

Subdural Hematoma

Traumatic venous injury · morphology · clinicopathologic correlation

Central nervous system pathology

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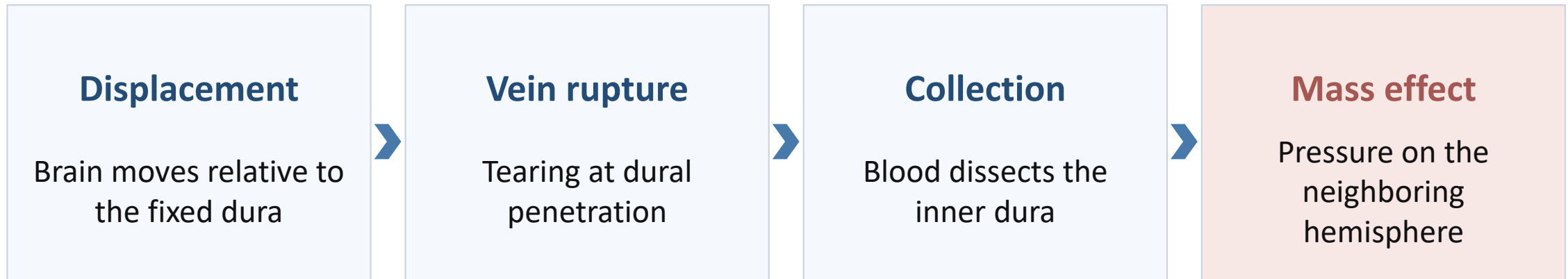
Learning objectives

Explain the mechanism and clinical importance of subdural bleeding.

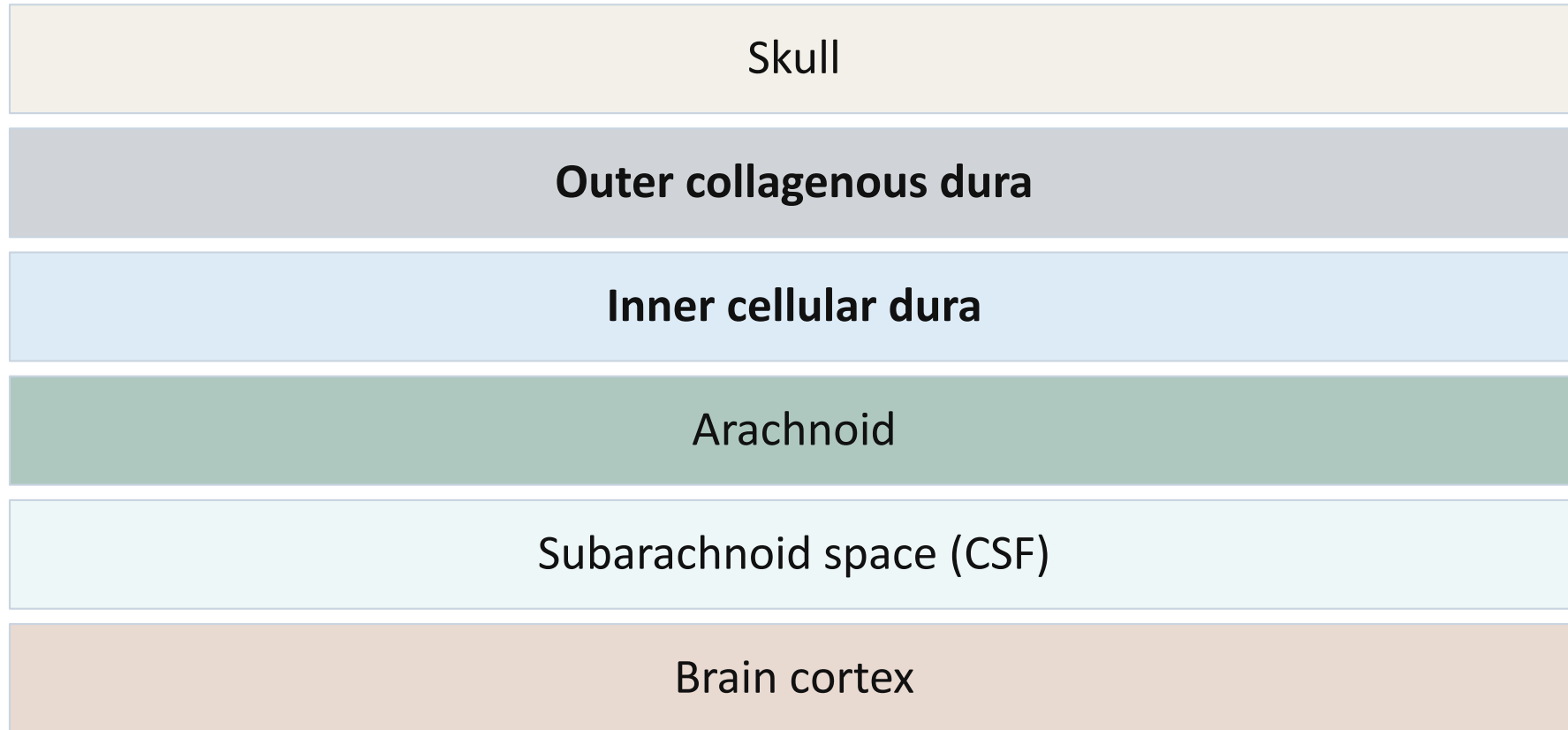
- Trace a bridging vein from cerebral surface to dural sinus.
- Describe acute gross appearance and sequential organization.
- Relate mass effect to symptoms and risk of repeat bleeding.
- Distinguish subdural from epidural hemorrhage.

A traumatic venous collection

Subdural hematoma is a consequence of disrupted bridging veins.



Meningeal compartments in context



Schematic — not a clinical specimen

The dura contains two distinct layers

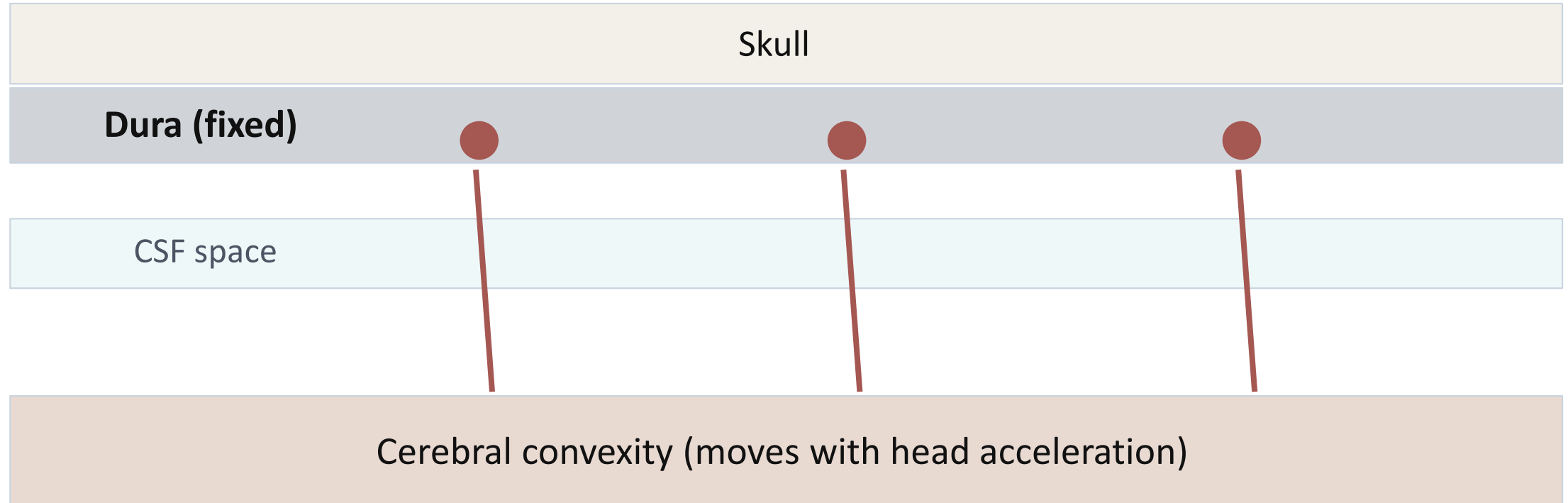
External layer

- Collagen-rich layer
- Firm association with inner skull
- Forms external dural component

