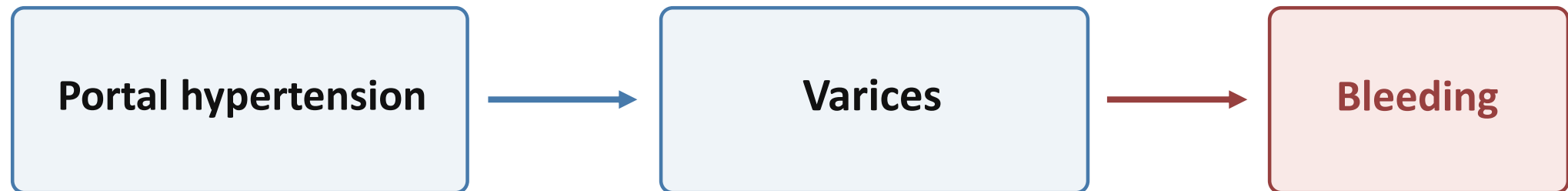


# Esophageal Varices

Portal hypertension · Vascular remodeling · Hemorrhage



Pathology and clinicopathologic integration

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## Learning objectives

- Explain how increased portal venous resistance produces gastroesophageal collaterals.
- Recognize variceal morphology in the mucosa and submucosa.
- Link vessel rupture to potentially fatal upper gastrointestinal hemorrhage.
- Interpret textbook prevalence, bleeding risks, recurrence, and management.

## Definition: Dilated distal esophageal veins

- Esophageal varices are abnormally dilated venous channels in the lower esophagus.
- The affected veins belong to the mucosal and submucosal plexuses.
- Varices arise as a consequence of portal hypertension and portosystemic diversion.
- The principal danger is rupture of a large varix, not mere venous dilation.

# From portal hypertension to varices

## **Impeded portal drainage**

Resistance to blood flow through the portal system and liver rises.



## **Collateral recruitment**

Portosystemic communicating channels carry redirected blood.



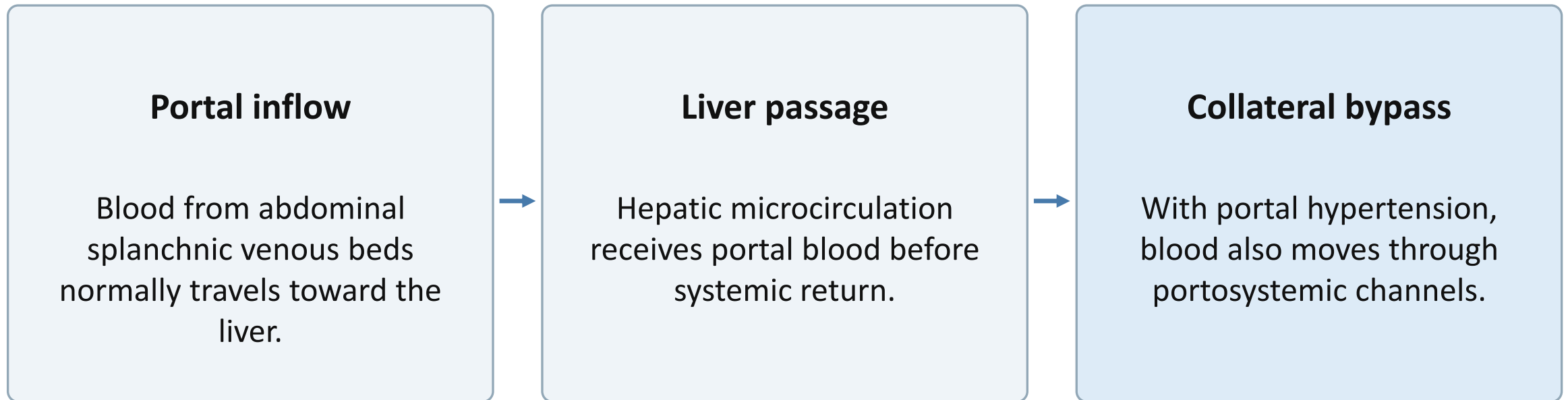
## **Esophageal congestion**

Subepithelial and submucosal plexuses expand into tortuous varices.

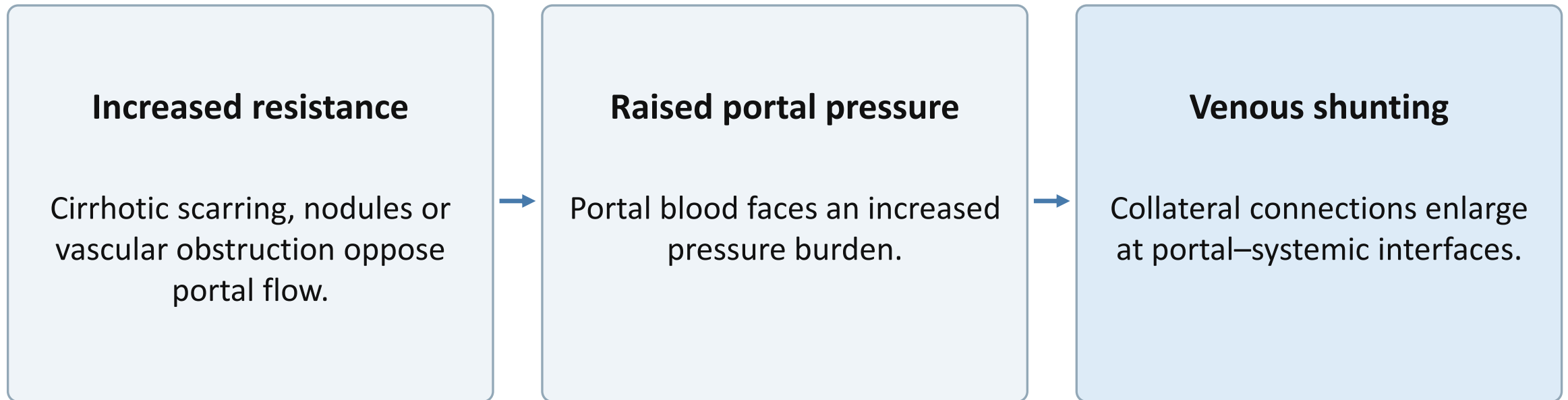
## Why the lower esophagus is vulnerable

