

Raised Intracranial Pressure and Herniation

Mechanisms, morphology, and clinicopathologic consequences

Dr Abu Suraih Sakhri

MBBS, MD (Pathology)

www.abusuraisakhri.com

Learning objectives

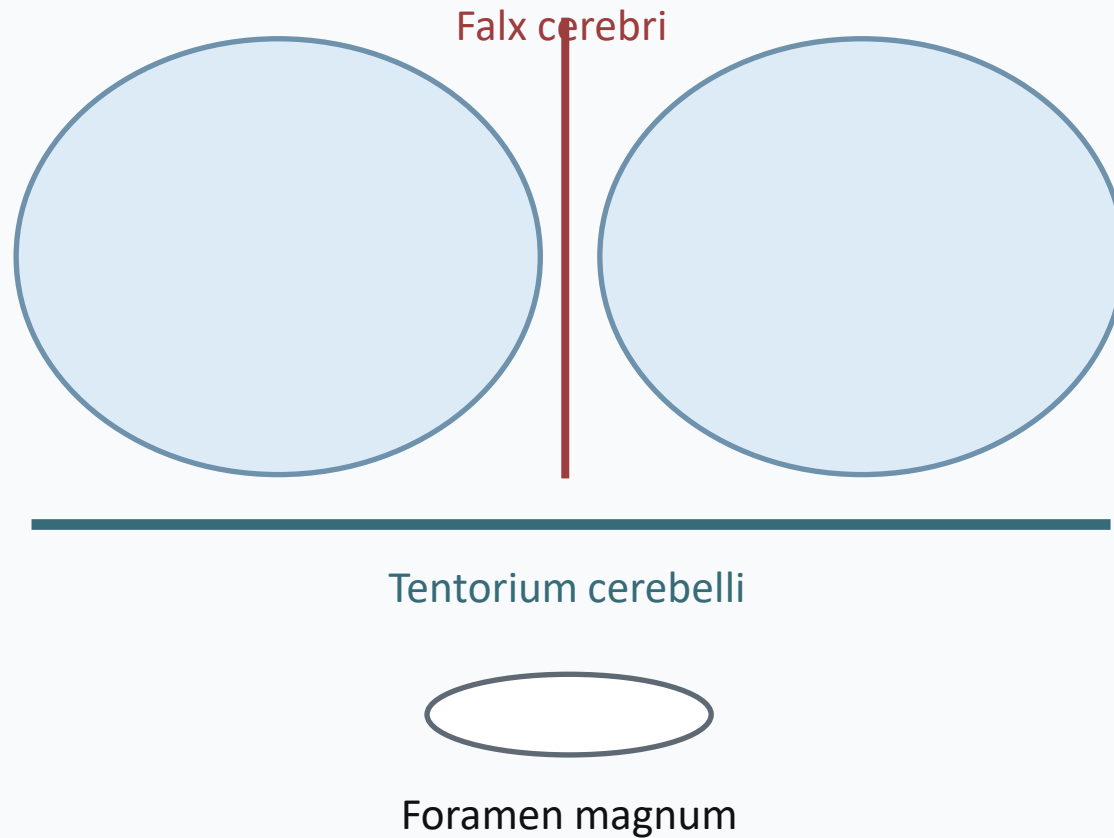
- Explain how added intracranial volume produces pressure and displacement.
- Distinguish vasogenic edema, cytotoxic edema, and hydrocephalus as contributors.
- Identify the three major anatomic routes of brain herniation.
- Correlate compressed neural and vascular structures with clinical findings.

Herniation is an anatomic displacement

- Brain tissue moves across rigid dural boundaries or through skull openings.
- The immediate driver is mass effect with increased intracranial pressure.
- The destination and structures compressed determine the consequences.

A mechanical event—not simply a pressure measurement.

Rigid boundaries create preferred routes



Mechanistic interpretation

- Falx cerebri separates the cerebral hemispheres.
- Tentorium cerebelli partitions supratentorial and infratentorial spaces.
- The foramen magnum provides an inferior opening.

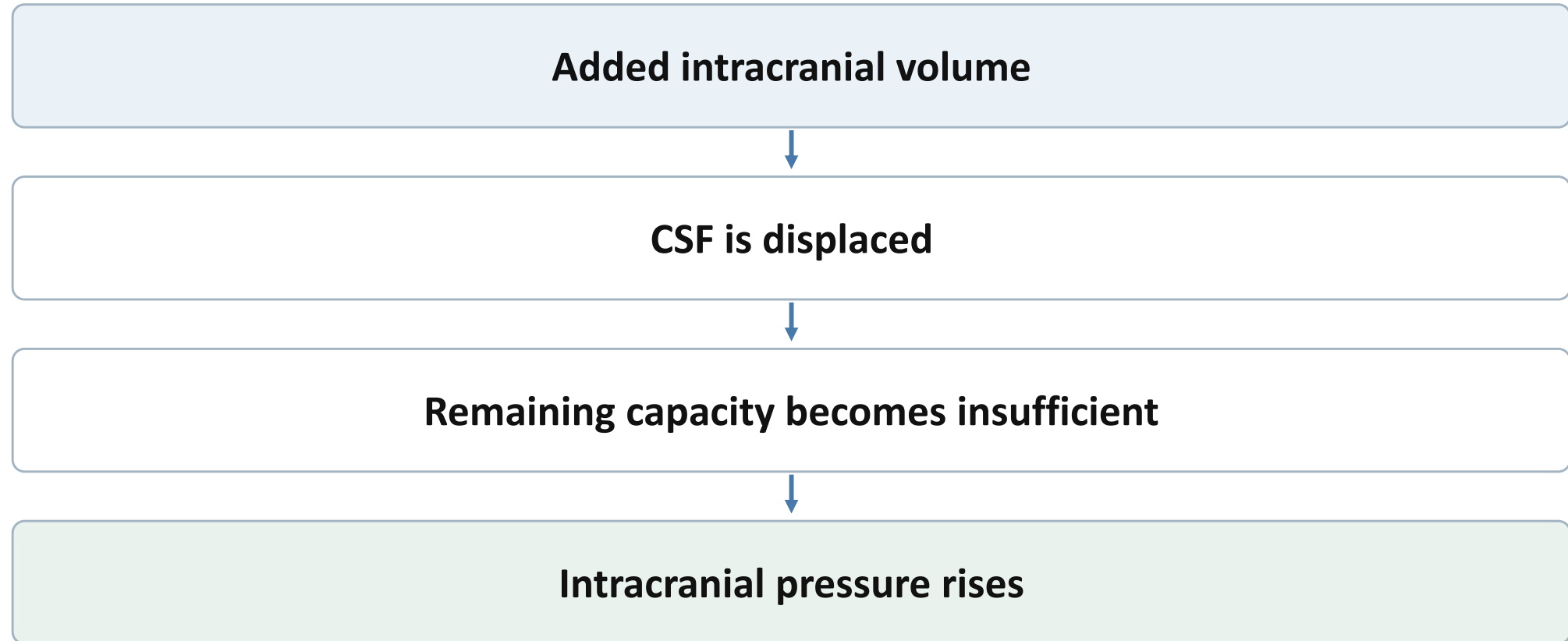
Schematic

The cranium has limited capacity to expand

- Brain parenchyma occupies a rigid cranial compartment.
- An additional mass or swollen brain increases the volume burden.
- Displacement of cerebrospinal fluid initially accommodates part of the change.