Internal layer

- More cellular component
- Contains fibroblasts
- Participates in organization

The route of the bridging veins



Bridging veins traverse CSF and penetrate the dura

Veins link the cerebral convexity to the dural sinus

Schematic — not a clinical specimen

A mechanical mismatch makes veins vulnerable

Brain

- Suspended in cerebrospinal fluid
- Can be displaced during trauma
- Moves relative to skull and dura

Venous sinus / dura

- Fixed relative to cranial dura
- Anchors the termination of the veins
- Limits motion at the entry point

How traumatic displacement tears a vein

Fixed dura and venous sinus

Bridging vein tear

Brain displaced in CSF

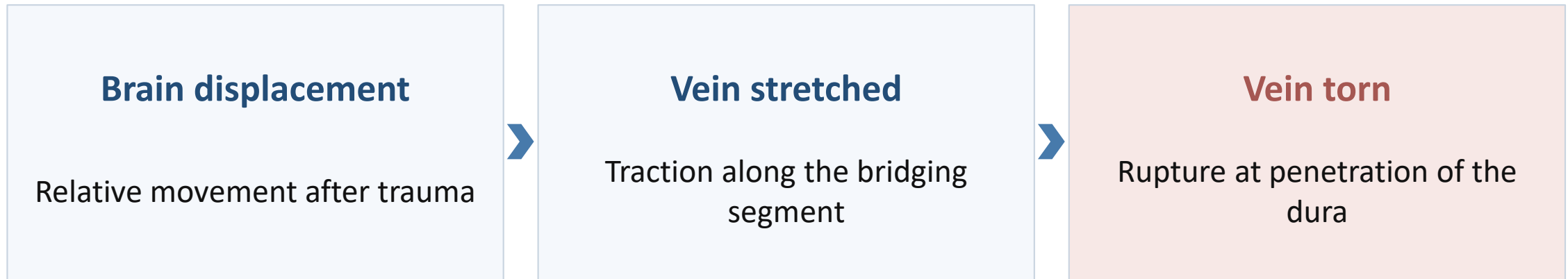


Relative movement creates traction on the traversing vein

Schematic — not a clinical specimen

The dural penetration is the weak point

Rupture occurs where a mobile venous segment enters fixed dural tissue.



Blood dissects into the dural border

The text describes blood tracking through the two dural layers.

Venous rupture

Blood leaves injured bridging
vein



Dissection

Blood opens a dural-border
tissue plane



Hematoma

Collection expands along the
surface

Classic location versus tissue-plane mechanism

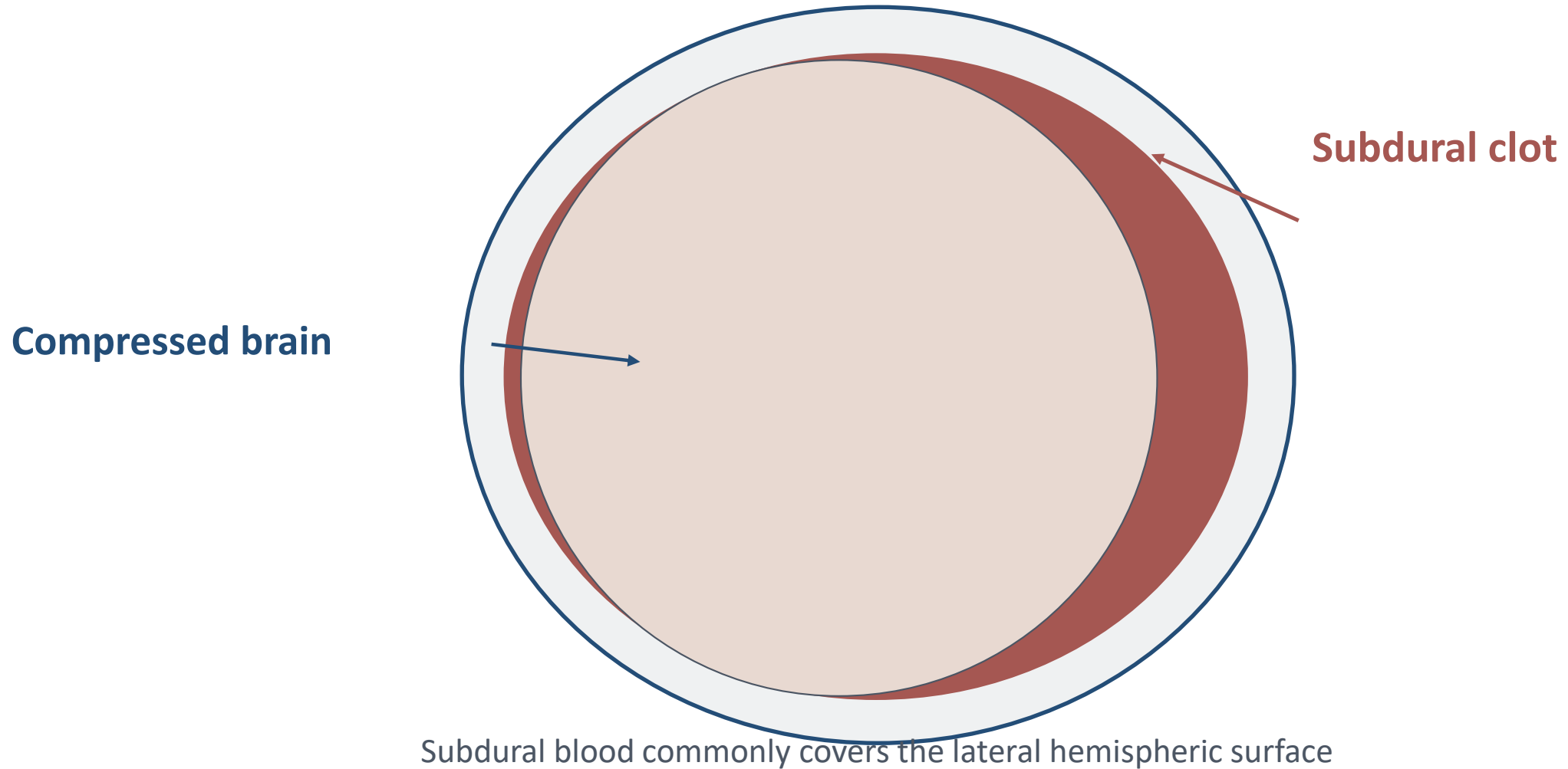
Anatomic shorthand

- Subdural: Between dura and arachnoid
- Helpful for categorizing hemorrhage
- Used in the accompanying figure caption

Pathologic description

- Inner dura is cellular and cleavable
- Blood dissects through dural layers
- The collection need not occupy a preformed space

Typical location: Cerebral convexities



Schematic — not a clinical specimen

Bilateral collections are possible

Bilateral subdural hematomas in about 10% of cases.

