

# Necrotizing Enterocolitis

Pathogenesis • Morphology • Clinicopathologic correlations

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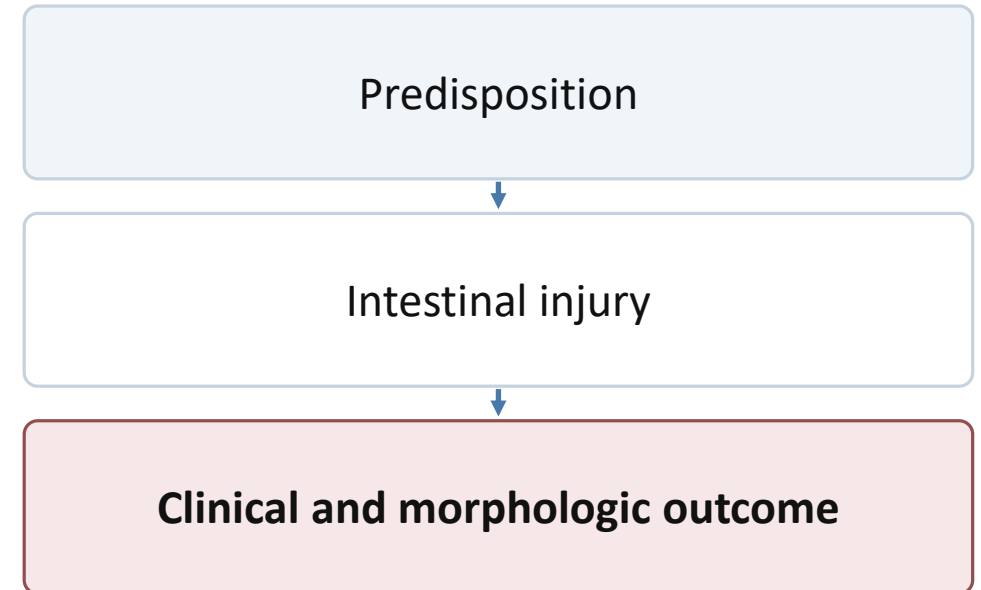
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# Learning objectives

## At the end of this session:

- Define NEC and its relationship to prematurity.
- Explain the proposed sequence from barrier injury to necrosis.
- Recognize the characteristic gross, microscopic, and clinical findings.
- Link healing to fibrosis and post-NEC stricture.



# Prematurity provides the disease context

**NEC is an important complication of preterm birth.**

- The prematurity chapter lists NEC among major hazards of immature newborns.
- Neonatal respiratory distress syndrome, sepsis, and hemorrhage are other hazards.
- This lecture examines NEC as an intestinal complication, not these separate conditions.

## Selected hazards of prematurity

Necrotizing enterocolitis

Neonatal respiratory distress syndrome

Sepsis

Germinal matrix / intraventricular hemorrhage

# What defines necrotizing enterocolitis?

The central pathological event is destructive intestinal injury.

- Inflammation and mucosal barrier failure are prominent in the proposed mechanism.
- Mucosal necrosis may extend through the entire bowel wall.
- Advanced injury can progress to perforation or systemic infection.



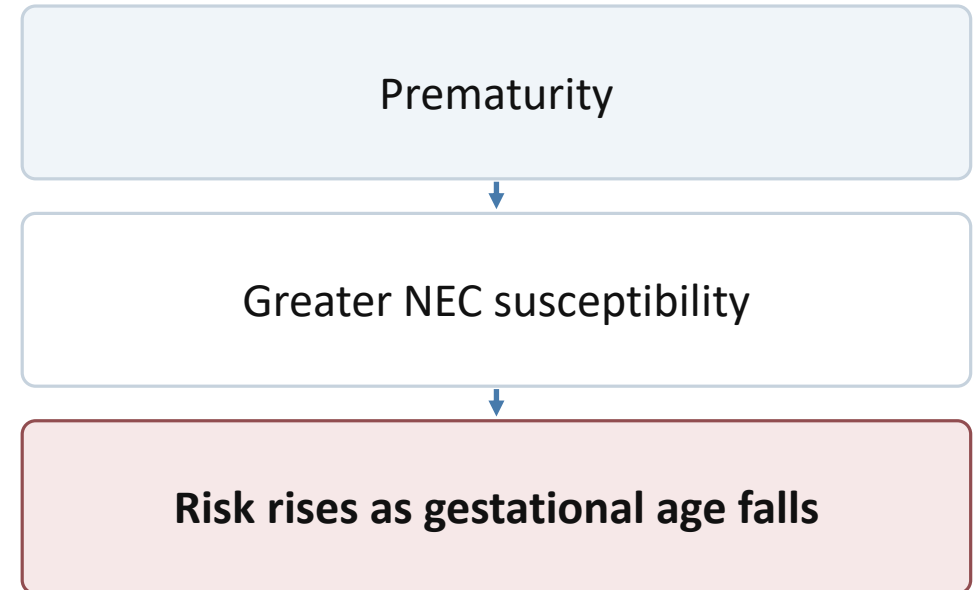
Injury begins at the barrier and may involve deeper layers

*Schematic — not a histologic or clinical specimen*

# Gestational age and susceptibility

NEC occurs most often in premature infants.

- NEC incidence increases as gestational age decreases.
- The source describes an inverse relationship, without a fixed week threshold.
- Gestational immaturity is a major predisposition, not a sufficient single cause.



# Very-low-birth-weight infants are prominent

The source uses the standard very-low-birth-weight category.

- Very low birth weight means birth weight under 1500 g.
- This population is specifically highlighted as highly affected.
- Do not confuse birth-weight classification with a causal mechanism.

**<1500 g**

Very-low-birth-weight definition

Textbook population descriptor

# The source describes a substantial disease burden

## Textbook frequency estimate for very-low-birth-weight infants:

- Approximately one in ten very-low-birth-weight infants develops NEC.
- Robbins also reports about 2500 U.S. Cases annually.
- These are textbook estimates, not independently verified current surveillance data.

**$\sim 1 / 10$**

Very-low-birth-weight infants

Source estimate; Population and period matter

# NEC ranges from mucosal injury to severe destruction

The depth and extent of intestinal injury vary.

- Necrosis may remain confined to mucosal tissue.
- In advanced lesions, the full wall becomes necrotic.
- Perforation, peritonitis, sepsis, and shock represent major complications.

## Increasing structural severity

Mucosal necrosis

Transmural necrosis

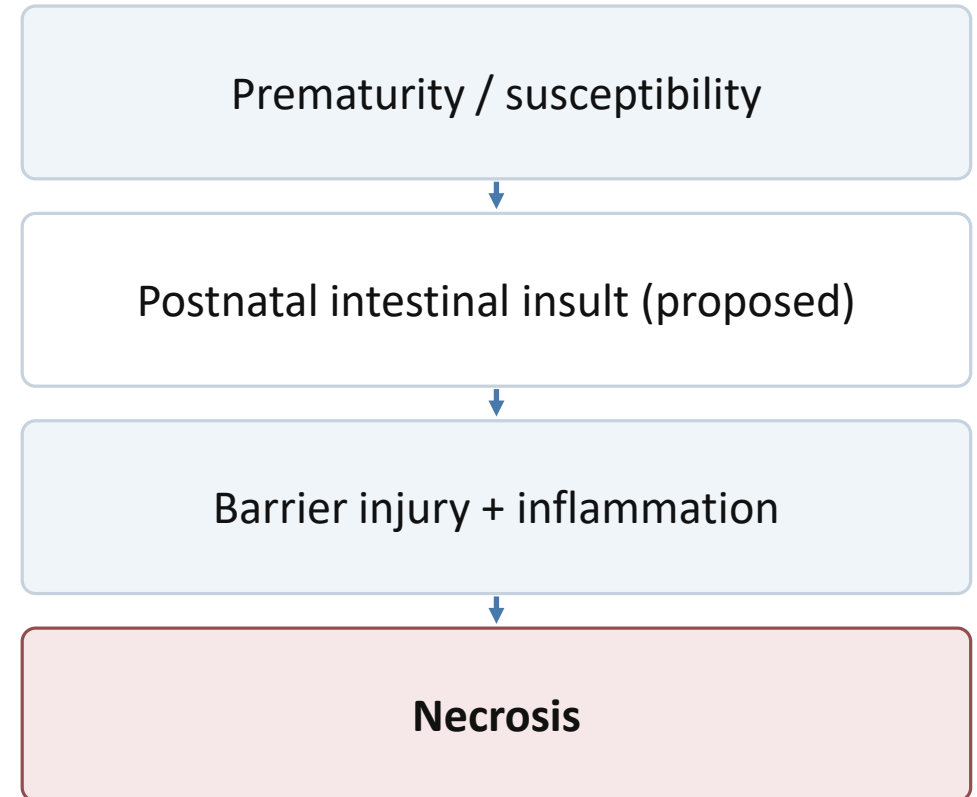
Possible perforation / peritonitis

Possible sepsis / shock

# Pathogenesis remains multifactorial

## Pathogenesis is unknown.

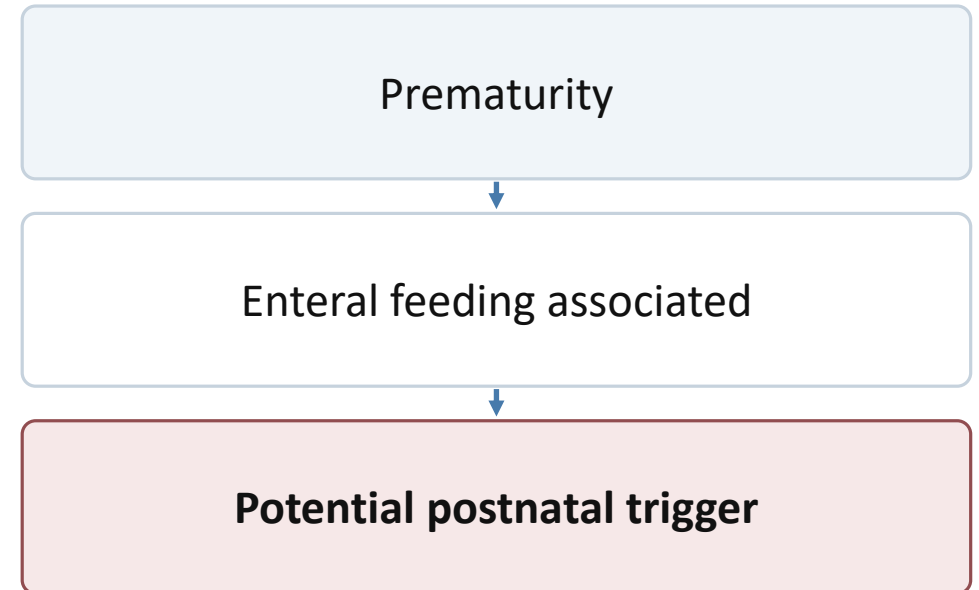
- Prematurity and enteral-feeding association are repeatedly observed.
- Potential microbial and inflammatory events are plausible contributors.
- No single initiating factor explains all cases.



# Enteral feeding is an important association

Most cases are associated with enteral feeding.

