

PYLORIC STENOSIS

Mechanisms, morphology and clinicopathologic diagnosis

Congenital hypertrophic and acquired disease

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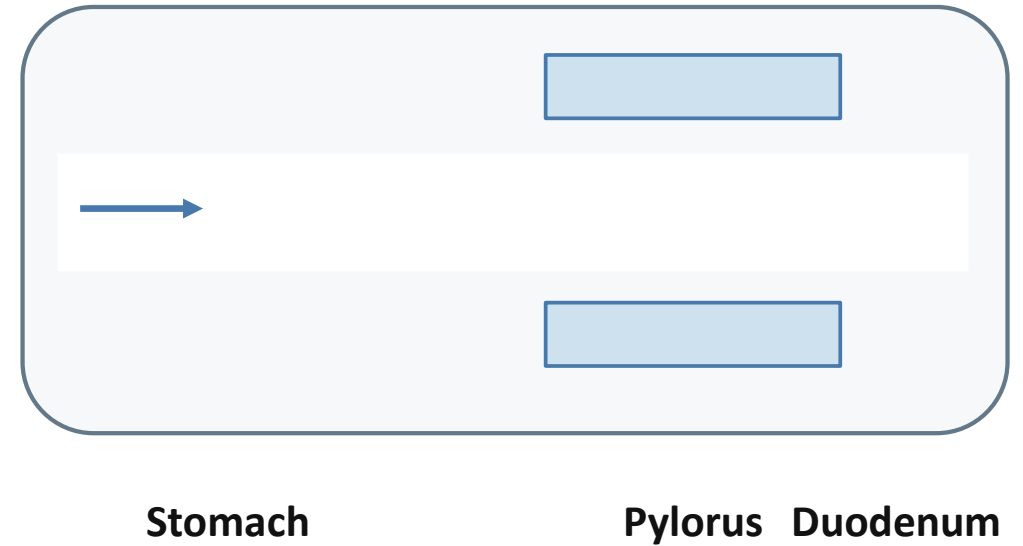
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Learning objectives

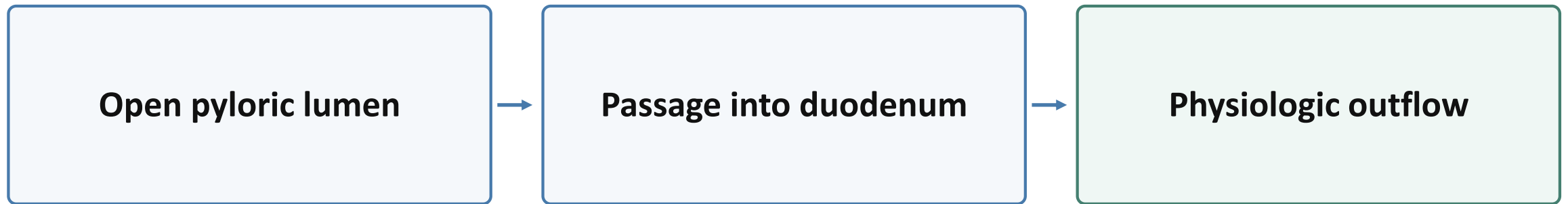
- Differentiate congenital hypertrophic stenosis from acquired pyloric narrowing.
- Explain how muscularis propria hyperplasia causes infantile gastric outlet obstruction.
- Connect the age profile, emesis, peristaltic waves and palpable “olive” to pathology.
- Interpret the anatomical rationale for ultrasonography and pyloromyotomy.
- Identify inflammatory, fibrotic and malignant causes of adult acquired disease.

The pylorus is the gastric outlet

- Pylorus links the distal stomach to the duodenum.
- The pyloric muscular wall surrounds the narrow outlet channel.
- Changes in its wall can restrict the passage of gastric contents.



Outlet caliber controls gastric emptying



- When the outlet narrows, the same gastric contents face greater resistance.

Two routes to pyloric stenosis

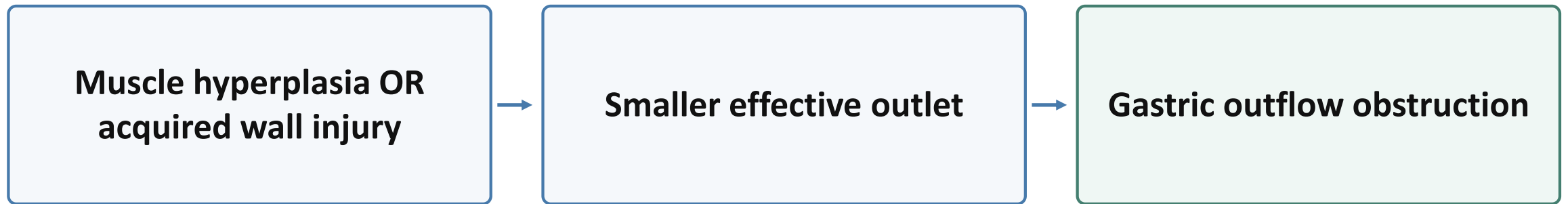
Congenital form

- Typically infantile hypertrophic pyloric stenosis.
- Dominant pathology: Hyperplasia of pyloric muscularis propria.
- Characteristic early-infant presentation.

Acquired form

- Usually considered in adults.
- Inflammation, ulcer-associated scarring or malignant disease.
- Luminal narrowing through fibrosis or infiltration.

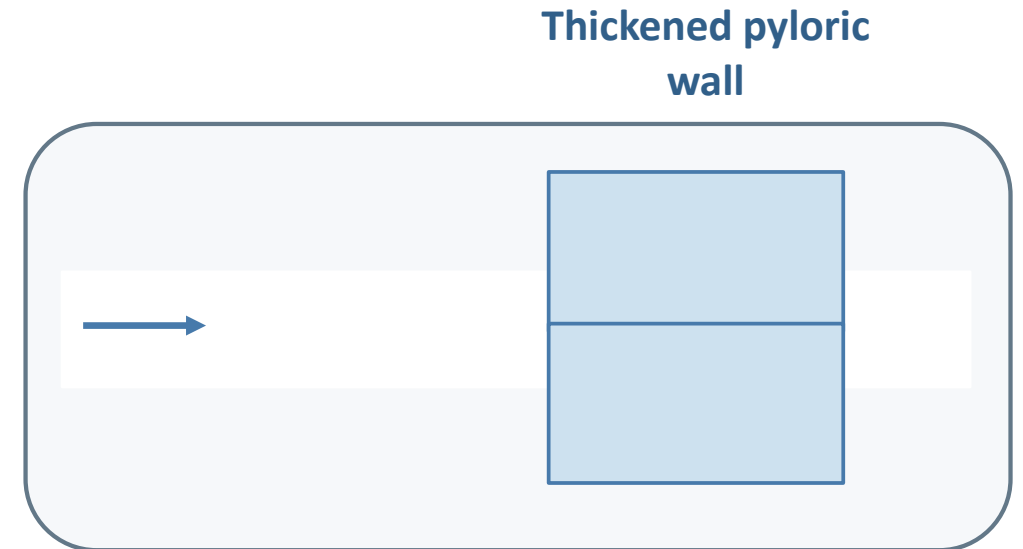
Different lesions, one shared endpoint



- Identical anatomical location does not imply identical cause.