- The gastroesophageal junction is a major site of communication between portal and systemic venous drainage.
- Elevated portal pressure diverts blood into collateral veins at this junction.
- Congested distal esophageal veins dilate within a relatively thin mucosal wall.
- Rupture opens a high-flow venous channel to the gastrointestinal lumen.

# Normal route and collateral bypass



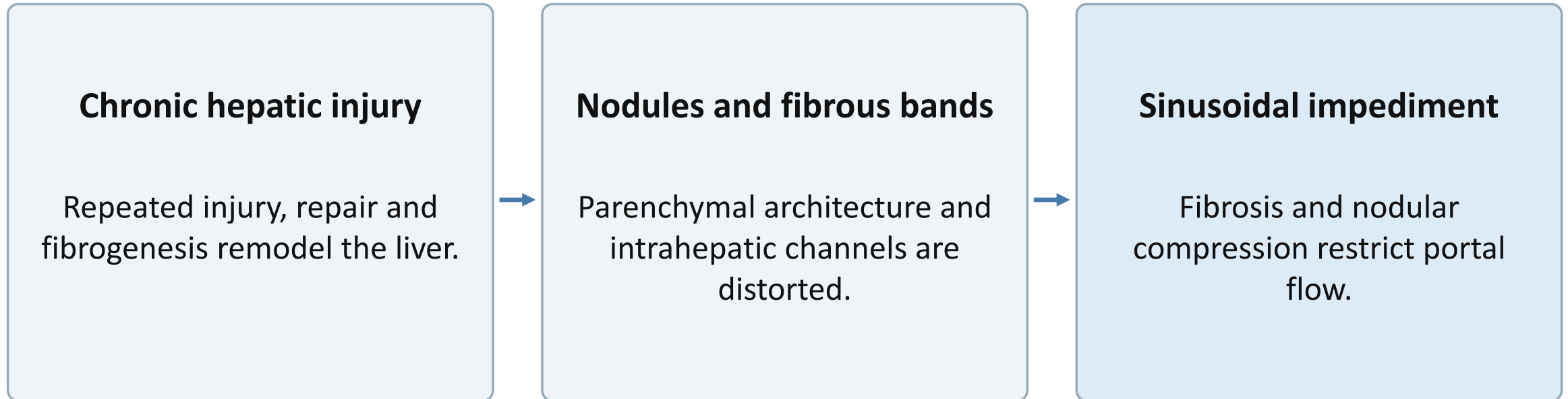
# Pressure–flow relation in portal hypertension



## Portal hypertension has multiple locations

- Prehepatic obstruction occurs before portal blood enters the hepatic microvasculature.
- Intrahepatic resistance develops within liver parenchymal and vascular pathways.
- Posthepatic obstruction affects venous drainage beyond hepatic sinusoids.
- All three can elevate portal pressure; Cirrhosis is the dominant intrahepatic cause.

# Cirrhosis: Structural resistance



## Cirrhosis is not synonymous with liver failure

- Cirrhosis is a diffuse architectural remodeling process with fibrosis and nodules.
- Its vascular resistance can produce portal hypertension and varices.
- The clinical degree of hepatic insufficiency varies among patients.
- Varices are therefore a vascular manifestation, not a histologic definition of cirrhosis.

## Liver disease contexts for esophageal varices

- Alcohol-associated cirrhosis is a common setting for gastroesophageal varices.
- Varices also complicate cirrhosis of other causes, including nonalcoholic fatty liver disease.
- Chronic viral hepatitis is another major contributor to cirrhosis and portal hypertension.
- Noncirrhotic portal hypertension must be considered when cirrhosis is absent.