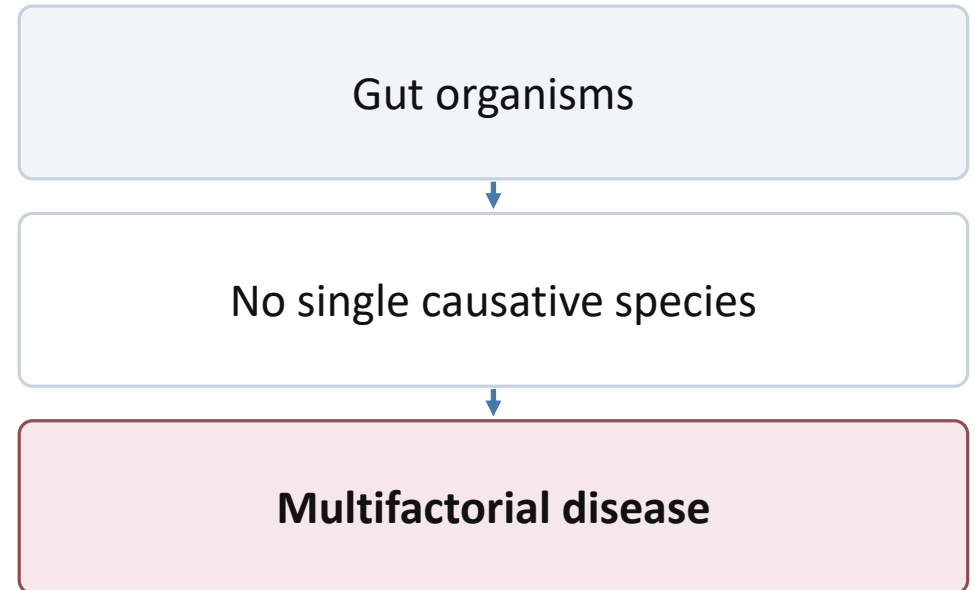
- The association suggests that postnatal luminal exposures may participate.
- The source does not establish feeding itself as an independently sufficient cause.
- The proposed insult may involve introduction of bacteria into the gut.



# Microbes may contribute without one universal pathogen

The microbial hypothesis is not a single-organism model.

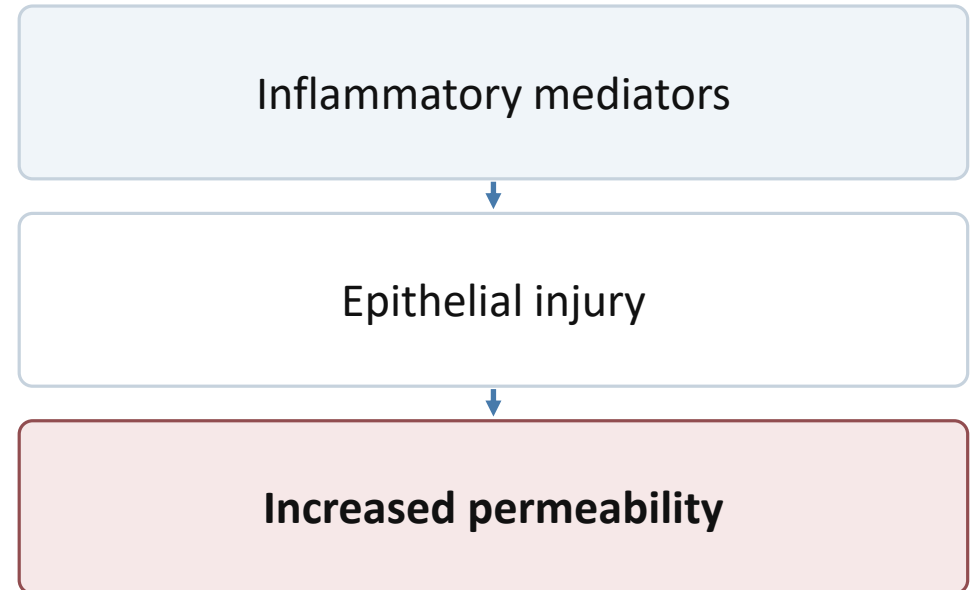
- Infectious agents may participate in NEC pathogenesis.
- No single bacterial pathogen has been linked to all NEC cases.
- The clinically relevant endpoint is bacterial access to injured tissues.



# Inflammatory mediators amplify injury

Many inflammatory mediators have been associated with NEC.

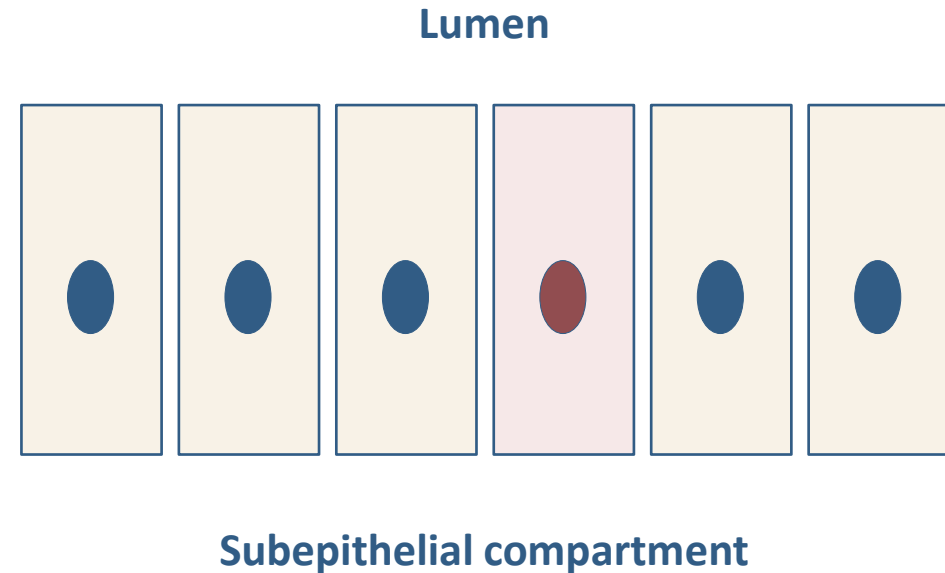
- The source proposes that mediators increase mucosal permeability.
- Enterocyte apoptosis and disrupted junctions provide possible routes.
- Mediator association is not proof of a unique initiating pathway.



# Enterocyte apoptosis compromises epithelial continuity

Epithelial cell loss can weaken the intestinal mucosal barrier.

- Robbins specifically implicates enterocyte apoptosis.
- Apoptotic loss reduces continuity of the epithelial covering.
- Barrier failure permits further inflammatory and microbial insult.



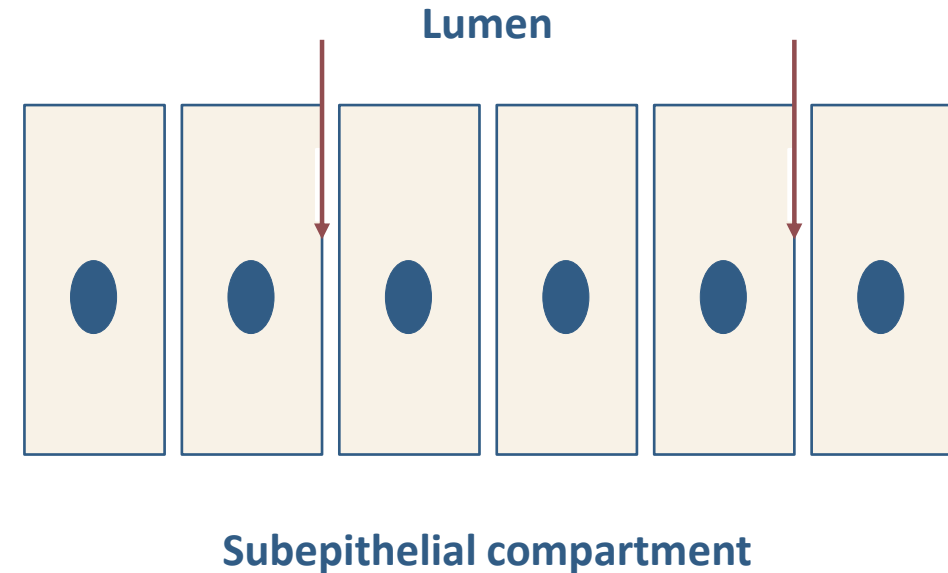
Cell loss contributes to barrier dysfunction

*Schematic — not a histologic or clinical specimen*

# Tight-junction disruption weakens the barrier

The intercellular seal is part of mucosal defense.

- Tight junctions normally limit movement between adjacent epithelial cells.
- The source proposes that NEC-associated injury compromises these contacts.
- Loss of junctional integrity increases mucosal permeability.



Interrupted junctions → increased paracellular entry

*Schematic — not a histologic or clinical specimen*

# Increased permeability is a pivotal event

Apoptosis and junctional defects converge on one consequence.

- The mucosal barrier becomes increasingly permeable.
- Luminal components gain improved access to deeper tissues.
- This can intensify injury already under way.

Enterocyte apoptosis + junction damage



Increased mucosal permeability

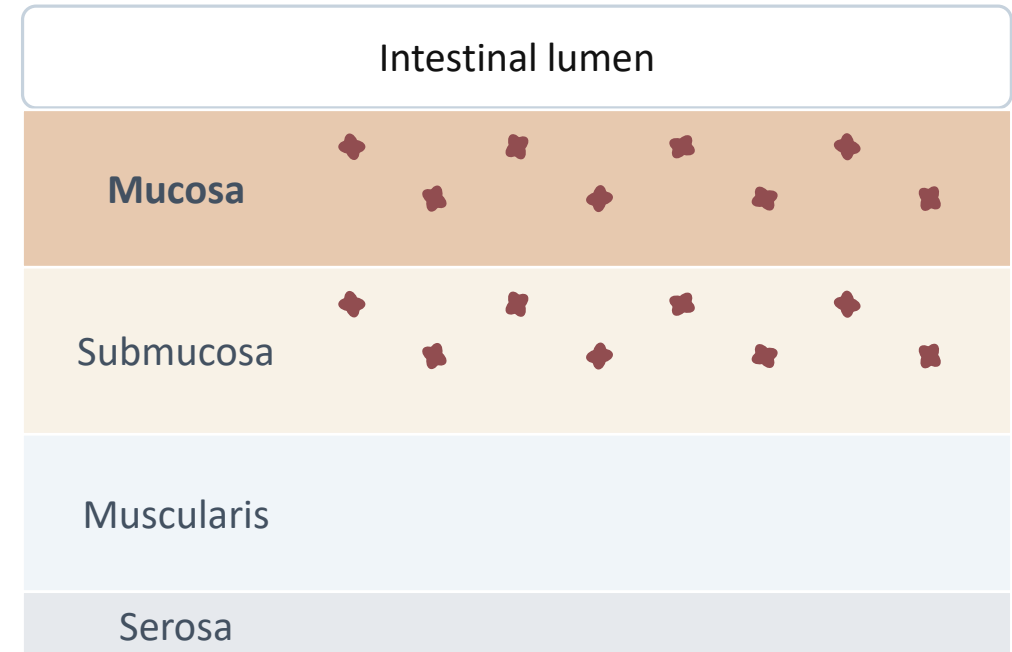


**Breakdown of barrier function**

# Barrier breakdown permits bacterial translocation

## Transluminal migration of gut bacteria.

- Intestinal bacteria cross the compromised mucosal barrier.
- Entry of gut organisms can intensify local inflammation.
- This process links epithelial dysfunction to intestinal tissue destruction.



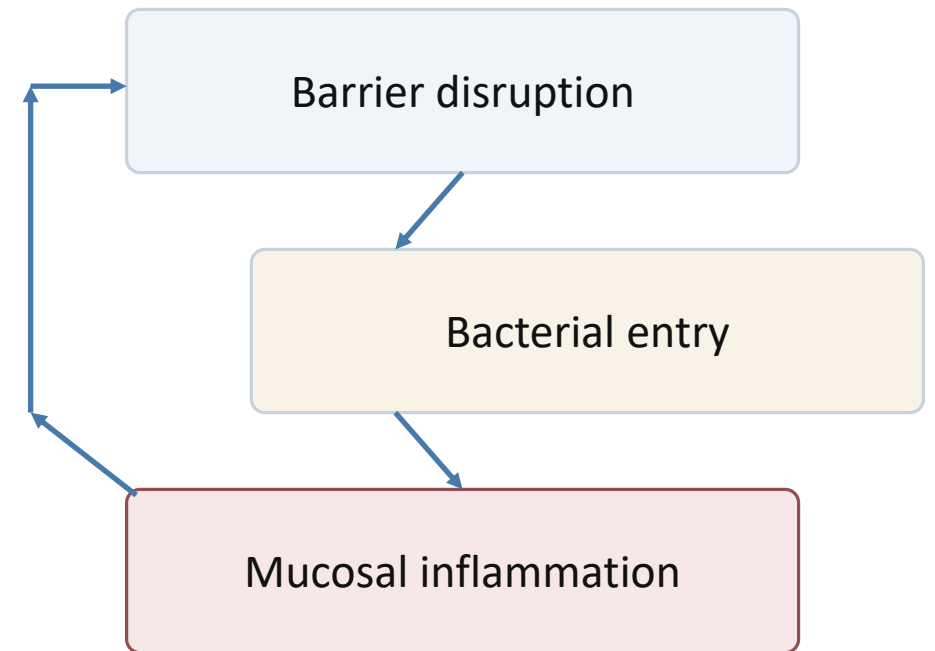
Bacteria are represented schematically, not by micrograph

*Schematic — not a histologic or clinical specimen*

# A positive-feedback loop perpetuates NEC

The injury and microbial-entry mechanisms reinforce each other.