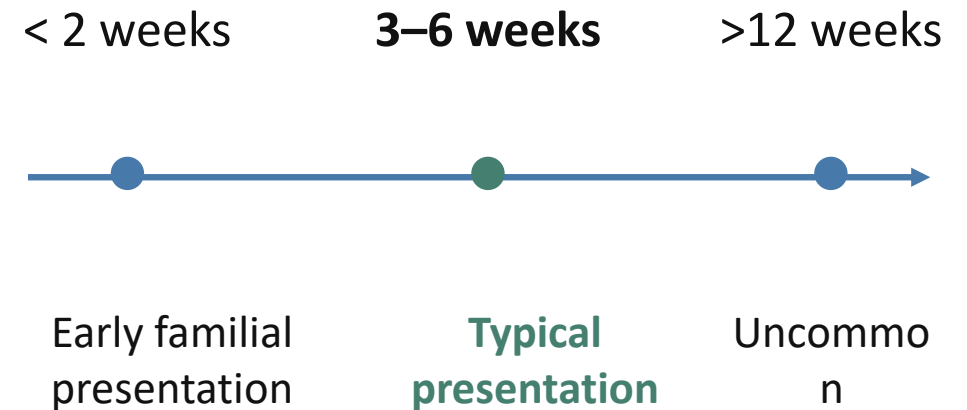
The infantile form is hypertrophic pyloric stenosis

- A congenital disorder of the pyloric muscular wall.
- The lumen becomes narrowed by expansion of the muscularis propria.
- The resulting lesion is a structural gastric outlet obstruction.



Typical presentation: Weeks 3–6

- Symptoms most commonly appear between the third and sixth weeks of life.
- The age distribution is a major clinical clue.



Pyloric stenosis after 12 weeks is uncommon

- Presentation after 12 weeks as rare.
- An older infant does not fit the classic timing pattern as closely.
- Age guides suspicion; It does not independently confirm or exclude the lesion.

Epidemiology: Recognizable but not ubiquitous

Sex distribution

- Male predominance is reported.
- Robbins gives approximately a threefold–fivefold excess among boys.

Occurrence

- The textbook provides an overall estimate of one case per 300–900 live births.
- This low baseline frequency contrasts with the higher risk seen in affected families.

Perinatal associations inform risk, not diagnosis

- First-born status is associated with the infantile form.
- Prematurity is another reported association.
- Young maternal age is also associated.

Familial

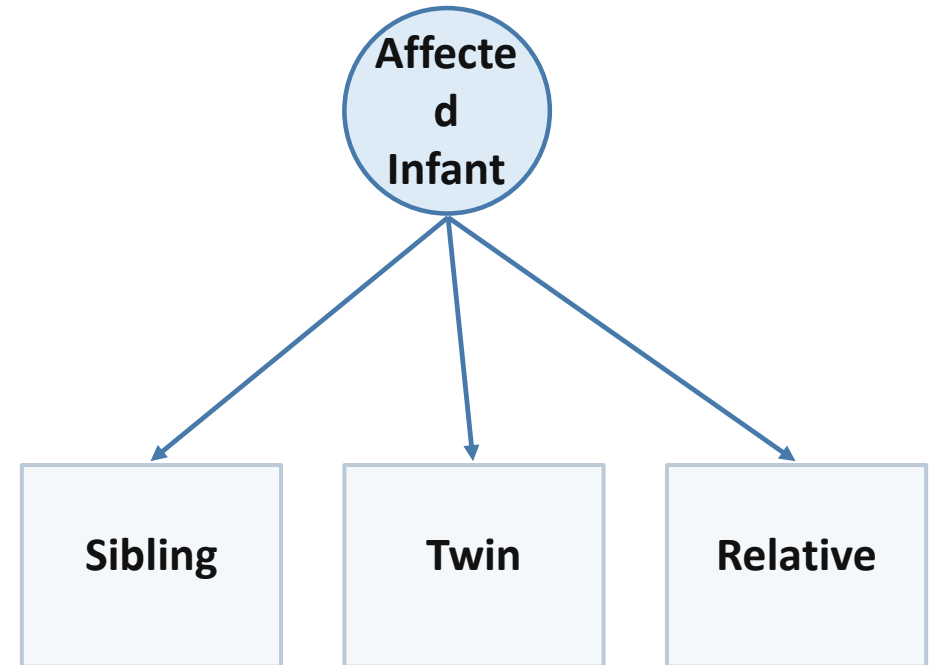
Demographic

Medication

Developmental

Familial clustering is well recognized

- Risk is elevated in siblings of affected patients.
- Family history changes prior clinical suspicion.
- Clustering alone does not establish a simple Mendelian inheritance pattern.



Clustering supports inherited susceptibility; It does not define a single-gene mechanism.

Family history: Population versus siblings

General population

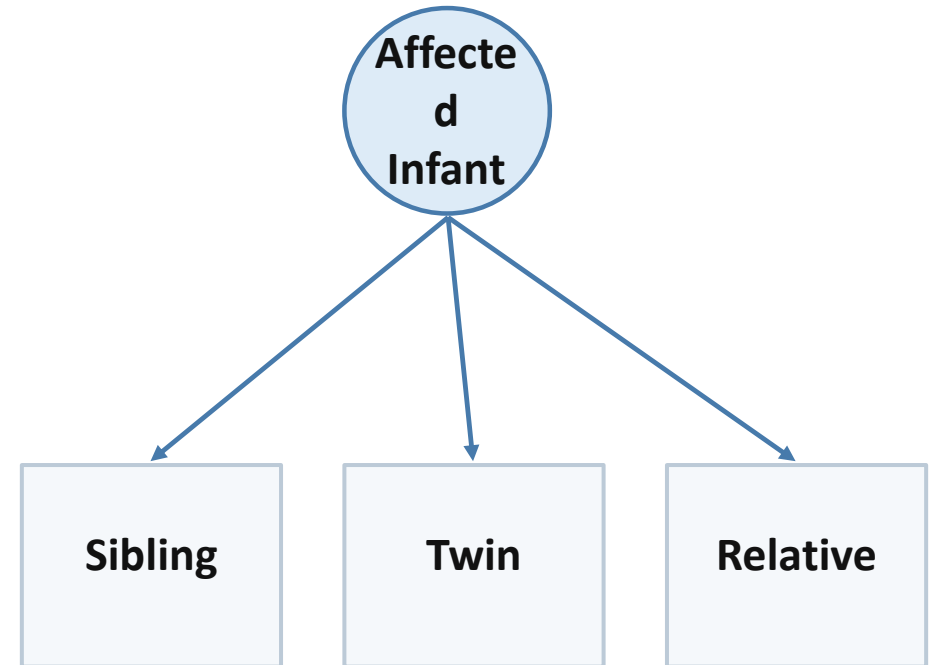
- Congenital hypertrophic pyloric stenosis is relatively uncommon.
- Most newborns do not develop the disorder.

