

Atherosclerotic Ischemic Renal Disease

Renal vascular pathology • Mechanisms, morphology, clinical correlation

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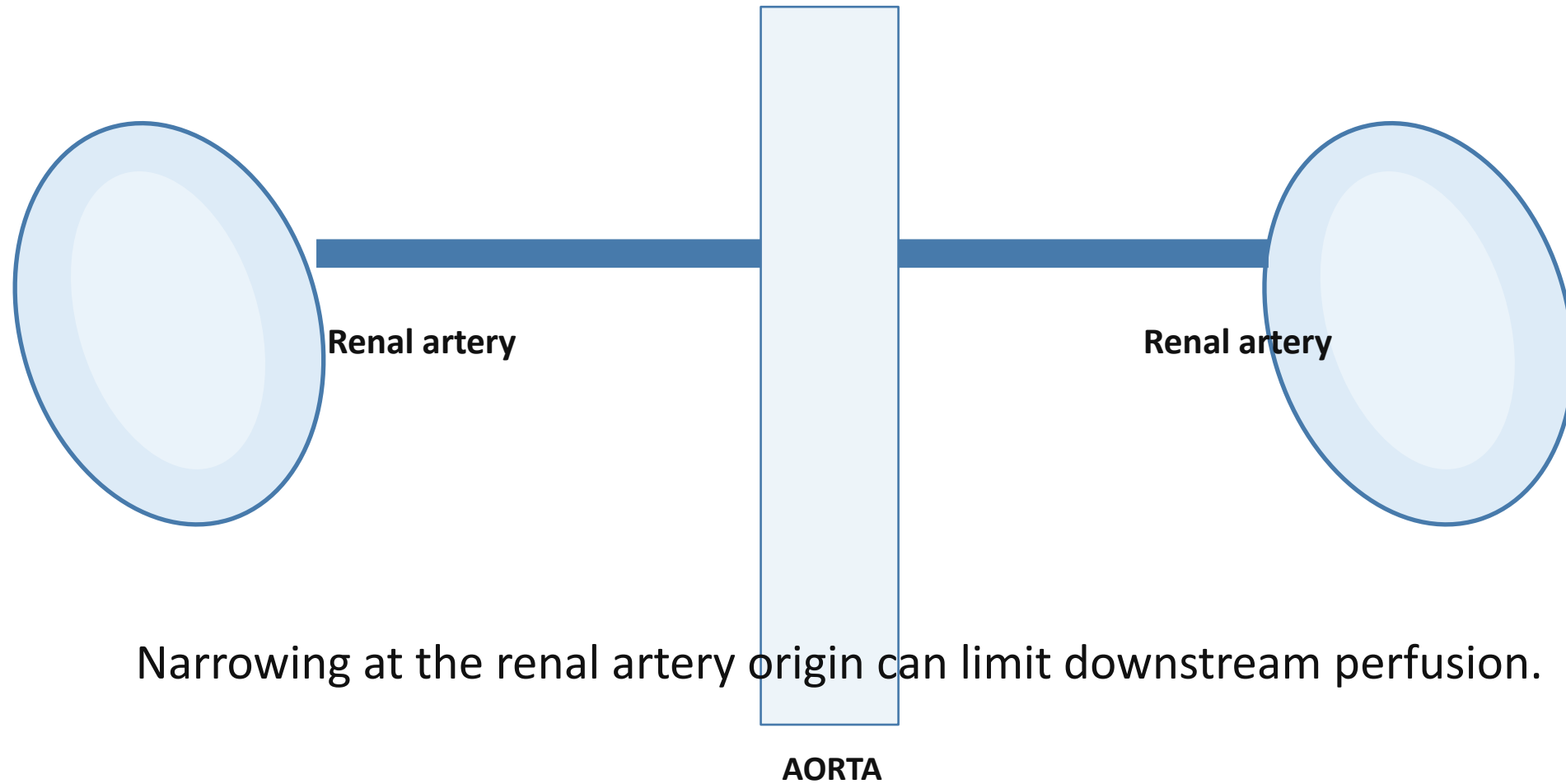
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Learning objectives

- Differentiate renovascular hypertension from ischemic renal insufficiency.
- Explain how atherosclerotic renal artery stenosis affects renal blood flow.
- Identify gross and microscopic consequences of chronic hypoperfusion.
- Distinguish large-artery stenosis from other renal vascular lesions.
- Interpret two-kidney physiology and relevant diagnostic principles.

Renal perfusion depends on large-artery inflow

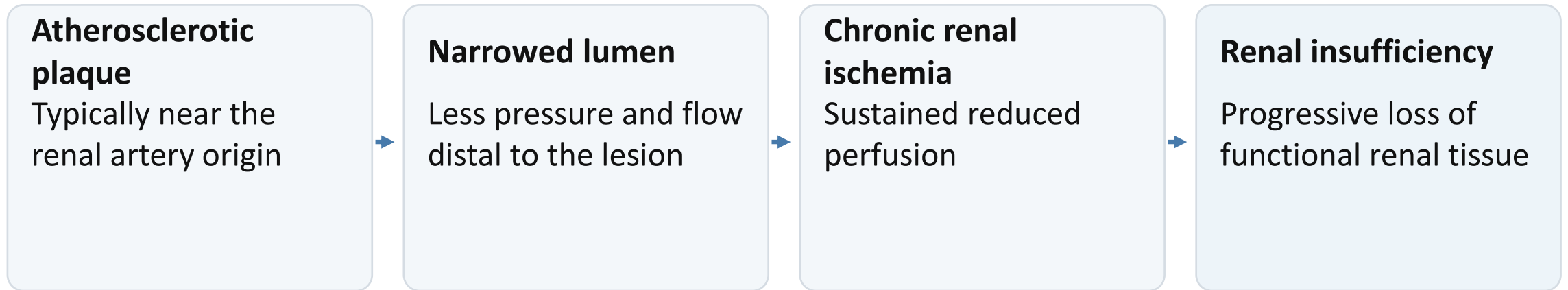


Definition and scope

Chronic renal hypoperfusion caused by atherosclerotic narrowing of the renal arteries

The clinical expression depends on lesion distribution, severity, duration, and preexisting parenchymal injury.