- Mucosal injury permits bacterial entry.
- Bacterial passage stimulates further inflammation.
- Further inflammation worsens mucosal injury and entry.

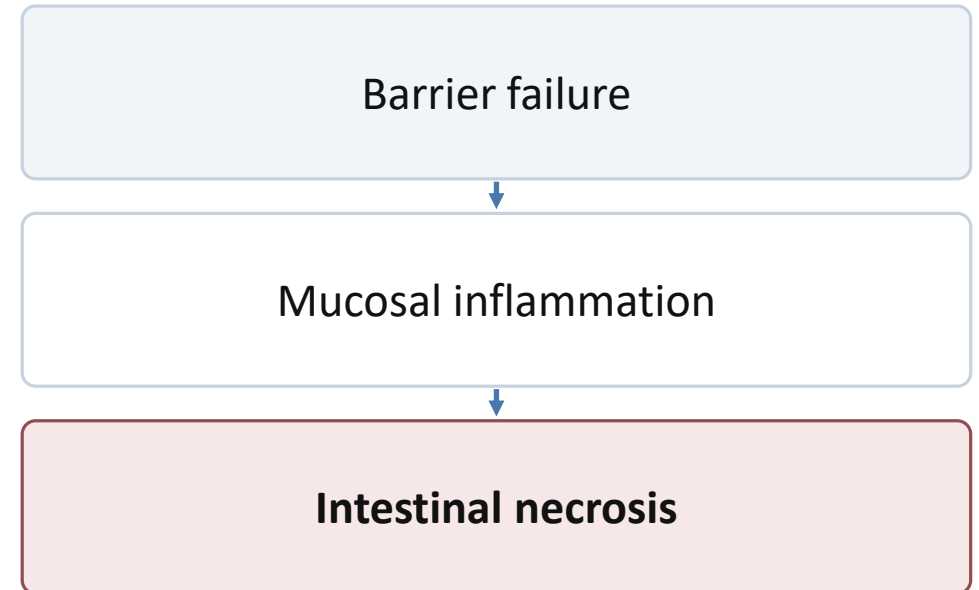


*Schematic — not a histologic or clinical specimen*

# Progressive inflammation produces necrosis

The source describes a cycle culminating in intestinal destruction.

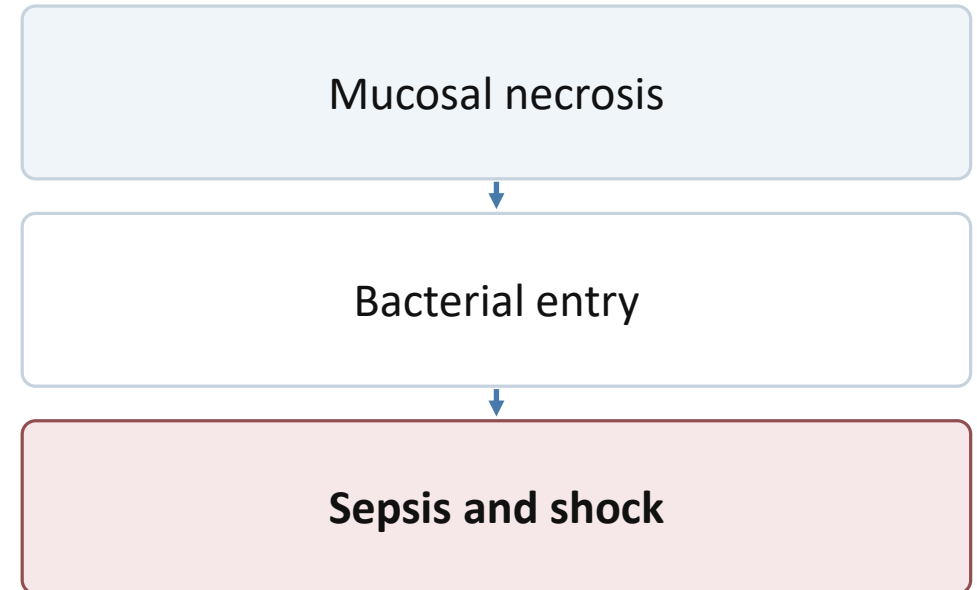
- Inflammation and continuing barrier failure promote mucosal necrosis.
- Necrosis may eventually extend through the entire wall.
- The observed depth and distribution determine the pathological appearance.



# Severe bowel injury can become systemic

The intestinal process may culminate in sepsis and shock.

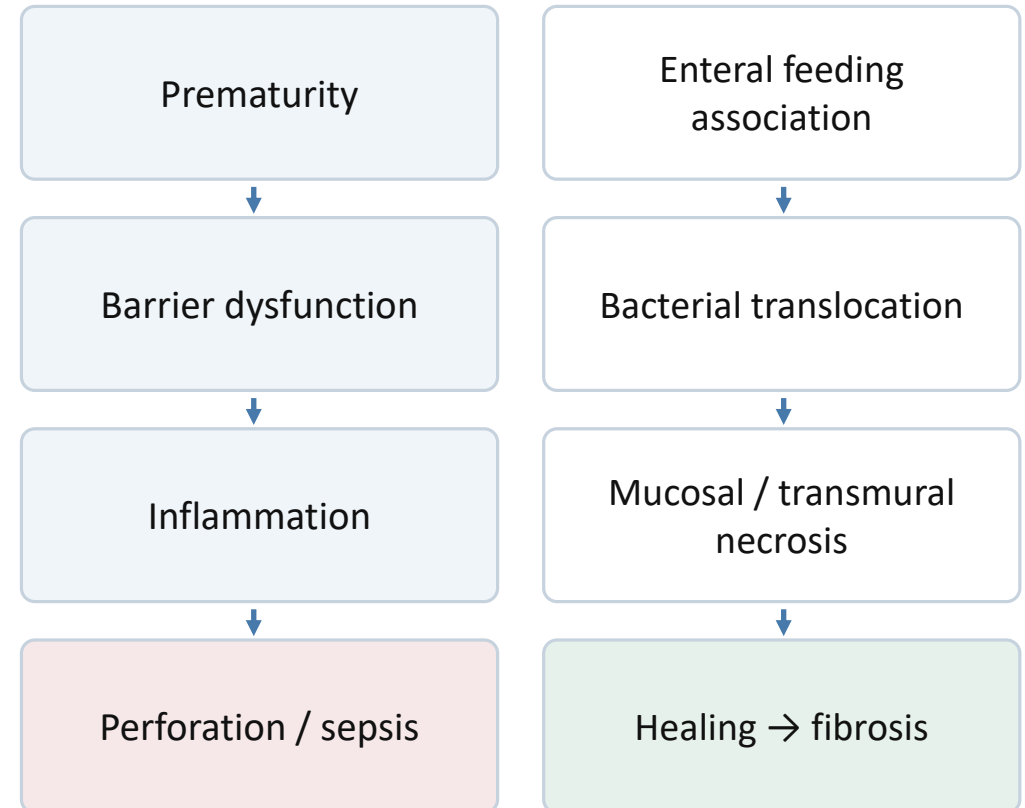
- Mucosal barrier failure permits microbial passage.
- Local inflammation and necrosis may progress to a systemic crisis.
- Circulatory collapse is a cardinal late clinical manifestation.



# An integrated proposed mechanism

This pathway is a model, not a proven single linear cause.

- Prematurity predisposes the infant to NEC.
- Feeding-associated postnatal exposures are implicated.
- Inflammatory barrier breakdown amplifies microbial entry.
- Necrosis may progress to sepsis and shock.

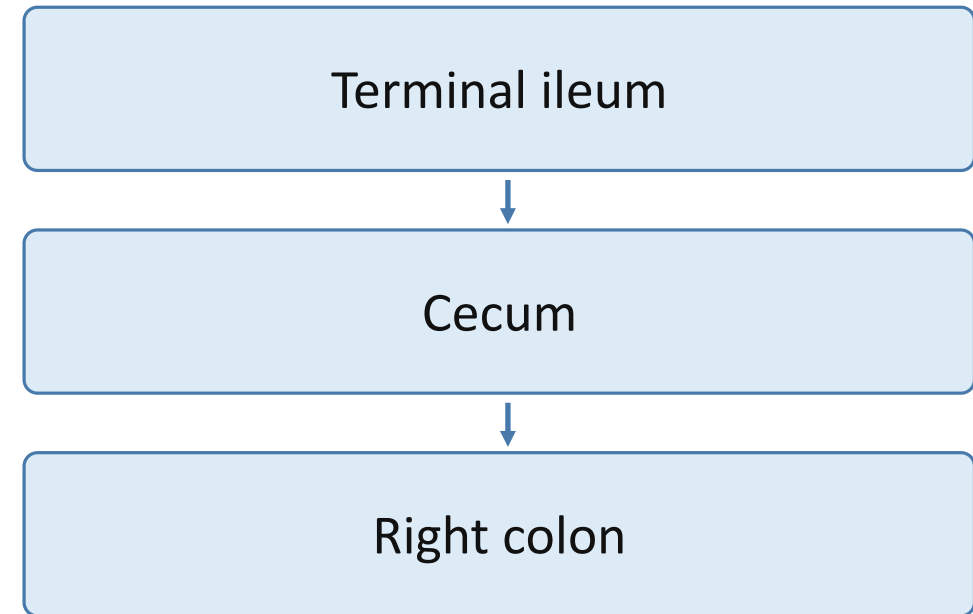


# Anatomic distribution has a characteristic emphasis

NEC most often involves the ileocecal region.

- Terminal ileum, cecum, and right colon are typical sites.
- Any small- or large-intestinal segment may be involved.
- The location alone does not define disease severity.

## Regional distribution (simplified)



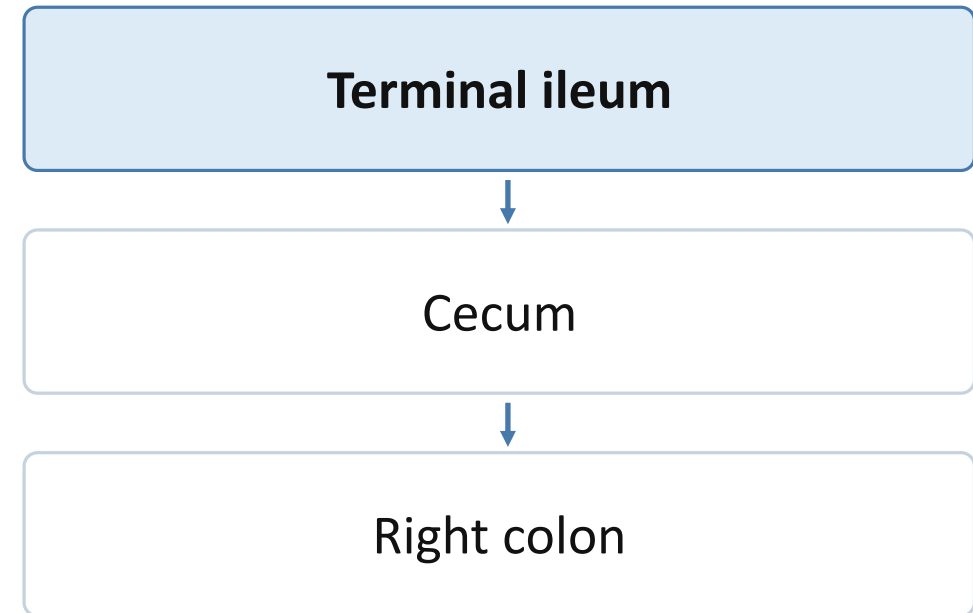
Other small- or large-bowel segments may also be involved

# Terminal ileum: A commonly involved segment

The terminal ileum is part of the typical NEC distribution.

