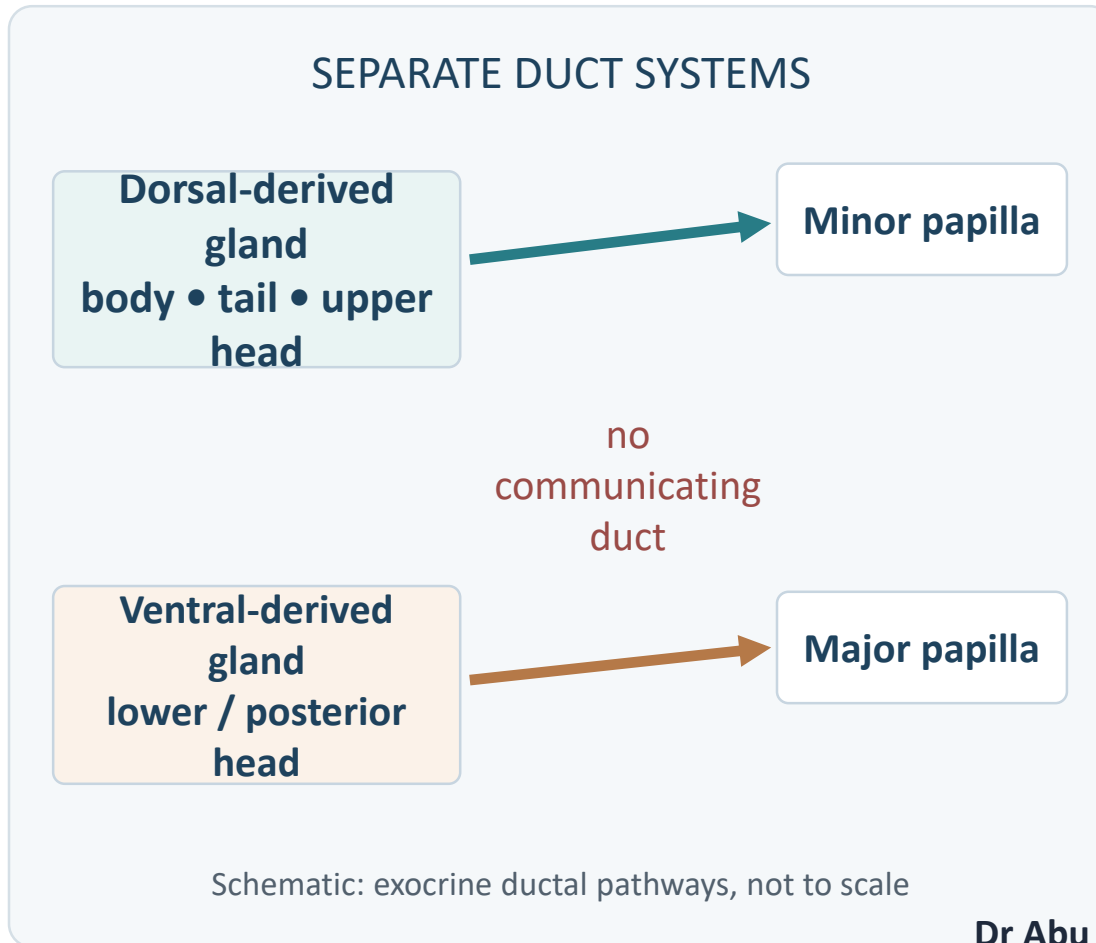


Pancreas divisum



- Congenital pancreatic ductal variant due to failed duct fusion.
- Most exocrine pancreatic drainage reaches the minor papilla.
- Clinical importance lies in the minority with pancreatitis.

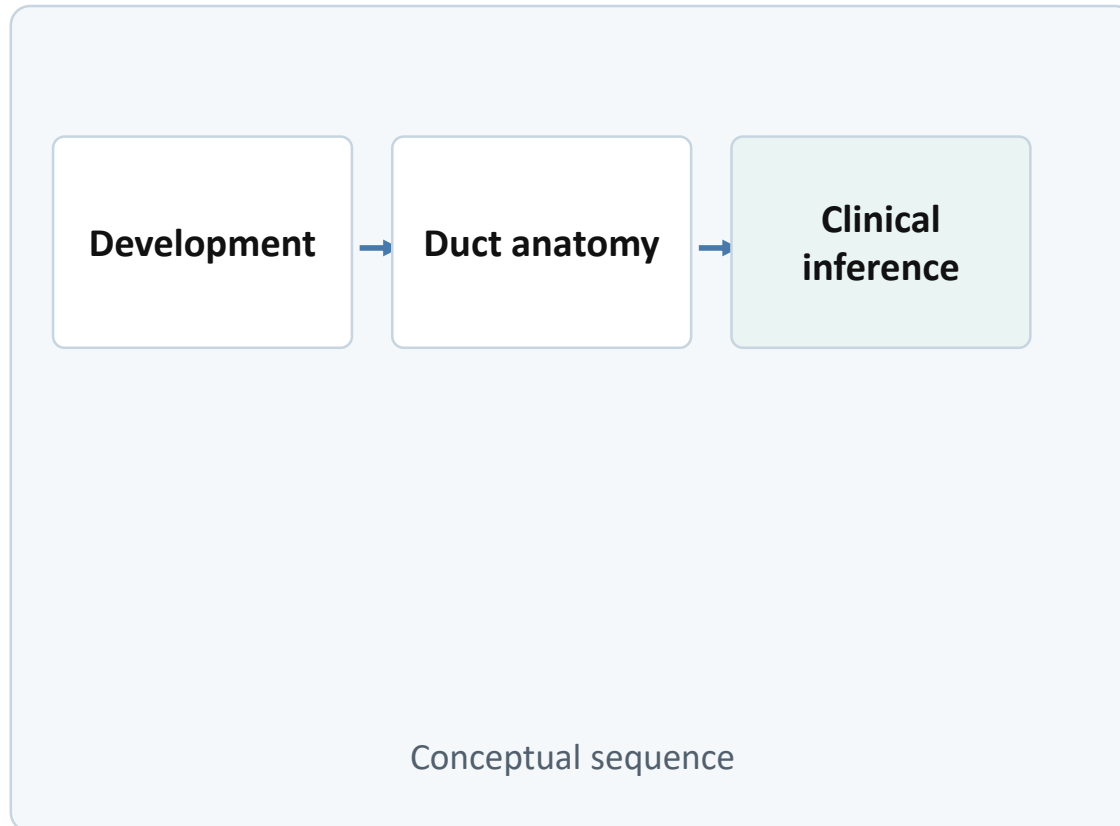
Schematic: dorsal and ventral pancreatic duct drainage.

Dr Abu Suraih Sakhri

MBBS, MD (Pathology)

www.abusuraihsakhri.com

Learning outcomes: Anatomy to clinical reasoning



- Trace dorsal and ventral pancreatic contributions to the adult gland.
- Compare pancreatic outflow in normal fusion and divisum.
- Interpret pancreatitis risk and the limitations of intervention evidence.

A ductal variant, not an inflammatory diagnosis

Congenital duct pattern

Anatomical finding

Acute pancreatitis

Inflammatory episode

Chronic pancreatitis

Irreversible parenchymal injury

- Divisum describes how ducts are connected and where they drain.
- Pancreatitis describes pancreatic injury, not a ductal phenotype.
- The two can coexist without a proven causal relationship in every case.

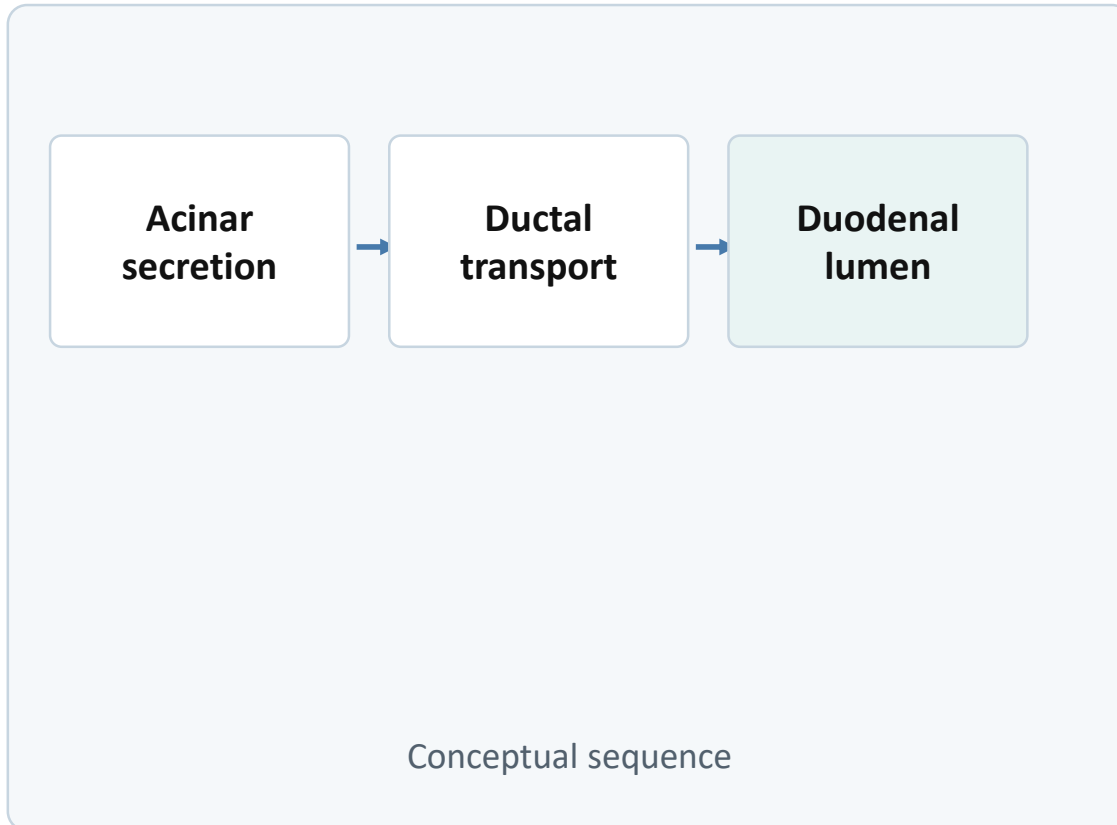
Pancreas divisum is the commonest congenital variant

3–10%

Frequency reported by Robbins (11th ed.)

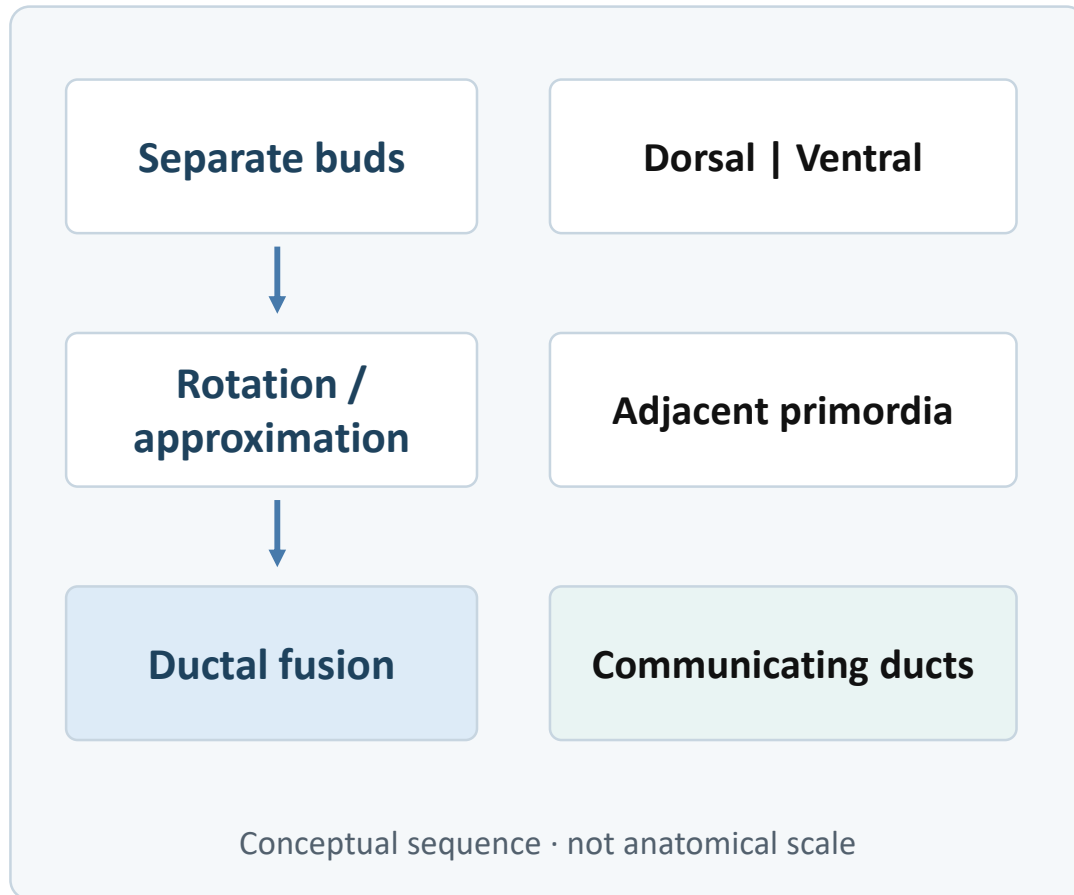
- Robbins characterizes divisum as the most common congenital pancreatic anomaly.
- Reported frequency reflects a congenital anatomic pattern.
- Prevalence is not the same as the risk of symptomatic disease.