## Two drivers of portal hypertension

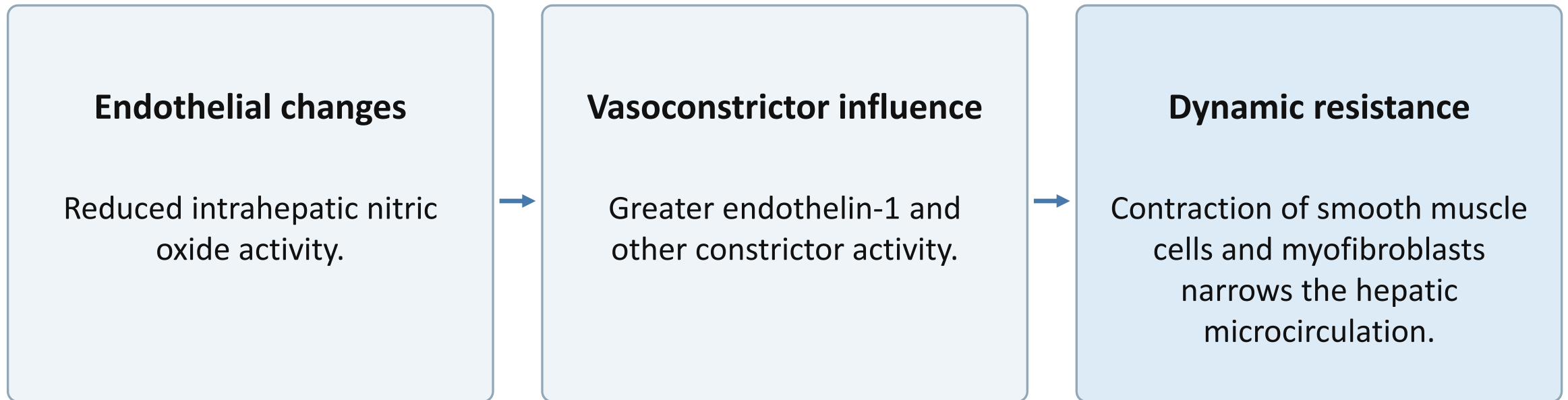
### Resistance within liver

Scarring, nodules and sinusoidal contraction obstruct portal passage.

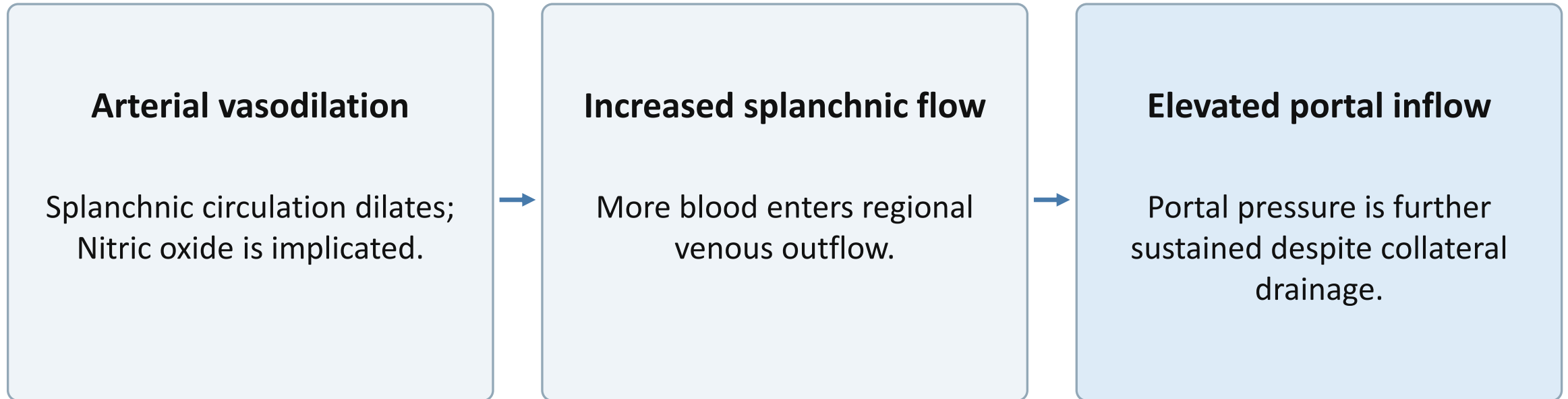
### Increased portal inflow

Splanchnic arterial vasodilation increases venous return to the portal system.

# Sinusoidal endothelial dysfunction



# Splanchnic hyperdynamic circulation



## Portal hypertension persists despite collaterals

- Collateral channels provide alternate routes for portal drainage.
- They do not reliably normalize resistance across a diseased liver.
- Persistently elevated inflow and resistance maintain pressure within the collateral bed.
- Esophageal varices consequently remain exposed to an abnormal hemodynamic load.

# Prehepatic causes of raised portal pressure

## Portal vein thrombosis

Obstructive thrombus limits flow before the hepatic portal branches.

## Portal vein narrowing

Structural narrowing can impede entry of portal blood.

## High splenic inflow

Marked splenomegaly may increase splenic venous delivery.

## Intrahepatic causes beyond cirrhosis

### Portal tract disorders

Schistosomiasis and nodular regenerative hyperplasia may increase resistance.

### Infiltrative disease

Amyloidosis, granulomatous disease and malignancy may impair flow.

### Other parenchymal disease

Massive fatty change or primary biliary cholangitis may contribute.

# Posthepatic causes of portal hypertension

## Hepatic vein outflow

Venous drainage obstruction, including Budd–Chiari syndrome.

## Cardiac causes

Severe right-sided heart failure elevates back pressure.

