

Cerebral Edema, Hydrocephalus, Raised Intracranial Pressure, and Herniation

An integrated clinicopathologic lecture

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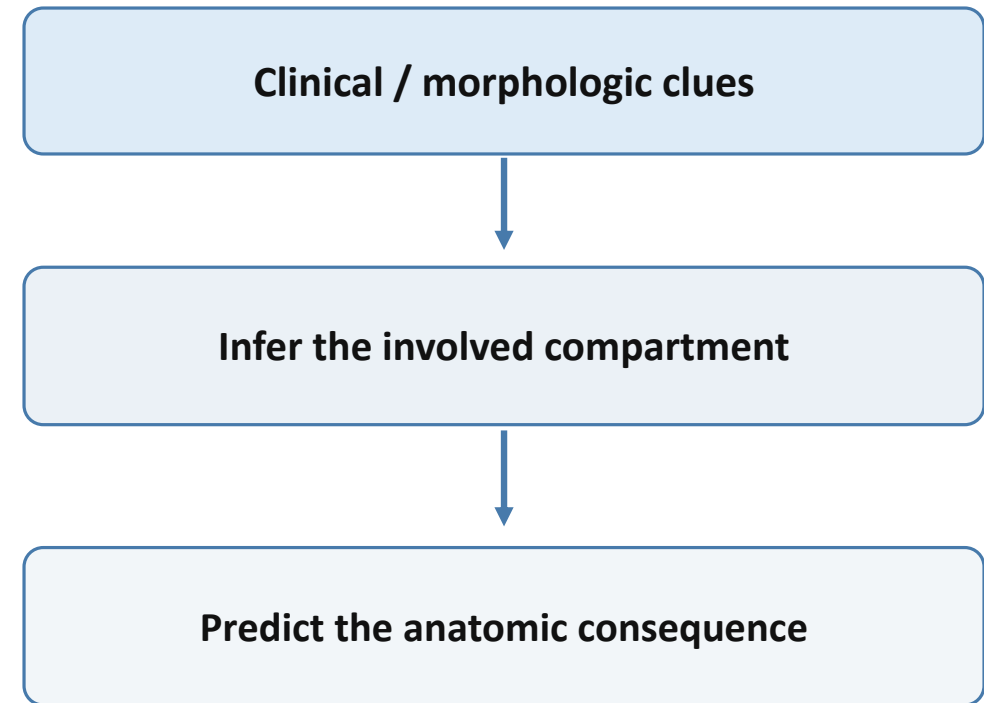
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Learning objectives: Four linked neuropathologic processes

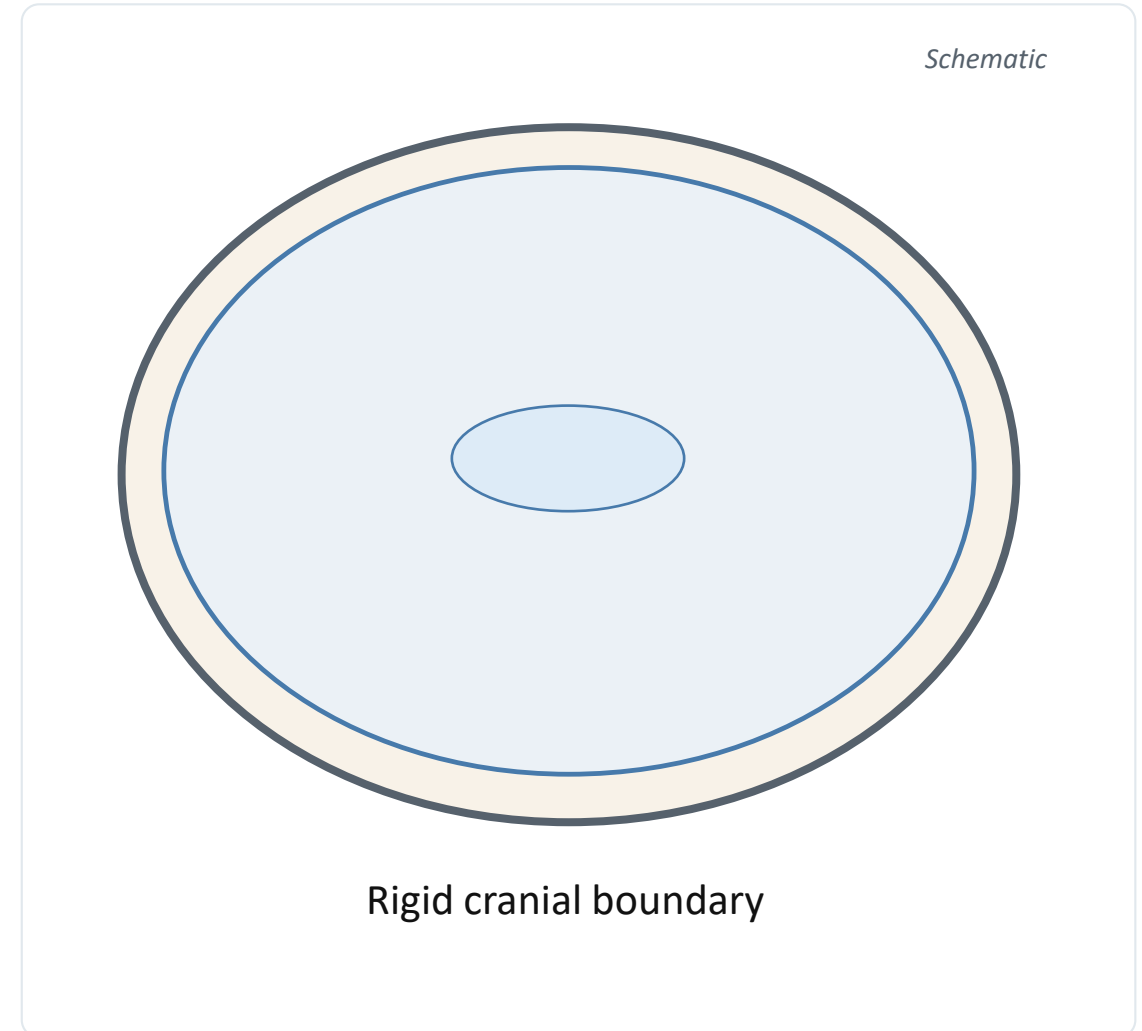
- Distinguish edema in parenchyma from excessive CSF in ventricles.
- Explain how additional intracranial volume produces pressure and ischemic injury.
- Classify major hydrocephalus patterns by CSF flow and resorption.
- Localize subfalcine, transtentorial, and tonsillar brain herniation.

Schematic



Rigid enclosure makes small expansions consequential

- The brain is enclosed by skull and dural reflections.
- Expansion cannot be relieved by skull enlargement after suture closure.
- Consequences depend on both speed and magnitude of pressure rise.



Three common settings raise intracranial pressure

- Generalized brain edema expands the parenchymal compartment.
- Increased cerebrospinal fluid volume expands the ventricular compartment.
- Focal tumor, abscess, or hemorrhage can act as a space-occupying lesion.

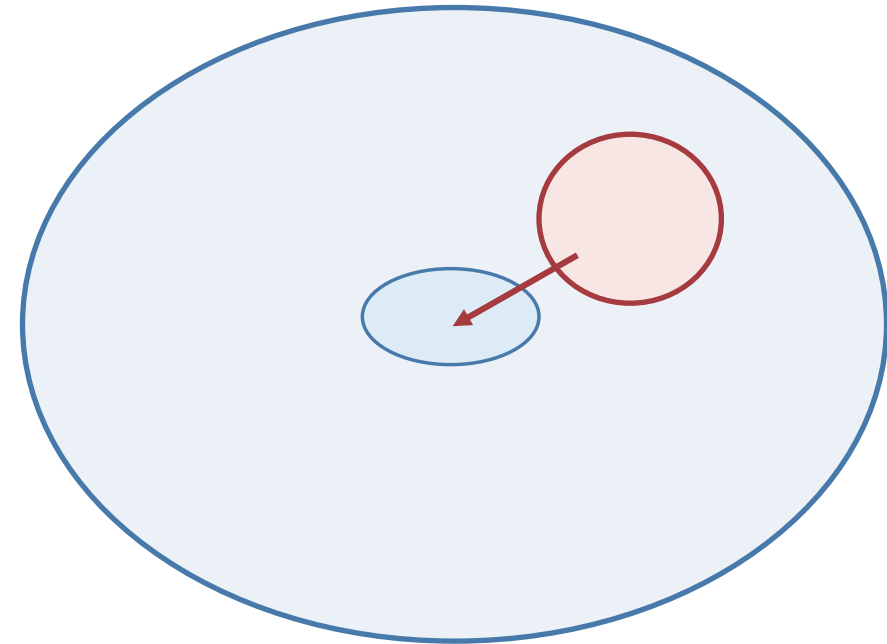
Schematic



Intracranial lesions may be diffuse or focal

- Diffuse processes enlarge the brain globally.
- A focal process produces regional mass effect and tissue displacement.
- Either pattern may progress to pressure-related vascular compromise.

Schematic

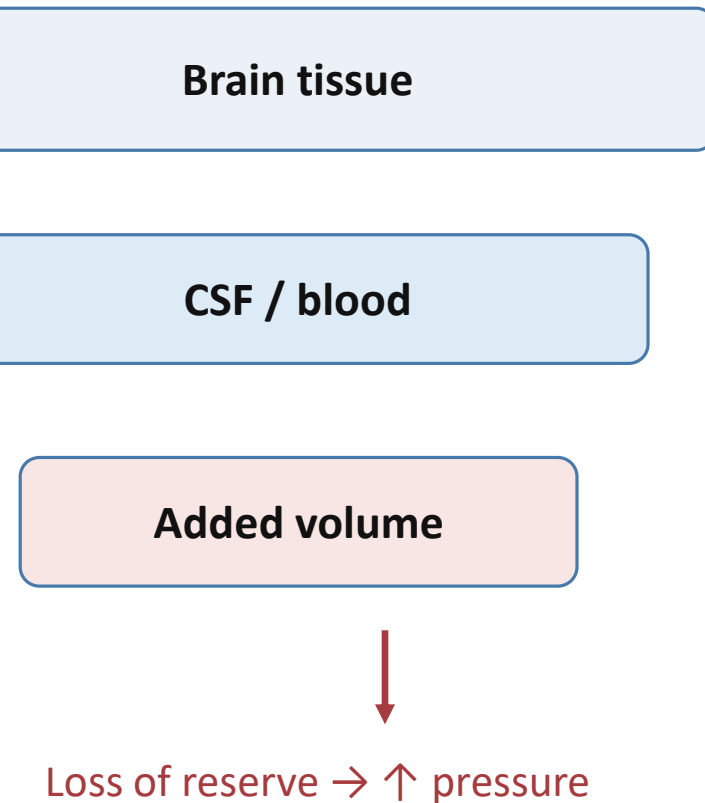


Expanding focal mass

The rate of intracranial expansion changes the outcome

- Brain volume can initially be accommodated by displacement of CSF.
- Further expansion eventually exceeds limited compensatory capacity.
- Rapid or severe pressure increases can produce catastrophic displacement.

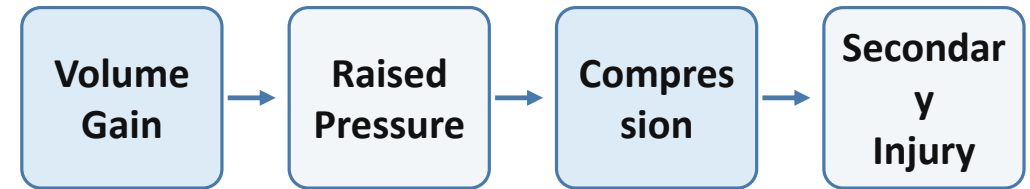
Schematic



A central sequence connects all four topics

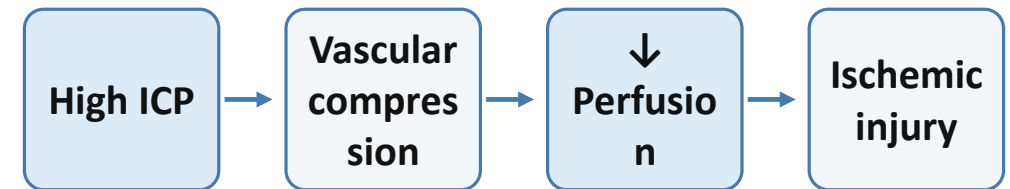
- Fluid or mass increases intracranial volume.
- Pressure rises as compensation becomes insufficient.
- Reduced perfusion and herniation amplify tissue injury.

Schematic



Mass effect creates a feedback loop of injury

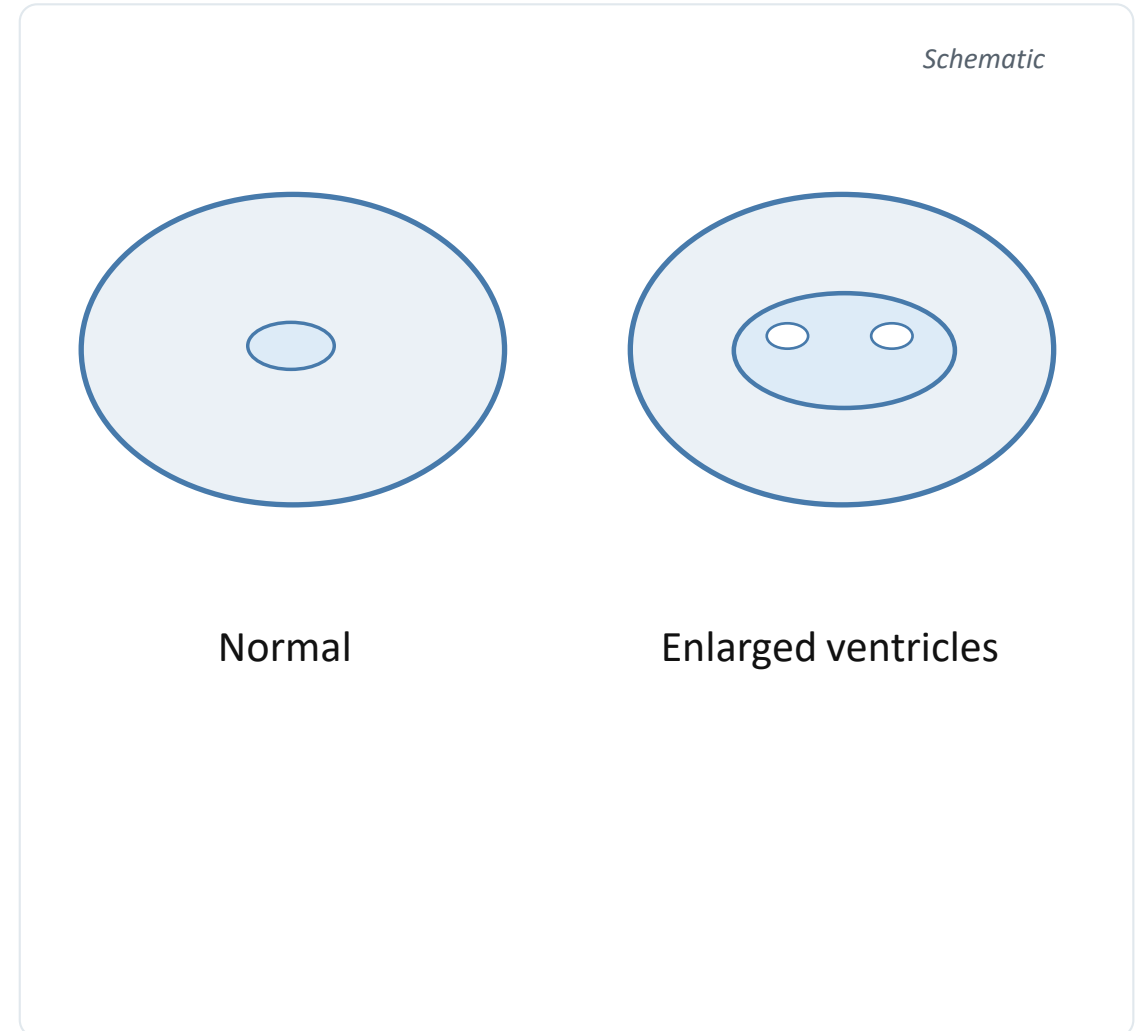
- Elevated intracranial pressure compresses cerebral vessels.
- Reduced cerebral perfusion promotes ischemic injury.
- Ischemia can aggravate edema, further intensifying mass effect.



Edema may intensify the cycle

First distinction: Parenchyma or ventricular CSF?

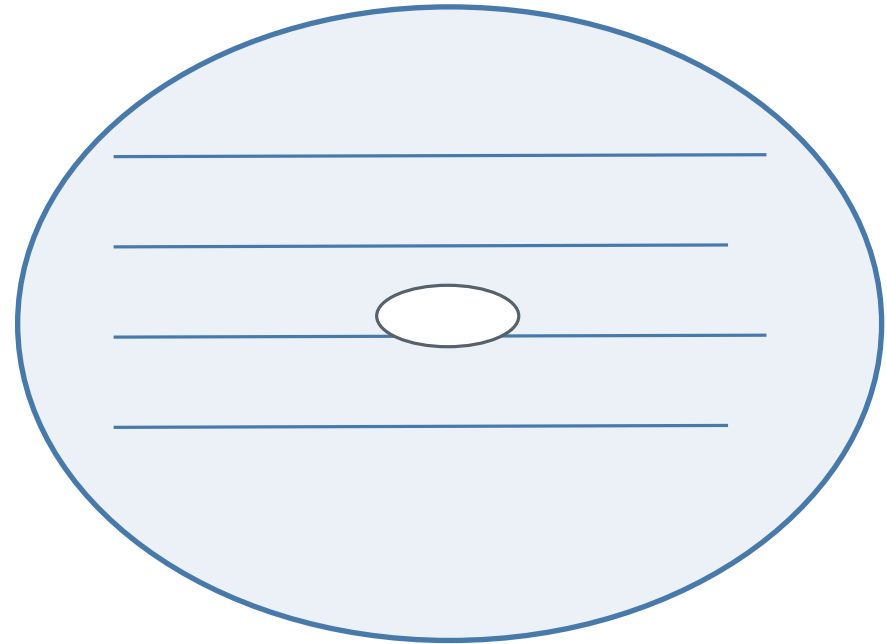
- Cerebral edema increases fluid within brain tissue.
- Hydrocephalus increases CSF in all or part of the ventricular system.
- Either may raise intracranial pressure through volume expansion.



Cerebral edema is excess fluid in brain parenchyma

- It arises from increased vascular fluid leakage and/or CNS cell injury.
- The resulting brain swelling is a morphologic as well as mechanical process.
- Generalized swelling may compress ventricles and predispose to herniation.

