



The Tract
Clinical Guides · No. 2



Intracranial Haemorrhage & Neuroanatomy

Mechanism first: the four bleeds, CSF and pressure

Epidural · Subdural · Subarachnoid · Intracerebral · CSF dynamics · ICP and herniation

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About this guide

Why an epidural kills faster than a subdural, what subarachnoid blood does to CSF, how to run the first hour of intracerebral haemorrhage, and how Monro–Kellie, compliance and herniation tie the four together. With MMA embolisation, INTERACT3, ANNEXA-I, ENRICH and SWITCH.

What changed. Version 6.1 corrects the STEM citation and the reading of the three middle meningeal artery embolisation trials, separates the epidural and subdural surgical thresholds, adds the global share of intracerebral haemorrhage, and resets the 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

Intracranial haemorrhage

Epidural · Subdural · Subarachnoid · Intracerebral

Four lesions, four planes. Each type of intracranial haemorrhage is named for the compartment it occupies, and almost every difference in shape, speed and behaviour follows from that compartment and from the pressure in the vessel that fills it. Learn the geometry once and the rest stops being a list.

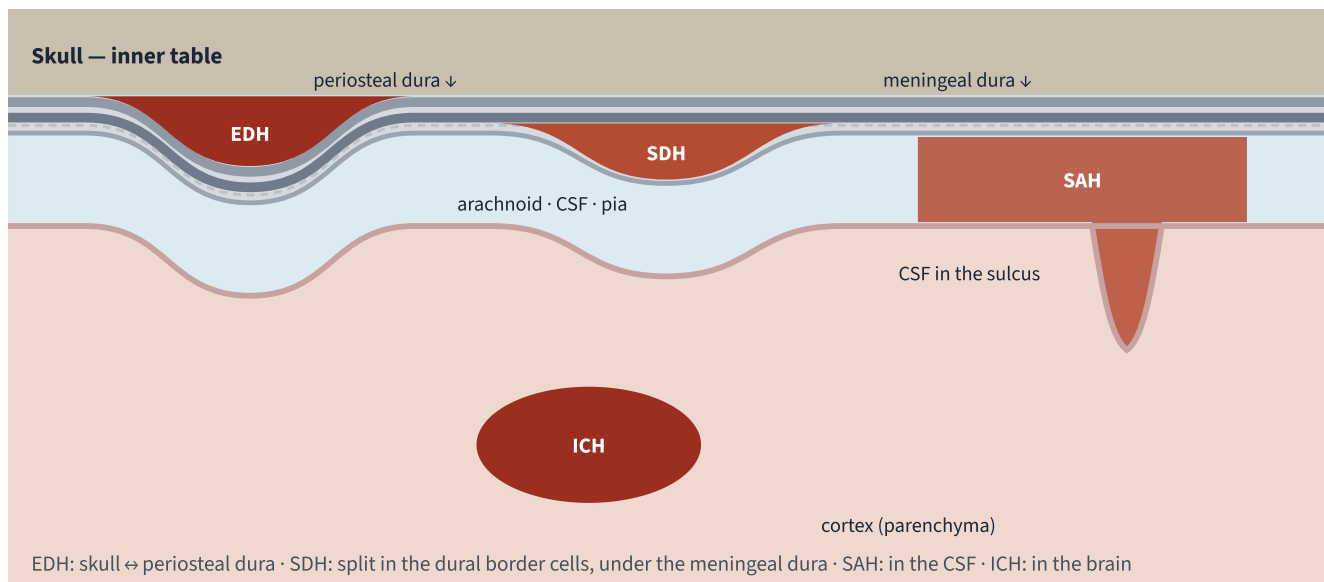


Figure 1. Each haemorrhage is defined by its plane. Two of those planes are potential rather than real, which is why epidural and subdural blood must make their own space, and why each is then stopped by a different anatomical attachment.

1.1 Epidural haematoma

Blood between the inner table and the outer, periosteal layer of the dura. It is nearly always traumatic, and the classic source is the **middle meningeal artery**, a branch of the maxillary artery that runs in a groove on the inner surface of the temporal squama, under the pterion where the bone is thinnest. Because the source is arterial, the collection expands under systemic pressure, and that single fact explains almost everything else.

Why the shape follows from the space

The extradural space is potential. Dura is bonded to the inner table, most tightly at the sutures, where the periosteal layer becomes continuous with the outer periosteum. An arterial jet has to strip dura off bone to make room, and it does so until it reaches a suture and stops. The result is the **biconvex lens**: sharply marginated, tall in the middle where the pressure head is applied.

The mirror-image rule is more useful than either half of it. **Epidural blood does not cross sutures but can cross the midline; subdural blood crosses sutures but is stopped at the falx and tentorium.** Hold that pair and you rarely need the shape.

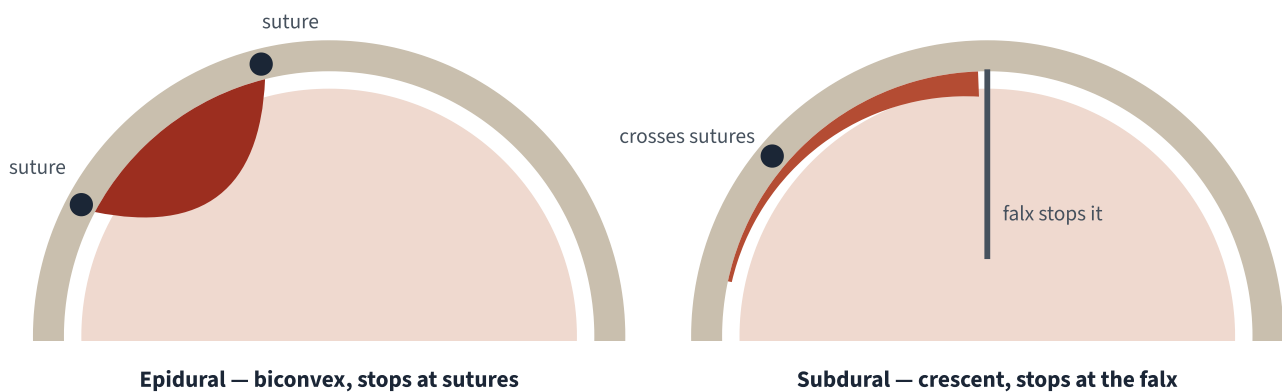


Figure 2. Two shapes, two attachment problems. Source pressure: middle meningeal artery at systemic pressure versus bridging veins at about 5–10 mmHg.

