

Acute Pyogenic (Bacterial) Meningitis

Etiology • CSF pathology • Morphology • Complications

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Learning outcomes

- Localize bacterial meningitis to the leptomeninges and CSF-containing subarachnoid space.
- Relate age-associated pathogens to presentation and characteristic CSF changes.
- Recognize gross and microscopic patterns and explain major neurologic and systemic complications.

Meningitis: An anatomic definition

- Inflammation involves the leptomeninges and the CSF within the subarachnoid space.
- Infection is the usual cause, but chemically induced and autoimmune reactions can produce meningitis.
- Anatomic localization is essential for interpreting exudate and CSF findings.

Anatomic substrate: Leptomeninges and CSF

Dura

Arachnoid

Subarachnoid space / CSF

Pia

Underlying brain parenchyma

The affected compartment lies between arachnoid and pia.

Schematic

Meningitis and meningoencephalitis are not synonyms

Meningitis

Leptomeninges + CSF
Primarily meningeal inflammation
Pyogenic disease may remain surface-limited

Meningoencephalitis

Meninges + brain parenchyma
Meningoencephalitis is most commonly viral
Brain tissue involvement is part of the definition

Fulminant bacterial meningitis can extend focally into parenchyma (cerebritis).

How CNS infections produce tissue injury

Direct injury

Pathogen-mediated injury to neurons or glia
Microbial products and toxic effects

Indirect injury

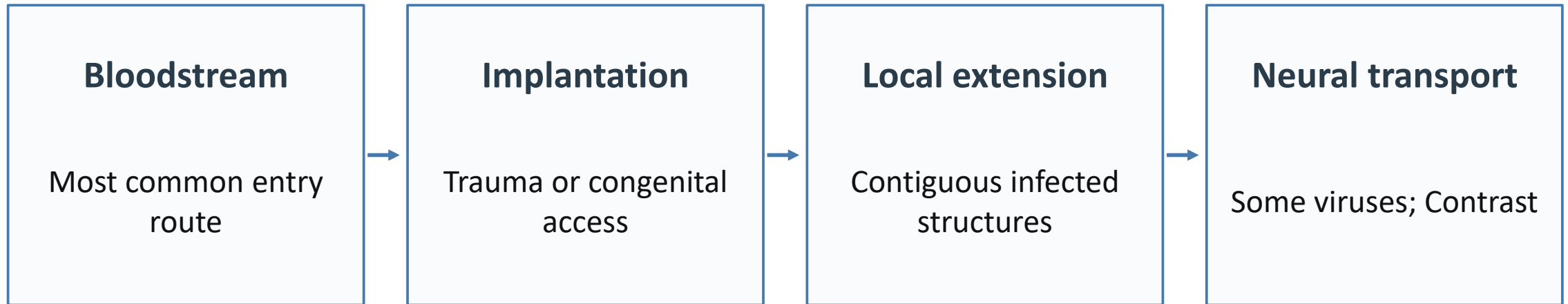
Destructive host inflammatory response
Immune-mediated effects

An effective host response can nevertheless damage vulnerable CNS structures.

Major infectious meningitis patterns

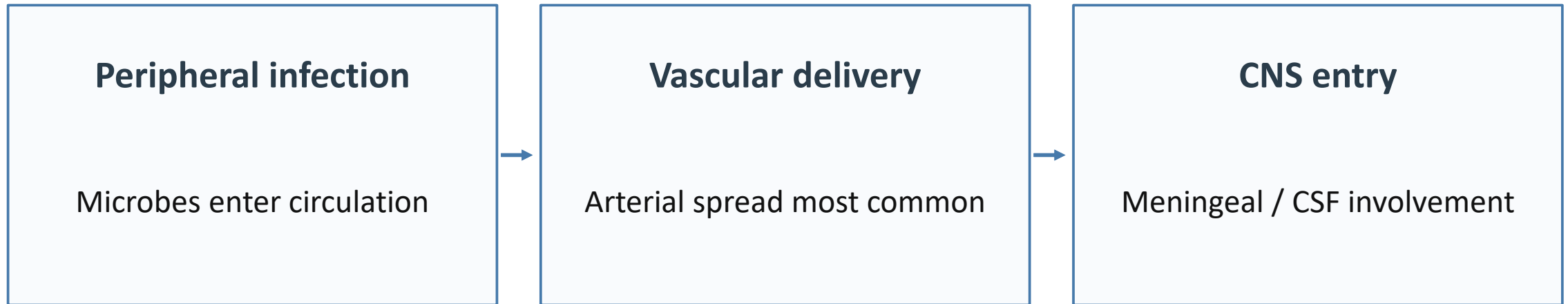
Pattern	Typical etiologic category	Distinguishing framing in Robbins
Acute pyogenic	Usually bacterial	Acute, purulent meningeal inflammation
Aseptic	Usually acute/subacute viral	Culture-negative clinical syndrome
Chronic	Tuberculous, spirochetal, cryptococcal	Chronic evolution

Routes by which agents reach the CNS



Different portals converge on meningeal or parenchymal compartments.

Hematogenous entry: The predominant route



Retrograde venous spread may occur through connections with facial veins.

Direct implantation bypasses protective barriers

- Head trauma can introduce microorganisms directly into CNS compartments.
- Congenital defects, including meningocele, may provide abnormal access.
- The route of entry differs from the hematogenous mechanism.

Contiguous spread from neighboring infection

- Infection may extend from paranasal air sinuses, teeth, skull, or vertebrae.
- Local anatomy can determine where infection first reaches the CNS.
- Direct extension is distinct from circulating bacteremia.

Transport along nerves: A contrast mechanism

Relevant to some viruses

Peripheral nervous system transport
Examples given: Rabies and herpes zoster

Acute pyogenic meningitis

Age-related bacterial organisms
Bloodstream and other non-neural routes
emphasized

The four CNS entry routes are not equally characteristic of every pathogen.