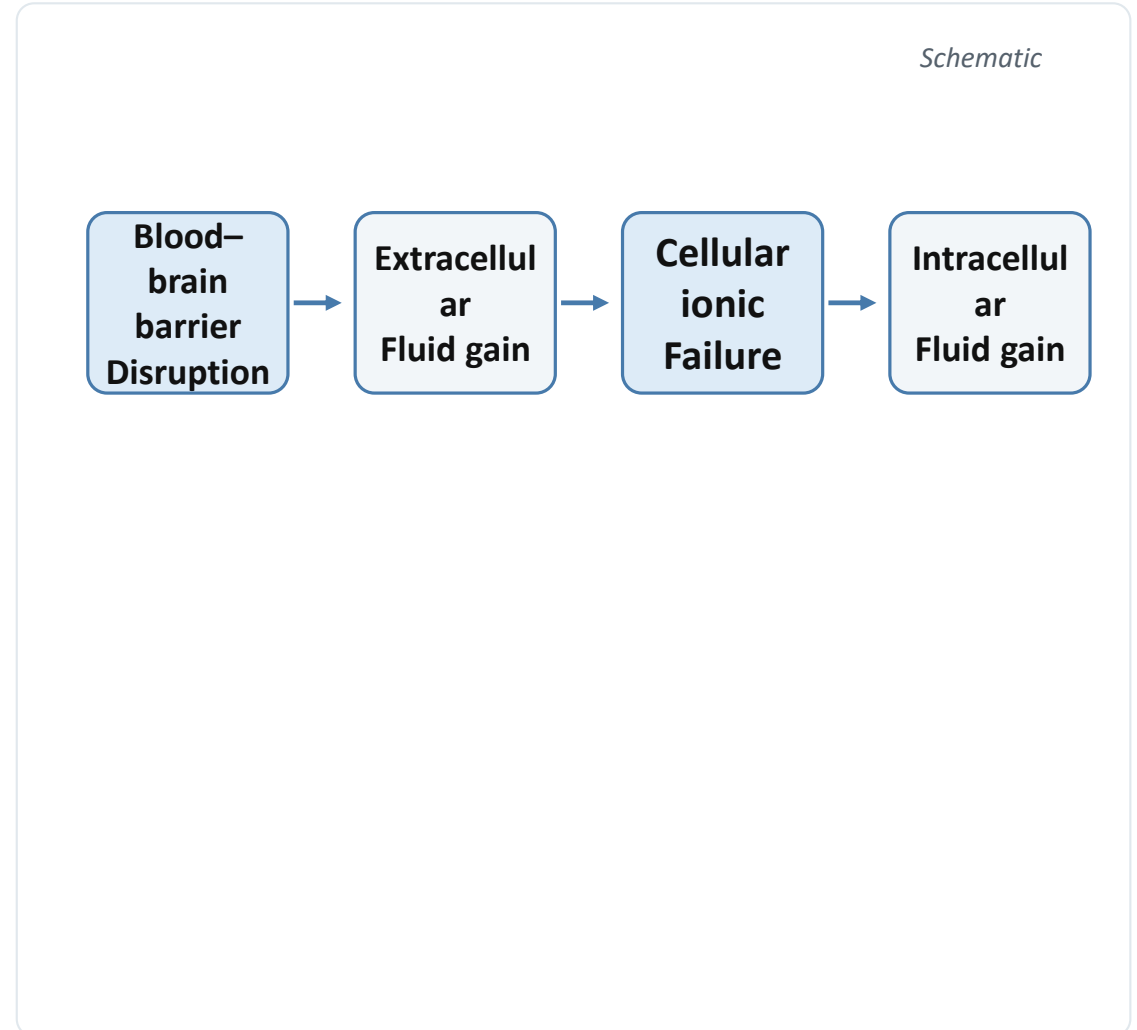
Schematic



Flattened gyri · narrowed sulci

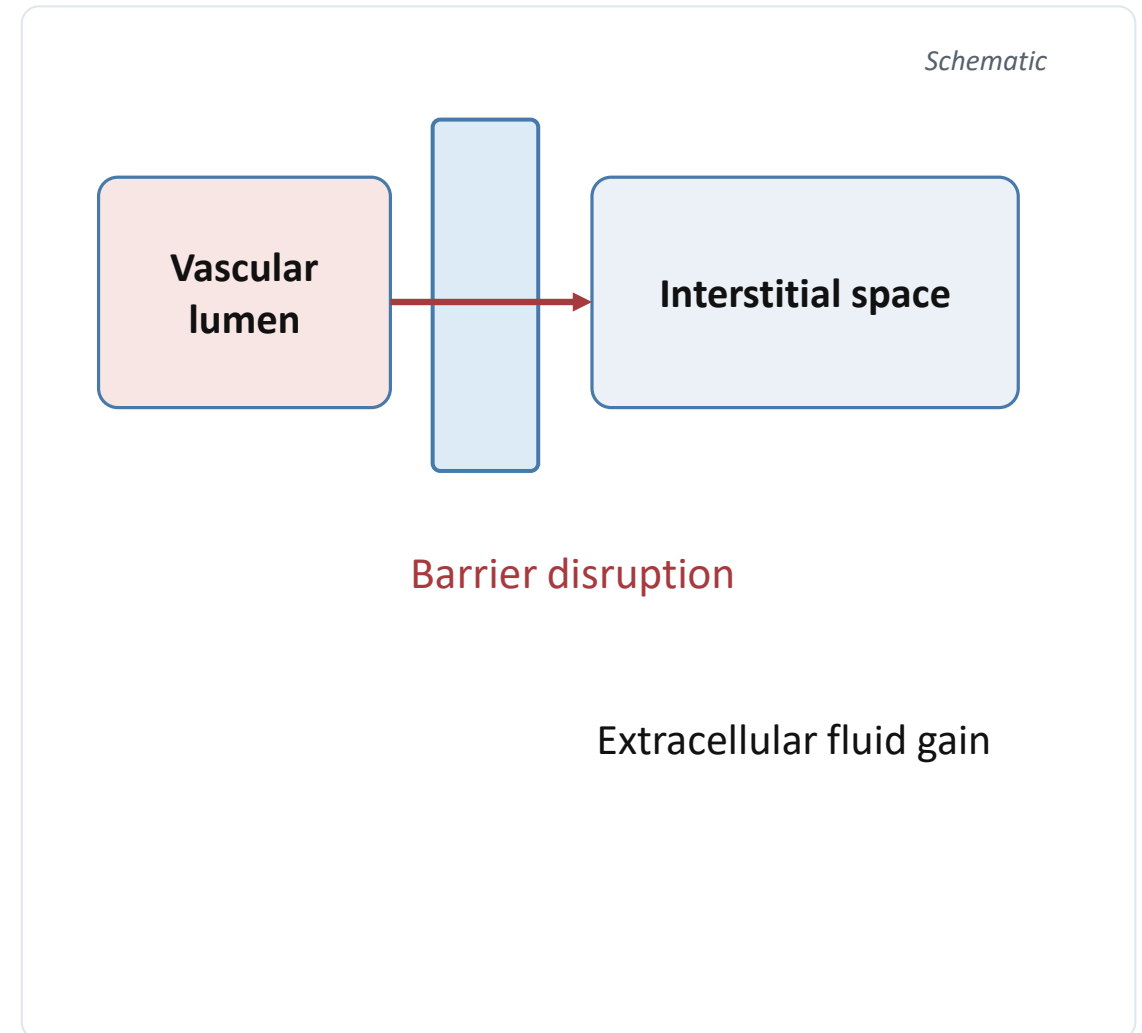
Two principal pathways generate cerebral edema

- Vasogenic edema adds fluid to extracellular tissue spaces.
- Cytotoxic edema adds fluid to the interiors of injured cells.
- Generalized edema often includes both mechanisms.



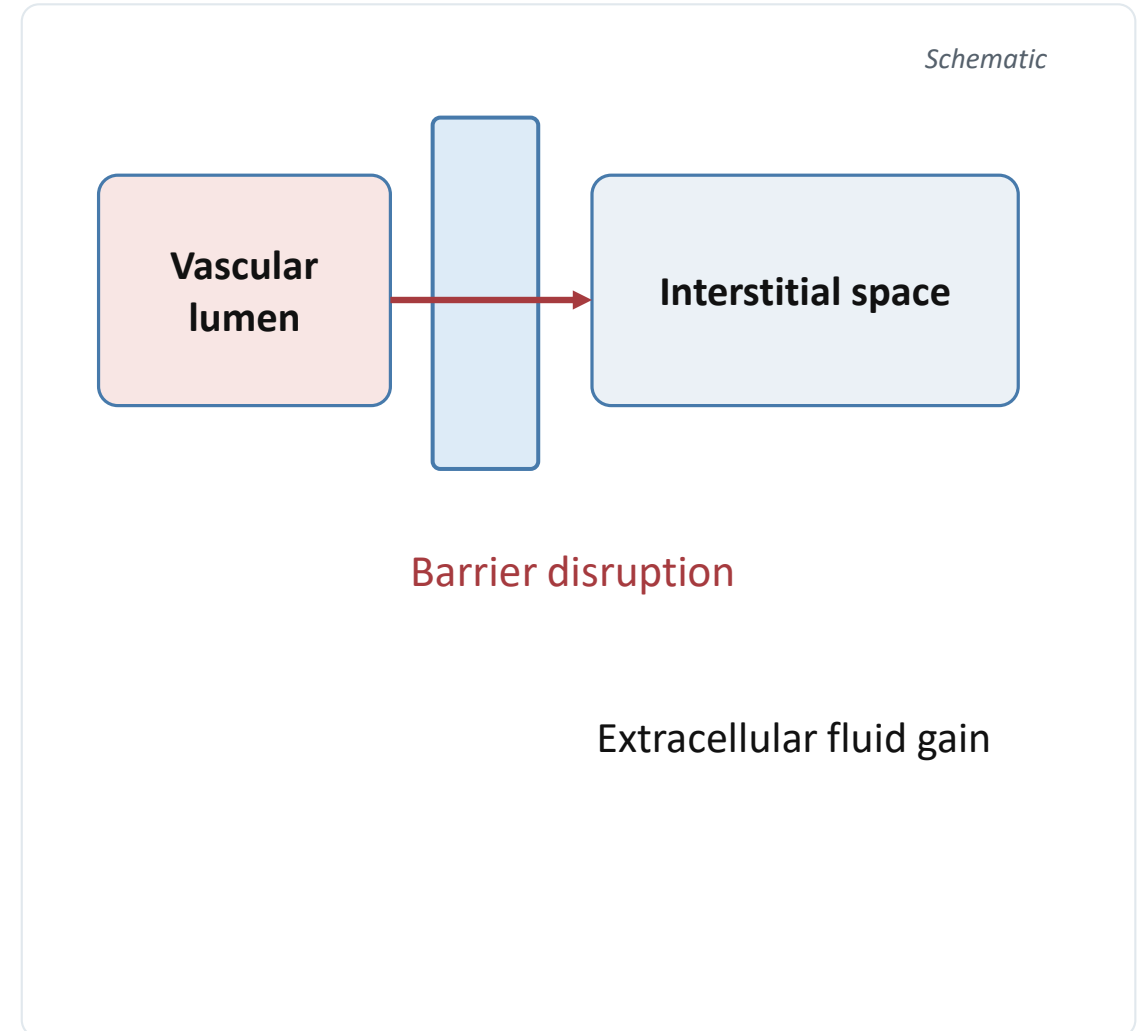
Vasogenic edema requires blood–brain barrier disruption

- Vascular permeability increases when the barrier is disturbed.
- Fluid leaves the intravascular compartment.
- Extracellular, intercellular fluid accumulates within brain parenchyma.



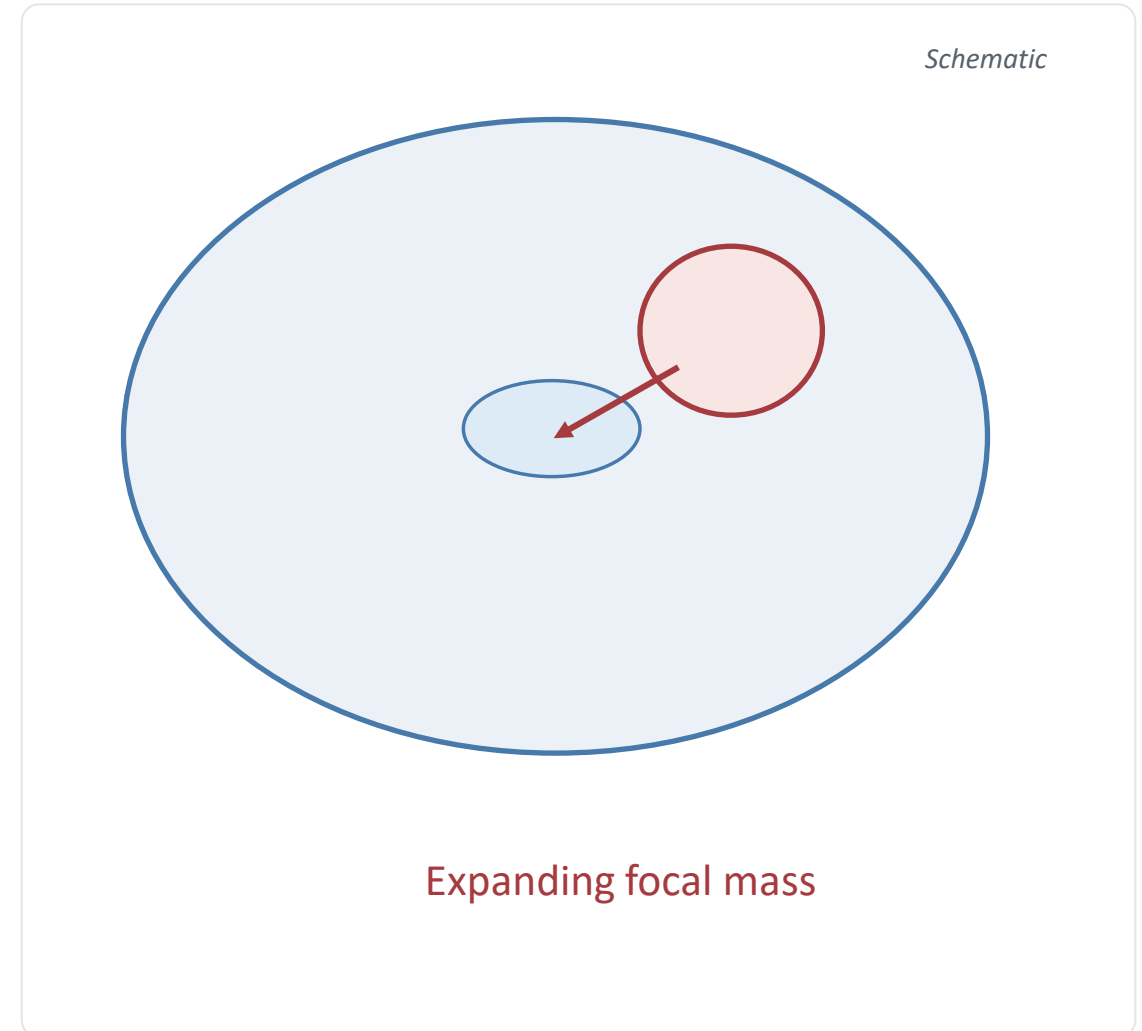
Vasogenic edema is extracellular in distribution

- The dominant expansion is between the cells, not initially within them.
- Its immediate source is leakage from permeable blood vessels.
- The location of the inciting lesion influences edema distribution.



Localized vasogenic edema surrounds focal insults

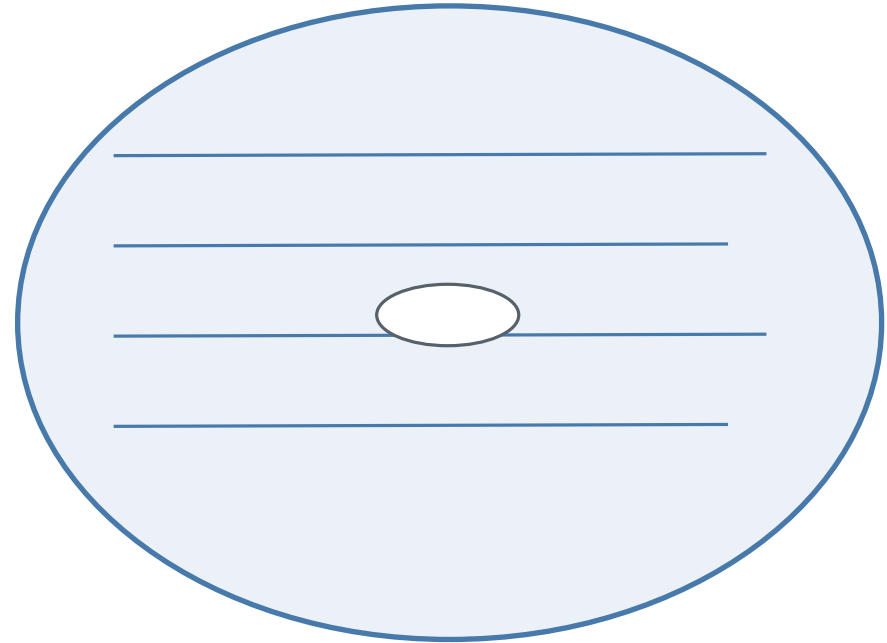
- Inflammation can disrupt local blood–brain barrier integrity.
- Neoplasms may increase permeability of adjacent vessels.
- Localized edema can intensify the mass effect of the underlying lesion.



Vasogenic edema can also be generalized

- The source describes generalized vasogenic edema after global ischemic injury.
- Extensive barrier dysfunction permits widespread extracellular fluid accumulation.
- Generalized swelling threatens intracranial volume reserve.

Schematic

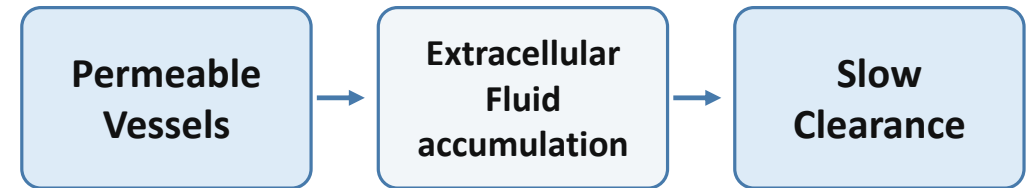


Flattened gyri · narrowed sulci

Brain interstitial fluid clears relatively slowly

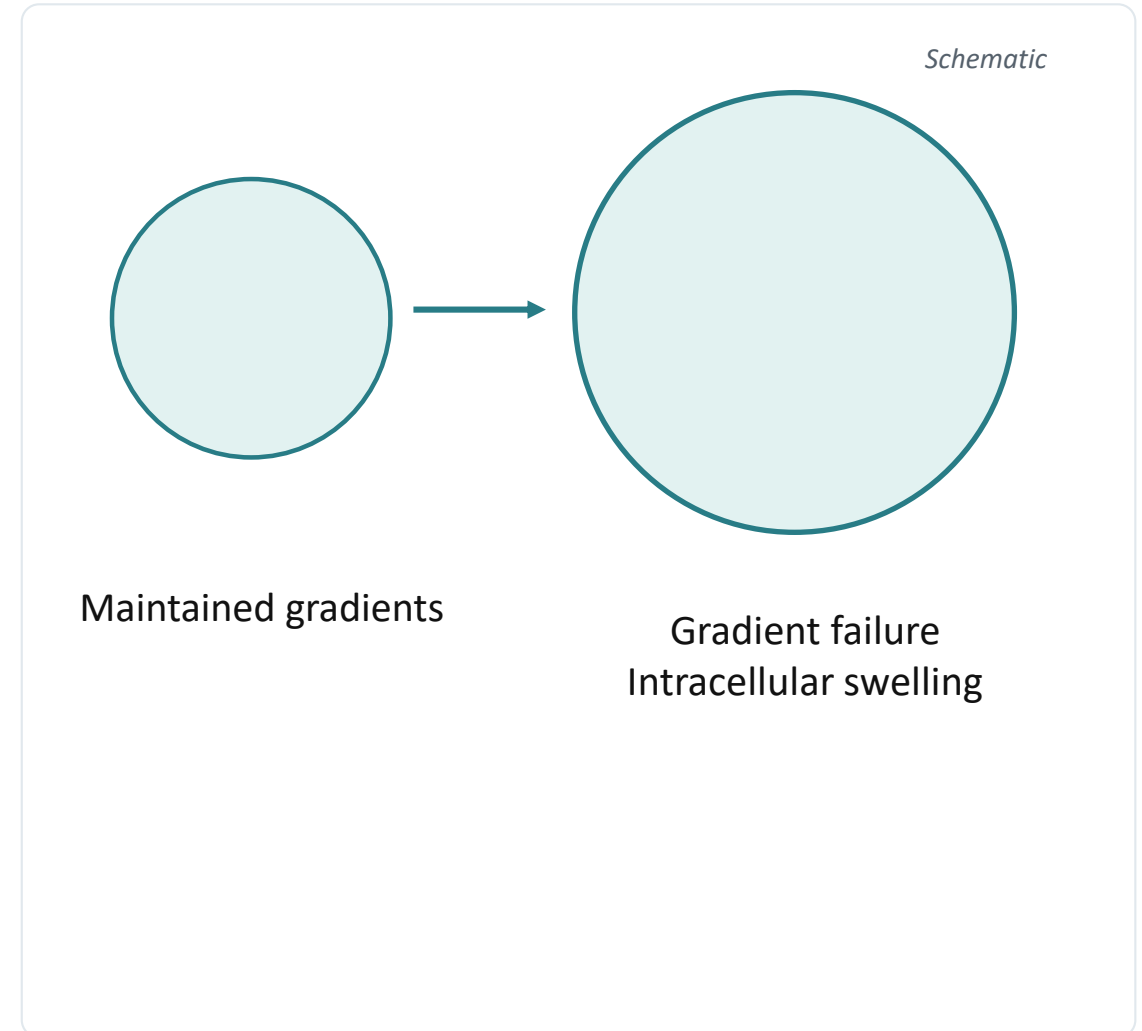
- The brain has relatively few conventional lymphatic vessels.
- This paucity slows resorption of excess extracellular fluid.
- Fluid removal can therefore lag behind ongoing vascular leakage.

