



The Tract
Clinical Guides · No. 1



Headache Decoded

A working reference to ICHD-3

Classification · Red flags · Migraine · TACs · Neuralgias · Secondary headache

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About this guide

ICHD-3 made usable at the bedside: red flags (SNNOOP10), the duration ladder, migraine, tension-type headache, the trigeminal autonomic cephalalgias, cranial neuralgias and the secondary headaches that must not be missed — updated to the 2026 AAN/AHS prevention guideline.

What changed. Version 2.1 corrects the tension-type headache criteria (bilateral location is one of four optional features, not a requirement), aligns migraine prevention with the 2026 AAN/AHS guideline, adds emergency-department parenteral treatment (AHS 2025), and resets the whole reference in The Tract house style.

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A teaching reference, not a protocol. Doses and thresholds are given to aid recognition; check them against current local guidance and prescribing information before acting on them.

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Part I

The framework

How ICHD-3 is built · Red flags · The duration ladder · The bedside route

Headache is the commonest neurological presentation in every setting that has patients, and almost all of it is diagnosed on the history. This guide follows the order in which a good history is taken: exclude the dangerous, then time the attack, then look for autonomic features. Four figures carry the structure; the tables carry the criteria, quoted from ICHD-3 and checked against it line by line.

1.1 How ICHD-3 is built

The third edition of the International Classification of Headache Disorders contains more than 300 diagnoses, which sounds unusable until you see that it has only three parts. Everything else is subdivision.

Part 1 • Primary The headache is the disease	Part 2 • Secondary The headache is a symptom	Part 3 • Neuropathies Pain in a nerve territory
1 Migraine 2 Tension-type headache 3 Trigeminal autonomic cephalalgias 4 Other primary headaches No cause to find. The diagnosis is made on the pattern alone.	5–7 Trauma, vascular, non-vascular 8 Substance or withdrawal 9–10 Infection, homeostasis 11 Cranial structures 12 Psychiatric disorder Treat the cause. Attribution needs a close temporal relationship.	13 Painful lesions of the cranial nerves and other facial pain 14 Other headache disorders Electric, brief, in a named nerve territory, often with trigger zones.

Figure 1. Learn the three parts and the numbering follows: codes 1–4 are primary, 5–12 secondary, 13–14 the painful cranial neuropathies and other facial pain. A patient can carry a primary and a secondary code at once.

Two rules govern the whole system. First, a secondary headache is diagnosed by its **temporal relationship** to another disorder: it appears in close relation to it and improves or resolves as that disorder is treated.

Second, when a patient with a pre-existing primary headache develops a new headache in close relation to another disorder, **both codes are given**. A patient can legitimately carry chronic migraine and medication-overuse headache at the same time.

1.2 Red flags — SNNOOP10

SNOOP is the version most of us learned. Do and colleagues extended it to SNNOOP10, which adds the features that most often precede a missed secondary headache. Apply it to every headache, every time; a single flag calls for investigation, not reassurance.

Flag	Feature	What it points to
S	Systemic symptoms, including fever	Meningitis, encephalitis, systemic infection, giant cell arteritis
N	Neoplasm in the history	Metastasis, haemorrhage into a metastasis
N	Neurological deficit or reduced consciousness	Mass lesion, stroke, encephalitis, raised intracranial pressure

Flag	Feature	What it points to
O	Onset sudden or thunderclap	Subarachnoid haemorrhage, RCVS, dissection, pituitary apoplexy, venous thrombosis
O	Older age at onset (over 50)	Giant cell arteritis, tumour, subdural haematoma
P	Pattern change, or a recent-onset progressive headache	Mass lesion, subdural collection, medication overuse
P	Positional	Intracranial hypotension if worse upright; raised pressure if worse lying down
P	Precipitated by sneezing, coughing or exercise	Posterior fossa lesion, Chiari malformation, raised pressure
P	Papilloedema	Raised intracranial pressure from any cause, including venous thrombosis
P	Pregnancy or puerperium	Venous thrombosis, pre-eclampsia, pituitary apoplexy, RCVS
P	Painful eye with autonomic features	Posterior fossa, pituitary or cavernous sinus lesion; angle-closure glaucoma
P	Post-traumatic onset	Subdural or extradural haematoma, dissection
P	Pathology of the immune system, including HIV	Opportunistic infection, lymphoma, abscess
P	Painkiller overuse, or a new drug at onset	Medication-overuse or drug-induced headache

Table 1.2 SNNOP10 (Do et al., Neurology 2019). The second N and the ten Ps are the point of the extension.

1.3 The duration ladder

If you ask only one question, ask how long a single *untreated* attack lasts. Duration separates the primary headaches more cleanly than site, quality or severity, and it does so across five orders of magnitude, which is why the axis below is logarithmic.