The reserve is limited, not unlimited.

Compensation precedes displacement



This sequence describes the mechanism; The boxes are not a calibrated pressure-volume curve.

Once accommodation is exceeded, pressure rises

- Brain swelling can no longer be accommodated simply by shifting CSF.
- Pressure increases within the cranial cavity.
- Pressure gradients and restricted openings favor tissue displacement.

Herniation indicates failure of spatial accommodation.

Diffuse and focal mass effects differ

Diffuse expansion

- Generalized cerebral edema enlarges brain parenchyma.
- Distributed swelling can compromise multiple compartments.

Focal expansion

- Tumor, abscess, or hemorrhage adds volume locally.
- Asymmetric pressure can preferentially displace adjacent tissue.

Both mechanisms may culminate in herniation.

Generalized edema is a diffuse driver

- Excess fluid accumulates within brain parenchyma.
- The swollen brain presses against the dura and inner skull surface.
- Flattened gyri, narrowed sulci, and ventricular compression reflect expansion.

The gross pattern supplies clues to intracranial mass effect.

Focal lesions distort tissue asymmetrically

- A tumor occupies intracranial volume.
- An abscess is a focal space-occupying lesion.
- Intracranial hemorrhage can produce abrupt mass effect.

Lesion site and direction of expansion matter.

Raised pressure is not synonymous with herniation

Raised intracranial pressure

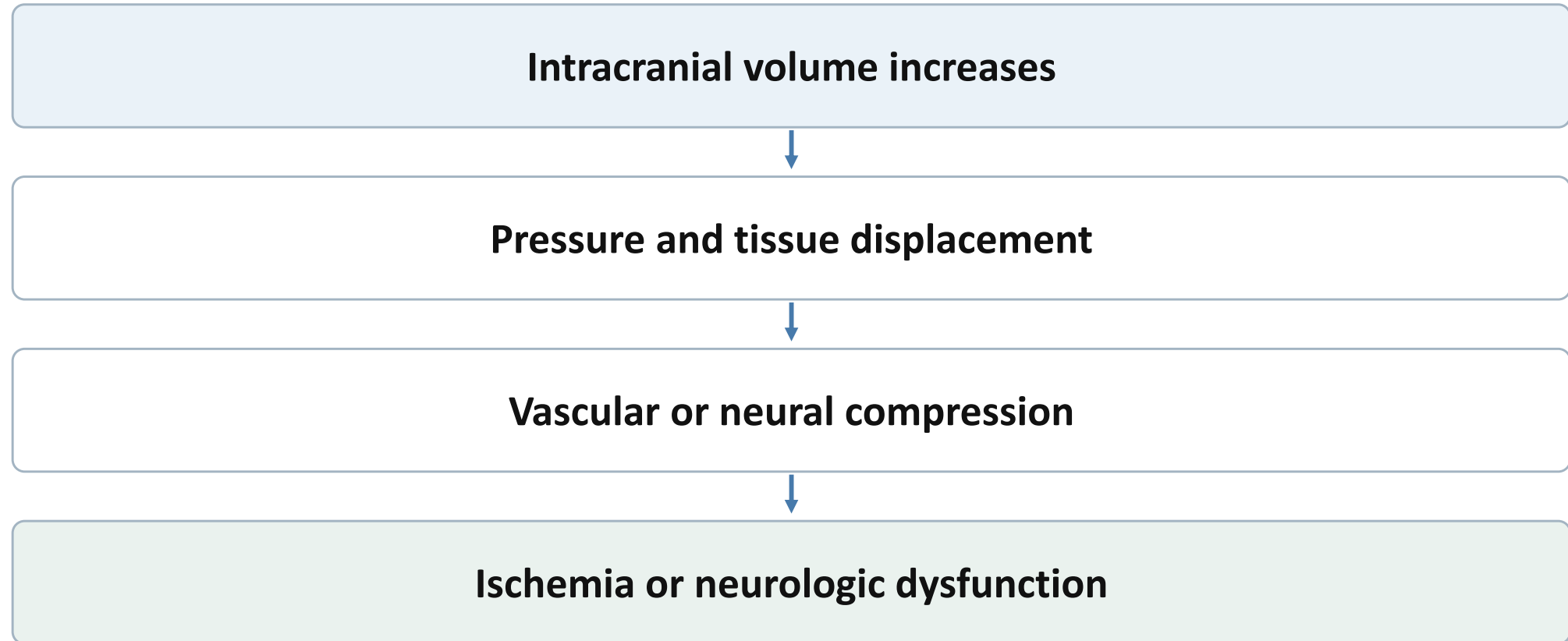
- A physiologic consequence of increased intracranial content.
- Can compromise perfusion even before obvious tissue displacement.

Herniation

- An anatomic shift across a dural fold or skull opening.
- Can directly compress vessels, nerves, and the brainstem.

Pressure and displacement can coexist and reinforce injury.

Two pathways connect mass effect to damage



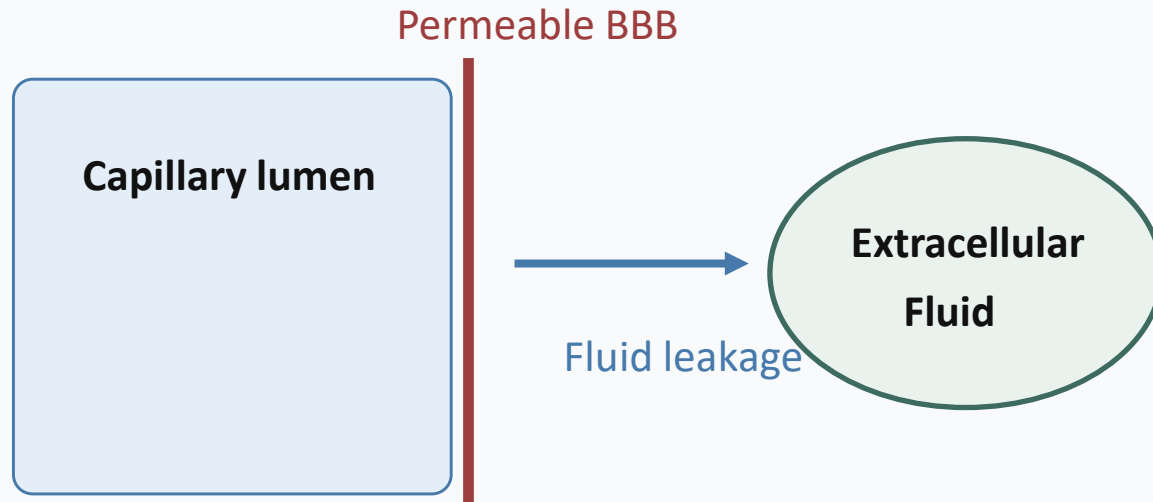
Clinical findings depend on which structures are compromised.

