

Varicose Veins

Venous hypertension, valvular incompetence, and tissue injury



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Learning objectives

- Explain pressure-induced venous dilation and valve incompetence.
- Connect venous congestion with skin changes and chronic ulcers.
- Distinguish superficial varicosity complications from deep-vein thrombosis.
- Trace the causes and consequences of esophageal varices and hemorrhoids.

Why venous pathology deserves attention

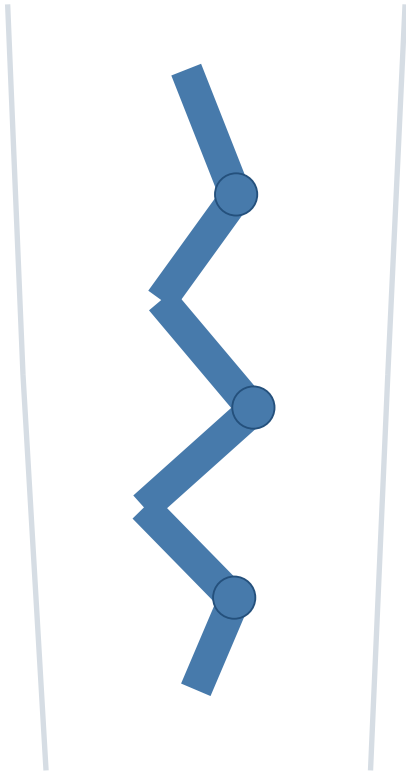
- Varicose veins and thrombophlebitis/phlebothrombosis dominate clinical venous disease.
- Together, these conditions account for at least 90% of clinical venous disease.
- Their principal hazards differ: Local tissue injury versus deep venous thromboembolism.

Definition: Two linked anatomic abnormalities

- A varicose vein is abnormally dilated and tortuous.
- The initiating setting is prolonged elevation of intraluminal pressure.
- Vessel dilation is followed by incompetence of venous valves.

What dilation and tortuosity mean

Schematic: Superficial lower-limb vein



Dilated and tortuous

- Dilation: Enlargement of the venous lumen.
- Tortuosity: Loss of a relatively straight course.
- Both can coexist within the same superficial vessel.

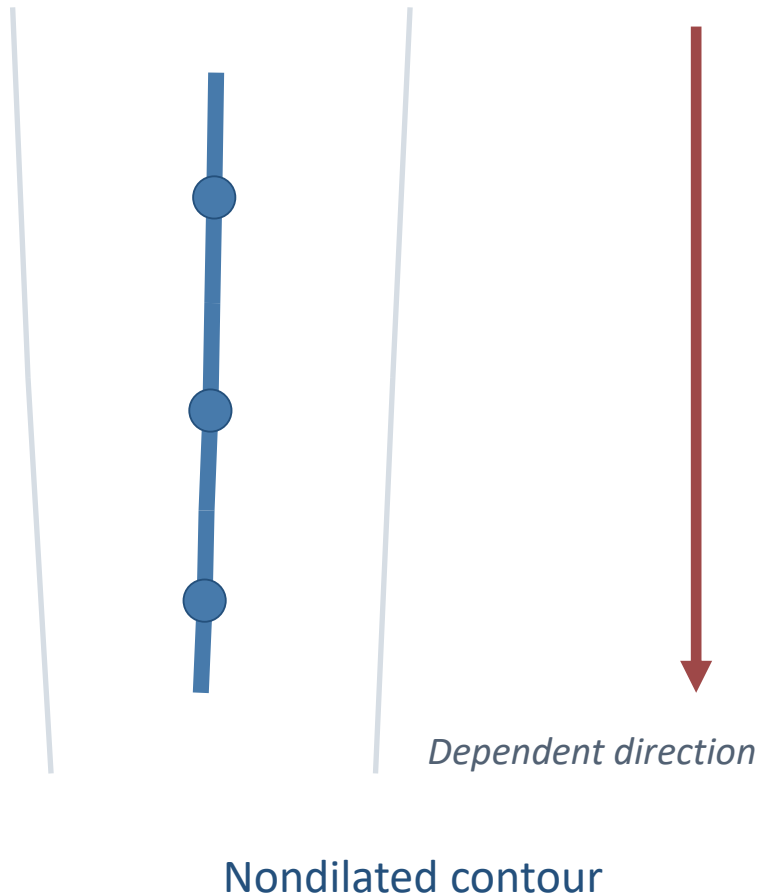
Sustained pressure is the upstream driver



- Prolonged venous hypertension loads the vessel wall.
- Repeated or persistent distension produces enlarged superficial veins.
- Valve incompetence compounds the resulting circulatory disturbance.