Schematic



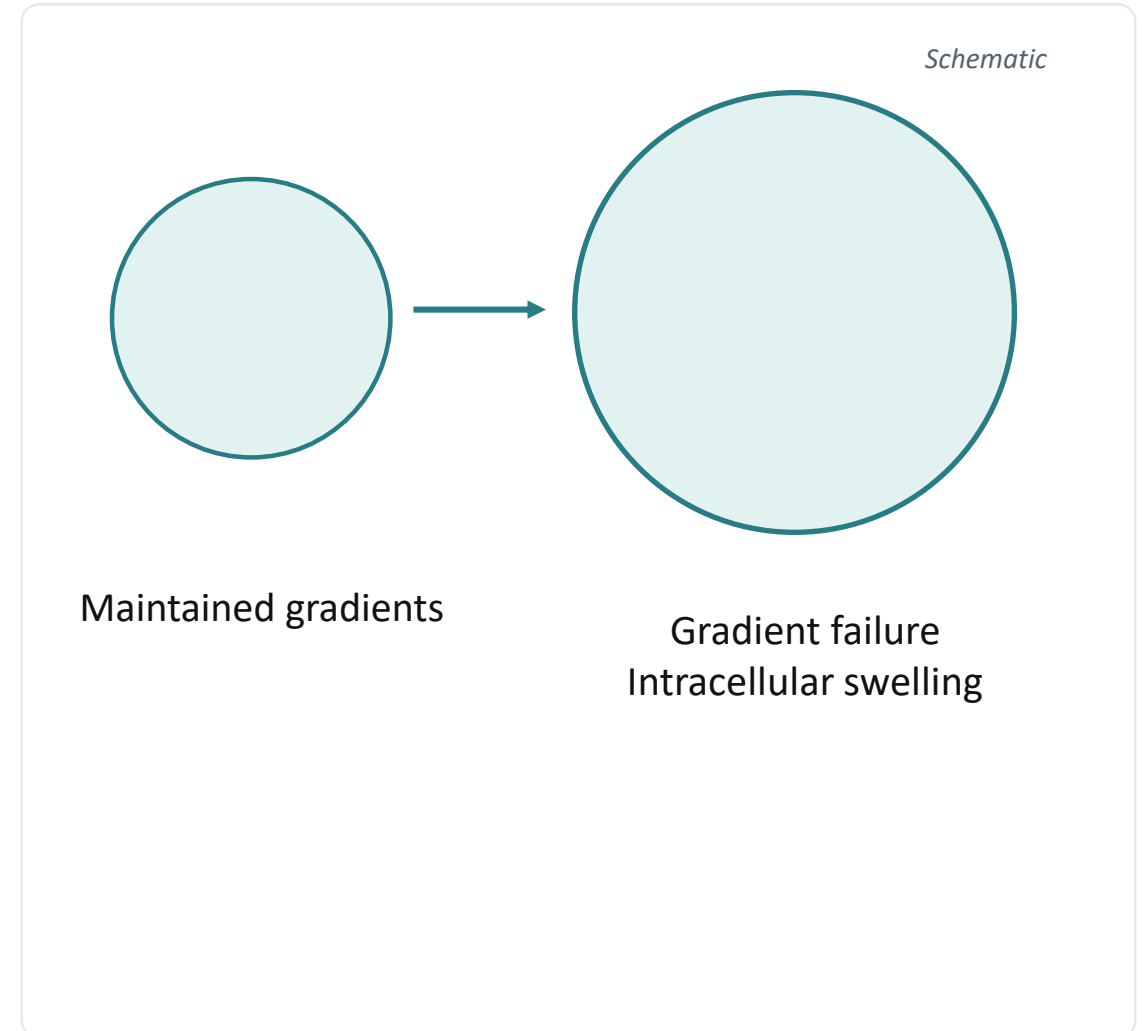
Cytotoxic edema begins with injury to cell membranes

- Neuron, glial, and endothelial cell membranes may be affected.
- Failure of normal ionic gradients permits intracellular fluid gain.
- Cells swell even without a primary permeability-driven leak.



Why loss of ionic gradients causes cell swelling

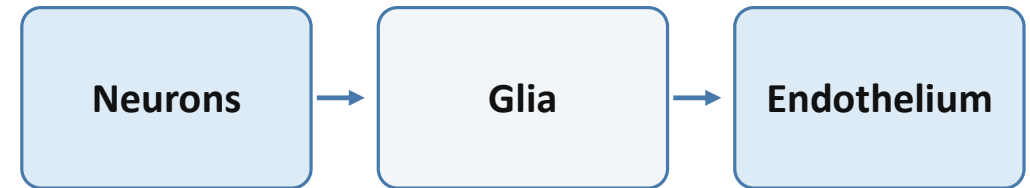
- Viable cells normally sustain transmembrane ionic differences.
- Hypoxic, ischemic, or metabolic disturbances can prevent their maintenance.
- The resulting intracellular water accumulation is cytotoxic edema.



Cytotoxic edema is not restricted to neurons

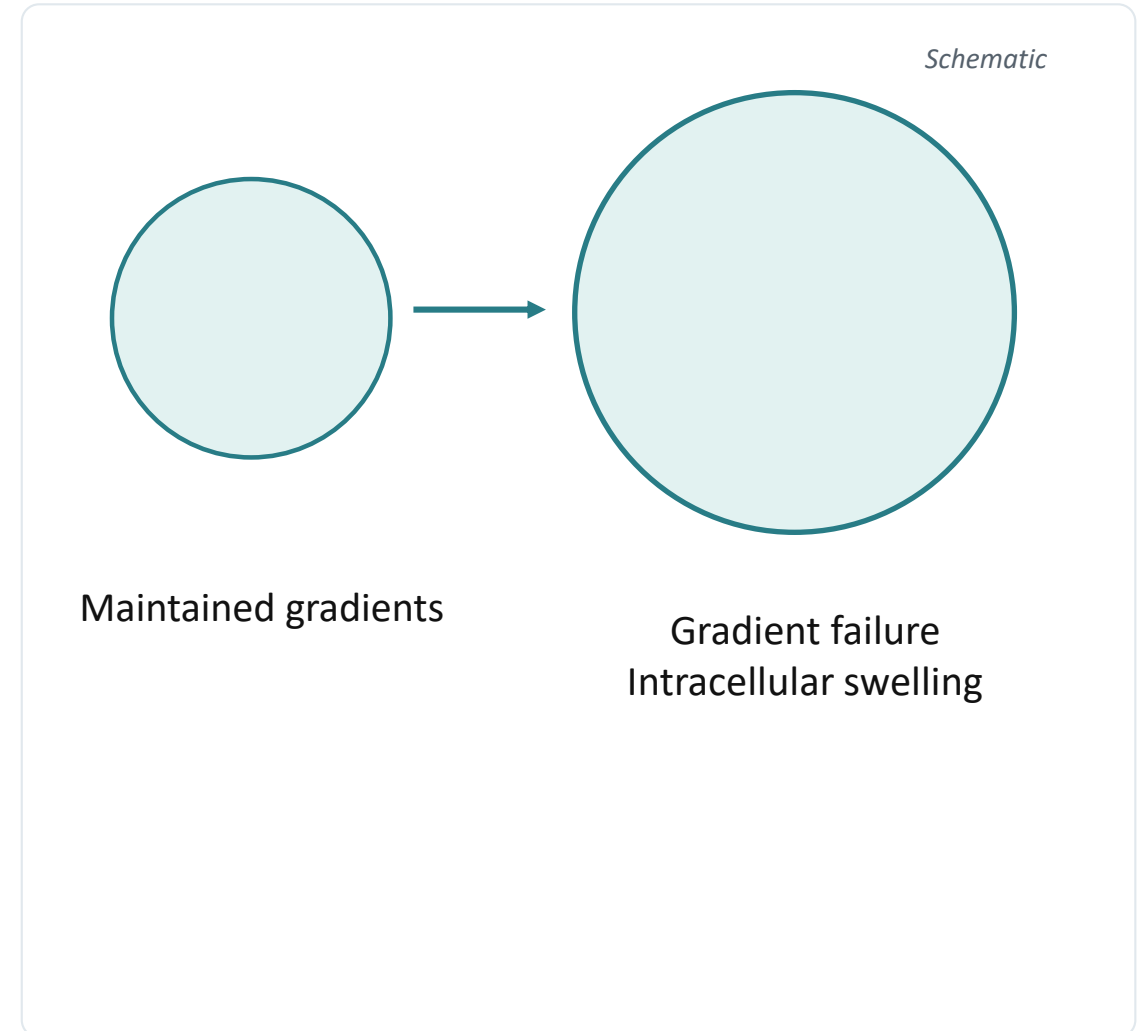
- Neurons may swell as membranes lose ionic regulation.
- Glial cells are also susceptible to intracellular fluid accumulation.
- Cerebral endothelial injury can contribute to the same mechanism.

Schematic



Ischemic and metabolic insults produce cytotoxic edema

- Generalized hypoxic/ischemic injury is a characteristic setting.
- Metabolic derangements that prevent normal gradients have similar consequences.
- The defining lesion is cell swelling from impaired membrane function.



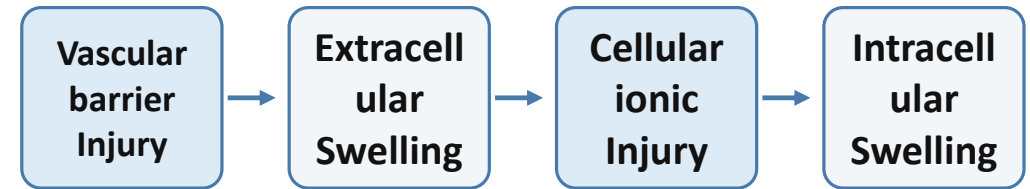
Vasogenic and cytotoxic edema differ by compartment

Feature	Vasogenic edema	Cytotoxic edema
Primary site	Extracellular spaces	Intracellular spaces
Proximal problem	Barrier permeability rises	Cell ionic gradients fail
Typical association	Inflammation or neoplasm	Hypoxia/ischemia or metabolic insult
Source of volume	Vascular leakage	Cellular water accumulation

Generalized edema frequently mixes both mechanisms

- Widespread CNS injury may disrupt vessels and cellular metabolism together.
- Extracellular and intracellular water accumulation can coexist.
- A single disease need not produce only one edema category.

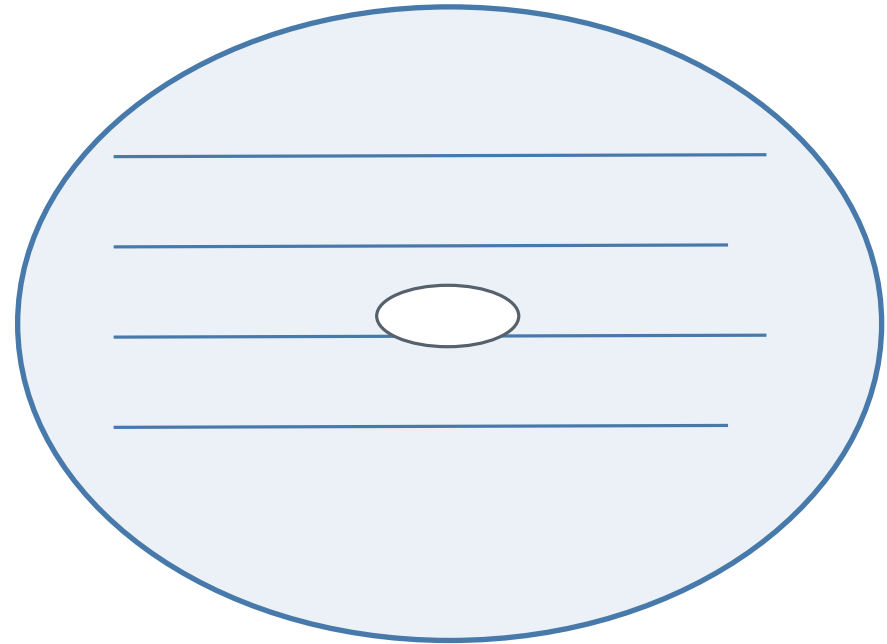
Schematic



Edematous gyri are flattened by the cranial boundary

- Swelling enlarges the brain within the rigid cranial cavity.
- The inner skull and dura compress the expanding cerebral surface.
- Consequently, gyri become flattened on gross examination.

Schematic

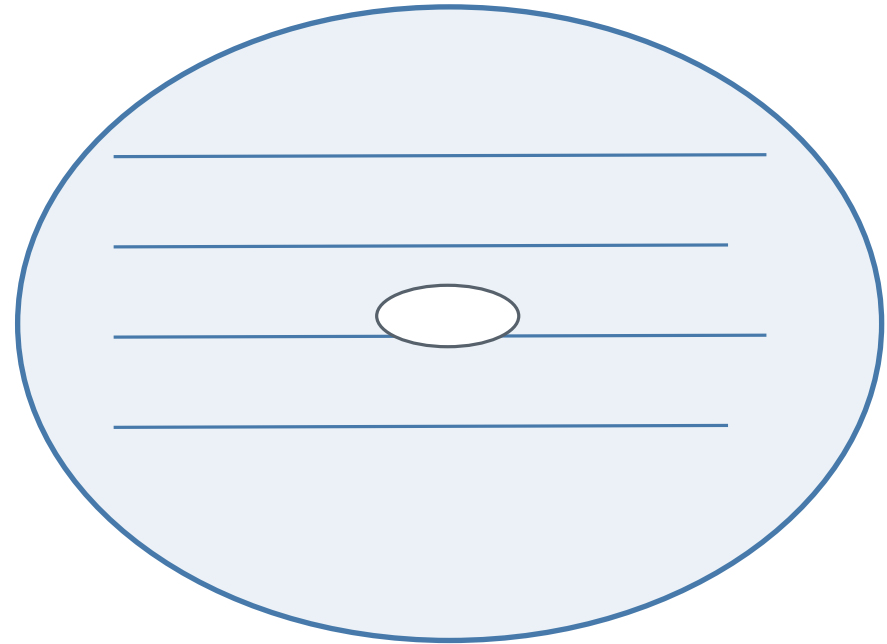


Flattened gyri · narrowed sulci

Generalized swelling narrows cerebral sulci

- The expanding gyri occupy their intervening grooves.
- The sulcal spaces become visibly reduced.
- Flattened gyri and narrowed sulci are complementary signs of swelling.

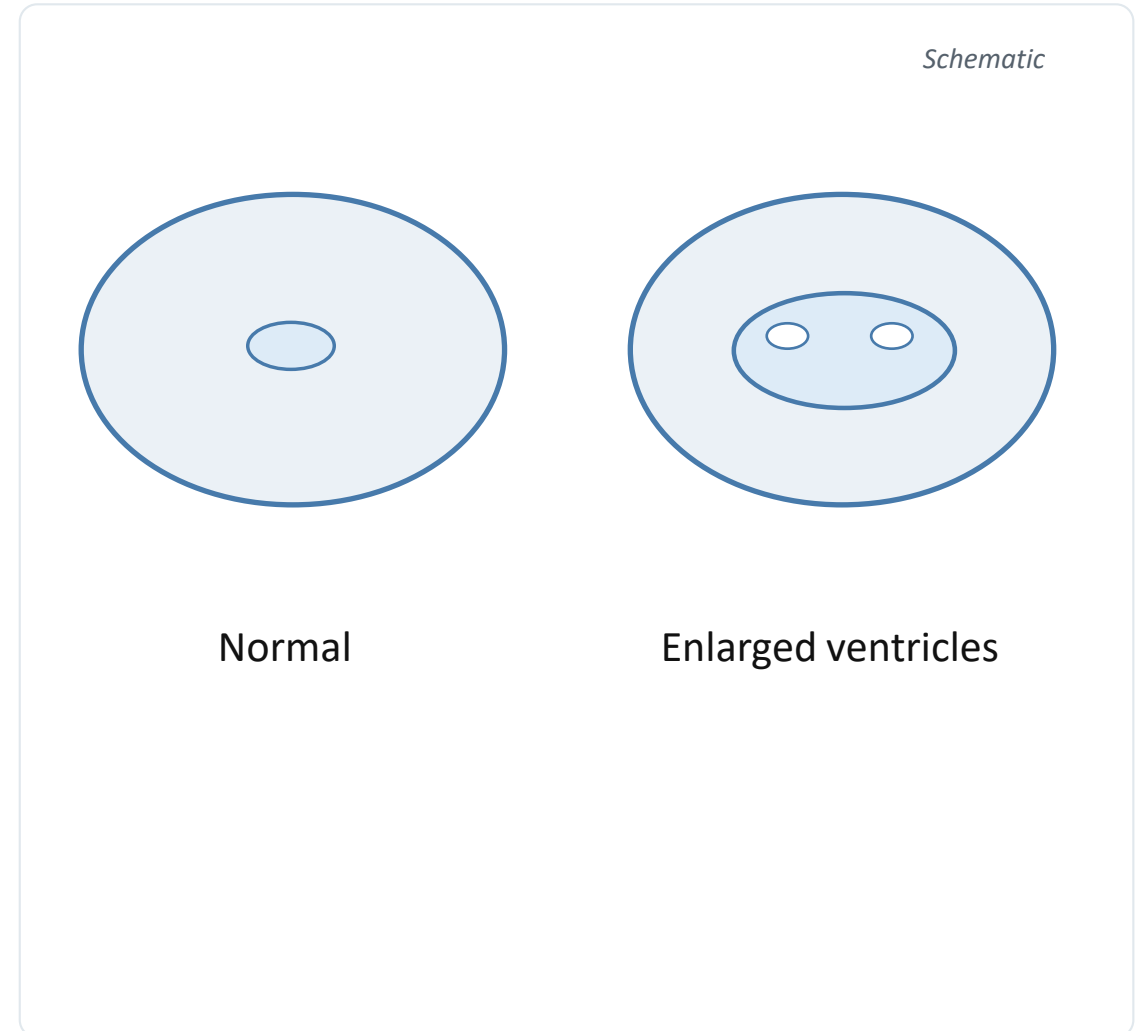
Schematic



Flattened gyri · narrowed sulci

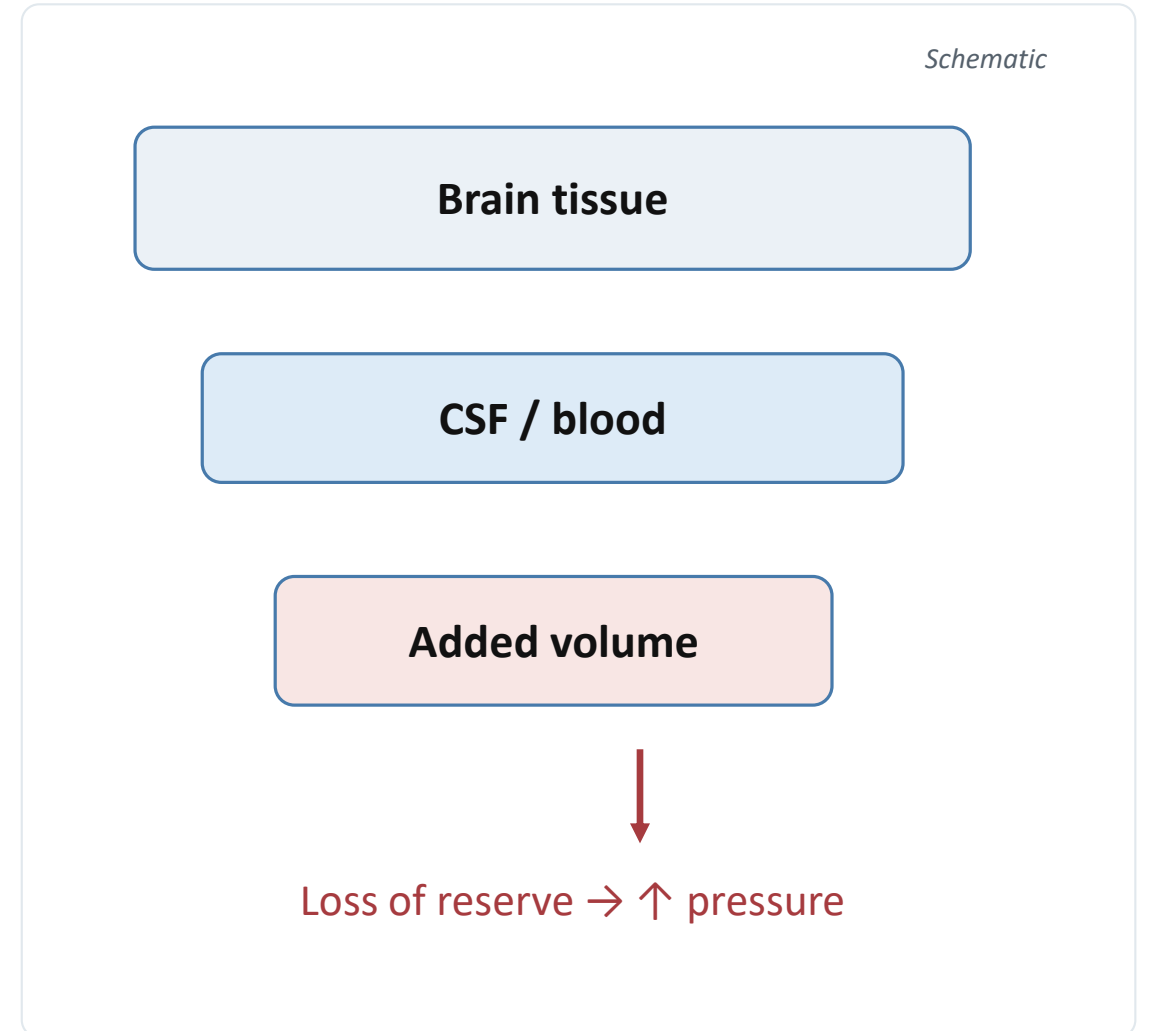
Severe swelling can compress the ventricular cavities