Cerebral edema is parenchymal fluid accumulation

- Fluid accumulation occurs within brain tissue, not merely the ventricles.
- Two major mechanisms: Vasogenic and cytotoxic.
- Both can contribute to a generalized swollen brain.

Edema is a central upstream mechanism.

Vasogenic edema: Fluid escapes the vessels



Schematic

Mechanistic interpretation

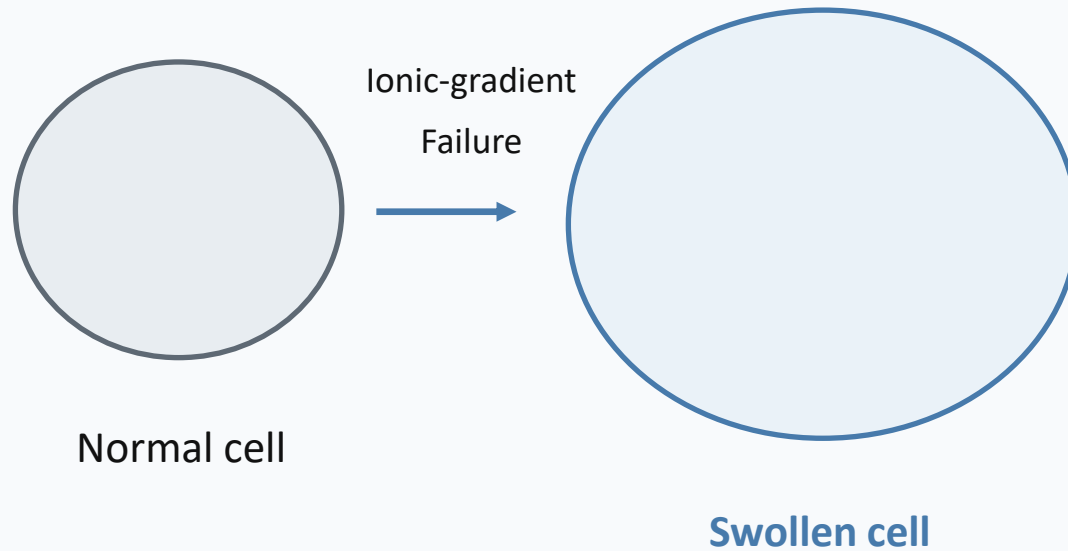
- Disruption of the blood–brain barrier increases permeability.
- Fluid leaves the intravascular space for the extracellular compartment.
- Limited brain lymphatic clearance slows removal of excess interstitial fluid.

Vasogenic edema may be local or widespread

- Localized vasogenic edema can occur adjacent to inflammation.
- Neoplasms can also be surrounded by vasogenic edema.
- Generalized vasogenic edema may follow a global ischemic insult.

Distribution influences the pattern of mass effect.

Cytotoxic edema: Cells swell



Schematic

Mechanistic interpretation

- Injury involves neuronal, glial, or endothelial cell membranes.
- Disturbed ionic gradients favor intracellular water accumulation.
- The expanded compartment is intracellular rather than primarily interstitial.

Cytotoxic edema accompanies failure of ionic balance

- Generalized hypoxic–ischemic injury is a textbook example.
- Metabolic derangements can prevent maintenance of membrane ionic gradients.
- Several CNS cell populations may become swollen together.

Mechanism explains why a diffuse insult can enlarge the whole brain.

The two edema mechanisms affect different spaces

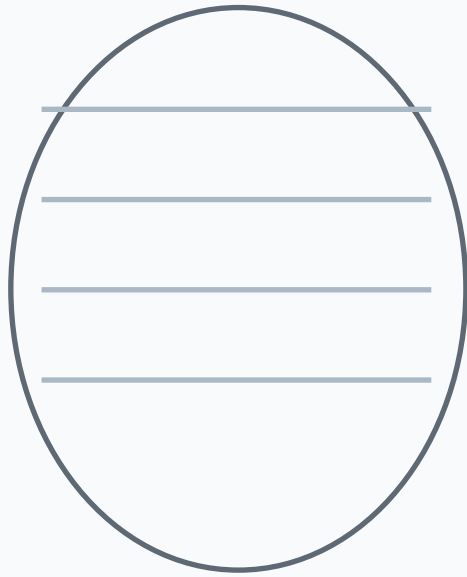
Feature	Vasogenic	Cytotoxic
Primary fluid location	Extracellular	Intracellular
Initiating disturbance	Blood–brain barrier permeability	Cellular membrane / ionic gradient injury
Illustrative settings	Neoplasm, inflammation, global ischemia	Hypoxia–ischemia, metabolic derangement
Shared downstream effect	Parenchymal expansion	Parenchymal expansion

Generalized edema often has mixed mechanisms

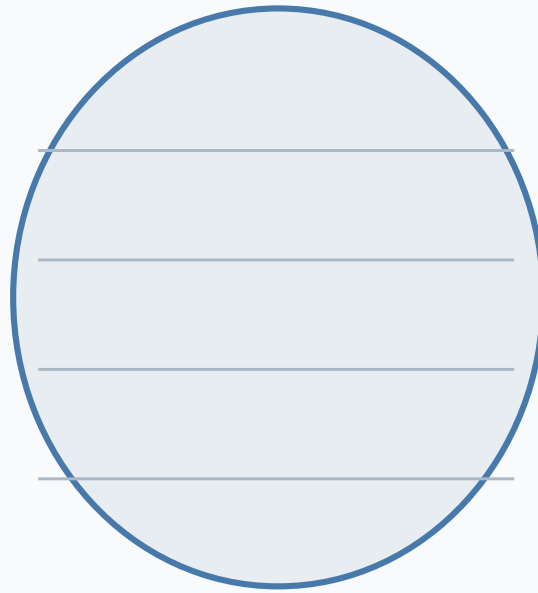
- Vasogenic and cytotoxic contributions may coexist.
- The bedside distinction is not an absolute separation into two diseases.
- The shared pathologic effect is an increase in brain volume.

Do not assume one mechanism excludes the other.

Gross edema: Swollen brain against the skull



Less compressed



Flattened gyri

Mechanistic interpretation

- The expanding brain is constrained by the dura and calvarium.
- Gyrus surfaces become flattened.
- Intervening sulci become narrowed.