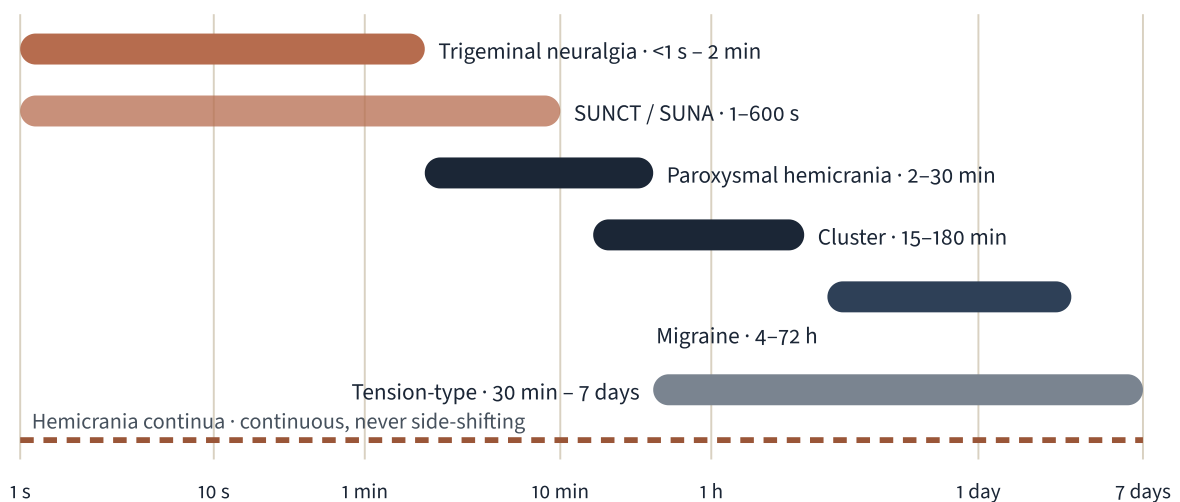


Figure 2. Seconds belong to the neuralgias and SUNCT/SUNA; minutes to the other trigeminal autonomic cephalalgias; hours to migraine and tension-type headache. Ask how long one attack lasts before you ask anything else.

1.4 The route through any headache

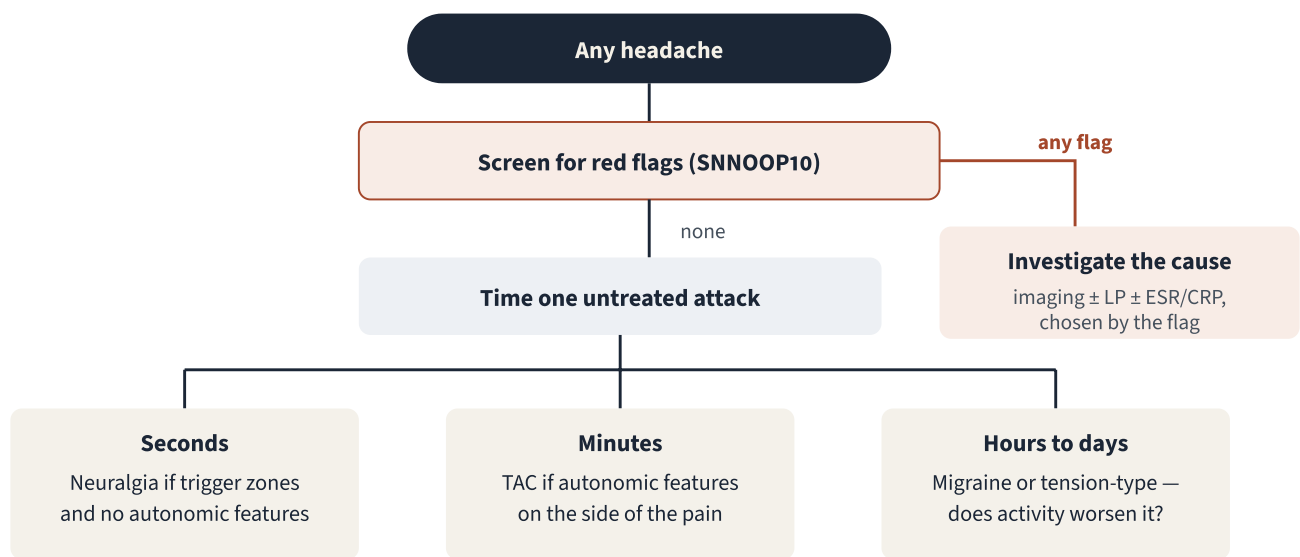


Figure 3. Exclude, then time the attack, then look for autonomic features and trigger zones. In that order the classification does most of the work.

1.5 The history that does the work

Examination is normal in every primary headache disorder, so the diagnosis rests on what the patient says. Eight questions settle most of it.

Ask	What the answer settles
How long does one attack last if you take nothing?	The place on the duration ladder. The single most useful question in the consultation.
How quickly did it reach its worst?	Under a minute is thunderclap and changes the whole pathway.
Is it always on the same side?	Strict side-locking points to a TAC, a neuralgia or a structural lesion. Migraine usually alternates between attacks.
Does moving about make it worse?	Worse with routine activity favours migraine; tension-type headache is not aggravated.
What do you do during an attack?	Lying still in a dark room: migraine. Pacing and rocking: cluster. Freezing and guarding the face: trigeminal neuralgia.
On the painful side, does the eye water or redden, the lid droop, the nostril block?	Cranial autonomic features move the diagnosis into the TAC group.
Does anything set it off — chewing, shaving, cold air, light touch?	Cutaneous trigger zones with an immediate response mean neuralgia.
On how many days a month do you take something for the pain?	Ten or more days of triptans, opioids or combinations, or fifteen of simple analgesics, defines medication overuse.

1.6 Master comparison

The four presentations that account for most diagnostic error. Read down a column to revise one entity, across a row to see what separates them.