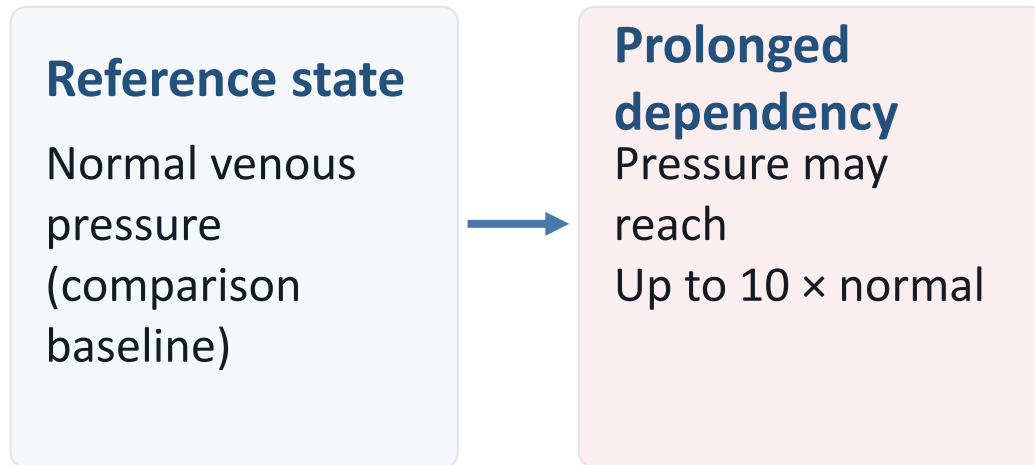
The dependent leg is a vulnerable site

Schematic: Superficial lower-limb vein



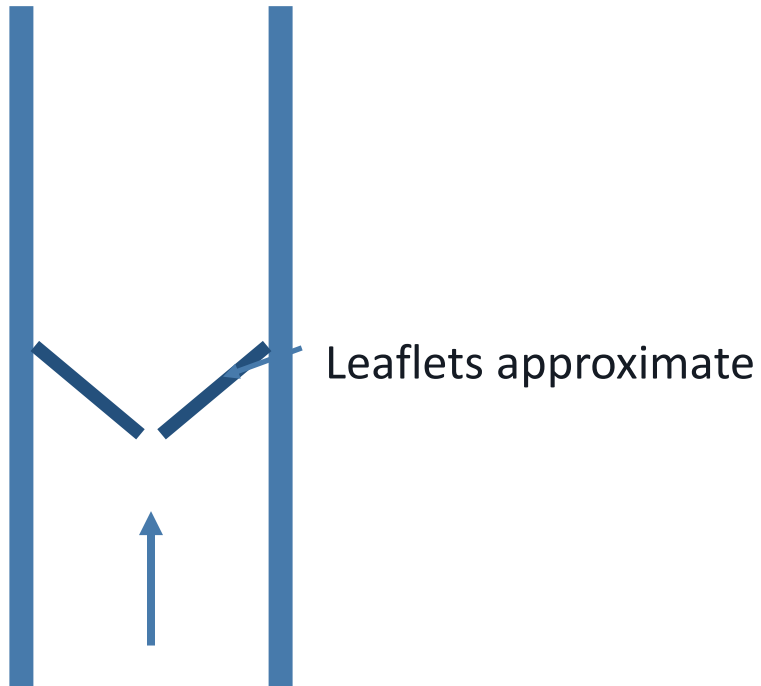
- The superficial veins of the upper and lower leg are frequently involved.
- Prolonged dependent posture markedly increases venous pressure.
- The anatomic site and posture explain the pattern of susceptibility.

How much can dependent pressure rise?



- Leg venous pressures reaching up to ten times normal.
- The comparison concerns prolonged dependent posture.
- This is a pressure effect—not a diagnostic threshold for an individual patient.

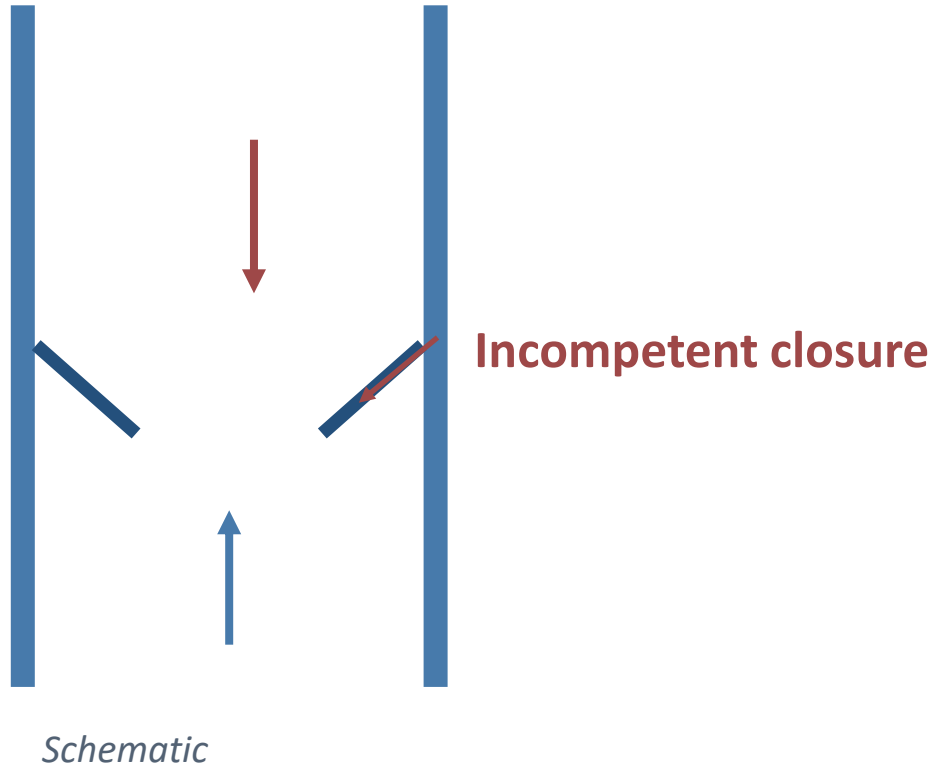
Venous valves normally enforce direction