The lucid interval

A minority of patients – series report 20–50% – show the classic sequence: brief unconsciousness from the impact, apparent recovery, then rapid secondary deterioration. The recovery is real. The patient is compensating on the flat part of the pressure–volume curve (Figure 4) while CSF is displaced into the spinal sac; deterioration is abrupt because the curve has a knee, not because anything new has happened to the bleed.

The temporal location puts the clot beside the uncus. Uncal herniation compresses the third nerve (an ipsilateral fixed, dilated pupil) and the cerebral peduncle (contralateral hemiparesis). Sometimes the shift presses the opposite peduncle against the tentorial edge and gives an ipsilateral hemiparesis – **Kernohan's notch**, a false-localising sign that has sent more than one patient to theatre on the wrong side.

◆ The hour you have

- Rising blood pressure, falling heart rate, irregular breathing: Cushing's response. Not impending herniation – herniation in progress.
- A new fixed, dilated pupil or a fall in GCS of two or more points: call the neurosurgeon now, not after the repeat scan.
- Brain Trauma Foundation thresholds: an epidural haematoma over 30 mL is evacuated whatever the GCS; GCS below 9 with anisocoria means evacuation as soon as possible. A haematoma under 30 mL, under 15 mm thick, with under 5 mm shift, GCS above 8 and no focal deficit can be observed with serial CT in a neurosurgical centre.
- Anisocoria and a temporal fracture in a patient who was talking: epidural haematoma until proved otherwise.

On the scan

- Hyperdense (about 60–90 HU) biconvex collection against the inner table, usually temporal or temporoparietal.
- A hypodense swirl inside the clot is unclotted blood – active bleeding, and an argument against watchful waiting.
- An overlying fracture in most adults; its absence in a child, whose skull deforms, does not exclude the diagnosis.

● Epidural pearls

- Untreated mortality approaches 100%; with timely evacuation before herniation it falls to single figures. Few operations have that gradient.
- The vein of Labbé is the inferior anastomotic vein draining to the transverse sinus – it has nothing to do with epidural haematoma, whatever the old mnemonic says.
- Venous epidural haematoma exists – from a torn sinus or diploic vein, at the vertex or in the posterior fossa. It grows slowly, may cross the midline, and is easy to under-call.
- Posterior fossa epidural haematoma is uncommon and disproportionately lethal: a small volume in a small compartment next to the fourth ventricle and medulla.

1.2 Subdural haematoma

Blood between dura and arachnoid, usually from rupture of the **cortical bridging veins** that cross from the brain surface to the superior sagittal sinus, tethered at both ends. They carry blood at 5–10 mmHg, so bleeding is slow, the course is variable, and the presentation is often nothing like the textbook trauma case.

The subdural space is not a space

Dura and arachnoid are apposed; there is no pre-existing cavity. What tears is the **dural border cell layer** – loosely joined cells with little extracellular collagen at the innermost dura. Blood cleaves this layer and makes the compartment as it goes, which is why it spreads over a convexity regardless of sutures and stops only at the dural reflections. The "true subdural space" of older texts is a dissection artefact (Haines et al., 1993).

Stage	Interval	On CT	HU	What it means
Acute	0–3 days	Hyperdense	50–80	Usually significant trauma, often with contusion beneath; high mortality.
Subacute	3 days – 3 weeks	Isodense to cortex	25–45	The dangerous window – CT can look normal. Look for effaced sulci and buckled grey–white junction; MRI settles it.
Chronic	> 3 weeks	Hypodense	10–22	Older, anticoagulated, atrophic brains. Membranes form and rebleed.
Acute on chronic	Any	Mixed, layered	Mixed	Fresh bleeding into a chronic collection – escalate.

Table 1.2 Severe anaemia lowers attenuation and can make acute blood look isodense.

Why older people

Atrophy lengthens the unsupported course of the bridging veins and lets the brain move inside the vault, so a trivial mechanism – a fall from standing, sometimes nothing remembered – tears them. The same atrophy leaves room for a large collection before anything localising appears, which is why chronic subdural haematoma presents as gait disturbance, apathy or confusion far more often than as hemiparesis. Anticoagulants, antiplatelets, alcohol and thrombocytopenia all raise the risk.

● Subdural pearls

- Bilateral collections can cancel each other's mass effect: effaced sulci and small ventricles with no midline shift. No shift is not reassurance.
- In a confused older patient with a "normal" CT, check whether the sulci reach the inner table. An isodense collection hides in plain sight.
- Surgical thresholds (acute): thickness over 10 mm or midline shift over 5 mm, whatever the GCS; or GCS $\leq$ 8 with deterioration of two points or more, fixed or asymmetric pupils, or ICP over 20 mmHg.
- Chronic subdural haematoma is a disease of the membrane as much as of the clot: leaky neovascular membranes drive recurrence after burr-hole drainage.

Middle meningeal artery embolisation — what the 2024 trials showed

The neomembranes of a chronic subdural haematoma are fed by the middle meningeal artery; embolising it removes the substrate for re-accumulation. Three randomised trials appeared together in November 2024.