Why pancreatic duct anatomy matters



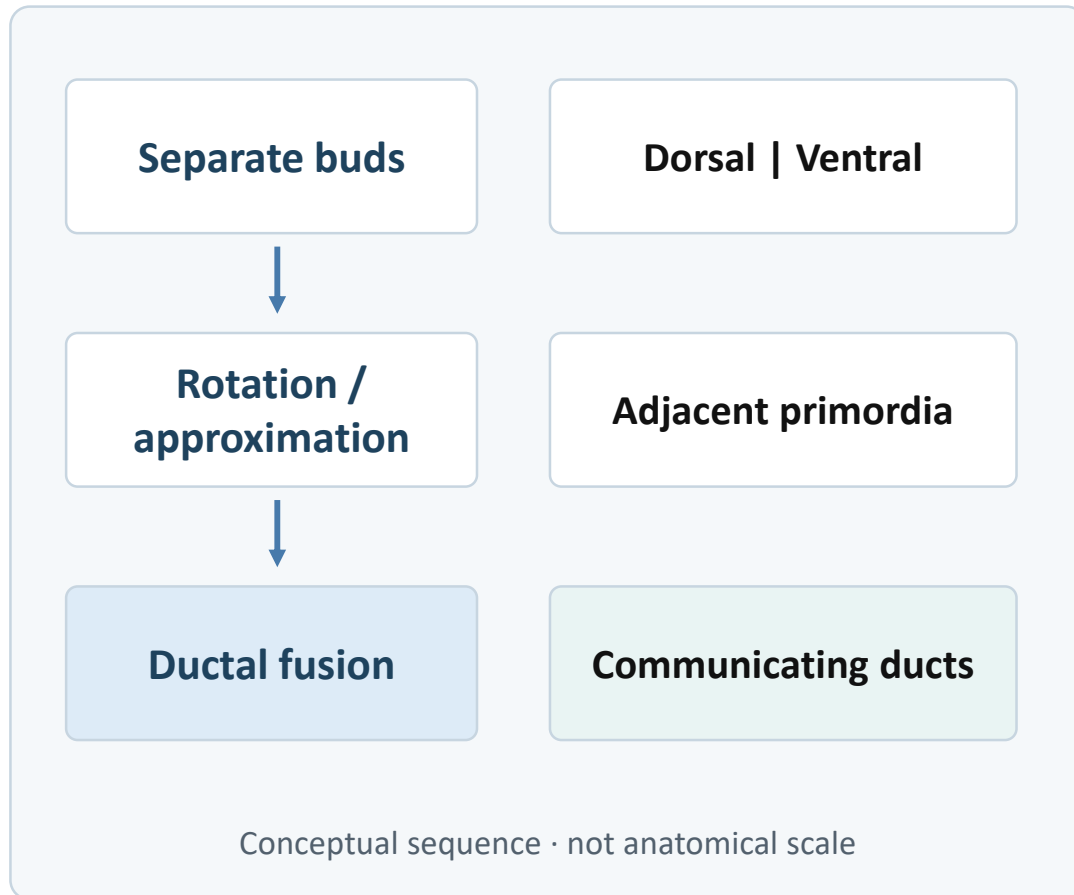
- Most pancreatic tissue is exocrine and makes digestive zymogens.
- Ductules and ducts carry secretions toward the duodenum.
- The final drainage route can matter when papillary outflow is limited.

Dorsal and ventral pancreatic primordia



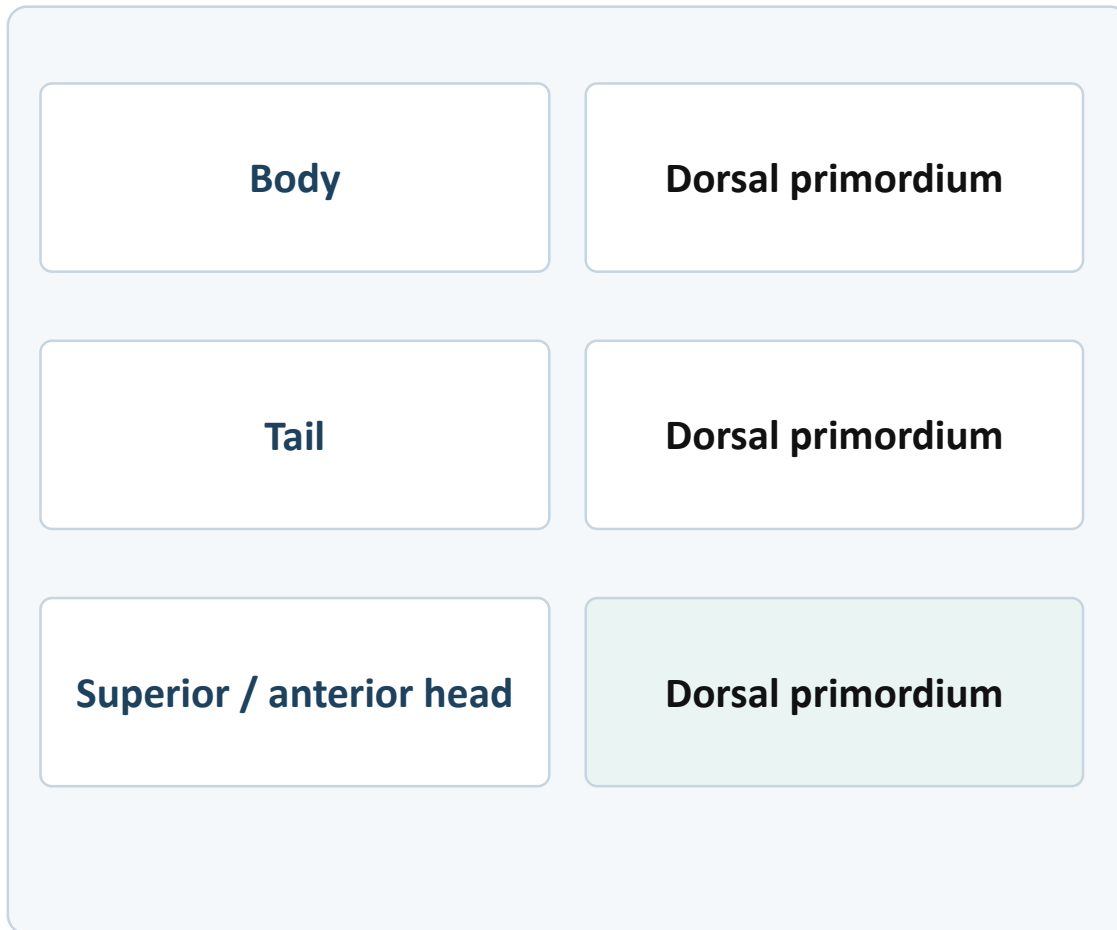
- The pancreas arises from two foregut outpouchings.
- The primordia normally merge as the pancreas develops.
- Their duct systems usually establish a communicating pathway.

The developmental fusion window



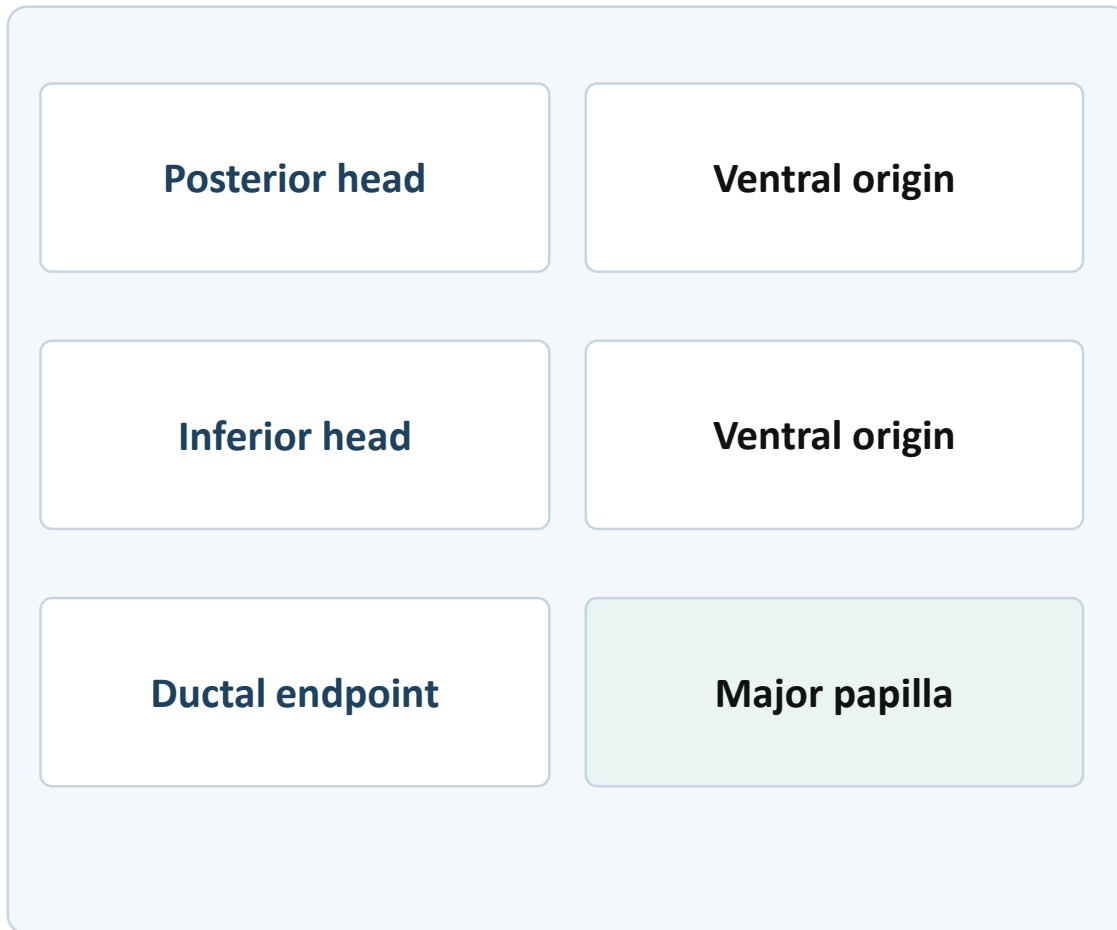
- Fusion between gestational weeks 5 and 8.
- This process involves coordinated rotation and approximation.
- Imperfection in development produces congenital ductal variants.

Dorsal primordium: Most of the gland



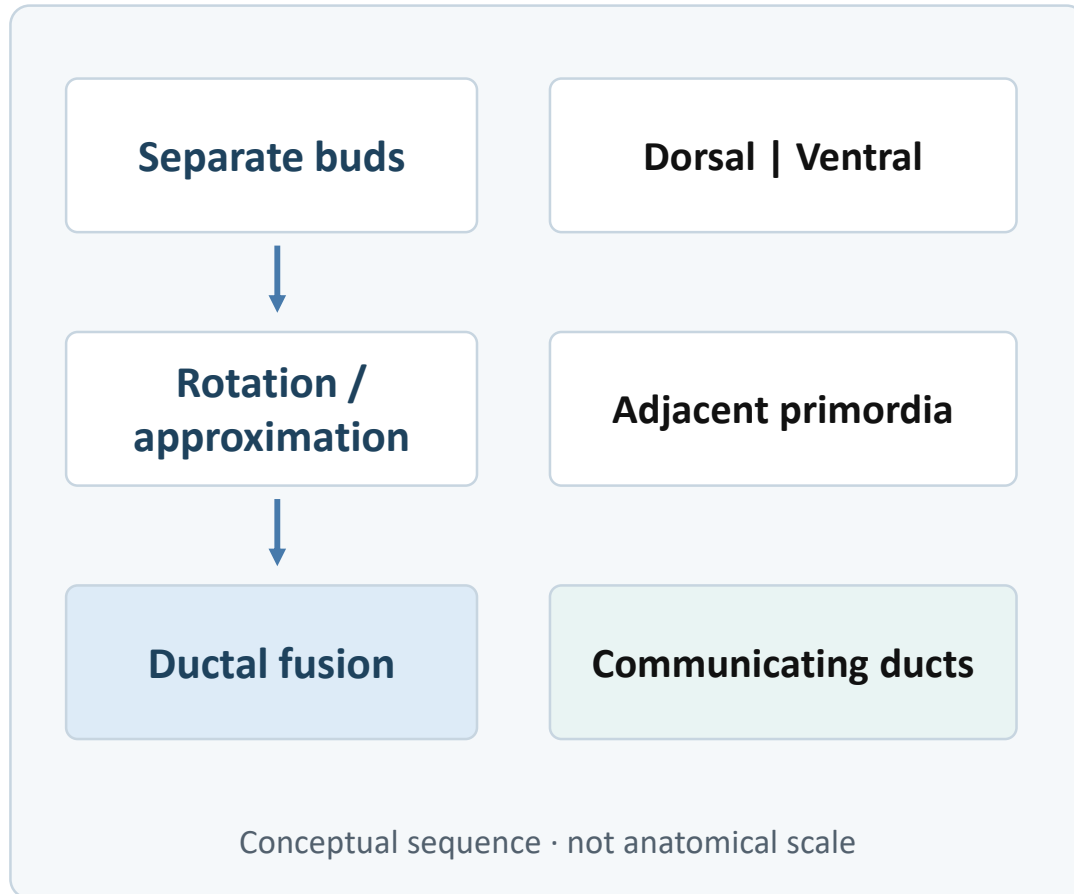
- The dorsal primordium forms the body and tail of the pancreas.
- It also contributes the superior/anterior portion of the head.
- Its large parenchymal contribution explains dominant dorsal drainage in divisum.