Schematic

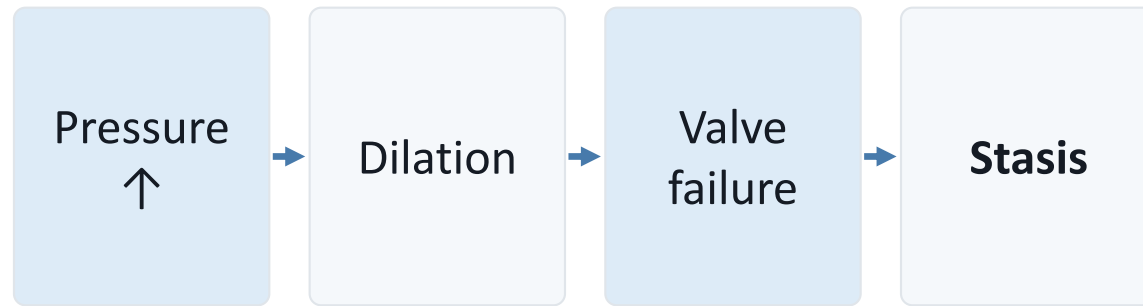
- Venous valves are arranged to favor directional return.
- Coaptation is necessary to oppose reverse movement of blood.
- Their failure is integral to varicose-vein pathogenesis.

Venous distension defeats valve closure



- Expansion separates the vessel walls and alters valve geometry.
- Leaflets no longer close effectively: Venous valve incompetence.
- Reverse flow and stasis then become possible.

A self-amplifying hemodynamic sequence



Conceptual pathogenesis sequence

- Elevated pressure causes dilation; Dilation compromises valves.
- Incompetent valves fail to prevent congestion and stasis.
- Persistent venous load reinforces clinically apparent varicosity.

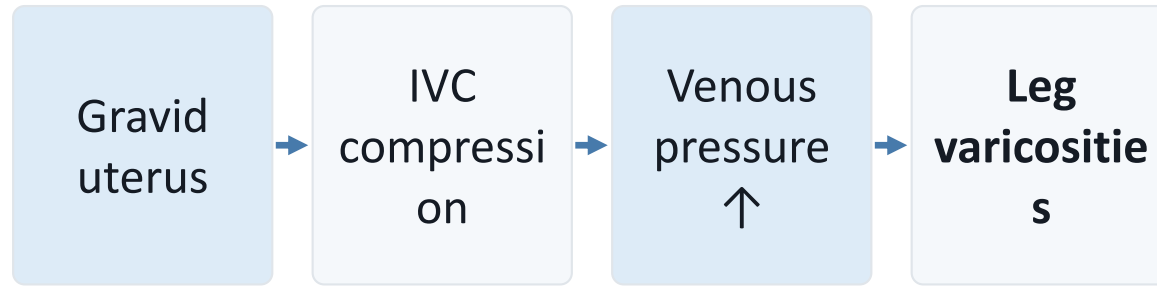
Familial susceptibility is structural

- A familial predisposition is recognized by Robbins.
- The textbook attributes susceptibility to faulty development of the venous wall.
- Familial risk can coexist with acquired elevations in venous pressure.

Obesity increases varicose-vein risk

- Obesity is specifically identified as a risk factor.
- The clinical association fits the overarching problem of venous pressure loading.
- Do not equate this risk factor with a single obligatory causal mechanism.

Pregnancy: A pressure-loading mechanism



- An enlarged uterus can compress the inferior vena cava.
- This raises venous pressure in the lower extremities over prolonged periods.
- Robbins invokes this mechanism to help explain increased frequency in women.

Risk factors organize into two mechanisms

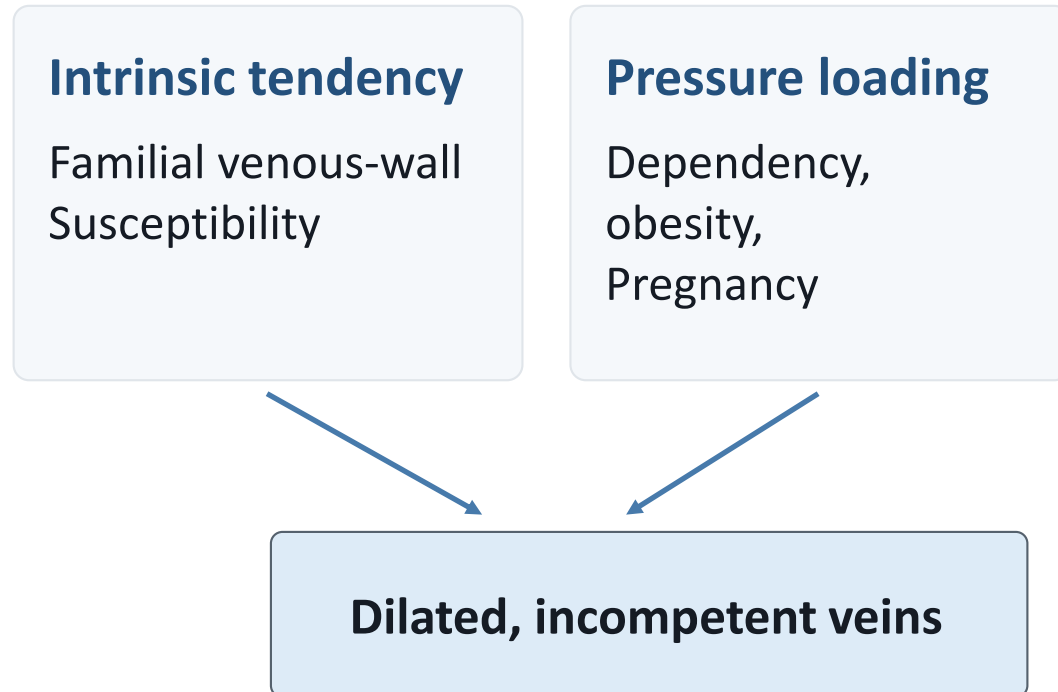
Predisposing influence	Robbins-supported mechanistic link
Prolonged dependency	Marked local venous pressure elevation
Pregnancy	Gravid uterine compression of the IVC
Familial susceptibility	Faulty development of the venous wall
Obesity	Increased risk; specific mechanism not detailed here

How common are lower-limb varicosities?