	Migraine	Tension-type	Cluster	Trigeminal neuralgia
ICHD-3 code	1	2	3.1	13.1.1

	Migraine	Tension-type	Cluster	Trigeminal neuralgia
One attack	4–72 h untreated	30 min – 7 days	15–180 min	Fraction of a second to 2 min
Quality	Pulsating	Pressing or tightening	Boring, excruciating	Electric shock, stabbing
Side	Unilateral in about 60%; bilateral allowed	Usually bilateral — one of four optional features	Strictly unilateral, side-locked	V2 or V3; V1 alone is uncommon
Severity	Moderate to severe	Mild to moderate	Severe to very severe	Very severe
Nausea	Yes, or photophobia and phonophobia together	None in the episodic form	Uncommon	No
Photo- / phonophobia	Both, typically	At most one	May occur, often one-sided	No
Routine activity	Worsens it	No effect	Cannot keep still	Talking or chewing triggers it
Autonomic features	Absent or mild	Absent	Prominent, ipsilateral — required	Absent (or minimal in V1)
Behaviour	Lies still in the dark	Carries on	Paces, rocks, agitated	Freezes, guards the face
Sex ratio	F : M about 3 : 1	F slightly more than M	M : F about 3 : 1	F : M about 3 : 2
Acute treatment	NSAID, triptan, gepant, lasmiditan	Simple analgesia, sparingly	High-flow oxygen + sumatriptan 6 mg SC	None — attacks are too brief
Prevention	See §2.5	Amitriptyline	Verapamil with ECG monitoring	Carbamazepine; decompression if refractory

Table 1.6 Trigeminal neuralgia is included because it is the entity most often mistaken for a primary headache; its drug treatment is preventive, not abortive.

Part II

Migraine

Criteria · Aura and spreading depolarisation · Acute and preventive treatment

2.1 Migraine without aura (1.1)

The commonest subtype, and the one most often mislabelled as sinus or tension headache. Every part of the criteria is doing work.

Criterion	Detail
A At least five attacks	Fulfilling B–D. Fewer than five, and the diagnosis is probable migraine at most.
B Lasting 4–72 hours	Untreated or unsuccessfully treated. In children and adolescents, 2–72 hours.
C At least two of four characteristics	Unilateral · pulsating · moderate or severe · aggravated by, or causing avoidance of, routine physical activity.
D At least one of	Nausea and/or vomiting; or photophobia <i>and</i> phonophobia.
E Not better accounted for by another ICHD-3 diagnosis	The clause that keeps you honest.

● What the criteria quietly permit

- **Bilateral pain does not exclude migraine.** Only two of the four C features are needed: a bilateral, throbbing, severe headache worsened by movement qualifies comfortably.
- **Most "sinus headache" is migraine.** Facial pain, nasal congestion and tearing come from parasympathetic activation in migraine as well as in the TACs.
- **A "tension headache" that sends the patient to bed in the dark is migraine.** Disability is the practical discriminator when the criteria feel borderline.

2.2 Migraine with aura (1.2)

Aura occurs in roughly a quarter to a third of people with migraine, and the same person often has attacks both with and without it. The criteria are commonly misquoted, so they are set out in full.

Criterion	Detail
A At least two attacks	Two, not five. Aura is distinctive enough that fewer attacks are needed.
B One or more fully reversible aura symptoms	Visual, sensory, speech or language, motor, brainstem or retinal. Anything that does not fully reverse needs imaging.
C At least three of six characteristics	(1) one symptom spreads gradually over ≥ 5 min; (2) two or more symptoms in succession; (3) each symptom lasts 5–60 min; (4) at least one is unilateral; (5) at least one is positive; (6) headache accompanies the aura or follows within 60 min.
D Not better accounted for by another diagnosis	Transient ischaemic attack is the differential that matters, and it is the one that does not spread gradually.

What aura is

Aura is **cortical spreading depolarisation**: a slow wave of intense neuronal and glial depolarisation followed by sustained suppression, moving across the cortex at about 3–5 mm per minute. It usually starts occipitally, which is why visual symptoms dominate, and moves forward. Leão described it in rabbit cortex in 1944; Hadjikhani and colleagues showed the matching signal in human visual cortex during aura in 2001. The wave activates the trigeminovascular system, which produces the headache.

The speed is the diagnostic point. A deficit that marches over five to twenty minutes is aura; one that is maximal at onset is vascular until proved otherwise.

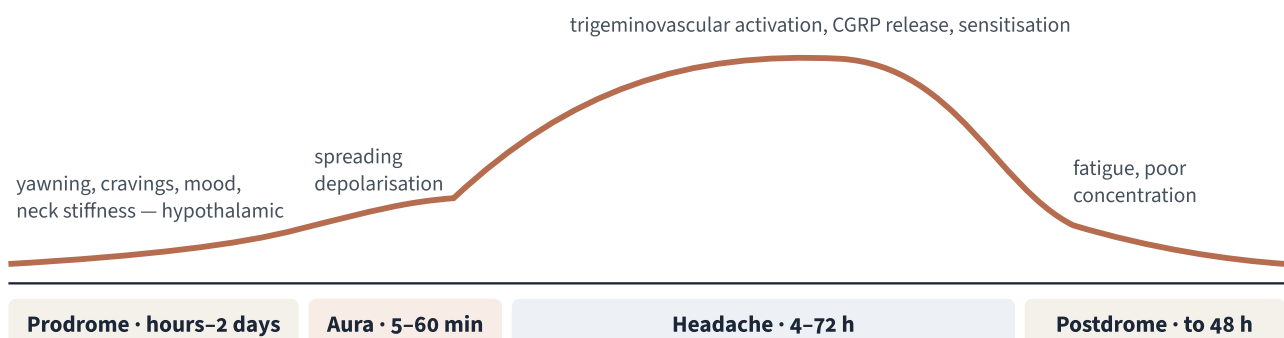


Figure 4. Prodrome and postdrome belong to the attack. Aura can occur without any headache; patients who recognise their prodrome can often treat earlier.

◆ Aura, contraception and stroke

- Migraine with aura roughly doubles the risk of ischaemic stroke; the risk multiplies with combined hormonal contraception and again with smoking.
- Combined hormonal contraception is generally avoided in migraine with aura at any age (UKMEC and US MEC category 4). Progestogen-only methods, the implant, the hormonal IUS and non-hormonal methods are acceptable.
- Migraine without aura carries no such restriction, so ask specifically about visual disturbance before prescribing.
- A first aura after 50, an aura always on the same side, or one lasting beyond an hour, should be imaged rather than reassured.