Ventral primordium: A smaller region



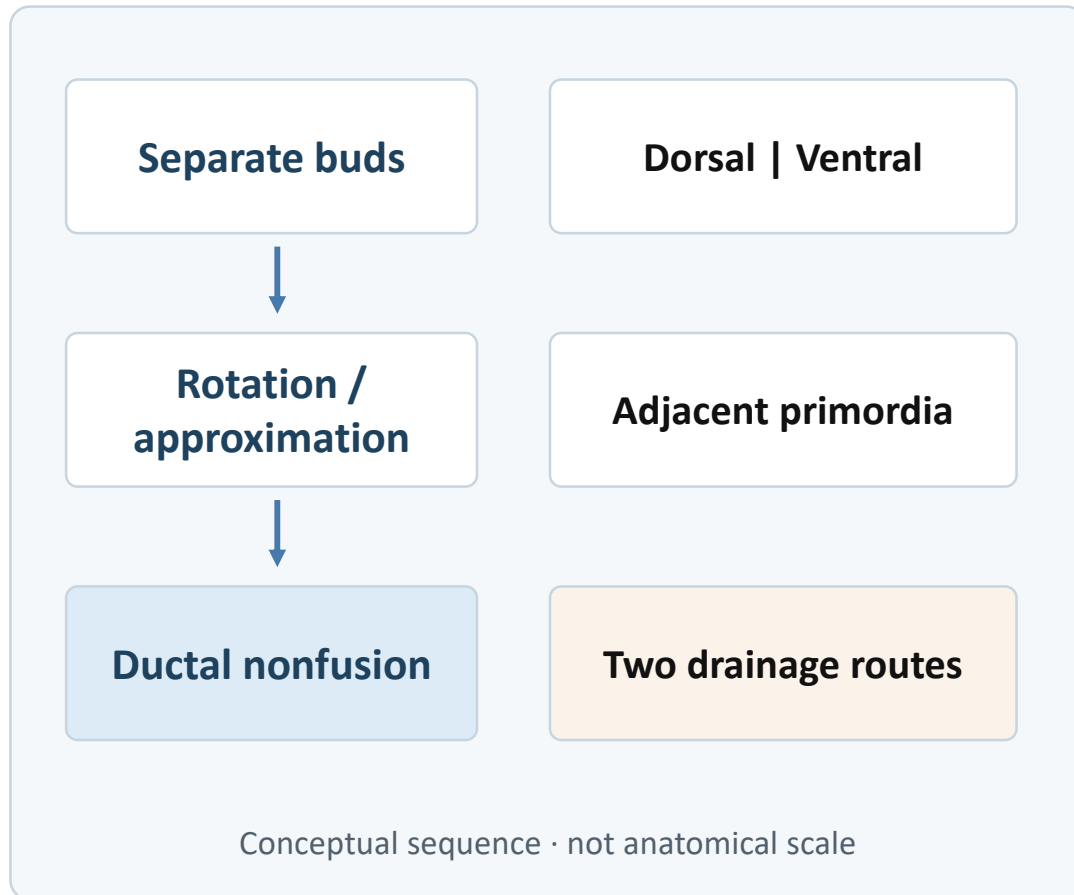
- The ventral primordium forms posterior/inferior pancreatic head.
- The ventral drainage pathway connects with the major papilla.
- In divisum, this route drains a relatively small portion of the gland.

Normal development produces ductal communication



- The two pancreatic primordia become anatomically united.
- Ductal fusion creates communication between their networks.
- Dominant exocrine outflow is ordinarily via the major papilla.

Pancreas divisum: Failure of ductal fusion



- The fetal dorsal and ventral duct systems fail to fuse.
- A dominant dorsal drainage route persists toward the minor papilla.
- Ventral drainage remains separate toward the major papilla.

Distinguish failure of fusion from failed organ growth

Divisum

Nonfused ducts

Annular pancreas

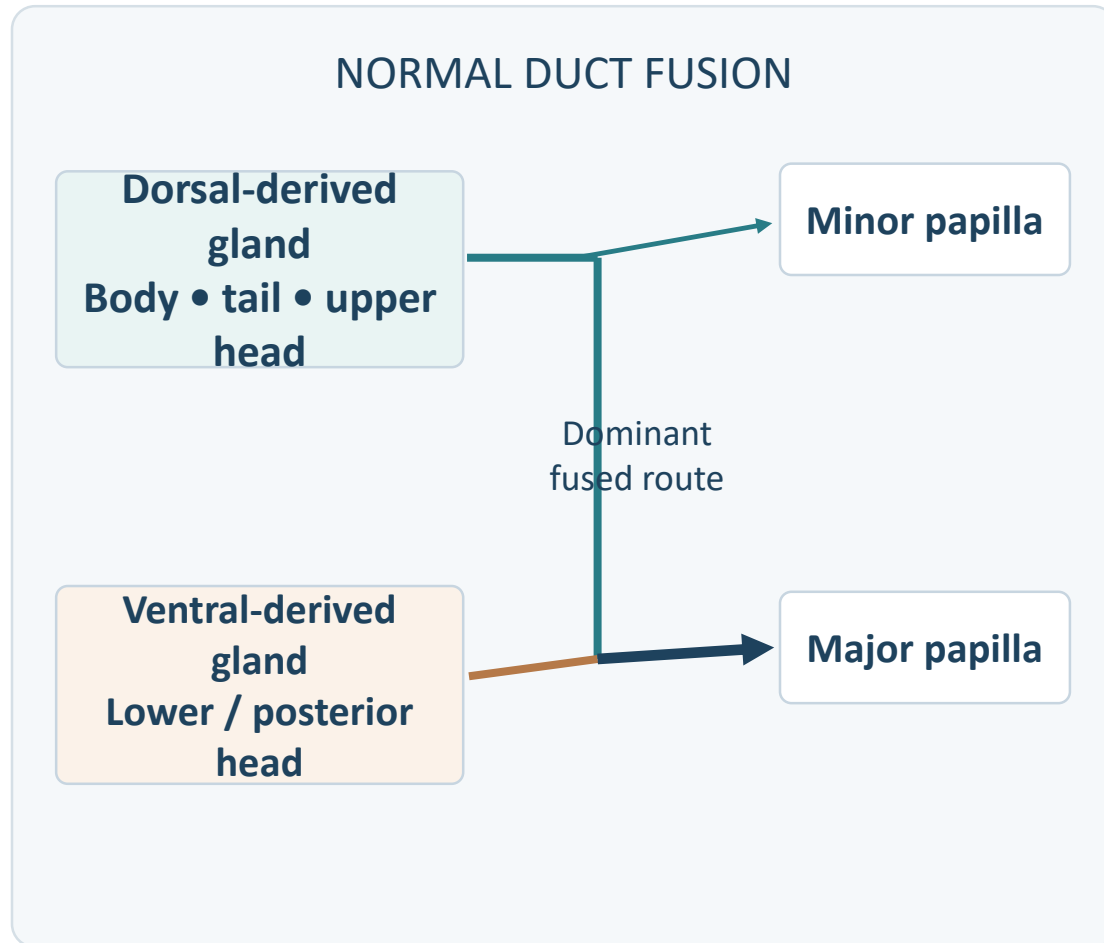
Ventral rotation anomaly

Agenesis

**Failed development of
gland**

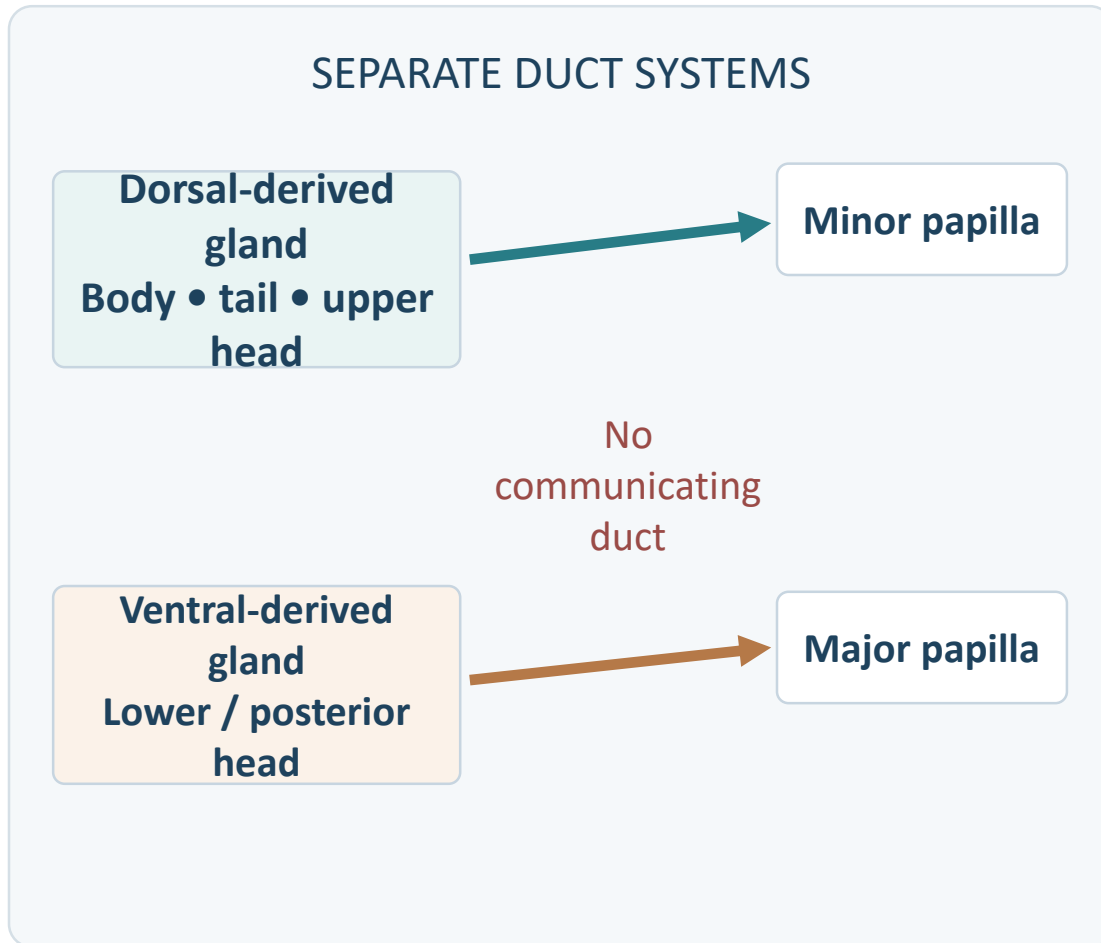
- Pancreas divisum concerns the exocrine ductal connections.
- An annular pancreas is a different congenital developmental problem.
- Pancreatic agenesis is not interchangeable with ductal divisum.

The duct of Wirsung



- Wirsung is the main pancreatic duct in normal fused anatomy.
- It joins the common bile duct near the major papilla of Vater.
- In divisum, its ventral component drains only a small head region.

The duct of Santorini



- Santorini is conventionally the accessory pancreatic duct.
- It reaches the duodenum through the minor papilla.
- In divisum it is the dominant pathway for most pancreatic secretions.