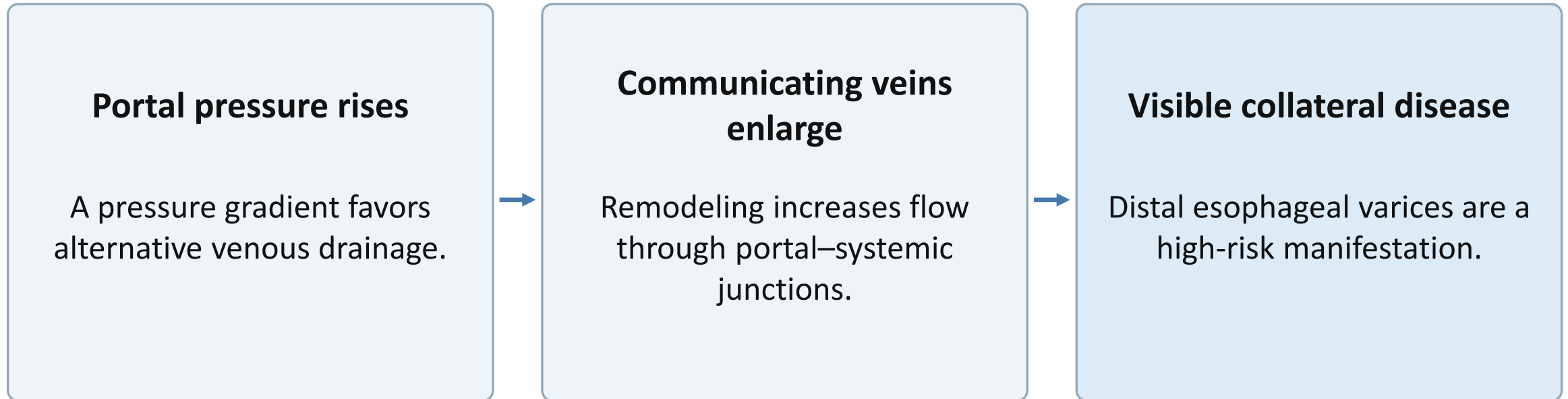
## Pericardial limitation

Constrictive pericarditis impedes systemic venous return.

## Localize the obstacle along portal flow

| Before liver                | Within liver                     | After liver               |
|-----------------------------|----------------------------------|---------------------------|
| Portal vein thrombus        | Cirrhosis                        | Hepatic vein obstruction  |
| Structural portal narrowing | Nodular regenerative hyperplasia | Right-sided heart failure |
| Increased splenic inflow    | Schistosomiasis / infiltration   | Constrictive pericarditis |

# Formation of portosystemic collateral channels



# Major sites of portosystemic shunting

## **Gastroesophageal junction**

Submucosal esophageal and gastric varices; Major bleeding hazard.

## **Periumbilical region**

Dilated abdominal wall collateral veins (caput medusae).

## **Rectal / retroperitoneal beds**

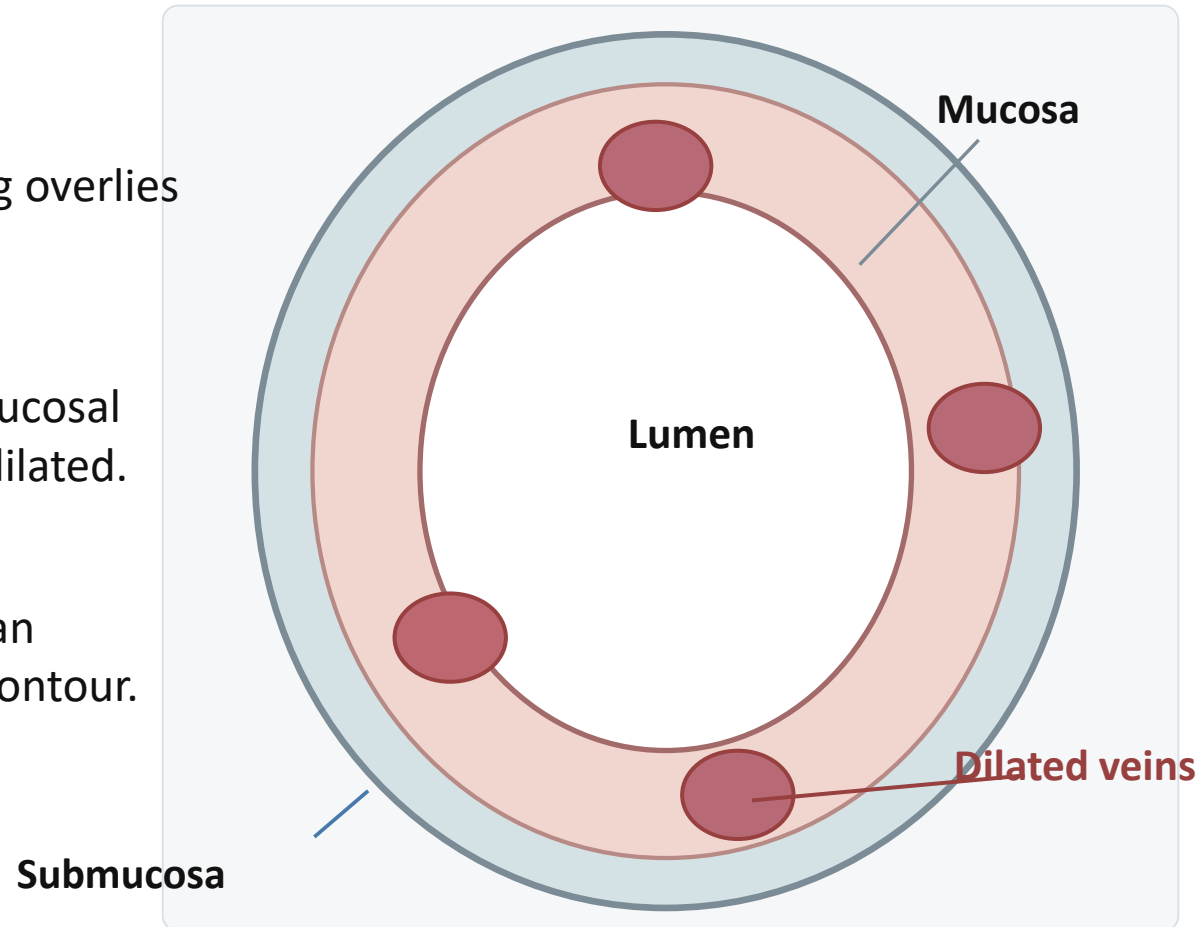
Additional collateral routes associated with portal hypertension.