The central disease pathway



The same lesion can also initiate renovascular hypertension.

Two overlapping presentations

Pattern	Dominant physiologic effect	Teaching consequence
Unilateral stenosis	Renin release from an ischemic kidney	Secondary renovascular hypertension
Bilateral disease	Perfusion compromise in both kidneys	Renal insufficiency; Hypertension may be absent
Coexisting lesions	Vascular and parenchymal processes overlap	Mixed clinical and morphologic phenotype

An important diagnostic paradox

Ischemic renal insufficiency may occur without hypertension

Bilateral atherosclerotic renal artery disease can be associated with renal dysfunction even when hypertension is absent.

The characteristic clinical setting

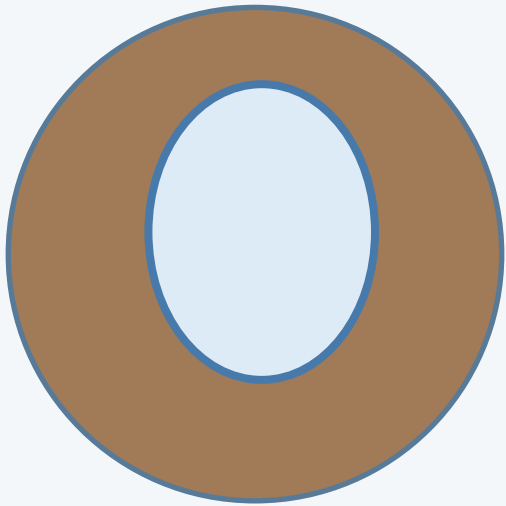
- Atherosclerotic narrowing is especially relevant in older individuals.
- The parent renal artery stenosis section describes increasing frequency with age and diabetes.
- Renal function may deteriorate gradually when both kidneys have impaired inflow.
- Hypertension is neither required nor sufficient to establish ischemic renal disease.

Atherosclerosis is the principal cause of renal artery stenosis

The typical plaque is located near the arterial origin

The Robbins source reports atherosclerotic narrowing at the renal artery origin as the most common cause of renal artery stenosis (approximately 70%).

Atherosclerotic lesions often occupy the renal artery origin



Schematic arterial cross-section

Plaque site

Near the aortic origin of the renal artery

Lumen

Narrowing reduces the perfusion pathway

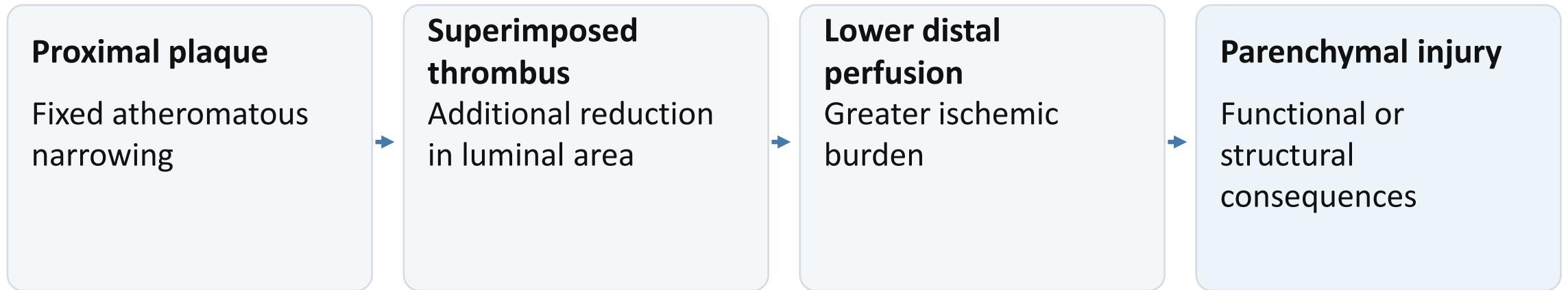
Superimposed change

Thrombosis may complicate the plaque

Ostial plaques: Important morphologic details

- The lesion often narrows the proximal artery at its origin from the aorta.
- The plaque is usually concentric.
- Thrombosis may be superimposed on the atheroma.
- Proximal inflow obstruction can affect the entire downstream kidney.

Atheroma plus thrombosis can intensify obstruction

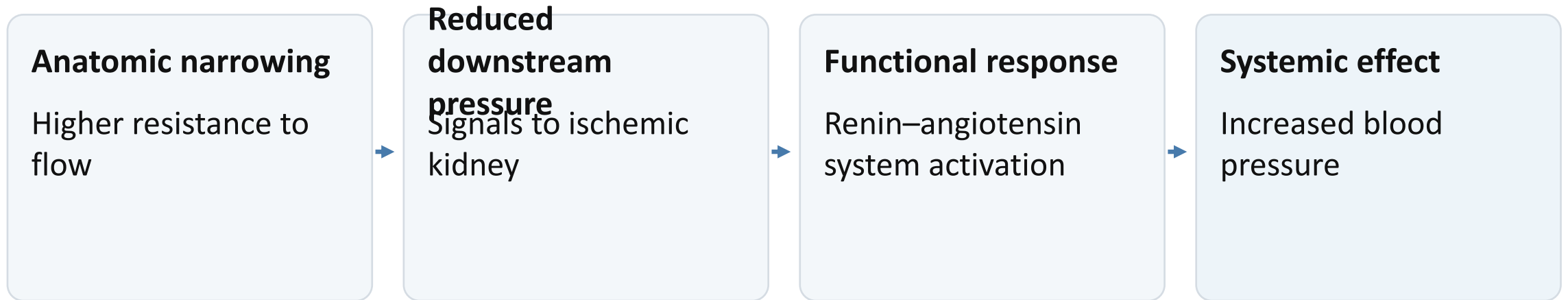


Thrombosis is described as a possible superimposed change, not a universal event.