EMBOLISE (400 patients, embolisation added to surgery) cut repeat surgery at 90 days from about 11% to 4%. **STEM** (310 patients, Squid embolic, added to surgical or medical care) reduced treatment failure from 36% to 16%. **MAGIC-MT** (722 patients in China, Onyx) did not reach significance on its primary outcome of symptomatic recurrence or progression (6.7% vs 9.9%), though serious adverse events were fewer.

Practical reading: adjunctive embolisation reduces recurrence and re-operation, with no shown effect on function or survival. It is a reasonable offer for a symptomatic collection that needs drainage, for recurrent collections, and where anticoagulation must restart. It does not replace decompressing a patient with mass effect and a falling GCS.

1.3 Subarachnoid haemorrhage

Bleeding into the CSF between arachnoid and pia. About 85% of spontaneous cases come from a ruptured saccular aneurysm, usually at a bifurcation on or near the circle of Willis; perimesencephalic non-aneurysmal haemorrhage and other causes make up the rest. Because the blood enters flowing CSF, it spreads within seconds through the basal cisterns, over the convexities and down the spinal canal – and disrupts CSF handling for weeks.

The headache

A headache maximal within seconds to a minute. Two mechanisms combine: an abrupt rise in ICP at rupture, and chemical irritation of the pain-sensitive meninges. Meningism develops over hours, so a supple neck early on is not reassuring. A sizeable minority describe a **sentinel headache** days or weeks earlier; recognising and investigating it is one of the few genuinely life-saving acts in this disease.

Imaging, and when the lumbar puncture is still needed

Non-contrast CT on a modern scanner, read by an experienced radiologist, is close to 100% sensitive within six hours of onset. Sensitivity falls after that as blood is cleared. If CT is negative and the story is convincing, **lumbar puncture at least 12 hours after onset** remains the standard next step. Xanthochromia comes from bilirubin made in vivo from oxyhaemoglobin by haem oxygenase – which is why it takes hours to appear and why it distinguishes true haemorrhage from a traumatic tap; spectrophotometry beats inspection. CT angiography is an alternative where expertise allows, at the cost of finding incidental aneurysms.

◆ The first 24 hours

- Rebleeding is the immediate threat – highest in the first hours, and lethal in about half of those it strikes. Secure the aneurysm, by coiling or clipping, as early as feasible and ideally within 24 hours.
- Until then keep systolic pressure below about 160 mmHg and avoid wide swings; reverse anticoagulation.
- Sudden deterioration with new headache after admission is rebleeding until the scan says otherwise.
- Acute hydrocephalus can appear within hours and is reversible with an external ventricular drain. A falling GCS with enlarging ventricles needs a drain, not a wait.

The later complications

- **Delayed cerebral ischaemia** peaks between days 4 and 14. Oxyhaemoglobin drives endothelin release and scavenges nitric oxide, narrowing large arteries – but delayed ischaemia and angiographic vasospasm are not the same thing. Clazosentan largely abolished vasospasm without improving outcome, the clearest demonstration that microvascular failure, microthrombosis and spreading depolarisation matter too. Treat the patient, not the angiogram.
- **Nimodipine** 60 mg orally every 4 hours for 21 days is the only drug with an outcome benefit; start on admission. Triple-H therapy is abandoned: keep euvolaemia, and induce hypertension when a new deficit appears; endovascular rescue for refractory cases.
- **Hydrocephalus** – early obstructive from intraventricular clot, later communicating as breakdown products block absorption. A falling conscious level, sometimes impaired upgaze. External drain acutely; a shunt in the minority who stay dependent.
- **Systemic complications** – neurogenic stunned myocardium, neurogenic pulmonary oedema, hyponatraemia (cerebral salt wasting more often than SIADH, so fluid restriction is harmful) and fever. These kill patients who survived the aneurysm.

Grading, and one negative trial

Hunt and Hess grades the clinical state; the WFNS scale is GCS-based and more reproducible; the modified Fisher scale grades blood on CT and predicts delayed ischaemia best. **ULTRA** tested ultra-early short-course tranexamic acid: fewer rebleeds, no better functional outcome. It is not routine.

1.4 Intracerebral haemorrhage

Bleeding into the brain itself. It causes 10–15% of strokes in high-income settings but about a quarter worldwide, with a higher share across sub-Saharan Africa and Asia, and 30-day mortality of 30–50% that has barely moved in decades. The dominant cause is **hypertensive small-vessel disease**: lipohyalinosis and fibrinoid necrosis of penetrating arteries, with Charcot–Bouchard microaneurysms. In older patients with lobar bleeds, cerebral amyloid angiopathy is the other major cause.

Site	Share	Vessels	Clinical signature
Putamen / basal ganglia	35–50%	Lenticulostriate (MCA)	Contralateral hemiparesis and sensory loss; eyes deviate towards the lesion.
Thalamus	15–20%	Thalamic perforators	Sensory loss dominates; upgaze palsy, small sluggish pupils, sometimes "wrong-way" eyes deviated away from the lesion.
Lobar	20–30%	Cortical branches	Amyloid angiopathy in older people – posterior predilection, multiple and recurrent; high rebleed risk on anticoagulation.

Site	Share	Vessels	Clinical signature
Cerebellum	about 10%	SCA, PICA, AICA branches	Vertigo, vomiting, ataxia, inability to stand, no hemiparesis. Deteriorates through brainstem compression and hydrocephalus — a surgical problem.
Pons	5–10%	Basilar perforators	Pinpoint reactive pupils, hyperthermia, quadriparesis, coma or locked-in state. The highest mortality.

Table 1.4 Ranges across series; they do not sum to 100.

