

Pulmonary Infections

Host defenses · pneumonia syndromes · bacterial community-acquired infection

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Learning objectives

- Explain innate and adaptive antimicrobial defenses in the respiratory tract.
- Relate host defects to characteristic classes of lung pathogens.
- Classify pneumonia by the clinical setting and recognize the principal community-acquired bacterial pathogens.
- Connect alveolar exudation, consolidation, diagnosis and complications.

Pneumonia is an infection of lung parenchyma

Pulmonary infection becomes pneumonia when infection involves the lung parenchyma.

- Upper-airway infections and parenchymal infections are not interchangeable diagnostic labels.

Pulmonary infections impose major clinical burden

- Respiratory infections are exceptionally common; Most are viral infections of the upper respiratory tract.
- Bacterial, viral, mycoplasmal and fungal pneumonias cause substantial morbidity.
- The source describes substantial mortality; Its numerical U.S. Estimate is not used as a current surveillance statistic.

Infection site determines the tissue response

Upper respiratory tract

Predominantly mucosal disease

Examples: Common cold; Pharyngitis

Lung parenchyma

Alveolar/interstitial involvement

Pneumonia with impaired gas-exchange surfaces

Multiple microbial classes cause pneumonia

Bacteria

Typical pyogenic infections; Also intracellular bacteria

Viruses

Respiratory epithelial infection; Possible bacterial superinfection

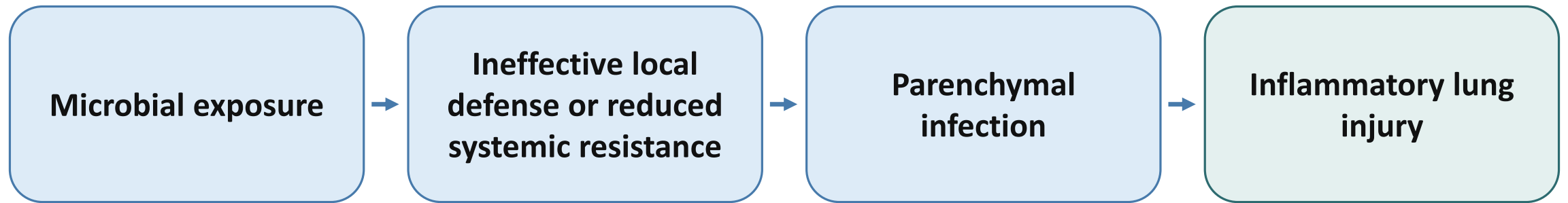
Mycoplasmas

Community-acquired pneumonia in specific epidemiologic settings

Fungi

Particularly important when immune control is impaired

Pneumonia develops when defenses are overcome



Mechanistic sequence · schematic

The respiratory tract relies on layered defenses

Mechanical

Cough; Mucus; Coordinated ciliary movement

Phagocytic

Resident macrophages; Recruited neutrophils

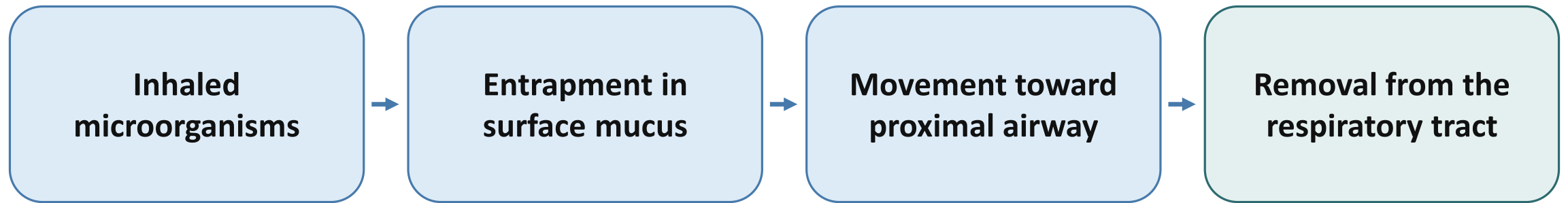
Humoral

Complement; IgA, IgG and IgM

Cellular

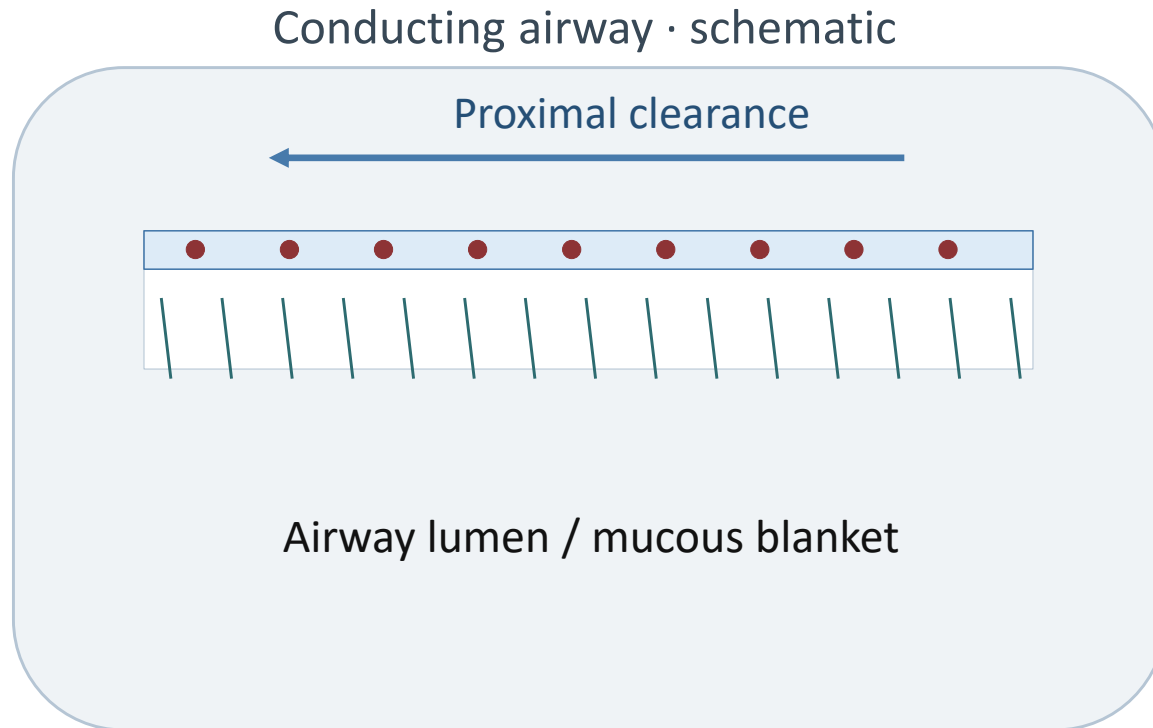
T-cell responses against intracellular pathogens

