

FAST FACTS

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*Indispensable
Guides to
Clinical
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Headaches

Second edition

by Richard Peatfield and David W Dodick

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and tension headaches 34

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Glossary

Arnold–Chiari malformation: a congenital disorder in which the base of the brain is distorted and the lower brainstem and part of the cerebellum protrude through the opening at the base of the skull

Aura: a premonitory, subjective sensation preceding an attack of migraine or epilepsy

AVM: arteriovenous malformation. Cluster of distended blood vessels that press on the brain

BIH: benign intracranial hypertension

CADASIL: cerebral autosomal-dominant arteriopathy with subcortical infarcts and leukoencephalopathy

CBF: cerebral blood flow

CDH: chronic daily headache

CGRP: calcitonin gene-related peptide

CH: cluster headache

CSF: cerebrospinal fluid

CT: computed tomography, a radiological method of imaging soft tissues

CVST: cerebral venous sinus thrombosis

Diplopia: double vision

Hemianopia: condition where half the normal field of vision is absent

Horner's syndrome: drooping of an upper eyelid, constriction of the pupil, and an absence of sweating over the affected side of the face. The syndrome is caused by a nervous disorder in the brainstem or neck

IHS: International Headache Society

5-HT: 5-hydroxytryptamine or serotonin, a widely distributed biochemical that may play a role in the modulation of craniovascular pain

MRI: magnetic resonance imaging, a non-radiological method of obtaining a cross-sectional image of tissues and systems

Neuralgia: a painful affection of the nerves

NO: nitric oxide, an endogenous mediator of physiological vasodilation

NSAIDs: non-steroidal anti-inflammatory drugs, commonly used for analgesia in headache

Papilledema: swelling of the first portion of the optic nerve

PET: positron emission tomography, a method of imaging to determine the metabolic activity of various regions of the brain

SAH: subarachnoid hemorrhage

SUNCT: short-lasting unilateral neuralgiform pain with conjunctival injection and tearing

Scotoma (plural scotomata): a small area in the visual field where vision is absent

Serotonin: *see* 5-HT

TTH: tension-type headache

Vasodilator: a nerve or agent bringing about vasodilation of blood vessels

Introduction

Most people experience at least occasional headaches during their lifetime. Although many sufferers can function normally using self-medication, the management of patients with disabling headaches forms a substantial proportion of the workload for family physicians and practicing neurologists. In a small number of patients, headache is a symptom of a potentially serious illness, and the first task of any physician is to identify and treat these conditions. Most patients seek reassurance that their headache is benign and that self-medication is appropriate. Many of these people experience disabling headaches, either continuously or as attacks, that justify careful assessment and specific intermittent or regular therapies available only on prescription.

This book describes the processes used to assess patients with headache, summarizes current thinking on pathogenesis and outlines the management of the common forms of disabling primary headache. Most assessment and treatment can, and indeed should, be undertaken within the primary care setting; only patients who are severely affected or refractory to treatment require specialist referral for investigation. The reassurance of people who are healthy but worried, and the proper investigation and treatment of those who are unwell are vital components of the role of any physician, and are particularly rewarding in patients with headache.

A positive diagnosis must be made at or soon after the first consultation with a patient seeking advice about headache. Most patients, especially with primary headache disorders, have few, if any, physical abnormalities to provide clinical clues, so the assessment must be derived largely from clinical history. A physical examination seldom provides much information, and special investigations are useful only in excluding specific structural secondary causes for headaches. It is, therefore, essential for both the family physician and the specialist to document the patient's complaint carefully, as this will usually be the sole basis on which the working clinical diagnosis can be made.

The clinician should record:

- the duration of headache
- the pattern of attacks, with their duration, severity and frequency
- the presence or absence of accompanying symptoms, such as nausea or vomiting, or visual, limb or speech disturbances.

The relationship of headache to coughing, foods, exercise, and neck and jaw movements may also be significant.

The majority of patients seen in the primary care setting have tension-type or migraine headache (Table 1.1); most recurrent, severe headaches are migrainous and are more likely to need to be referred on for specialist assessment and advice. In general, patients with recurrent non-progressive headache without significant disability or physical abnormalities should be reassured by the family physician and offered migraine-specific medication or appropriately potent analgesics, since the majority will have tried simple, non-prescription analgesics prior to seeking medical attention. In contrast, patients with any of the characteristics listed in Table 1.2 should be considered for referral for specialist care.

Secondary causes of headache (e.g. cervical spondylosis, post-traumatic headache and sinusitis) and headache associated with febrile illnesses, such as influenza, are responsible for no more than 5% of cases referred to a neurological outpatient clinic. Intracranial space-

TABLE 1.1

Differential diagnosis of headache

Primary

- Migraine*
 - episodic
 - chronic
- Tension-type headache*
 - episodic
 - chronic
- Cluster headache*
 - episodic
 - chronic
- Benign exertional headache

Secondary

- Cerebral tumors
- Hydrocephalus and CSF obstruction
- Low-pressure headache
- Spontaneous intracranial hypotension
- Arteriovenous malformations
- Cervicogenic headache
- Sinusitis
- Temporal arteritis
- Meningitis/encephalitis
- Subarachnoid hemorrhage
- Hypertension
- Cerebrovascular disease
 - transient ischemic attacks
 - stroke
 - carotid endarterectomy
 - cerebral venous sinus thrombosis
 - arterial dissection
- Cranial neuralgias
 - trigeminal
 - post-herpetic
- Facial and dental pain
- Analgesia-induced headaches
- Post-traumatic headaches
- Other benign headaches*
 - drug-induced headache
 - food- and alcohol-induced headache