Reported proportions describe varicose veins of the lower extremities.

Population described	Frequency reported by Robbins	Interpretive caution
Males	Up to 20%	Upper-bound estimate
Females	Approximately one-third	Textbook estimate; not an individual risk

Risk pathways converge on one lesion



- Constitutional wall susceptibility favors dilation.
- External or posture-associated pressure loading favors dilation.
- Both routes culminate in tortuous, valve-incompetent superficial veins.

The clinical cascade begins at the valves

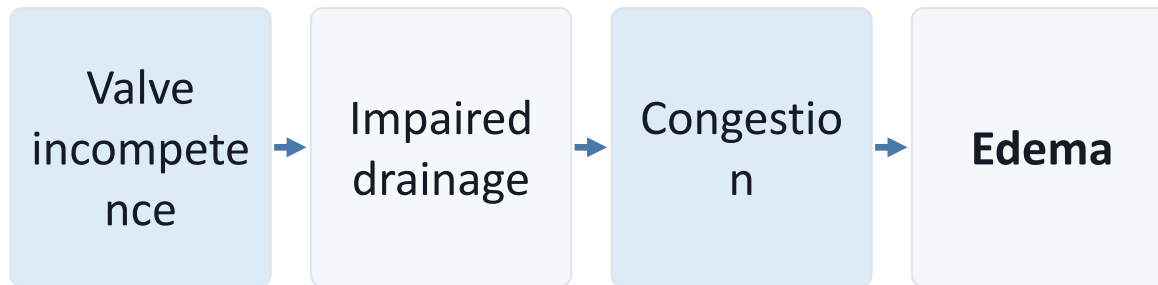


- Valve incompetence impairs effective venous drainage.
- Stasis and congestion are immediate functional consequences.
- Edema, pain, and thrombosis can follow.

Stasis and congestion are related—not identical

- Stasis denotes slowed or impaired venous blood movement.
- Congestion denotes accumulation of blood from impaired venous drainage.
- Both participate in lower-limb symptoms and tissue injury.

Why dependent edema accompanies varicosities

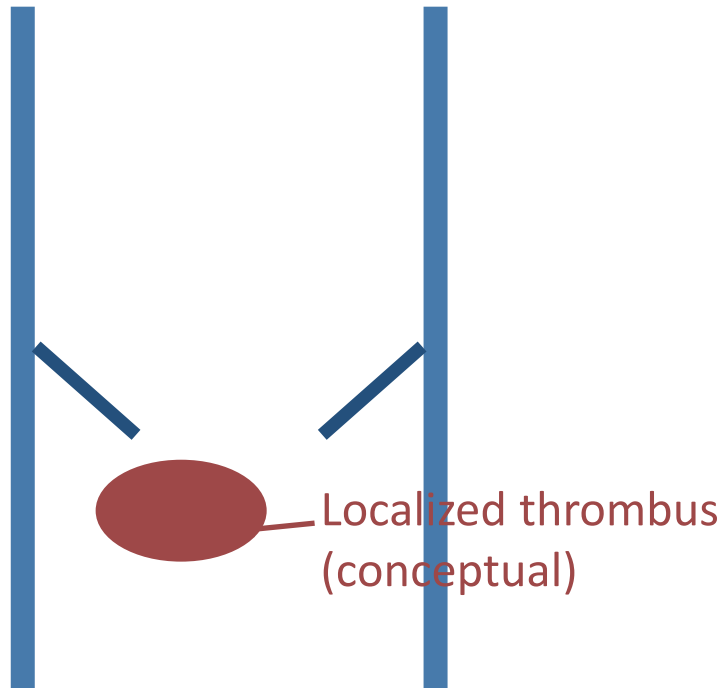


- Ineffective venous drainage promotes congestion.
- Fluid accumulation produces edema in affected tissues.
- Edema is a clinical manifestation of the underlying hemodynamic disturbance.

Pain is part of the clinical phenotype

- Pain among the consequences of valvular incompetence.
- Pain should be interpreted alongside visible varices, edema, and other complications.
- Pain alone does not distinguish varicose disease from deep venous thrombosis.

Thrombosis can complicate stagnant superficial veins



Schematic

- Stasis provides a local setting in which thrombosis can occur.
- The text lists thrombosis among consequences of incompetent valves.
- Superficial thrombosis must be distinguished from thrombosis of deep veins.

Superficial and deep venous disease differ

Feature	Superficial varicose veins	Deep-vein thrombosis
Core process	Chronic dilatation and valve incompetence	Thrombus formation in deep veins
Common consequences	Stasis, edema, skin injury	Potential pulmonary embolism
Embolism	Very rare from superficial varices	Relatively frequent from thrombosed deep veins

The embolic-risk distinction is essential

Superficial varicosity

Local stasis and tissue
injury
Embolism: Very rare

Deep-vein thrombosis

Major source of
pulmonary
Thromboembolism

- Pulmonary embolism from the described superficial varicosities is very rare.
- Thrombosed deep veins are a much more important source of thromboemboli.
- Never use a superficial varix as proof that deep venous thrombosis is absent.