Mucus traps organisms before distal migration



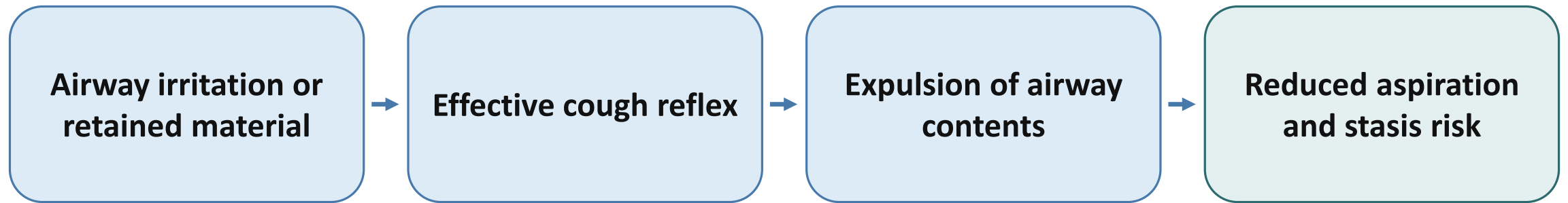
Mechanistic sequence · schematic

The mucociliary elevator clears trapped microbes



- Coordinated ciliary motion moves the mucus layer toward the upper airway.
- Successful clearance prevents retention and distal inoculation of organisms.

Cough is a protective expulsion reflex



Mechanistic sequence · schematic

Suppressed cough increases aspiration risk

Typical protective state

Intact sensorium and cough

Material more readily expelled

Vulnerable state

Coma, anesthesia, drugs, neuromuscular disease or painful respiration

Gastric/oropharyngeal material may enter lower airways

Cilia can be impaired by local exposures

Smoke

Damages mucociliary clearance

Irritant gases

Hot or corrosive inhalants affect surface defense

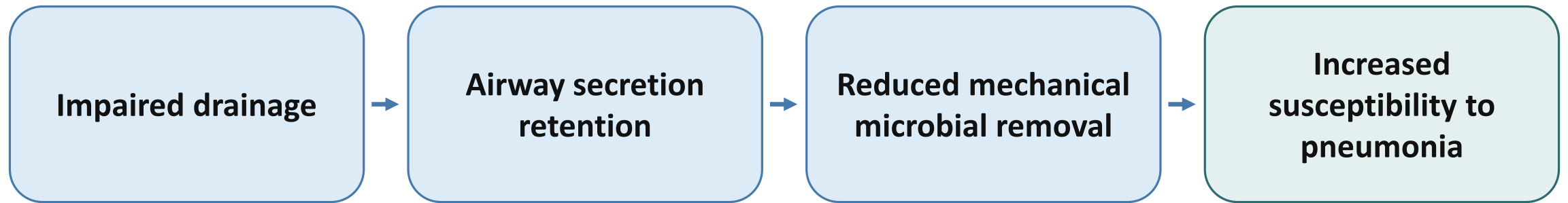
Respiratory viruses

Disrupt airway epithelial clearance

Ciliary disorders

Inherited dysfunction impairs transport

Retained secretions become a reservoir



Mechanistic sequence · schematic

Cystic fibrosis and obstruction impair clearance

Cystic fibrosis

Secretions accumulate because airway clearance is compromised.