Two duodenal papillae, different outflow routes

Minor papilla

**Dorsal drainage in
divisum**

Major papilla

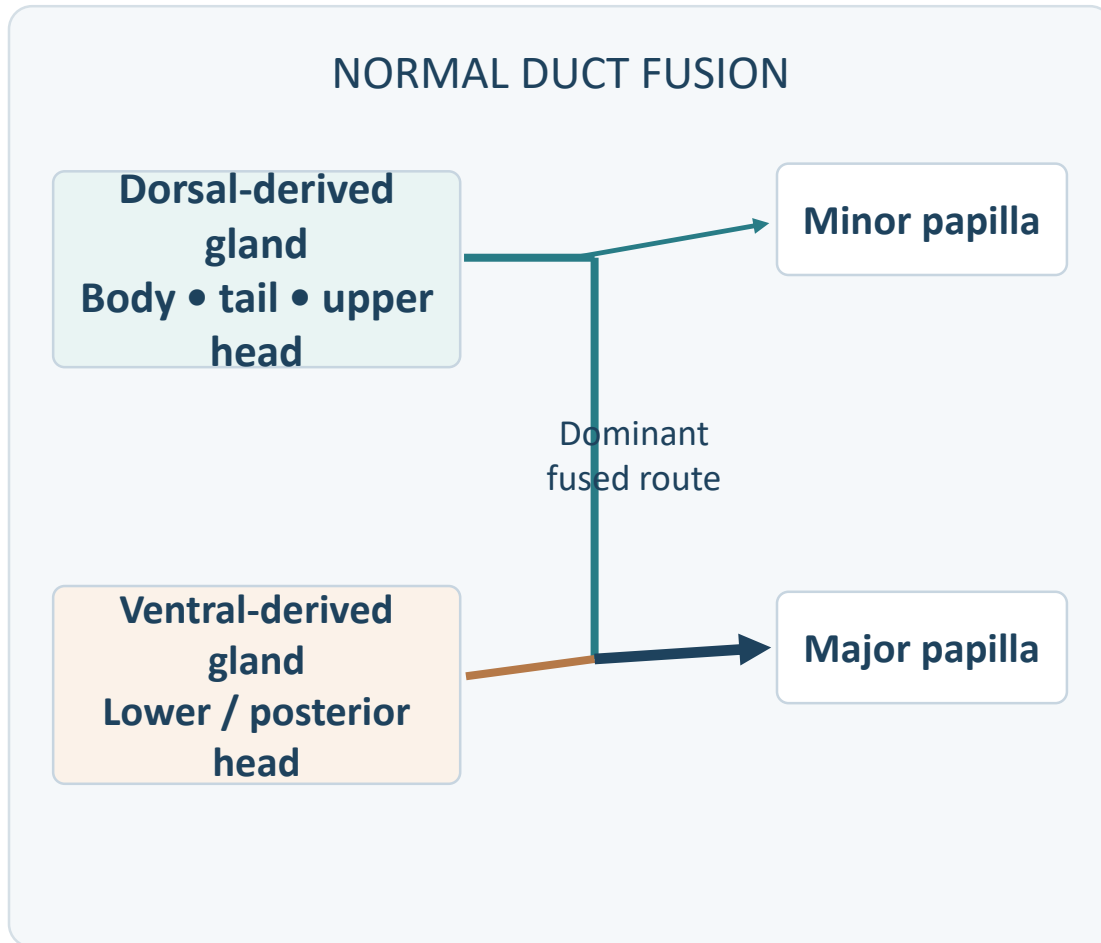
Bile + ventral drainage

Key distinction

Separate duct outlets

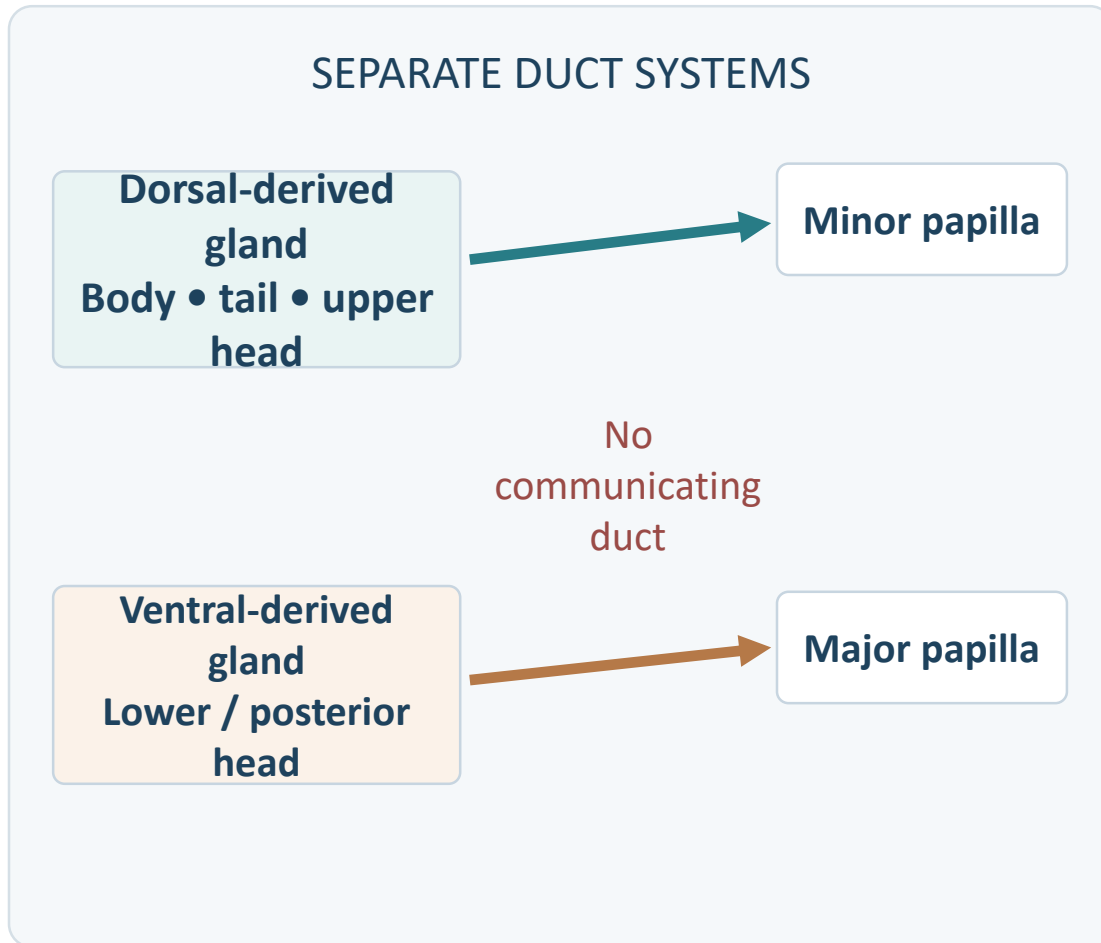
- The major papilla of Vater also receives the common bile duct.
- The minor papilla is the terminal outlet of the dorsal duct.
- In divisum, these two routes do not unite upstream.

Normal drainage: The fused configuration



- Main pancreatic duct drains predominantly via the major papilla.
- The accessory duct can drain through the minor papilla.
- Communicating ducts distinguish this from complete divisum.

Divisum drainage: The unfused configuration



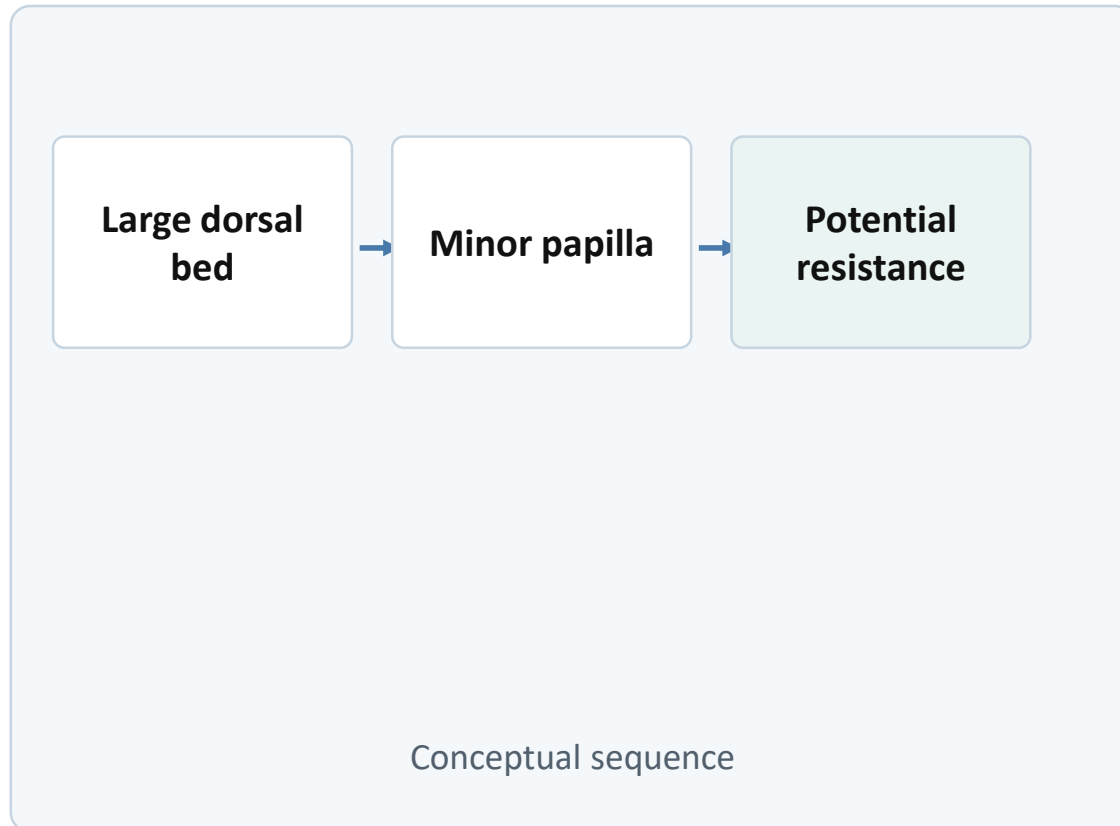
- Most tissue drains via Santorini and the minor papilla.
- The ventral duct of Wirsung drains a small part of the head.
- The ducts lack the usual major communication.

The dominant drainage route is reversed

Normal	Predominant → major papilla
Divisum	Predominant → minor papilla
Clinical question	Can minor outflow keep pace?

- The anatomical landmark is the papilla receiving bulk secretion.
- Major papilla predominance describes typical fused anatomy.
- Minor papilla predominance defines the functional drainage concern in divisum.

Why the small-caliber minor papilla is relevant



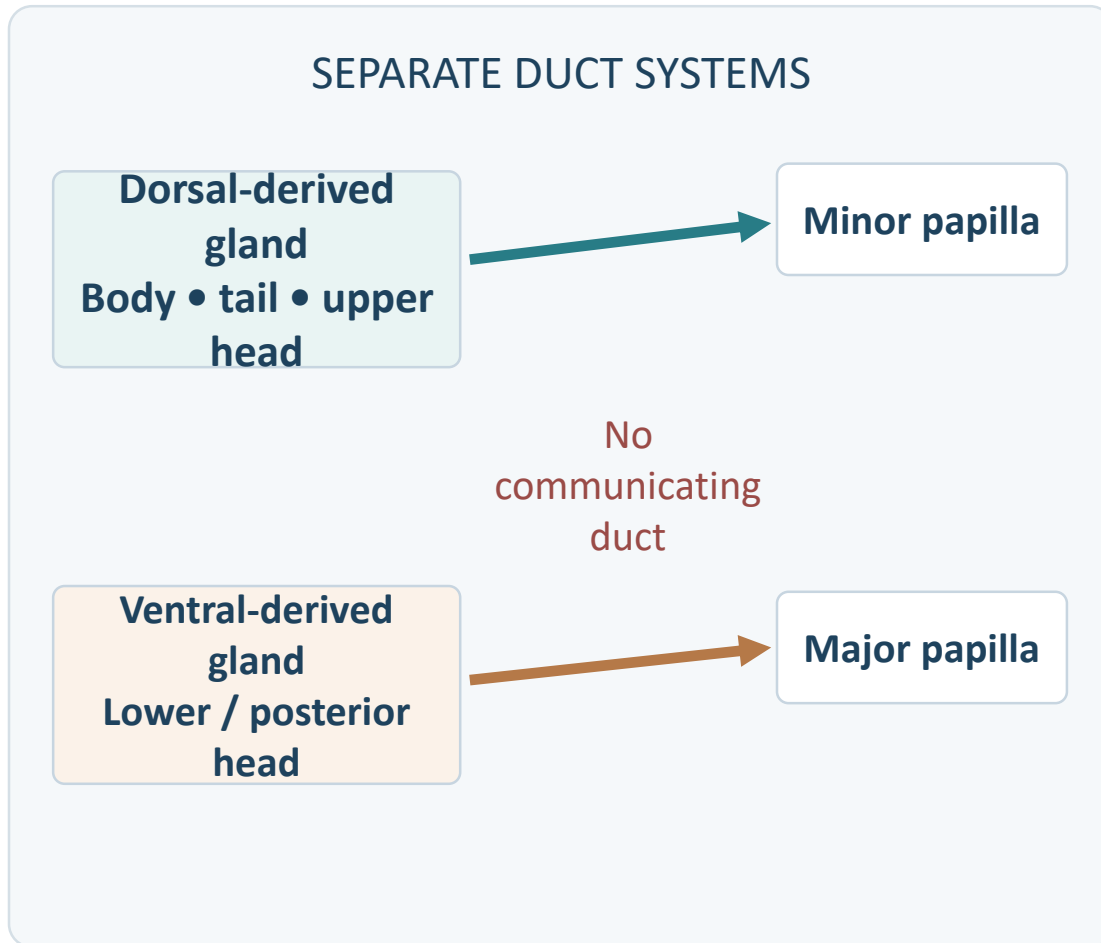
- The minor papilla as a small-caliber outlet.
- The dorsal primordium supplies most exocrine parenchyma.
- Relatively high flow through a narrow outlet motivates an outflow hypothesis.

The essential normal-versus-divisum comparison

Duct fusion	Present absent
Bulk drainage	Major minor
Ventral territory	Part of fused pathway small head area

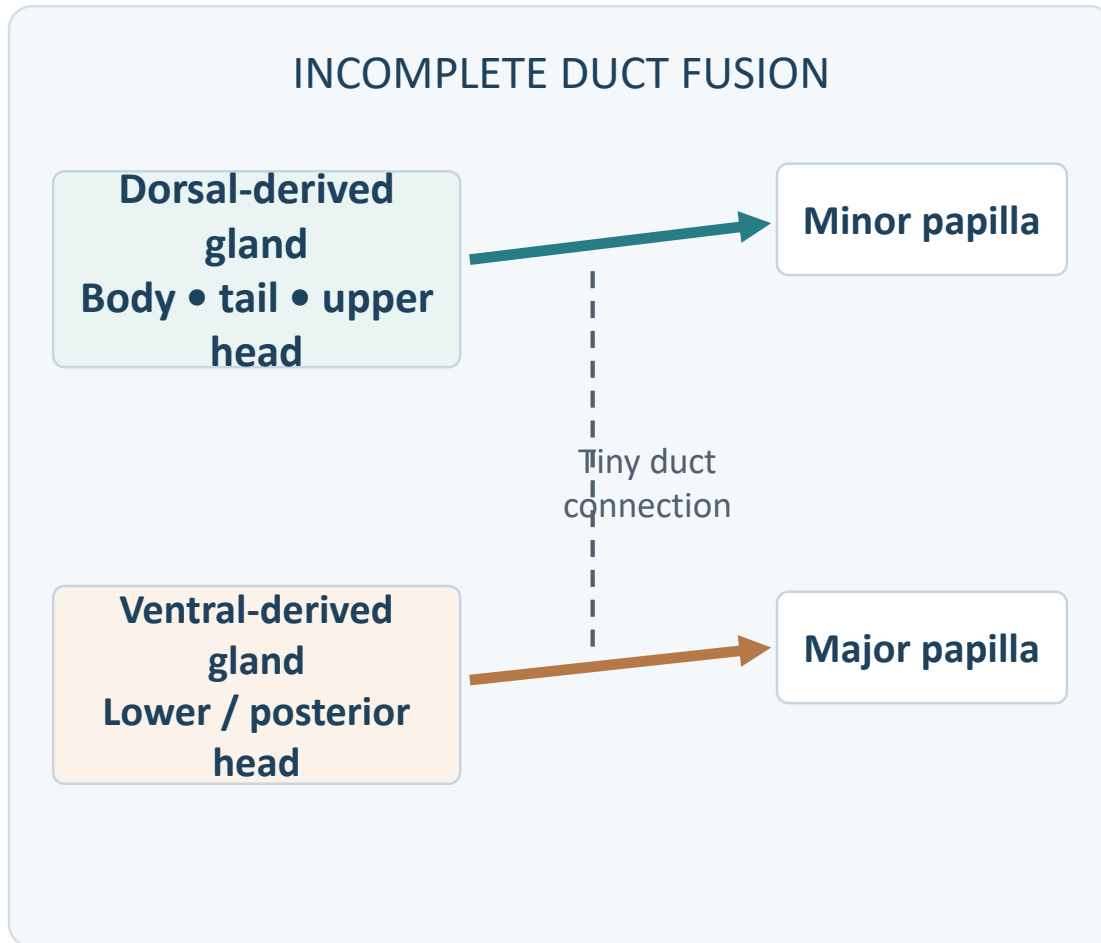
- Anatomical fusion—not pancreatic size—is the defining difference.
- The terminal papilla of dominant drainage changes.
- Both patterns can coexist with otherwise intact pancreatic tissue.