Case: Visible tortuous veins and leg heaviness

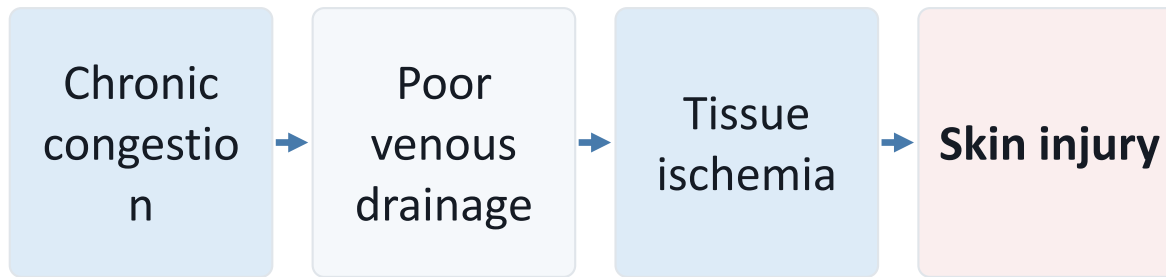
Hypothetical teaching case

An adult with obesity reports long periods standing. The lower-leg superficial veins are enlarged and tortuous, with dependent swelling.

Reasoning prompts

- Which factors increase venous loading?
- What structural change precedes valve failure?
- How is edema related to the venous lesion?

Persistent venous congestion impairs tissue oxygenation

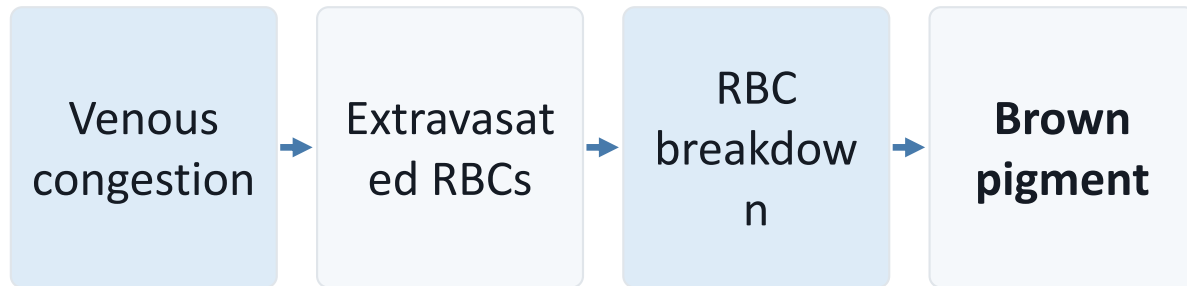


- Chronic venous congestion and poor drainage produce secondary tissue ischemia.
- The mechanism concerns impaired local tissue oxygen delivery despite venous blood pooling.
- Cutaneous damage becomes a downstream manifestation.

Stasis dermatitis follows chronic venous congestion

- Cutaneous inflammation and chronic change accompany impaired venous drainage.
- The term stasis dermatitis for this consequence.
- The characteristic discolored, indurated appearance is described as brawny induration.

Why the skin becomes brown and indurated



- Chronic venous hypertension favors red-cell extravasation into tissue.
- Breakdown of extravasated erythrocytes produces iron pigment.
- The pigment contributes to the brawny discoloration of stasis dermatitis.

Hemosiderin links congestion to visible pigmentation

**Venous
microenvironment**
Congestion and
vascular leakage



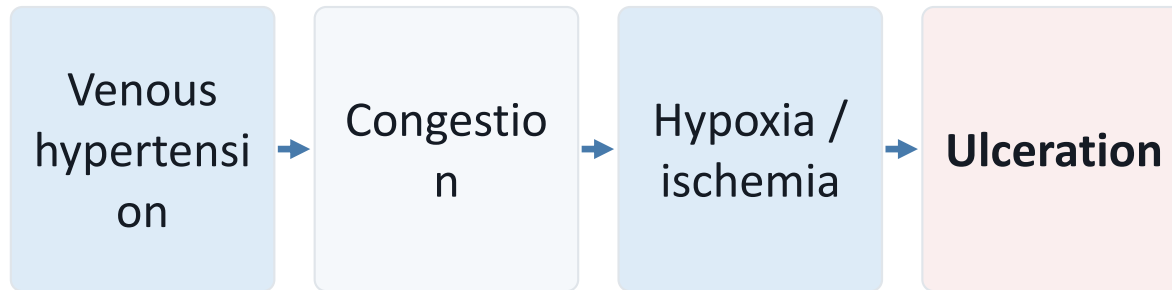
Tissue response
Red-cell breakdown
→ iron pigment

- Hemosiderin deposits in venous leg ulcers.
- The iron pigment is generated by breakdown of extravasated red cells.
- Deposits can coexist with chronic inflammation in damaged skin.