Affected family

- Siblings have a materially higher reported risk.
- The familial excess supports a susceptibility component.

Twin concordance supports inherited susceptibility

- Concordance is high among monozygotic twins.
- A smaller increase in risk occurs among dizygotic twins.
- This pattern is consistent with genetic contribution, not genetic determinism.



Clustering supports inherited susceptibility; It does not define a single-gene mechanism.

Syndromic associations are distinct from causation

Recognized associations

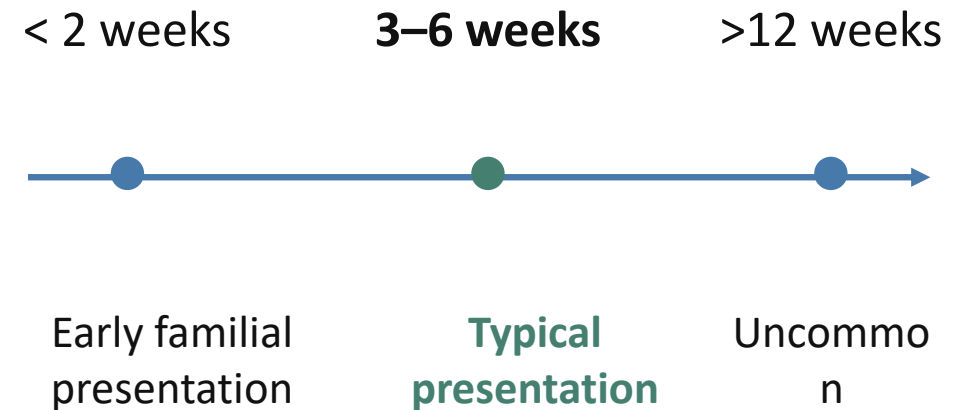
- Turner syndrome.
- Trisomy 18.

Interpretation

- Both are associated with increased risk.
- Their presence does not prove stenosis.
- Most infantile cases need not be syndromic.

Presentation before two weeks: Examine family history

- Very early onset is less typical than weeks 3–6.
- Presentation before two weeks is frequently associated with family history.
- Early symptoms should prompt careful attention to familial context.



Early macrolide exposure is an association

Antibiotics identified

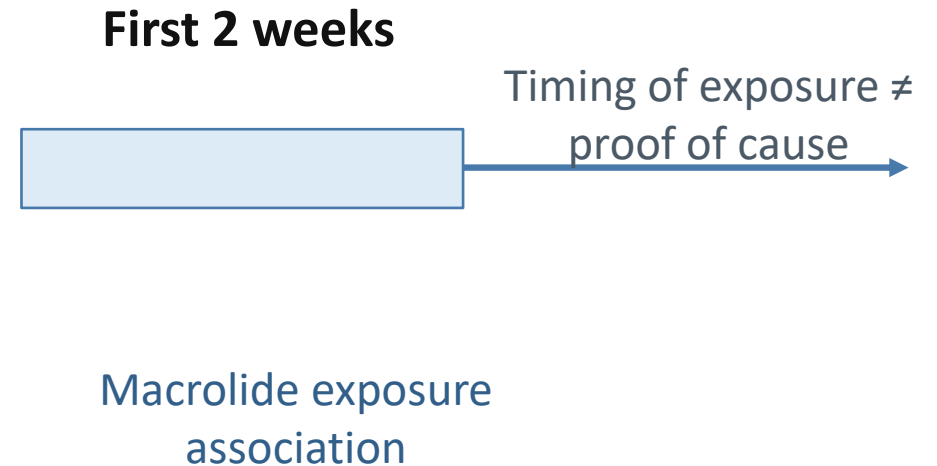
- Erythromycin.
- Azithromycin.

Reported exposure routes

- Direct oral infant exposure.
- Exposure through mother's milk.
- Association described when exposure occurs early in life.

The first two weeks define the reported window

- Robbins particularly identifies exposure in the first 2 weeks of life.
- The timing of exposure is part of the association.
- A history should identify both agent and route.



Association does not establish mechanism

What is reported

- Familial, chromosomal and perinatal correlations.
- Early macrolide exposure association.
- Development-linked genomic signals.

What remains uncertain

- The underlying cause is not fully understood.
- These observations do not establish one causal pathway.
- Do not infer patient-specific causation from a single exposure.

Genomic association studies identify developmental signals

- Genome-wide association studies implicate several genes related to gastrointestinal development.
- The textbook does not assign a complete causal pathway to these variants.
- It explicitly states that further investigation is needed.

A multifactorial susceptibility model is appropriate

- Inherited susceptibility is suggested by sibling and twin observations.
- Developmental context is suggested by chromosomal and genomic associations.
- Exposure associations add an environmental dimension without proving causality.

Familial

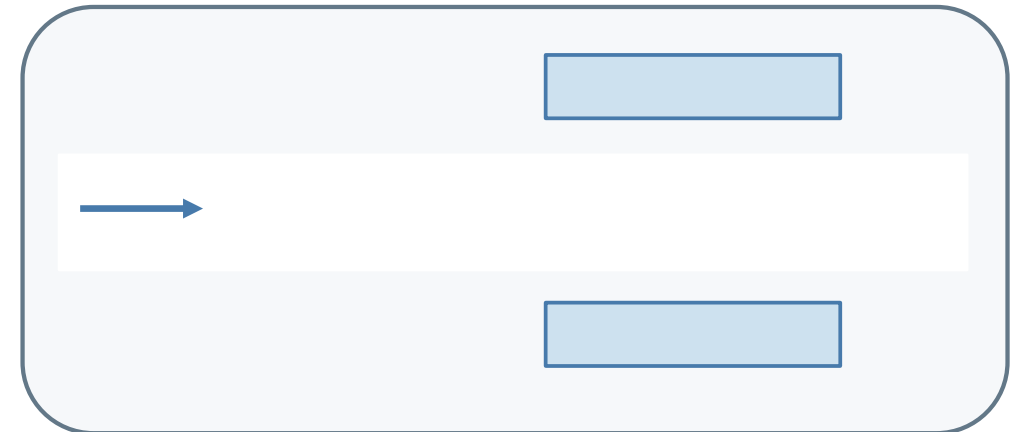
Demographic

Medication

Developmental

Anatomical wall layers locate the primary lesion

- Mucosa and submucosa line and support the outlet.
- Muscularis propria forms the contracting pyloric wall.
- The primary infantile lesion in the muscularis propria.