Age-associated organisms in acute pyogenic meningitis

Clinical group	Organisms highlighted
Neonates	Escherichia coli; group B streptococci
Adolescents / young adults	Neisseria meningitidis
Older adults	Streptococcus pneumoniae; Listeria monocytogenes

Neonates: Two important bacterial groups

Escherichia coli

Gram-negative enteric organism
Highlighted neonatal cause

Group B streptococci

Neonatal pathogen group
Also listed among infant causes

Age informs the differential; It does not by itself establish microbiologic identity.

Older adults: Pneumococcus and Listeria

Streptococcus pneumoniae

Highlighted organism in the elderly
Characteristic morphologic correlations later
in the lecture

Listeria monocytogenes

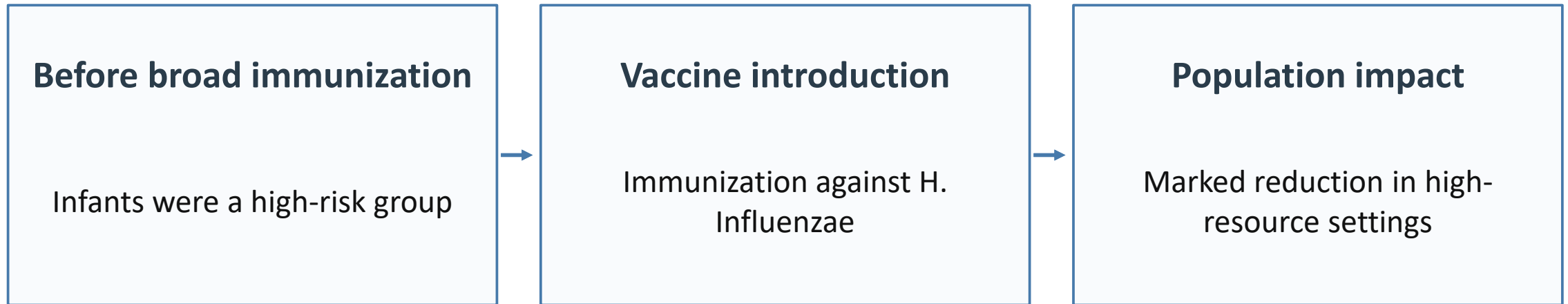
Also specifically associated with older adults
Must remain in the age-based differential

The age pattern is an educational association, not an exclusive host restriction.

Meningococcal meningitis in young adults

- *Neisseria meningitidis* is emphasized in adolescents and young adults.
- Clusters of meningococcal cases create significant public-health concerns.
- The same organism is important when assessing meningitis-associated septicemia.

Immunization changed the Haemophilus burden



The effect is substantial in high-resource settings; No numeric rates are shown.

Host immunosuppression changes the expected picture

Etiologic spectrum

Klebsiella and anaerobes may cause purulent meningitis
Organisms can extend beyond customary age patterns

Diagnostic presentation

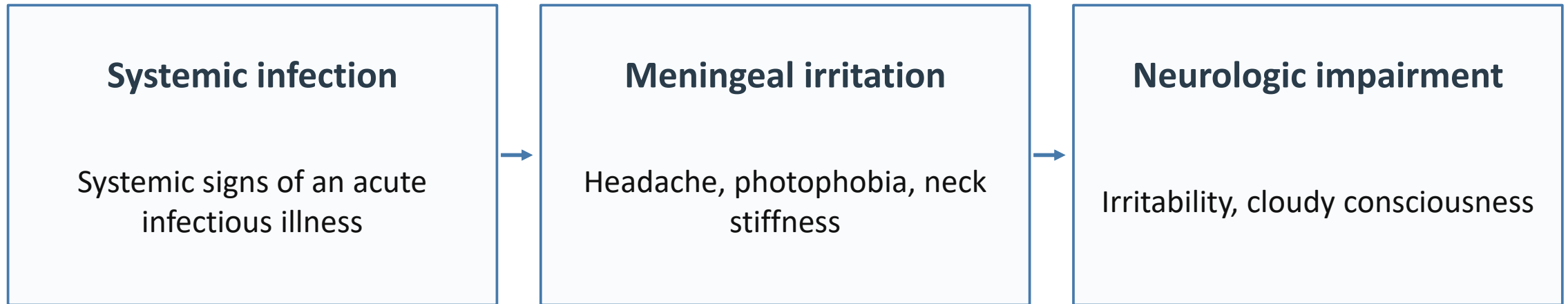
Course may be atypical
CSF findings may be uncharacteristic

Failure to recognize atypical disease may delay timely diagnosis.

Pathogen epidemiology is contextual, not determinative

- Age-associated organisms provide initial clinical context.
- Vaccination history and immunosuppression alter the expected organism spectrum.
- Clinical findings and CSF analysis remain central to assessing a suspected episode.

Two simultaneous clinical dimensions



A patient may exhibit findings from more than one dimension simultaneously.

Headache and photophobia signal meningeal irritation

- Headache is a common manifestation of the acute meningeal syndrome.
- Photophobia contributes to the constellation of meningeal irritation.
- The manifestations occur in a systemic infectious illness and must be interpreted together.

Neck stiffness: A characteristic meningeal symptom

- Neck stiffness is highlighted among manifestations of meningeal irritation.
- Its significance increases in the context of acute systemic illness and headache.
- It reflects meningeal involvement, not a direct measure of intracranial pus volume.

Neurologic dysfunction extends beyond meningeal pain

Behavioral / cognitive	Meningeal symptoms
Irritability Clouding of consciousness	Headache Photophobia Neck stiffness

Coexistence of these findings should raise concern for serious CNS infection.