Dermatitis and ulceration are linked stages

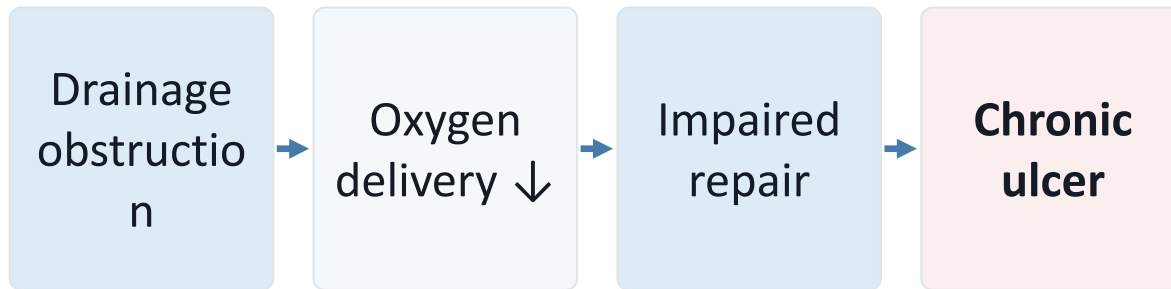
Change	Pathologic meaning	Clinical implication
Stasis dermatitis	Chronic skin effects of venous congestion	Brawny discoloration / induration
Venous ulcer	Loss of surface integrity in poorly perfused tissue	Difficult healing and infection risk

Why chronic venous disease can ulcerate



- Poor venous drainage creates secondary tissue ischemia.
- Sustained tissue injury may lead to skin ulceration.
- Severe varicose veins are one setting for venous leg ulcers.

A venous ulcer is a failure of local repair



- Poor healing to inadequate oxygen delivery to the wound.
- Obstructed venous drainage is listed as a factor impairing repair.
- Ulcers may persist while the underlying circulatory problem remains.

Venous ulcers are not caused by varices alone

- Chapter 3 also names congestive heart failure as a cause of chronic venous hypertension.
- The immediate pathway is venous pressure elevation and poor tissue oxygenation.
- An ulcer pattern identifies a mechanism, not always a single unique cause.

Venous and arterial ulcers: Different upstream lesions

Question	Venous leg ulcer	Arterial ulcer
Primary disturbance	Chronic venous hypertension	Atherosclerotic arterial insufficiency
Tissue problem	Poor oxygen delivery / impaired drainage	Ischemia, atrophy, necrosis
Source emphasis	Often severe varicosity or CHF	Commonly peripheral arterial disease, often with diabetes

Diabetic ulcers introduce additional mechanisms

Venous ulcer	Diabetic foot ulcer
Impaired venous drainage and chronic hypertension	Combination of vascular ischemia, neuropathy, and secondary infection
Hemosiderin deposition may accompany injury	Necrosis and failed healing occur in feet/lower extremities
Mechanisms may coexist clinically; the Robbins categories are instructive, not mutually exclusive.	The description does not establish diagnostic criteria or a fixed location for every lesion.

A pressure sore is another distinct chronic wound

Venous leg ulcer	Pressure sore
Pressure within a congested venous system	External compression of tissue against bone
Impaired venous drainage and tissue oxygen delivery	Local ischemia from sustained mechanical pressure
Varicose veins may be causal	Limited mobility and prolonged compression are typical settings

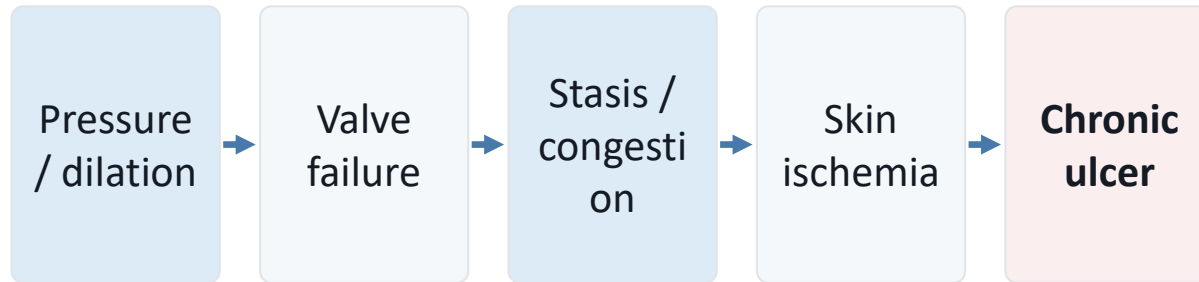
Chronic wound mechanisms at a glance

Wound type	Dominant injury mechanism	Robbins-associated context
Venous	Venous hypertension; poor drainage	Severe varicosities; CHF
Arterial	Atherosclerotic ischemia	Peripheral arterial disease; diabetes
Diabetic	Ischemia + neuropathy + infection	Lower limb, particularly foot
Pressure	Sustained tissue compression	Immobility against bony prominence

Infection is a downstream complication

- Chronic venous ulcers often have impaired healing.
- Superimposed infections are recognized complications.
- An infected nonhealing wound represents an added problem beyond venous dilation.

From varicosity to chronic ulcer: Integrated chain



- Pressure elevation and wall dilation cause valve incompetence.
- Stasis and congestion generate edema and tissue ischemia.
- Dermatitis, pigmentation, ulceration, and poor healing are downstream outcomes.

Case: Lower-leg pigmentation with an ulcer

Hypothetical teaching case

An older adult with longstanding varicose veins has dependent edema, brown skin discoloration, and a persistent lower-leg ulcer.

Reasoning prompts

- What causes the brown discoloration?
- Why may the ulcer fail to heal?
- Which complications should be considered?

Case reasoning: Interpret each finding

Hypothetical teaching case

The same hypothetical patient has varicose veins, edema, brown pigmentation, and a poorly healing skin defect.