Atherosclerosis versus fibromuscular dysplasia

Feature	Atherosclerosis	Fibromuscular dysplasia
Age setting	Typically older patients	Typically younger adults
Sex association	More frequent in males	More frequent in females
Lesion	Atheromatous plaque near origin	Fibrous/fibromuscular arterial thickening
Distribution	Often proximal ostial lesion	Can involve intima, media or adventitia

Stenosis creates a perfusion-pressure problem

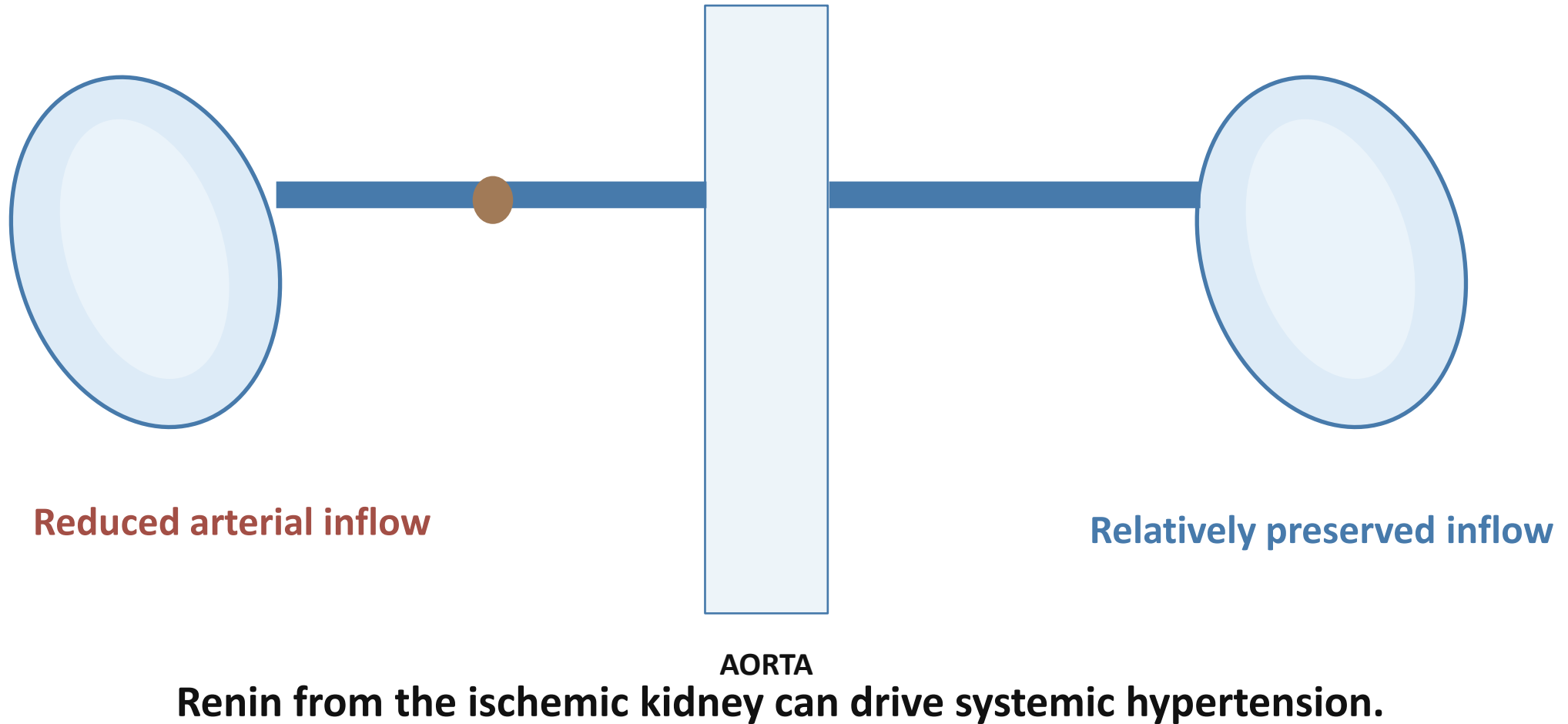


Systemic blood pressure does not equal intrarenal perfusion

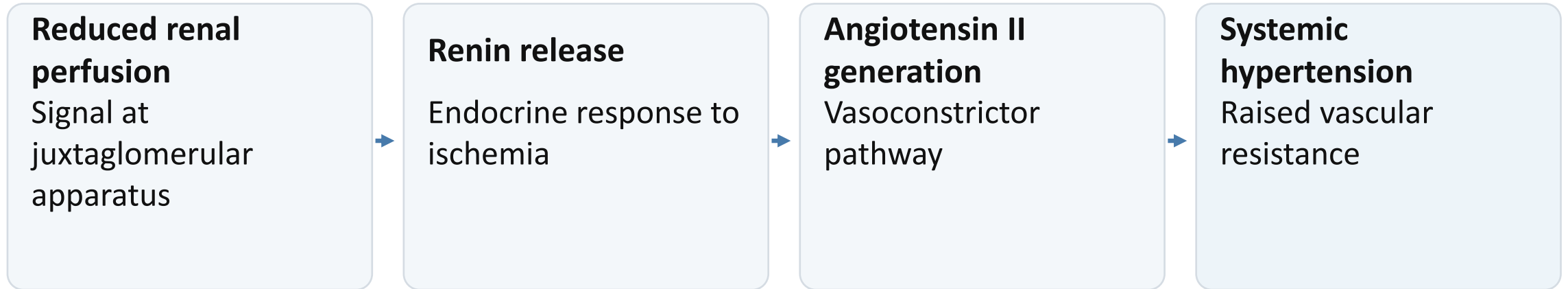
An elevated systemic pressure can coexist with a poorly perfused kidney

A proximal arterial lesion can separate pressure in the aorta from pressure experienced by the downstream renal circulation.