2.3 Chronic migraine (1.3)

Headache on 15 or more days a month for more than three months, with features of migraine on at least eight of those days. Two points are often got wrong. The threshold is *more than* three months. And medication overuse no longer has to be excluded first: ICHD-3 codes both together, which matters because withholding the chronic migraine label until withdrawal is complete delays effective prevention.

2.4 Acute treatment

Class	Examples	Notes
NSAIDs and aspirin	Ibuprofen 400–600 mg, naproxen, diclofenac, aspirin 900–1000 mg	Effective and cheap for mild to moderate attacks. Combine with a triptan for incomplete responders.
Triptans	Sumatriptan, rizatriptan, zolmitriptan, eletriptan, naratriptan, frovatriptan, almotriptan	5-HT _{1B/1D} agonists, first line for moderate to severe attacks. Take early in the headache. Failure of one does not predict failure of another — try more than one. Avoid in coronary, cerebrovascular and peripheral vascular disease and uncontrolled hypertension.
Gepants	Rimegepant, ubrogepant, zavegepant (nasal)	CGRP receptor antagonists without vasoconstriction, so usable where triptans are contraindicated. No signal of medication-overuse headache so far.
Ditan	Lasmiditan	5-HT _{1F} agonist, also non-vasoconstrictive. Dizziness and somnolence; no driving for eight hours after a dose.
Antiemetics	Metoclopramide, prochlorperazine, domperidone	Treat nausea and the gastric stasis that blocks absorption of everything else. Effective as monotherapy in the emergency department.
Avoid	Opioids, butalbital combinations	Poor efficacy, high risk of medication overuse, and they promote chronification.

Table 2.4 Keep acute drugs to fewer than 10 days a month (15 for simple analgesics).

In the emergency department

The American Headache Society's guideline on parenteral treatment (2025) gives the order of play for an adult with migraine in the emergency department: **offer** intravenous prochlorperazine or a greater occipital nerve block; metoclopramide, ketorolac, dexamethasone, subcutaneous sumatriptan and supraorbital nerve block are also effective; dexamethasone may be offered to reduce early recurrence. **Avoid** intravenous hydromorphone and morphine, and do not rely on diphenhydramine, octreotide or intravenous paracetamol. Give an antihistamine or anticholinergic with a dopamine antagonist only if akathisia is a concern.

2.5 Preventive treatment

The 2026 AAN/AHS guideline (Potrebic et al.) replaced the 2012 recommendations. **Offer prevention** to adults with four or more migraine days a month, four or more moderate-to-severe headache days a month, or migraine that substantially disrupts daily life. Choose by efficacy, tolerability, comorbidity, cost and preference: the CGRP monoclonal antibodies and atogepant sit alongside propranolol, topiramate and valproate among the options with the strongest evidence, and the guideline does not require older drugs to fail first, although funders often still do. Give each drug **8–12 weeks** at a tolerated dose before judging it (24 weeks for onabotulinumtoxinA), and offer prevention even when medication overuse is present.

Agent	Typical adult dose	Choose it when — and watch for
Propranolol	80–160 mg daily (modified release)	Coexisting tremor, anxiety or hypertension. Avoid in asthma and bradycardia.
Topiramate	50–100 mg daily	Weight gain is unwanted. Teratogenic; reduces the efficacy of hormonal contraception; cognitive slowing, paraesthesiae, renal stones.
Amitriptyline	10–75 mg at night	Coexisting tension-type headache, poor sleep or pain. Anticholinergic effects limit the dose.
Candesartan	16 mg daily	Well tolerated and often forgotten; reasonable where beta-blockade is unsuitable. Avoid in pregnancy.
Sodium valproate	500–1000 mg daily	Effective, but avoid in anyone who can become pregnant unless no alternative exists and a pregnancy prevention programme is in place.
CGRP antibodies	Erenumab 70 or 140 mg monthly · fremanezumab 225 mg monthly or 675 mg quarterly · galcanezumab 240 mg then 120 mg monthly · eptinezumab 100 or 300 mg IV quarterly	Among the best tolerated options. Erenumab targets the receptor, the others the ligand. Constipation and hypertension with erenumab; injection-site reactions across the class.
Gepants	Atogepant 60 mg daily (10–60 mg); rimegepant 75 mg every other day	Oral CGRP options for prevention; rimegepant doubles as acute treatment.
OnabotulinumtoxinA	155 U at 31 sites every 12 weeks (up to 195 U at 39)	Chronic migraine only. The PREEMPT paradigm is fixed; "follow the pain" sites are added to it, not substituted. Judge at 24 weeks.

Table 2.5 Reassess after six to twelve months of good control and consider a gradual taper.

Pregnancy

Avoid topiramate and valproate in anyone who can become pregnant wherever possible. If prevention is needed in pregnancy, maximise non-drug measures first; propranolol, metoprolol, nifedipine or onabotulinumtoxinA may be considered after a careful discussion of risk.

Part III

Tension-type headache

The commonest headache on earth, and a diagnosis of pattern

3.1 Criteria and subtypes

Tension-type headache is defined as much by what it lacks as by what it has. It is the most prevalent headache disorder in the world and the least studied, largely because it is the least disabling.