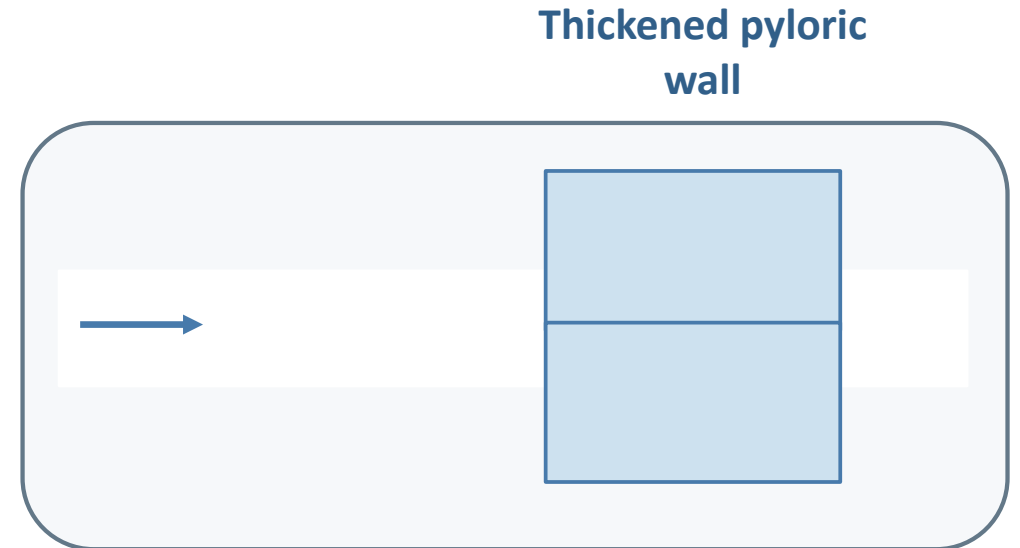


Stomach

Pylorus Duodenum

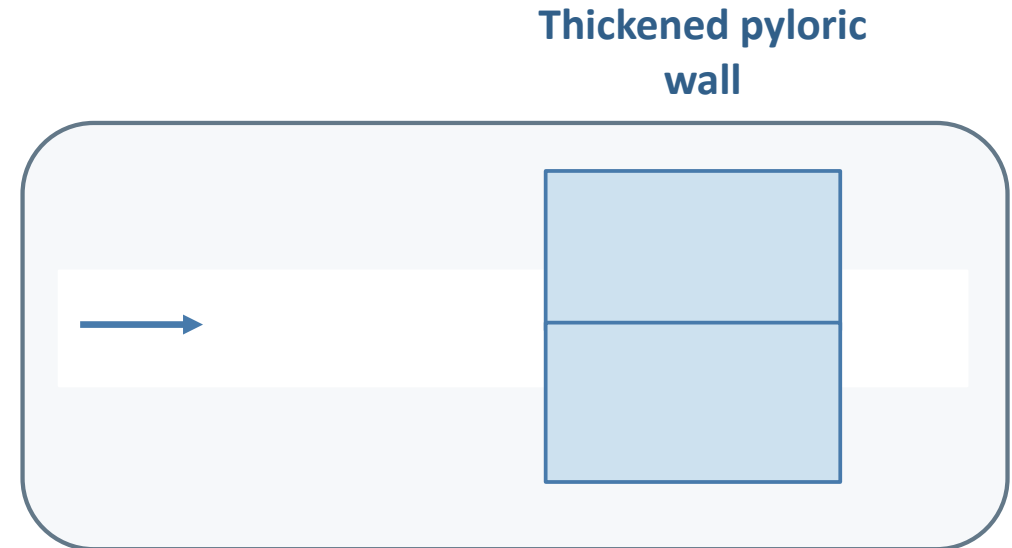
Muscularis propria hyperplasia is central

- Robbins specifically describes hyperplasia of the pyloric muscularis propria.
- Expansion of this wall compartment decreases effective luminal caliber.
- “Hypertrophic pyloric stenosis” is the clinical disease name.



Increased wall mass narrows the passage

- Concentric muscular expansion compresses the pyloric channel.
- The lesion is intramural rather than an intraluminal foreign body.
- Outflow obstruction follows from reduced caliber.



Edema and inflammation can worsen obstruction

Primary structural driver

- Hyperplasia of pyloric muscularis propria.
- Produces the initial mechanically narrowed outlet.

Additional contributors

- Mucosal and submucosal edema.
- Inflammation in these superficial layers.
- May exacerbate the obstruction.

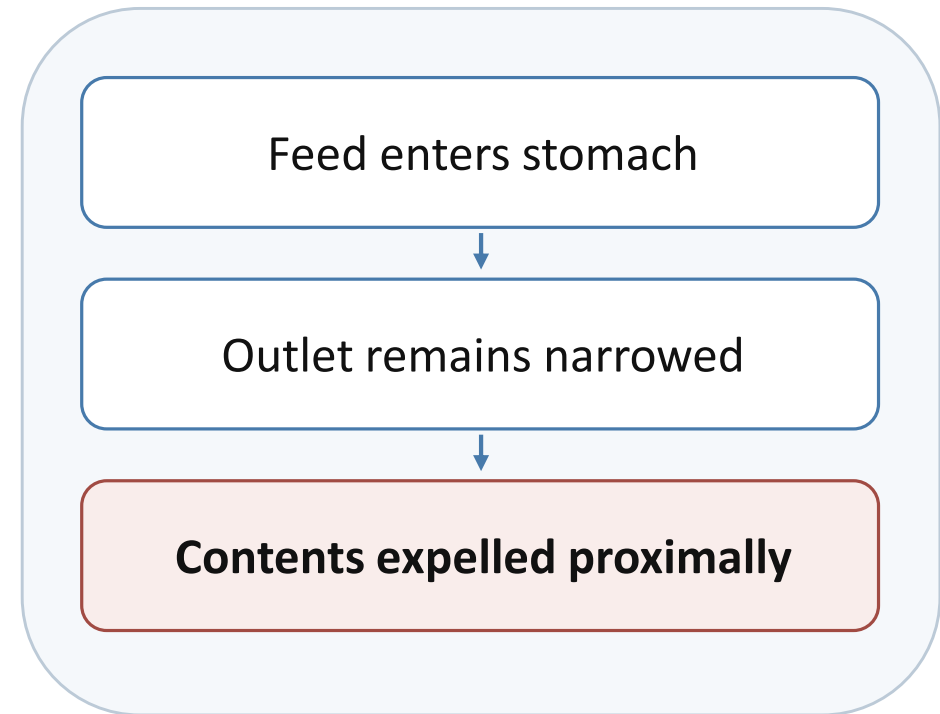
The congenital disease mechanism



- The structural mechanism connects gross anatomy directly to the feeding-related symptoms.