- Expansion of brain parenchyma displaces the intracranial contents.
- Ventricular cavities may become compressed rather than enlarged.
- This contrasts with the ventricular dilation typical of hydrocephalus.



Edema becomes dangerous when swelling causes displacement

- Expansion progressively exhausts cranial compensation.
- Mass effect can move brain across dural boundaries.
- Vascular compression and herniation are severe secondary consequences.



Clinicopathologic exercise: Peritumoral swelling

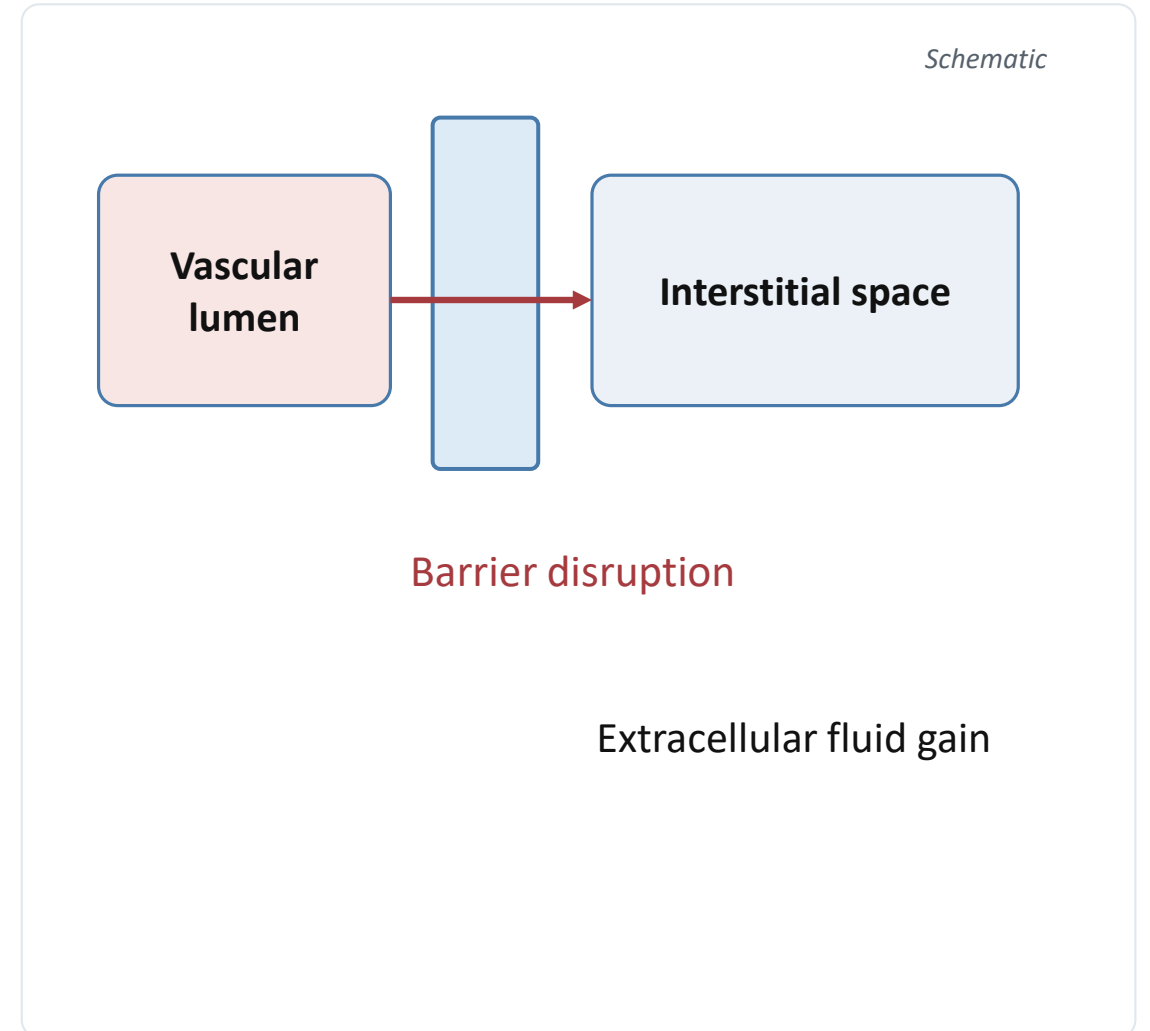
A cerebral mass is surrounded by swollen tissue. The surrounding microenvironment has abnormally permeable vessels, and extracellular fluid accumulates.

Which edema mechanism explains the perilesional swelling?

Reason from anatomy and mechanism before revealing the interpretation.

Peritumoral swelling indicates a vasogenic pathway

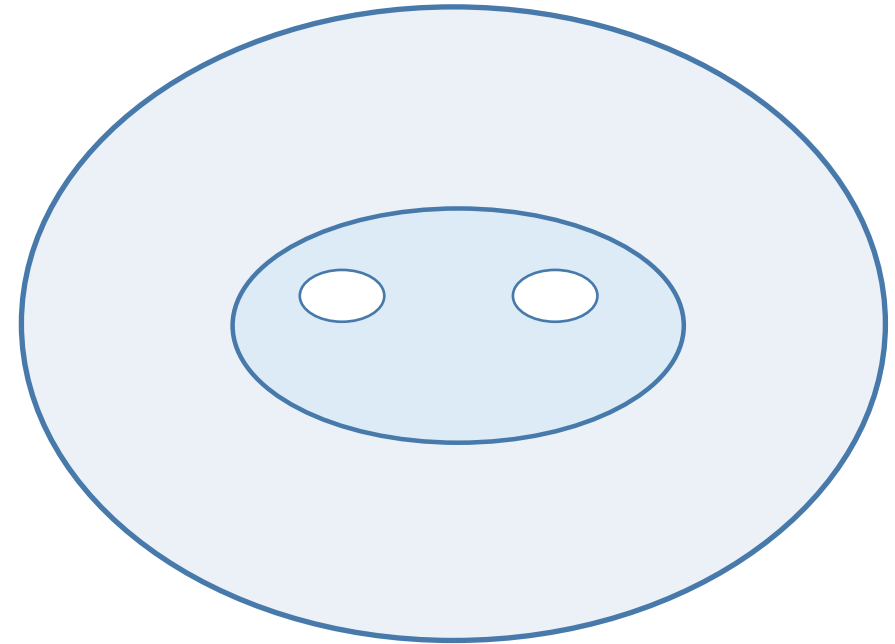
- A focal neoplasm is an established setting for localized vasogenic edema.
- Disrupted blood–brain barrier allows fluid into intercellular spaces.
- That fluid adds to the space-occupying effect of the lesion.



Hydrocephalus is excess CSF in the ventricular system

- CSF accumulation may involve all ventricles or only a portion.
- Ventricular expansion is the major gross morphologic consequence.
- The increase in CSF volume can raise intracranial pressure.

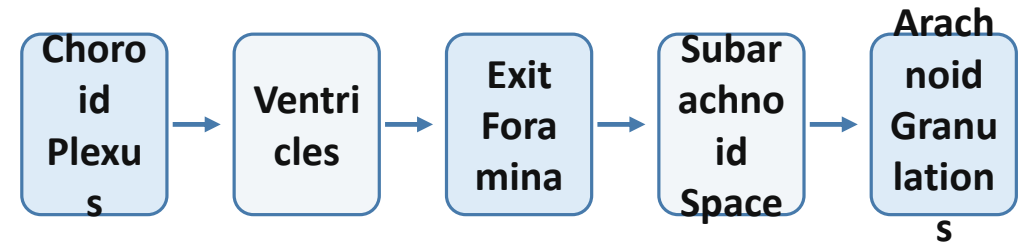
Schematic



Ventricular enlargement

The choroid plexus produces cerebrospinal fluid

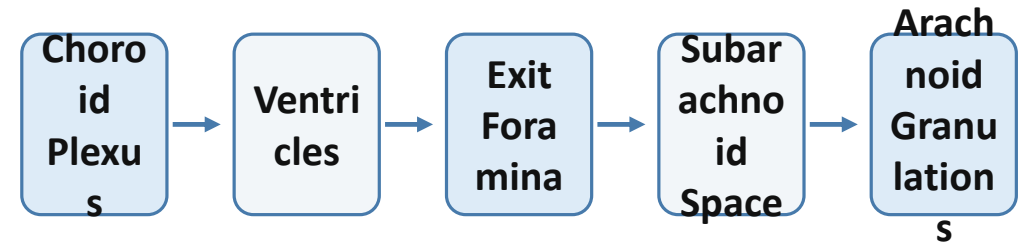
- CSF is generated within the ventricular system by the choroid plexus.
- CSF production is normally balanced by circulation and absorption.
- Disturbance of that balance is central to hydrocephalus.



Production → circulation → resorption

CSF circulates through the ventricular system

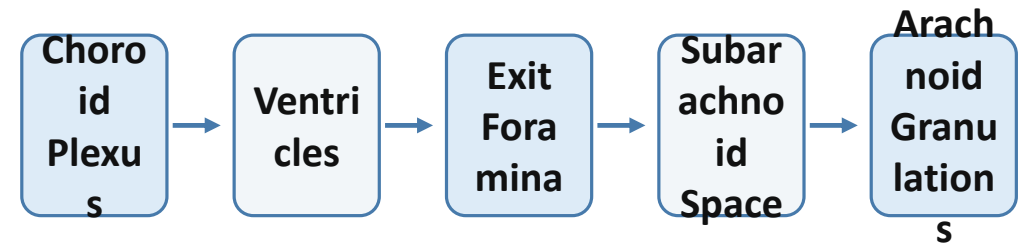
- Newly produced CSF passes through the interconnected ventricles.
- The ventricular fluid ultimately reaches the basal cisterns.
- A focal obstruction can block communication at a narrow passage.



Production → circulation → resorption

CSF leaves the ventricular system at the fourth ventricle

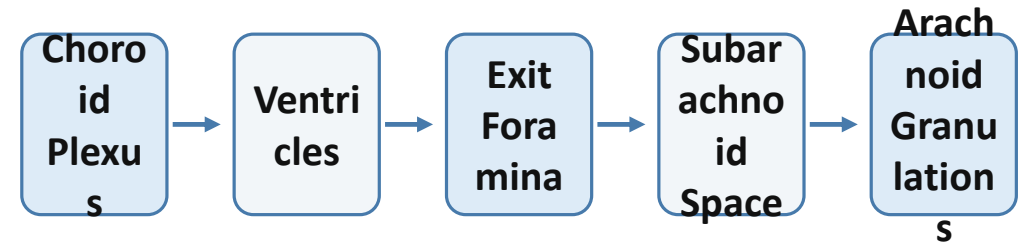
- CSF reaches the subarachnoid compartment through exit foramina.
- The foramina of Luschka and Magendie communicate with basal CSF spaces.
- Flow to the cisterna magna lies along the normal circulation pathway.



Production → circulation → resorption

Subarachnoid CSF reaches the cerebral convexities

- After leaving the ventricles, CSF bathes subarachnoid spaces.
- It circulates toward the superior cerebral convexities.
- Arachnoid granulations are the principal absorption pathway described.



Production → circulation → resorption

Arachnoid granulations absorb CSF

- Normal CSF volume depends on effective resorption.
- Arachnoid fibrosis after meningitis can impair this process.
- The ventricles may enlarge even though their exits remain patent.

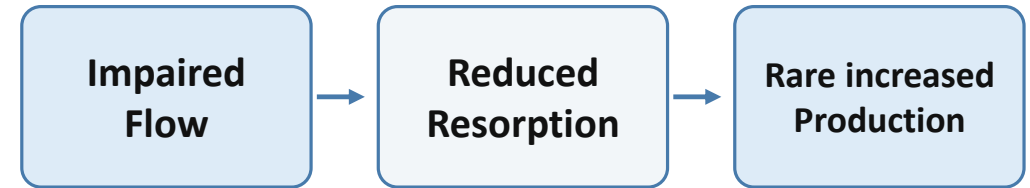
Schematic



Most hydrocephalus reflects impaired flow or resorption

- Obstruction may prevent CSF from reaching downstream spaces.
- Impaired arachnoid absorption may retain circulating CSF.
- Overproduction is less common and linked to choroid plexus tumors.

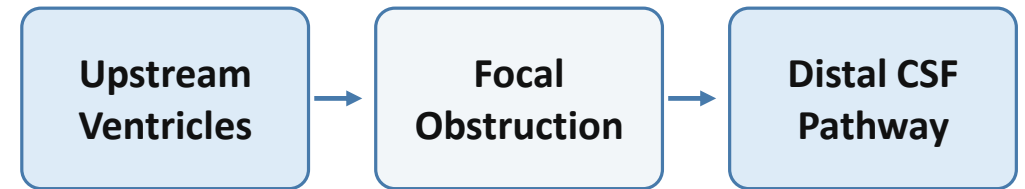
Schematic



Noncommunicating hydrocephalus has focal obstruction

- An obstruction interrupts continuity within the ventricular pathway.
- A mass in the third ventricle is one example.
- Stenosis of the cerebral aqueduct is another classic example.

Schematic

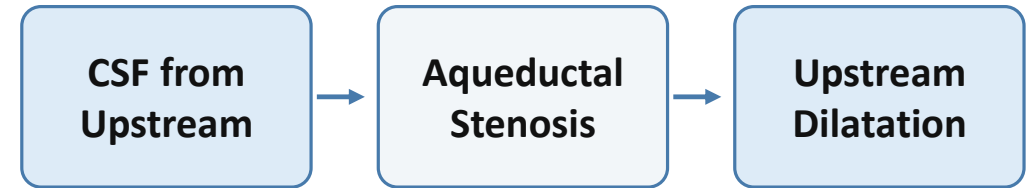


Production → circulation → resorption

Aqueductal stenosis illustrates obstructive hydrocephalus

- The cerebral aqueduct is a narrow CSF passage.
- Stenosis restricts outflow from upstream ventricles.
- CSF accumulates proximal to the blockage.

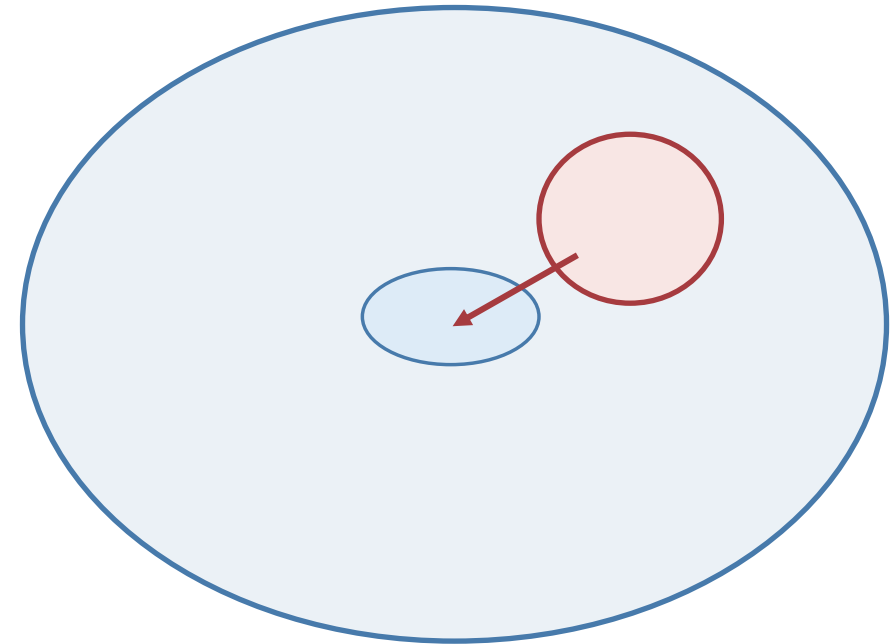
Schematic



A third-ventricular mass can obstruct CSF flow

- Space-occupying lesions may directly block a ventricular pathway.
- The blockage produces a noncommunicating pattern.
- Obstructed CSF accumulates proximal to the lesion.