Unilateral stenosis: Asymmetrical perfusion and pressure effects



The ischemic kidney activates renin secretion



Why renin matters in renovascular hypertension

- Increased renin production by the ischemic kidney as the principal initiating mechanism.
- Renin secretion activates the angiotensin pathway.
- Angiotensin II is a potent vasoconstrictor.
- The pressure response depends on the extent of arterial narrowing.

Renin–angiotensin sequence

Renal ischemia

Juxtaglomerular renin secretion



RAAS activation

Downstream hormonal cascade



Angiotensin II

Systemic arteriolar constriction



Hypertension

Rise in arterial pressure

Angiotensin II is the early vasoconstrictor effector

Arterial constriction raises systemic vascular resistance

Angiotensin II in the initial elevation of blood pressure following renal artery constriction.

Sodium retention contributes to sustained hypertension

- Renin–angiotensin activation initiates the pressure rise.
- Sodium retention as a contributor to the maintenance of hypertension.
- Long-standing hypertension need not remain dependent on one isolated signal.
- Mechanisms of pressure maintenance should be distinguished from the stenotic lesion itself.

Goldblatt physiology links stenosis to blood pressure

Renal artery constriction

Experimental change in inflow



Ischemic kidney

Release of renin



Angiotensin II

Hormone-mediated vasoconstriction



Blood pressure rise

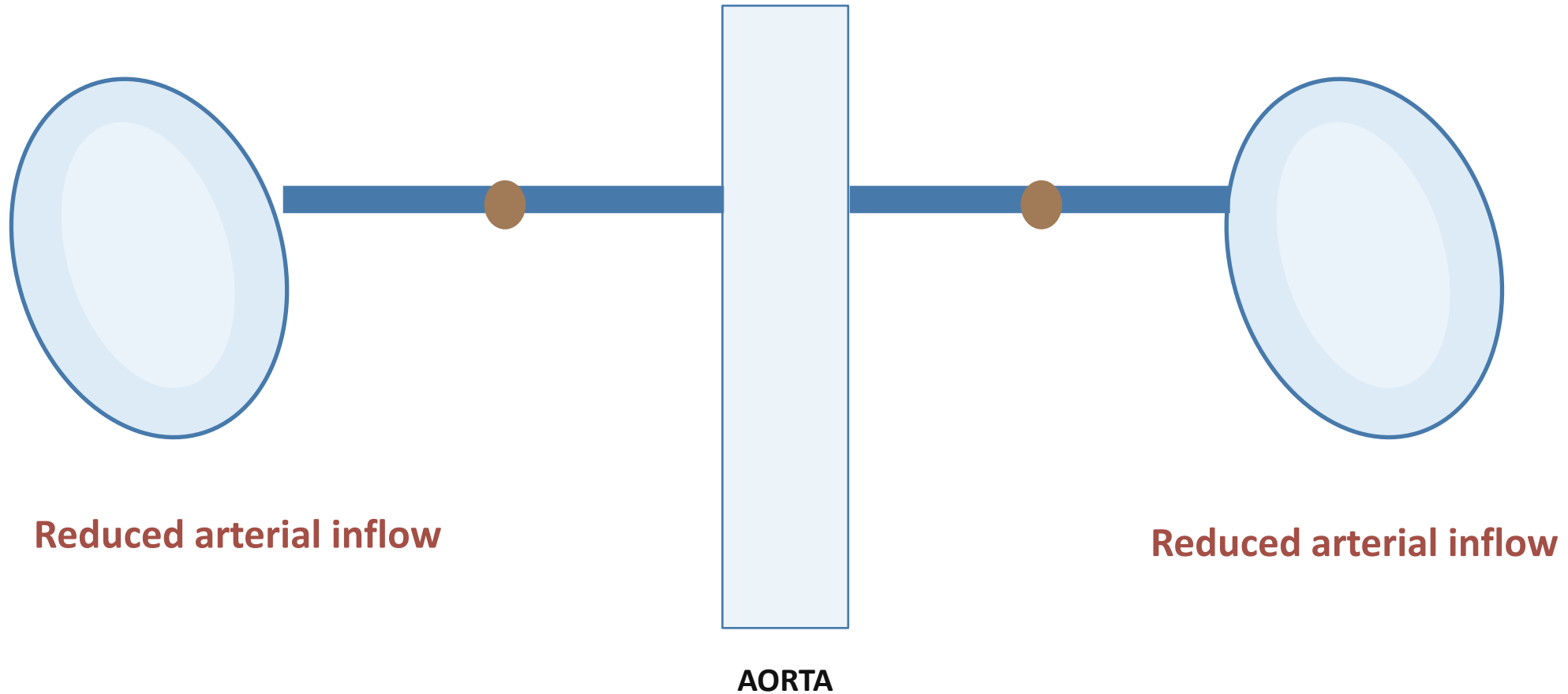
Effect scales with narrowing

The stenosis–hypertension relationship is not purely structural

A vascular lesion initiates endocrine and hemodynamic adaptation

Renin–angiotensin activation as an initiating mechanism, with other factors potentially maintaining hypertension.

Bilateral stenosis: Diminished inflow to both kidneys



Chronic ischemia can impair total renal function.