- Grossly, involved ileum can be congested and distended.
- The source figure describes hemorrhagic infarction and transmural necrosis in ileum.
- NEC is not confined to ileal tissue.

## Regional distribution (simplified)



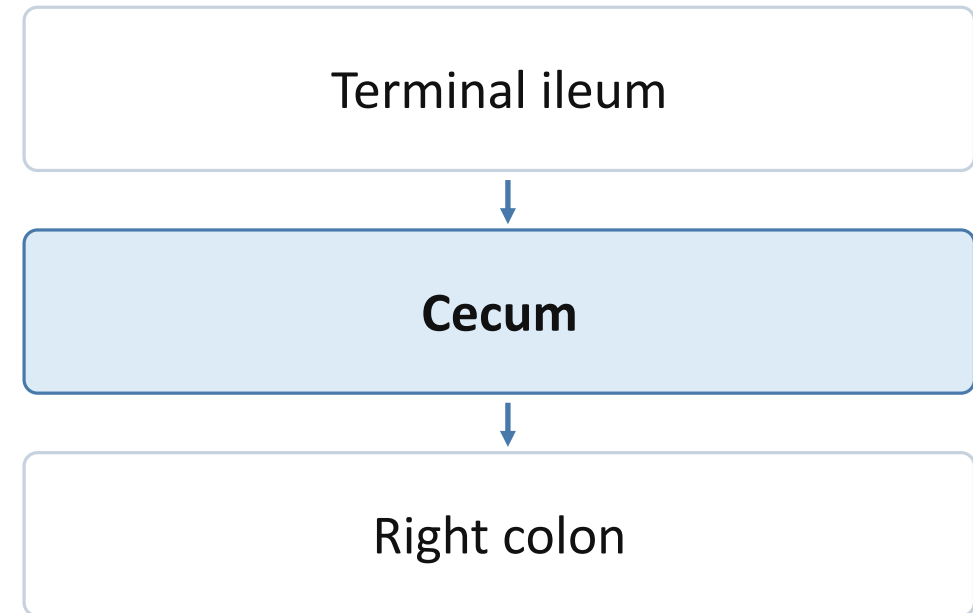
Other small- or large-bowel segments may also be involved

# Cecum and right colon also predominate

The ileocecal and proximal colonic region is frequently affected.

- Cecal involvement is specifically described.
- The right colon is another typical location.
- The rest of the bowel requires assessment because involvement can be wider.

## Regional distribution (simplified)



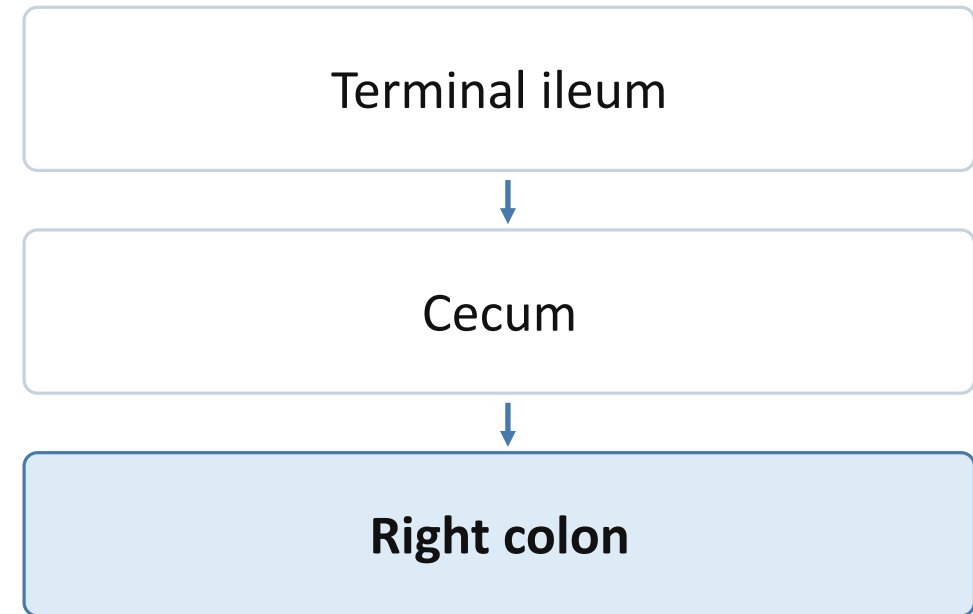
Other small- or large-bowel segments may also be involved

# The involved segment may vary in length

That any intestinal segment may be affected.

- Disease is not restricted to one fixed segment.
- Gross changes can involve the small intestine, large intestine, or both.
- Extensive disease is compatible with the documented severe-case appearance.

## Regional distribution (simplified)



Other small- or large-bowel segments may also be involved

# Gross morphology: Bowel distention

Affected bowel may appear markedly distended.

- Distention is a hallmark macroscopic finding in severe cases.
- The example shows massive small-bowel distention.
- Wall thinning may accompany extreme distention and signal perforation risk.

## Macroscopic observations

Expanded intestinal loops

Friability and congestion

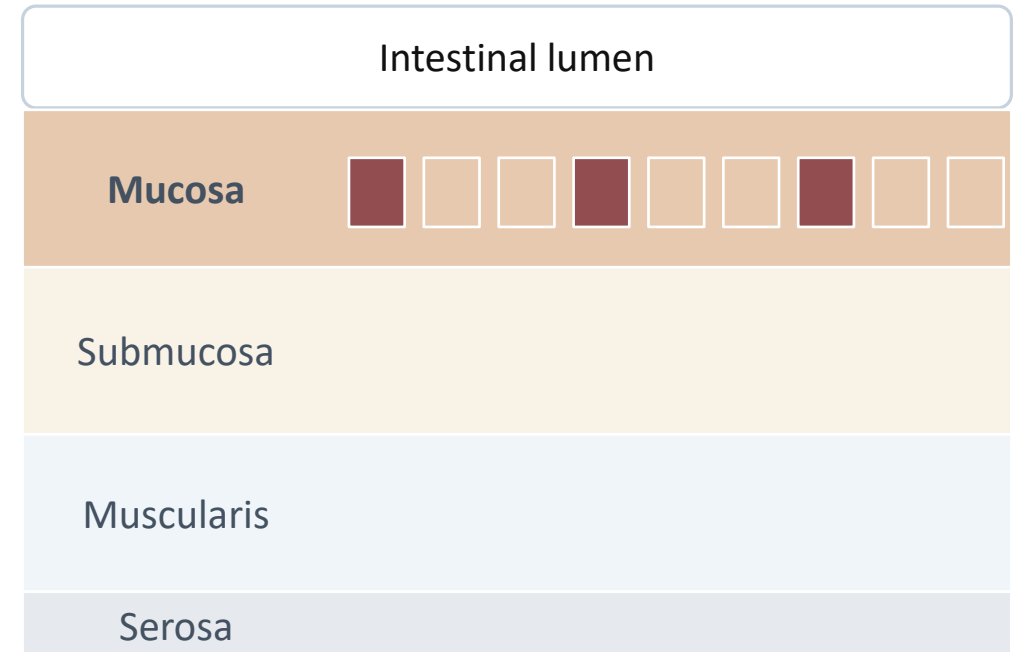
Potential wall thinning

Advanced gangrenous change

# Gross morphology: Friability and congestion

The bowel may be fragile and darkly congested.

- Friability reflects severe injury to the bowel segment.
- Congestion is a documented gross characteristic.
- The appearance may progress to frankly gangrenous tissue.



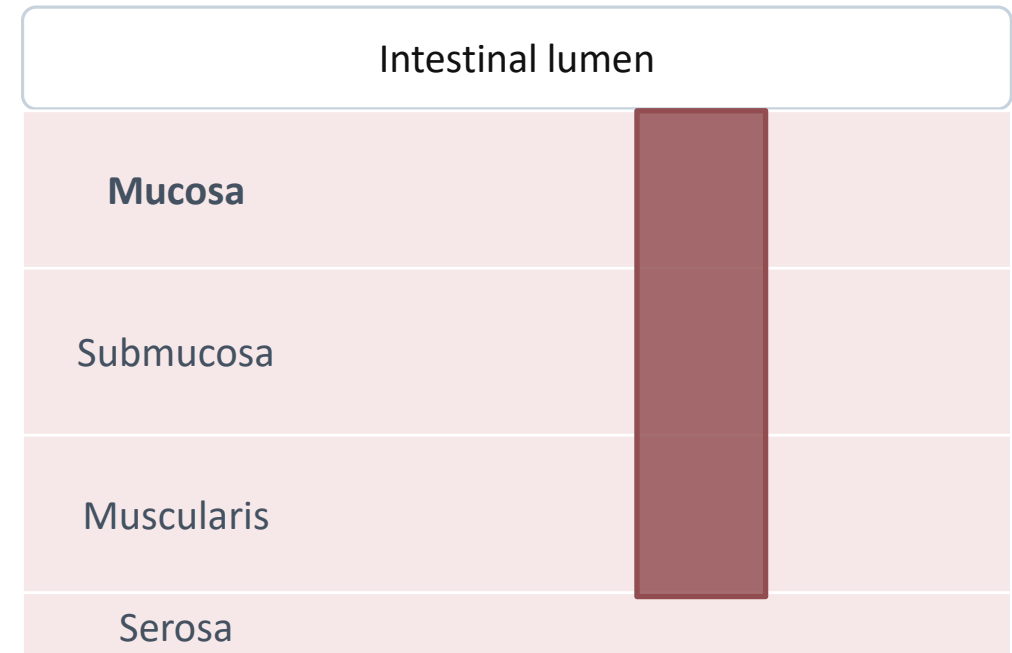
Conceptual wall section: Injury can compromise tissue integrity

*Schematic — not a histologic or clinical specimen*

# Hemorrhagic infarction can accompany necrosis

The source figure describes a congested ileal segment.

- The congested area corresponds to hemorrhagic infarction microscopically.
- Transmural necrosis is present in that severe example.
- Gross discoloration and microscopic destruction must be correlated.



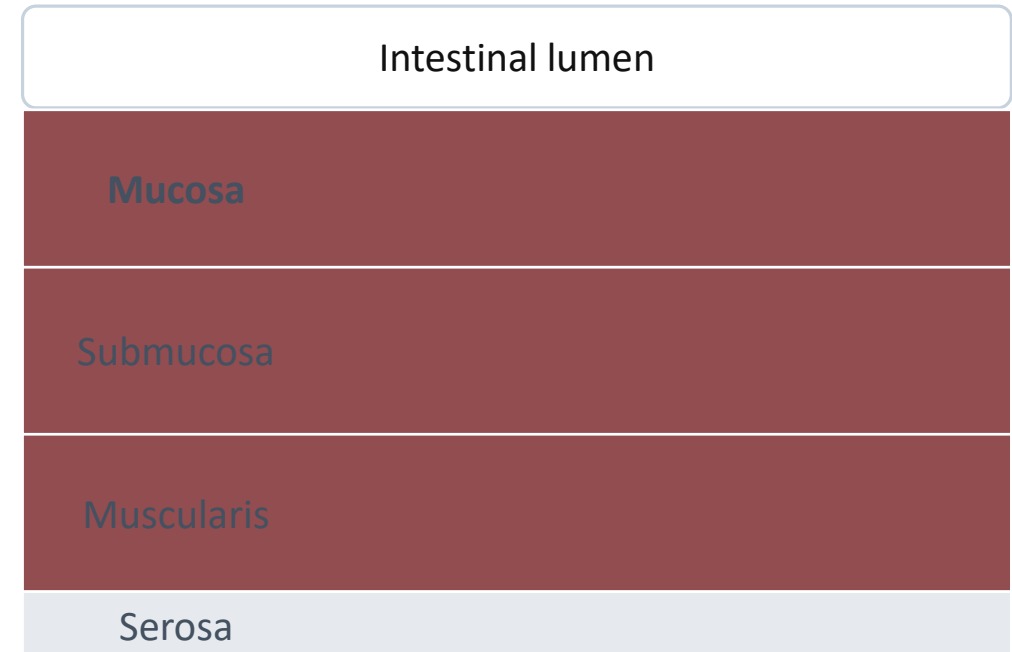
Severe transmural injury is shown as a schematic highlight

*Schematic — not a histologic or clinical specimen*

# Gangrenous change indicates advanced injury

NEC may produce frankly gangrenous intestinal tissue.