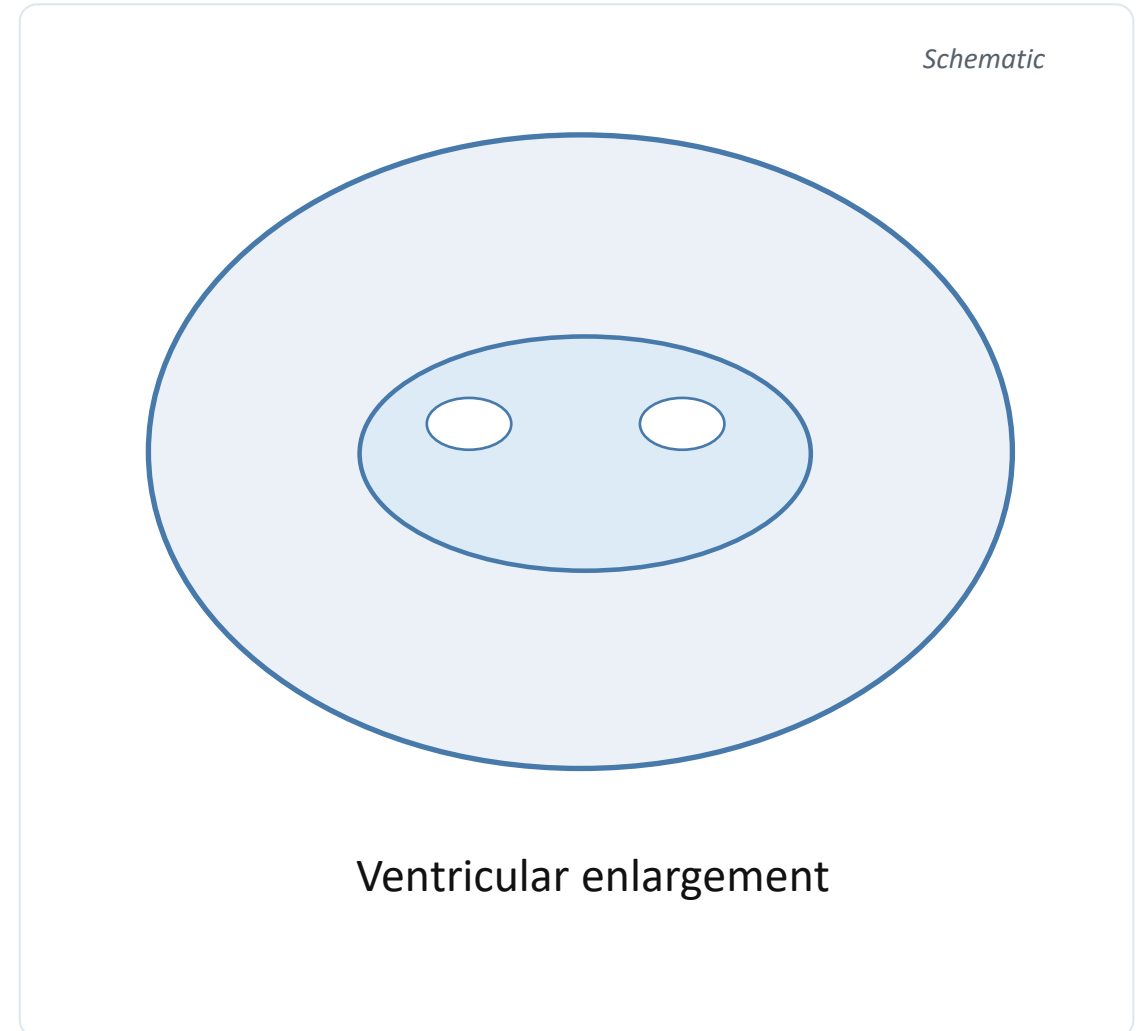
Schematic



Expanding focal mass

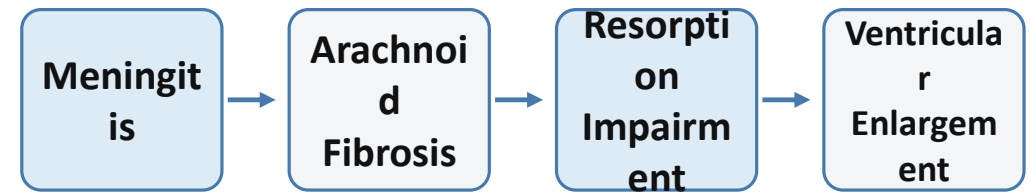
Communicating hydrocephalus retains ventricular continuity

- The ventricular system continues to communicate with the subarachnoid space.
- The entire ventricular system is enlarged.
- Impaired CSF resorption is an important mechanism.



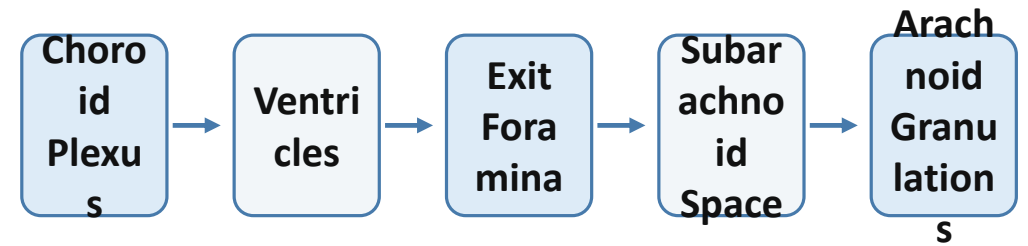
Postmeningitic arachnoid fibrosis impairs resorption

- Meningitis can cause fibrosis in arachnoid tissues.
- Fibrosis reduces normal CSF absorption.
- CSF retention occurs despite an intact ventricular outflow pathway.



Choroid plexus tumors may cause CSF overproduction

- Excess CSF production is an uncommon cause of hydrocephalus.
- The source associates it with tumors of the choroid plexus.
- Increased production can enlarge the ventricular system.



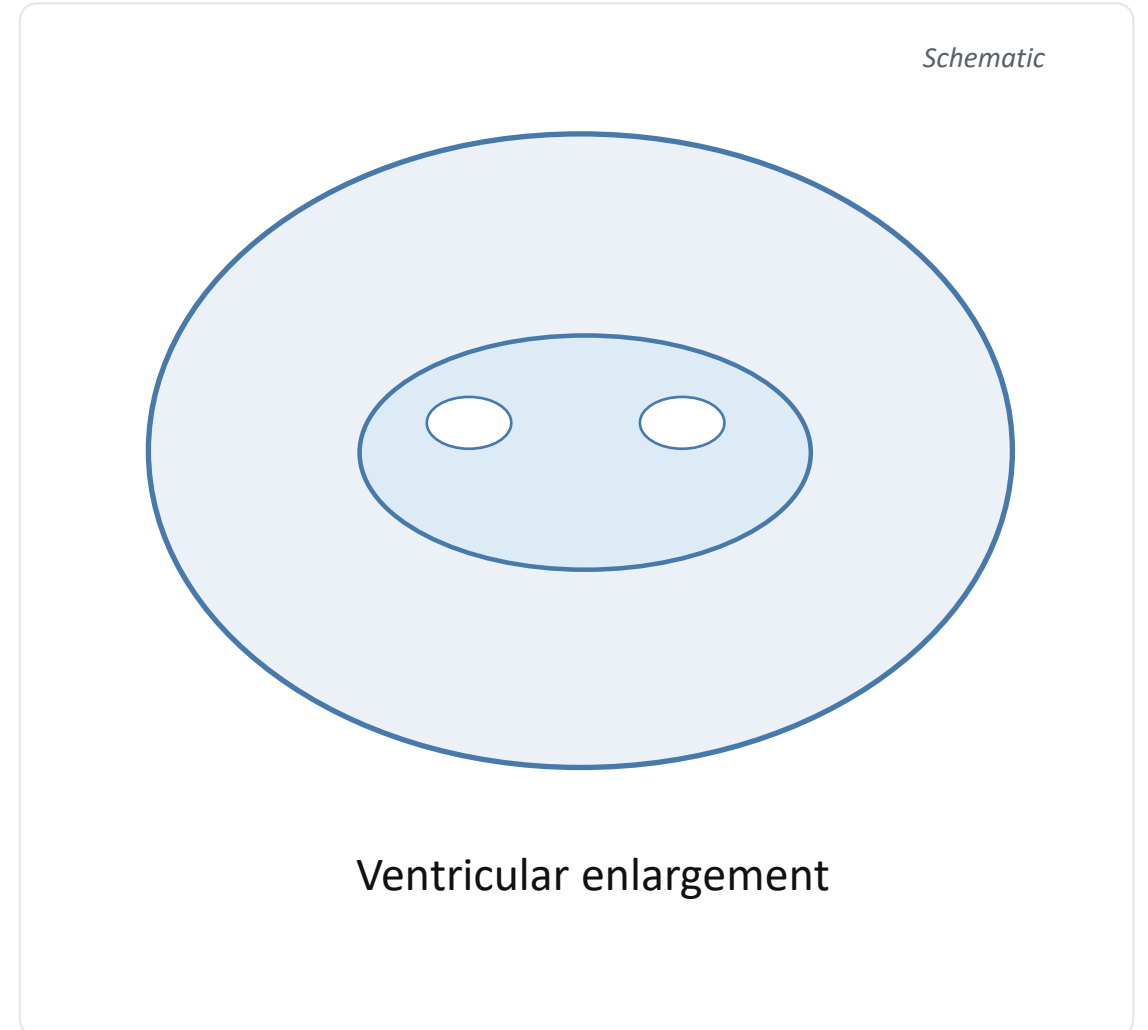
Production → circulation → resorption

Hydrocephalus patterns follow the site of dysfunction

Pattern	Where the problem lies	Expected consequence
Noncommunicating	Focal ventricular obstruction	Upstream ventricles expand
Communicating	Absorption failure / rare excess production	Entire ventricular system expands
Ex vacuo	Loss of surrounding brain parenchyma	Compensatory ventricular enlargement

Hydrocephalus expands the ventricular cavities

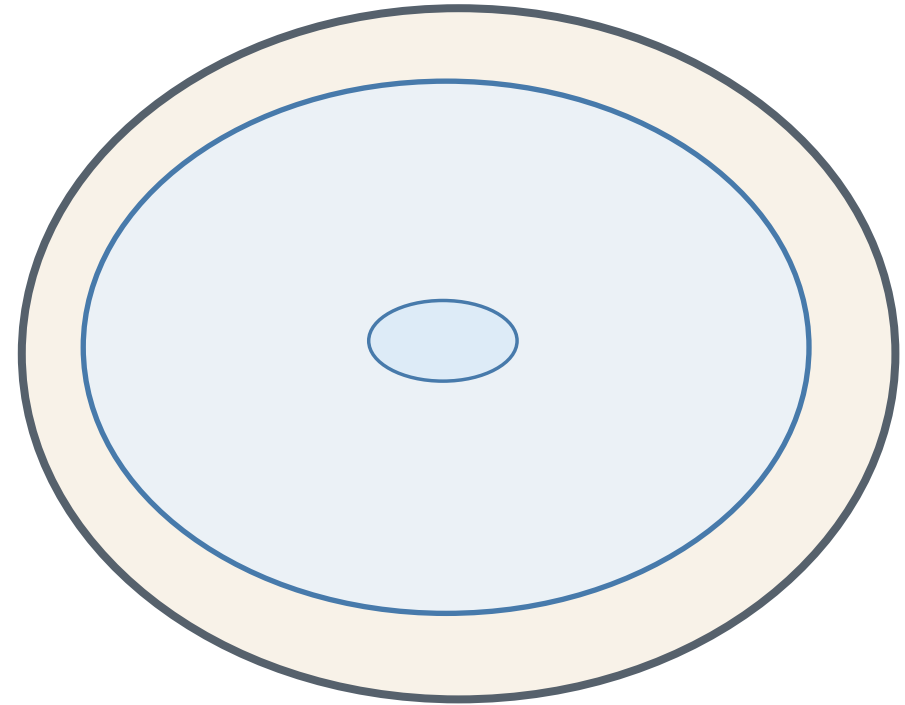
- The defining morphologic change is enlargement of CSF spaces within the ventricles.
- In coronal section, dilated lateral ventricles may be conspicuous.
- The ventricular compartment must be distinguished from tissue swelling.



Hydrocephalus before suture closure enlarges the head

- The developing skull has cranial sutures that have not yet closed.
- Ventricular expansion may increase head circumference in infancy.
- This presentation differs from hydrocephalus in the mature skull.

Schematic

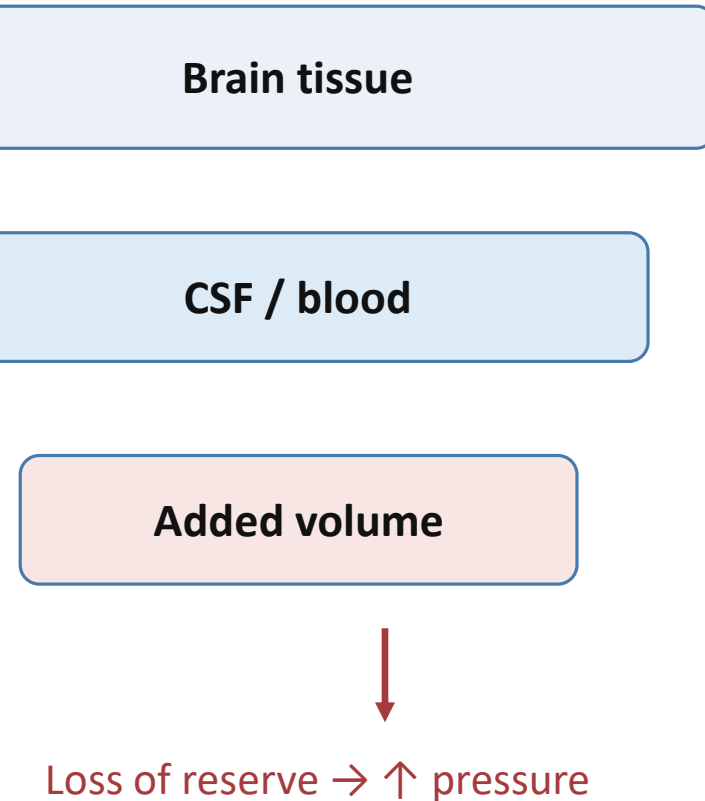


Volume expansion within rigid skull

Hydrocephalus after suture closure raises pressure

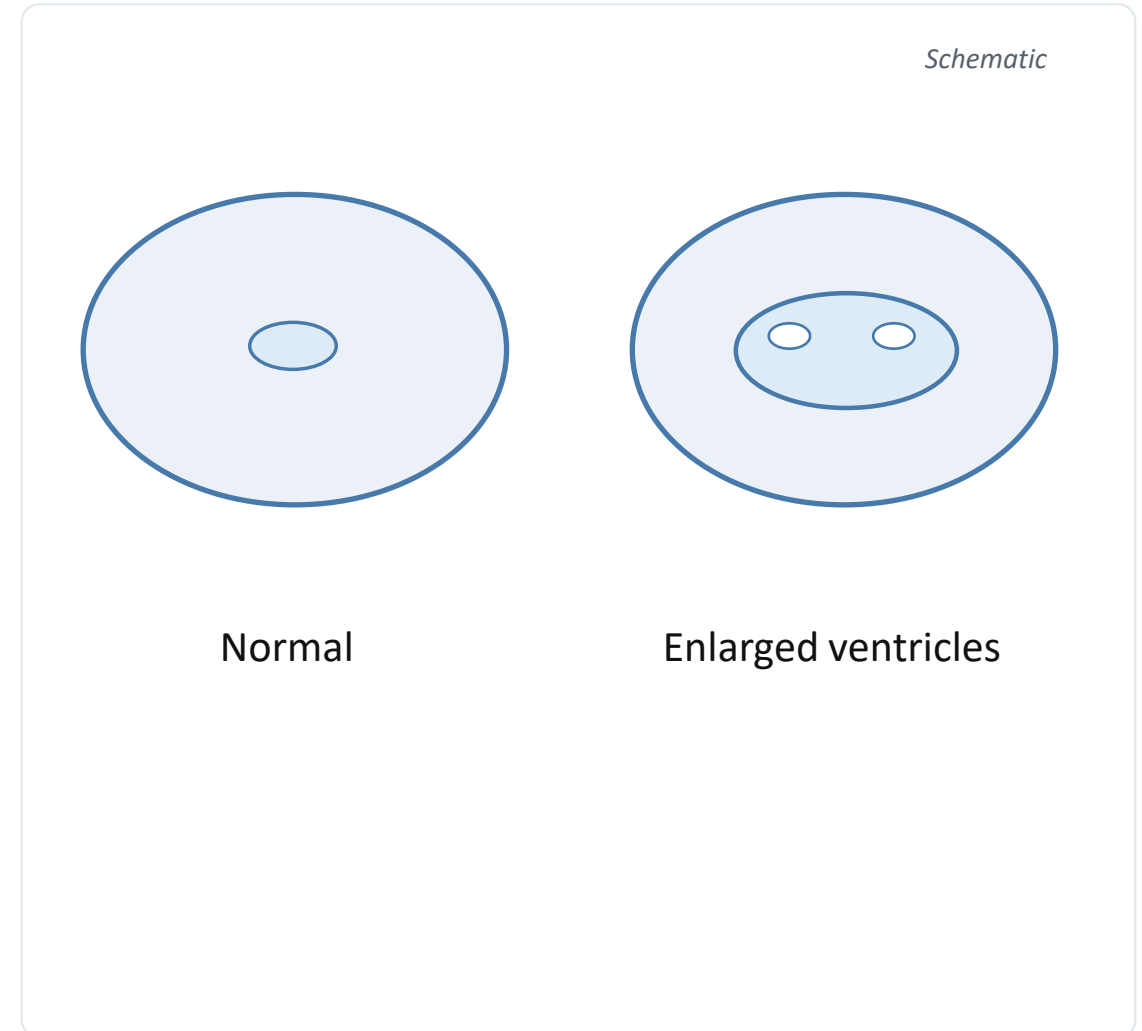
- After suture closure, cranial circumference does not enlarge.
- The ventricles expand inside a mechanically constrained compartment.
- Intracranial pressure may rise as CSF volume increases.

Schematic



Hydrocephalus ex vacuo is a compensatory enlargement

- Loss of brain parenchyma leaves additional intracranial space.
- Ventricular CSF volume increases to occupy that space.
- This is not equivalent to excess CSF from an obstructed pathway.



Ex vacuo enlargement and true hydrocephalus differ

True hydrocephalus

- Excess CSF volume from altered flow, absorption, or production.
- Ventricular enlargement may increase intracranial pressure.
- Identify the abnormal CSF pathway.

Hydrocephalus ex vacuo

- Brain tissue loss precedes ventricular enlargement.
- Enlargement compensates for loss of parenchymal volume.
- Not primarily a CSF flow-obstruction disorder.

Clinicopathologic exercise: Aqueductal narrowing

A ventricular imaging study shows dilation of CSF spaces above a narrowed cerebral aqueduct. The downstream pathway is not the primary site of disease.

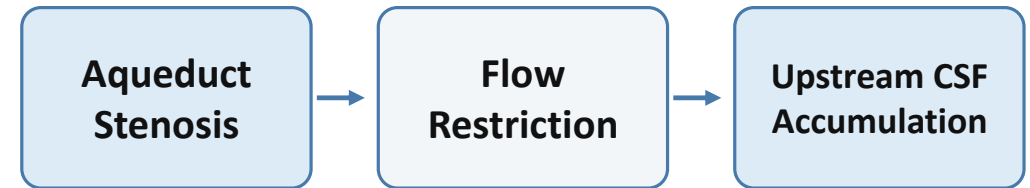
Which hydrocephalus category best fits the lesion?

Reason from anatomy and mechanism before revealing the interpretation.

Aqueductal narrowing causes noncommunicating hydrocephalus

- A focal ventricular conduit is obstructed.
- CSF accumulates upstream, producing ventricular dilation.
- The lesion is classified as noncommunicating (obstructive) hydrocephalus.

Schematic



Raised intracranial pressure reflects exhausted capacity

- Expanding tissue, excess CSF, or a focal mass adds to intracranial volume.
- Displacement of CSF provides only limited accommodation.
- Increasing pressure can ultimately displace brain tissue.