Clinical-to-pathology correlation

- Leptomeningeal inflammation produces a syndrome of meningeal irritation.
- The illness also generates systemic signs of infection.
- Altered consciousness reflects neurologic impairment; The symptom alone does not prove parenchymal invasion.

Lumbar puncture evaluates the CSF compartment

- Lumbar puncture samples the CSF compartment involved in meningitis.
- CSF may appear cloudy or frankly purulent.
- The diagnostic pattern integrates cellular, chemical, and pressure findings.

CSF appearance and cells

Macroscopic appearance

Cloudy CSF
Frankly purulent fluid in severe cases

Cellular component

Numerous neutrophils
Neutrophilic pleocytosis is characteristic

The visible turbidity accords with the intense inflammatory cellular response.

Raised CSF pressure

- Increased CSF pressure is part of the typical textbook pattern.
- It accompanies inflammatory pathology within the craniospinal CSF compartment.
- Pressure must be interpreted with the other CSF and clinical abnormalities.

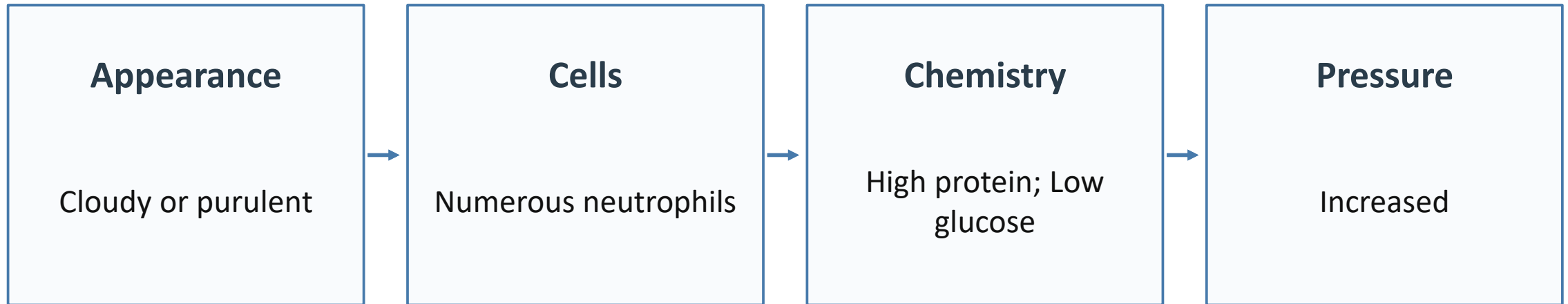
CSF protein rises in pyogenic meningitis

- Increased protein concentration is a characteristic laboratory finding.
- This change accompanies pronounced meningeal inflammation and exudation.
- Protein elevation alone is insufficient to identify a specific organism.

CSF glucose is markedly reduced

- Markedly diminished CSF glucose is highlighted in acute bacterial meningitis.
- This finding complements neutrophilic pleocytosis and increased protein.
- Interpret the entire CSF profile; One result alone does not identify an organism.

Integrated CSF signature



The constellation is more informative than any individual feature.

Pyogenic and viral/aseptic CSF patterns

Feature	Acute pyogenic (bacterial)	Typical aseptic (often viral)
Dominant cells	Neutrophils	Lymphocytes
Protein	Increased	Moderately increased
Glucose	Markedly decreased	Nearly always normal
Fluid appearance	Cloudy / purulent	Not specified in excerpt

Chemical meningitis can imitate infectious disease

Trigger	CSF profile
Chemical irritant in subarachnoid space Ruptured epidermoid cyst	Sterile fluid with neutrophils Increased protein; Glucose usually normal

Sterile neutrophilic CSF is not, by itself, proof of bacterial infection.

Recognize the seriousness of untreated infection

Without treatment

Acute pyogenic meningitis may be fatal
The illness can be fulminant

With effective antibiotics

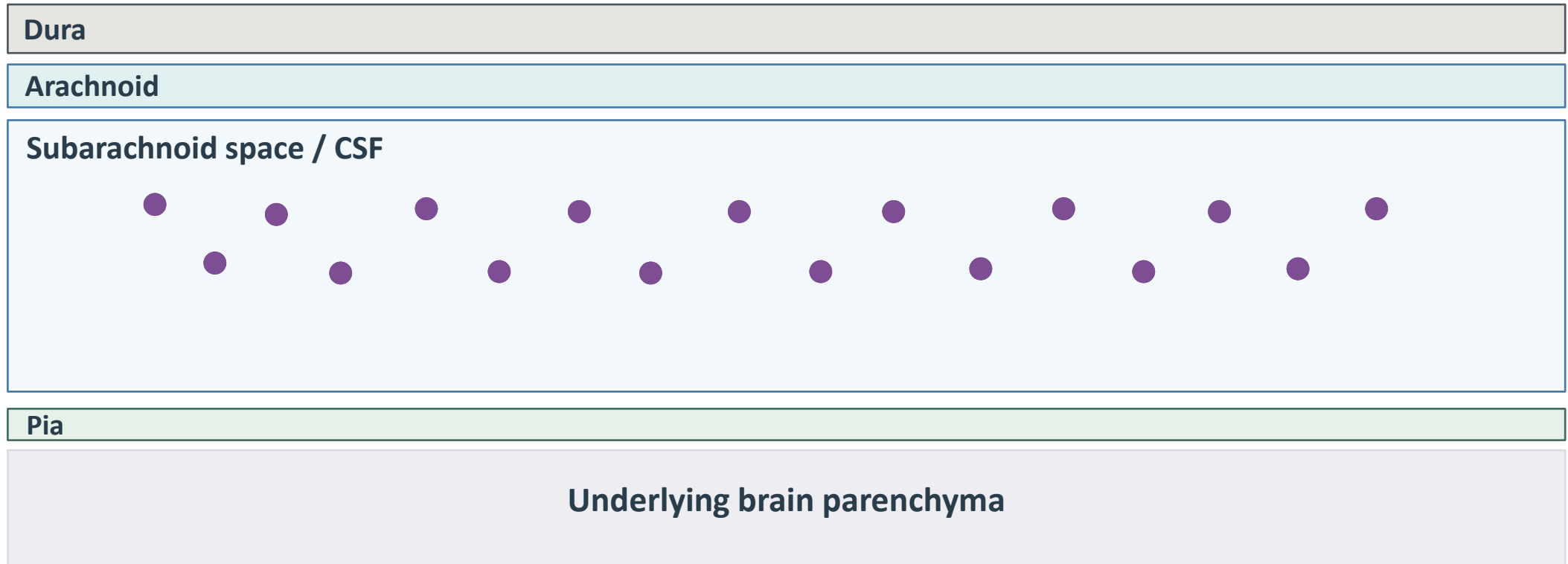
Mortality is sharply diminished
Treatment efficacy is emphasized by Robbins