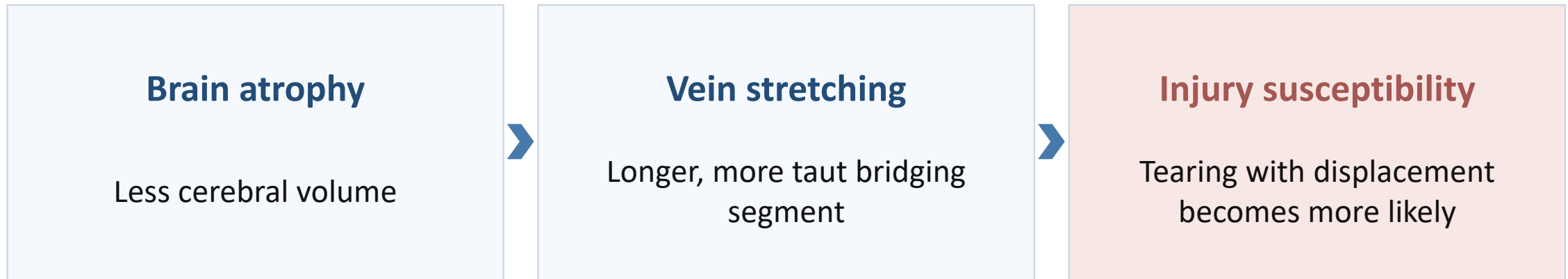
- A lateral hemispheric collection is the common pattern.
- Bilateral involvement is less common but explicitly recognized.
- Do not assume all subdural collections are unilateral.

Susceptibility reflects vascular and brain anatomy

Context	Structural explanation	Consequence
Older age	Cerebral atrophy stretches veins	Greater susceptibility
Infancy	Bridging veins are thin-walled	Greater susceptibility
Coagulopathy	Bleeding tendency after injury	Even minor trauma may suffice

Why cerebral atrophy raises vulnerability

Age-associated volume loss changes mechanical strain on the veins.



Infancy: A different structural vulnerability

Older individuals

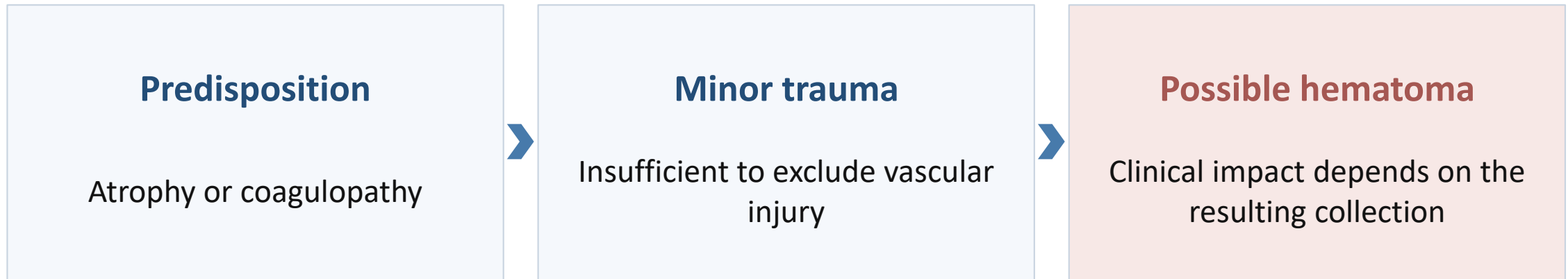
- Cerebral atrophy is emphasized
- Stretched bridging veins
- Trauma may be minor

Infants

- Bridging veins are thin-walled
- Particular susceptibility to tearing
- Same venous bleeding mechanism

Minor trauma can still produce hemorrhage

Predisposing conditions alter the consequences of mechanical injury.



Anatomy explains why mechanism matters

Head trauma is not a single uniform vascular insult.

- Ruptured bridging veins produce subdural blood.
- Lacerated meningeal arteries produce epidural blood.
- The injured vessel determines the initial collection compartment.
- Both lesions may compress the brain.

The epidural comparison: Vessel origin

Subdural

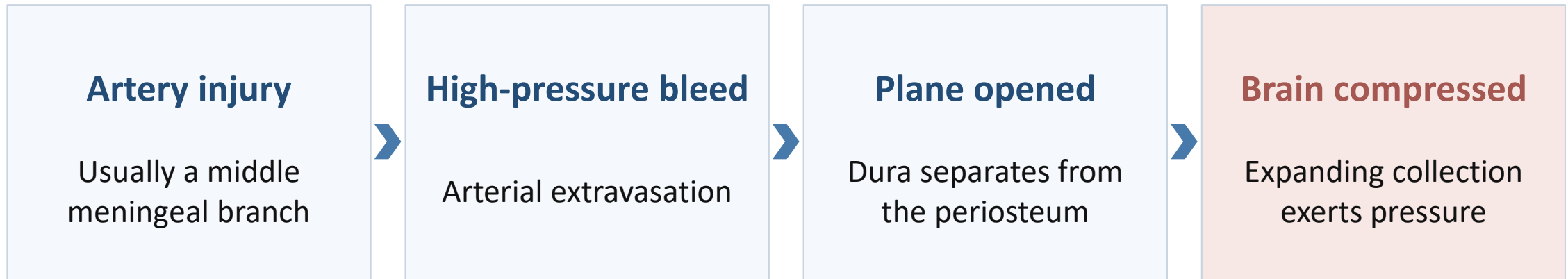
- Bridging veins are disrupted
- Venous blood forms the collection
- Traction at dura entry is central

Epidural

- Meningeal artery is disrupted
- Arterial blood separates dura from skull
- Temporal fracture often injures the middle meningeal artery

An epidural hematoma is a different plane

Arterial blood lifting the dura away from the skull.



Why the temporal skull is relevant to epidural bleeding

The middle meningeal artery lies in a vulnerable dural course.

- Temporal fractures may cross the vessel.
- An arterial tear can occur at that site.
- In children, skull deformation may tear a vessel without fracture.
- These epidural mechanisms should not be assigned to bridging-vein rupture.

Different vessels, different pathogenesis

Subdural mechanism

- Traction causes a bridging-vein tear
- Collection tracks along inner dura
- Venous bleeding commonly limits itself

Epidural mechanism

- Meningeal arterial rupture
- Blood displaces dura from skull
- Expanding arterial collection can evolve rapidly