Predisposition to recurrent infection

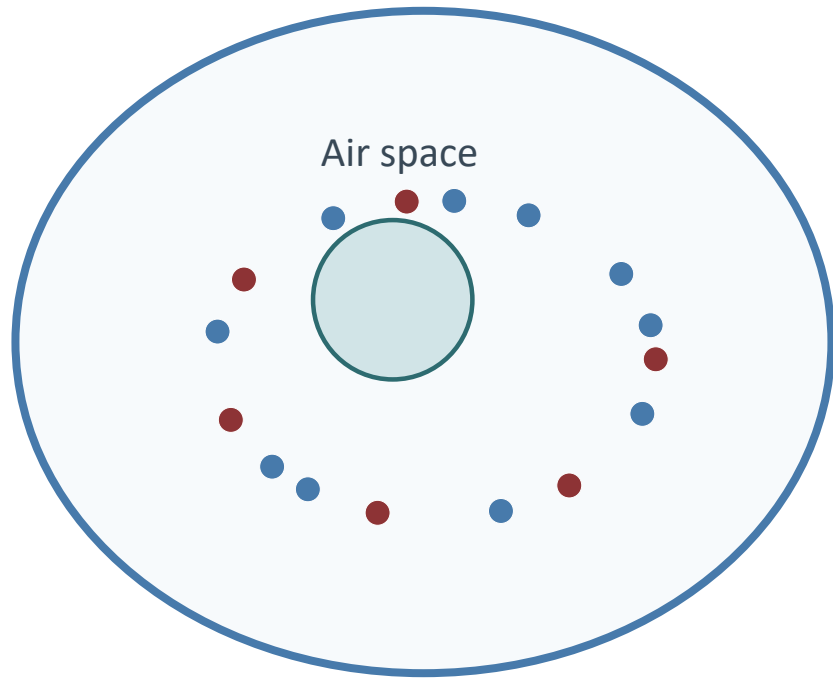
Bronchial obstruction

Distal secretions cannot drain effectively.

Localized infectious susceptibility

Alveolar macrophages defend the air spaces

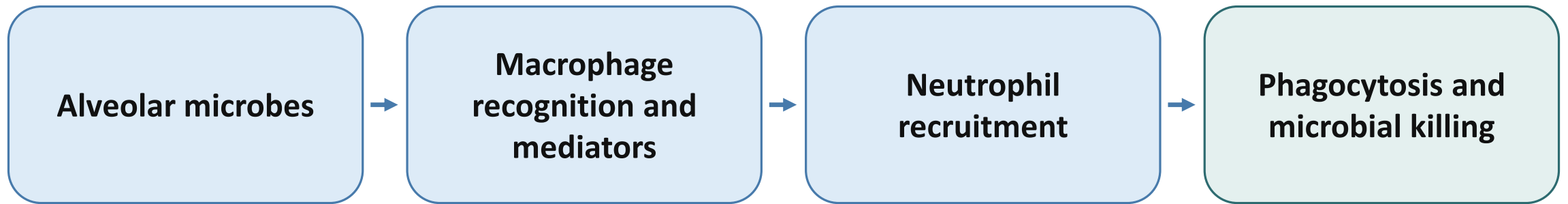
Alveolar air-space schematic



Macrophage / inflammatory cells (stylized)

- Resident alveolar macrophages ingest and degrade arriving microbes.
- They also recruit additional inflammatory effector cells.

Macrophage recruitment amplifies innate defense



Mechanistic sequence · schematic

Macrophage function is susceptible to injury

Alcohol

Interferes with macrophage antimicrobial function

Tobacco smoke

Impedes phagocytic and bactericidal activity

Anoxia

Compromises macrophage effectiveness

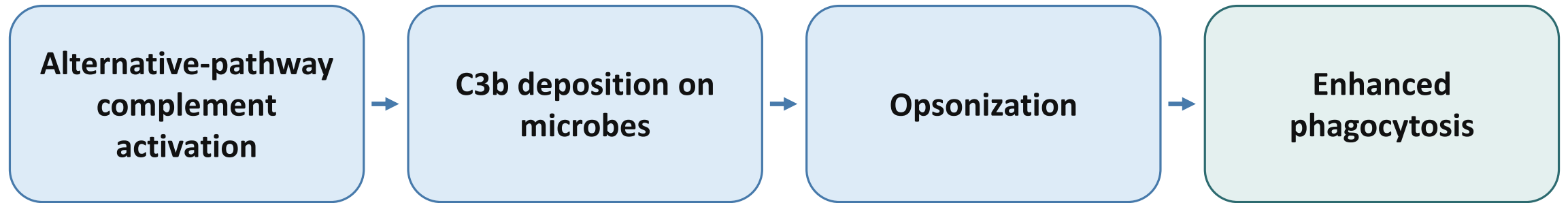
Excess oxygen exposure

Can interfere with macrophage defense

Neutrophils are essential against pyogenic bacteria

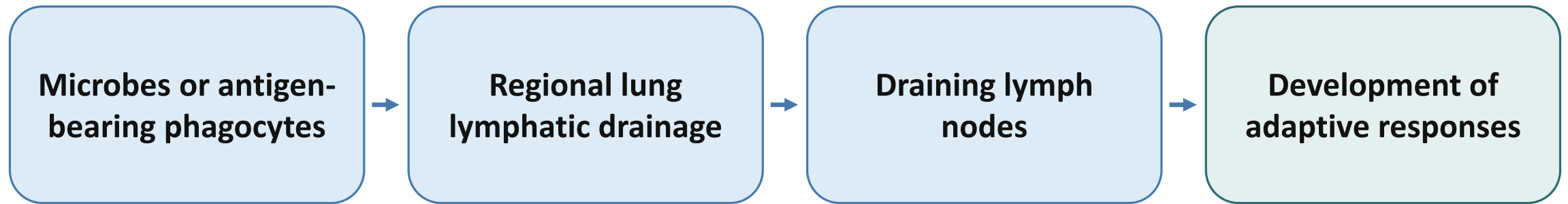
- Recruited neutrophils phagocytose and kill organisms.
- Neutrophil dysfunction or leukopenia reduces antibacterial resistance.
- A neutrophil-rich exudate is a typical tissue response to pyogenic bacterial pneumonia.