Effective treatment markedly changes the clinical prognosis.

Gross hallmark: A suppurative meningeal exudate

- A thick purulent layer may cover the surface of the brain.
- The exudate occupies the leptomeninges and makes them appear thickened.
- The characteristic gross alteration is at the brain surface, not an intraparenchymal abscess.

Where the purulent exudate accumulates



Neutrophils occupy the CSF-containing subarachnoid space.

Schematic

Meningeal vessels become engorged

- Prominent, engorged meningeal vessels accompany the acute exudate.
- The vascular pattern can be conspicuous on the brain surface.
- Surface vascular involvement helps explain the later vascular complications.

Exudate is not uniformly distributed

- The amount and distribution of meningeal pus vary between cases.
- Some pathogens show characteristic regional tendencies.
- Regional patterns are descriptive gross observations—not stand-alone microbiologic tests.

H. Influenzae: Often a basal exudate

Qualitative gross-distribution tendency

Cerebral convexities near sagittal sinus

Superior surface of cerebral hemispheres

Basal leptomeninges

Brain-base meningeal compartment

H. Influenzae: Exudate is often basal.

Pneumococcal meningitis: Convexity accentuation

Qualitative gross-distribution tendency

Cerebral convexities near sagittal sinus

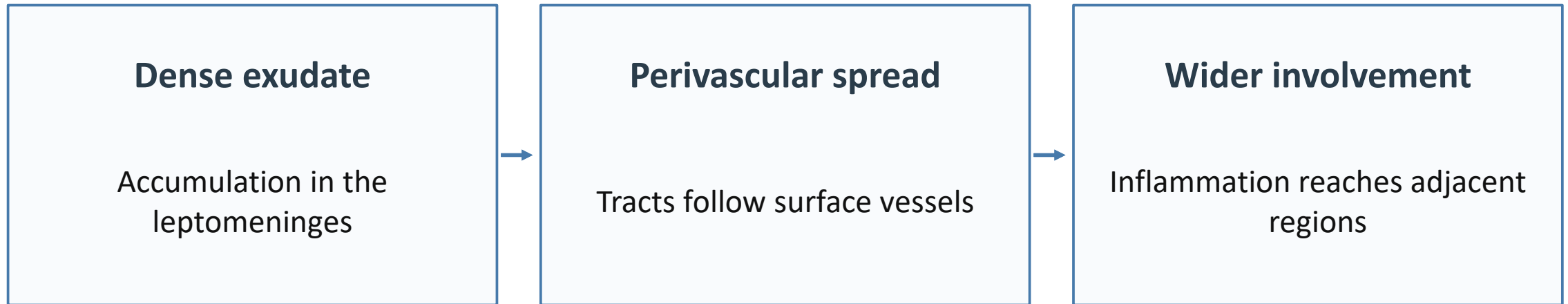
Superior surface of cerebral hemispheres

Basal leptomeninges

Brain-base meningeal compartment

S. Pneumoniae: Exudate may be densest over cerebral convexities.

Pus can extend along surface vessels



The anatomic course follows meningeal vascular structures.