* Discussed in other chapters

occupying lesions, such as gliomas, meningiomas, cerebral abscesses and hematomas, are unusual in an outpatient setting. Although the majority of patients seen in a hospital emergency department complaining of ‘the worst headache of my life’ are still more likely to have a primary headache disorder, a higher proportion than in an

TABLE 1.2

Characteristics of patients requiring further investigation and referral

- Suspected recent subarachnoid hemorrhage or meningitis
- Abnormal neurological physical signs (e.g. papilledema, hemiparesis, permanent visual loss and ataxia)
- Decrease in visual acuity or temporary loss of vision
- Persistent or increasing vomiting
- Headache of recent onset or increasing frequency or severity
- Seizures
- Endocrine disturbances (e.g. acromegaly, diabetes insipidus, amenorrhea, galactorrhea, impaired male sexual function or beard growth and poor growth in children)
- Relevant past or family history, such as previous malignancies or neurofibromatosis

outpatient clinic have a secondary headache, particularly sinusitis, subarachnoid hemorrhage or meningitis (Figure 1.1). New-onset headache or a change from a previous pattern should, therefore, always prompt neuroimaging and/or CSF studies before it is deemed primary.

Cerebral tumors

Headache is a common symptom of space-occupying intracranial lesions, such as:

- primary brain tumors
- cerebral metastases
- other masses, including subdural hematomas and cerebral abscesses.

The presence of headache becomes more likely as the tumor expands, and is almost universal in the terminal stages. Headache is seldom the only reason why a patient with a cerebral tumor seeks advice. Most patients present with either seizures or a focal cerebral dysfunction reflecting tumor invasion, rather than distortion of intracranial structures (Figure 1.2). Investigations should be directed at the cause of these symptoms or signs, as the reason for the headache then often becomes evident.

The underlying mechanisms of the migrainous headache, aura and associated symptoms are becoming better understood, though no unifying hypothesis has yet emerged.

The migraine aura

The term 'aura' has been used for nearly two thousand years to denote the sensory hallucinations immediately preceding certain epileptic seizures. For over a century, the term has been used to signify analogous symptoms which inaugurate certain migraine attacks. The transient neurological symptoms of the migraine aura are among the most striking features of migraine and frequently motivate patients to seek consultation with a physician. The most common aura is the visual hallucination which may take a variety of forms such as a dance of brilliant stars, sparks or flashes of light, blind spots, and complex geometric patterns. A variety of other symptoms may occur during the aura, including a tingling sensation often in the face and upper extremity, speech impairment or weakness of one side of the body (hemiparesis). These symptoms usually precede the headache phase by 20–60 minutes, and reflect focal dysfunction of brain cortex. According to the traditional vascular theory, the symptoms of the migraine aura are caused by vasoconstriction with resulting cerebral ischemia, while the aftercoming headache is a result of reflex vasodilation of large, cranial vessels as a response to the presumed ischemia of the constrictor phase.

However, many of the clinical features of migraine cannot be explained by the mechanism of ischemia of cerebral tissue espoused by the vascular theory.

- Aura is experienced by only approximately 15% of patients. Moreover, even in these patients, aura only occurs during some attacks.
- The majority of migraine patients report a constellation of premonitory features which precede the actual headache by hours or days. These include a variety of symptoms such as fluid retention,

thirst, food cravings, elation, depression and drowsiness. It would be impossible to account for these vegetative and affective symptoms on the basis of cerebral ischemia.

- Medications such as non-steroidal anti-inflammatory agents, neuroleptic agents and the anti-epileptic drug divalproex sodium, which have no vasoconstrictor activity, are extraordinarily effective in relieving the headache and associated symptoms during a migraine attack.
- The results from a variety of functional neuroimaging studies have shown only a small, albeit significant, decrease in cerebral blood flow (CBF) at the time of the migraine aura, in an area corresponding to the symptoms (Figure 4.1), which migrates across the cerebral cortex. The headache phase of migraine begins while CBF is still reduced, and the earliest phase of the aura is associated with an increase rather than a decrease in CBF.

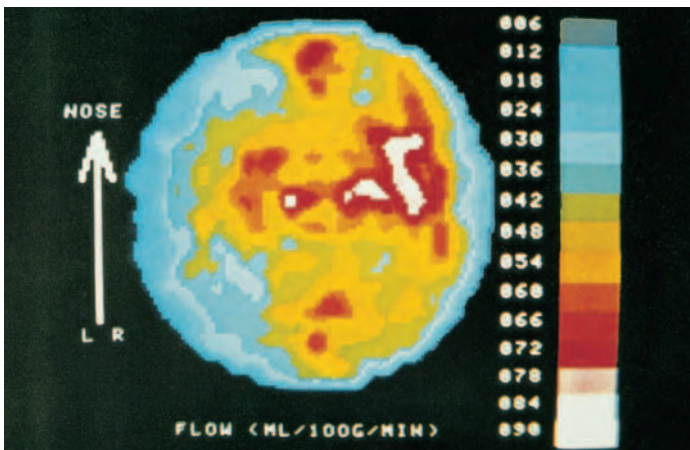


Figure 4.1 Cerebral blood flow tomogram of a patient with classical migraine. A 24-year-old woman with familial hemiplegic migraine arrived at the clinic with right scintillations, hemiparesis and aphasia. At the time of the study she had right arm paresthesias, left-sided headache, nausea and photophobia. The tomogram 5 cm above the orbitomeatal line shows reduced blood flow corresponding to the tomographic representation of left lateral temporal, parietal and frontal cortex. Reproduced with permission from Lauritzen M and Olesen J. *Brain* 1984;107:447.