Complete divisum: No ductal communication



- Complete divisum means the dorsal and ventral ducts remain separate.
- The dorsal duct reaches the minor papilla independently.
- A small ventral duct empties through the major papilla.

Incomplete divisum: A residual communication



- Incomplete divisum retains a small duct connecting both systems.
- Despite this connection, dorsal-to-minor drainage may predominate.
- The distinction requires careful visualization of the tiny connecting duct.

Ductal phenotype must be described precisely

Complete

No dorsal–ventral link

Incomplete

**Small communicating
branch**

Report

**Papillary drainage +
connection**

- A report should describe the observed communication status.
- Prominent dorsal drainage may persist despite incomplete fusion.
- Avoid inferring complete divisum when image resolution is inadequate.

Divisum usually remains asymptomatic

Most

Individuals with this congenital variant have no symptoms

- Many cases are incidental ductal findings.
- Anatomical nonfusion alone does not establish obstructive disease.
- A clinical assessment depends on actual pancreatitis or other objective findings.

Pancreatitis develops in a minority

≈5%

Patients with pancreatitis, as reported by
Robbins

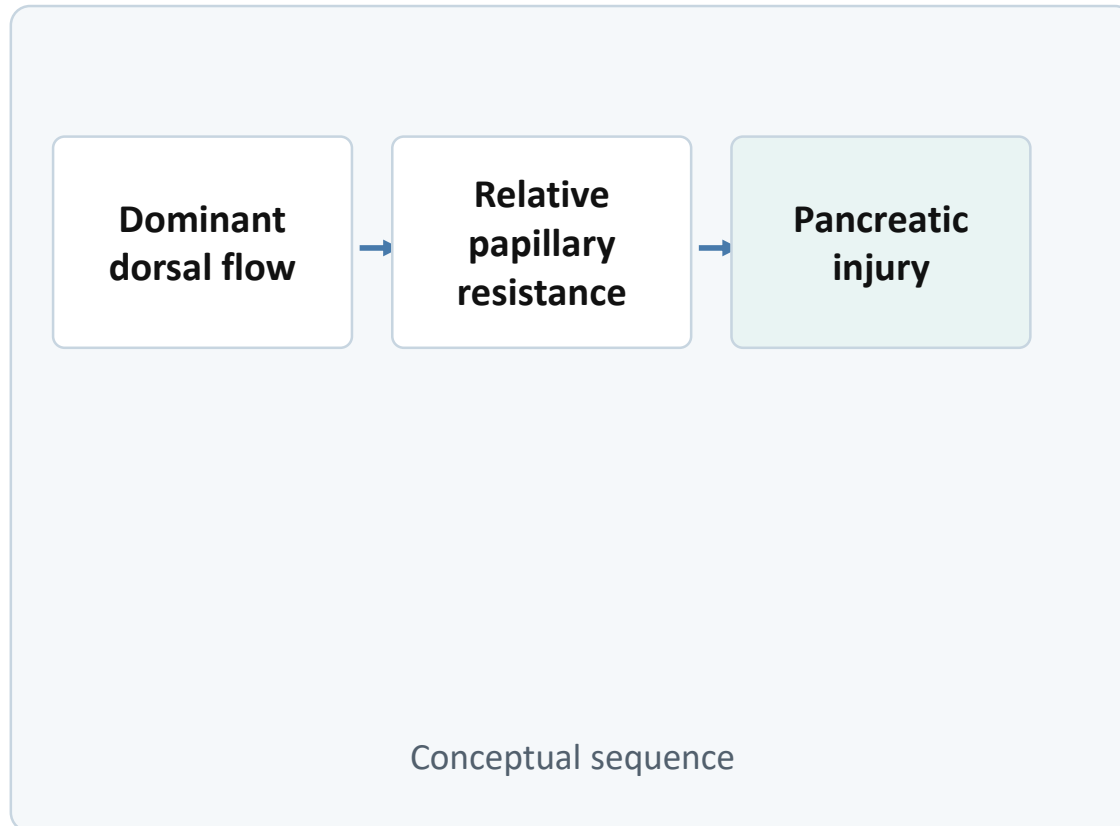
- Only about 5% develop pancreatitis.
- The majority never experience the proposed drainage complication.
- Do not present divisum as an inevitable cause of pancreatic injury.

Why does a common variant seldom cause disease?

Anatomy	Nonfusion is common enough
Phenotype	Most remain asymptomatic
Mechanism	Outflow limitation is proposed
Modifiers	Coexisting susceptibility matters

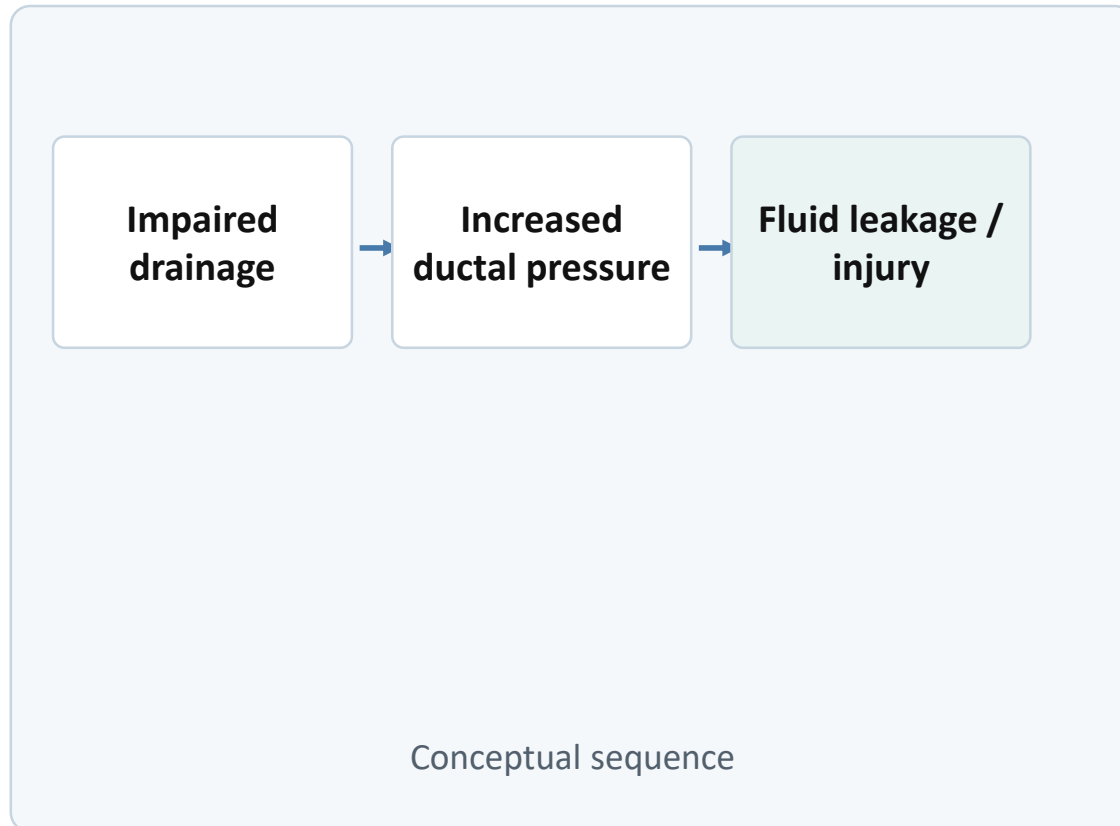
- Anatomical configuration and clinical illness are not synonymous.
- The literature proposes rather than proves minor papillary obstruction in every case.
- Other pancreatitis etiologies and genetic modifiers need evaluation.

The proposed outflow-obstruction mechanism



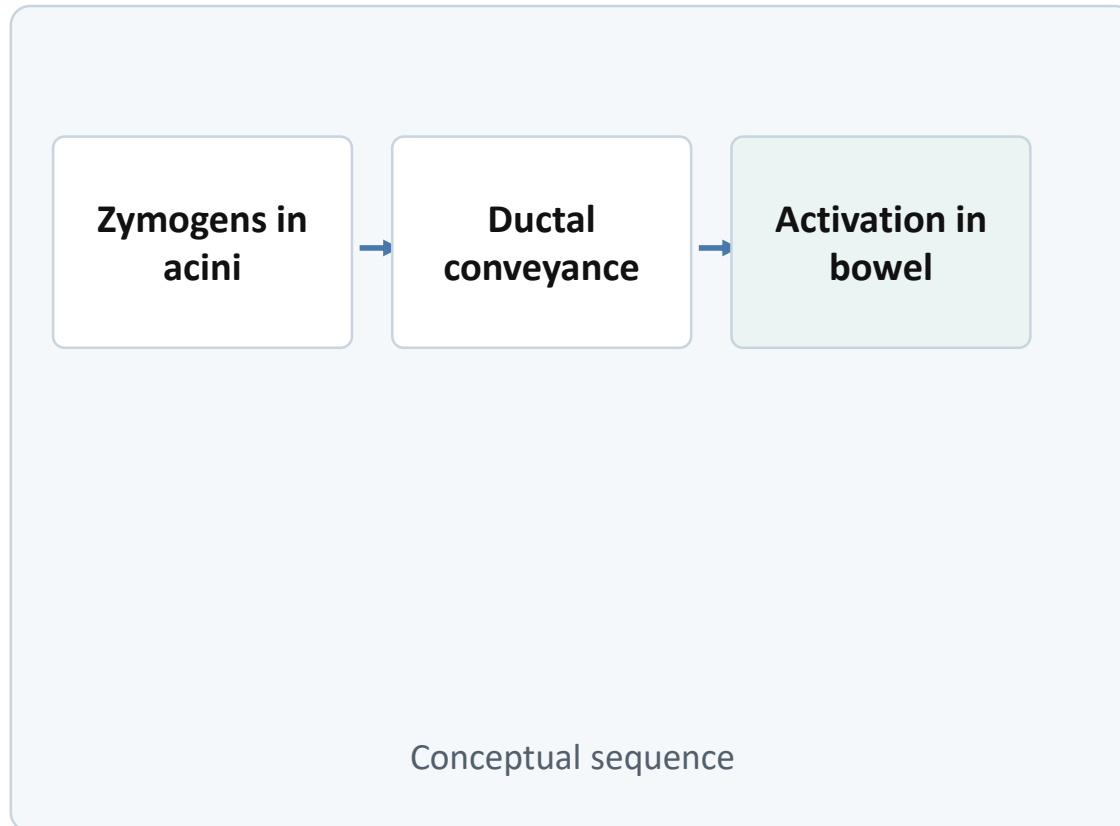
- Most secretion passes through the minor papilla.
- Limited drainage may increase ductal pressure in susceptible patients.
- Obstructive stress is a proposed route to pancreatitis, not a universal outcome.