Histologic intensity changes the distribution of cells

Severely involved region

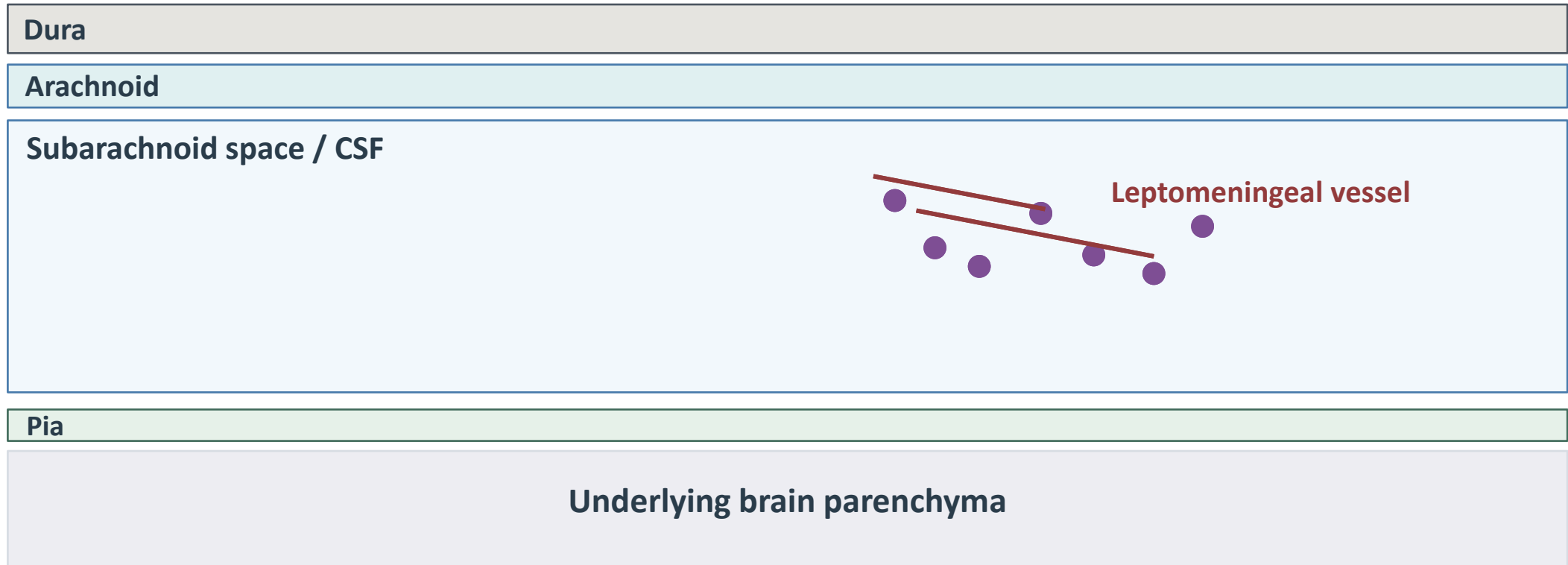
Subarachnoid space filled by neutrophils
Dense suppurative infiltrate

Less involved region

Neutrophils concentrated around vessels
Perivascular inflammatory pattern

These are degrees of the same meningeal inflammatory process.

Microscopy concept: Perivascular neutrophils



Inflammation may cluster around leptomeningeal blood vessels.

Schematic

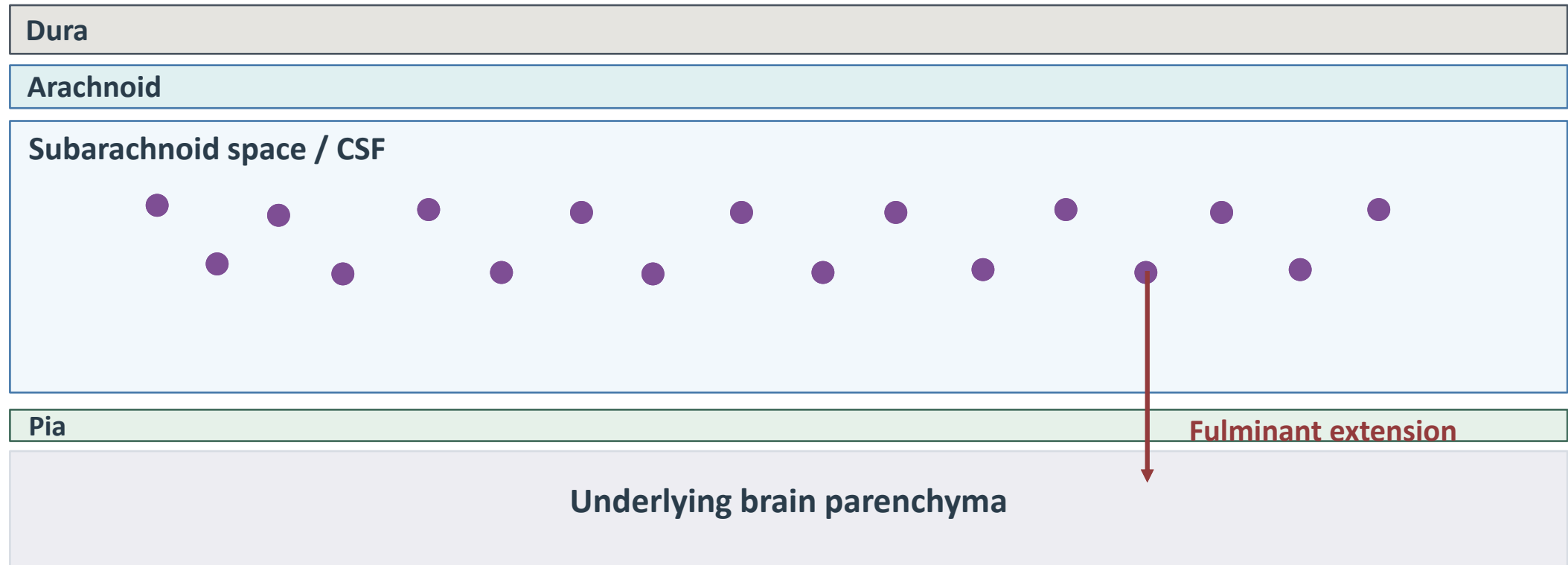
Gram stain may demonstrate bacteria

- Variable numbers of organisms may be visible on Gram stain.
- Bacteria may be demonstrable, particularly in untreated infection.
- A negative or low-yield illustrative field cannot replace the wider diagnostic context.

The pia is usually not breached

- Bacterial inflammation is most prominent in leptomeninges and the subarachnoid space.
- The pia is rarely crossed in ordinary bacterial meningitis.
- Fulminant cases may show a different, more invasive pattern.