Criterion	Detail
A Episodes fulfilling B–D	At least ten episodes for the episodic forms.
B Lasting 30 minutes to 7 days	Hours to continuous in the chronic form.
C At least two of four characteristics	Bilateral · pressing or tightening, non-pulsating · mild or moderate · not aggravated by routine physical activity. Unilateral tension-type headache exists ; bilateral location is one option, not a requirement.
D Both of	No nausea or vomiting (mild nausea is allowed in the chronic form); and no more than one of photophobia or phonophobia.
E Not better accounted for by another diagnosis	

Subtype	Frequency	Note
Infrequent episodic (2.1)	< 1 day a month on average (< 12 a year)	Near-universal; rarely reaches a clinic.
Frequent episodic (2.2)	1–14 days a month for more than 3 months	The point at which prevention is worth discussing.
Chronic (2.3)	≥ 15 days a month for more than 3 months	Screen for medication overuse, depression and sleep disorder.

Table 3.1 Pericranial tenderness on manual palpation is recorded as a qualifier and is the only useful physical sign.

● The three discriminators

- **Activity.** Routine physical activity worsens migraine and does not worsen tension-type headache.
- **The photophobia rule.** Tension-type headache allows at most one of photophobia or phonophobia. Both present: think migraine.
- **Triptans.** They do not work in tension-type headache. A clear response should make you revisit the label.

3.2 Treatment

- **Episodic attacks.** Ibuprofen 400 mg, aspirin 500–1000 mg or paracetamol 1000 mg; caffeine combinations add efficacy and add overuse risk. Keep total use below 15 days a month.
- **Chronic tension-type headache.** Amitriptyline is first line, starting at 10 mg at night and titrating slowly; mirtazapine or venlafaxine where it is not tolerated.
- **Non-drug measures work here more than anywhere else:** physiotherapy, exercise, relaxation, biofeedback and cognitive behavioural approaches.
- **No opioids.** There is no role for them in any primary headache disorder.

Part IV

Trigeminal autonomic cephalalgias

Cluster · Paroxysmal hemicrania · SUNCT and SUNA · Hemicrania continua

4.1 The shared mechanism

All four share one circuit. Activation of first-division trigeminal afferents drives the **trigeminal–autonomic reflex**: a brainstem link to the superior salivatory nucleus and, through the sphenopalatine ganglion, to cranial parasympathetic outflow. That produces the ipsilateral tearing, conjunctival injection, nasal congestion and rhinorrhoea; sympathetic dysfunction adds ptosis and miosis. Functional imaging during attacks shows activation in the posterior hypothalamic region, which fits the striking circadian and circannual rhythm of cluster headache.

What separates the four is arithmetic: how long each attack lasts, how many occur in a day, and whether indometacin abolishes them.

	One attack	Frequency	Treatment that defines or leads
SUNCT / SUNA (3.3)	1–600 seconds	At least once a day; often dozens to hundreds	Lamotrigine; IV lidocaine for attack clusters
Paroxysmal hemicrania (3.2)	2–30 minutes	More than 5 a day for over half the time	Indometacin — absolute response is a criterion
Cluster headache (3.1)	15–180 minutes	One every other day to 8 a day	Oxygen plus subcutaneous or nasal triptan
Hemicrania continua (3.4)	Continuous for > 3 months	Exacerbations on a constant background	Indometacin — absolute response is a criterion

Table 4.1 The TAC ladder: as attacks get shorter, they get more frequent.

4.2 Cluster headache (3.1)

Strictly unilateral, orbital, supraorbital or temporal, very severe, with ipsilateral autonomic features and restlessness. Episodic bouts usually last weeks to a few months and often recur at the same season; attacks often come at the same hour, frequently from sleep. The male predominance once quoted as 5–6 : 1 is now nearer 3 : 1, probably because women were misdiagnosed with migraine. Chronic cluster (about 10–15%) has remissions shorter than three months.

Step	What to do
Acute	100% oxygen at 12–15 L/min through a non-rebreather mask for 15–20 minutes, sitting up — not nasal cannulae. Plus sumatriptan 6 mg subcutaneously, or zolmitriptan 5 mg or sumatriptan 20 mg intranasally. Oral triptans act too slowly.
Transitional	A short oral corticosteroid course, or a greater occipital nerve block on the affected side, to suppress attacks while prevention is titrated.
Preventive	Verapamil first line: 80 mg three times daily, increased stepwise, sometimes beyond 480 mg daily under ECG monitoring. Lithium and topiramate second line. Galcanezumab is licensed in the United States for episodic cluster headache.
Refractory	Occipital nerve stimulation; sphenopalatine ganglion stimulation; non-invasive vagus nerve stimulation; hypothalamic deep brain stimulation in highly selected patients.

◆ Verapamil needs an ECG, not just a prescription

- High-dose verapamil causes dose-dependent heart block, often silent. Baseline ECG, then about ten days after each increment, and periodically once stable.
- Cluster doses far exceed cardiology practice. Escalating without monitoring is unsafe.
- Home oxygen in a smoker needs a documented fire-risk assessment.

4.3 Paroxysmal hemicrania (3.2)

Shorter and more frequent than cluster: attacks of 2–30 minutes, more than five a day for more than half the time, with the same unilateral autonomic features. The diagnosis is defined by treatment: an **absolute response to indometacin** is a criterion, not a bonus.

Start indometacin 25 mg three times daily, rising to 50 and then 75 mg three times daily if the response is partial (maximum around 225 mg a day). Always give gastric protection; long-term use raises blood pressure and harms the kidney.

4.4 SUNCT and SUNA (3.3)

Short-lasting Unilateral Neuralgiform headache attacks with Conjunctival injection and Tearing. When only one of those two, or other cranial autonomic features, accompany the pain, the term is SUNA (with cranial Autonomic symptoms). ICHD-3 sets attack duration at 1–600 seconds, wider than the older 5–240 seconds; attacks can number in the hundreds a day.

The trap is trigeminal neuralgia, also brief, electric and triggered. The discriminators: **autonomic features**, which SUNCT has and trigeminal neuralgia lacks; and the **refractory period**, which trigeminal neuralgia has and SUNCT lacks.