Both lesions may become neurologic emergencies

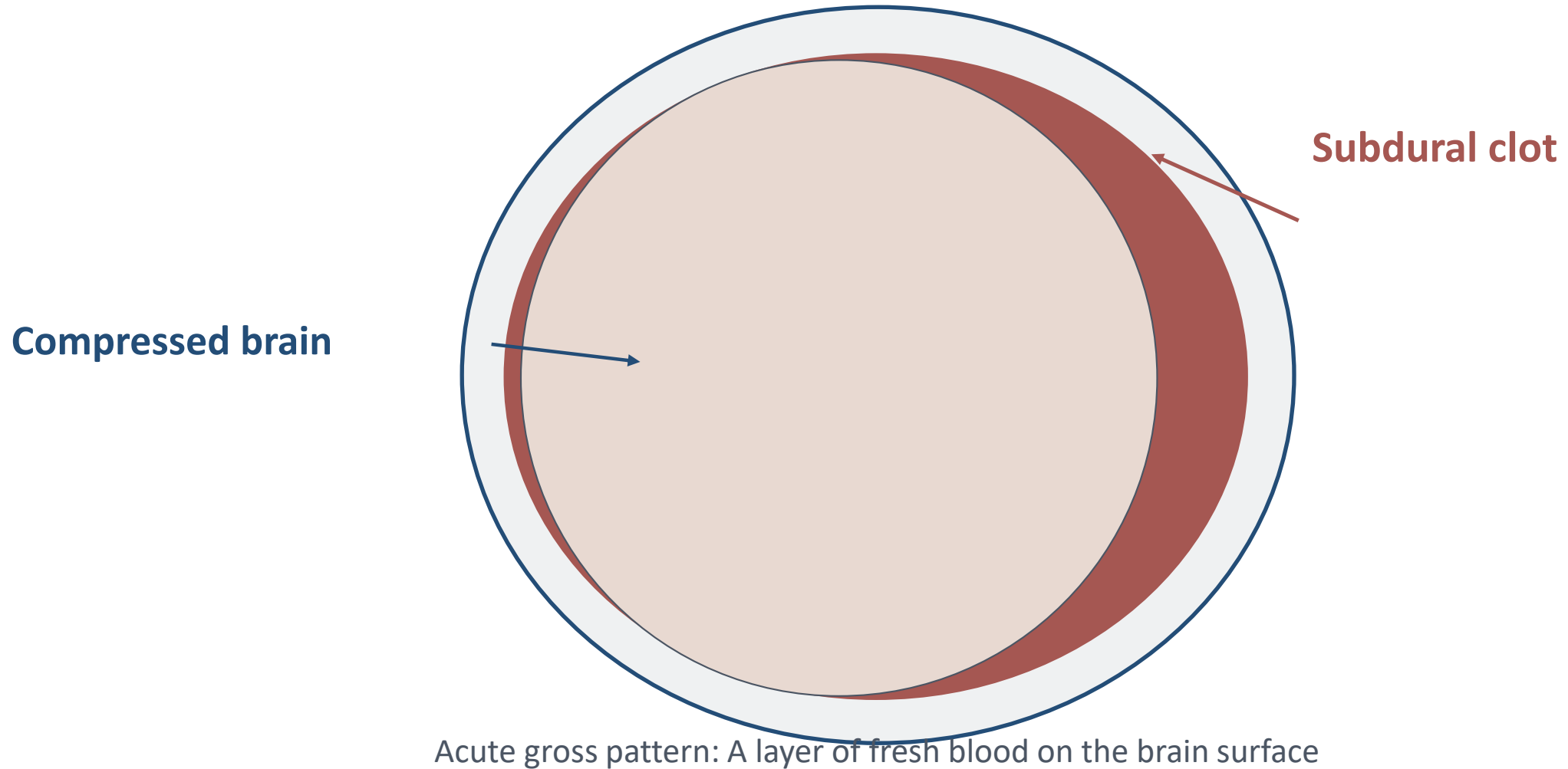
The shared risk is pressure on the intracranial contents.

- Epidural blood can expand rapidly under arterial pressure.
- Subdural blood may cause progressive deterioration.
- Acute decompensation can occur in subdural hematoma.
- Clinical course must be interpreted in its vascular context.

Hemorrhage compartments: Keep the terms distinct

Compartment	Anatomic description	Context from Robbins
Epidural	Skull–dura interface	Meningeal artery injury
Subdural	Inner dural border / arachnoid interface	Bridging-vein injury
Subarachnoid	CSF-containing space around brain	May accompany trauma
Intraparenchymal	Within brain tissue	Parenchymal vascular injury

Fresh subdural blood lies along the surface



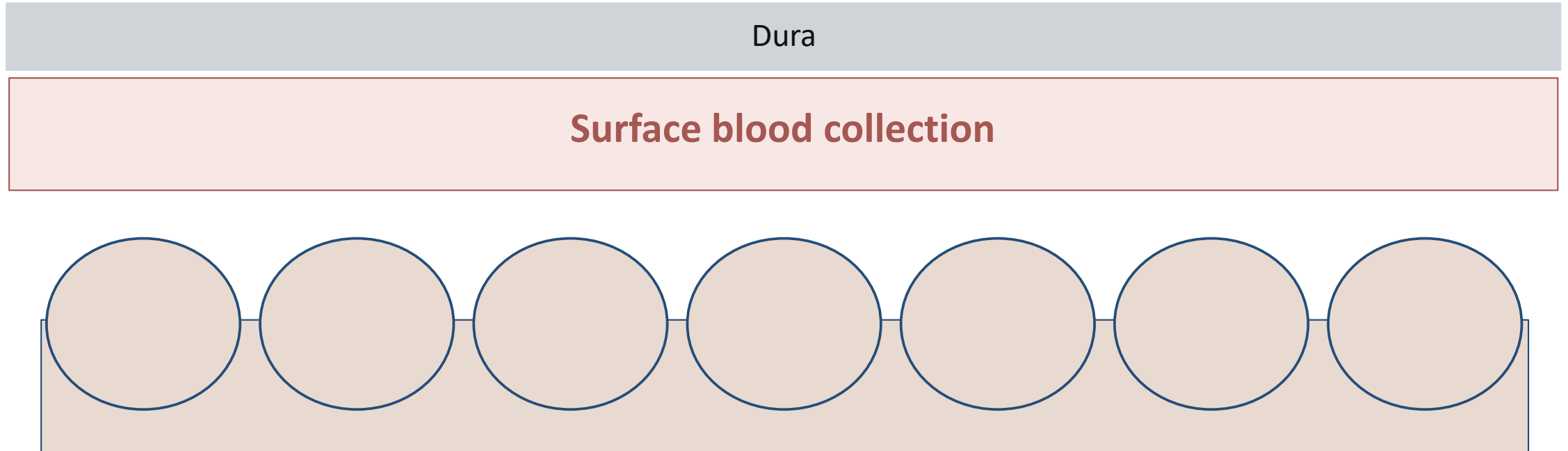
Schematic — not a clinical specimen

Acute hematoma: Freshly clotted blood

The acute specimen is a surface collection of recently extravasated blood.

- The blood is freshly clotted in the acute stage.
- It follows the surface rather than penetrating brain parenchyma.
- Its location is assessed relative to the meninges and sulci.

