internal jugular vein valve incompetence using duplex ultrasound in 70% of patients with exertional headache and 20% of controls.

Hypnic headache

Hypnic headache is a rare primary headache disorder occurring mainly in older patients (average age of onset is about 60 years), characterised by exclusively sleep-related headache attacks.[22] The headache may be unilateral or bilateral and is usually severe or at least moderately severe. The headache attacks are very disruptive as they typically occur once or twice each night and most patients have to get up and move about; many patients then drink coffee as caffeine appears to provide some relief.

Prophylactic treatments that are sometimes effective include coffee before going to bed, lithium, topiramate, indomethacin, melatonin and amitriptyline (all off label uses).

Nummular headache

Nummular headache is a rare primary headache disorder in which there is a focal 'coin-shaped' circumscribed area of pain, 2 to 6 cm in diameter (from the Latin *nummulus*, a small coin).[23] Nummular headache is typically persistent in type with some superimposed exacerbations of more severe pain (which is still usually described as a dull ache). There is only rarely sharper pain or local tenderness.

No treatment is reliably effective. There is an anecdotal report of benefit from botulinum toxin injection in a small number of patients.[24] The natural history is very variable: some cases resolve spontaneously but others may continue for years.

Exploding head syndrome

Sufferers of exploding head syndrome describe alarming attacks of a painless explosion within their head. Attacks tend to occur at the onset of sleep, even at the start of daytime naps.

This condition is probably not rare: when Pearce first described 10 cases with this evocative name, it received substantial press coverage and he was then inundated with correspondence describing similar cases, so that within a year he was able to publish a review of 50 well-documented cases.[25] The cause is uncertain but the timing suggests a similarity to the other physiological phenomena, such as nocturnal myoclonus, that mark the transition from wakefulness to stage 1 sleep.

No reliable treatment is known but clomipramine has been suggested anecdotally.

Ice-cream headache

Ice-cream headache is so common that it is almost a normal phenomenon. It is termed officially (but more prosaically) as 'headache attributed to ingestion or inhalation of a cold stimulus'. The short-lasting pain, which may be severe, is induced in susceptible individuals by the passage of cold material over the palate and/or posterior pharyngeal wall.

Ice-cream headache affects about 40% of adolescents.[26] Like exertional headache, it occurs more often in individuals with migraine: in one study, 55% of people with migraine reported ice-cream headache compared with 29% of those with no background history of headache.[26] In another study cold-induced headache was triggered experimentally in 74% of people with migraine (who often reported unilateral throbbing pain) and in only 32% of patients with tension-type headache (whose pain was usually bilateral and not throbbing).[27]

SUMMARY

Although some of the primary headache disorders are relatively uncommon, GPs will at times see patients with these conditions. The strictly unilateral nature and associated autonomic features of the TACs are strong diagnostic clues. For many of the other headaches, the specific trigger factors (sexual activity, exertion, sleep or coughing) would suggest the correct diagnosis. The flowchart on page 43 provides a diagnostic approach to patients presenting with recurrent headaches. MT

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A list of references is included in the website version (www.medicinetoday.com.au) and the iPad app version of this article.

COMPETING INTERESTS: Associate Processor Stark has received lecture fees from Allergan, Janssen-Cilag, MSD and Pfizer, and consultancy fees from Allergan, Janssen-Cilag and Pfizer. Dr Hutton: None.

Unusual primary headaches

ELSPETH HUTTON MB BS, BMedSci(Hons), FRACP, **RICHARD STARK** MB BS, FRACP, MACLM

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