Duct pressure and interstitial enzyme exposure



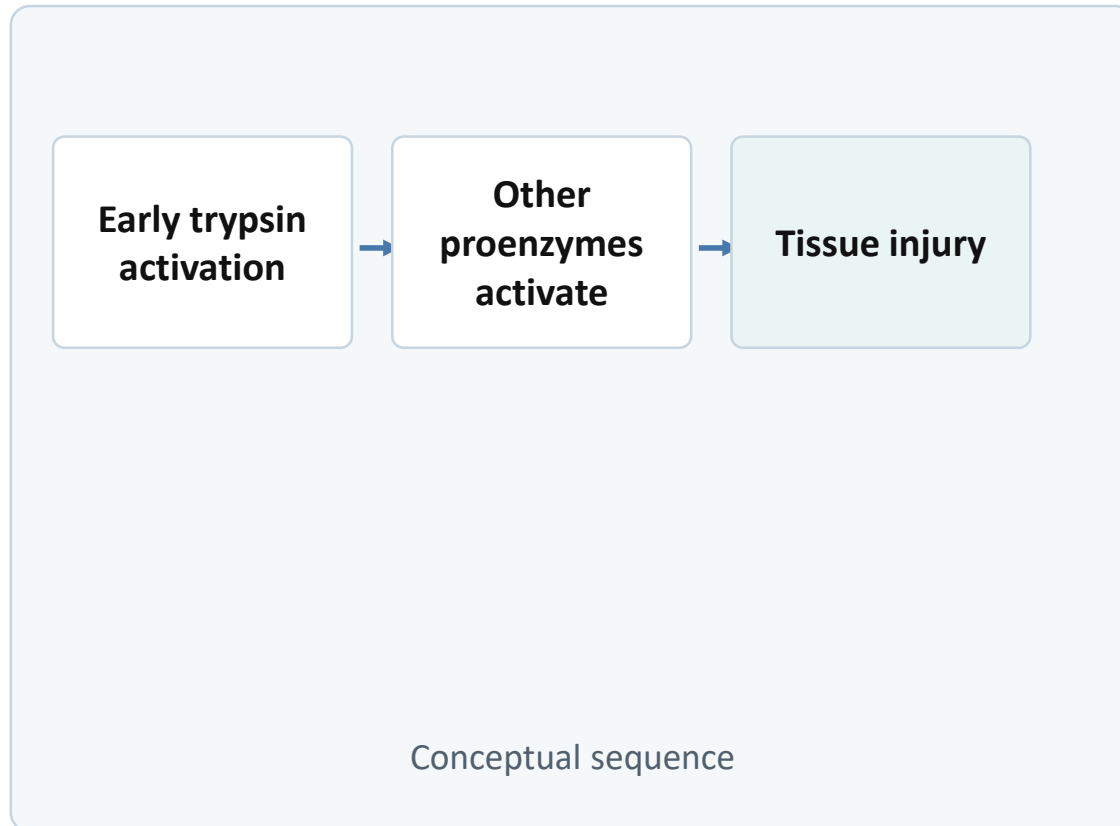
- Obstructive pancreatitis to increased ductal pressure.
- Enzyme-rich pancreatic fluid may leak into the interstitium.
- Subsequent tissue injury may amplify inflammation.

Digestion requires protected enzyme handling



- Digestive enzymes are predominantly synthesized as inactive precursors.
- Secretion normally travels in ducts before intestinal activation.
- Premature activation within pancreas predisposes to autodigestion.

The role of trypsin in pancreatic injury



- Inappropriate intrapancreatic trypsin activity activates other enzymes.
- Enzyme-mediated injury can affect parenchyma and fat.
- The ensuing inflammation may further injure acinar cells.

Why a duct variant is only one risk modifier

Ductal	Possible outflow resistance
Genetic	Protease regulation susceptibility
Clinical	Competing common etiologies
Inference	Assess the whole causal picture

- Pancreatitis is multifactorial, including mechanical and genetic factors.
- Divisum can be an anatomical contributor rather than a sufficient cause.
- Clinical causation cannot be assigned from imaging alone.

Genetic susceptibility: Textbook context

PRSS1

Trypsin pathway

SPINK1

Trypsin inhibition

CFTR / CASR

**Duct physiology /
calcium regulation**

- Inherited susceptibility in pancreatitis pathogenesis.
- The pancreas divisum section proposes interaction with genetic defects.
- A gene list is context—not a divisum-specific molecular signature.

Acute pancreatitis is reversible injury

Injury

**Enzyme-mediated
damage**

Response

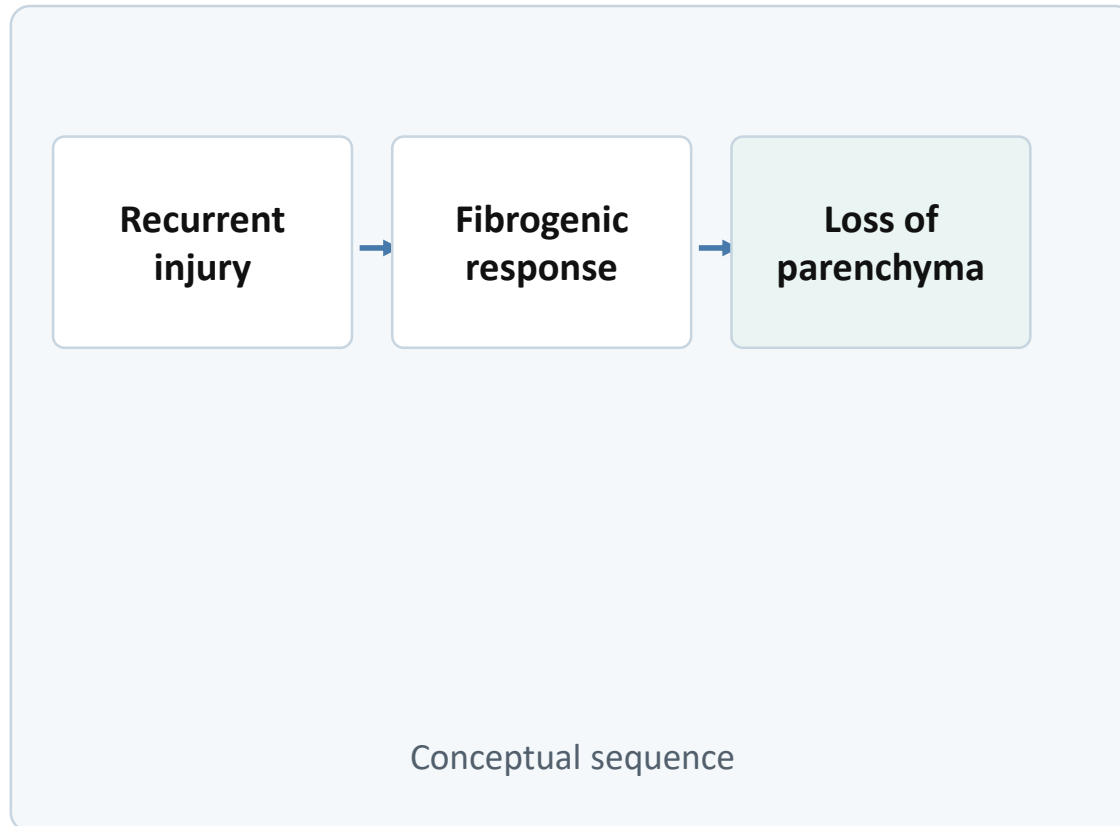
Acute inflammation

Outcome

Can resolve or recur

- Acute attacks involve inflammatory pancreatic parenchymal injury.
- Triggering mechanisms include duct obstruction and other causes.
- Divisum is considered within a broader pancreatitis differential.

Repeated attacks may culminate in chronic disease



- Repeated acute injury may initiate persistent remodeling.
- Pancreatic stellate-cell activation promotes fibrosis.
- Chronic pancreatitis involves irreversible exocrine tissue loss.

Chronic pancreatitis: What pathology demonstrates

Fibrosis

Stromal replacement

Acinar dropout

Loss of exocrine tissue

Duct distortion

**Altered excretory
architecture**

- These changes describe chronic pancreatitis in general.
- They are not diagnostic morphological markers of the disease itself.
- Causation requires clinical, radiologic, and etiologic correlation.

Divisum is not diagnosed on routine histology

Primary lesion

Duct topology

Key modality

Ductal imaging

Histology

Secondary injury if present

Limitation

No unique H&E signature

- The defining finding is the arrangement of the duct systems.
- Parenchymal tissue may be normal in asymptomatic divisum.
- Fibrosis or acinar loss indicates chronic injury, not its specific cause.

Competing causes of pancreatitis must be assessed

Mechanical	Gallstones / periampullary obstruction
Toxic	Alcohol exposure
Metabolic	Hypercalcemia / hyperlipidemia
Genetic	Inherited pancreatitis susceptibility

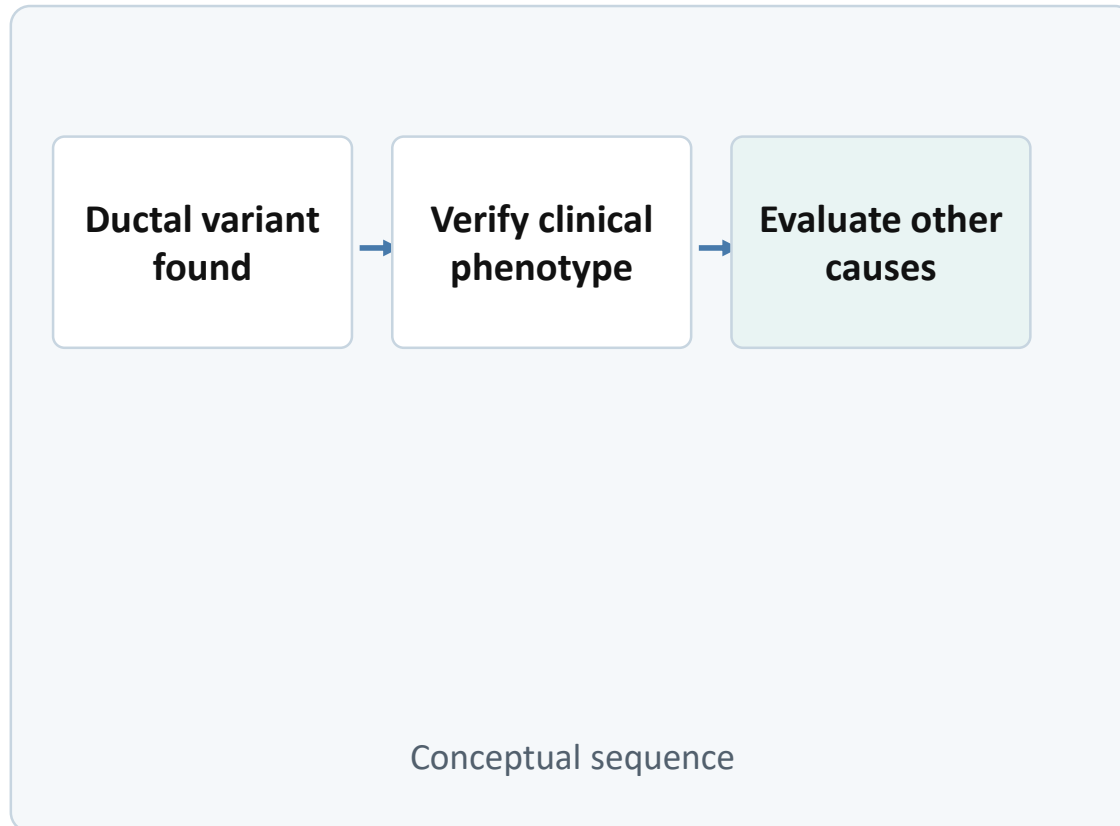
- Multiple causes of acute pancreatic injury.
- A divisum finding must not prematurely close the etiologic workup.
- Common explanations may account for attacks despite coincident divisum.

Clinical presentations form a spectrum

Incidental	No symptoms
Recurrent acute	Documented inflammatory attacks
Chronic injury	Structural / functional sequelae
Pain alone	Not proof of obstructed duct

- Incidental divisum generally needs no intervention.
- Episodes of proven pancreatitis warrant systematic etiologic review.
- Pain without pancreatitis is a different management situation.

First decision: Incidental or clinically implicated?



- Do not equate incidental anatomy with an actionable disease.
- Recurrent documented pancreatitis raises a different question from pain alone.
- Alternative and coexisting etiologies remain central.