- Gangrene is part of the described gross pathological spectrum.
- Advanced damage may involve all layers of the bowel wall.
- Loss of structural integrity predisposes to perforation.



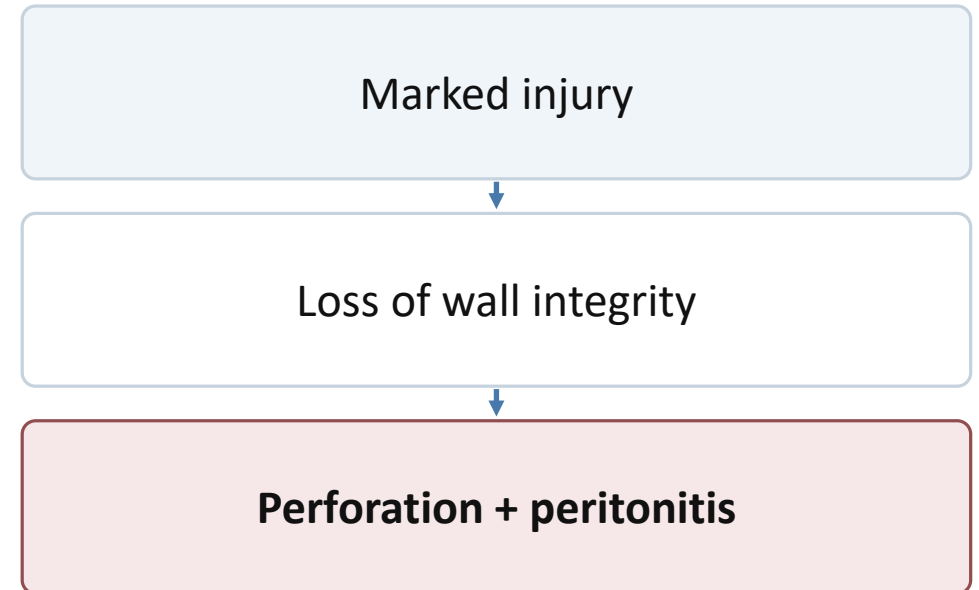
Darkened bands mark affected wall layers schematically

*Schematic — not a histologic or clinical specimen*

# A perilously thin wall raises concern for perforation

The severe example in Fig. 10.8 shows this finding.

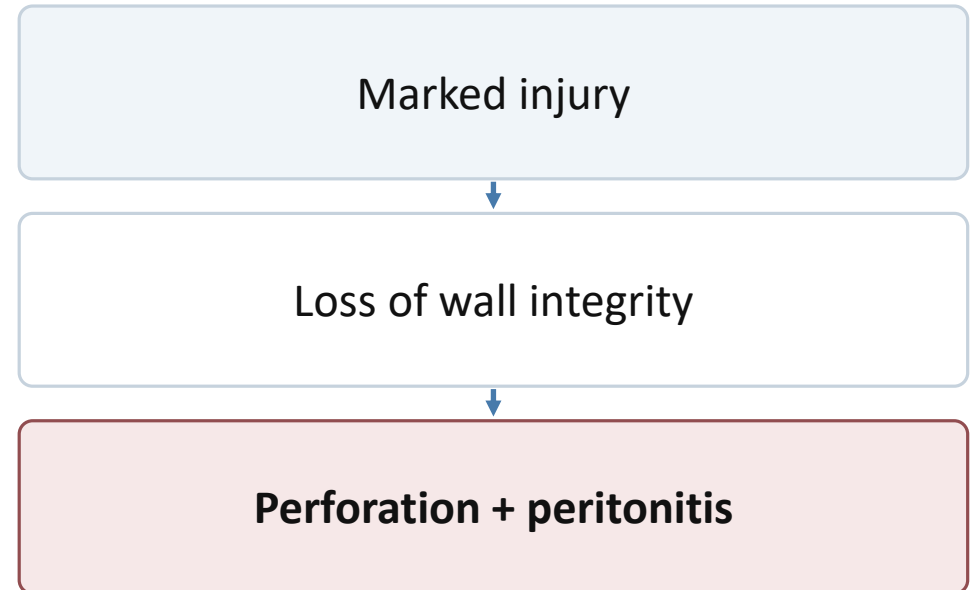
- Extreme distention is accompanied by a markedly attenuated wall.
- Robbins interprets this appearance as usually implying impending perforation.
- Thin-wall imagery should not be mistaken for a quantified diagnostic threshold.



# Perforation may cause peritonitis

Transmural necrosis can disrupt full-thickness wall integrity.

- Intestinal perforation is a recognized complication of NEC.
- Peritonitis may accompany perforation.
- The complication indicates an advanced destructive process.



# Microscopy: Mucosal coagulative necrosis

NEC can cause coagulative necrosis of intestinal mucosa.

- The mucosal layer may become necrotic without the entire wall being involved.
- The source also describes ulceration and bacterial colonization.
- Mucosal injury provides the pathological substrate for barrier failure.



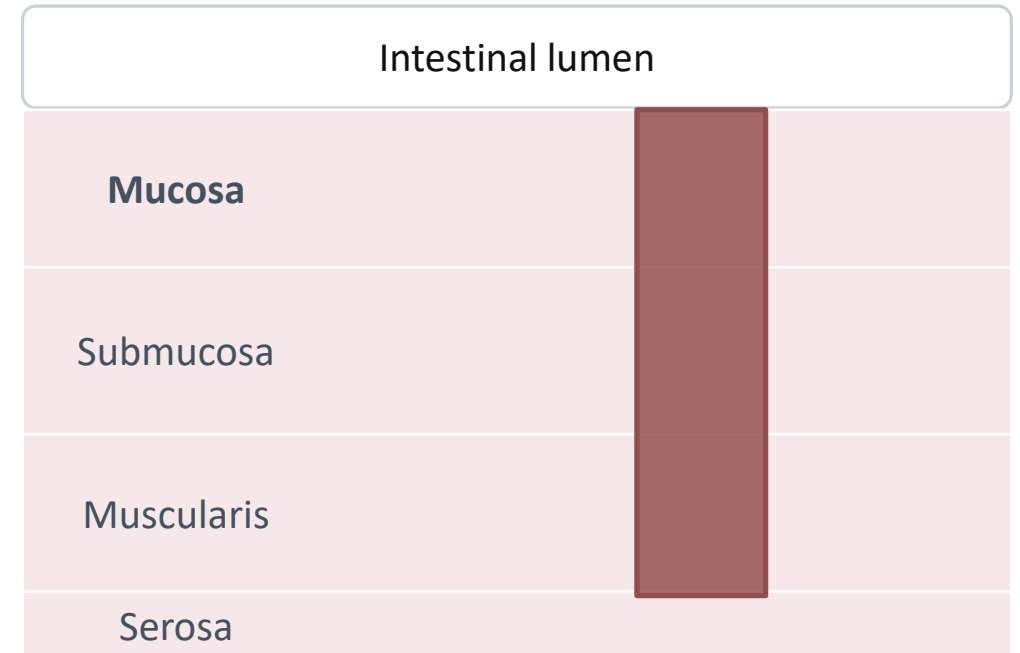
Colored mucosal layer represents injury, not actual H&E staining

*Schematic — not a histologic or clinical specimen*

# Microscopy: Transmural necrosis

Severe lesions extend through the full thickness of the bowel.

- Transmural coagulative necrosis.
- Full-wall injury explains why structural failure is possible.
- Perforation and peritonitis are major potential consequences.



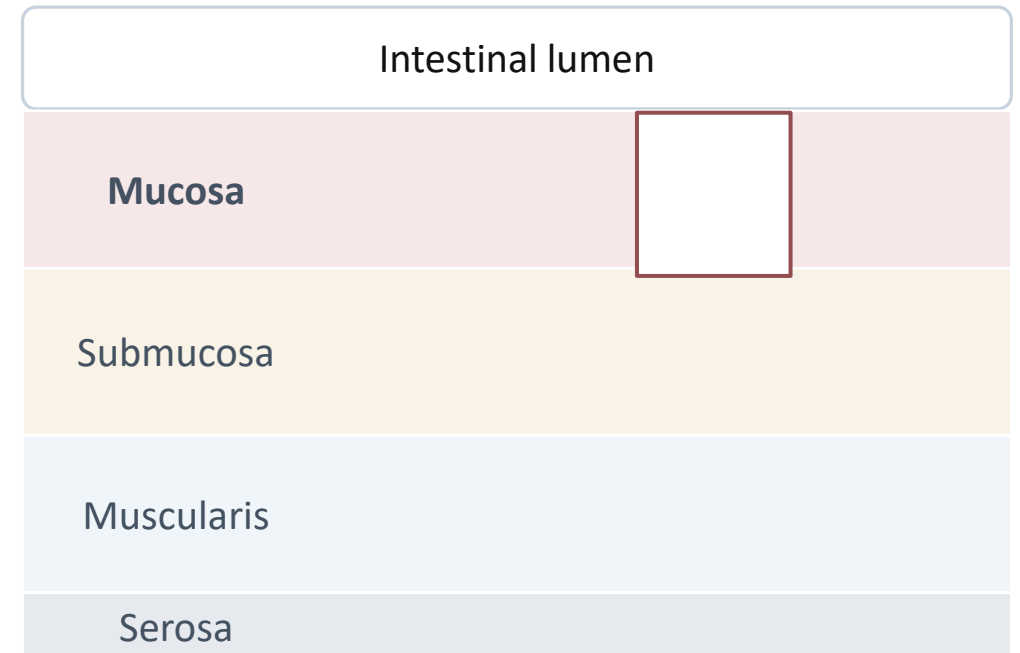
Highlighted column represents full-thickness injury

*Schematic — not a histologic or clinical specimen*

# Ulceration accompanies epithelial loss

Loss of mucosal tissue is a reported microscopic feature.

- NEC microscopy may demonstrate ulceration.
- Ulceration removes a portion of the protective lining.
- Barrier breakdown creates conditions for further bacterial access.



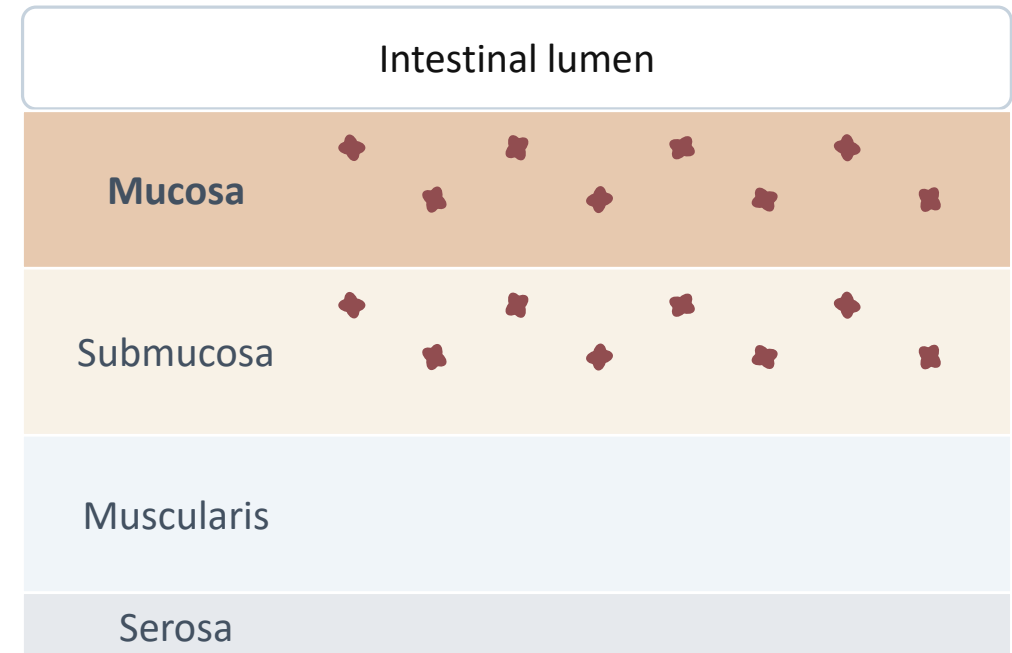
Gap represents a conceptual ulcerated mucosal surface

*Schematic — not a histologic or clinical specimen*

# Bacterial colonization appears in affected tissue

Microscopy can show bacteria in the involved bowel.