Schematic

Ventricular compression can signal parenchymal swelling

- Expansion of the surrounding brain can narrow the ventricular cavities.
- This contrasts with ventricular enlargement caused by excess CSF.
- The distinction requires identifying what compartment has expanded.

Edema and hydrocephalus may produce different ventricular appearances.

How edema progresses toward herniation

Vasogenic and/or cytotoxic edema



Brain volume increases



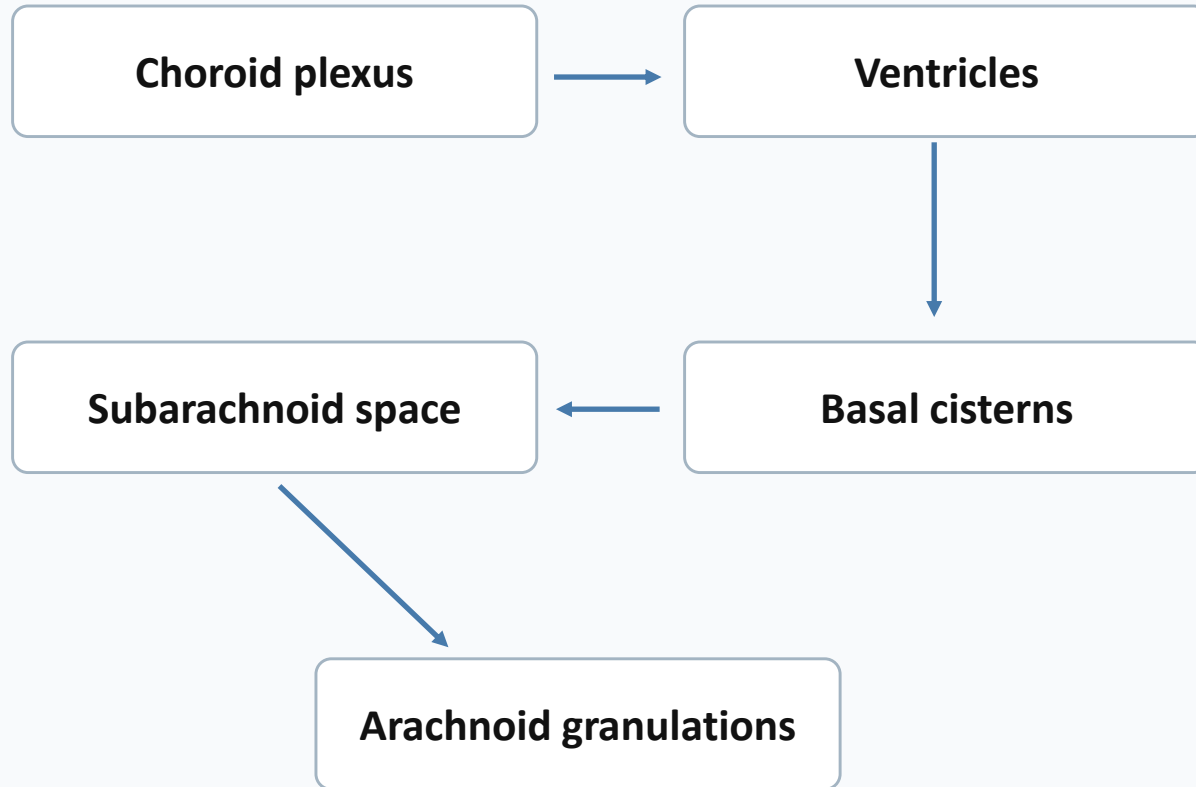
CSF accommodation is exhausted



Tissue displaces across rigid boundaries

The route is conditioned by local anatomy and the distribution of swelling.

Normal CSF circulation supplies context



Schematic

Mechanistic interpretation

- CSF is produced by choroid plexus within the ventricular system.
- It exits toward basal cisterns through the foramina of Luschka and Magendie.
- Subarachnoid CSF is absorbed by arachnoid granulations.

Hydrocephalus expands the ventricular CSF volume

- Hydrocephalus means excessive CSF accumulation within ventricles.
- Increased volume expands the ventricular system.
- It can increase intracranial pressure, especially with closed cranial sutures.

The expansion is initially ventricular rather than interstitial.

Obstruction causes noncommunicating hydrocephalus

A focal blockage interrupts ventricular CSF flow



CSF accumulates upstream



Affected ventricles enlarge



Intracranial pressure may rise

The precise enlargement pattern depends on obstruction location.

An aqueductal stenosis can obstruct CSF flow

- Stenosis prevents normal passage through the cerebral aqueduct.
- The ventricular system is functionally divided by the obstruction.
- This is a textbook example of noncommunicating hydrocephalus.

An anatomic blockage is the defining feature.

A third-ventricular mass can obstruct flow

- A mass in the third ventricle can impede the normal CSF pathway.
- The resulting hydrocephalus is noncommunicating if flow is focally blocked.
- Mass effect and excess upstream CSF may coexist.

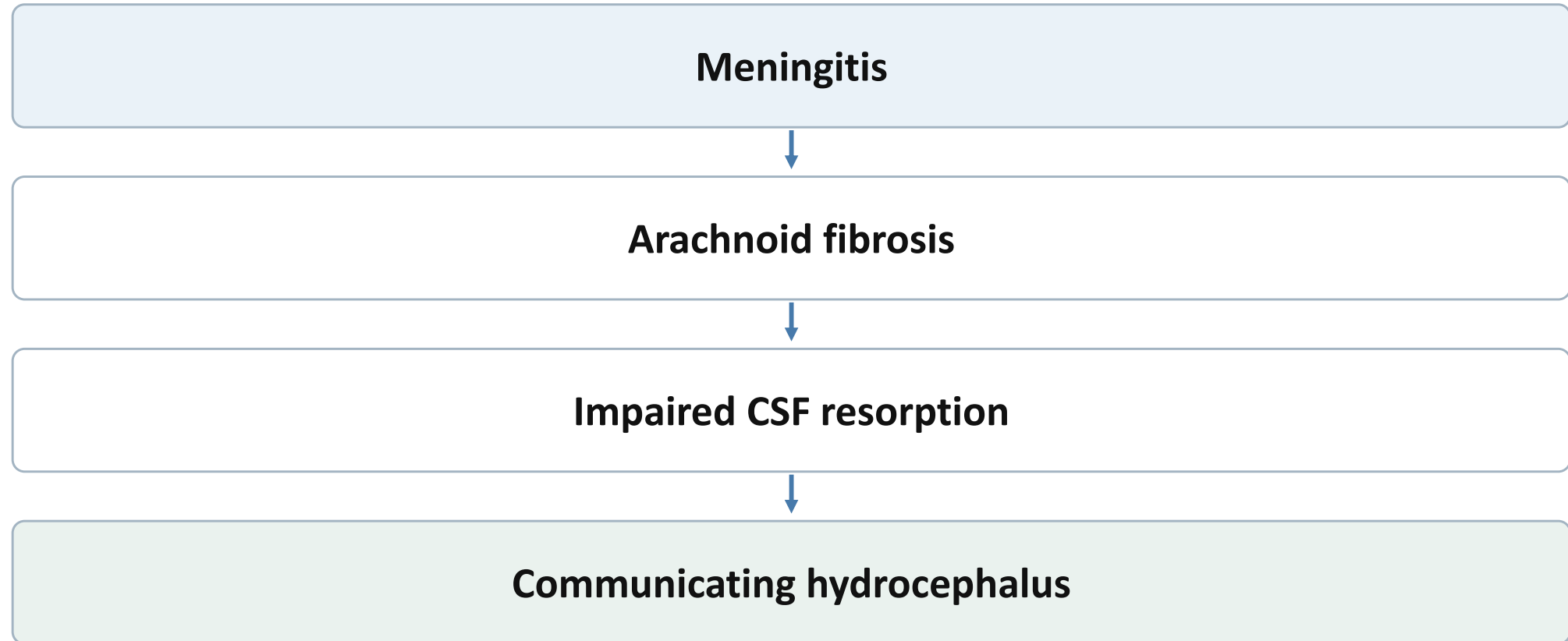
One lesion can contribute volume by more than one route.