Noninvasive visualization of ductal anatomy

MRCP

Ductal fluid anatomy

S-MRCP

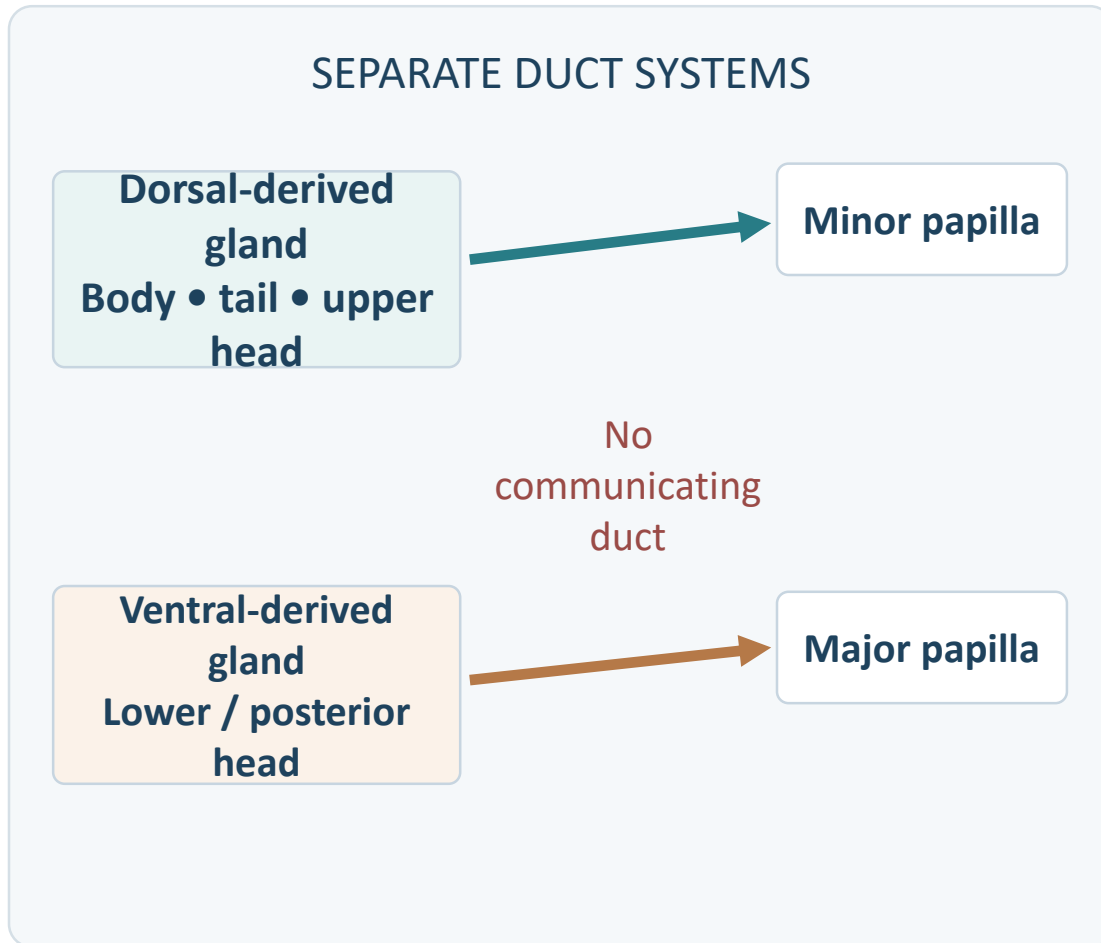
**Secretin-enhanced
visibility**

EUS

**Detailed pancreatic
assessment**

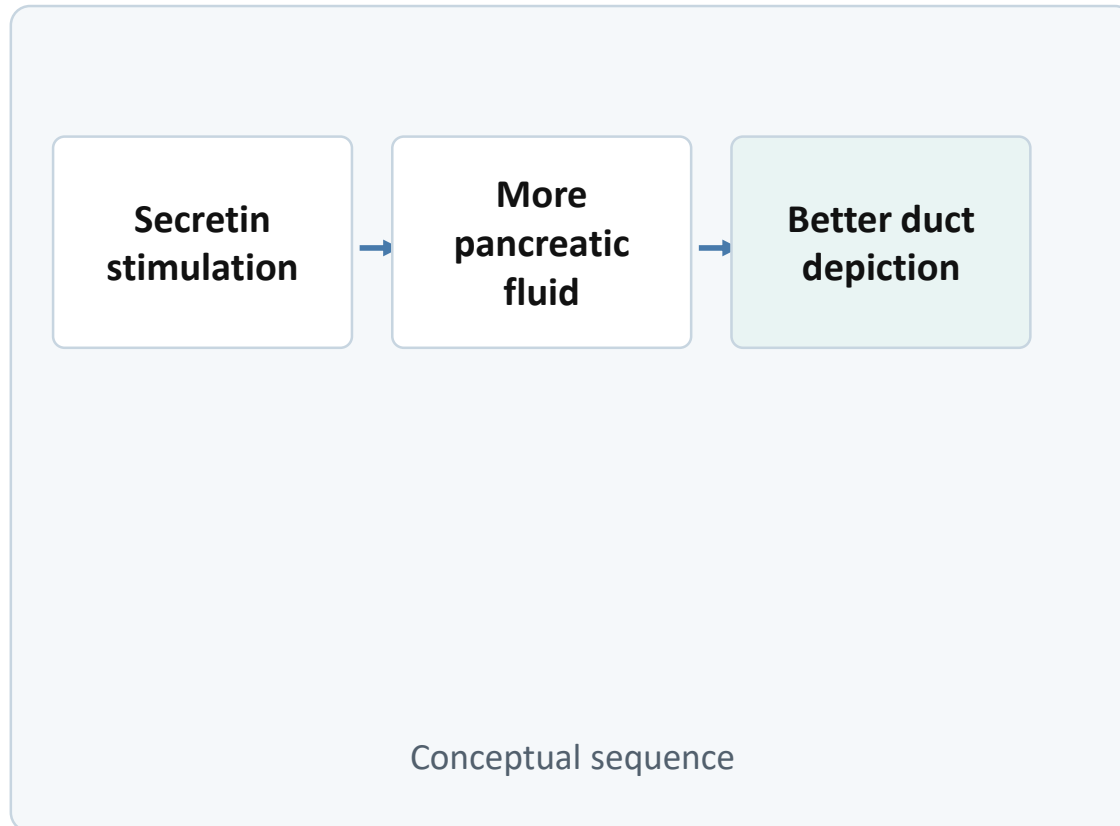
- MRCP can map dominant dorsal and ventral duct pathways.
- Secretin stimulation can enhance fluid signal and duct visibility.
- Endoscopic ultrasound helps evaluate unexplained recurrent pancreatitis.

MRCP: Follow the dorsal duct to its endpoint



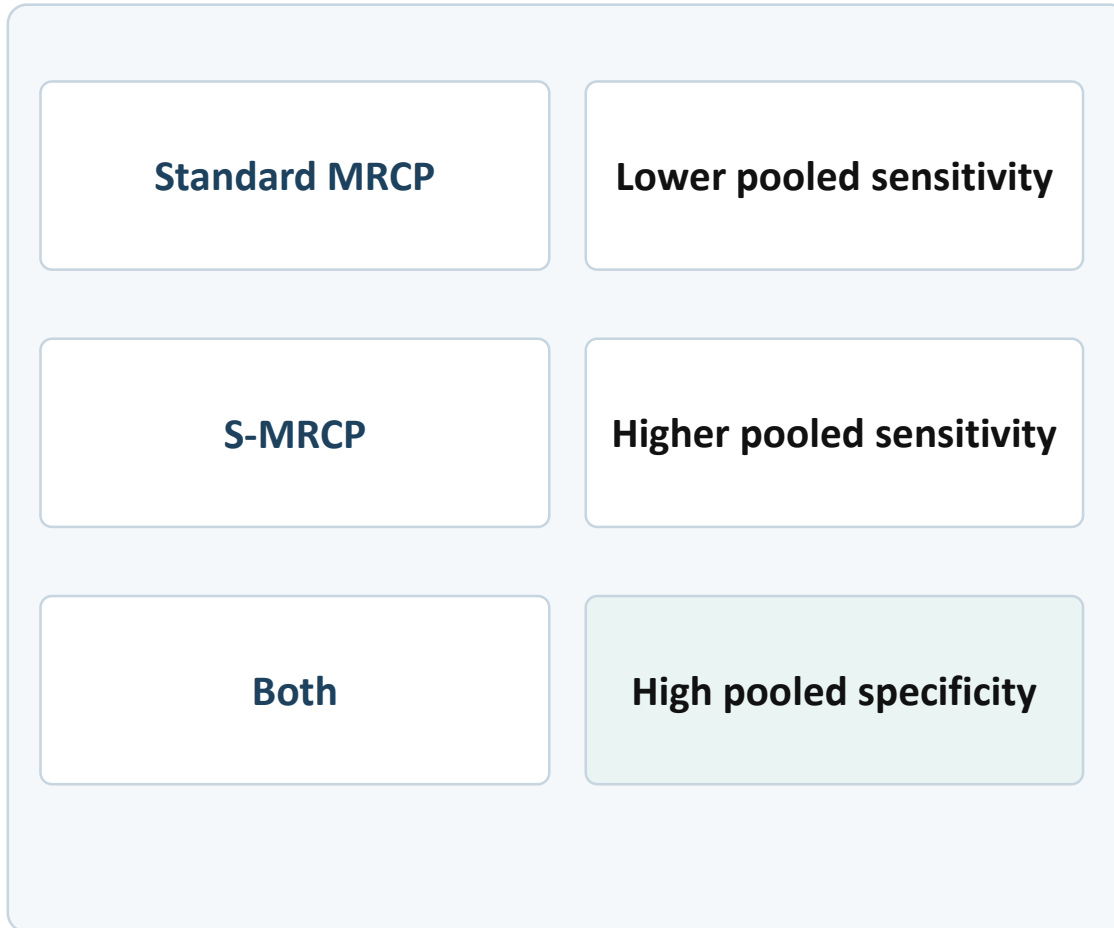
- Identify the dominant duct from the body and tail of the gland.
- Trace its course to the duodenal minor papilla.
- Assess whether there is a ductal connection to the ventral system.

Secretin-enhanced MRCP: The physiologic principle



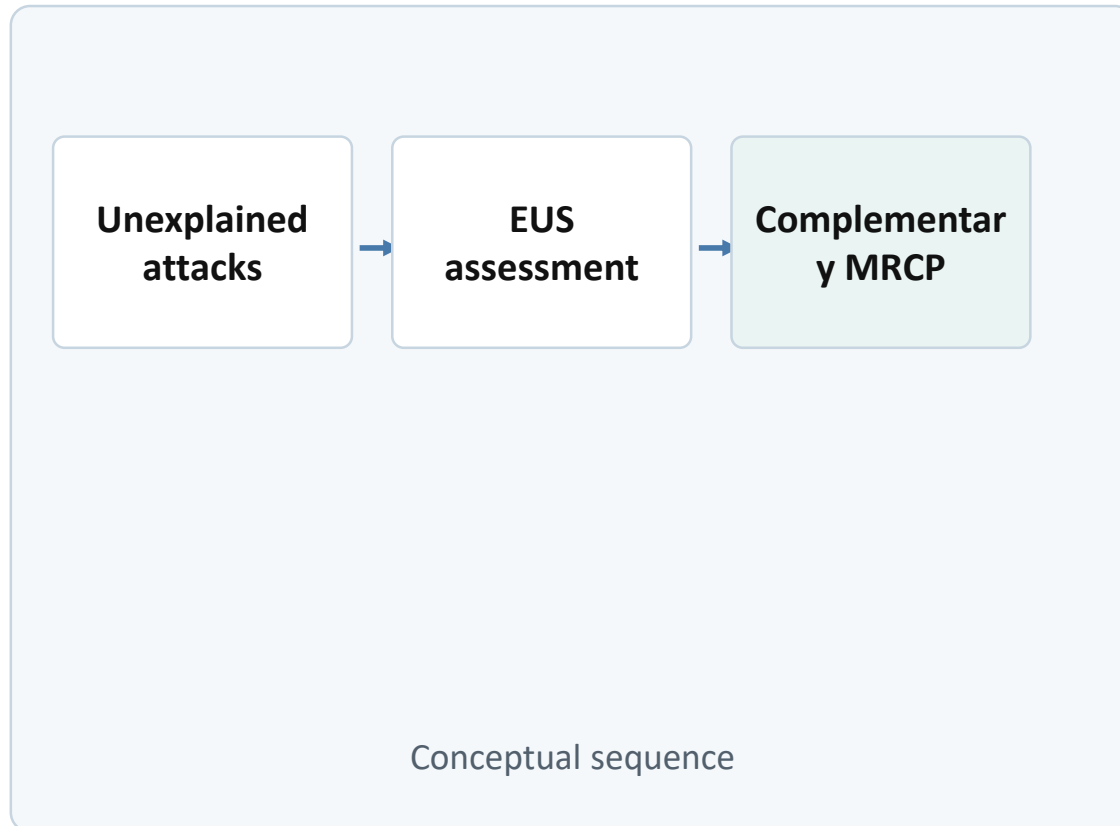
- Secretin increases pancreatic exocrine fluid secretion.
- Fluid-sensitive MR imaging accentuates duct lumen signal.
- The technique may improve visualization when standard MRCP is inconclusive.

MRCP and S-MRCP: Accuracy is not identical



- 2016 meta-analysis: Sensitivity 0.59 for MRCP, 0.83 for S-MRCP.
- Pooled specificities were 0.99 for both approaches.
- These summary estimates are context-dependent, not a universal guarantee.

EUS complements ductal imaging



- AGA 2022 prefers EUS after an unrevealing initial pancreatitis evaluation.
- Contrast MRI/MRCP is a reasonable complementary or alternative test.
- Local expertise and availability influence investigation choice.

Beware false anatomical interpretation

Small duct

Communication may be hard to see

Chronic injury

Ductal stricture may mislead

Variant loop

Can mimic duct anatomy

Response

Correlate with expert imaging

- Ductal anatomy may be incompletely visualized on standard sequences.
- Stricture or an ansa loop can complicate interpretation of divisum.
- An equivocal report should express uncertainty rather than overclassify.

A radiology report should answer three questions

Drainage

Which papilla receives dorsal flow?

Connection

Is dorsal–ventral fusion seen?

Consequences

Dorsal dilation or santorinicele?

Context

Any alternative explanation?

- The report should establish the relevant drainage topology.
- Look for objective signs of potential outflow obstruction.
- Interpret duct configuration alongside other pancreatic findings.