- Bacterial colonization is a recognized morphologic feature.
- Its presence supports the interaction between luminal organisms and injured mucosa.
- It does not identify a single universal NEC pathogen.



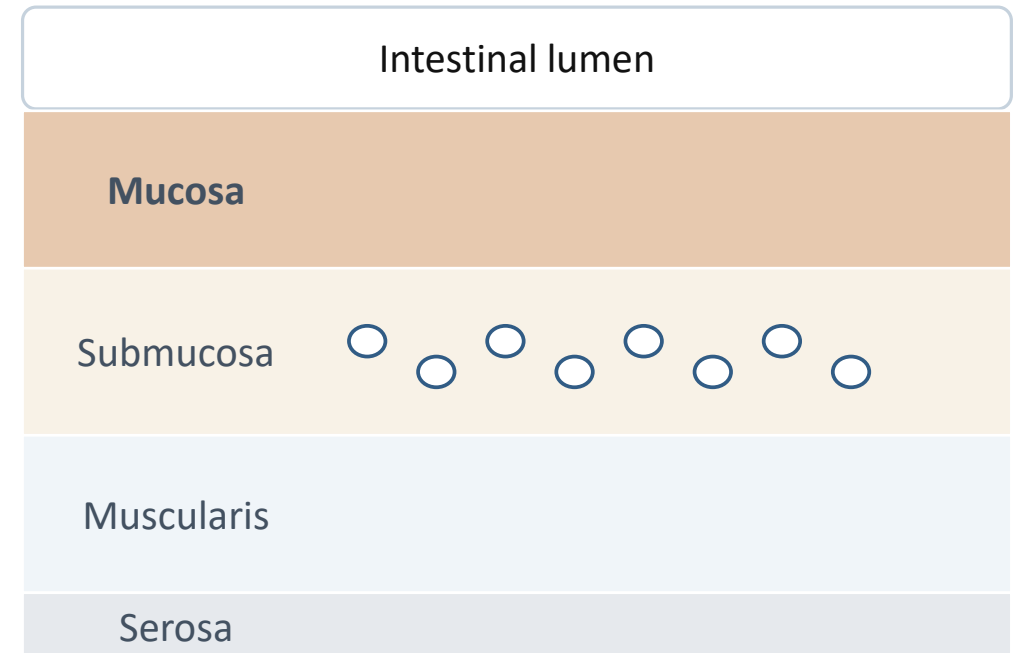
Schematic bacterial rods are not an observed tissue section

*Schematic — not a histologic or clinical specimen*

# Submucosal gas bubbles are a tissue finding

Gas may be visible beneath the mucosa.

- Submucosal gas bubbles microscopically.
- The associated radiographic term is pneumatosis intestinalis.
- The gas is within the intestinal wall rather than merely in the lumen.



Circular spaces depict the submucosal gas concept

*Schematic — not a histologic or clinical specimen*

# Mucosal versus transmural involvement

The depth of necrosis has an anatomic meaning.

- Mucosal necrosis remains in the lining.
- Transmural necrosis extends across the entire bowel wall.
- Full-thickness involvement explains the risk of wall perforation.

## Depth of injury

Mucosal: Limited to intestinal lining

Transmural: Full bowel-wall thickness

Perforation: Wall continuity is lost

Healing: Reparative tissue may create fibrosis

# Bloody stools are a characteristic presentation

The source identifies onset of bloody stools in a typical course.

- Mucosal disruption and hemorrhagic injury provide pathological context.
- Bleeding should be considered together with abdominal and systemic signs.
- No single symptom alone establishes the tissue diagnosis.

Bloody stools

Abdominal distention

**Potential circulatory collapse**

The findings occur in a potentially progressive clinical course

# Abdominal distention reflects bowel involvement

Distention is both a clinical and pathological observation.

- A distended abdomen is typical in the NEC clinical course.
- Grossly, the involved intestinal segment can be markedly dilated.
- The visible finding must be interpreted in the full clinical context.

Clinical: Distended abdomen

Gross: Distended bowel loops

**Advanced: Thin bowel wall**

The findings occur in a potentially progressive clinical course

# Circulatory collapse marks serious systemic progression

## Circulatory collapse in the clinical course.

- Severe NEC can culminate in sepsis and shock.
- The systemic deterioration follows escalating barrier and tissue injury.
- Clinically apparent collapse indicates a potentially life-threatening stage.

Intestinal necrosis

Bacterial entry / sepsis

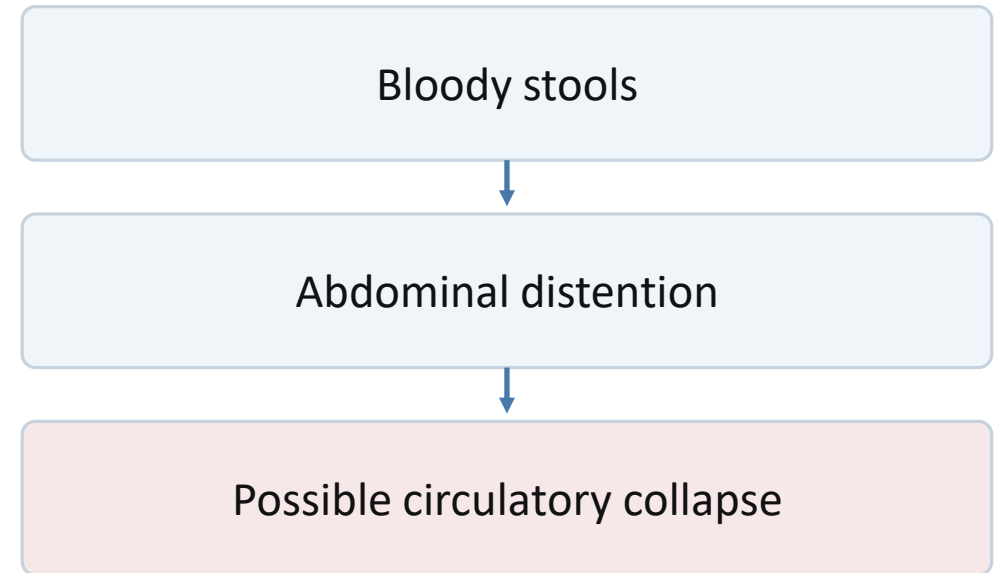
**Circulatory collapse**

The findings occur in a potentially progressive clinical course

# The typical clinical pattern is progressive

## Gut symptoms with later systemic deterioration.

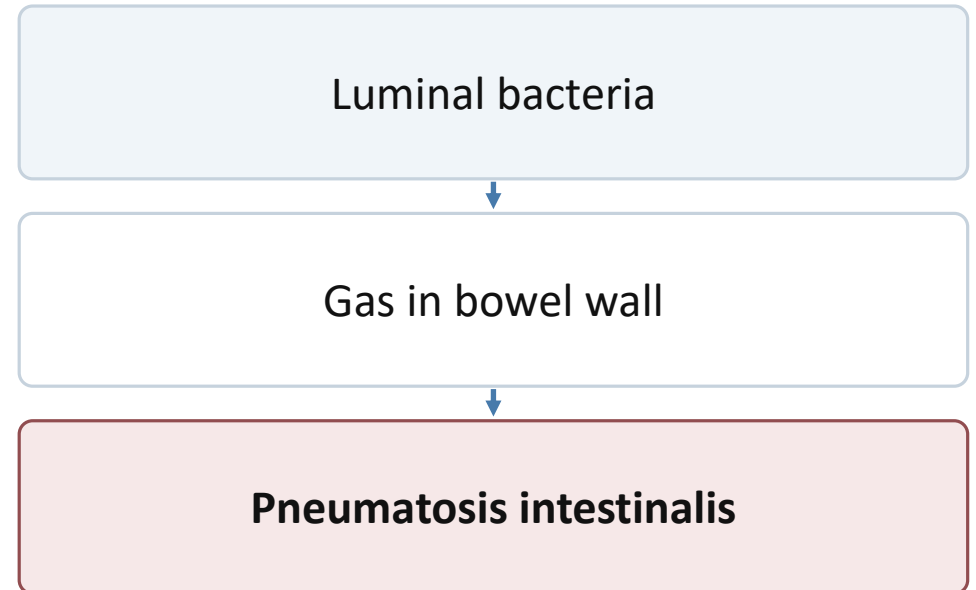
- Bloody stools indicate gastrointestinal injury.
- Abdominal distention points to affected bowel.
- Circulatory collapse signals severe systemic involvement.



# Pneumatosis intestinalis is a radiographic clue

Abdominal radiographs often show gas in the intestinal wall.

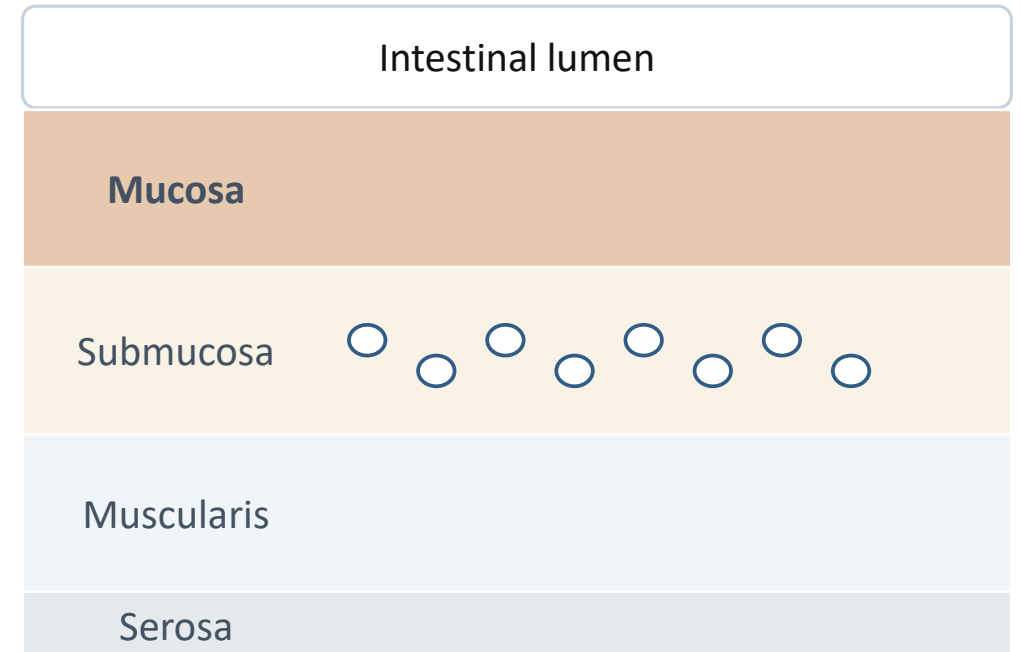
- The term pneumatosis intestinalis denotes mural gas.
- It correlates with microscopic submucosal gas bubbles.
- A teaching schematic cannot replace an authentic radiograph.



# Mural gas differs from normal bowel-lumen gas

The location of gas is the crucial distinction.

- Luminal gas lies inside the intestinal cavity.
- Pneumatosis refers to gas within wall tissue.
- Robbins localizes microscopic bubbles to the submucosa.