Communicating hydrocephalus preserves pathway continuity

- The ventricular system remains in continuity with subarachnoid CSF.
- The entire ventricular system may enlarge.
- Impaired resorption or, rarely, excessive production contributes.

Do not confuse communicating with pressure-free.

Post-meningitic fibrosis can impair CSF absorption



Arachnoid fibrosis after meningitis as an example.

Choroid plexus tumors can rarely increase CSF production

- Most hydrocephalus reflects impaired flow or resorption.
- CSF overproduction is an uncommon alternative mechanism.
- A choroid plexus tumor is the textbook example.

Mechanistic classification should not imply equal frequency.

Hydrocephalus ex vacuo is different

Hydrocephalus from altered CSF dynamics

- Excess ventricular CSF can expand the ventricles.
- Pressure may rise as CSF volume increases.

Hydrocephalus ex vacuo

- Loss of brain parenchyma leaves additional intracranial space.
- Ventricular enlargement is compensatory, not evidence by itself of raised pressure.

Interpret ventricular size in relation to parenchymal volume.

Open and closed cranial sutures change the response

Before suture closure

- Head enlargement can accompany hydrocephalus.
- The enlarging skull alters the visible presentation.

After suture closure

- Head circumference does not increase in the same way.
- The ventricles enlarge within a rigid vault; ICP may rise.

Same fluid disorder, different mechanical constraints.

Separate the three hydrocephalus labels

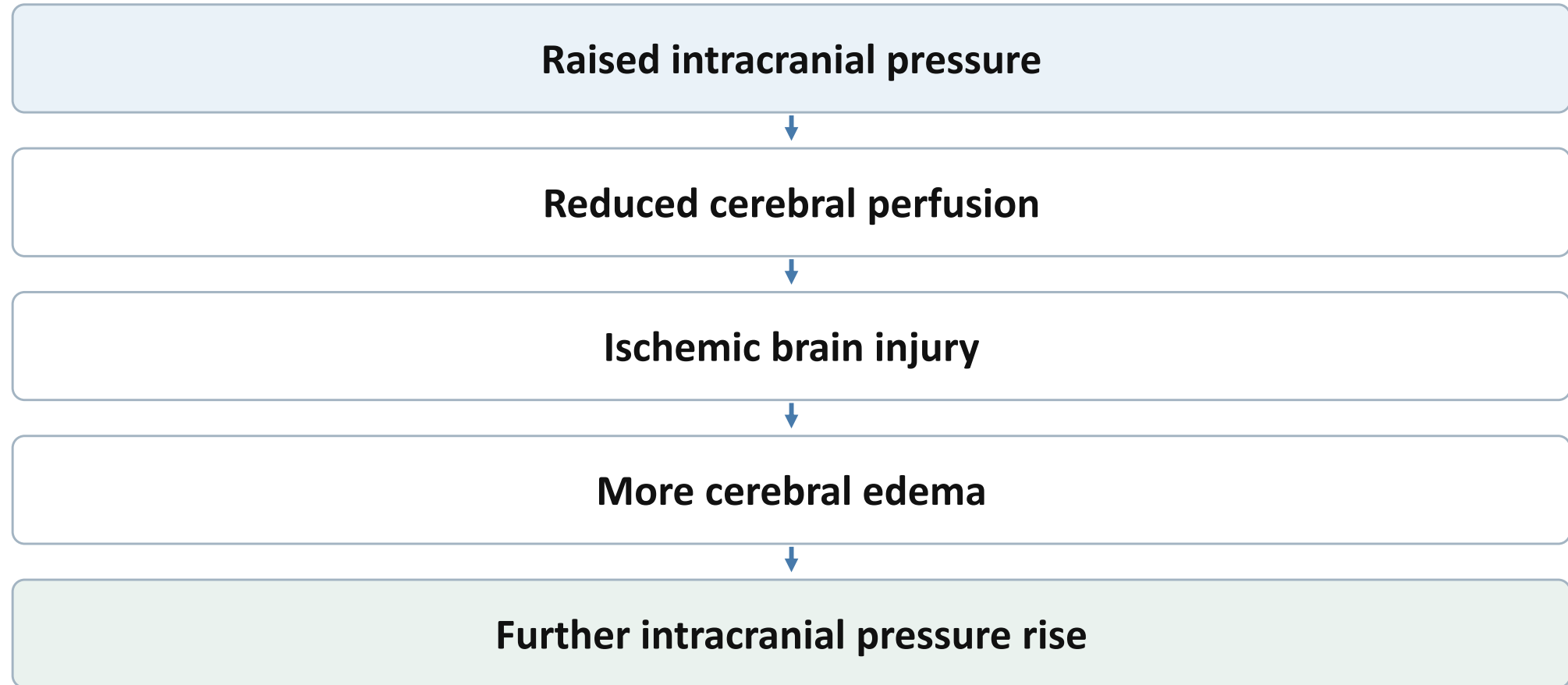
Pattern	Defining feature	Illustrative mechanism
Noncommunicating	Focal block in CSF flow	Aqueductal stenosis or third-ventricular mass
Communicating	Ventricular–subarachnoid continuity preserved	Arachnoid fibrosis after meningitis
Ex vacuo	Ventricular enlargement accompanying tissue loss	Compensatory change after parenchymal loss

Raised pressure can compress cerebral vessels

- Intracranial pressure acts on the cerebral circulation.
- Vascular compression reduces perfusion of brain tissue.
- Less perfusion increases susceptibility to ischemic injury.

High ICP causes harm beyond physical displacement.

The secondary injury loop can reinforce itself



This positive feedback mechanism.