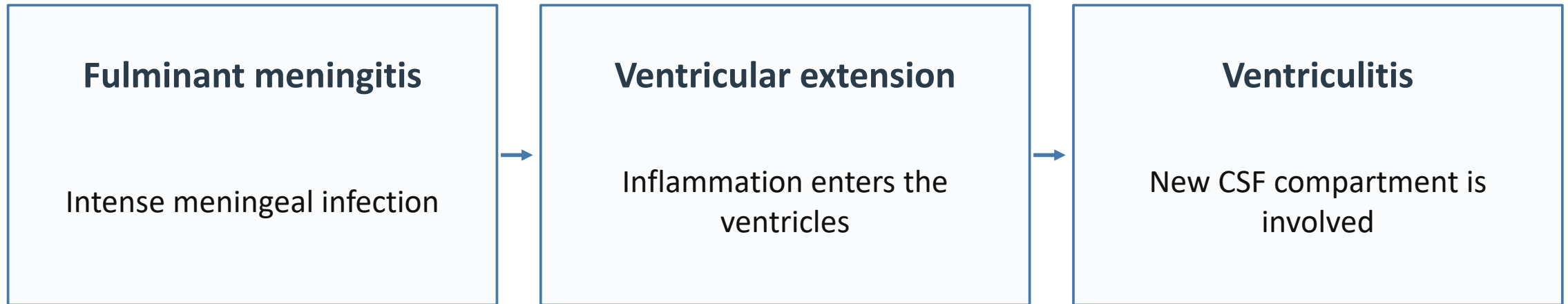
Fulminant extension: Focal cerebritis



The pia is usually preserved; Severe inflammation can extend into brain.

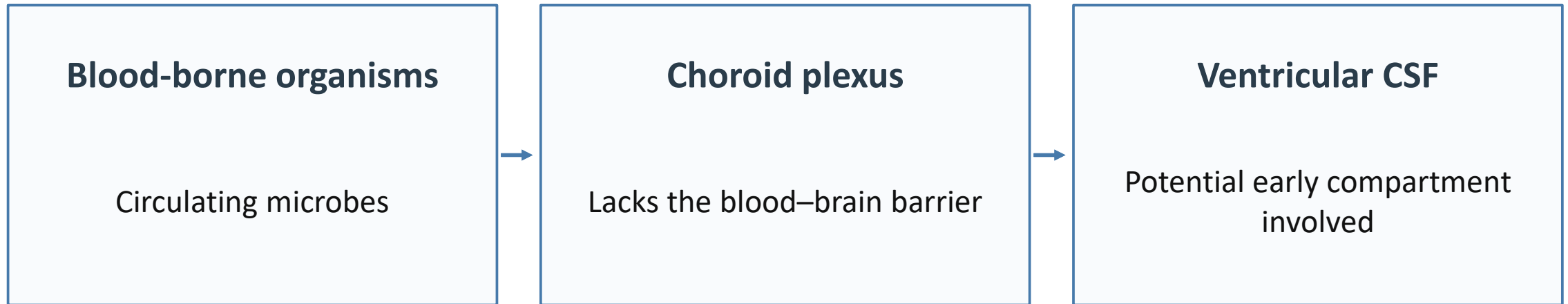
Schematic

Ventriculitis: Spread to a CSF-containing cavity



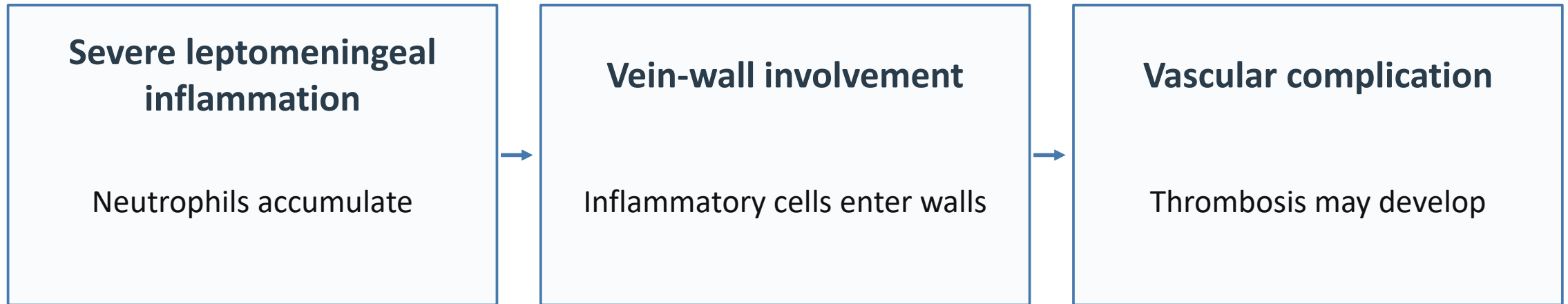
The source also describes a separate blood-borne route to ventricular CSF.

Choroid plexus as a portal for blood-borne infection



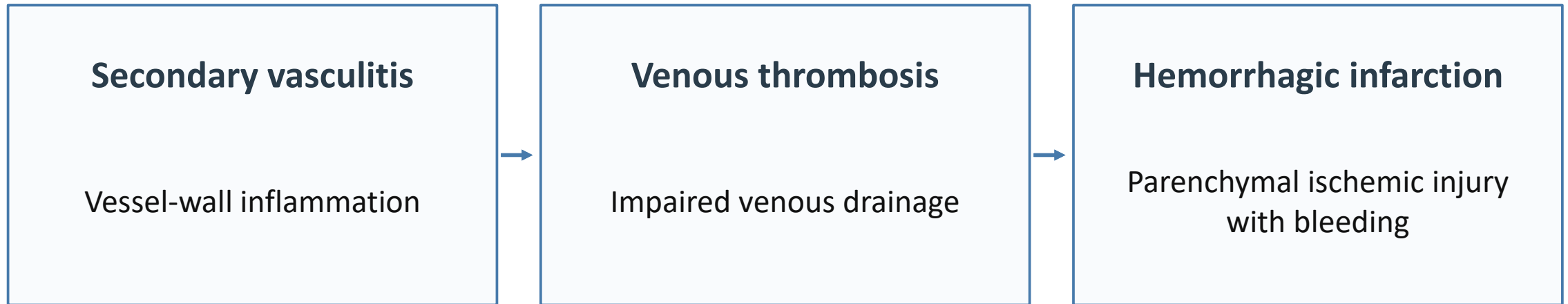
This is an entry route, distinct from extension of pre-existing meningitis.