Submucosa (not bowel lumen) contains the illustrated bubbles

*Schematic — not a histologic or clinical specimen*

# Clinical, imaging, and pathology findings converge

A source-derived triad supports integrated interpretation.

- Clinical: Bloody stools and abdominal distention.
- Radiograph: Intramural intestinal gas may be present.
- Pathology: Mucosal or transmural necrosis and submucosal bubbles.

## Evidence across domains

Clinical: Blood in stool / distention

Radiographic: Pneumatosis intestinalis

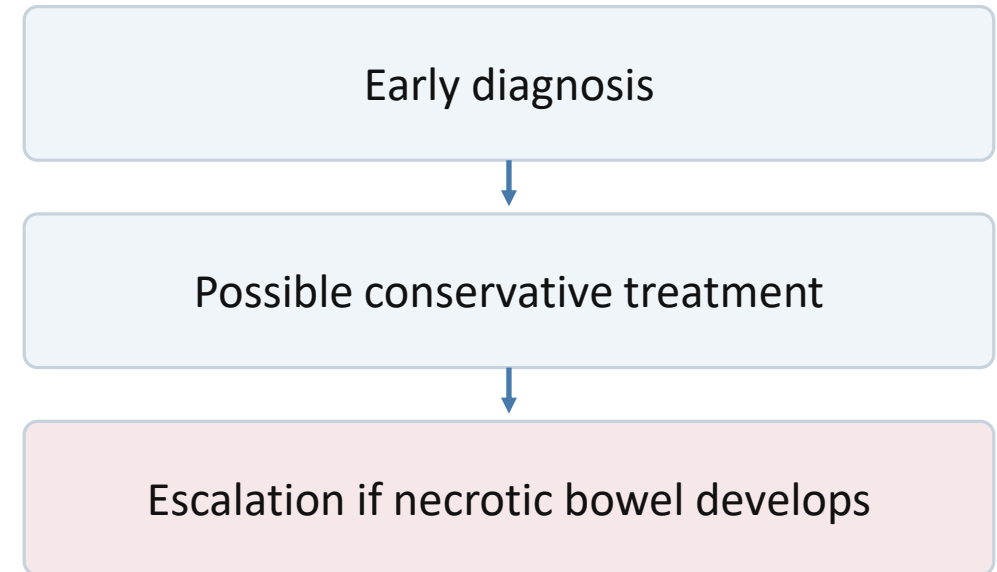
Gross: Distention, friability, congestion

Microscopic: Necrosis, ulceration, gas

# Early recognition can change the clinical course

The source allows conservative management when NEC is detected early.

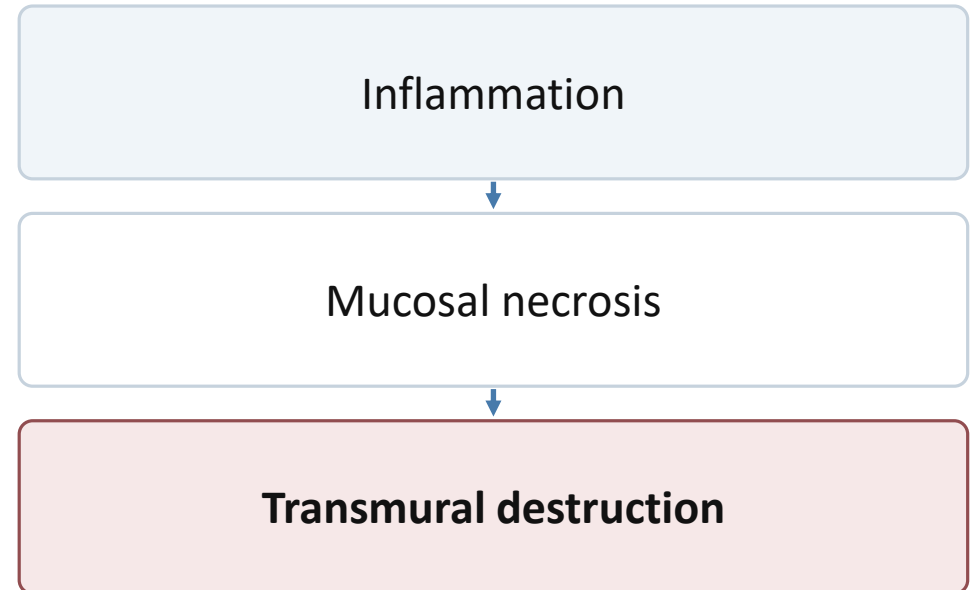
- Not every episode requires resection.
- Progressive necrosis increases the possibility of surgery.
- The excerpt does not specify a detailed management protocol.



# Conservative management may suffice early

## The timing of recognition matters.

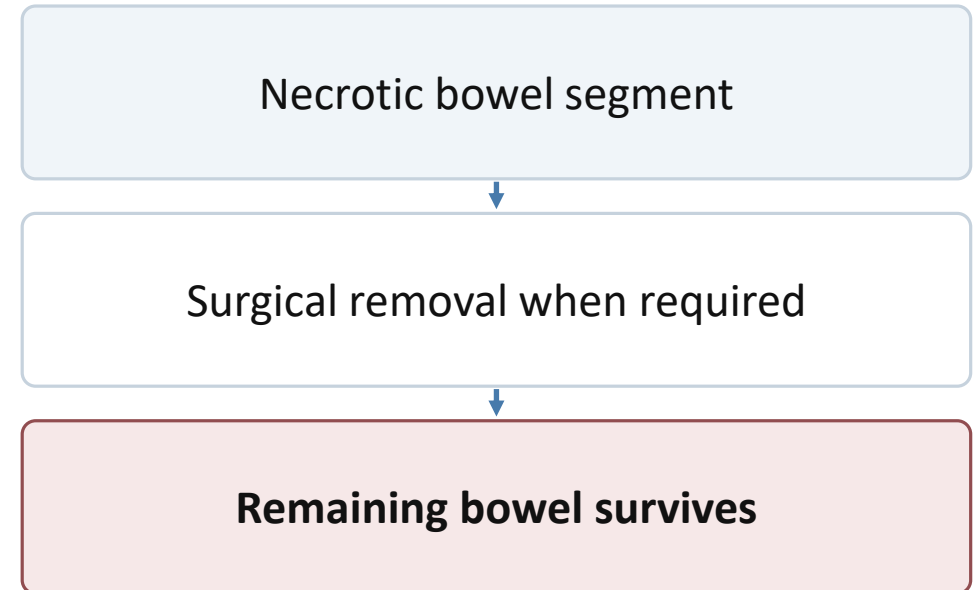
- Early NEC can often be managed conservatively.
- This is a general outcome statement, not an individualized treatment algorithm.
- Morphology and progression explain why close clinical assessment matters.



# Necrotic bowel may require surgical resection

Severe NEC sometimes necessitates removal of bowel segments.

- Advanced injury can produce nonviable intestinal tissue.
- Resection targets necrotic segments when surgery is required.
- The surgical extent depends on the involved bowel; The source gives no operative algorithm.



# The source reports wide variation in resection frequency

## Reported range in the Robbins text:

- Between 20% and 60% of cases require resection of necrotic bowel.
- The broad range should not be interpreted as a precise current institutional rate.
- The key clinicopathologic point is that substantial necrosis may demand surgery.

**20–60%**

Resection frequency reported by Robbins

Textbook range; Not a patient-specific prediction

# Severe NEC has substantial mortality

The source describes high perinatal mortality.

- Perforation, peritonitis, sepsis, and shock are serious complications.
- The excerpt does not provide a mortality percentage.
- Do not infer an exact prognostic rate without an appropriate cohort.

## Severe outcomes

Intestinal perforation

Peritonitis

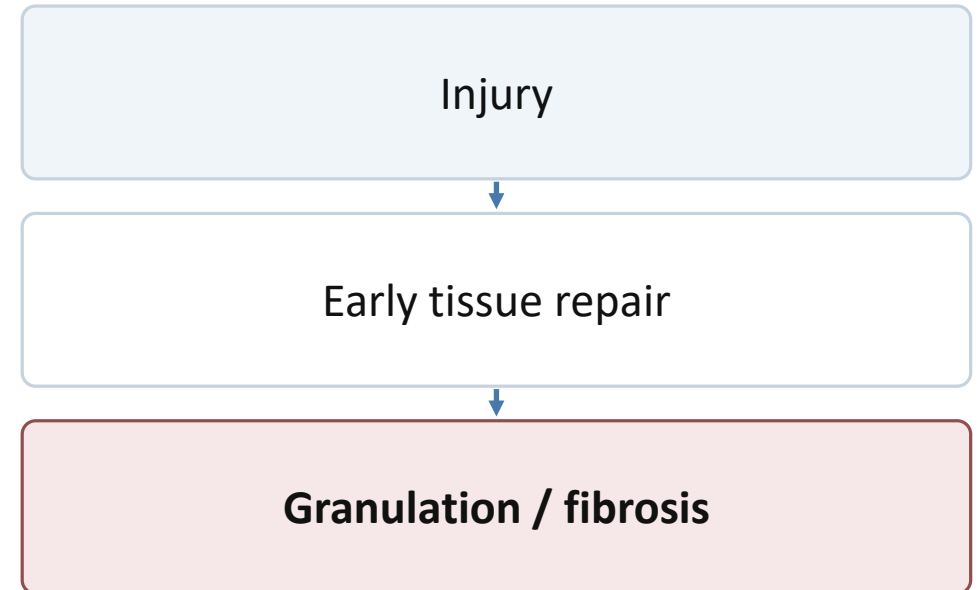
Sepsis and shock

High perinatal mortality

# Tissue repair begins soon after acute injury

Reparative changes shortly after an episode.

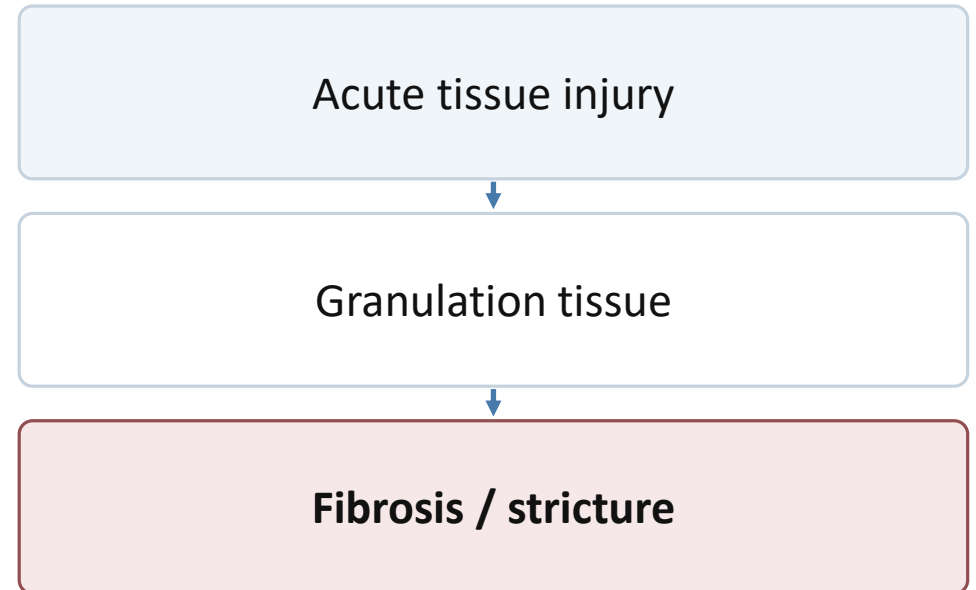
- Granulation tissue can appear in involved bowel.
- The repair process follows acute mucosal or deeper necrosis.
- Fibrosis may develop as healing proceeds.



# Fibrosis is a consequence of healing

Repair does not always restore the original wall structure.