Blood does not extend into the depths of sulci



The blood remains above the cortical folds; Sulcal depths are not filled

This gross feature helps localize the hemorrhage

Schematic — not a clinical specimen

Surface blood differs from sulcal blood

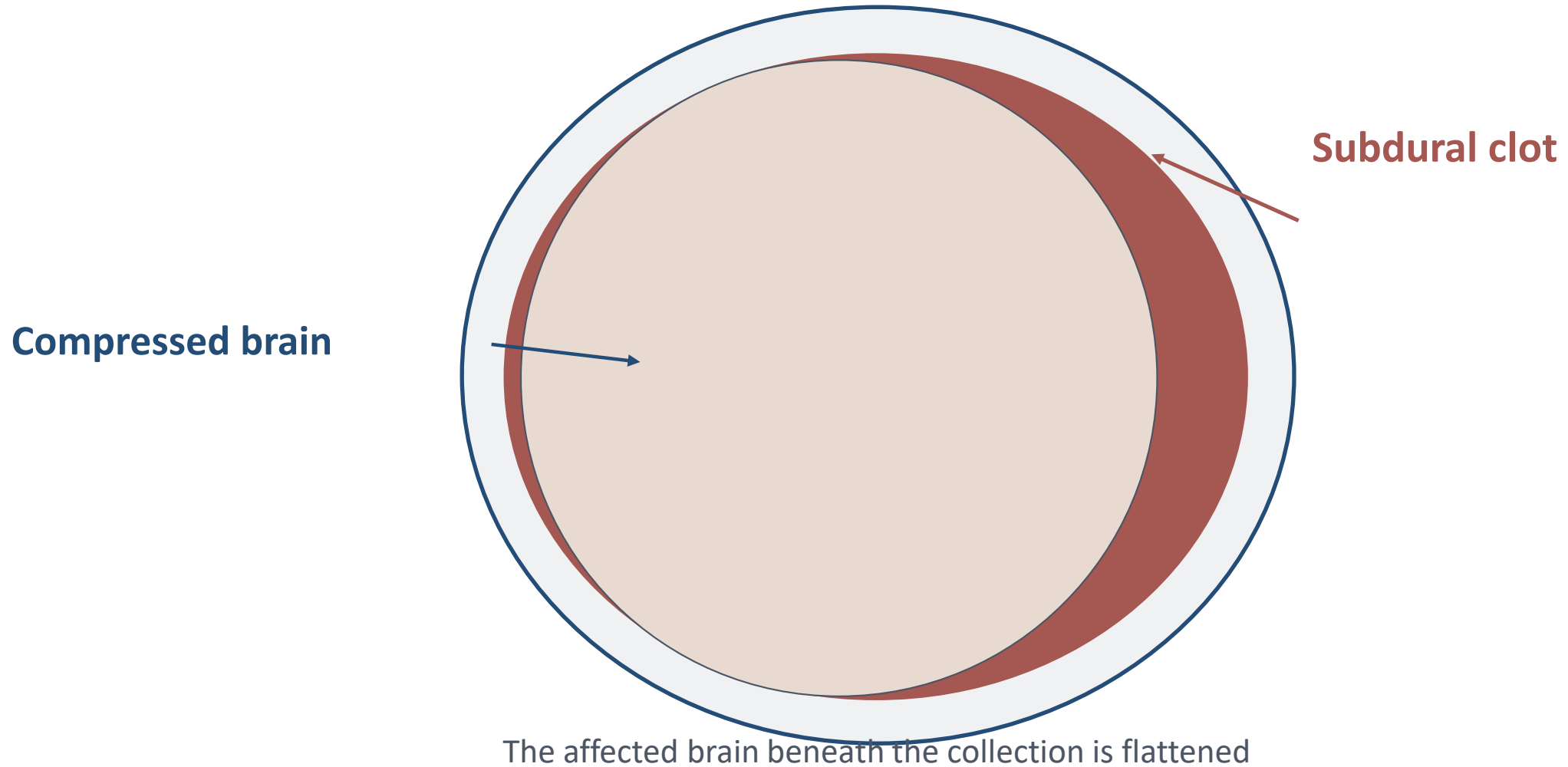
Subdural morphology

- Clot runs over the cerebral surface
- Does not descend into sulcal depths
- Subarachnoid space often appears clear

Subarachnoid compartment

- Anatomically follows the sulcal spaces
- May contain blood in other hemorrhagic conditions
- Do not assign sulcal blood to subdural clot

Compression alters underlying brain contours



Schematic — not a clinical specimen

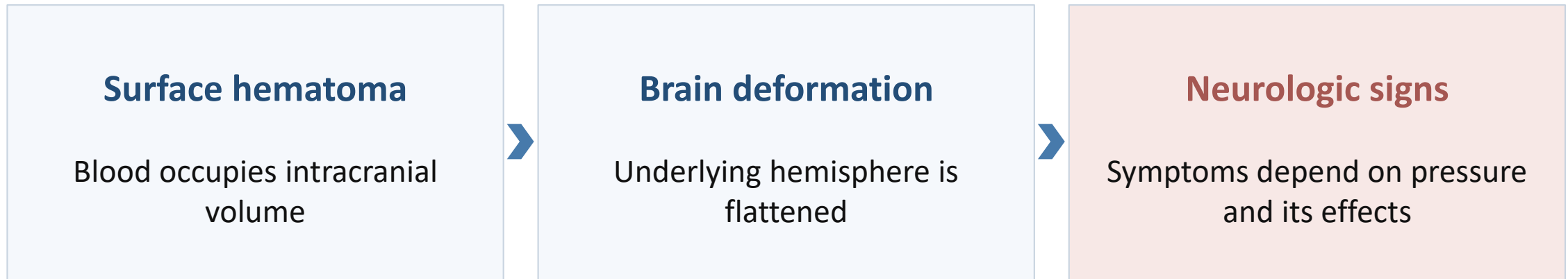
A clear subarachnoid space supports localization

The underlying subarachnoid compartment is often clear.

- A surface clot may overlie a relatively clear arachnoid/CSF compartment.
- This observation follows from the location of the dural-border collection.
- The distinction is gross and anatomic, not a marker of severity.

Mass effect links morphology to symptoms

Neurologic findings arise from pressure exerted on the adjacent brain.



Clinical manifestations can be focal

The collection exerts a local mechanical effect on nearby neural tissue.

- Robbins acknowledges focal neurologic signs.
- Focality is compatible with a local compressive lesion.
- The excerpt does not require a particular focal deficit for diagnosis.

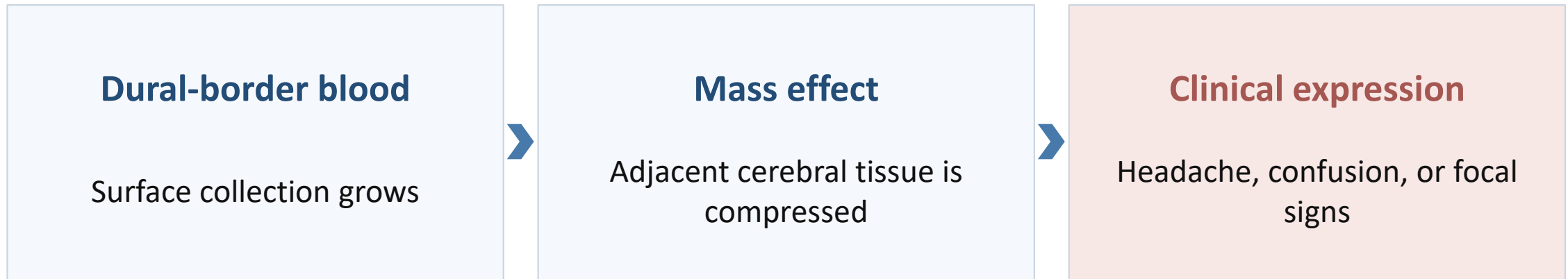
Clinical manifestations are often nonlocalizing

A localized collection does not always produce a localizing examination.

- Headache is a prominent nonlocalizing manifestation.
- Confusion is another explicitly described presentation.
- A nonfocal examination does not negate the presence of a collection.

Why headache and confusion fit the pathology

A teaching interpretation of the pressure–symptom relationship.



Symptoms commonly appear within 48 hours

Time course stated by Robbins; Not a mandatory diagnostic cutoff.