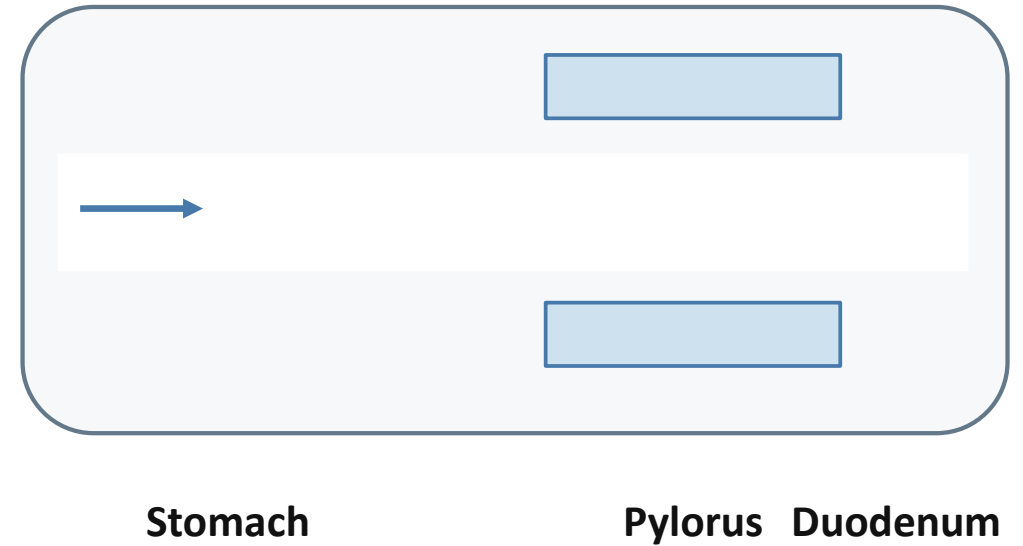
Vomiting occurs after feeding

- Feeding delivers gastric contents to an obstructed outlet.
- The failure of normal onward passage precedes expulsion.
- The feeding–vomiting relationship supports an outlet-level lesion.



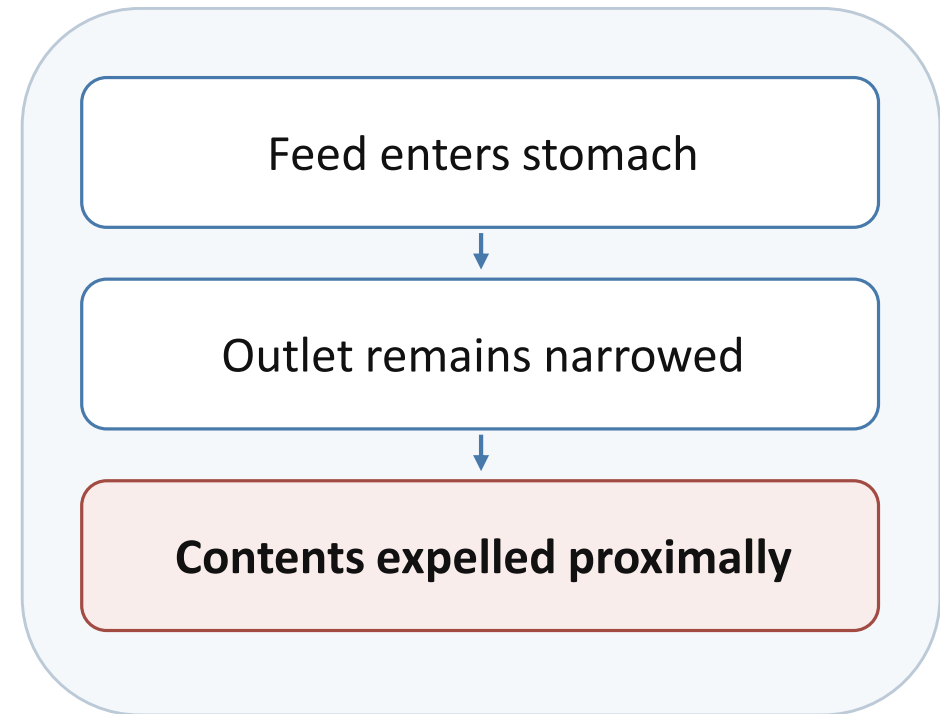
The vomitus is characteristically nonbilious

- The narrowed pylorus is proximal to the duodenal site of bile entry.
- Gastric contents may be expelled before mixing with bile.
- Nonbilious emesis is therefore anatomically coherent.



Forceful projectile emesis is characteristic

- Forceful, projectile vomiting.
- This is the classic postprandial infantile presentation.
- Projectile quality should be considered with age and the remaining examination.



Persistent hunger after vomiting is a diagnostic clue

- Affected infants may demand feeding again soon after emesis.
- The feeding drive may persist despite ineffective gastric emptying.
- Refeeding demand is part of the classic syndrome, not an isolated diagnostic test.

Visible peristaltic waves reflect outflow effort

- Abnormal left-to-right upper-abdominal waves may be observed.
- They appear just before vomiting in some cases.
- The observation links gastric motor activity to distal resistance.

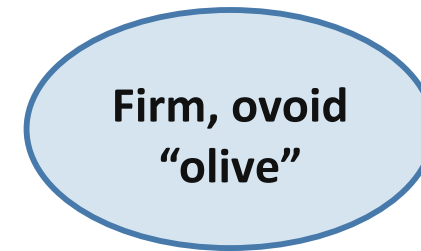
Visible waves across upper abdomen



Left → right, before emesis

An abdominal “olive” indicates a palpable lesion

- A firm, ovoid mass may be felt on examination.
- It represents the thickened pyloric region.
- The described size is approximately 1–2 cm.



Approximately 1–2 cm

Palpable mass is a clinical finding, not an imaging criterion.

The olive is nearly pathognomonic when present

- The finding in up to 90% of cases.
- A clearly palpable olive strongly favors the infantile hypertrophic form.
- Its diagnostic weight depends on an appropriate clinical setting.



Approximately 1–2 cm

Palpable mass is a clinical finding, not an imaging criterion.

A missing olive does not rule out stenosis

Present

- Firm ovoid lesion is a major positive bedside sign.
- Supports diagnosis in a compatible infant.

Not palpable

- Not every case has an identifiable mass.
- Absence should not override the remainder of the clinical and imaging evidence.

The clinical pattern is a constellation

Historical pattern

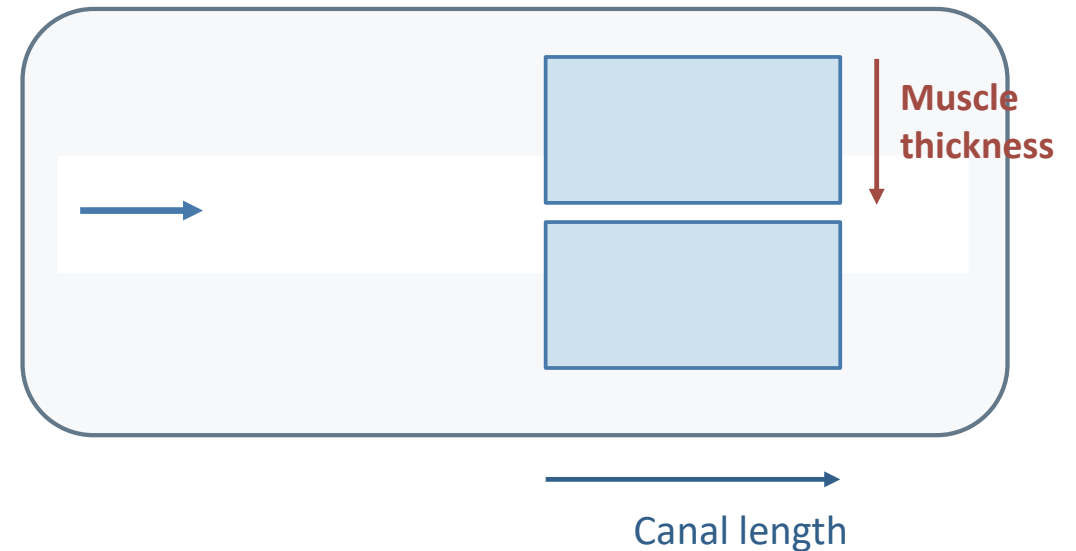
- Age generally 3–6 weeks.
- Projectile, nonbilious, post-feed emesis.
- Often renewed feeding demand.