## Esophageal and proximal gastric continuity

- Increased portal pressure affects veins on both sides of the gastroesophageal junction.
- Submucosal and subepithelial veins are particularly prominent sites of congestion.
- Esophageal and proximal gastric varices therefore share a hemodynamic origin.
- The involved segment is primarily distal esophagus and adjacent stomach.

# Esophageal wall: Venous compartments

- Intact luminal lining overlies vulnerable vessels.
- Mucosal and submucosal channels become dilated.
- Tortuosity creates an irregular vascular contour.



SCHEMATIC · NOT HISTOLOGY

# Evolution of the venous plexus

## Communicating vein

A small venous channel drains across the portal–systemic interface.



## Progressive congestion

Higher pressure and collateral flow expand the channel.



## Established varix

Enlarged tortuous mucosal or submucosal vessel protrudes toward the lumen.

## Morphology: Serpiginous, dilated veins

- Histologically, veins are enlarged and tortuous within the mucosa and submucosa.
- A dense tangle of vascular lumina may produce submucosal expansion.
- The mucosa can remain intact before bleeding occurs.
- A prominent varix represents vascular dilation rather than a neoplastic mass.

## Correlating views of a varix

### Endoscopy

Protruding submucosal nodules may be seen beneath the surface.

### Vascular imaging

Tortuous venous pathways demonstrate collateral circulation.

### Microscopy

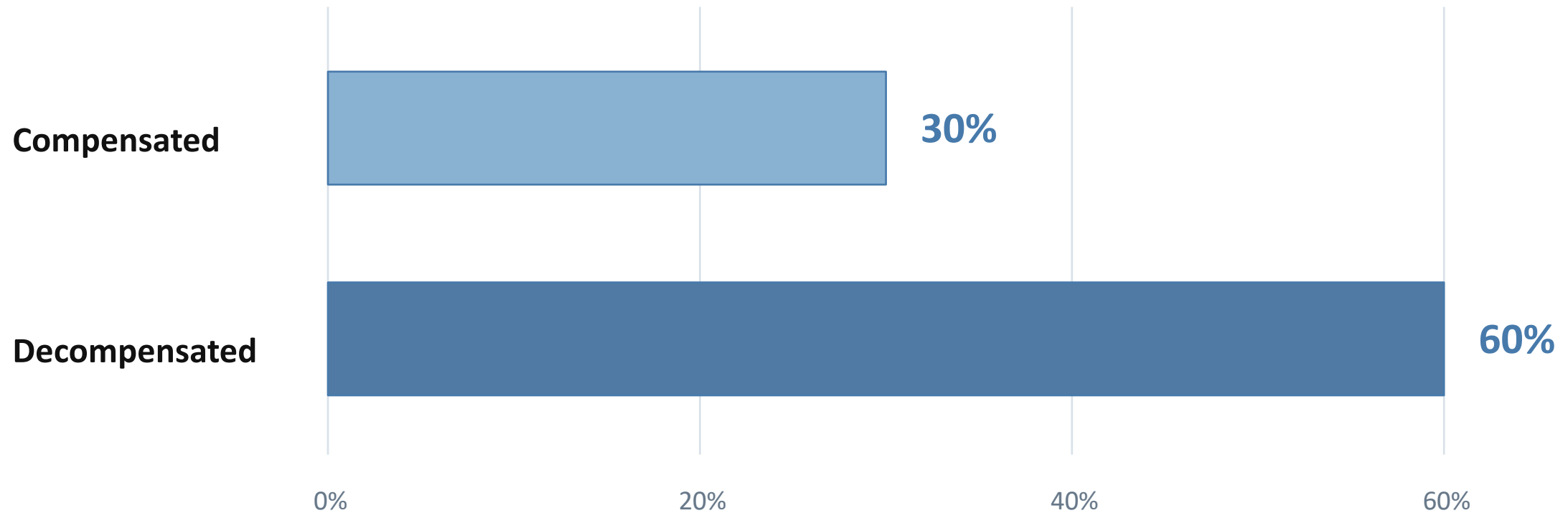
Dilated veins are located below squamous mucosa.

## Small varices often remain unruptured

- Most small varices never bleed.
- Presence of a varix is not equivalent to immediate hemorrhage.
- Size, tortuosity and hemodynamic conditions influence bleeding risk.
- Morphologic risk and underlying liver disease must be considered together.