Haematoma expansion is the treatable enemy

About a third of patients have meaningful growth, most of it within the first 3–6 hours; each 10% increase in volume adds roughly 5% to mortality. The window in which anything you do can change the outcome is open when the patient arrives. The CT angiographic **spot sign** marks active extravasation — good specificity, modest sensitivity; useful for prognosis, not for triage.

Estimate volume with **ABC/2**: A, the largest diameter on the slice with the biggest clot; B, the diameter perpendicular to it; C, the number of slices with clot multiplied by slice thickness. It approximates an ellipsoid and over-estimates irregular clots.

◆ The first-hour bundle

- **Blood pressure.** For systolic 150–220 mmHg, lower towards 140 mmHg and hold it steadily (acceptable 130–150), starting within two hours and reaching target within one. INTERACT3 showed a bundle built around this improves function.
- **Do not overshoot.** ATACH-2 found no benefit and more renal harm below 120 mmHg; large, fast drops seem to be what hurts. Avoid falling below 130 mmHg.
- **Reverse anticoagulation now.** Warfarin: four-factor prothrombin complex concentrate plus intravenous vitamin K, aiming for an INR below 1.3. Dabigatran: idarucizumab. Factor Xa inhibitors: andexanet alfa where available, otherwise prothrombin complex concentrate.
- **Glucose, temperature and a swallow screen** are part of the same bundle and the parts most often forgotten.

Andexanet alfa — read ANNEXA-I before you reach for it

ANNEXA-I (2024) compared andexanet with usual care, mostly prothrombin complex concentrate, in factor Xa inhibitor-associated haemorrhage. Haemostatic efficacy was better and the trial stopped early — but thrombotic events, including ischaemic stroke, were more frequent, and function and mortality did not differ. The drug works and has a real thrombotic cost; cost and availability make prothrombin complex concentrate the pragmatic choice in many systems.

Surgery, reconsidered

- **Cerebellar haemorrhage** over 3 cm, or with deteriorating consciousness, brainstem compression or hydrocephalus, is evacuated urgently. A ventricular drain alone is not enough for a compressing posterior fossa clot.
- **Supratentorial haemorrhage** has a long record of neutral trials of open craniotomy (STICH, STICH II). **ENRICH** (2024) changed the picture for a defined group: minimally invasive trans-sulcal parafascicular evacuation within 24 hours of onset improved outcome, driven by lobar haemorrhages of 30–80 mL; deep bleeds did not show the same benefit.

- **Decompressive craniectomy** without evacuation was tested in **SWITCH** (2024) for severe deep haemorrhage: the result favoured surgery but missed conventional significance. A rescue option, not a standard.
- **No prophylactic antiseizure drugs.** Treat clinical seizures, and electrographic seizures found on EEG when consciousness is depressed out of proportion.

When it is not hypertensive

Attributing every bleed to hypertension is the commonest diagnostic error in this disease. Suspect a secondary cause in patients under 55 or without hypertension, and when the haematoma is lobar, purely intraventricular, oddly shaped, multiple, surrounded by disproportionate oedema, or accompanied by subarachnoid blood.

- **Vascular malformation** – arteriovenous malformation, cavernoma, dural fistula. The commonest secondary cause in the young.
- **Aneurysm** – especially with subarachnoid blood, or a frontal or temporal clot next to the circle of Willis.
- **Venous sinus thrombosis** – haemorrhagic venous infarction that ignores arterial territories, often parasagittal or temporal, with disproportionate oedema. The treatment is anticoagulation – the opposite of the reflex.
- **Tumour** – melanoma, renal, thyroid, choriocarcinoma and lung metastases bleed; so does glioblastoma.
- **Drugs and coagulopathy** – sympathomimetics, anticoagulants, thrombolysis, and haemorrhagic transformation of an infarct.
- **Vasculopathy** – RCVS, vasculitis, moyamoya; consider them in recurrent or multifocal bleeds.

● The delayed scan

CT angiography in the acute phase finds the lesions that bleed at pressure. Acute blood hides everything else. Repeat MRI with susceptibility-weighted imaging at 6–8 weeks, once the clot has resolved, to unmask a cavernoma, a tumour, or the lobar microbleeds and cortical siderosis of amyloid angiopathy – which change anticoagulation decisions for the rest of the patient's life.

1.5 Master comparison

	Epidural	Subdural	Subarachnoid	Intracerebral
Plane	Skull ↔ periosteal dura	Cleaved dural border cells	Within the CSF	Brain parenchyma
Vessel	Middle meningeal artery	Bridging veins	Saccular aneurysm (~85%)	Penetrating arterioles; amyloid vessels
Pressure	Arterial	Venous, 5–10 mmHg	Arterial, into CSF	Arterial, into tissue
Shape on CT	Biconvex lens	Crescent	Cast of cisterns and sulci	Rounded or irregular mass
Limited by	Sutures	Falx and tentorium	Nothing	Tissue planes
Crosses	The midline	Sutures	Everything	Into the ventricles
Presentation	Impact, lucid interval, abrupt decline	Insidious confusion, gait change	Thunderclap	Focal deficit, headache, vomiting
Early threat	Uncal herniation within hours	Mass effect over days	Rebleeding	Expansion in 3–6 h

	Epidural	Subdural	Subarachnoid	Intracerebral
Later threat	Seizures	Recurrence from membranes	Delayed ischaemia; hydrocephalus	Oedema peaking at days 3–5
First move	Evacuate	Evacuate if symptomatic; consider embolisation	Secure aneurysm; nimodipine	SBP towards 140 within the hour; reverse anticoagulation

Table 1.5 Almost every difference traces to two variables: the pressure in the bleeding vessel and the plane the blood is forced into.