ERCP is not a routine test for incidental divisum

MRCP / EUS

Diagnostic investigation

ERCP

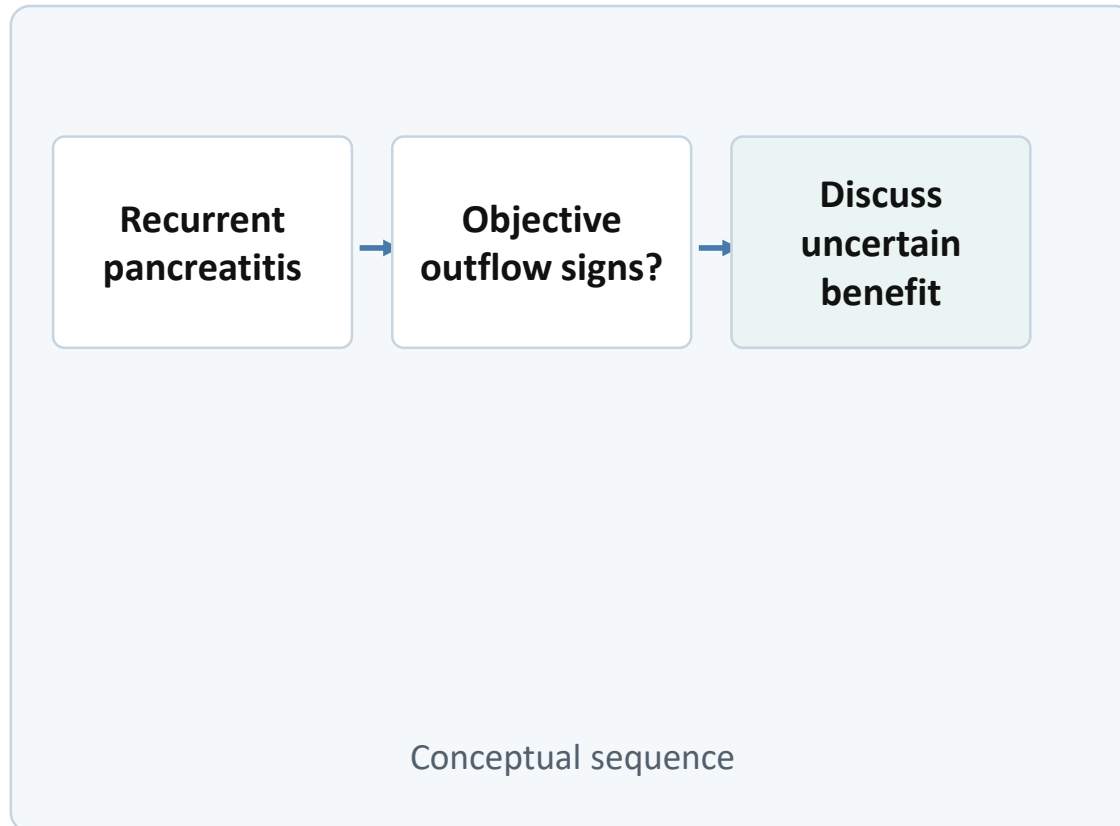
**Potential therapeutic
procedure**

Risk

**Procedure-related
pancreatitis**

- Noninvasive and less-invasive tests can establish duct anatomy.
- ERCP has procedure-related risks and uncertain preventive benefit.
- A purely incidental finding is not an indication for papillary therapy.

AGA 2022: A selective endoscopic approach



- AGA considered minor-papilla endotherapy only in selected patients.
- Possible objective markers included dorsal duct dilatation or santorinicele.
- The guidance explicitly described benefit as controversial.

Pain alone is not an ERCP indication

Pain only

No ERCP for divisum

Documented RAP

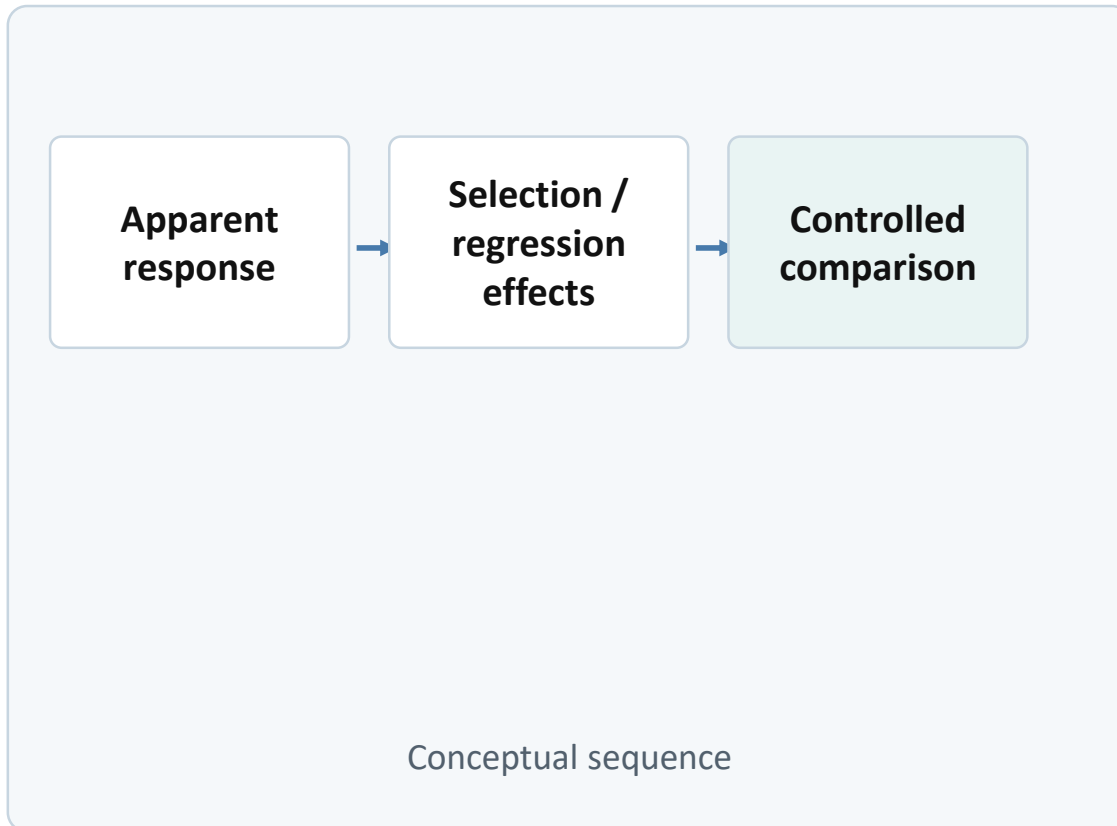
Evaluate etiologies

Outflow signs

**Potentially relevant, not
definitive**

- AGA 2022 states there is no role for ERCP to treat pain alone.
- Symptom severity must not substitute for objective pancreatic injury.
- Decisions need shared discussion of uncertainties and harm.

Why sham-controlled evidence is important



- Uncontrolled improvement cannot isolate benefit of papillotomy.
- Recurrent pancreatitis may vary over time without intervention.
- A randomized sham comparison tests benefit beyond these effects.

SHARP trial: Direct test of minor papillotomy

Population	148 with divisum and RAP
Design	Randomized, sham-controlled
Intervention	ERCP + minor papillotomy
Outcome	New acute pancreatitis >30 d

- The multicenter trial was published online January 2026.
- Participants had unexplained recurrent acute pancreatitis and divisum.
- The primary end point was subsequent acute pancreatitis after day 30.

SHARP trial: No statistically significant reduction

Observed recurrence after 30 days

Minor papillotomy



Sham ERCP



Trial: N = 148; Adjusted HR 0.83 (95% CI 0.49–1.41)

- Papillotomy: 26/75 patients (34.7%) developed acute pancreatitis.
- Sham: 32/73 patients (43.8%) developed acute pancreatitis.
- Adjusted HR 0.83 (95% CI 0.49–1.41): Benefit was not established.

SHARP trial: Procedure-related injury matters

Papillotomy arm

**11/75 acute attacks ≤ 30
d**

Sham arm

6/73 acute attacks ≤ 30 d

Interpretation

**Consider procedural
harm**

- Acute pancreatitis within 30 days occurred in both trial groups.
- The papillotomy arm had numerically more early attacks.
- Risk–benefit decisions must include ERCP complications.

How the 2026 trial changes clinical interpretation

AGA 2022

**Endotherapy: Uncertain,
selective**

JAMA 2026

**No demonstrated
preventive effect**

Practice reasoning

**Avoid routine minor
papillotomy**

- Older expert advice allowed carefully selected minor papillary intervention.
- The newer sham-controlled trial did not show significant prophylactic benefit.
- Evidence should be updated without rewriting what Robbins actually states.

Evidence limits: Do not overgeneralize SHARP

Studied	Idiopathic recurrent acute pancreatitis
Excluded	Other etiologies / calcific CP
Interval	Outcome beyond 30 days
Inference	Applies to trial-like populations

- The trial was not a study of all patients with divisum.
- Its results do not imply every duct intervention is ineffective in all contexts.
- It does challenge routine papillotomy to prevent recurrent attacks.

Vignette: Incidental ductal variant

TEACHING VIGNETTE (HYPOTHETICAL)

Asymptomatic patient

MRCP shows dorsal-to-minor drainage

No documented pancreatitis

- First establish that the finding is incidental.
- The anatomical variant alone is not evidence of ductal obstruction.
- Avoid unnecessary ERCP or elective minor-papilla intervention.

Vignette: Recurrent acute pancreatitis

TEACHING VIGNETTE (HYPOTHETICAL)

Multiple documented acute attacks

Divisum on ductal imaging

Other causes incompletely assessed

- The proper task is a comprehensive etiologic assessment.
- Evaluate gallstone, alcohol, metabolic and inherited susceptibility.
- Do not assume a minor papillotomy will prevent recurrence.

Vignette: Fibrosis with pancreas divisum

TEACHING VIGNETTE (HYPOTHETICAL)

Chronic pancreatic insufficiency

Fibrosis / acinar dropout

Divisum detected on MRCP

- Fibrosis establishes chronic pancreatic injury, not its cause.
- The pancreatic ductal variant may be contributory or incidental.
- Seek other etiologies and objective evidence of outflow abnormality.

Differentiate the nearby congenital anomalies

Divisum

Failure of duct fusion

Annular pancreas

Encircles 2nd duodenum

Ectopic pancreas

Pancreatic tissue at other sites

- Annular pancreas can produce duodenal obstruction.
- Ectopic pancreas is heterotopic pancreatic tissue, often incidental.
- Only divisum is defined by dominant dorsal drainage to the minor papilla.

Clinical interpretation: Five essential distinctions

Anatomic	Divisum ≠ disease
Frequency	Common variant ≠ common pancreatitis
Causality	Coexistence ≠ proof
Procedure	ERCP ≠ benign diagnostic test

- A fifth distinction is evidence: Expert consensus differs from randomized outcomes.
- Record the precise observed duct pattern and clinical phenotype.
- Use a complete pancreatitis evaluation before attributing causation.

Review questions: Anatomy and clinical decisions

TEACHING VIGNETTE (HYPOTHETICAL)

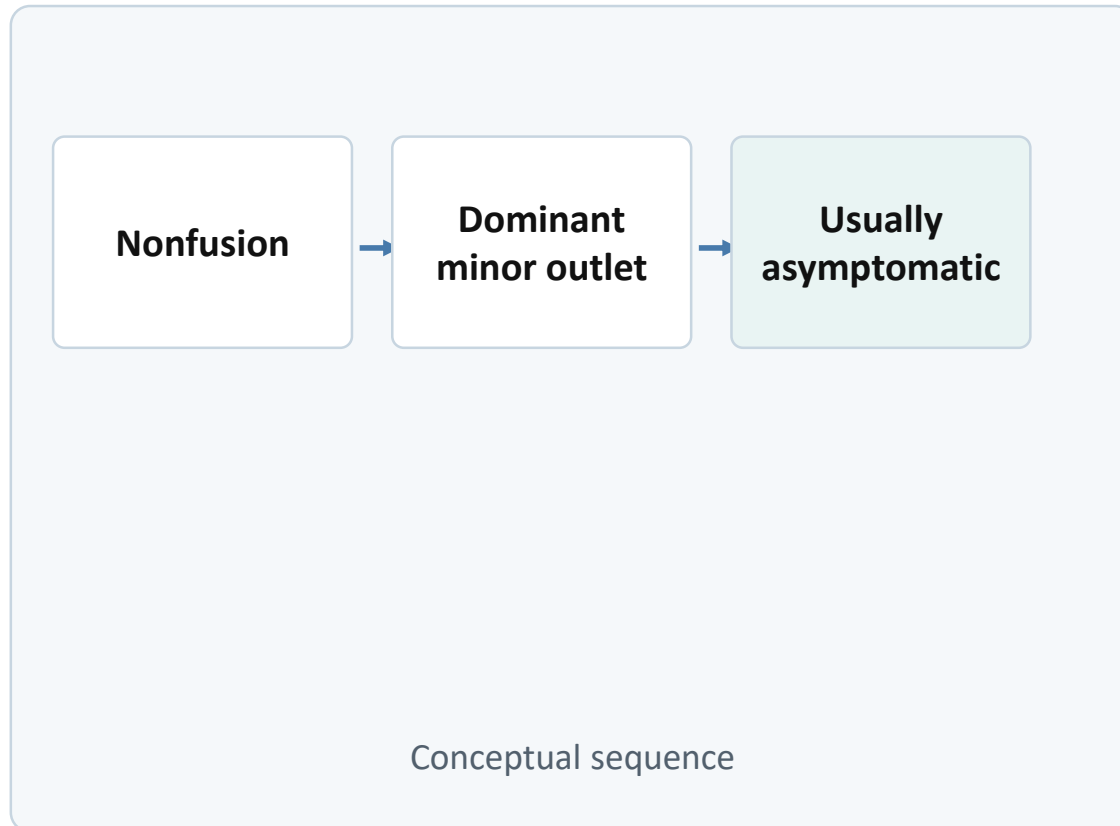
Most pancreatic output in divisum?

What defines complete divisum?

ERCP for pain alone?

- Dorsal-derived gland → duct of Santorini → minor papilla.
- Complete divisum lacks an interductal dorsal–ventral connection.
- Pain alone does not justify ERCP for pancreas divisum.

Pancreas divisum: Integrated conclusions



- Robbins: Congenital ductal nonfusion; Reported frequency 3–10%.
- An outflow contribution to pancreatitis is proposed in a minority.
- JAMA 2026: Routine minor papillotomy did not demonstrate preventive benefit.

Selected references and source boundaries

Textbook	Robbins 11e, Ch 19 (2026)
Imaging	Shen et al., Sci Rep 2016
Guidance	AGA CPU, Gastro 2022
Trial	JAMA SHARP, 2026

- Primary anatomical and pathology teaching derives from Robbins.
- Diagnostic imaging and procedural advice are documented external context.
- The 2026 randomized trial informs, but does not replace, the textbook anatomy.

Core anatomical text: Robbins 11th edition; diagnostic and trial findings: cited supplementary studies.