Trauma

Venous injury may begin a surface collection

Within 48 h

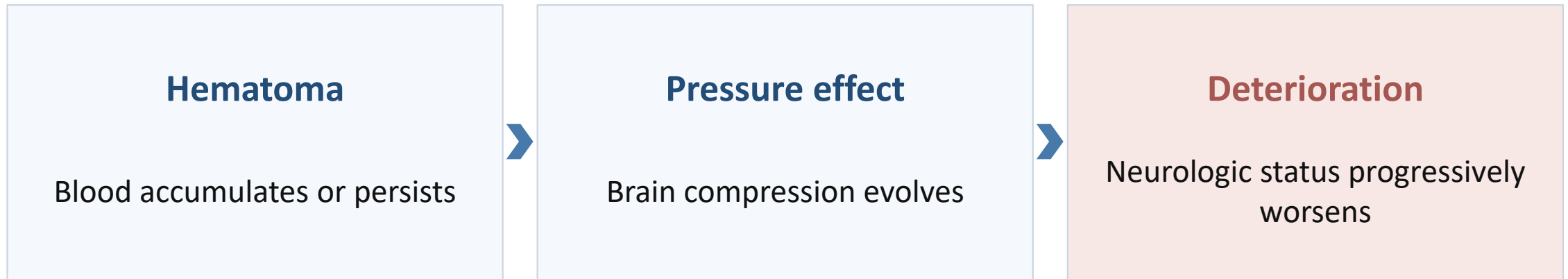
Symptomatic cases typically declare themselves

Later course

Deterioration may progress or abruptly worsen

A progressive course is characteristic

Slowly evolving neurologic deterioration.



Acute deterioration remains possible

Typical progression

- Clinical change can be gradual
- Manifestations may be nonlocalizing
- Progressive neurologic decline

Acute decompensation

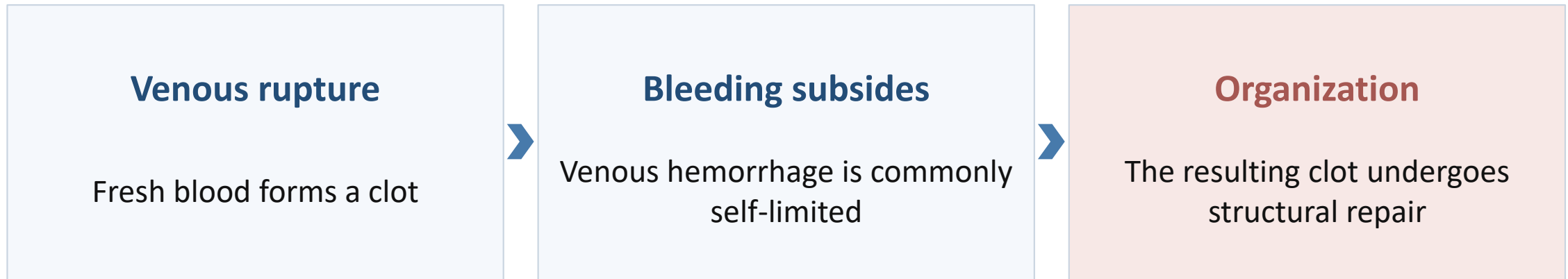
- Sudden worsening can occur
- Does not exclude a subdural lesion
- Reflects potential severity of intracranial pressure effects

Timing and severity are separate dimensions

Question	Textbook-supported answer
When do symptoms usually begin?	Typically within 48 hours
How can neurologic signs evolve?	Often progressively
Can sudden worsening occur?	Yes, acute decompensation is described
Are focal signs obligatory?	No; nonlocalizing symptoms may predominate

Venous bleeding often becomes self-limited

The usual natural history is organization rather than indefinite active bleeding.



Organization follows a characteristic sequence

Approximate time intervals from the textbook, not exact staging criteria.

~1 week

Lysis of the clot

~2 weeks

Fibroblasts extend from the
dural surface

1–3 months

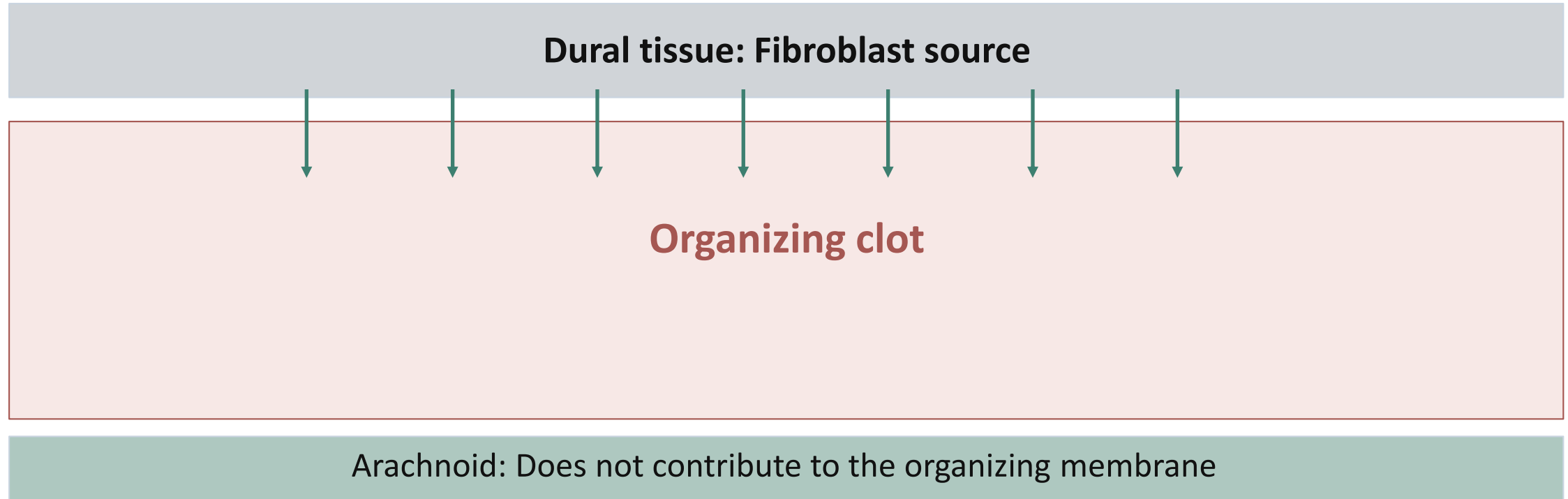
Early hyalinized connective
tissue develops

Clot lysis begins the reparative phase

Lysis of the clot at approximately one week.

- Fresh blood is altered before a mature fibrous membrane develops.
- The reported interval is approximate, not a clock for individual cases.
- The next stage involves fibroblast ingrowth from the dura.

Fibroblasts grow from the dural surface



Fibrous tissue grows from the dural surface into the collection

Early connective-tissue organization is dural in origin

Schematic — not a clinical specimen

Fibroblast ingrowth appears near two weeks

The dura contributes the cellular reparative response.

- Fibroblasts extend from the dural surface into the hematoma.
- This creates an increasingly organized collection.
- The arachnoid does not provide the organizing fibroblasts.