Part II

Meningeal architecture

The layers · The dural venous sinuses · The blood–brain barrier

2.1 The meningeal layers

Dura mater

A dense fibrous membrane in two layers: the outer periosteal layer, which is the periosteum of the inner table, and the inner meningeal layer, which continues as the spinal dura. They are fused everywhere except where they part to enclose the venous sinuses. The meningeal layer folds inward as the falx cerebri, tentorium cerebelli and falx cerebelli. The tentorial notch, through which the midbrain passes, is the choke point in uncal herniation – the reason a supratentorial mass kills through a brainstem it never touches.

The dura is the main pain-sensitive covering of the brain. Above the tentorium it is supplied by the trigeminal nerve; below, by the upper cervical roots and branches of the vagus and glossopharyngeal nerves. Supratentorial pathology refers pain to the front of the head; posterior fossa pathology to the occiput and neck.

Arachnoid mater

Thin, avascular, held against the dura by CSF pressure. It bridges over the sulci rather than following them, creating the cisterns wherever the brain surface falls away. Fine trabeculae cross the subarachnoid space to the pia. The arachnoid granulations project into the venous sinuses and act as one-way valves, opening when CSF pressure exceeds sinus pressure by a few mmHg.

Pia mater

The innermost layer, adherent to every gyrus and sulcus. As an artery dives into the parenchyma it carries a sleeve of pia, forming the perivascular (Virchow–Robin) space – the anatomical substrate of glymphatic exchange.

2.2 The dural venous sinuses

Valveless, rigid channels between the two dural layers that receive cerebral venous blood and CSF. Their rigidity is the point: they do not collapse when ICP rises, which keeps venous outflow possible until pressures become extreme.

- **Superior sagittal sinus** – along the attached margin of the falx to the torcular; the main recipient of arachnoid granulations; usually drains preferentially to the right transverse sinus. Thrombosis gives bilateral parasagittal venous infarcts, often haemorrhagic, and can cause convexity subarachnoid haemorrhage.

- **Transverse and sigmoid sinuses** – with the superior sagittal sinus, the commonest sites of venous thrombosis. A hypoplastic left transverse sinus is a normal variant and a frequent source of false-positive reports.
- **Cavernous sinuses** – either side of the sella. The oculomotor and trochlear nerves and the ophthalmic and maxillary divisions run in the lateral wall; the abducens nerve runs free within the sinus beside the internal carotid artery, so an isolated sixth nerve palsy can be the first sign of cavernous sinus disease. Septic thrombosis from mid-facial infection gives painful ophthalmoplegia, proptosis and chemosis.
- **Straight sinus** – draining the vein of Galen and so the deep white matter, basal ganglia and thalami. Deep venous thrombosis gives bilateral thalamic oedema and a falling conscious level – an easily missed and treatable cause of coma.

2.3 The blood–brain barrier

Tight junctions between specialised endothelial cells – claudins and occludin anchored by zonula occludens proteins – supported by pericytes, a basal lamina and astrocytic end-feet. It excludes most hydrophilic molecules, ions and proteins, including most drugs.

In haemorrhage the barrier fails locally. Thrombin, complement and iron released from degrading haemoglobin drive **perihæmatomal oedema**, which peaks around days 3–5 and can exceed the volume of the clot. This secondary injury is the most attractive therapeutic target in the disease and, so far, the least successfully hit.

Part III

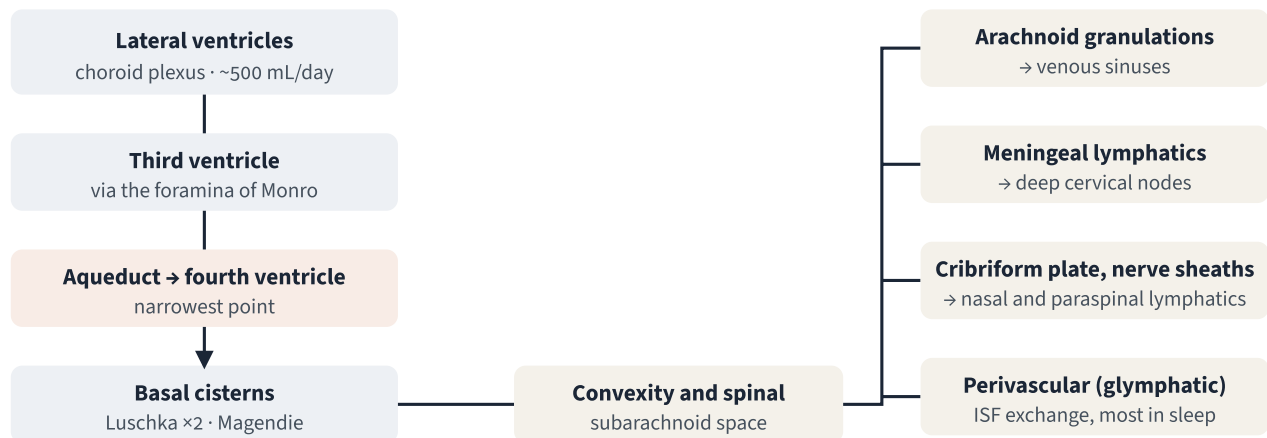
CSF dynamics

Production · Circulation · Absorption and clearance · Reference values

3.1 Production

CSF is secreted by the choroid plexus: a sealed epithelium over leaky, fenestrated capillaries – the reverse of the usual arrangement, which puts the blood–CSF barrier at the epithelium. Secretion is active: basolateral Na^+/K^+ -ATPase sets the gradient, carbonic anhydrase supplies bicarbonate and protons, aquaporin-1 provides the water route. Because it is active, production is largely independent of pressure – which is why a blocked pathway causes relentless hydrocephalus.