Schematic

Brain tissue

CSF / blood

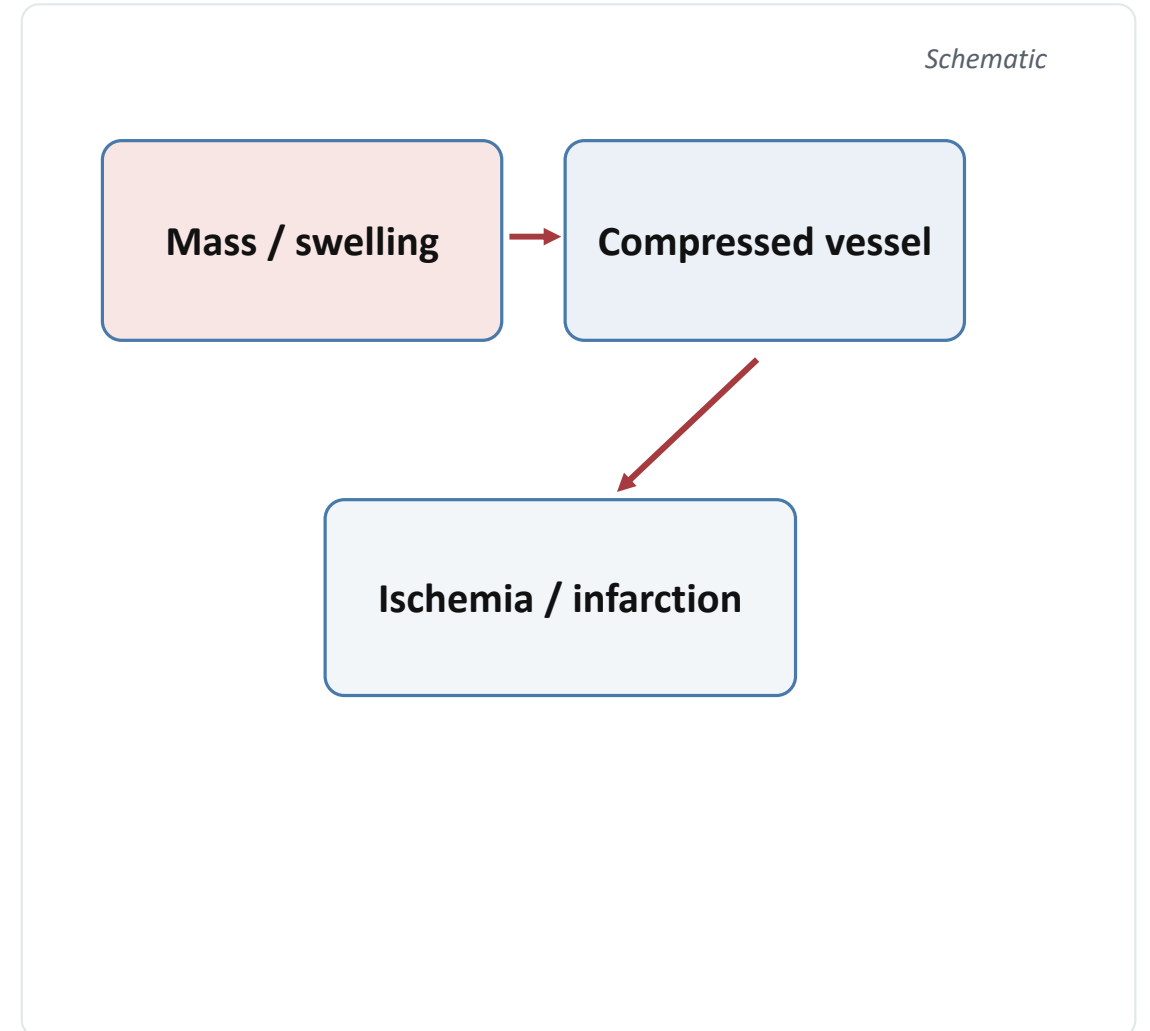
Added volume



Loss of reserve → ↑ pressure

Large or expanding lesions threaten cerebral perfusion

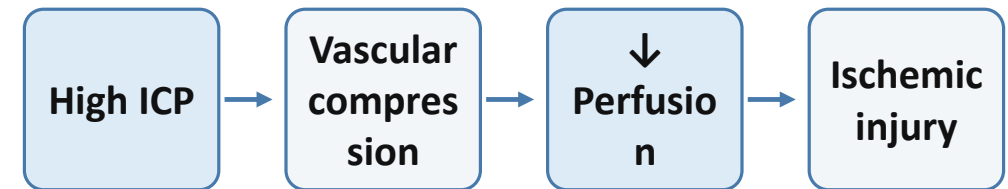
- High intracranial pressure can compress cerebral vasculature.
- Reduced perfusion promotes ischemic tissue injury.
- Infarction may develop as a secondary consequence of mass effect.



Pressure-induced ischemia can amplify cerebral swelling

- An expanding lesion raises intracranial pressure.
- Reduced blood flow can aggravate cellular and vascular damage.
- Secondary edema causes further volume expansion.

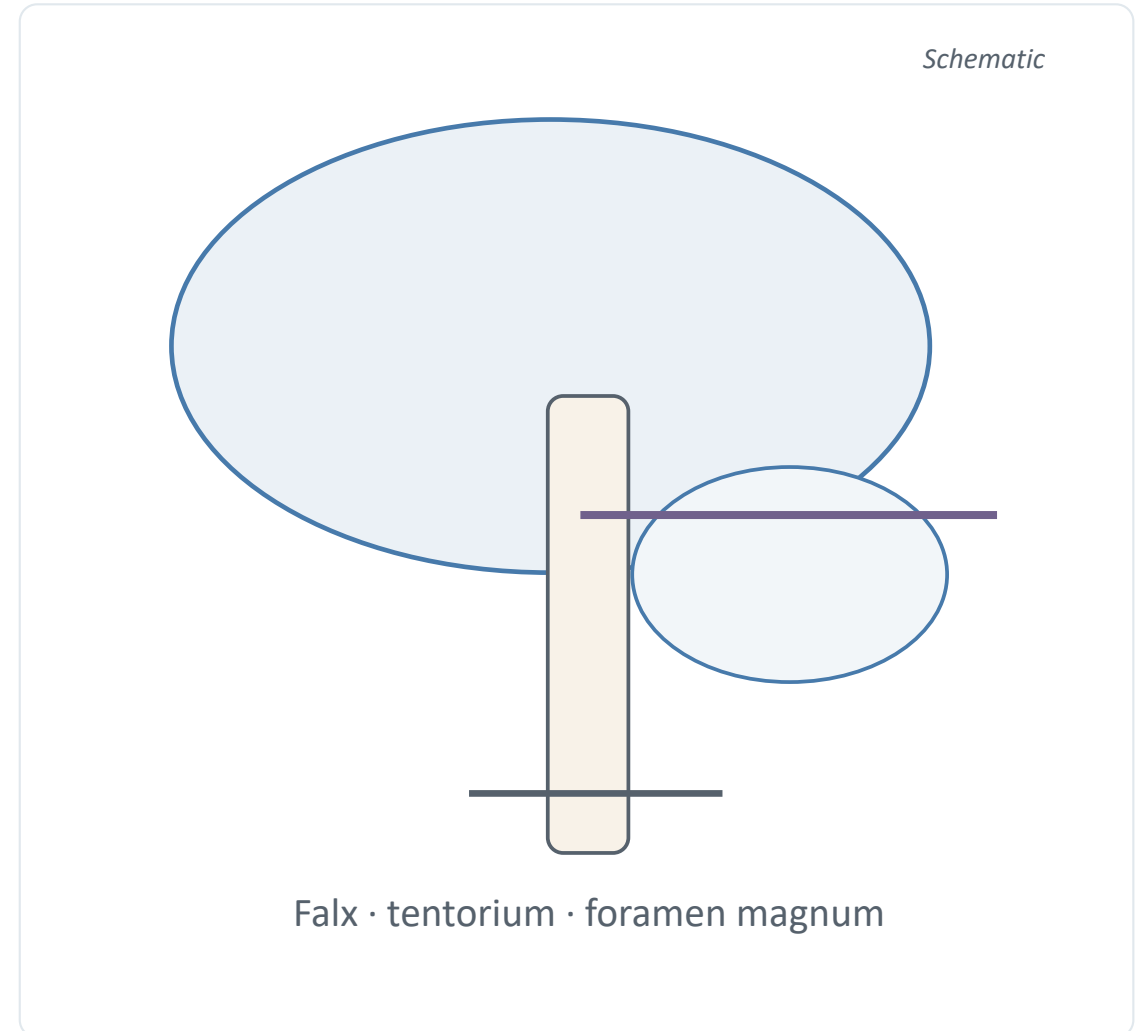
Schematic



Edema may intensify the cycle

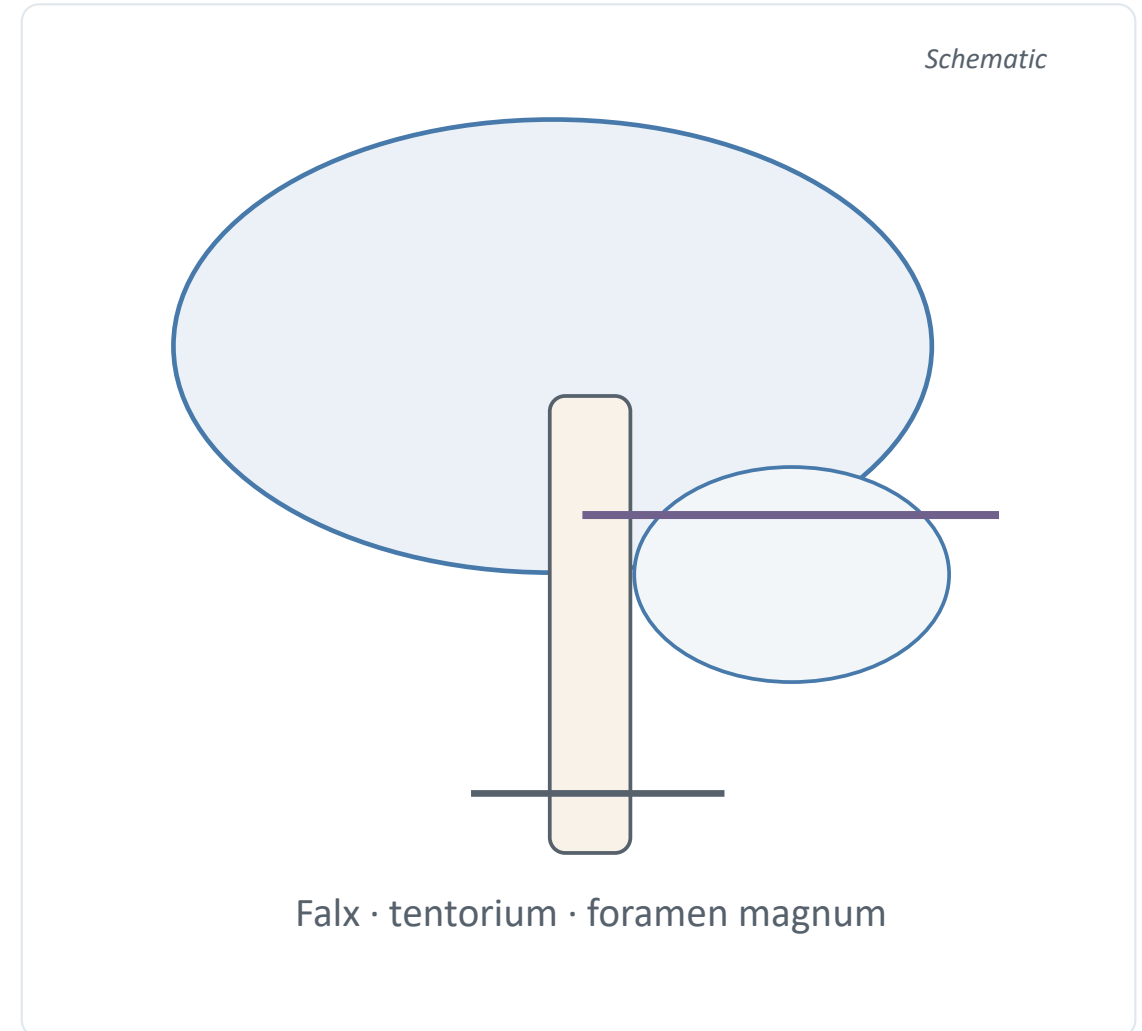
Herniation is brain movement across fixed boundaries

- Brain tissue moves past the falx or tentorium, or through skull openings.
- Displacement occurs when the brain cannot accommodate pressure elevation.
- The direction of movement determines the endangered structures.



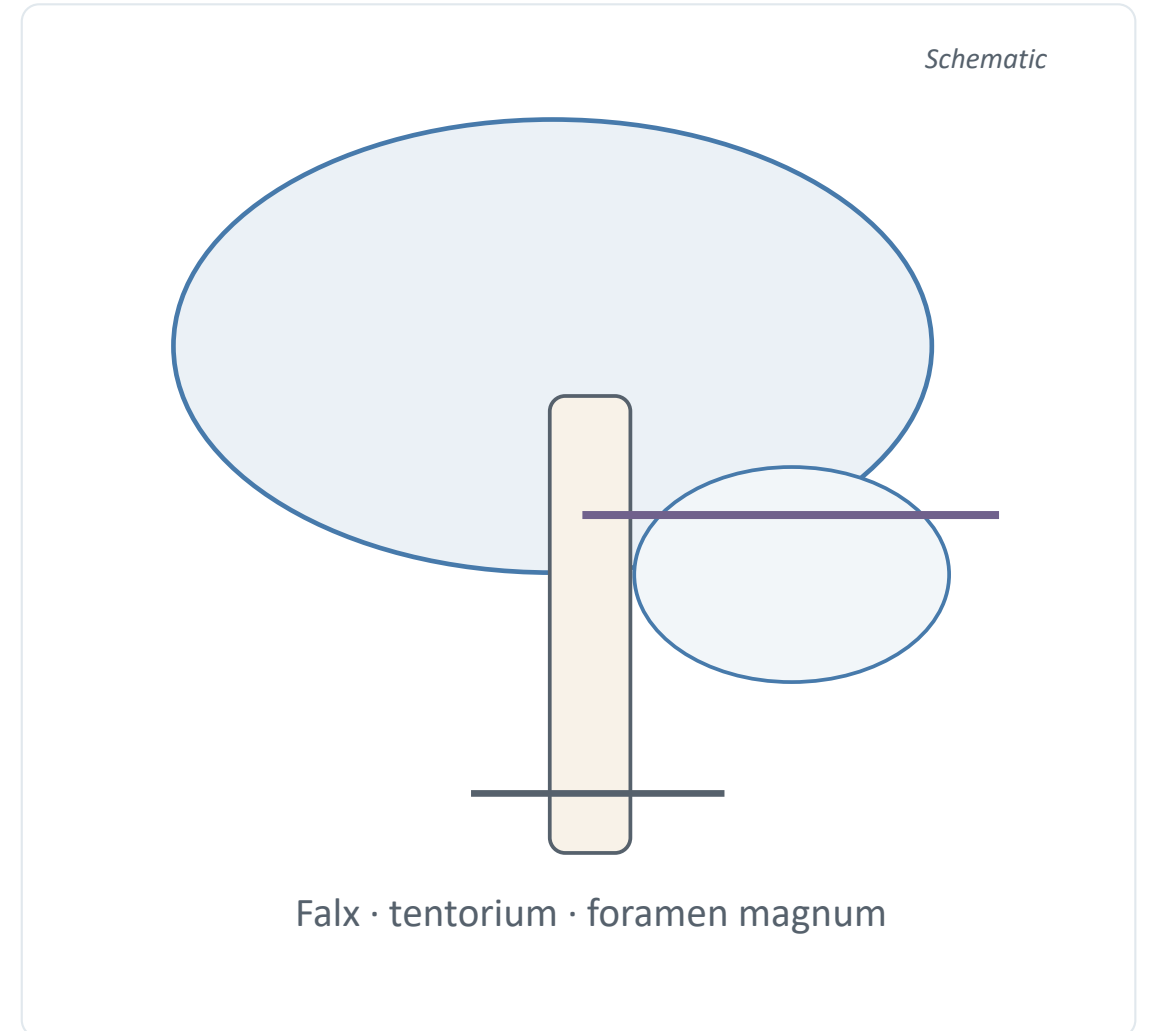
Major anatomic boundaries determine herniation patterns

- The falx cerebri separates the cerebral hemispheres.
- The tentorium cerebelli forms a rigid intracranial partition.
- The foramen magnum provides the inferior opening into the spinal canal.



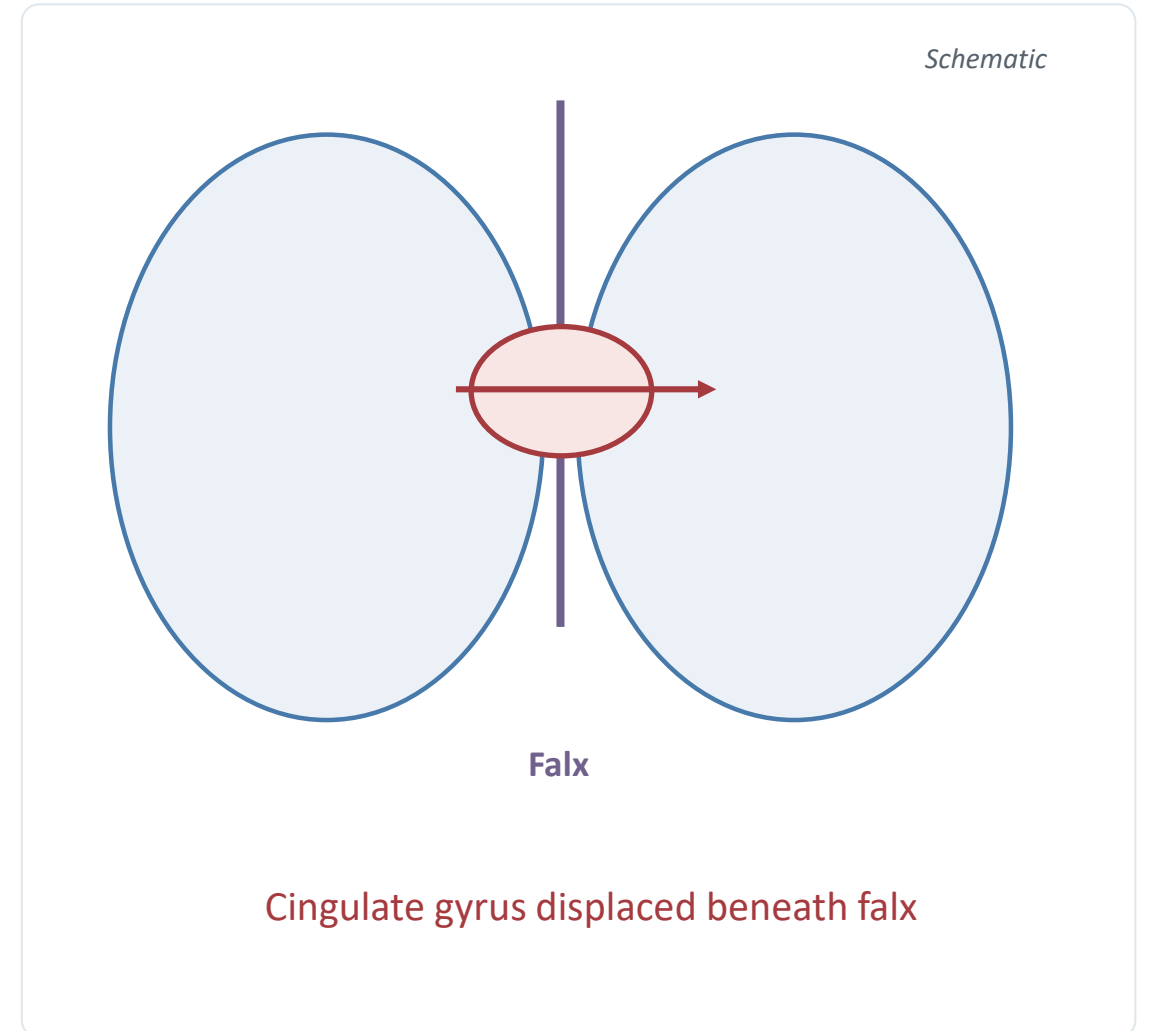
Severe mass effect may generate multiple herniations

- Expanding brain tissue can shift along more than one route.
- Subfalcine, transtentorial, and tonsillar displacement may coexist.
- Pattern recognition must include both displaced and compressed structures.