Hyalinized connective tissue develops later

Early

Freshly clotted blood

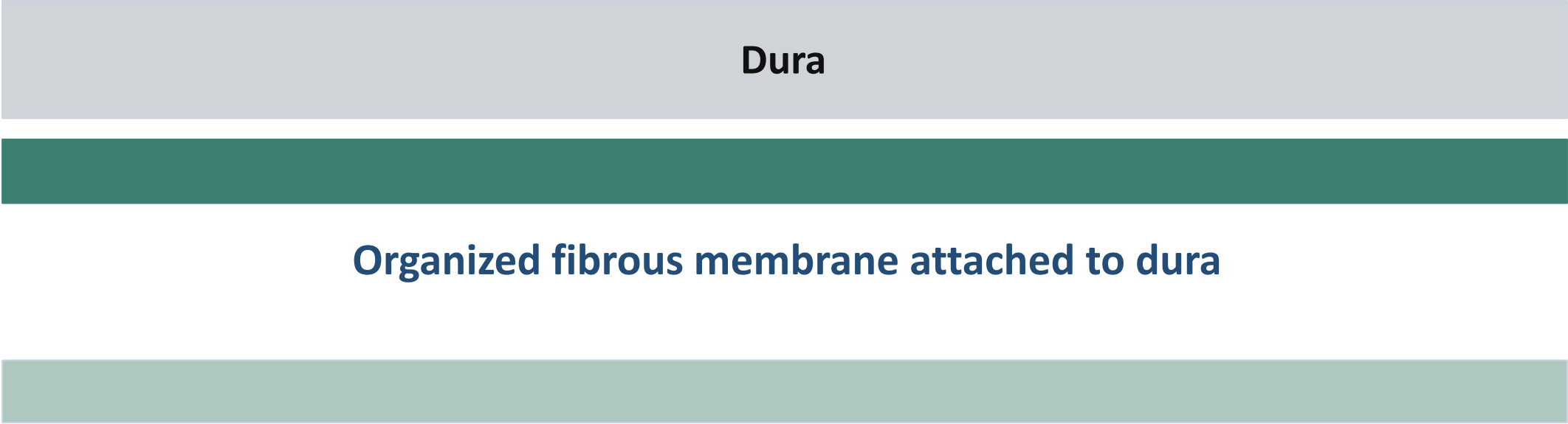
Interim

Clot lysis and fibroblast
ingrowth

1–3 months

Early hyalinized connective
tissue

The organizing hematoma is dura-attached



The diagram consists of three horizontal bars. The top bar is light gray and labeled 'Dura'. The middle bar is dark green and labeled 'Organized fibrous membrane attached to dura'. The bottom bar is light green and labeled 'Arachnoid remains separate'.

Dura

Organized fibrous membrane attached to dura

Arachnoid remains separate

A thin reactive membrane may remain after the hematoma retracts

Organization secures the lesion to the inner dura

Schematic — not a clinical specimen

The arachnoid does not participate in healing

Dura

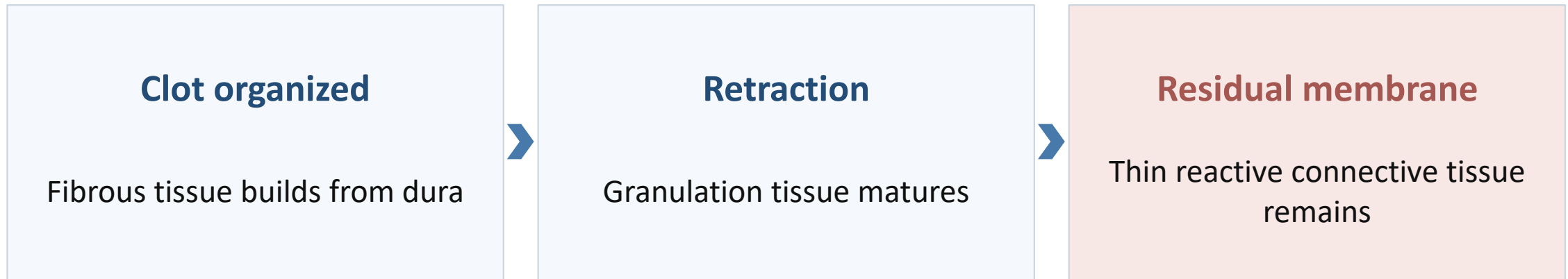
- Provides ingrowing fibroblasts
- Anchors the organized collection
- Forms the attached fibrous membrane

Arachnoid

- Remains free of the organized lesion
- Does not contribute to repair
- Provides a different anatomic boundary

A mature membrane can remain after retraction

The organized hematoma may contract with connective-tissue maturation.



Two later courses: Resolution or recurrent bleeding

Organizing / resolving

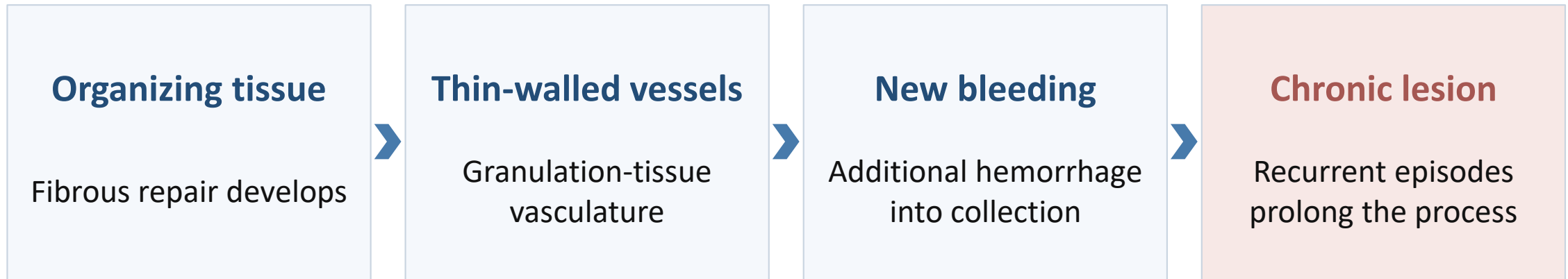
- Clot lysis and fibroblast ingrowth
- Fibrous tissue matures and retracts
- Thin subdural membrane may persist

Recurrent / chronic

- Thin-walled granulation vessels persist
- Bleeding episodes can recur
- Chronic subdural hematoma develops

Fragile granulation vessels enable rebleeding

Chronic recurrences to thin-walled vessels in granulation tissue.



Chronic subdural hematoma is a dynamic lesion

Organization does not guarantee permanent cessation of bleeding.

- Repeated bleeding can occur during attempted repair.
- The suspected source is fragile granulation-tissue vasculature.
- The chronic collection therefore reflects both blood and organized tissue.

The recurrence window extends for months

Risk of repeat bleeding is greatest in the first few months.

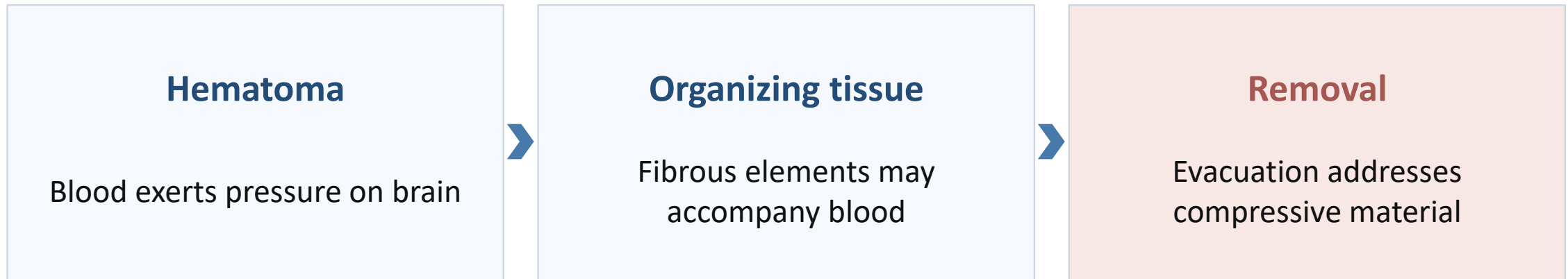
- The period overlaps the formation of organizing connective tissue.
- Thin-walled repair vessels provide a plausible bleeding source.
- The time statement is descriptive, not an individual risk estimate.