Bilateral stenosis threatens total functional renal mass

Both renal arteries affected

Inflow to both kidneys impaired



Sustained hypoperfusion

Chronic parenchymal ischemia



Atrophy and scarring

Reduced functional nephron mass



Renal insufficiency

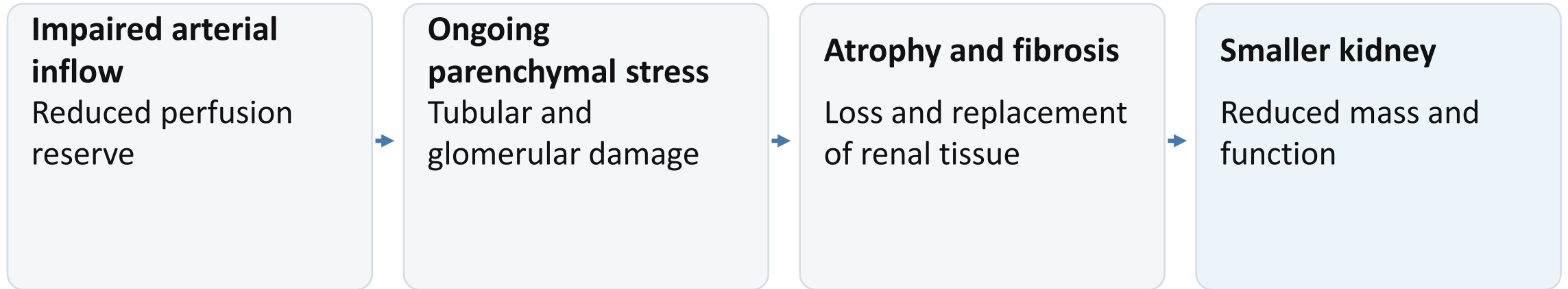
Declining aggregate function

Why hypertension can be absent in bilateral disease

Renal insufficiency is a perfusion-related outcome, not a diagnostic synonym for hypertension

Robbins explicitly separates chronic ischemic renal insufficiency from the obligatory presence of hypertension.

Persistent ischemia produces chronic renal injury



The ischemic kidney becomes smaller

- The ischemic kidney as reduced in size.
- Diffuse atrophy reflects loss of viable renal parenchyma.
- Tubular atrophy and interstitial fibrosis are central correlates.
- Kidney size alone cannot identify the precise cause of chronic injury.

Renal atrophy is a tissue consequence, not a plaque image

A small kidney demonstrates chronic loss of parenchyma

Direct demonstration of the arterial narrowing and evaluation of competing causes
are distinct diagnostic tasks.