◆ Image every SUNCT and SUNA

MRI brain with attention to the posterior fossa and pituitary before accepting the diagnosis: a structural cause – pituitary adenoma, posterior fossa malformation, demyelination, neurovascular compression – is found more often than in the other TACs. Lamotrigine is first line and must be titrated slowly because of rash.

4.5 Hemicrania continua (3.4)

A continuous, strictly unilateral headache for more than three months that never switches side and never fully remits, with exacerbations carrying ipsilateral autonomic features or restlessness. It is under-diagnosed because the background pain is moderate and patients present with the exacerbations. A complete response to indometacin is required; celecoxib, topiramate, melatonin or occipital nerve block are fallbacks when it cannot be tolerated.

● The indometacin rule

Paroxysmal hemicrania and hemicrania continua are both defined by an absolute response to an adequate dose. Partial or no response means the diagnosis is wrong, and the next step is imaging. The corollary: a trial counts as negative only at full dose, for long enough, taken as prescribed.

Part V

Cranial neuralgias

Electric · territorial · triggered

5.1 What makes a neuralgia

Feature	Why it matters
Electric, stabbing, lancinating	Not dull, throbbing or pressing. Patients use the words shock, jolt or stab.
Very brief — a fraction of a second to two minutes	Separates neuralgia from every primary headache and every TAC except SUNCT/SUNA.
Trigger zones	Light touch, chewing, talking, shaving, cold air. The response is immediate.
Refractory period	A quiet interval during which the trigger fails. Present in trigeminal neuralgia, absent in SUNCT.
Confined to a named territory	Trigeminal divisions, or the occipital territory. Pain that ignores these borders is something else.
No cranial autonomic features	Tearing, injection, ptosis or congestion move the diagnosis to the TACs.

5.2 Trigeminal neuralgia (13.1)

Recurrent paroxysms of electric pain in one or more trigeminal divisions, usually V2 or V3, lasting a fraction of a second to two minutes, triggered by innocuous stimuli. ICHD-3 divides it into **classical** (neurovascular compression with morphological change of the root on MRI), **secondary** (multiple sclerosis, a cerebellopontine angle tumour) and **idiopathic**.

	Detail
Imaging	MRI with high-resolution steady-state sequences (CISS, FIESTA) through the cerebellopontine angle; the superior cerebellar artery is the usual culprit. Not optional — a secondary cause is found in a meaningful minority.
Suspect secondary	Onset before 40, bilateral pain, sensory loss, V1 alone, or no refractory period. Bilateral trigeminal neuralgia is multiple sclerosis until proved otherwise.
First line	Carbamazepine (from 100 mg twice daily) or oxcarbazepine. Monitor sodium, blood count and liver tests. Test for HLA-B*15:02 before carbamazepine in people of Han Chinese, Thai, Malay and other South-East Asian ancestry.
Second line	Lamotrigine, baclofen, gabapentin, pregabalin, often as add-ons.
Surgery	Microvascular decompression gives the most durable result when compression is confirmed — about 70% pain-free at ten years — at the cost of a posterior fossa operation. Radiosurgery and percutaneous procedures are less invasive, with shorter benefit and more numbness.
Crisis	Unable to eat or drink is an admission. IV fosphenytoin or lidocaine can break a crisis while oral therapy is restored.

Table 5.2 Trigeminal neuralgia carries a high burden of depression; screen for it.

5.3 The other neuralgias

Neuralgia	Territory and triggers	Management
Occipital (13.4)	Greater, lesser or third occipital nerve. Occiput to vertex, sometimes referred to the eye through trigeminocervical convergence. Tender nerve.	Occipital nerve block — diagnostic and therapeutic. Amitriptyline or gabapentin if recurrent.
Glossopharyngeal (13.2)	Ear, tonsillar fossa, base of tongue, angle of jaw. Triggered by swallowing, coughing, talking or yawning.	Carbamazepine. Attacks can cause bradycardia and syncope. Decompression if refractory.
Nervus intermedius (13.3)	Deep in the ear canal. May follow herpes zoster (Ramsay Hunt).	Carbamazepine or oxcarbazepine; surgery in refractory cases.

Part VI

Secondary headache

The ones that must not be missed

6.1 Thunderclap headache

A headache that reaches maximum intensity in under a minute is a presentation, not a diagnosis, with a differential to be worked through in order.

Cause	What points to it	What to do
Subarachnoid haemorrhage	Meningism, brief loss of consciousness, vomiting, focal signs. The commonest serious cause, still a minority of thunderclaps.	Non-contrast CT: close to 100% sensitive within 6 hours on a modern scanner read by an expert. Later, or if doubt remains, lumbar puncture ≥ 12 h after onset for xanthochromia, or CT angiography.
RCVS	Recurrent thunderclaps over days to weeks, triggered by exertion, sex, bathing, coughing or vasoactive drugs; puerperium.	Angiography shows segmental "string of beads", often normal in the first days — repeat it. Stop the trigger; nimodipine is widely used; avoid steroids, which worsen outcome.
Cervical artery dissection	Neck or face pain, Horner syndrome, pulsatile tinnitus, minor trauma or neck manipulation. A leading cause of stroke under 50.	CT or MR angiography of the neck (MRI with fat-saturated sequences). Antithrombotic treatment.
Venous sinus thrombosis	Progressive or thunderclap headache, papilloedema, seizures, deficits that cross arterial territories. Pregnancy, puerperium, oestrogen, dehydration, thrombophilia, cancer.	CT or MR venography — plain CT is not enough. Anticoagulate, including when there is haemorrhagic venous infarction.
Pituitary apoplexy	Sudden headache with visual field loss, ophthalmoplegia, hypopituitarism; may be the first sign of an adenoma.	Pituitary MRI. Hydrocortisone immediately; endocrine and neurosurgical review.
Others	Intracerebral or intraventricular haemorrhage, posterior fossa infarct, spontaneous intracranial hypotension, colloid cyst, PRES.	Imaging chosen by the story.