Mechanistic interpretation

- Edema: Congestion from impaired drainage.
- Brown pigment: RBC breakdown with hemosiderin.
- Ulcer: Chronic ischemic tissue injury.
- Persistence/infection: Impaired repair, secondary infection.

Varicosities also occur outside the leg

Lower limb

Valve failure,
edema,
dermatitis,
ulcers

Gastroesophageal

Portal
collaterals;
Risk of major
bleeding

Anorectal

Dilated
plexus;
Bleeding,
thrombosis,
pain

- The Robbins section discusses esophageal varices and hemorrhoids.
- Shared concept: Venous dilatation under prolonged abnormal pressure.
- Different circulatory beds create different clinical dangers.

Portal hypertension recruits alternative venous routes

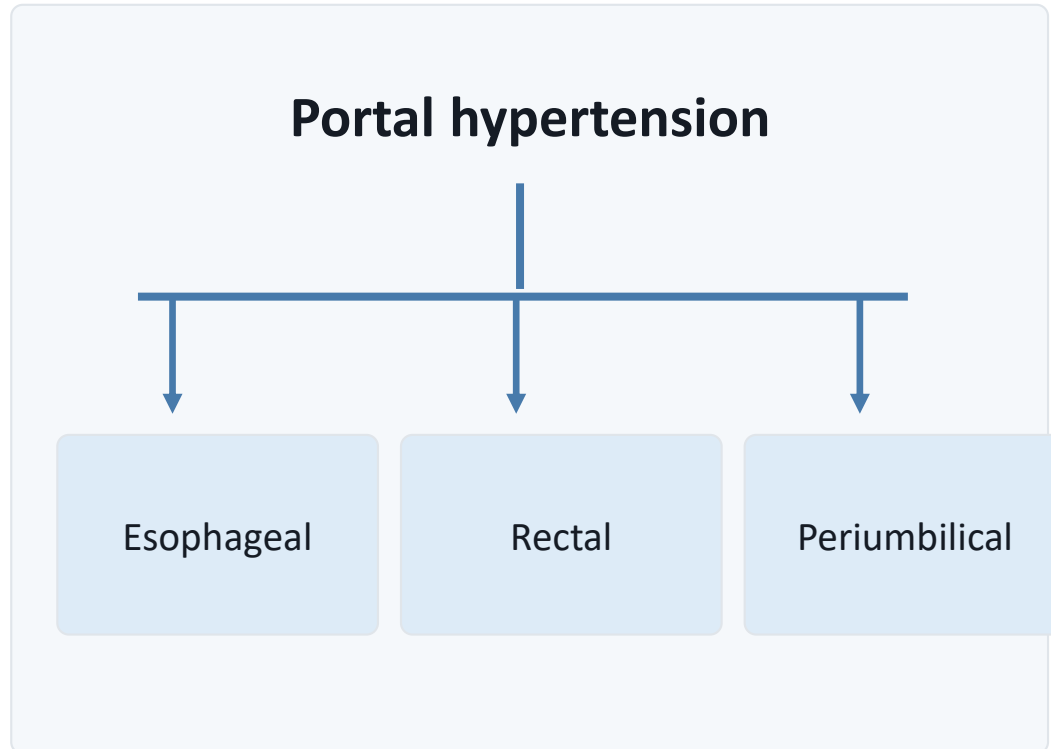


- Cirrhosis is a major cause of portal hypertension.
- Elevated portal pressure promotes opening of portosystemic shunts.
- Collateral veins enlarge in characteristic anatomical regions.

Causes leading to esophageal varices

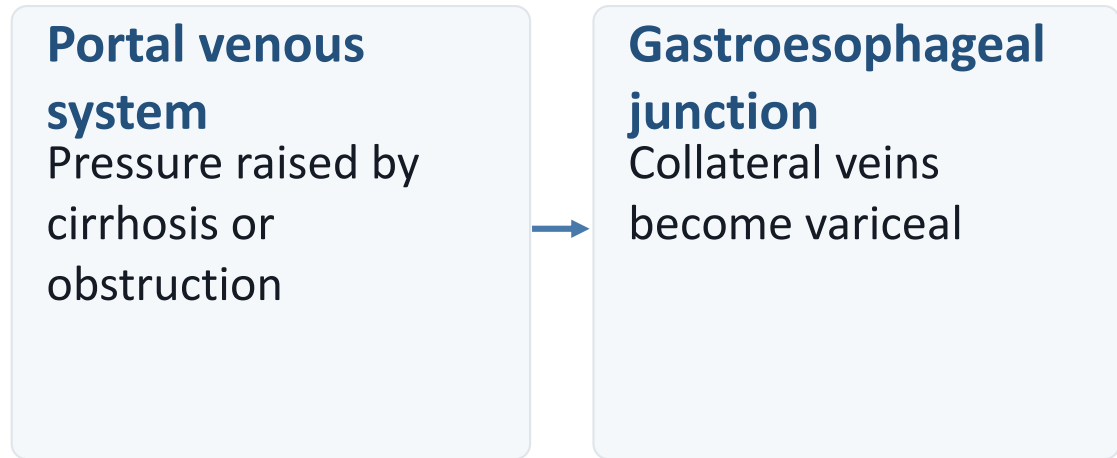
Cause described in Robbins	Mechanistic relation
Liver cirrhosis	Common cause of portal hypertension
Portal-vein obstruction	Less frequent route to portal hypertension
Hepatic-vein thrombosis	Less frequent route to portal hypertension