# Variceal prevalence increases with decompensation

## Gastroesophageal varices in cirrhosis

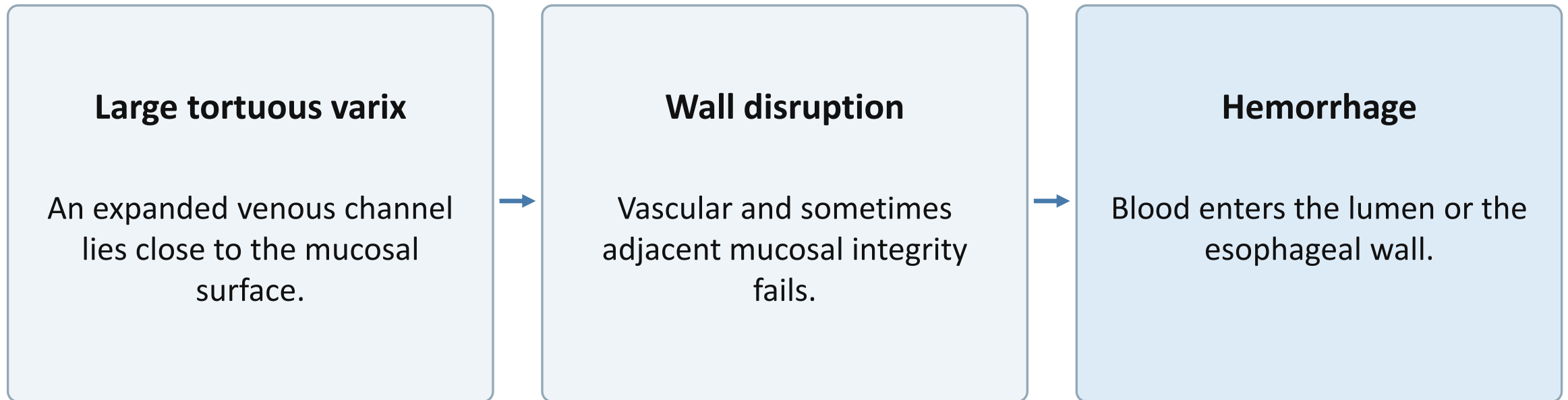


Source categories are compensated and decompensated cirrhosis; This is prevalence, not bleeding probability.

## Prevalence is not bleeding risk

- The 30% and 60% figures describe varices among two cirrhosis categories.
- They do not state the annual probability of rupture.
- Varix morphology and pressure-related factors must be assessed separately.
- Avoid interpreting prevalence as a prognostic event rate.

# Mechanistic pathway to variceal rupture



## Risk factors for variceal hemorrhage

### **Variceal anatomy**

Large caliber or tortuous configuration.

### **Hemodynamics**

Elevated hepatic venous pressure gradient.

### **Clinical history / liver status**

Previous bleeding and advanced liver disease.

## Variceal size and tortuosity matter

- Large veins have greater potential for severe bleeding when they rupture.
- Tortuous vessels reflect pronounced collateral venous remodeling.
- Both large and tortuous varices as risk features.
- A visually abnormal vein still requires clinical context for risk assessment.

## Hepatic venous pressure gradient: Risk signal

- An elevated hepatic venous pressure gradient is associated with bleeding risk.
- It reflects the pressure burden across the hepatic vascular bed.
- The provided source does not supply a risk-stratification threshold.
- A numerical cutoff is deliberately omitted rather than inferred.

## Previous hemorrhage changes risk assessment

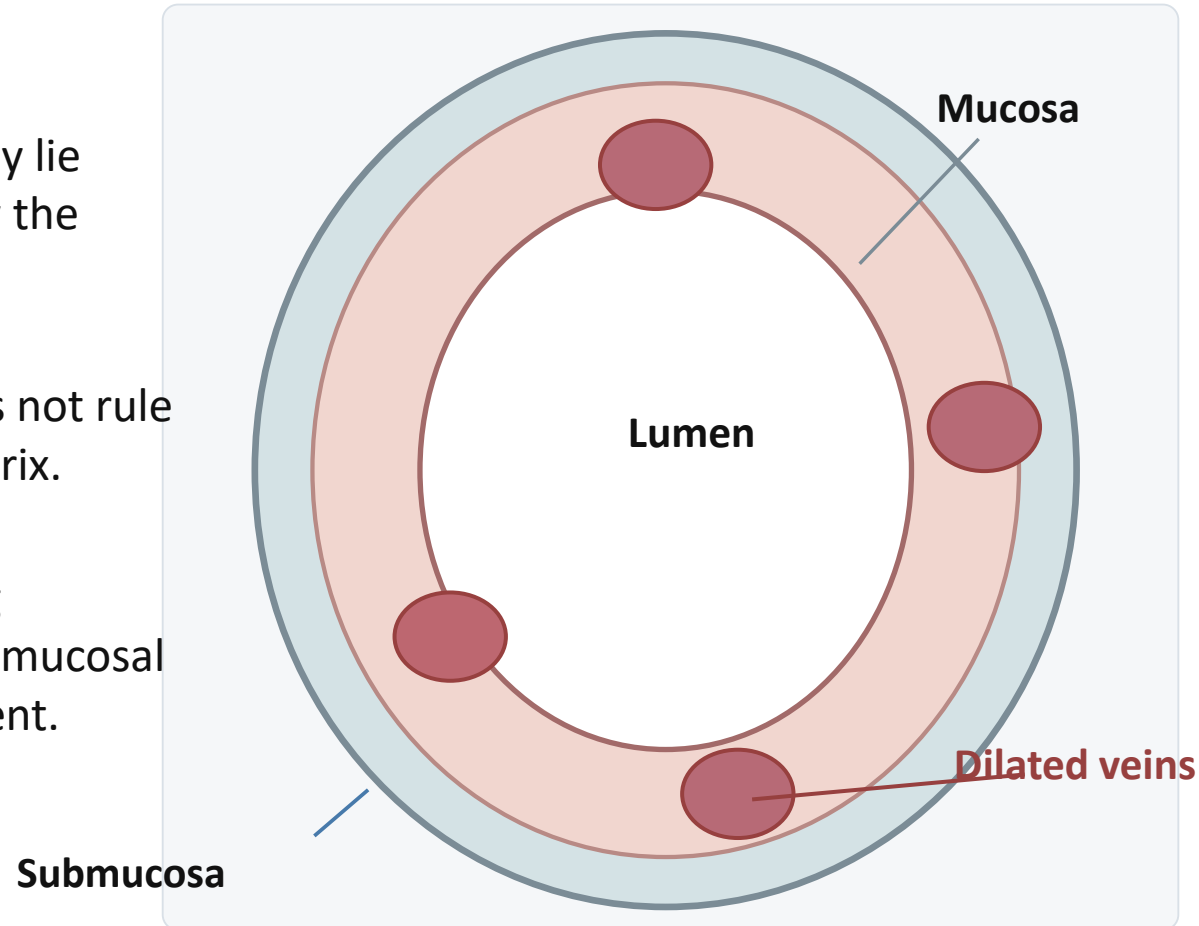
- A history of variceal bleeding is itself a recognized risk factor.
- Prior bleeding implies that the collateral bed has already ruptured.
- Recurrent events are common among survivors.
- Prevention of further bleeding is a distinct clinical goal.