Ischemic injury can generate further edema

- Reduced perfusion deprives tissue of adequate blood supply.
- The resulting ischemic injury can amplify brain swelling.
- Swelling further worsens cranial volume and pressure constraints.

The cause becomes a consequence and then another cause.

Direct compression and secondary ischemia are distinct

Mechanical injury

- Displaced brain directly presses on a nerve or the medulla.
- Produces deficits according to local anatomy.

Vascular injury

- Compressed arteries reduce perfusion to downstream territory.
- Secondary infarction can add deficits beyond the lesion itself.

The same herniation may produce both.

Herniation patterns follow anatomic passages

- Subfalcine: Cingulate gyrus shifts beneath the falx.
- Transtentorial: Mesial temporal structures shift at the tentorial edge.
- Tonsillar: Cerebellar tonsils pass through the foramen magnum.

The boundary crossed identifies the herniation type.

Three principal routes of brain herniation

SUBFALCINE



Under falx



UNCAL



At tentorium



TONSILLAR



Foramen magnum

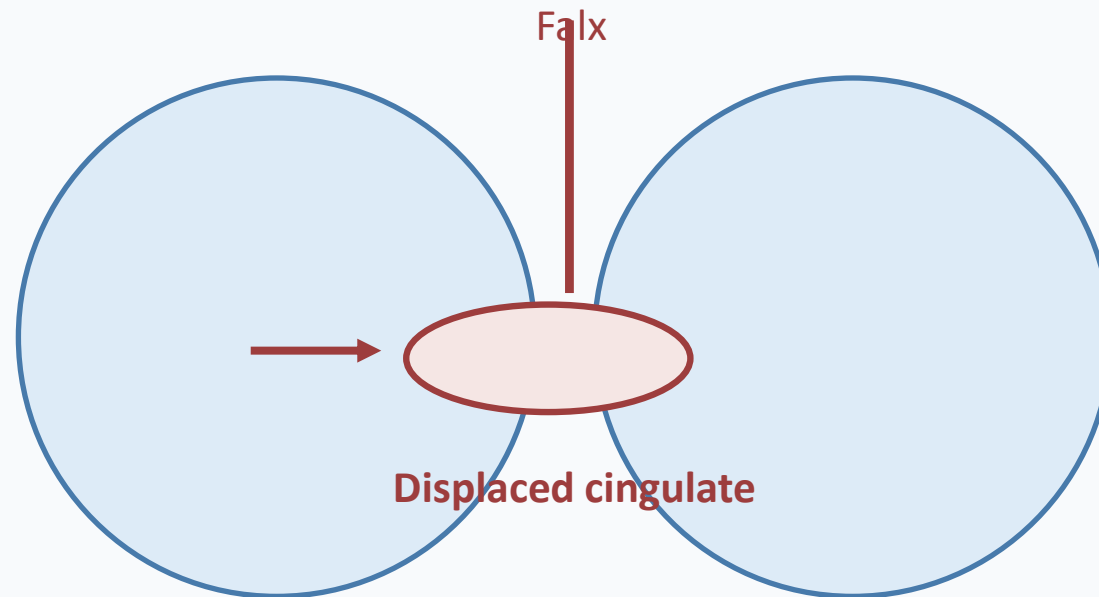


Mechanistic interpretation

- Subfalcine—beneath the falx cerebri.
- Transtentorial—past the free tentorial edge.
- Tonsillar—through the foramen magnum.

Schematic

Subfalcine herniation: Cingulate under the falx



ACA at risk

Mechanistic interpretation

- The cingulate gyrus is displaced below the lower edge of the falx.
- The driving expansion is unilateral or asymmetric.
- The threatened arterial distribution is the anterior cerebral circulation.

Schematic

Asymmetric hemispheric expansion drives subfalcine shift

- A unilateral mass raises pressure on one side.
- The hemisphere displaces toward the midline.
- The cingulate region may pass beneath the falx.

The direction of movement follows the anatomy of the dural partition.

Subfalcine herniation threatens anterior cerebral flow

Cingulate shifts under falx



Anterior cerebral artery or branches compressed



Perfusion of supplied territory decreases



Secondary infarction may occur

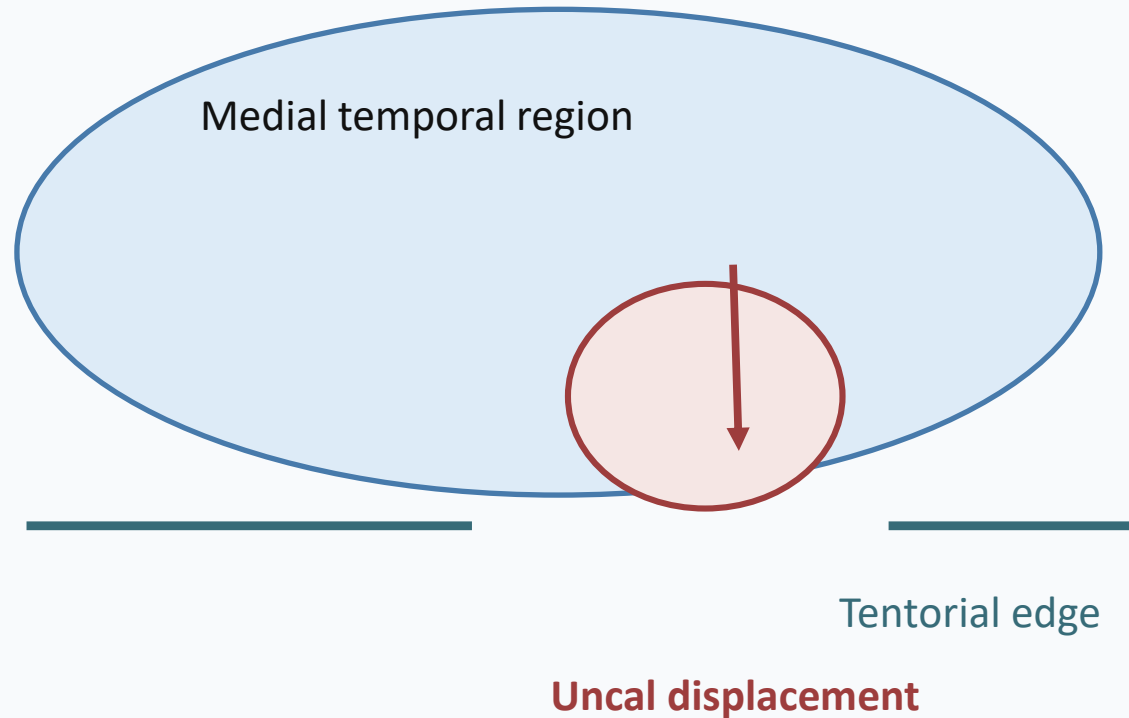
The vascular event is a consequence of herniation.