Venous wall inflammation creates vascular risk



Inflammation and thrombosis are key bridges from meningeal disease to tissue injury.

Vasculitis, thrombosis, and cerebral infarction

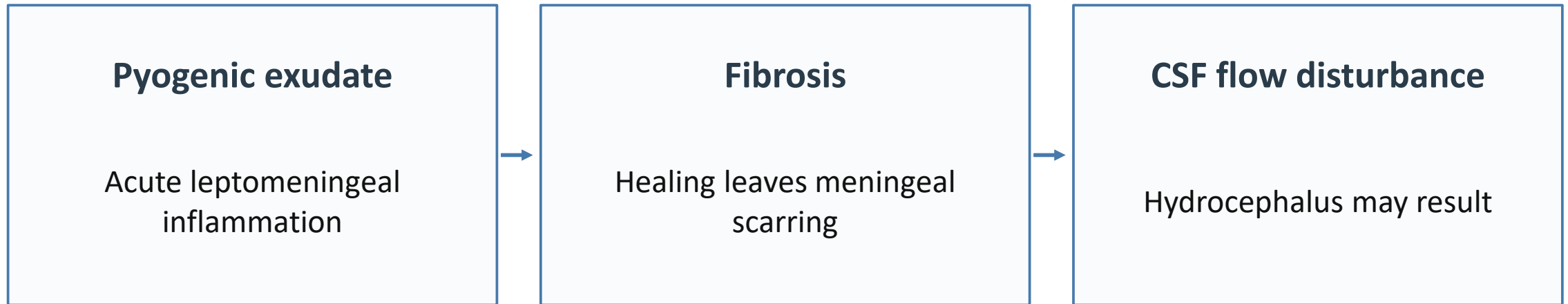


Meningeal disease can progress to devastating cerebral vascular injury.

Why cerebral hemorrhagic infarction matters

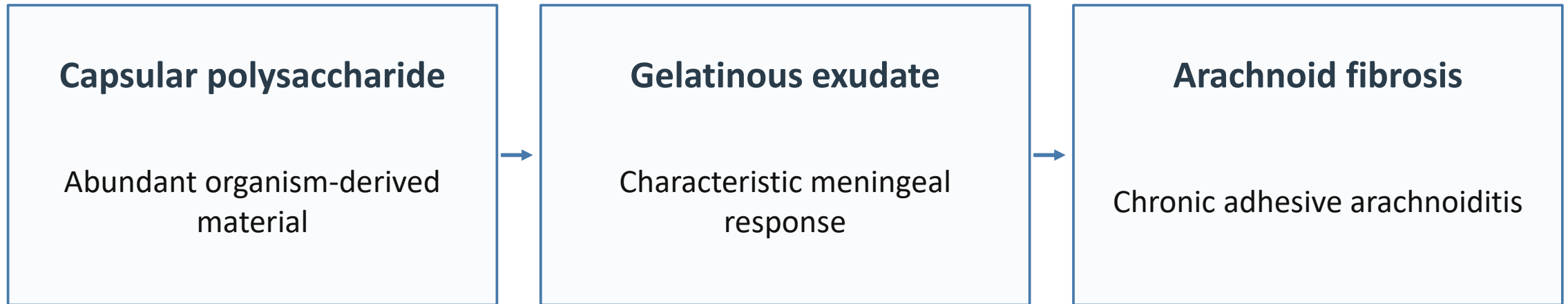
- The primary infection begins in the meninges, but vascular injury can damage brain tissue.
- Thrombosis and vasculitis provide a path to hemorrhagic cerebral infarction.
- A focal parenchymal lesion does not necessarily represent a primary brain abscess.

Inflammation can produce delayed hydrocephalus



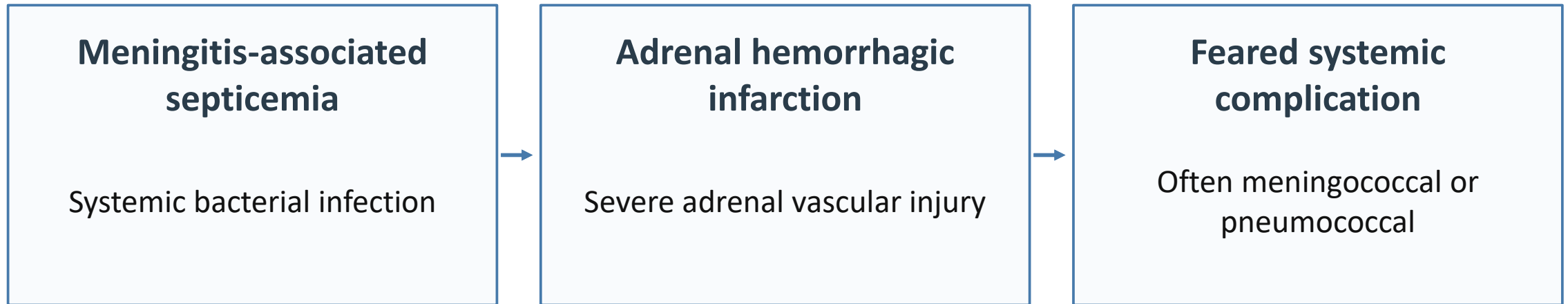
Leptomeningeal fibrosis is a distinct late effect of the acute inflammatory episode.

Pneumococcus: An adhesive arachnoiditis pathway



Chronic fibrosing inflammation is termed adhesive arachnoiditis.

Waterhouse–Friderichsen syndrome



This is an extracranial complication—not purulent material in the adrenal glands.