## Advanced liver disease amplifies clinical risk

- Advanced liver disease is associated with variceal bleeding risk.
- The underlying hepatic disorder also affects physiologic reserve.
- A similar anatomical lesion may have different consequences in different patients.
- Risk discussion must consider both vascular morphology and liver status.

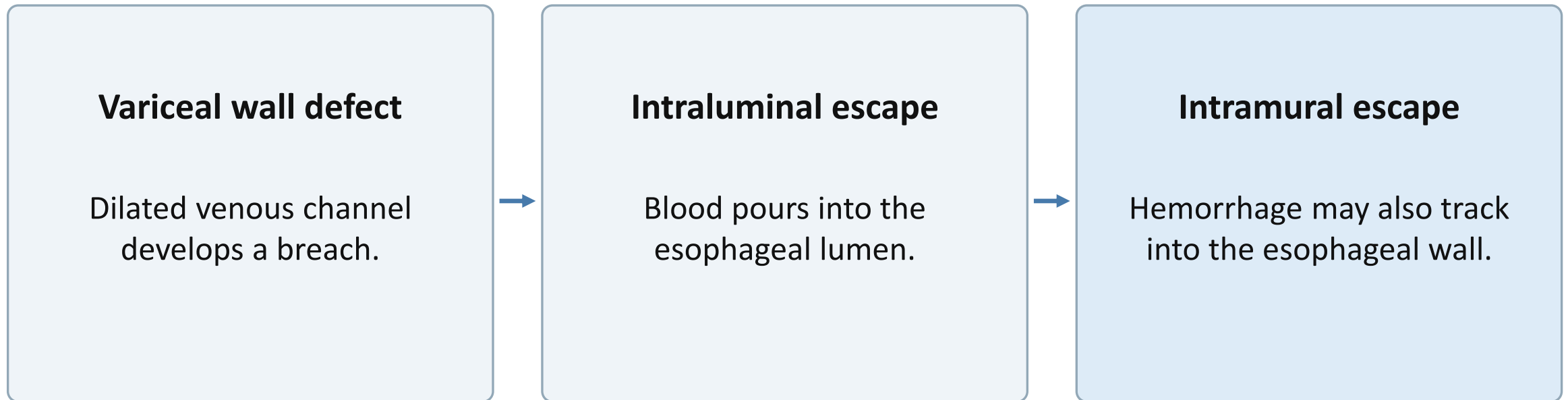
## Before rupture: Intact squamous mucosa

- A dilated vessel may lie immediately below the epithelium.
- Intact mucosa does not rule out a prominent varix.
- Endoscopic bulging corresponds to submucosal vascular enlargement.



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## After rupture: Two hemorrhage locations



## Rupture may damage overlying mucosa

- Variceal hemorrhage can coexist with mucosal ulceration.
- Necrosis may accompany local tissue injury.
- These surface changes are consequences or accompaniments of rupture.
- Do not mistake associated ulceration for the primary vascular lesion.

## Thrombosis after previous bleeding

- Venous thrombosis may be found if varices ruptured previously.
- A thrombus documents vascular luminal alteration rather than simple dilation.
- Prior hemorrhage can coexist with inflammatory change.
- Morphologic interpretation benefits from treatment history.

## Inflammation and repair in a treated varix

- Inflammation may surround veins affected by earlier hemorrhage.
- Sclerotherapy intentionally produces local thrombosis and sclerosis.
- Previously treated varices can differ markedly from untreated vessels.
- A biopsy or postmortem specimen should be interpreted in clinical context.

## Histologic pattern: Untreated versus previously treated

### Untreated or unruptured

Large, tortuous  
mucosal/submucosal veins;  
Epithelium may be intact.

### Previously ruptured

Thrombosis, hemorrhage,  
inflammation, ulceration or  
necrosis may be present.

### Previously sclerosed

Therapeutic vascular  
alteration can add scarring  
and thrombotic change.

## Endoscopic appearance versus histologic substrate

- Endoscopy shows protruding submucosal nodules rather than the entire venous network.
- Angiographic imaging illustrates the tortuous collateral pathways.
- Microscopy identifies dilated vessels below squamous mucosa.
- These views are complementary; None is a substitute for the others.

# Why variceal bleeding can be catastrophic

## **Portal hypertensive varix**

An enlarged collateral receives blood under abnormal pressure.



## **Vessel rupture**

Rapid loss of blood into the upper GI tract may follow.