Complement makes organisms easier to ingest



Mechanistic sequence · schematic

Lymphatic drainage links innate and adaptive defense



Mechanistic sequence · schematic

Innate defenses operate as a coordinated system

- 1 Airway entrapment and clearance
- 2 Macrophage uptake in distal alveoli
- 3 Neutrophil recruitment and killing
- 4 Complement-mediated opsonization
- 5 **Lymph node delivery for immune priming**

Adaptive immunity adds antigen-specific protection

Secretory IgA

Limits microbial attachment to upper-airway epithelium

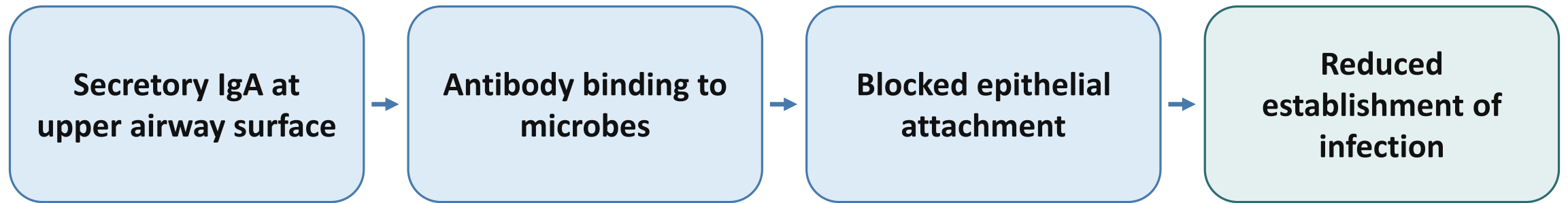
IgM / IgG

Act in lower-airway lining fluid

T cells

Control viral and other intracellular infection

Secretory IgA blocks epithelial attachment



Mechanistic sequence · schematic

Lower-airway antibody supports opsonization

- IgM and IgG are present in the alveolar lining fluid.
- Antibodies activate classical-pathway complement.
- IgG itself serves as an opsonin; The resulting C3b also enhances phagocytosis.

Humoral defenses act through two pathways

IgG

Direct opsonization of organisms

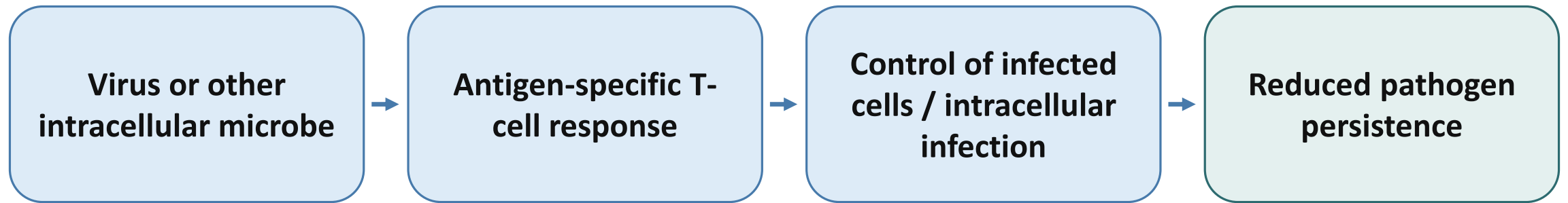
Facilitates phagocyte recognition

IgM and IgG

Activation of classical complement pathway

Generates complement opsonins including C3b

T cells control intracellular lung pathogens



Mechanistic sequence · schematic

Local defense differs by airway compartment

Location	Dominant protection	Functional goal
Conducting airways	Mucus, cilia, cough, secretory IgA	Prevent attachment and distal carriage
Distal alveoli	Macrophages, neutrophils, complement	Eliminate inhaled microbes
Infected tissue	T cells and specific antibody	Contain intracellular and extracellular infection

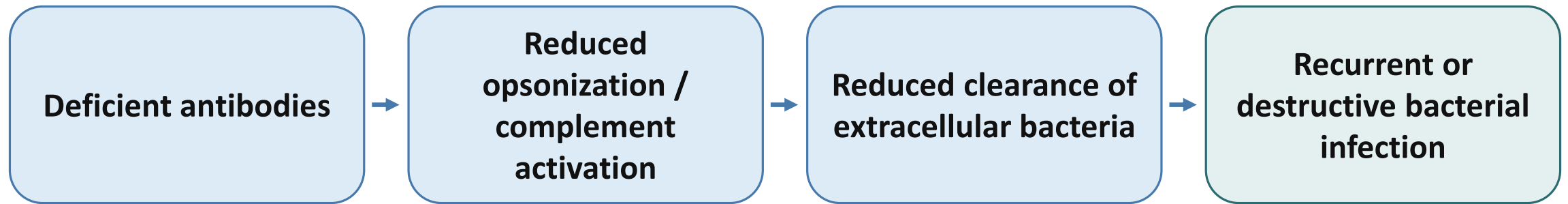
Defect type predicts pathogen susceptibility

Defective arm	Characteristic susceptibility
Neutrophils / complement	Pyogenic bacterial infection
Humoral immunity	Pyogenic bacterial infection
Cell-mediated immunity	Intracellular microbes and opportunists

Neutrophil defects favor invasive pyogenic disease

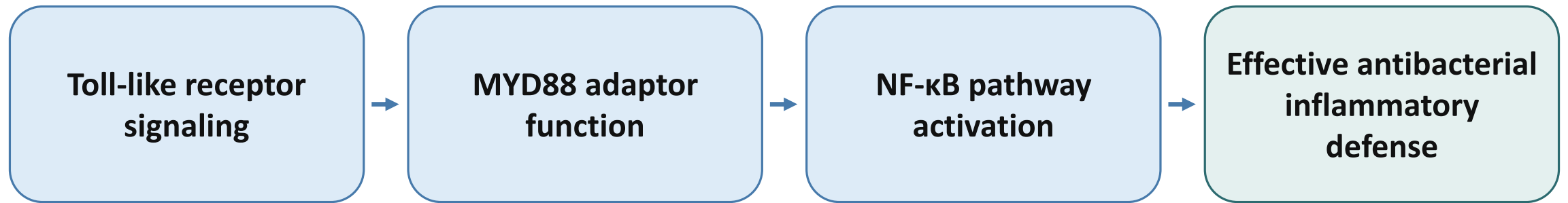
- Innate defects reduce effective early microbial clearance.
- Loss of neutrophil antibacterial activity particularly increases pyogenic infections.
- Interpret a recurrent bacterial pneumonia history in the context of the patient's immune function.