- About 500 mL a day from a standing volume of about 150 mL: three to four turnovers daily, about 0.35 mL a minute.
- Most is made in the lateral ventricles; the rest in the third and fourth ventricles, with an extrachoroidal contribution.
- Acetazolamide reduces production modestly – the basis of its use in idiopathic intracranial hypertension.



Block above the cisterns: obstructive (non-communicating). Block at the exits: communicating — as after subarachnoid haemorrhage.

Figure 3. The granulation route is one of several exits. The distinction that matters clinically is where the block sits relative to the cisterns.

3.2 Circulation

From the lateral ventricles CSF passes through the foramina of Monro into the third ventricle, then through the **aqueduct of Sylvius** — narrow and the most stenosis-prone segment of the pathway. Aqueductal stenosis, pineal tumours and tectal gliomas obstruct here. The fourth ventricle empties through the paired foramina of Luschka and the midline foramen of Magendie. Obstruction at this level — posterior fossa tumour, cerebellar haematoma, Chiari malformation — is dangerous out of proportion to its size, because the compartment has no room and the medulla is next door. Beyond, CSF flows through the cisterns, over the convexities and into the spinal subarachnoid space, the compliant reservoir that buys time when intracranial volume rises.

3.3 Absorption and clearance

The classical account gives all absorption to the arachnoid granulations. That model is correct but incomplete.

- **Glymphatic exchange** — aquaporin-4-dependent movement of CSF along perivascular spaces into the interstitium and out again, carrying solutes such as amyloid- β . In mice it rises sharply in sleep, when the interstitial space expands by about 60% (Xie et al., 2013).
- **Meningeal lymphatics** — described in 2015, running along the dural sinuses to the deep cervical nodes.
- **Cribriform plate and nerve sheaths** — CSF tracks along olfactory and spinal nerve sheaths into lymphatics, proportionally more in the young.

Why the taxonomy earns its keep

After subarachnoid haemorrhage, breakdown products impair absorption while the pathway stays open: CSF circulates but cannot leave — communicating hydrocephalus, which can appear weeks after the aneurysm is secured. In aqueductal stenosis or fourth-ventricular outlet obstruction, CSF cannot reach the exits at all — non-communicating hydrocephalus, in which lumbar drainage is dangerous.

3.4 Reference values

Parameter	CSF	Serum	Interpretation
Opening pressure	6–25 cmH ₂ O, lateral decubitus, legs extended	—	Above 25 supports raised pressure (25–28 is a grey zone in obesity). Below 6 suggests intracranial hypotension.
Protein	15–45 mg/dL	6–8 g/dL	Raised in infection, haemorrhage, tumour, Guillain–Barré; rises with a traumatic tap.
Glucose	About 60% of serum (ratio $\geq$ 0.6)	Paired sample	Low (ratio under 0.4–0.5) in bacterial, tuberculous, fungal and carcinomatous meningitis.
White cells	< 5 per μ L, mononuclear	—	Neutrophils are not normal. Correct for a traumatic tap before calling a pleocytosis.
Red cells	0	—	Xanthochromia, not falling red cell counts, distinguishes haemorrhage from a traumatic tap.
Lactate	About 1–2.5 mmol/L	—	Above about 3.5 mmol/L favours bacterial meningitis and discriminates well even after antibiotics have started.

Table 3.4 Adult values; use your laboratory's ranges where they differ.

Part IV

Intracranial pressure

Monro–Kellie · Compliance and waveforms · High- and low-pressure headache

4.1 The Monro–Kellie doctrine

After the sutures fuse, the skull is a rigid box holding three effectively incompressible contents: brain (about 1,400 mL), blood (about 150 mL) and CSF (about 150 mL). An increase in one must be matched by a decrease in another or pressure rises. Normal ICP in a supine adult is 5–15 mmHg.

The doctrine's real value is that it explains **timing**. Compensation is finite and is spent in order: CSF is displaced into the spinal sac (the largest, fastest buffer), CSF absorption rises, venous blood is squeezed out, and production falls slightly. Once that reserve is used, each further millilitre costs disproportionate pressure.

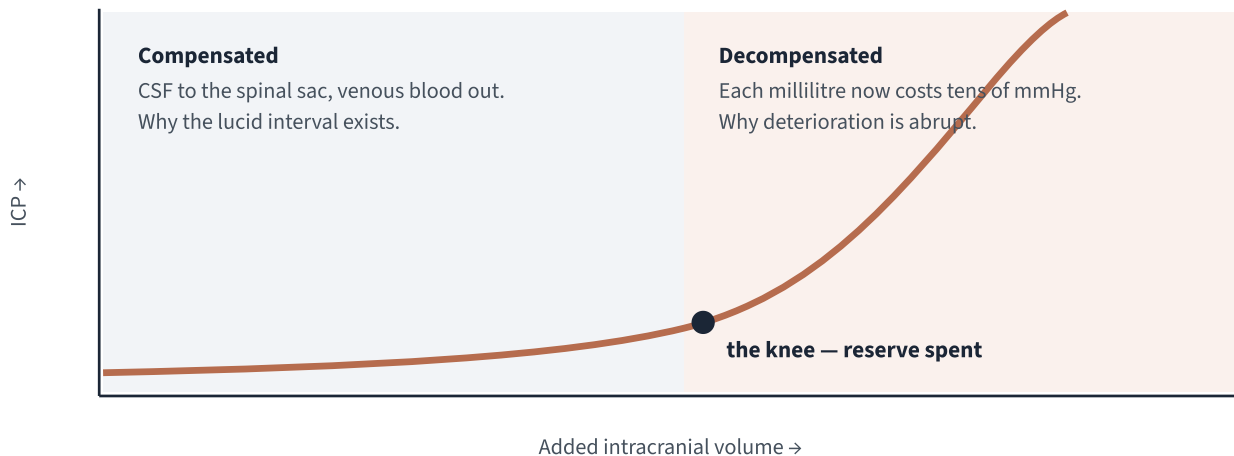


Figure 4. The same curve explains the lucid interval, the abruptness of deterioration, and why a P2-dominant waveform is worth noticing before the mean pressure rises.