Acute and chronic subdural lesions differ

Feature	Acute	Organizing / chronic
Dominant material	Fresh clotted blood	Clot plus fibrovascular organization
Gross relation	Surface collection	Fibrous attachment to inner dura
Potential fate	Clot breakdown	Retraction or repeat hemorrhage
Mechanistic source	Bridging vein tear	May involve granulation-tissue vessels

Management addresses blood and organizing tissue

Treatment involves removal of the collection and associated organizing material.



Why tissue organization is relevant to treatment

A chronic collection is not merely a pocket of fresh blood.

- The inner dural surface becomes attached to organized tissue.
- Persistent membranes may remain after the clot retracts.
- Robbins explicitly includes associated organizing tissue in removal.
- Detailed operative techniques are outside the supplied excerpt.

Case: Older adult after a minor injury

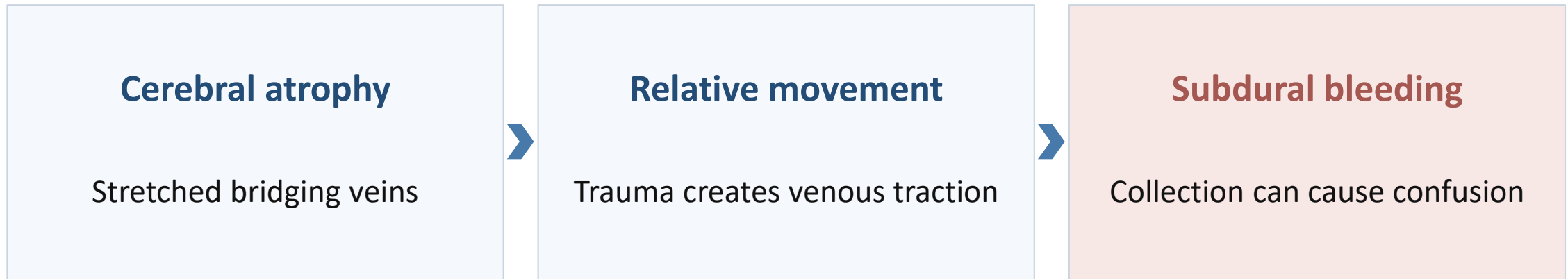
Teaching vignette

An older person with cerebral atrophy develops confusion after a seemingly minor head injury. The examination is not strongly localizing.

Which anatomic feature makes subdural hemorrhage plausible?

Case reasoning: Cerebral atrophy stretches veins

The case does not require major trauma to make the mechanism plausible.



Case: Infant with traumatic head displacement

Teaching vignette

An infant develops a subdural collection following head trauma. The mechanism is assessed without assuming the adult atrophy pattern.

What age-specific feature does Robbins emphasize?

Case reasoning: Thin-walled bridging veins

The same venous pathway is vulnerable for a different reason.

- Infant bridging veins are relatively thin-walled.
- Traumatic displacement can still tear the veins.
- The resulting blood accumulates at the dural border.
- No unique injury mechanism can be inferred from this feature alone.

Case: Gradual symptoms followed by sudden decline

Teaching vignette

A patient has headache and progressive confusion after trauma, then acutely deteriorates.

Does acute decompensation rule out a subdural hematoma?

Case reasoning: Clinical tempo varies

Compatible with subdural hematoma

- Progressive deterioration is common
- Headache and confusion may predominate
- Acute worsening is explicitly described

Incorrect inference

- Only focal deficits are allowed
- Only one progression rate is possible
- Sudden worsening excludes a subdural collection

Case: Recurrent symptoms after prior hematoma

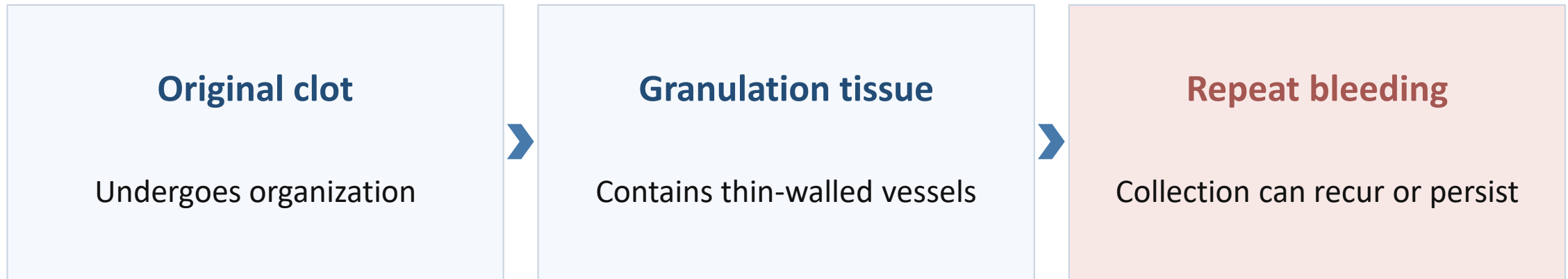
Teaching vignette

Following an initially organizing subdural hematoma, a patient develops renewed neurologic symptoms within the first months.

What tissue component may be responsible for repeat bleeding?

Case reasoning: Granulation-tissue vessels

The source links recurrent episodes to newly formed fragile vessels.

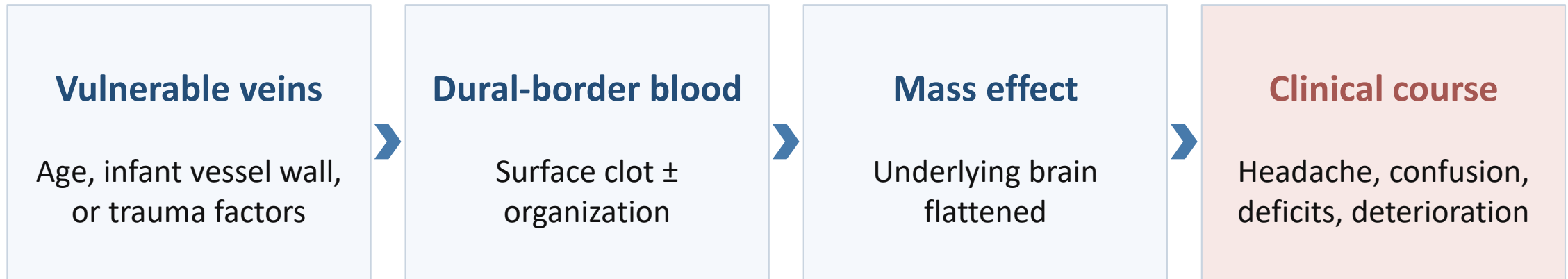


Integrated morphology review

Time / setting	Characteristic finding	Teaching implication
Acute	Fresh clot over hemisphere; sulci spared	Localize the bleed
~1 week	Clot lysis	Repair begins
~2 weeks	Dural fibroblast ingrowth	Organization is dura-derived
1–3 months	Early hyalinized connective tissue	Membrane maturation
Chronic/recurrent	Fragile granulation vessels	Further hemorrhage possible

Clinical-pathologic synthesis

One continuous sequence links anatomy, injury, morphology, and symptoms.



Check your understanding

What vessel usually produces a traumatic subdural hematoma?

Bridging vein

Does fresh subdural clot fill sulcal depths?

Usually no

Where do organizing fibroblasts originate?

Dural surface

What favors recurrent chronic bleeding?

Fragile vessels

Principal references and image status

Primary scientific content is based on the supplied Robbins source.

- Robbins, Cotran & Kumar: Pathologic Basis of Disease, 11th ed. (2026).
- Chapter 28: The Central Nervous System — pp. 1145–1146.
- Relevant parent context: traumatic vascular injury and epidural hematoma.
- All shown diagrams are original, editable educational schematics; no textbook figures are reproduced.