Subfalcine herniation shifts the cingulate gyrus

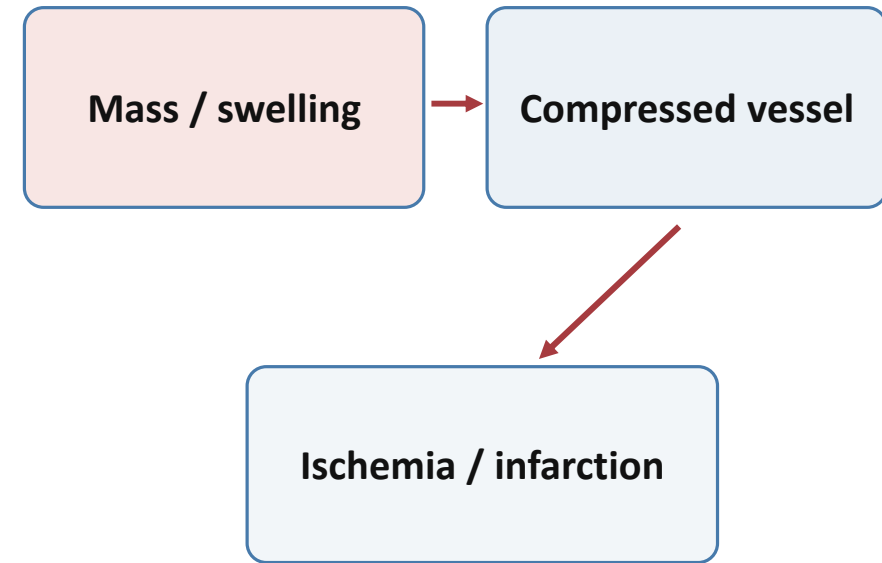
- Unilateral or asymmetric cerebral expansion pushes tissue medially.
- The cingulate gyrus moves beneath the falx cerebri.
- The adjacent anterior cerebral artery branches may be compressed.



Anterior cerebral artery compression threatens infarction

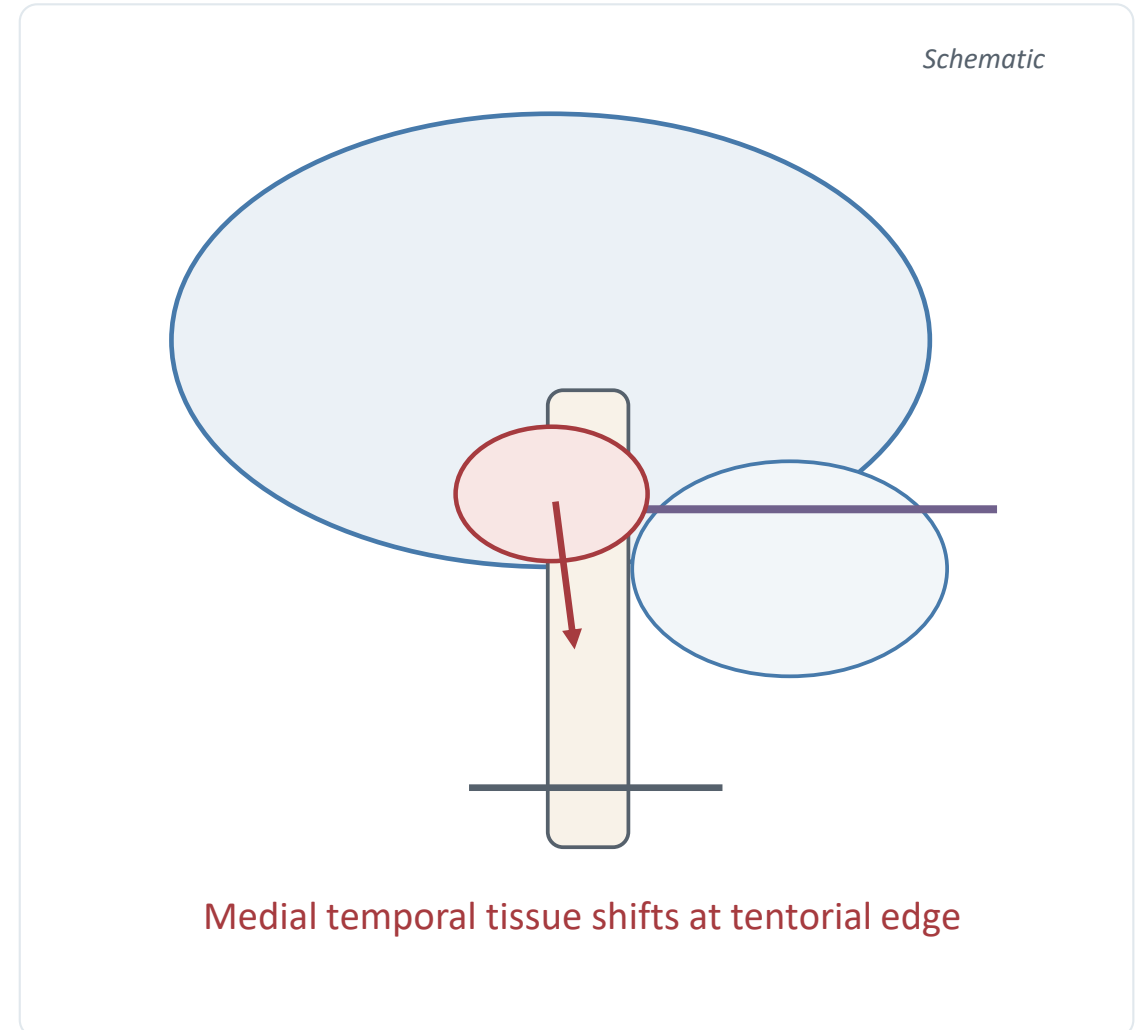
- Subfalcine tissue movement occurs close to anterior cerebral vessels.
- Vascular compression may impair downstream perfusion.
- Secondary infarcts can complicate the original mass lesion.

Schematic



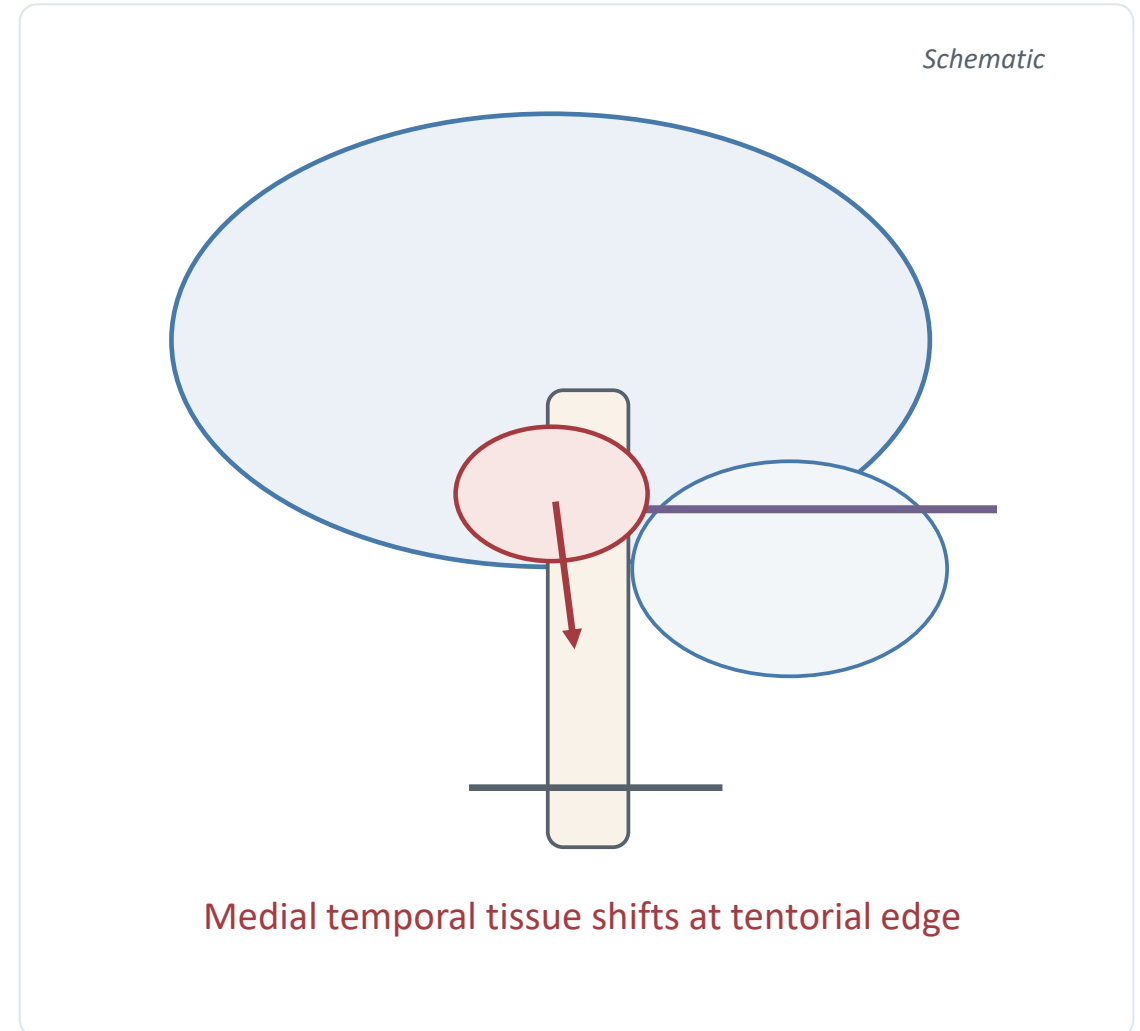
Transtentorial herniation compresses the medial temporal lobe

- The uncus and mesial temporal structures move toward the tentorial edge.
- The free tentorial margin compresses adjacent neural and vascular structures.
- Neurologic findings reflect the structures compromised by displacement.



Uncal herniation can compromise cranial nerve III

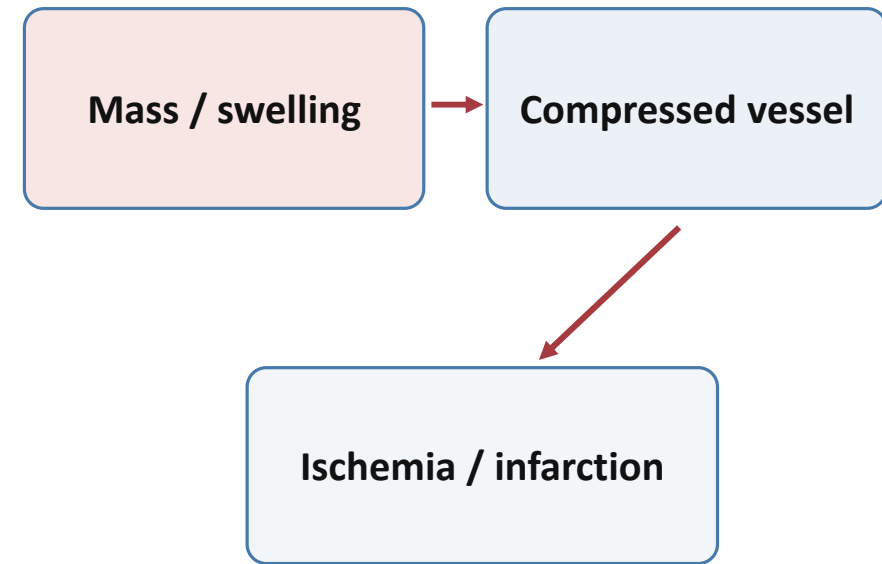
- The oculomotor nerve lies at risk beside the tentorial opening.
- Compression causes pupillary dilation on the side of the lesion.
- Ipsilateral ocular movement impairment may accompany the pupil change.



Posterior cerebral artery compression affects vision

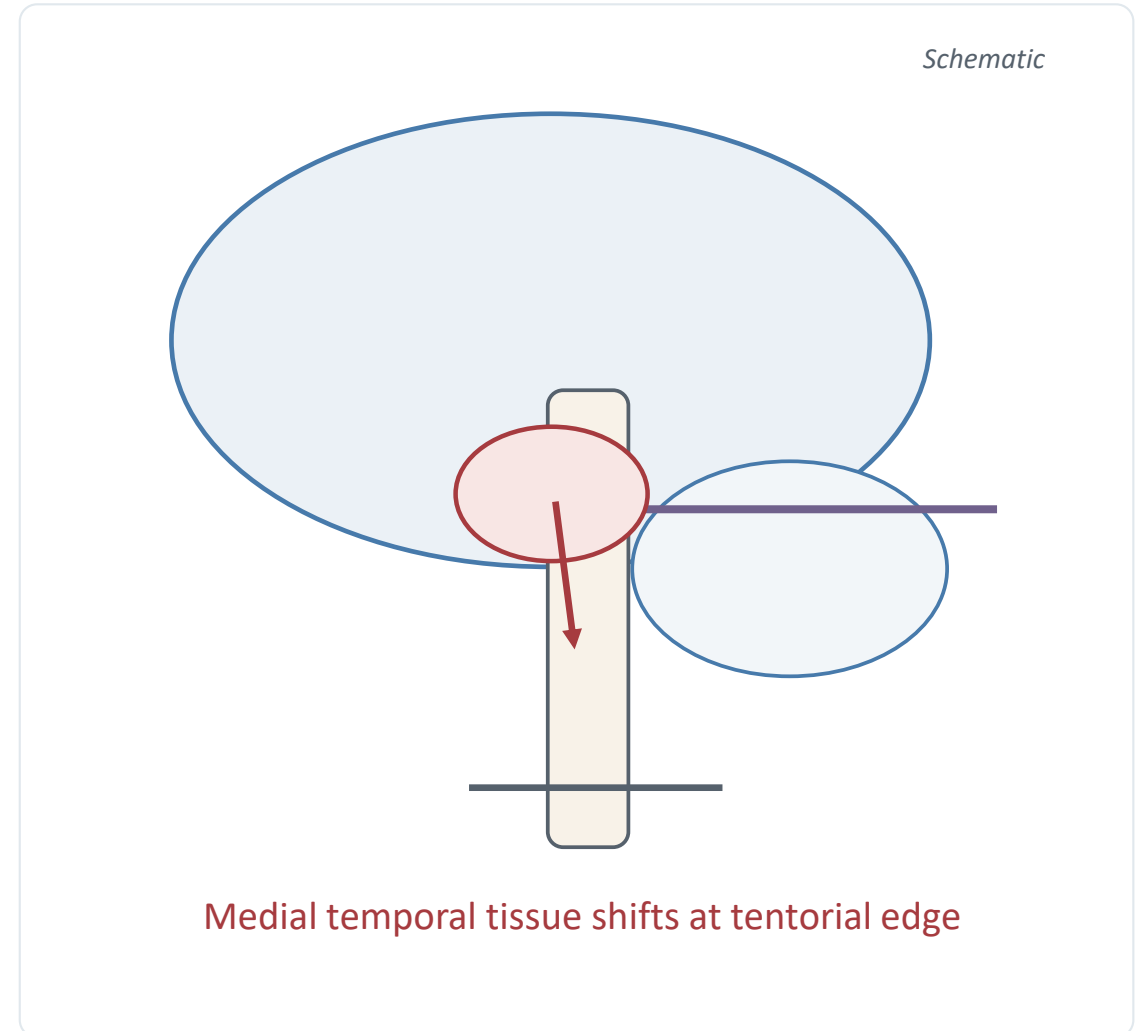
- The ipsilateral posterior cerebral artery may be compressed.
- Its territory includes primary visual cortex.
- Ischemia can produce contralateral homonymous hemianopia.

Schematic



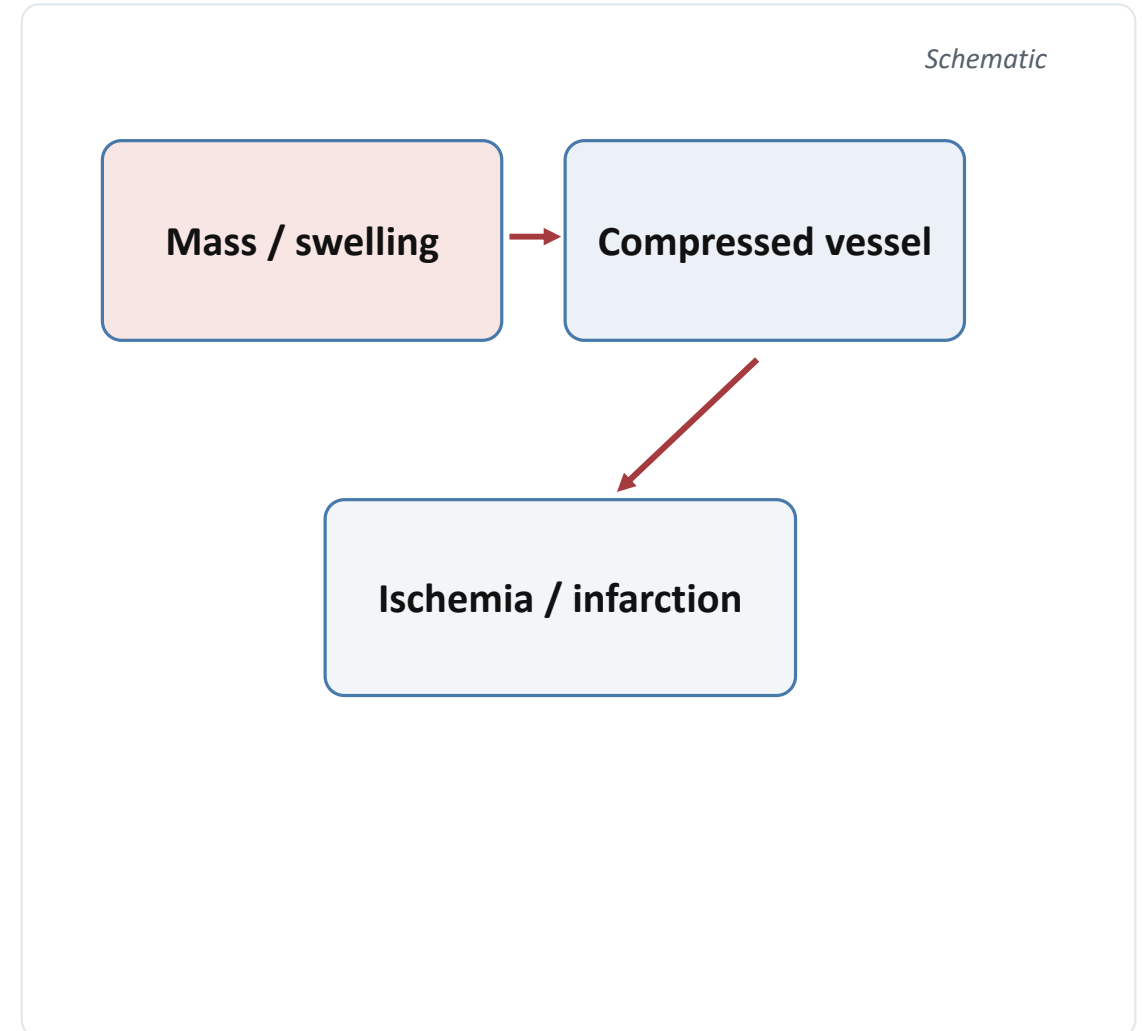
Kernohan notch can create false localizing weakness

- Major transtentorial shift can compress the opposite cerebral peduncle.
- The resulting hemiparesis can occur ipsilateral to the mass lesion.
- This paradox is called the Kernohan notch phenomenon.