Humoral immunodeficiency favors pyogenic bacteria



Mechanistic sequence · schematic

MYD88 helps link TLR sensing to inflammation



Mechanistic sequence · schematic

MYD88 defects can predispose to severe pneumonia

- Germline MYD88 mutations with destructive bacterial pneumonia.
- Pneumococcal pneumonia is specifically highlighted.
- This illustrates that an innate signaling defect may be important even without structural airway disease.

Cell-mediated deficits shift the pathogen spectrum

Mycobacteria

Intracellular pathogens
become more consequential

Herpesviruses

Increased susceptibility
when T-cell control is
impaired

Pneumocystis jirovecii

Low-virulence opportunist
in immunodeficient hosts

Pneumocystis illustrates opportunistic susceptibility

Effective cell-mediated defense

Low-virulence organisms may be controlled

Cell-mediated immunodeficiency

P. Jirovecii may cause serious lung infection

Systemic resistance modifies pneumonia risk

Chronic disease

Overall physiologic defense compromised

Immunodeficiency

Inadequate immune effector response

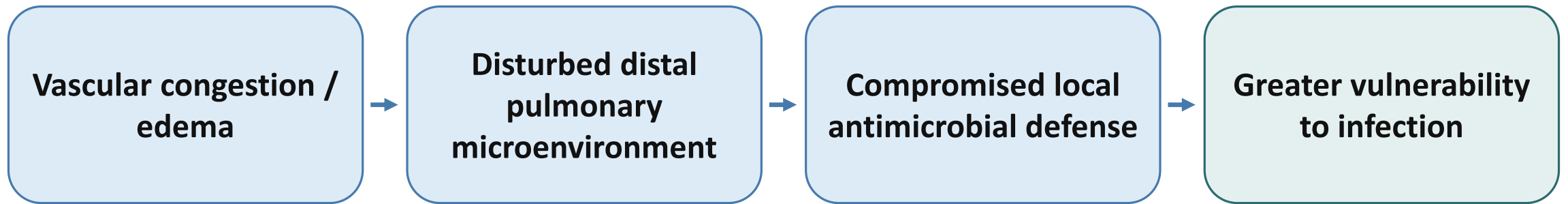
Immunosuppressive therapy

Iatrogenic reduction in pathogen control

Leukopenia

Fewer effective circulating leukocytes

Pulmonary congestion and edema reduce resistance

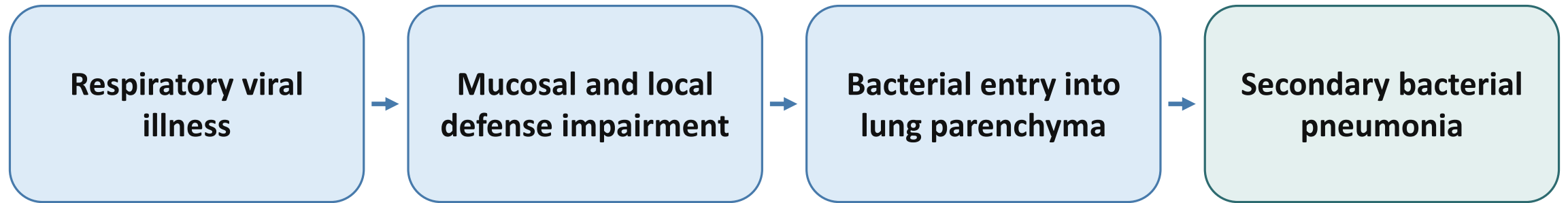


Mechanistic sequence · schematic

Tobacco smoke produces several host-defense insults

- Mucociliary dysfunction impairs mechanical removal.
- Alveolar macrophage phagocytic function may be suppressed.
- These failures may act together rather than as isolated defects.

Viral epithelial injury facilitates bacterial invasion



Mechanistic sequence · schematic

Post-influenza bacterial pneumonia can be lethal

- The source emphasizes bacterial superinfection during influenza epidemics.
- An initial viral respiratory illness can be followed by severe bacterial pulmonary infection.
- The mechanism is both epithelial injury and compromised antimicrobial defenses.

Pulmonary infection can arrive through blood

Respiratory entry

Microbes reach the lung through inhalation or aspiration.

Common route in bacterial pneumonia

Hematogenous seeding

Infection spreads to lungs from an extrapulmonary focus.

Can resemble primary pulmonary infection

The hospital environment adds distinctive risks

Antibiotic resistance

Hospital organisms may be resistant

Increased transmission

Patients and equipment facilitate spread

Invasive procedures

Intubation / interventions permit entry

Contaminated apparatus

Respiratory-care equipment may harbor microbes

Etiology and setting are complementary classifications

Etiologic approach

Classify by isolated causative organism.