A secondary infarct can enlarge the lesion's impact

- The initial lesion causes displacement.
- Arterial compression may injure a different downstream territory.
- Clinicopathologic interpretation must account for both primary and secondary damage.

Mass effect can propagate injury beyond its original site.

Uncal herniation occurs at the tentorial edge



Schematic

Mechanistic interpretation

- The medial temporal lobe moves against the free margin of the tentorium.
- Local pressure jeopardizes nearby cranial nerve and vascular structures.
- Increasing shift may distort the midbrain and pons.

The uncus is the relevant mesial temporal structure

- Transtentorial herniation is also termed uncal or mesial temporal herniation.
- The free tentorial margin is the critical rigid boundary.
- Compression effects depend on the amount and direction of displacement.

Identify the temporal lobe–tentorium relationship before inferring a deficit.

Third-nerve compression explains ocular findings

Mesial temporal displacement



Ipsilateral oculomotor nerve (CN III) compromised



Pupillary dilation and impaired eye movements



Signs localize the pressure effect to the affected side

The clinical correlation is explicit.

Pupillary dilation is ipsilateral to an uncal lesion

- The third cranial nerve is compromised on the side of herniation.
- Ipsilateral pupillary dilation is the textbook correlation.
- It may accompany impairment of ipsilateral ocular movements.

Laterality is a key part of the mechanism.

PCA compression produces a visual pathway deficit

Ipsilateral posterior cerebral artery compressed



Its downstream territory undergoes ischemia/infarction



Primary visual cortex may be involved



Contralateral homonymous hemianopia results

The artery is ipsilateral, whereas the visual field loss is contralateral.

Visual loss follows the posterior cerebral territory

- The PCA supplies cortex that includes primary visual areas.
- Arterial compression can produce secondary infarction.
- Contralateral homonymous hemianopia is the described consequence.

This deficit reflects vascular injury, not just cranial nerve pressure.

Kernohan notch: A false-localizing motor sign

Right-sided mass



Brainstem shifts leftward



Left cerebral peduncle compressed



Right-sided weakness

Mechanistic interpretation

- Severe shift compresses the contralateral cerebral peduncle.
- The result may be hemiparesis ipsilateral to the herniation.
- Motor laterality can therefore falsely suggest the side of the primary lesion.

Schematic

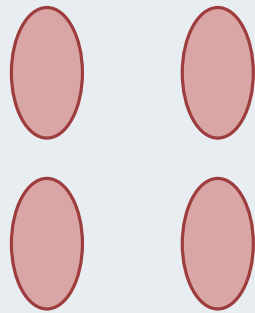
Why the motor sign can seem paradoxical

- A lesion on one side can displace the brainstem toward the opposite tentorial edge.
- Compression involves the cerebral peduncle contralateral to the herniation.
- The resulting weakness may appear on the lesion's side.

This is the Kernohan notch phenomenon.

Duret hemorrhages occur within the upper brainstem

Brainstem schematic



Midline / paramedian
Hemorrhages

Schematic

Mechanistic interpretation

- Linear or flame-shaped hemorrhages affect the midbrain and pons.
- They occur characteristically in midline and paramedian regions.
- They accompany progression of severe transtentorial herniation.

Duret hemorrhages reflect vascular distortion

Downward brain displacement



Stretching or tearing of penetrating brainstem vessels



Hemorrhage in midbrain and pons



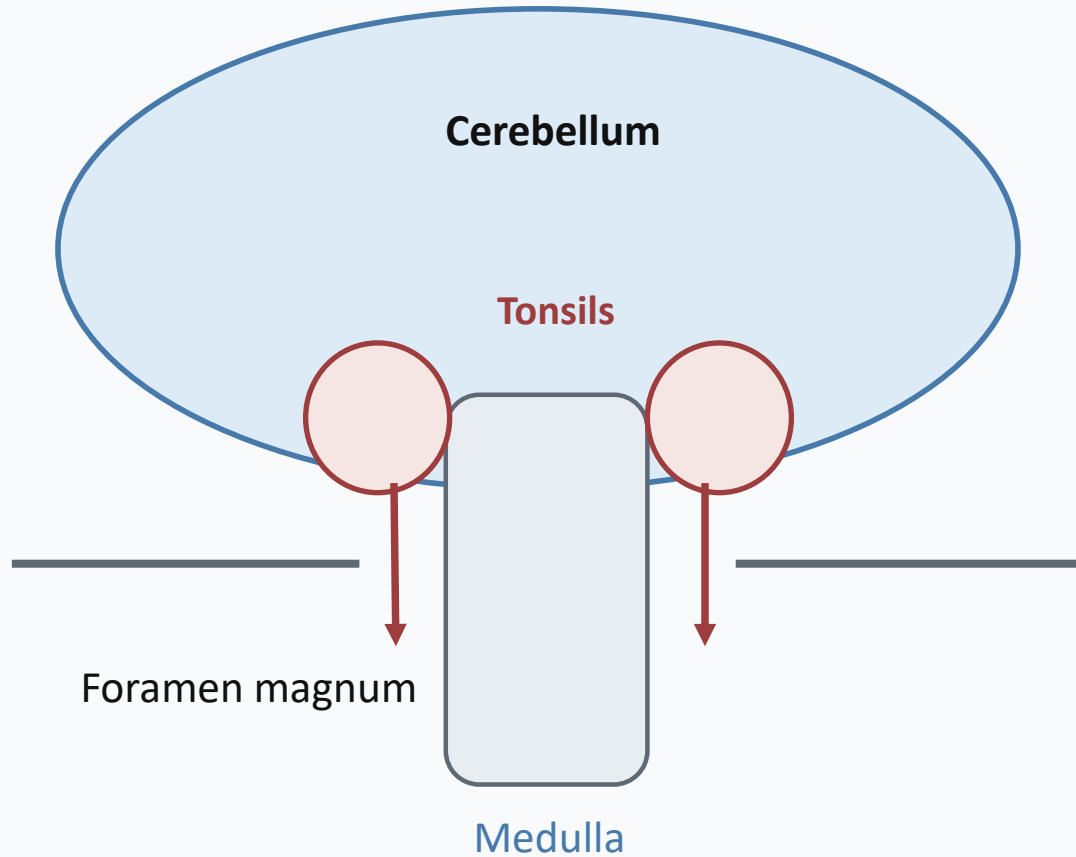
Secondary destructive brainstem injury

The proposed mechanism involves arteries and veins entering the upper brainstem.

Uncal herniation can affect several structures

Compressed structure	Mechanism	Characteristic consequence
Cranial nerve III	Local nerve compromise	Ipsilateral dilated pupil and impaired eye movements
Posterior cerebral artery	Vascular compression	Contralateral homonymous hemianopia
Contralateral cerebral peduncle	Kernohan notch	Ipsilateral hemiparesis
Upper brainstem perforators	Distortion or tearing	Duret hemorrhages

Tonsillar herniation passes through the foramen magnum



Schematic

Mechanistic interpretation

- Cerebellar tonsils are displaced inferiorly.
- They pass through the skull-base opening around the medulla.
- Compression of the lower brainstem creates the major hazard.