Glomerular crowding reflects loss of surrounding parenchyma

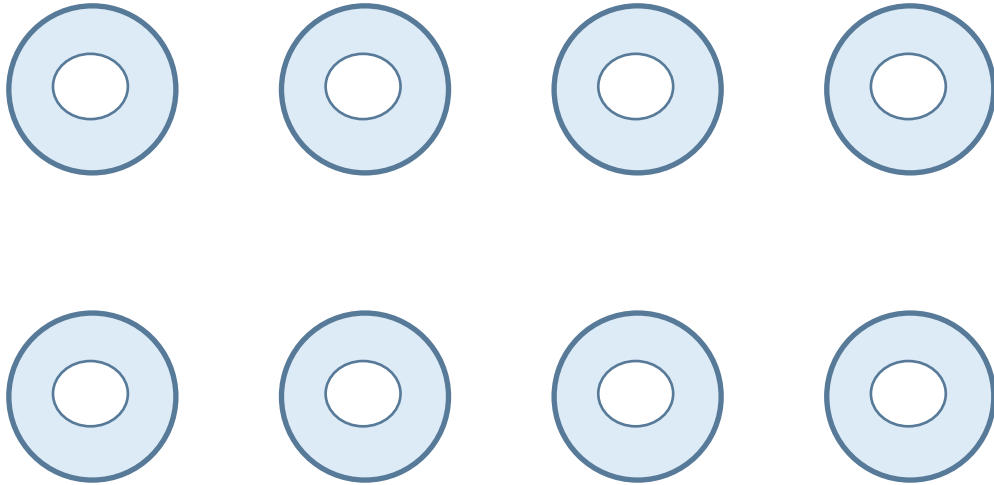


Illustration only — not tissue microscopy

Atrophic ischemic kidney

Reduced tubular volume

Glomeruli appear closer together

Tubular atrophy is a major component of chronic ischemia

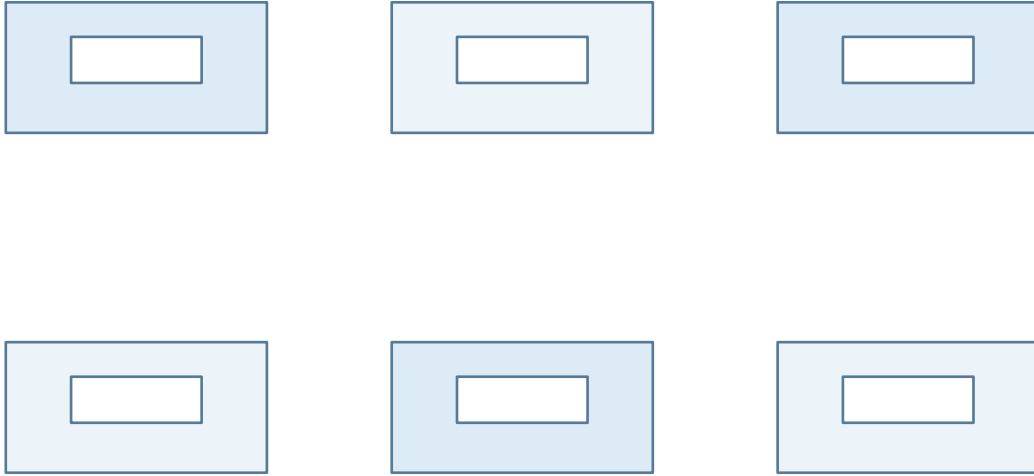


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Sustained reduced flow

Tubular epithelial loss and shrinkage

Reduced tubular profiles and
function

Interstitial fibrosis records chronic parenchymal damage

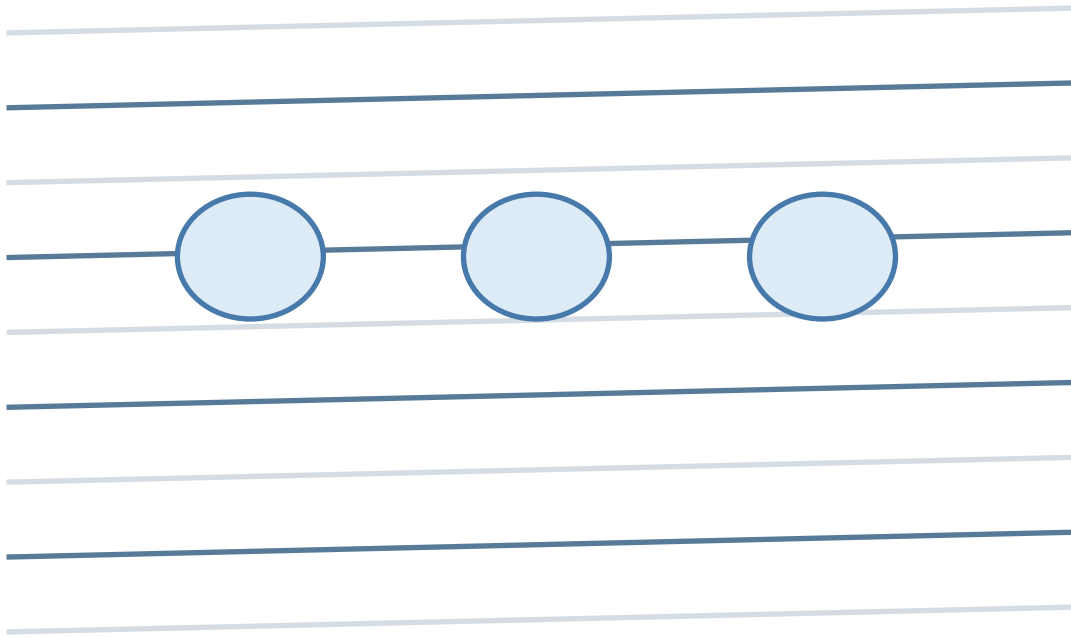


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Prolonged vascular insufficiency

Tubulointerstitial injury

Fibrous replacement of lost tissue

Focal inflammatory infiltrates may accompany chronic injury

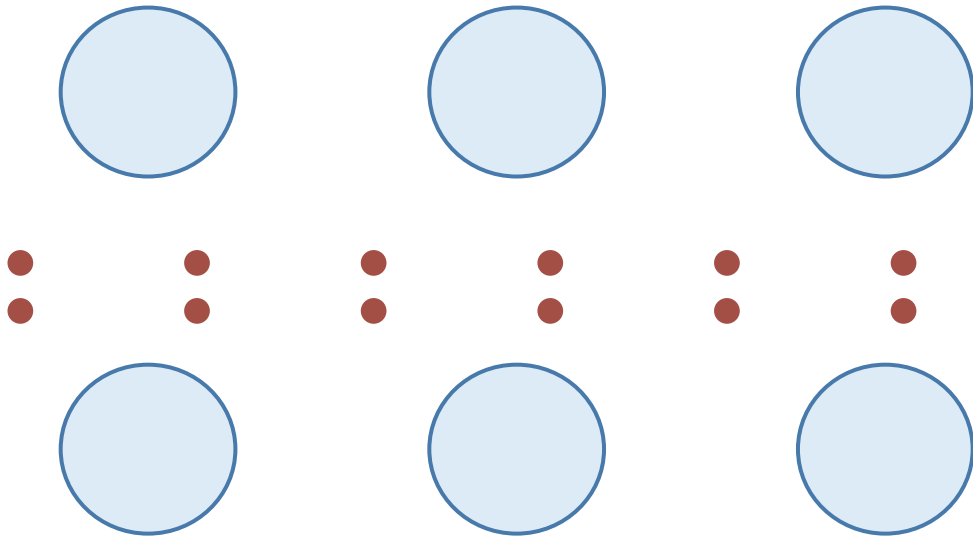


Illustration only — not tissue microscopy

Chronic ischemic injury

Interstitial response

Focal inflammatory infiltrates

Arterioles downstream may be relatively protected

- The stenotic kidney receives less of the systemic pressure load.
- Its arterioles commonly show only mild arteriolosclerosis.
- This can coexist with pronounced parenchymal atrophy.
- Severity of pressure-related arteriolar change need not parallel ischemic tissue loss.

The contralateral kidney experiences a different hemodynamic environment

The well-perfused kidney may show more severe arteriolosclerosis

The severity depends on the hypertension experienced by the contralateral kidney.

Two kidneys in unilateral renovascular hypertension

Tissue feature	Stenotic kidney	Contralateral kidney
Pressure exposure	Reduced by upstream stenosis	Exposed to systemic hypertension
Size and atrophy	Often small and diffusely atrophic	Not necessarily ischemic from this lesion
Arteriolosclerosis	May be comparatively mild	May be more severe
Dominant driver	Hypoperfusion	Pressure-related vascular injury

The morphologic triad is not etiologically specific

Glomerular changes + tubular atrophy + interstitial fibrosis

Atherosclerotic ischemic disease must be interpreted together with arterial disease and the distribution of renal injury.

Nephrosclerosis overlaps with chronic ischemic renal injury

- Nephrosclerosis includes glomerulosclerosis, tubulointerstitial scarring, arteriosclerosis and arteriolosclerosis.
- Hypertension and age are important associations, but not exclusive explanations.
- Small-vessel narrowing produces focal renal ischemia.
- Large-artery atherosclerotic stenosis represents a different anatomic level of perfusion limitation.