Specific when adequate laboratory evidence exists

Syndromic approach

Classify by acquisition/clinical setting.

Restricts plausible pathogen spectrum

Seven clinical pneumonia syndromes are described

1 Community-acquired acute

2 Healthcare-associated (textbook category)

3 Hospital-acquired

4 Aspiration

5 Chronic

6 Necrotizing / lung abscess

7 Immunocompromised host

Community-acquired pneumonia has varied etiologies

Pyogenic bacteria

S. Pneumoniae; H. Influenzae; M. Catarrhalis;
S. Aureus

Other bacteria

Legionella; Mycoplasma; Chlamydia; Coxiella

Gram-negative pathogens

Klebsiella and Pseudomonas among listed
agents

Respiratory viruses

RSV, metapneumovirus, influenza and
adenovirus among examples

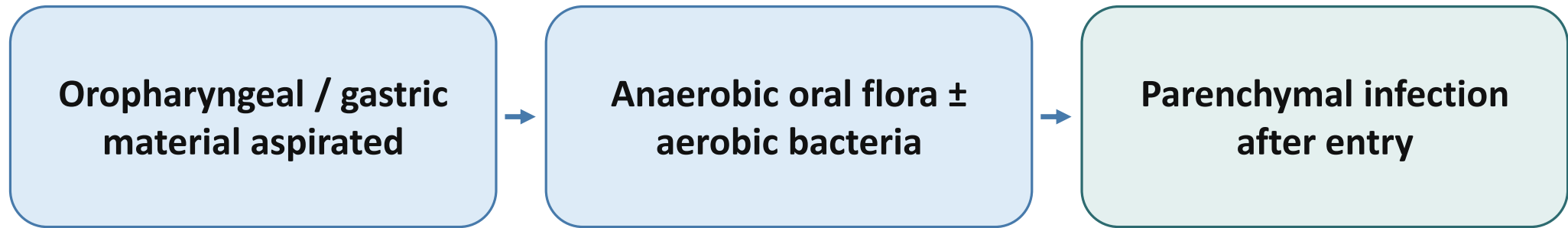
Healthcare-associated is a source-table category

- Robbins Table 15.7 separately lists healthcare-associated pneumonia.
- The organisms in that table include susceptible/resistant *S. Aureus*, *Pseudomonas* and pneumococcus.
- This slide records the textbook taxonomy; It is not a substitute for contemporary treatment guidelines.

Hospital-acquired pathogens reflect care exposure

Pathogen group	Examples from the source table
Gram-negative rods	Klebsiella, Serratia, E. Coli, Pseudomonas
Gram-positive cocci	S. Aureus, often methicillin-resistant

Aspiration pneumonia commonly involves mixed flora



Mechanistic sequence · schematic

Chronic pneumonia suggests a different differential

Nocardia / Actinomyces

Named causes of chronic pulmonary infection

Mycobacteria

Tuberculosis and nontuberculous mycobacteria

Endemic fungi

Histoplasma, Coccidioides, Blastomyces

Necrotizing pneumonia and lung abscess

- The source groups necrotizing pneumonia with lung abscess.
- Anaerobic bacteria frequently contribute, often with mixed infection.
- Listed additional causes include *S. Aureus*, *K. Pneumoniae*, *S. Pyogenes* and type 3 pneumococcus.

Immunocompromised pneumonia has a broad spectrum

Viruses

Cytomegalovirus

Fungi

Pneumocystis; Invasive aspergillosis;
Candidiasis

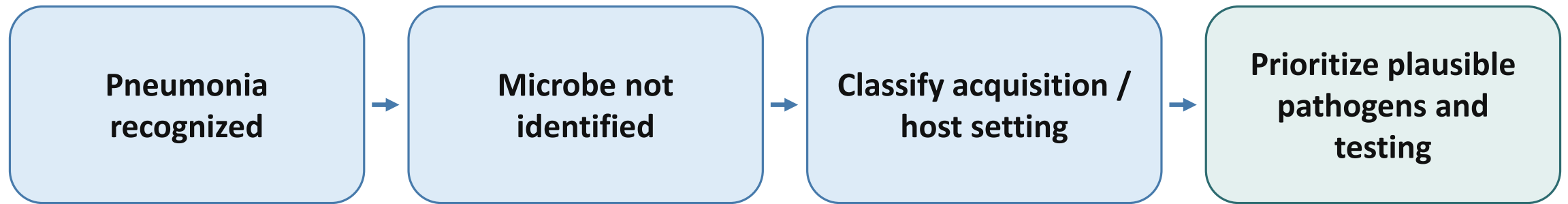
Mycobacteria

M. Avium–intracellulare complex

Conventional pathogens

Usual bacterial and viral pathogens still occur

Clinical setting helps when etiology is undetermined



Mechanistic sequence · schematic

Community-acquired acute pneumonia: Core concept

- Acquired in the community rather than during hospitalization.
- May be caused by bacteria or viruses.
- The source frames community-acquired bacterial pneumonia as infection in previously healthy individuals acquired from the usual environment.