The medulla contains vital cardiorespiratory centers

- Inferior tonsillar displacement compresses the brainstem.
- Vital respiratory and cardiac control centers are threatened.
- This makes tonsillar herniation life-threatening.

The risk is fundamentally different from an isolated focal cortical deficit.

Several herniation patterns may coexist

- Severe brain expansion may cross more than one anatomic boundary.
- The observed deficits can reflect multiple simultaneous compression sites.
- A single presenting lesion need not produce only one pattern.

Recognizing one pattern does not exclude another.

Herniation patterns: Morphology to consequence

Pattern	Displaced tissue / opening	Critical complication
Subfalcine	Cingulate beneath falx	ACA compression and secondary infarct
Transtentorial	Medial temporal lobe at tentorial margin	CN III, PCA, peduncle, Duret hemorrhages
Tonsillar	Cerebellar tonsils via foramen magnum	Medullary compromise of vital centers

Clinical application: Asymmetric cerebral mass

An expanding mass occupies the right cerebral hemisphere. The cingulate gyrus is driven beneath the falx.

Which artery is at risk, and what kind of secondary tissue injury can follow?

Case interpretation: Subfalcine mechanism

Anterior cerebral artery compression

- The displaced cingulate lies near the falx and ACA circulation.
- Arterial compromise may produce a downstream cerebral infarct.
- The infarct is secondary to the mass effect rather than the original mass.

Clinical application: Ipsilateral dilated pupil

A patient with major right mesial temporal displacement develops a dilated right pupil and impaired right ocular movements.

Identify the compressed structure and herniation type.

Case interpretation: Uncal cranial-nerve effect

Right oculomotor nerve (CN III)

- The right medial temporal lobe impinges on the right tentorial edge.
- Third-nerve compromise explains ipsilateral pupillary dilation.
- Impaired eye movements accompany the same local injury.

Clinical application: A misleading motor deficit

A severe right-sided mass shifts the midbrain so that the left cerebral peduncle is compressed. Right-sided weakness develops.

Why is the weakness on the same side as the original mass?

Case interpretation: Kernohan notch

Compression of the contralateral peduncle

- The left cerebral peduncle is compressed by displacement.
- The resulting right hemiparesis is ipsilateral to the primary right-sided lesion.
- This false-localizing pattern is Kernohan notch phenomenon.

Clinical application: Threat to vital function

A patient with profound posterior fossa mass effect shows inferior displacement of the cerebellar tonsils through the foramen magnum.

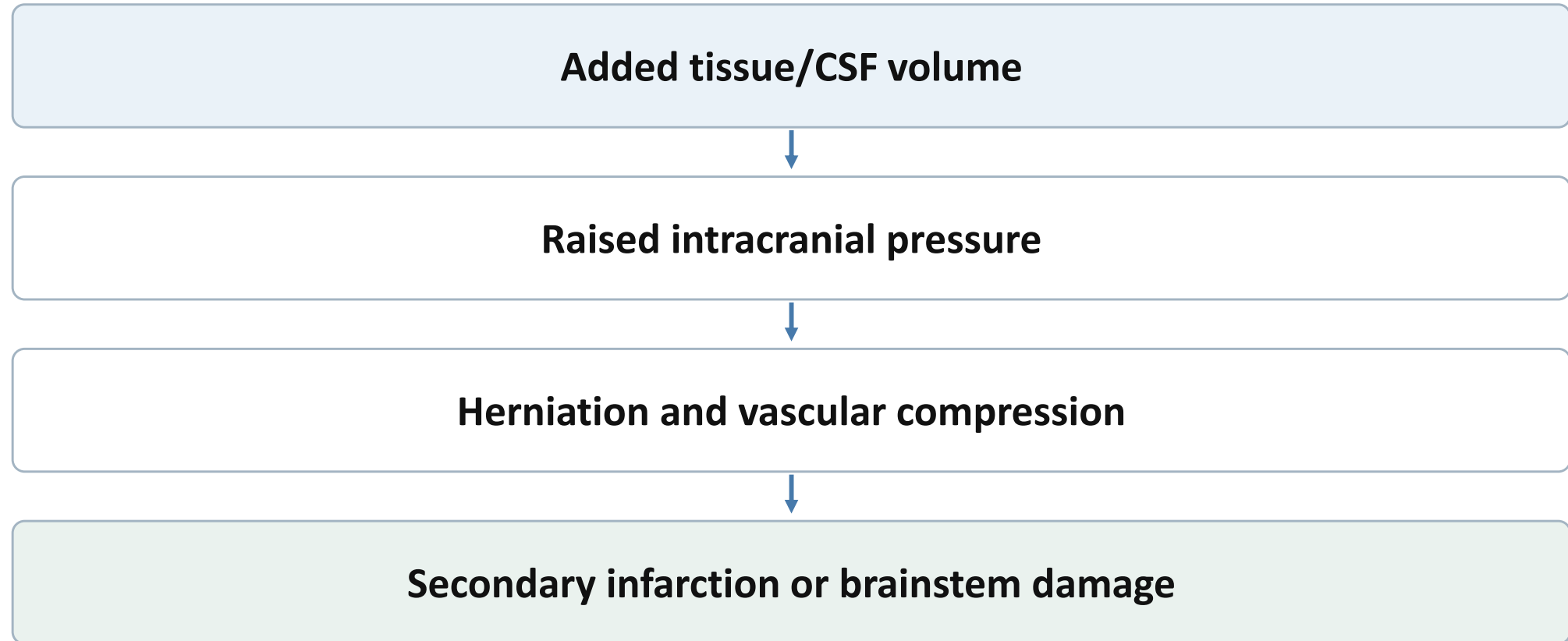
Which region is endangered, and why is deterioration potentially fatal?

Case interpretation: Tonsillar compression

Medulla and vital cardiorespiratory centers

- The tonsils move inferiorly through the skull-base opening.
- The compressed medulla contains centers essential for respiratory and cardiac function.
- The mechanism is direct brainstem compromise.

Integrated injury model



The model summarizes the causal relationships emphasized in the source.

Essential clinicopathologic synthesis

- Identify the expanded compartment: Parenchyma, ventricular CSF, or focal mass.
- Determine the direction of displacement relative to falx, tentorium, and foramen magnum.
- Predict vascular, cranial-nerve, peduncular, or medullary consequences.
- Remember that ischemia may amplify edema and the pressure burden.

Anatomic localization converts morphology into clinical reasoning.

Primary reference

Robbins, Cotran & Kumar. Pathologic Basis of Disease. 11th ed. Chapter 28, The Central Nervous System.

Conceptual diagrams in this lecture are original editable schematics, not diagnostic specimens.