Table 6.1 A normal CT excludes subarachnoid haemorrhage (within its window); it excludes nothing else on this list.

6.2 Giant cell arteritis

New headache after 50, often temporal, with scalp tenderness and jaw claudication; jaw claudication and visual symptoms carry the strongest likelihood ratios. Inflammatory markers are usually raised, but normal values do not exclude it.

- **Treat before you confirm.** Prednisolone 40–60 mg daily on suspicion. With visual loss or amaurosis fugax, give intravenous methylprednisolone: the affected eye is usually lost, and the aim is to save the other.
- **Then confirm.** Temporal and axillary artery ultrasound for the halo sign is first line in many centres; biopsy remains informative for at least two weeks after steroids start, so treatment never waits for it.
- **Plan the course.** Treatment runs a year or more. Tocilizumab is licensed as a steroid-sparing agent; give bone and gastric protection from day one.

6.3 Meningitis and encephalitis

- **The classic triad is unreliable.** Fever, neck stiffness and altered mental state occur together in fewer than half of adults with bacterial meningitis, but almost all have at least two of headache, fever, neck stiffness and altered mental state.
- **Do not delay antibiotics for imaging or lumbar puncture.** Ceftriaxone; add amoxicillin or ampicillin for *Listeria* over 50 and in immunosuppression, vancomycin where penicillin-resistant pneumococci are common, and aciclovir if encephalitis is possible.
- **Dexamethasone** 10 mg six-hourly for four days, with or just before the first antibiotic dose. The benefit is clearest in pneumococcal meningitis.
- **Bacterial CSF:** turbid, neutrophils, high protein, CSF : serum glucose ratio below about 0.4, lactate raised – lactate discriminates well in partly treated cases.

6.4 Disorders of CSF pressure

	Idiopathic intracranial hypertension	Spontaneous intracranial hypotension
Typical patient	Woman of child-bearing age with obesity or recent weight gain	Any adult; connective tissue laxity; often after a trivial strain
Headache	Daily, worse lying down and on waking, worse with Valsalva	Worse within minutes to hours of standing, better lying flat; the pattern fades in chronic leaks
Other features	Transient visual obscurations, pulsatile tinnitus, sixth nerve palsy, papilloedema	Neck pain, muffled hearing or tinnitus, nausea, interscapular pain
Imaging	Empty sella, flattened globes, distended optic nerve sheaths, transverse sinus stenosis. MR venography to exclude venous thrombosis.	Pachymeningeal enhancement, brain sag, venous engorgement, subdural collections. Then find the leak in the spine.
Pressure	Opening pressure ≥ 25 cmH ₂ O with normal CSF	< 6 cmH ₂ O supports it, but a normal pressure does not exclude it
Treatment	Weight loss is disease-modifying; acetazolamide; topiramate as an alternative. Sight-threatening disease: venous stenting, shunting or optic nerve sheath fenestration.	Conservative measures, then epidural blood patch — targeted if the leak is found. Repeat patching, fibrin glue or surgical repair follow.

Table 6.4 Repeated therapeutic lumbar puncture is no longer recommended as maintenance treatment in IIH.

6.5 Medication-overuse headache (8.2)

Headache on 15 or more days a month in someone with a pre-existing headache disorder, developing as a consequence of regular overuse of acute medication for more than three months: ten or more days a month for triptans, ergots, opioids or combination analgesics; fifteen or more for simple analgesics. Opioids and butalbital combinations carry the highest risk and the hardest withdrawal.

The traditional approach was supervised withdrawal, during which headache worsens for one to two weeks before improving. Current evidence supports starting effective prevention at the same time as withdrawal – or, with CGRP antibodies, even without enforced withdrawal. Either way, explain why the drug that helps each attack is driving the pattern.

6.6 Other never-miss causes

- **Acute angle-closure glaucoma** – unilateral headache with a red eye, hazy cornea, fixed mid-dilated pupil, haloes and vomiting. Measure the pressure; it is an ophthalmic emergency, and the vomiting often sends it to the wrong team.
- **Carbon monoxide** – headache, nausea and poor concentration in several people in one household, better outdoors, worse in winter. Ask about heaters, generators and charcoal indoors.
- **Hypertensive emergency and PRES** – headache with visual disturbance, seizures or confusion; in pre-eclampsia at pressures that look unremarkable.
- **Sphenoid sinusitis** – the one sinusitis that presents with headache alone, vertex or retro-orbital, without nasal symptoms.
- **Third nerve palsy with a dilated pupil** – a posterior communicating artery aneurysm until angiography says otherwise.

Part VII

Self-assessment

Eight vignettes against ICHD-3

Q1 • Migraine criteria

A 32-year-old woman describes six attacks of unilateral throbbing headache, each lasting six to eight hours, with nausea and photophobia. Climbing stairs makes it much worse. How many attacks does ICHD-3 require for migraine without aura?

- A. One
- B. Three
- C. Five
- D. Ten

C. Five. Criterion A requires at least five attacks fulfilling B–D; with fewer, the label is probable migraine. Aggravation by routine activity is what separates this from tension-type headache.

Q2 • Differentiating the TACs

A 45-year-old man has right periorbital attacks lasting about 90 minutes, twice a day, with ipsilateral tearing and conjunctival injection. He paces during attacks. This has run for eight weeks; he had an identical bout last spring. Best acute treatment?