Imaging cannot reliably determine microbial class

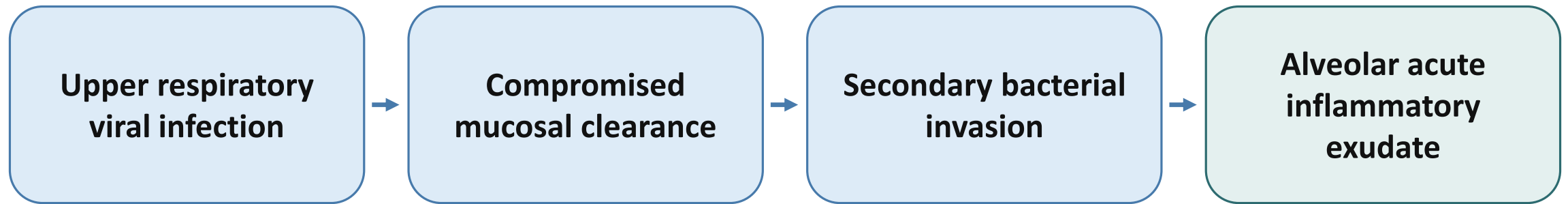
Clinical and radiologic features

Often overlap between viral and bacterial pneumonias

Microbiologic investigations

Culture or other assays may be needed for etiologic diagnosis

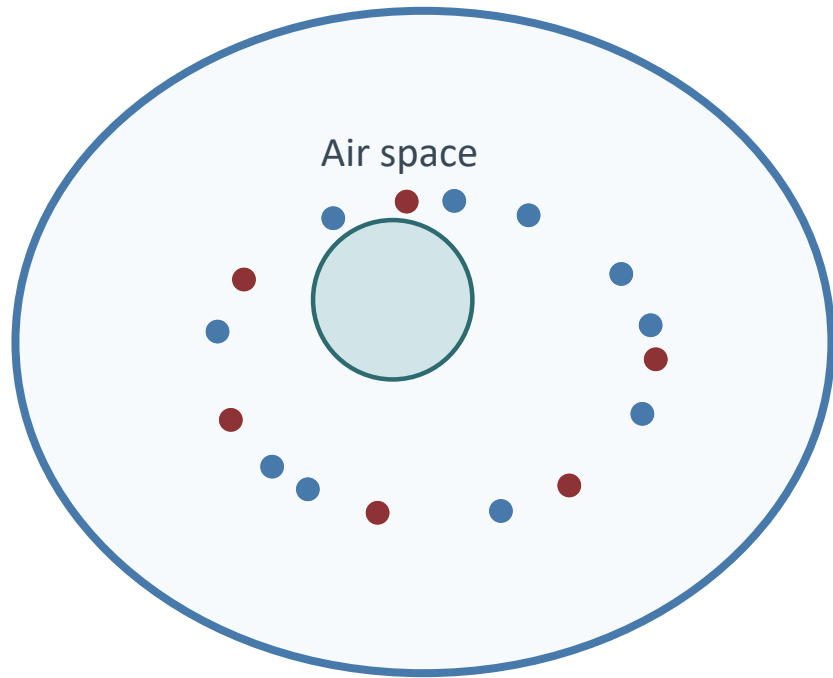
Community-acquired bacterial disease may be postviral



Mechanistic sequence · schematic

Alveolar exudate creates pulmonary consolidation

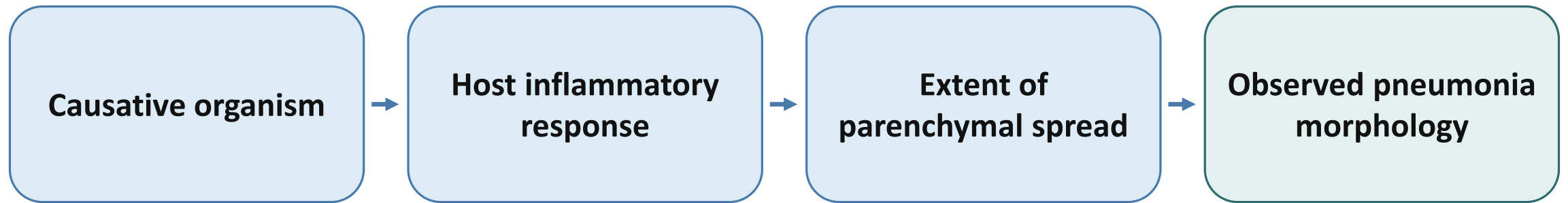
Alveolar air-space schematic



Macrophage / inflammatory cells (stylized)

- Bacterial infection causes inflammatory material to accumulate in the alveolar air spaces.
- Replacing air with exudate produces a firmer, less aerated lung: Consolidation.

Host and organism jointly determine the pattern



Mechanistic sequence · schematic

Predispositions to community-acquired bacterial infection

Age

Children and older adults

Comorbidity

CHF, COPD and diabetes

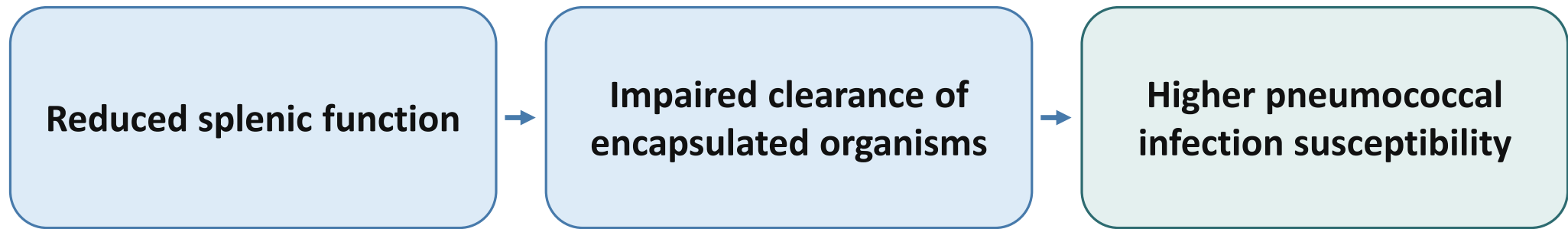
Immunity

Congenital or acquired immune defects

Spleen

Decreased or absent splenic function

Asplenia heightens the risk from encapsulated bacteria



Mechanistic sequence · schematic

Pneumococcus is a key community-acquired pathogen

- *S. Pneumoniae* is highlighted by Robbins as the leading cause of community-acquired acute bacterial pneumonia.
- This gram-positive diplococcus is also a potential colonizer of the upper respiratory tract.
- Diagnostic interpretation requires both the organism and quality/context of the specimen.