Examination pattern

- Visible left-to-right waves in some.
- Firm palpable olive in some.
- Ultrasound evaluates anatomical narrowing.

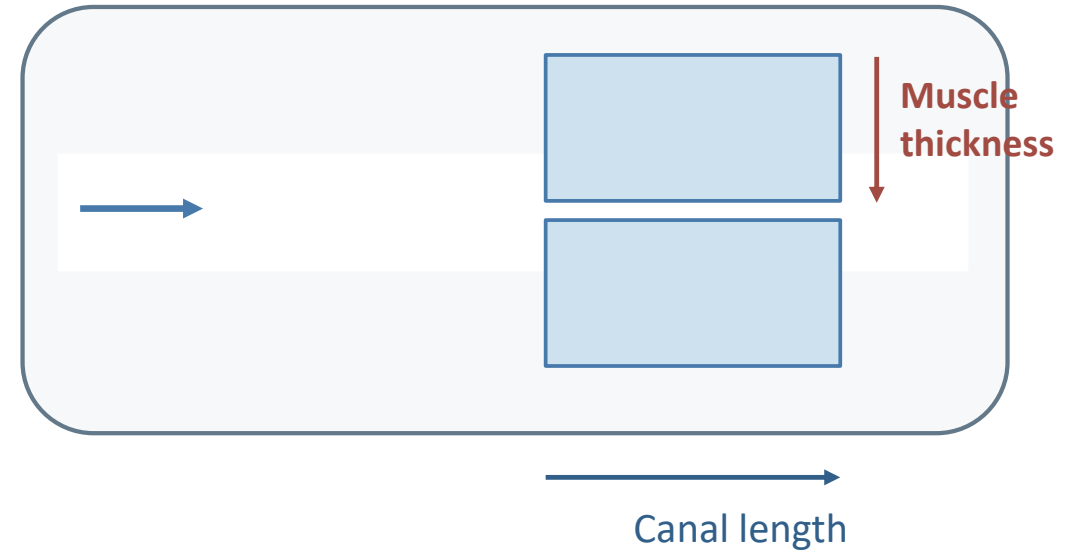
Ultrasonography evaluates the pyloric anatomy

- Ultrasound is described as sensitive and specific.
- The assessment targets the pyloric muscle and channel.
- The technique can support diagnosis without relying solely on palpation.

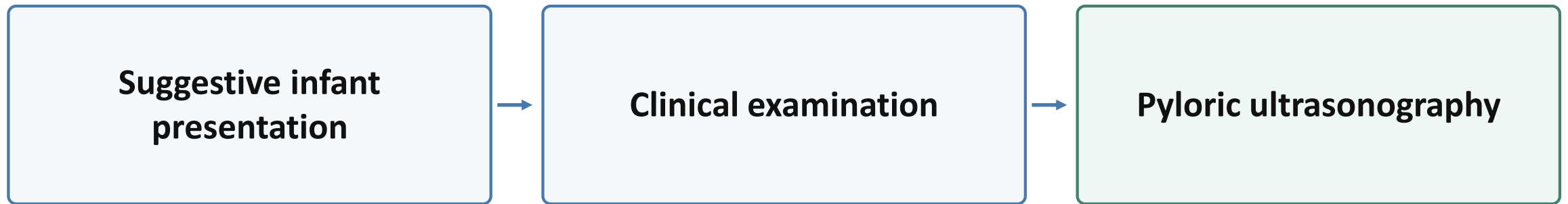


Two sonographic features are central

- Thickness of the pyloric muscle.
- Length of the pyloric canal.
- The supplied textbook passage does not state numerical cutoffs.



Clinical suspicion leads to anatomical confirmation



- Clinical and sonographic data address different elements of the same obstruction.

Link the imaging target to the tissue lesion

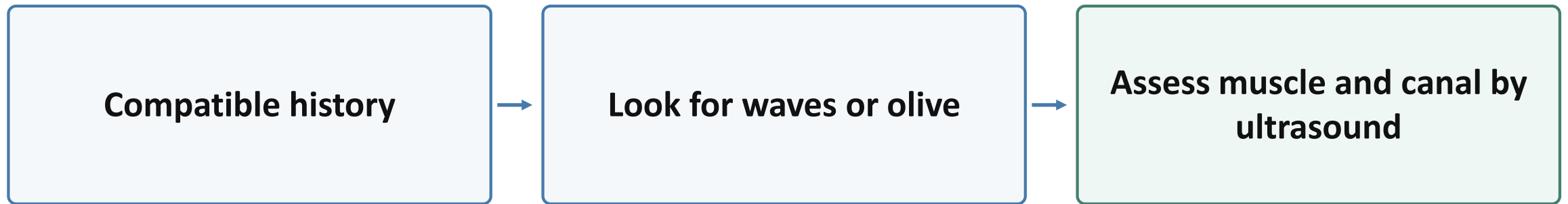
Histopathologic process

- Muscularis propria hyperplasia.
- Secondary edema or inflammation may accentuate narrowing.

Ultrasound targets

- Muscle thickness.
- Pyloric canal length.
- Two complementary anatomical dimensions.

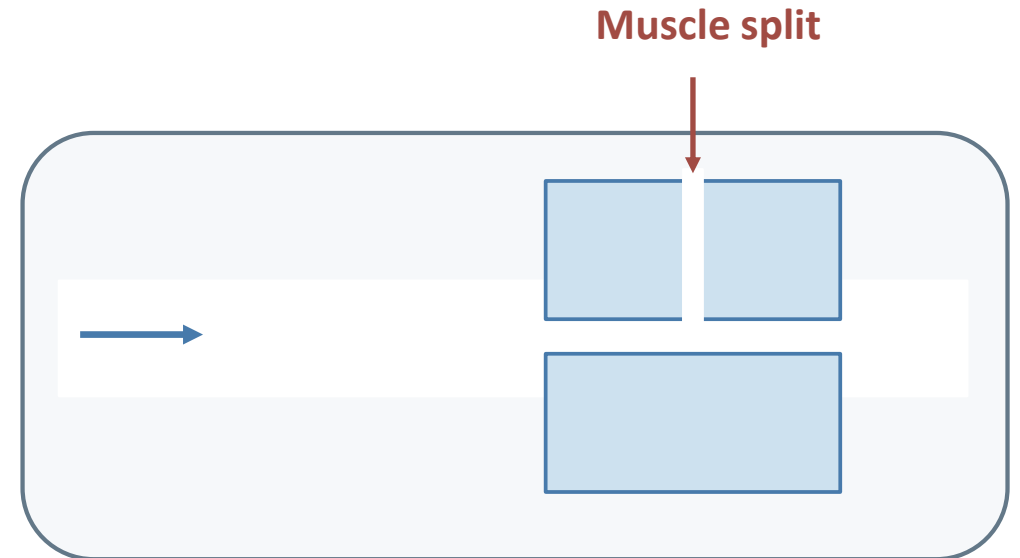
A restrained diagnostic reasoning sequence