4.2 Waveforms and plateau waves

A monitored ICP trace is pulsatile, synchronous with the heartbeat and modulated by breathing. Three peaks are described: **P1**, the percussion wave of the arterial pulse; **P2**, the tidal wave, which reflects intracranial compliance; and **P3**, the dicrotic wave after aortic valve closure. Normally $P1 > P2 > P3$. As compliance falls, P2 rises above P1 – often before the mean ICP moves, which makes it one of the few early warnings available.

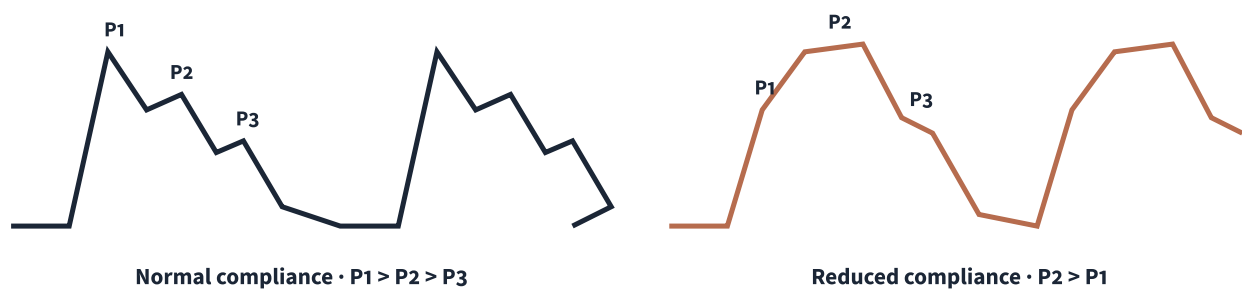


Figure 5. Morphology before magnitude: a rounded, P2-dominant trace at a normal mean pressure belongs to a patient who has already spent their reserve.

- **Lundberg A (plateau) waves** – sustained rises to 50–100 mmHg for 5–20 minutes, then abrupt return. They reflect exhausted compliance with intact but overwhelmed autoregulation, and are an emergency.
- **Lundberg B waves** – rhythmic oscillations at 0.5–2 per minute, marking reduced compliance.
- **Lundberg C waves** – 4–8 per minute, linked to systemic arterial (Traube–Hering–Mayer) waves; of little significance.

4.3 High-pressure headache and its treatment

Sustained raised pressure produces headache by distending and pulling on the pain-sensitive dura, sinuses and proximal arteries; the parenchyma has no nociceptors.

- **Worse in the morning and lying flat** – recumbency impedes venous drainage, and mild nocturnal hypoventilation raises PaCO_2 and cerebral blood volume.
- **Worse with Valsalva** – coughing, straining and bending obstruct jugular outflow.
- **Papilloedema** takes days to develop; its absence never excludes acutely raised pressure.

- **Sixth nerve palsy** – the classic false-localising sign.
- **Vomiting**, often without much nausea; **Cushing's response** – hypertension, bradycardia, irregular breathing – late and pre-terminal.

Treat the perfusion, not the number

Cerebral perfusion pressure = mean arterial pressure – ICP. The usual target is 60–70 mmHg, defended by supporting arterial pressure as much as by lowering ICP. Where autoregulation is intact, raising MAP lowers ICP by vasoconstriction; where it is lost, the same manoeuvre raises ICP. Autoregulation-guided targets (pressure reactivity, "optimal CPP") are the direction of travel.

Tier	Trigger	Interventions (after SIBICC, 2019)
Baseline	Every monitored patient	Head up 30°, neck neutral, nothing tight around the neck. Normoxia, PaCO ₂ 35–45 mmHg, normothermia, normoglycaemia, sodium normal or high. Analgesia and sedation. Treat pain, fever, seizures and agitation first.
Tier 1	ICP > 22 mmHg sustained	Deepen sedation; drain CSF if there is a ventricular drain; osmotherapy — mannitol 0.25–1 g/kg or hypertonic saline — watching sodium and osmolality.
Tier 2	Refractory	Mild hypocapnia (PaCO ₂ 32–35 mmHg) with brain oxygen monitoring where available; neuromuscular blockade trial; MAP challenge to test autoregulation.
Tier 3	Refractory to tier 2	Decompressive craniectomy, barbiturate coma titrated to effect, or mild hypothermia. Each carries real harm; the decision is as much about trajectory and goals as about pressure.
Rescue	Herniation in progress	Brief hyperventilation as a bridge measured in minutes, with an osmotic bolus, while definitive treatment is arranged.

4.4 Low-pressure headache

Intracranial hypotension is usually a CSF leak – after lumbar puncture, spontaneously through a spinal dural tear or CSF–venous fistula, or after trauma or surgery. An opening pressure below 6 cmH₂O supports it, but many patients with proven leaks have normal pressures.

The brain weighs about 1,400 g in air and about 50 g floating in CSF. Lose the buoyancy and it sags on standing, pulling on the bridging veins, dura and pain-sensitive nerves. Monro–Kellie demands compensation for the lost volume, delivered as venous engorgement – which produces the MRI picture: smooth pachymeningeal enhancement, subdural collections, engorged sinuses, pituitary swelling and brain sag with tonsillar descent that can be mistaken for Chiari I.