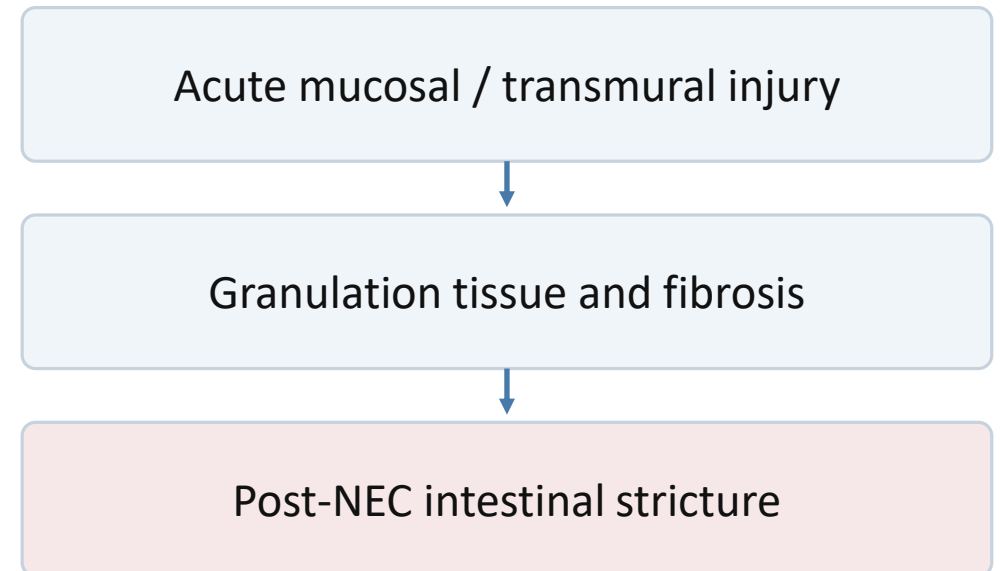
- The source identifies fibrosis among reparative changes.
- Collagenous scarring can narrow a previously damaged segment.
- This provides a pathological explanation for post-NEC strictures.



# Post-NEC stricture follows scar formation

**Fibrosis is the mechanism emphasized.**

- Surviving infants can develop intestinal strictures.
- Stricture arises from scarring during healing of the injured segment.
- The chronic consequence contrasts with acute necrosis and perforation.



# Prevention research is not settled in this source

## An area under evaluation.

- Probiotic therapies are being evaluated for NEC prevention.
- The excerpt does not establish effectiveness, product, dosage, or guideline status.
- Any clinical recommendation would require separately verified contemporary evidence.

### What the textbook actually says

Potential preventive approach: Probiotics

Status: Under evaluation in source

Efficacy / optimal regimen: Not specified

No clinical protocol claimed in this lecture

# Case exercise: A preterm infant with gut signs

Hypothetical teaching vignette (not a real patient).

- A very-low-birth-weight premature infant develops bloody stools.
- The abdomen becomes markedly distended.
- Abdominal radiography suggests gas within the intestinal wall.
- Question: Which diagnosis and tissue phenomenon best unify the findings?

Prematurity

Bloody stools + distention

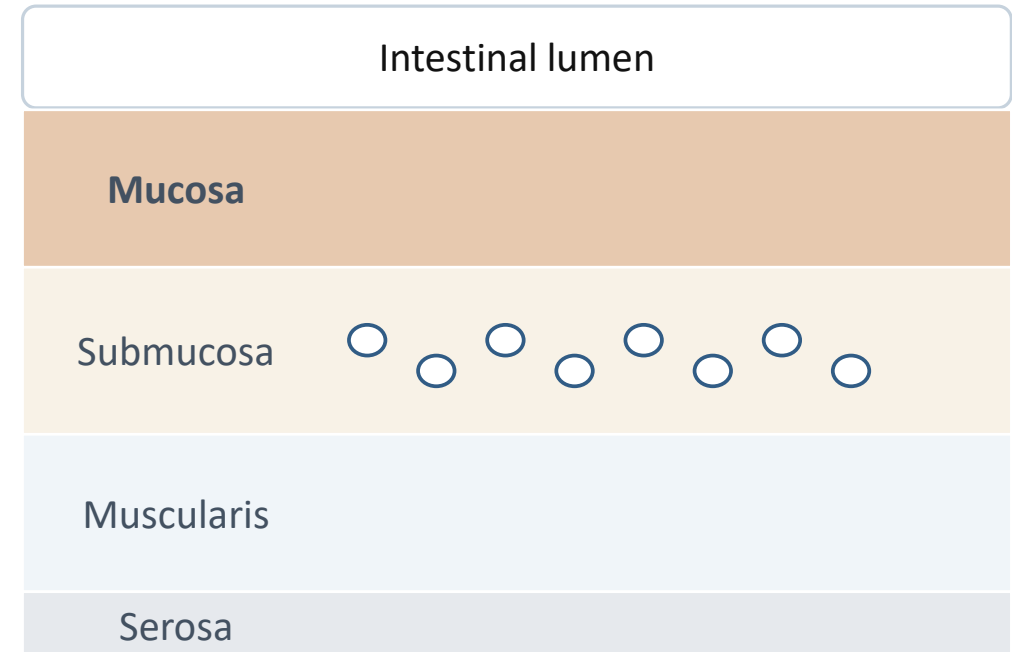
**Intramural intestinal gas**

The findings occur in a potentially progressive clinical course

# Case analysis: The findings support NEC

The vignette combines the textbook's defining clues.

- Prematurity provides the susceptibility context.
- Bloody stools and distention match the typical presentation.
- Mural gas corresponds to pneumatosis intestinalis.
- The proposed pathology includes barrier injury and intestinal necrosis.



Clinical mural gas ↔ submucosal gas spaces

*Schematic — not a histologic or clinical specimen*

# Case exercise: A severe necrotic segment

## Hypothetical teaching vignette (not a real patient).

- An infant with NEC deteriorates with circulatory collapse.
- At examination, bowel appears congested and gangrenous.
- A markedly thin segment raises concern for wall perforation.
- Question: What complications and histologic depth are most concerning?



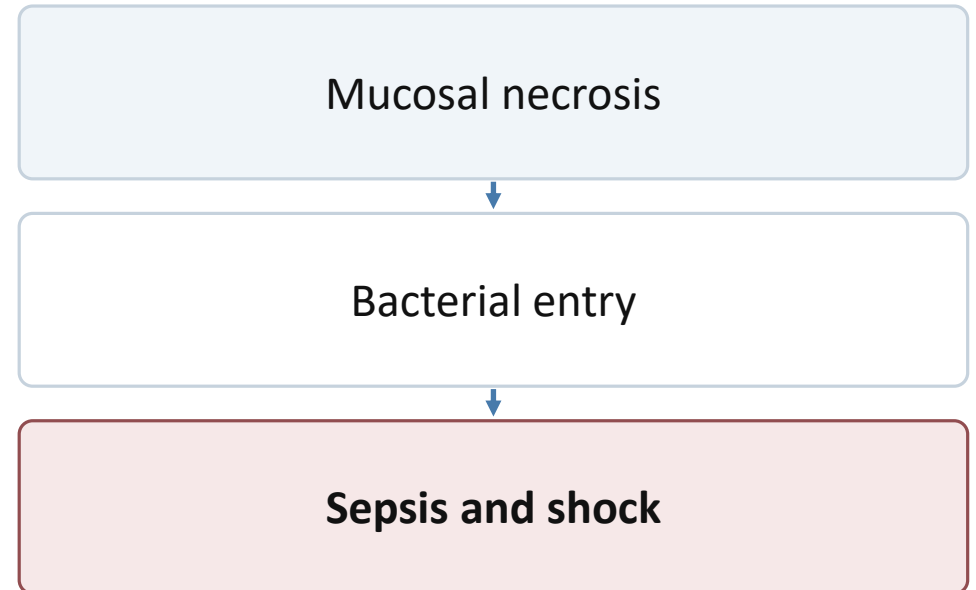
Severe tissue injury and loss of structural integrity

*Schematic — not a histologic or clinical specimen*

# Case analysis: Full-wall damage and systemic crisis

The findings describe the severe end of the NEC spectrum.

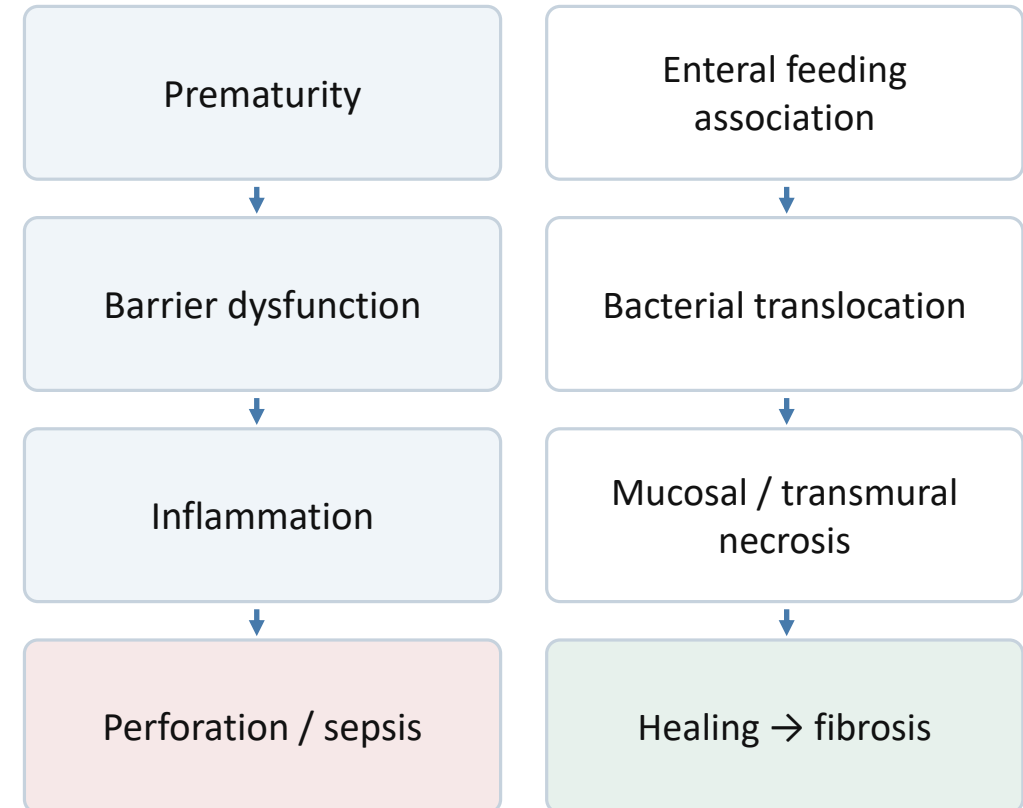
- Transmural coagulative necrosis may underlie gangrene.
- Perforation may produce accompanying peritonitis.
- Microbial translocation can culminate in sepsis and shock.
- Extensive necrotic tissue may require resection.



# Morphology links mechanisms to manifestations

All major pathologic observations fit a causal narrative.

- Barrier failure and bacterial entry amplify inflammation.
- Mucosal or transmural necrosis produces destructive intestinal lesions.
- Pneumatosis is mural gas; Advanced injury may perforate.
- Fibrotic healing explains later strictures.



# Five essential points about NEC

## A focused clinicopathologic synthesis.

- NEC predominantly affects premature infants.
- Pathogenesis is multifactorial; Barrier failure and bacterial translocation reinforce inflammation.
- Mucosal to transmural necrosis can produce perforation, sepsis, and shock.
- Pneumatosis intestinalis is gas within the bowel wall.
- Survivors may develop fibrotic intestinal strictures.

### The diagnostic chain

Predisposition: Prematurity

Mechanism: Barrier injury and bacteria

Morphology: Necrosis / pneumatosis

Sequelae: Perforation or stricture

# Reference and figure limitations

## Teaching source and verification limits.

- Robbins, Cotran & Kumar: Pathologic Basis of Disease, 11th edition (2026).
- Necrotizing enterocolitis section (11th edition, 2026).
- Original photomicrographs and Fig. 10.8 were not available in the provided files.
- Anatomical diagrams are editable teaching schematics, not diagnostic specimens.

### Material included

Source-derived medical statements

Original editable PowerPoint schematics

Hypothetical teaching cases

No synthetic microscopy or invented radiographs