## **Major hemorrhage**

Severe bleeding may progress to exsanguination.

## Clinical presentation: Upper GI hemorrhage

- Bleeding esophageal varices may manifest as massive hematemesis.
- The source characterizes variceal hemorrhage as a medical emergency.
- Large-volume bleeding reflects disruption of the submucosal venous collateral.
- Immediate control of hemorrhage is the clinical priority.

# Mortality of a bleeding episode

**15–20%**

**Per-episode mortality**

The risk remains substantial despite available interventions.

## Risk after an initial bleed

**>50%**

**Recurrent bleeding within 1 year**

Among patients who survive a first variceal hemorrhage.

## First bleed versus recurrent hemorrhage

### Initial episode

Potentially life-threatening acute loss of blood; Risk factors include large varices.

### After survival

More than half have recurrence within one year in the chapter's description.

# Acute care targets the bleeding circuit

## **Reduce splanchnic flow**

Medical vasoconstriction diminishes delivery into the portal system.



## **Control varix locally**

Endoscopic treatment targets the affected venous channel.



## **Prevent recurrence**

Portal-flow reduction and planned ligation address future episodes.

## Medical therapy: Splanchnic vasoconstriction

- The source identifies pharmacologic splanchnic vasoconstriction as acute therapy.
- Vasoconstriction reduces inflow contributing to portal venous pressure.
- This treats a hemodynamic driver rather than the histologic scar of cirrhosis.
- Specific medication dosing is outside the supplied textbook excerpt.

# Endoscopic variceal ligation

- Ligation constricts the variceal channel locally using endoscopically applied bands.
- The affected vessel is mechanically occluded to stop or prevent bleeding.
- Ligation as both treatment and preventive strategy.
- Past ligation may leave recognizable treatment-related changes.

## Sclerotherapy: Thrombotic obliteration

- The source describes injection of thrombotic agents into varices.
- The intended effect is local thrombosis and obliteration of the target channel.
- Microscopy may subsequently show treatment-associated changes.
- Sclerotherapy is distinct from systemic management of portal pressure.

## Balloon tamponade: Mechanical hemorrhage control

- Balloon tamponade is listed as an emergency endoscopic/mechanical option in the source.
- Applied pressure can temporarily compress a bleeding variceal region.
- It targets active hemorrhage rather than correcting chronic portal hypertension.
- Its role is a bridging hemorrhage-control concept, not a definitive cure.

## Compare strategies by immediate target

| Approach          | Primary action            | Pathologic target       |
|-------------------|---------------------------|-------------------------|
| Vasoconstriction  | Reduces splanchnic inflow | Portal hemodynamic load |
| Band ligation     | Mechanical occlusion      | Individual varix        |
| Sclerotherapy     | Induced thrombosis        | Individual varix        |
| Balloon tamponade | Pressure compression      | Active bleeding site    |

## $\beta$ -blockers and prevention of bleeding

- $\beta$ -blockers as treatment to reduce portal blood flow.
- The strategy acts upstream of the dilated collateral veins.
- Endoscopic variceal ligation provides local preventive therapy.
- Preventive treatment is important because recurrent bleeding is frequent.

# Case: Massive hematemesis

## Clinical vignette

A patient with established cirrhosis suddenly develops a large-volume upper gastrointestinal hemorrhage.

## Interpretation task

What structural lesion and mechanism should be suspected?

## Teaching answer

Portal hypertension with a ruptured distal esophageal submucosal varix.

## Case: Post-treatment esophageal tissue

### Clinical vignette

A previously treated patient has dilated veins with thrombus and adjacent inflammatory change in an esophageal specimen.

### Interpretation task

Which clinical history most directly explains this combination?

### Teaching answer

Prior variceal rupture and/or sclerotherapy may explain thrombosis and inflammation.

## Case: Different stages of cirrhosis

### Clinical vignette

Two patient cohorts have compensated and decompensated cirrhosis, respectively.

### Interpretation task

Which cohort has the higher reported prevalence of gastroesophageal varices?

### Teaching answer

Decompensated cirrhosis: 60% versus 30% in compensated cirrhosis, as reported.

# Esophageal varices versus other portosystemic signs

## Gastroesophageal varices

Most concerning for potentially massive upper-GI hemorrhage.

## Caput medusae

Distended abdominal wall collaterals radiating near umbilicus.

## Rectal collaterals

Portosystemic channels around rectal region; Different clinical bleeding profile.

# Integrate lesion, mechanism and outcome

## Hepatic vascular resistance

Cirrhosis or another obstructive process increases portal pressure.



## Collateral remodeling

Venous channels at the distal esophagus progressively dilate.



## Variceal hemorrhage

Rupture creates a potentially fatal GI bleeding emergency.

## Five discriminating pathology facts

- A varix is a dilated vein in distal esophageal mucosa/submucosa.
- Portal hypertension drives collateral recruitment at the gastroesophageal junction.
- Varices are commoner in decompensated than compensated cirrhosis.
- Rupture may cause massive bleeding, mucosal injury and thrombosis.
- A prior bleed and advanced liver disease indicate greater clinical concern.

## Principal references

### **Robbins, Cotran & Kumar**

Pathologic Basis of Disease · 11th edition (2026)

- Chapter 17: Esophageal Varices, pp. 691–693, with Fig. 17.8 description.
- Chapter 18: Cirrhosis and Portal Hypertension, pp. 753–755.
- Chapter 11: Portosystemic shunts as a vascular complication, pp. 471–472.