Nephrosclerosis can amplify parenchymal ischemia

Intimal and medial thickening

Narrowing of small arteries



Arteriolar hyalinization

Further luminal reduction



Focal hypoperfusion

Ischemic nephron injury



Scarring

Glomerulosclerosis and tubular loss

Hyaline arteriolosclerosis acts within the microcirculation

Protein-rich hyaline material thickens arteriolar walls

The narrowed lumens contribute to focal parenchymal ischemia. This is distinct from an ostial renal artery plaque.

Fibroelastic hyperplasia affects intrarenal arteries

- Interlobular and arcuate arteries can show medial hypertrophy.
- Replication of the internal elastic lamina and intimal tissue accumulation narrow lumens.
- These changes increase with age and hypertension.
- Unlike renal artery ostial atheroma, this is an intrarenal vessel-wall process.

From microvascular hypoperfusion to glomerular loss

Small-vessel narrowing

Focal reduction in
perfusion



Glomerular ischemia

Capillary tuft and
GBM changes



Sclerosis

Collagen and nephron
loss



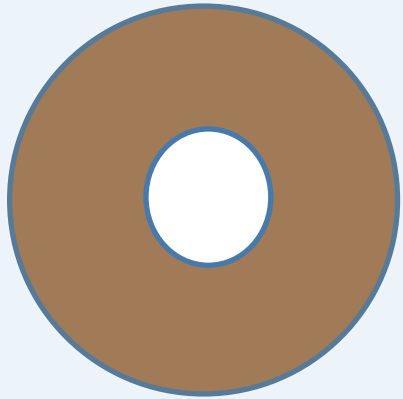
Tubular dropout

Regional interstitial
scar

Chronic atrophy versus renal infarction

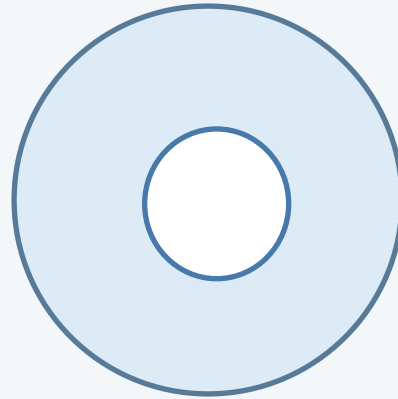
Feature	Chronic ischemic atrophy	Renal infarction
Time course	Persistent or progressive hypoperfusion	Abrupt vascular occlusion
Morphology	Diffuse or patchy atrophy and fibrosis	Coagulative necrosis and later scar
Gross pattern	Renal shrinkage	Classically wedge-shaped pale lesion
Clinical link	Progressive renal insufficiency	May be painful or clinically silent

Large-vessel stenosis and microvascular narrowing act at different levels



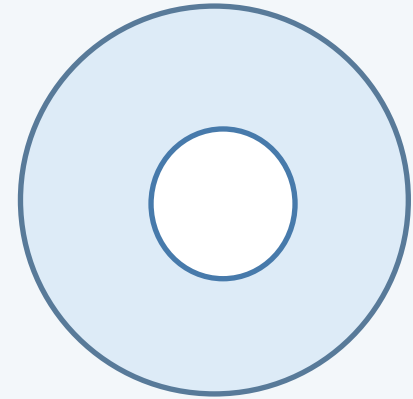
Renal artery

Ostial atheromatous plaque



Small artery

Fibroelastic intimal changes



Arteriole

Wall hyalinization

Clinical expression is often nonspecific

- Older patients with renal artery stenosis may resemble those with essential hypertension.
- Atherosclerotic bilateral disease may present primarily as renal insufficiency.
- Symptoms and examination alone do not define arterial anatomy.
- Diagnostic interpretation must connect vascular findings with renal functional impairment.

No classic symptom reliably establishes renal artery stenosis

Clinical suspicion and anatomic localization are separate steps

That distinctive clinical features are few, even in renovascular hypertension.

A renal bruit is a possible clue, not proof

- A bruit may occasionally be heard over an affected kidney.
- Auscultation does not locate or grade an arterial lesion.
- Absence of a bruit is not an exclusion criterion.
- A bruit belongs in a broader evaluation rather than functioning as a stand-alone diagnosis.

Renin testing reflects physiology, not arterial anatomy

- Renovascular hypertension is initiated by renin from the ischemic kidney.
- Elevated plasma or renal-vein renin as potentially helpful.
- Renin values do not, by themselves, demonstrate the location of stenosis.
- Endocrine and structural assessments address different questions.

Response to angiotensin blockade supports pathway involvement

Functional response is not equivalent to lesion localization

Blood pressure reduction with medications that block angiotensin II activity in renovascular hypertension; This slide is mechanistic, not prescribing advice.

Anatomic and functional evaluation answer different questions

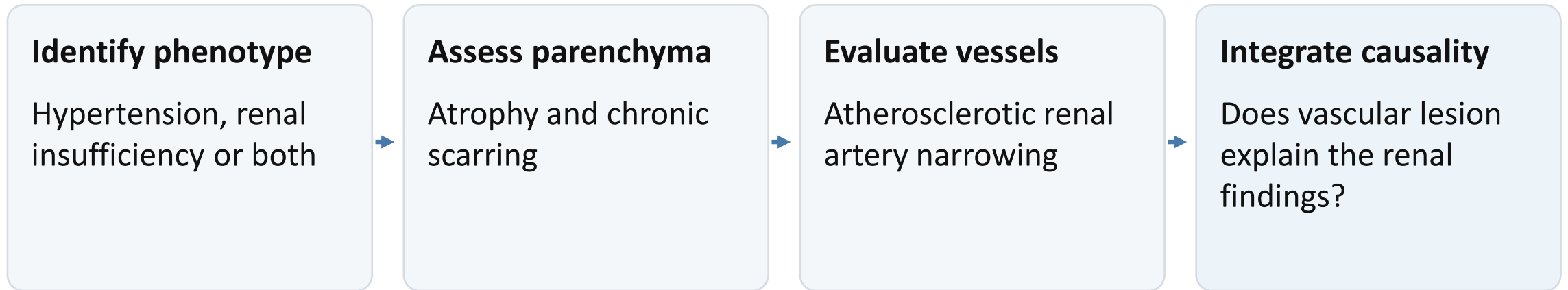
- Morphology suggests the effect of chronic vascular insufficiency on parenchyma.
- Renal scans and intravenous pyelography are mentioned clinical discussion.
- These tests are described as diagnostic aids, not as histologic evidence.
- The cited source uses arteriography for definitive anatomic localization.