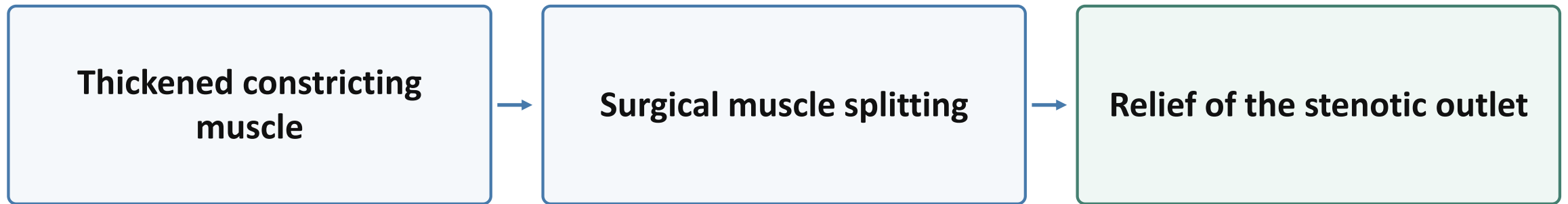
- Neither historical risk factors nor a single absent bedside sign is conclusive.

Pyloromyotomy treats the muscular obstruction

- Treatment splits the hypertrophic pyloric muscularis.
- The operation addresses the anatomical narrowing rather than a presumed single gene or exposure.
- This procedure is generally curative.



Why mechanical release restores outflow



- This therapeutic mechanism follows directly from the location of the lesion.

Case: Classic infantile presentation

Clinical vignette

- A 4-week-old boy vomits forcefully after feeding.
- The material is nonbilious.
- He repeatedly seeks another feed.
- A small firm epigastric mass is palpable.

Interpretation

- Age and symptoms match the classic pattern.
- The palpable “olive” adds high diagnostic weight.
- Evaluate pyloric thickness and channel length by ultrasound.

Case: Presentation earlier than expected

Clinical vignette

- A 10-day-old infant develops post-feed vomiting.
- An older sibling had congenital hypertrophic pyloric stenosis.
- No specific cause can be assigned to the family history alone.

Interpretation

- The onset is unusually early.
- Robbins relates presentation before two weeks to family history.
- Clinical examination and ultrasound remain relevant.

Case: Compatible symptoms, no palpable olive

Clinical vignette

- A 5-week-old infant has projectile nonbilious emesis.
- Examination does not identify a discrete ovoid mass.
- The history remains highly suggestive.

Interpretation

- The mass is not present or detectable in every case.
- Do not use palpation alone to exclude the disease.
- Ultrasound assesses the primary anatomical lesion.

Acquired stenosis has a different causal landscape

Inflammatory causes

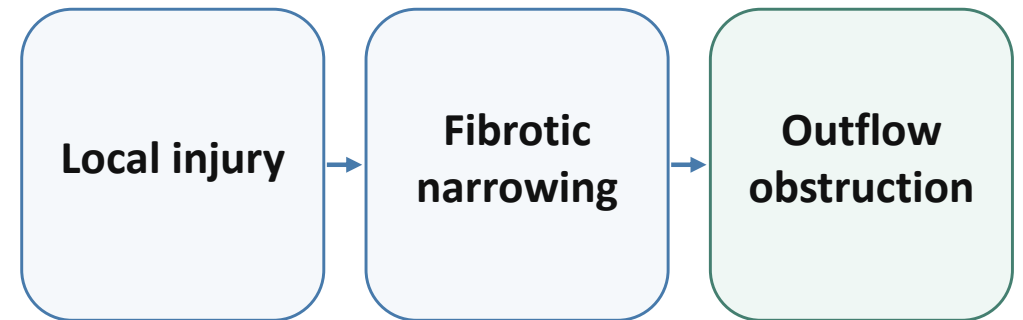
- Antral gastritis.
- Peptic ulcer close to the pylorus.
- Fibrotic narrowing may follow local injury.

Neoplastic causes

- Distal gastric carcinoma.
- Pancreatic carcinoma.
- Malignant infiltration or fibrosis can narrow the channel.

Antral gastritis is a recognized acquired cause

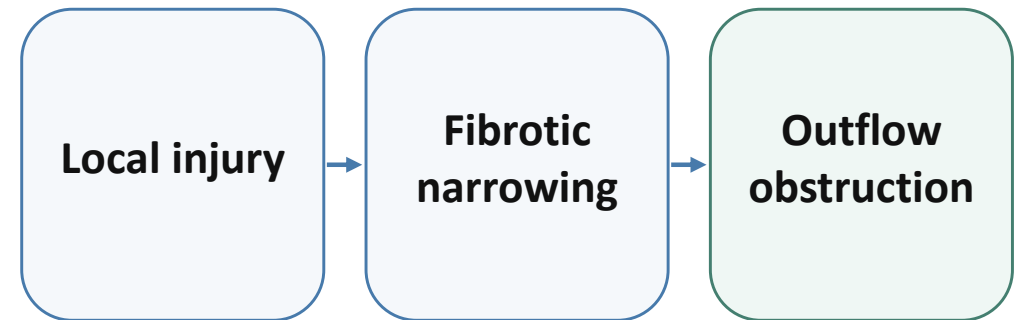
- Inflammation near the gastric outlet may eventually narrow the pyloric channel.
- Robbins specifically lists antral gastritis among adult causes.
- The mechanism is distinct from congenital muscular hyperplasia.



Schematic causal sequence

Peptic ulcer disease can narrow the outlet

- Ulcers close to the pylorus are an acquired cause.
- Healing-associated tissue change may contribute to the stenotic lesion.
- Proximity of the injury to the pyloric canal is mechanistically important.