Three sites reflect portosystemic collateralization



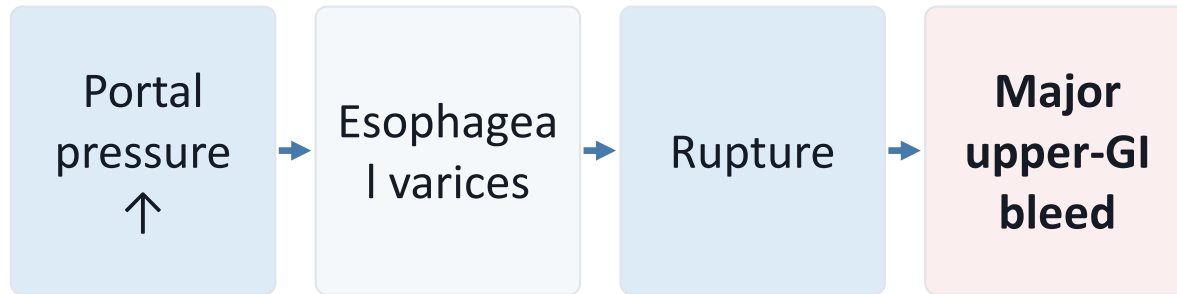
- Gastroesophageal junction: Esophageal varices.
- Rectal venous drainage: Anorectal collaterals.
- Periumbilical abdominal-wall veins: Caput medusae.

Esophageal varices are portal-systemic collaterals



- Portal hypertension increases venous flow through gastroesophageal connections.
- The enlarged veins are situated at the gastroesophageal junction.
- These differ in cause and danger from primary superficial leg varicosities.

Rupture makes esophageal varices high-risk



- Variceal rupture can release blood into the upper gastrointestinal tract.
- The resulting hemorrhage may be massive and fatal.
- This hemorrhagic hazard drives the special importance of esophageal varices.

Caput medusae is a visible collateral pattern

- Portal hypertension may open periumbilical abdominal-wall collateral veins.
- The visible dilated venous network is called caput medusae.
- It indicates a different hemodynamic territory from lower-limb superficial varicosities.

Rectal collateral vessels in portal hypertension



- Portal hypertension may increase flow through venous shunts at the rectum.
- Robbins groups this region among characteristic portosystemic collateral sites.
- The separate possibility of primary hemorrhoidal plexus dilation must also be understood.

Hemorrhoids may arise from primary plexus dilation

Anorectal venous plexus

Local vessel dilation
with venous
congestion



Hemorrhoid

Potential local
bleeding, thrombus,
and ulceration

- The anorectal junction contains a venous plexus.
- Primary varicose dilation can produce hemorrhoids.
- This may develop with prolonged pelvic venous congestion.

Pelvic pressure loading favors hemorrhoids

- Pregnancy is associated with prolonged pelvic venous congestion.
- Straining to defecate is another recognized precipitating circumstance.
- Both provide examples of pressure-related anorectal venous dilation.

Hemorrhoid complications are predominantly local

Pathologic event	Clinical manifestation
Surface / venous injury	Bleeding
Thrombosis of dilated plexus	Painful thrombotic event
Ulceration	Potentially painful surface defect

Rectal bleeding should not be attributed automatically

- Chapter 17 notes that hemorrhoids may cause bright-red rectal bleeding.
- It also emphasizes that colon cancer can coexist and must be excluded.
- Visible hemorrhoids do not conclusively explain all episodes of anorectal bleeding.

Varicosities: Mechanism and danger depend on site

Anatomic site	Driver	Dominant Robbins complication
Superficial leg	Dependent pressure; valve failure	Stasis dermatitis / venous ulcer
Esophagus	Portal pressure and shunts	Potentially fatal GI hemorrhage
Anorectal plexus	Pelvic congestion or portal pathways	Bleeding; thrombosis; painful ulceration

Case: Cirrhosis and upper-GI hemorrhage

Hypothetical teaching case

A patient with cirrhosis develops portal hypertension and then presents with severe upper gastrointestinal bleeding.

Reasoning prompts

- Which variceal site is most concerning?
- What vascular connection becomes enlarged?
- Why is this unlike a typical leg varicosity?

Case: Pregnancy and anorectal symptoms

Hypothetical teaching case

During pregnancy, a patient develops uncomfortable anorectal swelling with episodic bleeding during defecation.

Reasoning prompts

- Which venous plexus can be involved?
- What pressure-related factors are present?
- Why should bleeding not be assumed benign?

Mechanism check: Predict the next consequence

- Dilated superficial vein + failed valve closure → predict the hemodynamic change.
- Chronic congestion + red-cell extravasation → predict the pigment and skin appearance.
- Cirrhosis + portal shunting + gastroesophageal dilation → identify the feared event.

The unifying idea is pressure—but effects diverge



- Leg veins: Dilation and incompetence impair drainage and injure skin.
- Portal collaterals: Dilation creates vulnerable veins that may rupture.
- Anorectal plexus: Dilation may produce bleeding, thrombosis, and ulceration.

Core Robbins references

Robbins, Cotran & Kumar: Pathologic Basis of Disease, 11th edition (2026).

- Chapter 11, Blood Vessels: “Varicose Veins,” p. 471.
- Chapter 11: “Thrombophlebitis and Phlebothrombosis,” pp. 471–472.
- Chapter 3, Inflammation and Repair: “Defects in Healing: Chronic Wounds,” pp. 98–99.
- Chapter 17, The Gastrointestinal Tract: “Hemorrhoids,” p. 745.