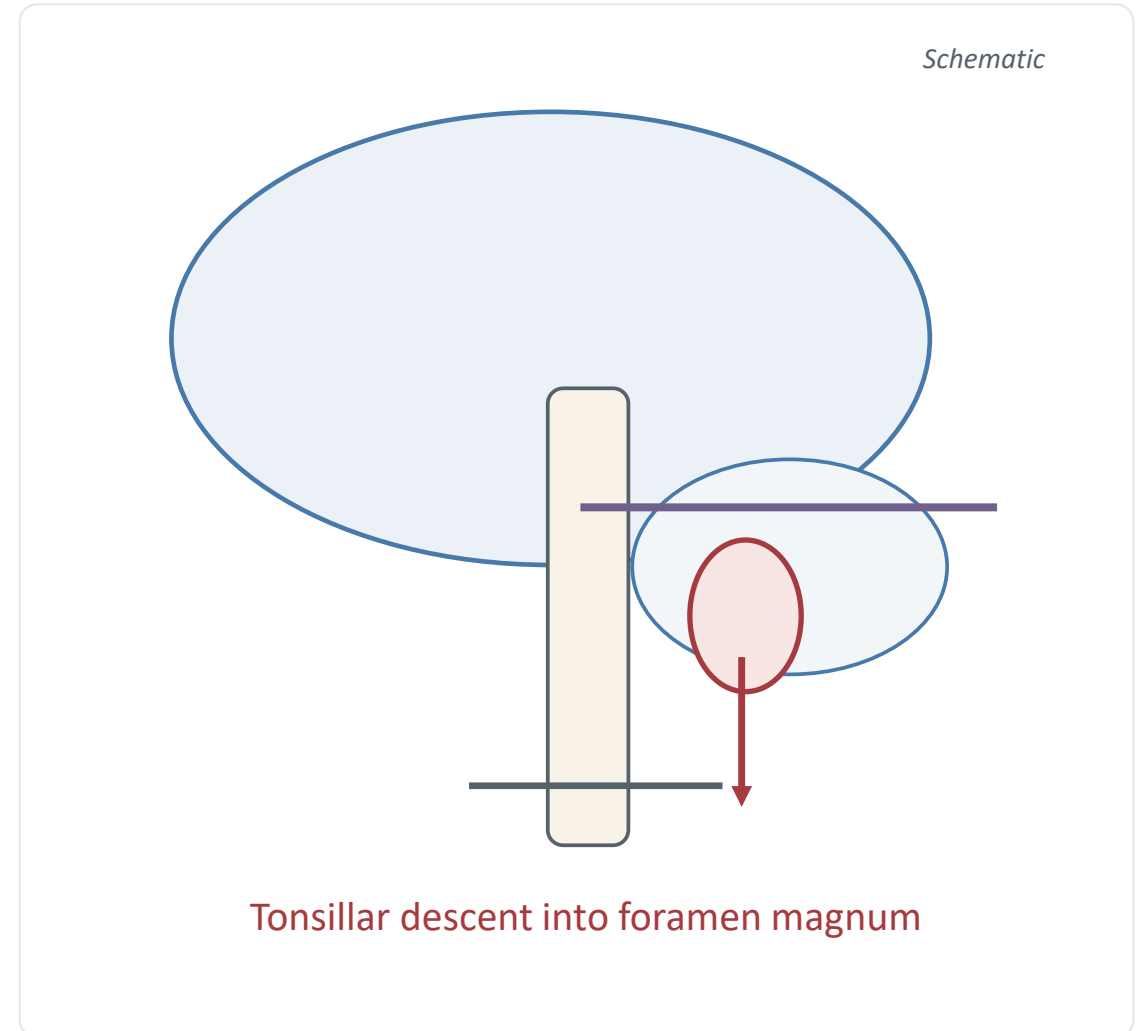
Duret hemorrhages accompany severe downward displacement

- Transtentorial shift can distort penetrating brainstem vessels.
- Linear or flame-shaped hemorrhages form in midline or paramedian midbrain/pons.
- The source attributes them to vascular tearing or distortion.



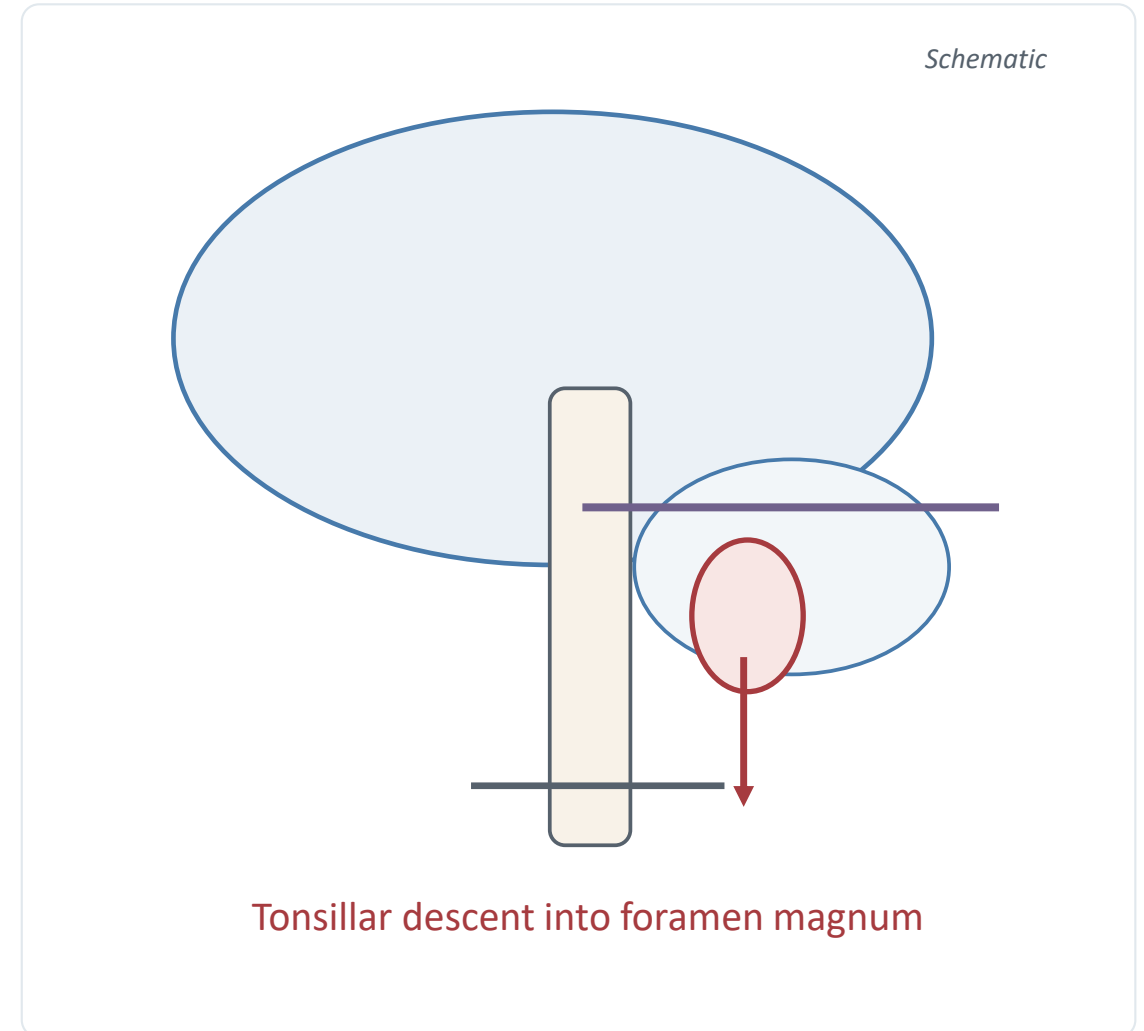
Tonsillar herniation displaces cerebellar tonsils inferiorly

- Cerebellar tonsils descend through the foramen magnum.
- The cervicomedullary region is compressed by the displacement.
- This pattern represents a major threat to brainstem function.



Tonsillar herniation jeopardizes vital medullary centers

- Compression involves the medulla at the craniocervical junction.
- Respiratory and cardiac control centers may be compromised.
- For this reason, the morphology is immediately life-threatening.



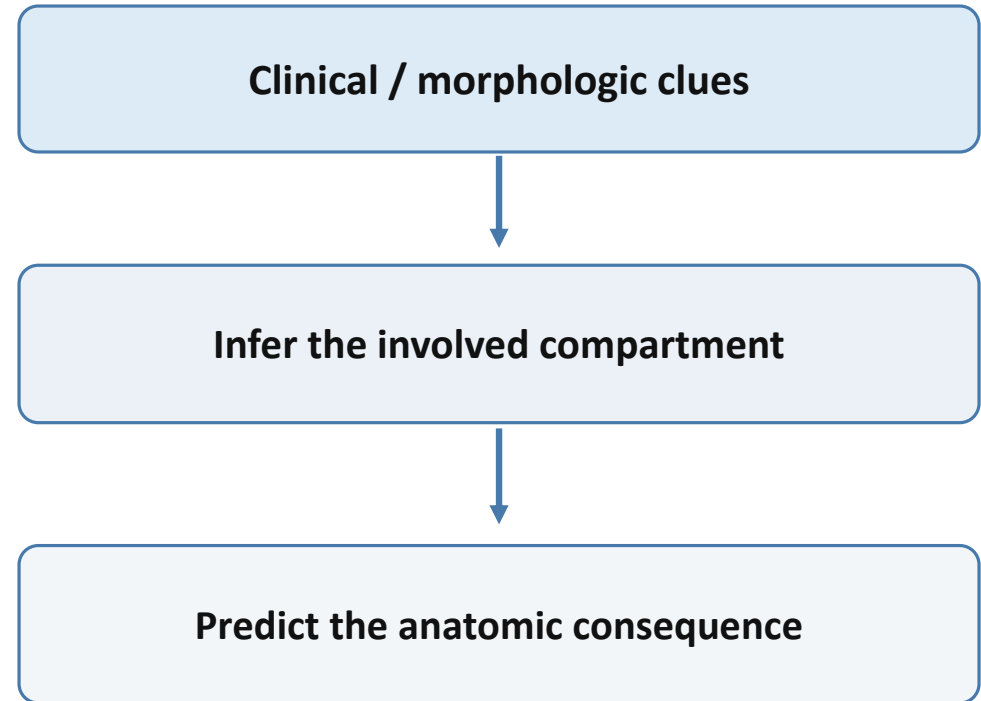
The three principal herniations produce different injuries

Type	Displacement	Principal danger
Subfalcine	Cingulate beneath falx	Anterior cerebral artery compression
Transtentorial	Medial temporal through tentorial notch	CN III, PCA, peduncle, brainstem injury
Tonsillar	Cerebellar tonsils through foramen magnum	Medullary compression

Clinical localization begins with the displaced structure

- Cingulate movement suggests risk to anterior cerebral artery branches.
- Medial temporal displacement suggests oculomotor, PCA, and brainstem effects.
- Tonsillar descent suggests compromise of vital medullary centers.

Schematic



Clinicopathologic exercise: Dilated pupil and a mass

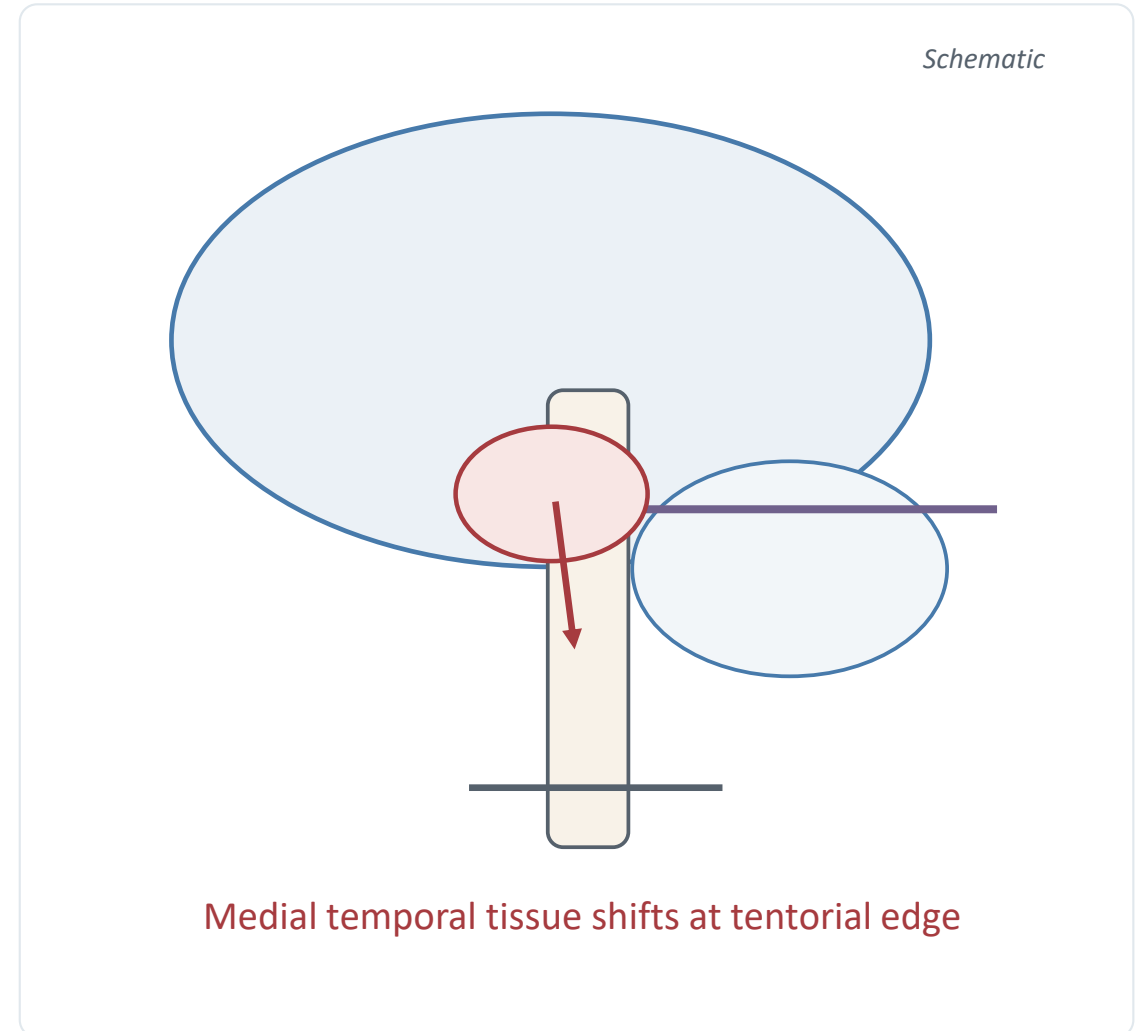
A unilateral temporal mass causes ipsilateral pupillary dilation and impaired ocular movements. Progressive midline displacement is suspected.

Which herniation route and compressed nerve best explain the findings?

Reason from anatomy and mechanism before revealing the interpretation.

A dilated ipsilateral pupil localizes uncus displacement

- The medial temporal lobe shifts against the tentorium.
- Compression of ipsilateral cranial nerve III impairs pupil and eye movements.
- Additional PCA and brainstem injury may follow with progression.



Clinicopathologic exercise: Downward posterior fossa shift

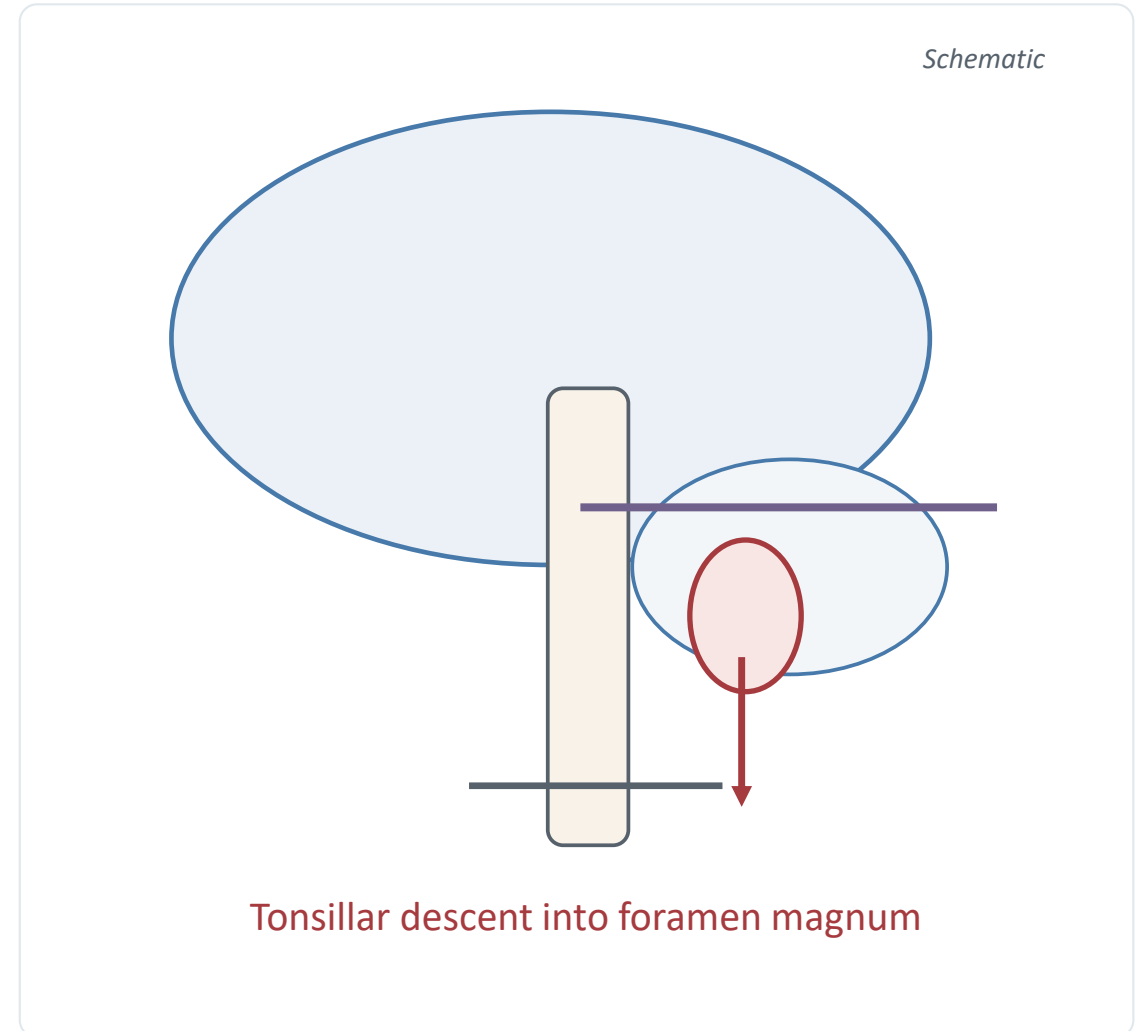
There is marked mass effect in the posterior fossa. The cerebellar tonsils descend through the skull base opening and medullary function becomes threatened.

What is the herniation pattern, and why is it life-threatening?

Reason from anatomy and mechanism before revealing the interpretation.

Foramen magnum descent endangers medullary functions

- The displacement is tonsillar herniation.
- Descending cerebellar tonsils compress the medulla.
- Vital cardiorespiratory centers may fail.



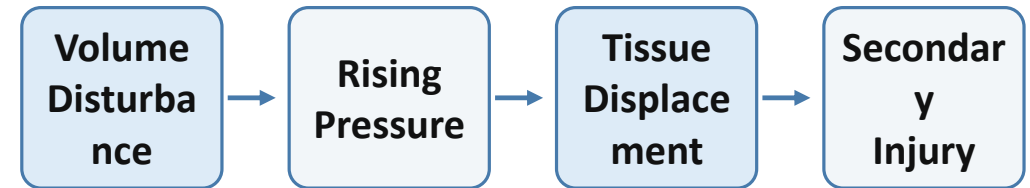
Integrating the disorders: Compartment predicts morphology

Primary volume change	Typical morphology	Mechanical consequence
Parenchymal edema	Flattened gyri, narrowed sulci, compressed ventricles	Raised pressure; displacement
Hydrocephalus	Enlarged ventricular cavities	Raised pressure when volume increases
Focal mass lesion	Asymmetric mass effect	Regional shift; vascular injury

The essential neuropathologic causal chain

- Cerebral edema and hydrocephalus are distinct volume disorders.
- Added volume may raise pressure within a rigid cranial compartment.
- Pressure and displacement cause vascular compression, ischemia, and herniation.
- The specific herniation route predicts focal neurologic consequences.

Schematic



Selected source and figure provenance

- Primary text: Robbins, Cotran & Kumar, Pathologic Basis of Disease, 11th edition (2026), Chapter 28.
- Teaching scope: Cerebral Edema; Hydrocephalus; Raised Intracranial Pressure and Herniation (pp. 1132–1133).
- All schematics are original editable vector constructions; they are not clinical or histologic specimens.

Schematic

Clinical / morphologic clues



Infer the involved compartment



Predict the anatomic consequence