● Post-dural puncture headache

- Prevention beats treatment: an atraumatic (pencil-point) needle roughly halves the incidence compared with a cutting needle.
- Bed rest and aggressive hydration do not prevent or shorten it; caffeine gives brief relief at best.
- The epidural blood patch is the definitive treatment; durable relief after a first patch is lower than the often-quoted 90%, and a second patch rescues most of the rest.
- In spontaneous intracranial hypotension, image the whole spine and consider dynamic myelography to find a leak or CSF–venous fistula before targeted treatment.

Part V

Synthesis

Rates and pressures · Herniation · CT chronology · Prognosis

5.1 Why the epidural kills faster than the subdural

The answer is hydraulic. The middle meningeal artery delivers blood at systemic pressure; bridging veins at 5–10 mmHg. Flow through a breach scales with the pressure gradient, so an epidural collection fills many times faster and decompensation arrives in minutes to hours rather than days to weeks. Location compounds it: an epidural haematoma sits over the temporal squama, directly lateral to the uncus. And the brains differ: the young brain that suffers an epidural has no atrophic reserve; the atrophic brain that develops a chronic subdural has room to spare.

5.2 Subarachnoid haemorrhage – sabotage of the drainage system

Subarachnoid haemorrhage is the only one of the four that attacks CSF handling directly. Fibrin and blood products obstruct the arachnoid granulations and provoke fibrosis, so absorption stays impaired long after the aneurysm is secured. Meanwhile the cisternal blood narrows large arteries and disturbs the microcirculation. Four waves arrive in sequence – the ictus, rebleeding, delayed ischaemia from day 4 to 14, and hydrocephalus that may take weeks – each with its own window and its own intervention.

5.3 Herniation syndromes

Syndrome	Movement	Structures compressed	Signs
Uncal (lateral transtentorial)	Medial temporal lobe through the tentorial notch	CN III, cerebral peduncle, PCA, midbrain reticular formation	Ipsilateral dilated pupil first, then contralateral hemiparesis and falling consciousness; occipital infarction. Kernohan's notch gives ipsilateral weakness.
Central (downward)	Both hemispheres downward	Diencephalon, then midbrain, pons, medulla	Small reactive pupils becoming mid-position and fixed; decorticate then extensor posturing; Cheyne–Stokes then irregular breathing.
Subfalcine	Cingulate gyrus under the falx	Anterior cerebral artery	Often silent early; leg weakness and abulia if ACA territory infarcts.
Tonsillar	Cerebellar tonsils into the foramen magnum	Medulla, respiratory and vasomotor centres	Neck stiffness, head tilt, then apnoea, bradycardia and death — often without warning.
Upward	Posterior fossa contents through the notch	Superior cerebellum, SCA, aqueduct, dorsal midbrain	Obstructive hydrocephalus, impaired upgaze. Classically after draining supratentorial CSF above a posterior fossa mass.

5.4 CT chronology of blood

Stage	Interval	Attenuation	Why
Hyperacute	< 6 hours	30–45 HU	Liquid blood, protein not yet concentrated; may be under-estimated.
Acute	6 hours – 3 days	50–90 HU	Clot retraction concentrates globin — peak density.
Early subacute	3–7 days	35–50 HU	Lysis begins at the periphery; fading from outside in.

Stage	Interval	Attenuation	Why
Late subacute	1–3 weeks	20–35 HU	Passes through isodensity with cortex — CT is least reliable here.
Chronic	> 3 weeks	< 20 HU	Liquefaction; a serous collection, sometimes with a membrane.

Table 5.4 Severe anaemia (haemoglobin below about 8–9 g/dL) and coagulopathy lower attenuation throughout.

5.5 Prognosis, and the trap inside it

The **ICH score** (Hemphill, 2001) estimates 30-day mortality in seconds and makes handovers precise.

Component	Finding	Points
Glasgow Coma Scale	13–15 · 5–12 · 3–4	0 · 1 · 2
Haematoma volume	< 30 mL · ≥ 30 mL	0 · 1
Intraventricular extension	No · yes	0 · 1
Infratentorial origin	No · yes	0 · 1
Age	< 80 · ≥ 80	0 · 1
30-day mortality in the derivation cohort	Score 0: 0% · 1: 13% · 2: 26% · 3: 72% · 4: 97% · 5: 100%	

◆ The self-fulfilling prophecy

The scores that predict death were derived in cohorts where care was often withdrawn early. Applied as a verdict, the number helps produce the outcome it predicts. The AHA/ASA guideline advises aggressive full care in the first days and against new do-not-resuscitate decisions or limits of care based on a score alone. Treat first; decide later, with the family, on the trajectory you actually see.

● The integrative frame

- **Epidural** – arterial pressure into a potential space bounded by sutures. Minutes are the unit of decision.
- **Subdural** – venous pressure into a cleaved cell layer, in a brain with room to spare. Slow, silent, a disease of the membrane.
- **Subarachnoid** – arterial blood into circulating CSF. Four waves of injury, four windows.
- **Intracerebral** – a diseased penetrating artery, clot toxicity and barrier failure. The window that matters is the first hours.
- All four converge on one pathway: rising ICP, falling perfusion pressure, global ischaemia. **Monro–Kellie** is the clock behind every decision.

Appendix

Abbreviations and sources

For checking

A Abbreviations

ABC/2	bedside haematoma volume formula
CAA	cerebral amyloid angiopathy
CPP	cerebral perfusion pressure
CTA	CT angiography
DCI	delayed cerebral ischaemia
EDH	epidural (extradural) haematoma
EVD	external ventricular drain
ICH	intracerebral haemorrhage
ICP	intracranial pressure
ISF	interstitial fluid
MAP	mean arterial pressure
MMA	middle meningeal artery
PCC	prothrombin complex concentrate
SAH	subarachnoid haemorrhage
SDH	subdural haematoma
SIBICC	Seattle International Severe Traumatic Brain Injury Consensus Conference
WFNS	World Federation of Neurosurgical Societies

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