Complications and their pathologic mechanisms

Complication	Mechanistic association
Ventriculitis	Inflammation reaches ventricular CSF
Cerebritis	Focal penetration into parenchyma in fulminant cases
Hemorrhagic cerebral infarction	Vasculitis and venous thrombosis
Hydrocephalus	Leptomeningeal / arachnoid fibrosis
Waterhouse–Friderichsen	Septicemia with adrenal hemorrhagic infarction

Distinguish meningeal infection from focal suppuration

Entity	Dominant compartment	Characteristic finding
Pyogenic meningitis	Leptomeninges + CSF	Diffuse or regionally concentrated surface exudate
Brain abscess	Parenchyma	Localized necrotic focus with acute inflammation
Subdural empyema	Subdural space	Purulent collection after contiguous spread
Extradural abscess	Extradural space	Often from adjacent infection or procedure

Brain abscess and meningitis differ structurally

Pyogenic meningitis

Exudate on leptomeninges
Neutrophils within subarachnoid CSF

Brain abscess

Localized parenchymal necrosis
Focal acute inflammation

An abscess can secondarily rupture, causing ventriculitis or meningitis.

Subdural empyema and meningitis differ by space

Meningitis

Inflamed leptomeninges
Subarachnoid CSF is involved

Subdural empyema

Infected collection in subdural space
Underlying arachnoid often remains unaffected

Anatomic localization changes interpretation of findings and complications.

Case application: Neonatal meningitis

Hypothetical case

A newborn develops an acute infectious illness and clinical concern for meningitis. CSF is cloudy and neutrophil-rich, with high protein and low glucose.

Which organisms should be considered?

Interpretation

E. Coli and group B streptococci are important neonatal causes. The CSF pattern supports pyogenic disease but does not identify the organism.

Teaching exercise, not a documented patient or a diagnostic substitute.

Case application: Meningococcal clustering

Hypothetical case

Several adolescents develop a compatible acute meningitis syndrome within a population cluster. Clinicians recognize a potential community concern.

Which organism is highlighted?

Interpretation

Neisseria meningitidis is prominent in this age range; Clusters raise public-health concern. Age and clustering do not alone prove etiology.

Teaching exercise, not a documented patient or a diagnostic substitute.

Case application: Gross meningeal morphology

Hypothetical case

An older patient with acute pyogenic meningitis has a thick meningeal exudate concentrated over the cerebral convexities near the sagittal sinus.

Which source-associated organism fits?

Interpretation

S. Pneumoniae has the stated gross distribution tendency. It is not the only possible pathogen in an older adult.

Teaching exercise, not a documented patient or a diagnostic substitute.

Case application: Atypical immunosuppressed host

Hypothetical case

An immunosuppressed patient has suspected purulent meningitis, but the symptoms and CSF pattern are not characteristic of the usual syndrome.

What diagnostic pitfall arises?

Interpretation

Atypical presentations can delay recognition. Klebsiella, anaerobes and other organisms may be involved.

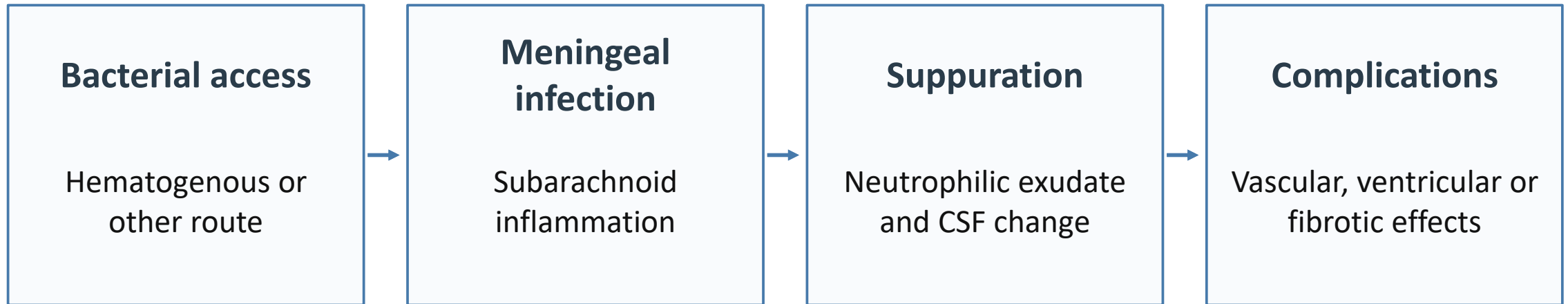
Teaching exercise, not a documented patient or a diagnostic substitute.

Integrated recognition: Compare three disease dimensions

Clinical	CSF	Morphology
Systemic infection Meningeal irritation Neurologic impairment	Purulent appearance Neutrophilic pleocytosis High protein / low glucose	Surface exudate Engorged vessels Neutrophils in subarachnoid space

Converging patterns support the clinicopathologic diagnosis.

A unifying pathologic sequence



Clinical signs and laboratory findings emerge within this wider tissue-level process.

Synthesis: Five points that define the disease

- Acute bacterial meningitis affects leptomeninges and the subarachnoid CSF compartment.
- The dominant pathogens differ across neonatal, young-adult and older-adult groups.
- Characteristic CSF: Neutrophils, increased protein/pressure and decreased glucose.
- Morphology: Suppurative surface exudate, vascular engorgement, neutrophils.
- Serious complications include ventriculitis, infarction, hydrocephalus and septicemic adrenal injury.

Suggested reference for further reading

- Robbins, Cotran & Kumar: Pathologic Basis of Disease, 11th edition (2026).
- Section: The Central Nervous System — Infections — Acute Meningitis.
- Core reading: Acute Pyogenic (Bacterial) Meningitis and its morphology.