Schematic causal sequence

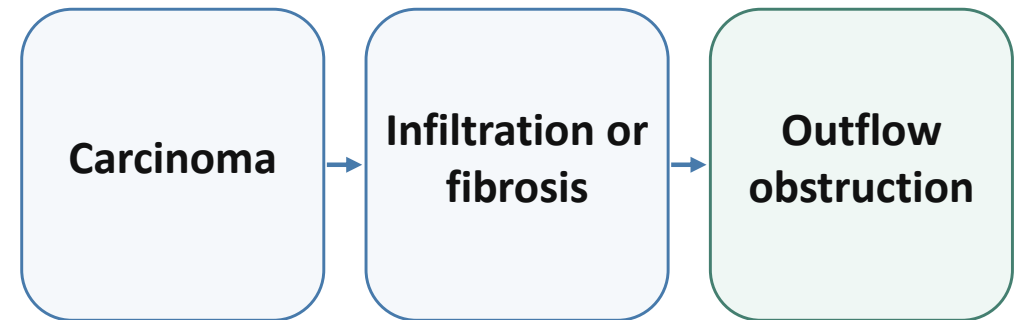
Fibrosis converts injury into fixed narrowing



- Adult narrowing may persist because the wall architecture is remodeled.

Distal gastric carcinoma may cause stenosis

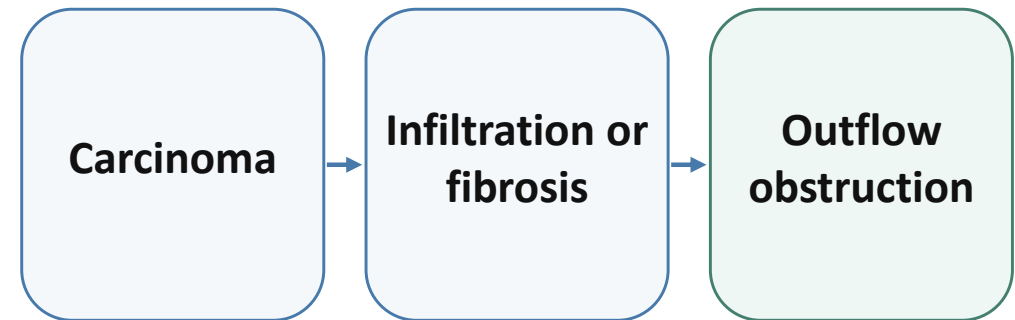
- A carcinoma in the distal stomach can affect the pyloric outlet.
- Luminal narrowing may reflect malignant infiltration.
- Fibrosis may also contribute to obstruction.



Schematic causal sequence

Pancreatic carcinoma can obstruct the pyloric region

- Pancreatic carcinoma among acquired causes.
- Tumor-related infiltration or fibrosis may restrict the outflow channel.
- An adult stenotic lesion is not necessarily a primary muscular disorder.



Schematic causal sequence

Malignant infiltration and fibrosis are not identical

Malignant infiltration

- Neoplastic tissue encroaches on or involves the pyloric region.
- The disease process is malignant.

Fibrotic narrowing

- Collagenous remodeling reduces the effective passage.
- Can accompany inflammatory injury or malignancy.

Ectopic pancreatic tissue can narrow the pylorus

Location and process

- Ectopic pancreatic tissue may occur in the stomach or pylorus.
- At the pylorus, local injury and inflammation can develop.

Potential effect

- Scarring can narrow the gastric outlet.
- An ectopic rest may also mimic more serious pathology.

Acquired etiologies by mechanism

Source lesion	Mechanism near pylorus	Adult implication
Antral gastritis	Inflammation / injury	Benign inflammatory cause
Juxtapyloric peptic ulcer	Local injury → fibrosis	Benign fibrotic cause
Distal gastric carcinoma	Infiltration and/or fibrosis	Malignant cause
Pancreatic carcinoma	Infiltration and/or fibrosis	Malignant cause

Case: Adult with pyloric ulceration

Clinical vignette

- An adult has a peptic ulcer located near the pyloric channel.
- A narrowed outlet is identified anatomically.
- The history suggests a locally acquired lesion.

Mechanistic answer

- An ulcer close to the pylorus can cause acquired stenosis.
- Fibrotic repair offers a structural explanation.
- Do not label this congenital muscular hyperplasia.

Case: Adult with distal gastric carcinoma

Clinical vignette

- An adult has a distal gastric carcinoma.
- The tumor lies close to the outlet and gastric emptying is obstructed.
- The cause is not necessarily a benign scar.

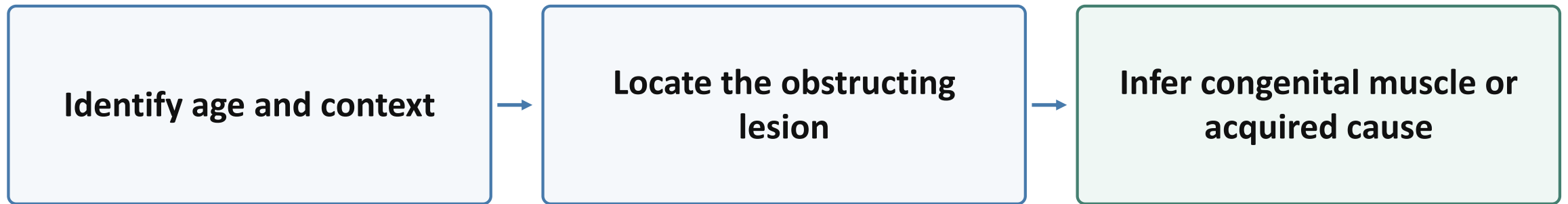
Mechanistic answer

- Malignant infiltration may narrow the pyloric channel.
- Associated fibrosis can contribute.
- Classify the lesion as acquired malignant obstruction.

Congenital and acquired stenosis compared

Feature	Congenital hypertrophic	Adult acquired
Typical setting	Infancy; Weeks 3–6	Adulthood
Primary pathology	Muscularis propria hyperplasia	Inflammation, fibrosis, infiltration
Clinical clue	Projectile nonbilious emesis	Context of gastritis, ulcer or carcinoma
Confirmation emphasis	Pyloric ultrasound	Evaluation of underlying cause
Treatment principle	Pyloromyotomy generally curative	Depends on cause (not detailed in source)

Mechanism is the unifying diagnostic framework



- A diagnosis of “pyloric stenosis” is anatomically useful but etiologically incomplete.

Final synthesis: Infant and adult disease

Infantile hypertrophic

- Muscularis propria hyperplasia.
- Weeks 3–6; Projectile nonbilious emesis.
- Ultrasound of muscle and channel.
- Pyloromyotomy generally curative.

Acquired

- Antral inflammation or near-pyloric ulceration.
- Fibrotic narrowing.
- Distal gastric or pancreatic carcinoma.
- Possible malignant infiltration.

Interpretation exercise: What does the clue imply?

Clinical vignette

- Clue 1: Palpable 1–2 cm ovoid mass in a vomiting 4-week-old.
- Clue 2: Adult with antral gastritis and a narrowed channel.
- Clue 3: Tumor infiltrating a distal gastric outlet.

Answer

- 1 → hypertrophic muscular lesion likely.
- 2 → acquired inflammatory or fibrotic stenosis.
- 3 → acquired malignant narrowing.

Reference and source scope

Primary textbook

Robbins, Cotran & Kumar: Pathologic Basis of Disease. 11th ed. (2026). Chapter 17: The Gastrointestinal Tract. Section “Pyloric Stenosis,” p. 686.

The lecture uses original editable schematics; authentic histology or ultrasonography was not supplied.