Arteriography localizes a stenotic arterial lesion

A demonstrable narrowing establishes anatomy; Attribution of renal dysfunction requires interpretation

Arteriography as necessary to localize stenosis in its renal artery stenosis discussion and commonly used for bilateral disease.

Clinicopathologic diagnostic synthesis



Do not assume every detected stenosis is causative.

Recognizing revascularizable disease matters

- Surgical revascularization can prevent further loss of renal function in selected ischemic disease.
- This is a conditional statement of potential benefit, not a universal indication.
- Established parenchymal scarring and competing injury may limit functional recovery.
- Contemporary procedural decisions require evidence and guidelines beyond this textbook excerpt.

Vascular correction and parenchymal recovery are not synonymous

**Restoring arterial inflow does not automatically reverse
established renal scarring**

This is a pathophysiologic inference from the parent morphology section; Robbins gives no universal recovery guarantee for ischemic renal disease.

Differential: Atherosclerotic stenosis and fibromuscular dysplasia

Axis	Atherosclerotic disease	Fibromuscular dysplasia
Typical context	Older; Age and diabetes association	Younger; Female predominance
Wall pathology	Atheromatous plaque	Fibrous/fibromuscular thickening
Usual mechanism	Large-artery inflow limitation	Large-artery inflow limitation
Interpretation	Can impair bilateral renal function	More often taught as a cause of renovascular HTN

Differential: Fixed stenosis and atheroembolic disease

Axis	Atherosclerotic ischemic disease	Atheroembolic renal disease
Vascular process	Fixed renal artery narrowing	Plaque material embolizes distally
Anatomic site	Proximal renal artery	Intrarenal arterial branches
Histologic clue	Chronic parenchymal atrophy	Cholesterol-cleft emboli
Clinical setting	Chronic ischemic insufficiency	Often after arterial instrumentation

Differential: Large-artery disease and thrombotic microangiopathy

Axis	Atherosclerotic ischemia	TMA
Principal level	Renal artery inflow lesion	Capillaries and arterioles
Characteristic injury	Chronic hypoperfusion and atrophy	Microthrombi and endothelial injury
Associated pattern	Renovascular HTN or renal insufficiency	Hemolysis, thrombocytopenia, ischemia
Specific mechanism	Atheromatous narrowing	Diverse toxin/complement/platelet pathways

Differential: Chronic ischemia and acute infarction

Axis	Chronic atherosclerotic hypoperfusion	Renal infarct
Vascular event	Sustained stenosis	Usually embolic occlusion
Parenchymal lesion	Atrophy, fibrosis and renal shrinkage	Wedge-shaped ischemic coagulative necrosis
Clinical course	Can be insidious	May cause costovertebral pain and hematuria
Pathologic point	Viable but chronically injured nephrons	Established necrosis and subsequent scarring

Case 1: Asymmetric renovascular hypertension

Clinical vignette

An older patient with hypertension has severe proximal narrowing of one renal artery. The affected kidney is smaller; Its intrarenal arteriolar disease is comparatively mild.

Interpret

How can severe hypertension coexist with relatively little arteriolosclerosis in the affected kidney?

Explanation

Upstream stenosis reduces pressure exposure in that kidney; Renin release raises systemic blood pressure, affecting the contralateral kidney.

Case 2: Renal insufficiency without hypertension

Clinical vignette

An older individual has slowly declining renal function and bilateral atherosclerotic renal artery disease, but does not have sustained hypertension.

Interpret

Does the absence of hypertension rule out ischemic renal disease? Which mechanism is most relevant?

Explanation

No. Bilateral compromised inflow can produce chronic renal ischemia and insufficiency even without hypertension.

Case 3: Contrasting renal vascular lesions

Clinical vignette

A patient has unilateral renal artery narrowing, a small ischemic kidney, and pronounced arteriolosclerosis in the opposite kidney.

Interpret

Which lesion is attributed mainly to hypoperfusion, and which to systemic pressure?

Explanation

Ischemic atrophy is expected in the stenotic kidney; More marked pressure-related arteriolosclerosis may develop contralaterally.

Synthesis: Three useful vascular patterns

Pattern	Primary lesion	Dominant pathology
Renal artery atheroma	Proximal large-vessel narrowing	Renin-driven hypertension or chronic ischemia
Hypertensive nephrosclerosis	Arteries and arterioles	Patchy ischemic atrophy and glomerulosclerosis
Atheroembolism	Distal arterial plaque fragments	Cholesterol-cleft emboli; Variable AKI

Final clinicopathologic synthesis

- Atherosclerotic renal artery narrowing often occurs near the arterial origin.
- Unilateral disease may produce renin-mediated hypertension.
- Bilateral disease can cause chronic renal insufficiency even without hypertension.
- Chronic ischemia produces renal shrinkage, tubular atrophy and interstitial fibrosis.
- Distinguish fixed stenosis from nephrosclerosis, atheroembolism and infarction.

Primary source and interpretation boundaries

Robbins, Cotran & Kumar: Pathologic Basis of Disease

11th edition (2026) • Chapter 20: The Kidney

Atherosclerotic Ischemic Renal Disease — p. 855

Parent sections: Nephrosclerosis (pp. 851–852) and Renal Artery Stenosis (pp. 852–853).

Adjacent differential context: Atheroembolic Renal Disease and Renal Infarcts.

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