- A. Oral sumatriptan 50 mg
- B. Indometacin 75 mg three times daily
- C. 100% oxygen by non-rebreather plus sumatriptan 6 mg subcutaneously
- D. Lamotrigine 25 mg

C. Cluster headache: 15–180-minute attacks, seasonal bouts, restlessness and ipsilateral autonomic features. Attacks of 2–30 minutes, more than five a day, would point to paroxysmal hemicrania and an indometacin trial. Oral triptans are too slow.

Q3 • The indometacin rule

A woman has a continuous right-sided headache that never resolves, with daily exacerbations of severe right periorbital pain, tearing and nasal congestion. It has never changed side. Diagnosis and treatment?

- A. Cluster headache — verapamil
- B. SUNCT — lamotrigine
- C. Hemicrania continua — indometacin
- D. Chronic migraine — topiramate

C. Continuous, side-locked, with autonomic exacerbations. A complete response to indometacin is required; if an adequate trial fails, image.

Q4 • Giant cell arteritis

A 68-year-old woman has a new bitemporal headache for three weeks and jaw fatigue when eating. ESR 78 mm/h, CRP 42 mg/L. Vision is normal. Most urgent step?

- A. MRI brain and routine neurology referral
- B. Amitriptyline for chronic tension-type headache
- C. Temporal artery biopsy, then steroids when the result returns
- D. Prednisolone 40–60 mg daily now, with ultrasound or biopsy within days

D. The risk is ischaemic optic neuropathy and blindness. Treat on suspicion; biopsy stays informative for at least two weeks. Visual symptoms would call for intravenous methylprednisolone.

Q5 • Mechanism of aura

A slowly spreading wave of neuronal and glial depolarisation, followed by suppression, starting occipitally and moving forward at 3–5 mm per minute, is:

- A. Trigemino-vascular activation
- B. Cortical spreading depolarisation
- C. Central sensitisation
- D. The trigeminal–autonomic reflex

B. Described by Leão (1944) and shown in human visual cortex during aura by Hadjikhani and colleagues (2001). Central sensitisation explains allodynia; the trigeminal–autonomic reflex produces the TAC features.

Q6 • Trigeminal neuralgia

A 65-year-old man has right cheek electric shocks lasting two to three seconds, triggered by chewing and light touch, with a refractory period. MRI shows an artery indenting the trigeminal root entry zone. First-line drug and best long-term surgical option?

- A. Amitriptyline, then radiosurgery
- B. Gabapentin, then percutaneous rhizotomy
- C. Carbamazepine, then microvascular decompression
- D. Lamotrigine, then occipital nerve block

C. Carbamazepine (or oxcarbazepine) first; microvascular decompression gives the most durable relief when compression with root distortion is shown.

Q7 • SUNCT

A patient has 30-second attacks of severe periorbital pain with conjunctival injection and tearing, about 50 times a day, with no refractory period. Before lamotrigine is started, which investigation is essential?

- A. EEG
- B. CT angiography of the circle of Willis
- C. Lumbar puncture
- D. MRI brain with attention to the posterior fossa and pituitary

D. Secondary SUNCT/SUNA is relatively common – pituitary and posterior fossa lesions especially. ICHD-3 attack duration is 1–600 seconds.

Q8 • Chronic migraine prevention

A 38-year-old woman has headache on 18 days a month, with migraine features on 10. Topiramate and amitriptyline failed at adequate doses. She is not overusing acute drugs and uses an implant for contraception. Most appropriate next step?

- A. OnabotulinumtoxinA or a CGRP monoclonal antibody
- B. Lithium
- C. Verapamil 240 mg daily
- D. Carbamazepine 400 mg daily

A. Chronic migraine. Both are recommended for chronic migraine, and under the 2026 AAN/AHS guideline a CGRP antibody could have been chosen even before the oral drugs. Lithium and verapamil belong to cluster headache; carbamazepine to trigeminal neuralgia.

Appendix

Mnemonics, abbreviations and sources

For revision and for checking

A Mnemonics and one-liners

	POUND — migraine	Note
P	Pulsating	Throbbing rather than pressing
O	One-day duration	4–72 hours untreated
U	Unilateral	Bilateral does not exclude migraine
N	Nausea	Or vomiting
D	Disabling	Interferes with activity

POUND. Four or five of the five features give a positive likelihood ratio of about 24 for migraine (Detsky et al., JAMA 2006).

Migraine vs cluster	The migraine patient lies still in the dark; the cluster patient paces.
Photophobia rule	Tension-type allows at most one of photophobia or phonophobia.
TAC vs neuralgia	TACs have cranial autonomic features; neuralgias are electric, seconds long, triggered, and have none.
Indometacin pair	Paroxysmal hemicrania and hemicrania continua: complete response, or the diagnosis is wrong.
Giant cell arteritis	Over 50, jaw claudication, raised markers: steroids before biopsy; visual loss means intravenous.
Bilateral trigeminal neuralgia	Multiple sclerosis until proved otherwise.
Aura and the pill	Migraine with aura: avoid combined hormonal contraception.
Thunderclap	A normal early CT excludes subarachnoid haemorrhage and nothing else.

B Abbreviations

CGRP calcitonin gene-related peptide

CSD	cortical spreading depolarisation
CSF	cerebrospinal fluid
GCA	giant cell arteritis
ICHD-3	International Classification of Headache Disorders, 3rd edition
IIH	idiopathic intracranial hypertension
LP	lumbar puncture
MOH	medication-overuse headache
PRES	posterior reversible encephalopathy syndrome
RCVS	reversible cerebral vasoconstriction syndrome
SIH	spontaneous intracranial hypotension
SUNA	short-lasting unilateral neuralgiform attacks with cranial autonomic symptoms
SUNCT	... with conjunctival injection and tearing
TAC	trigeminal autonomic cephalalgia
TTH	tension-type headache

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