Pneumococcal sputum has a recognizable Gram stain

Suggestive morphology

Gram-positive, lancet-shaped diplococci
inside neutrophils

Diagnostic limitation

Sputum positivity must be weighed
against upper-airway carriage

Pneumococcal colonization can mislead sputum testing



Mechanistic sequence · schematic

Blood culture has different diagnostic trade-offs

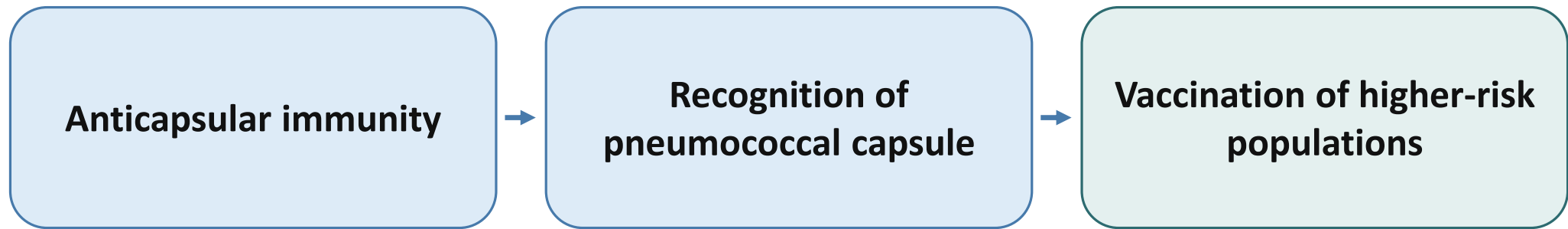
Positive pneumococcal blood culture

Higher organism specificity than expectorated sputum

Negative blood culture

Does not exclude pneumococcal pneumonia; Source notes limited early sensitivity

Capsular polysaccharide is central to prevention



Mechanistic sequence · schematic

Haemophilus influenzae has two capsular groups

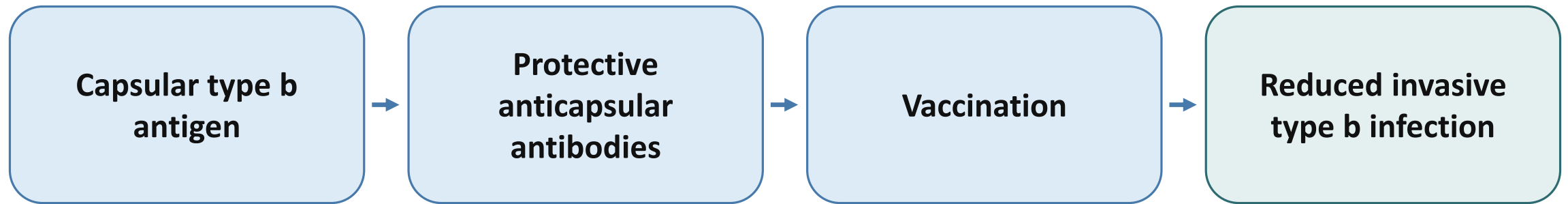
Encapsulated forms

Six serotypes (a–f); Type b emphasized as more virulent

Nonencapsulated forms

Also called nontypeable; Important in mucosal respiratory infections

Hib vaccination changed the disease spectrum



Mechanistic sequence · schematic

Nontypeable H. Influenzae causes mucosal disease

Otitis media

Middle-ear infection

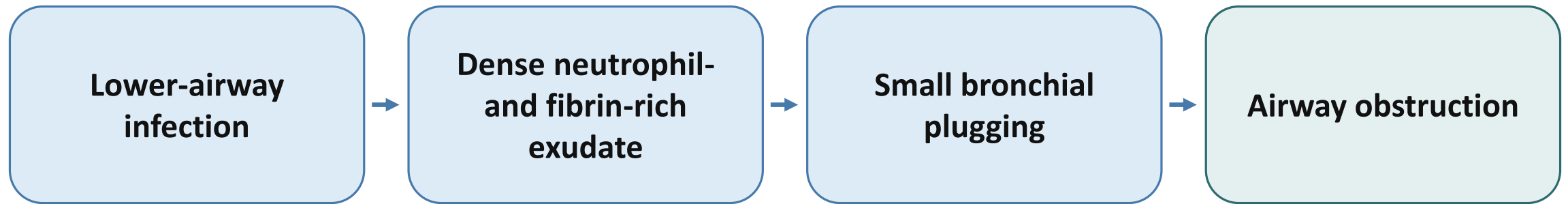
Sinusitis

Inflammation of paranasal sinuses

Bronchopneumonia

Patchy distal airway and alveolar infection

H. Influenzae can obstruct small airways



Mechanistic sequence · schematic

H. Influenzae pneumonia is consequential in children

- Neonates and children with prematurity, malignancy or immunodeficiency are vulnerable to invasive infection.
- The source describes severe pediatric pneumonia, sometimes after viral infection.
- Its morphology can be patchy or confluent rather than exclusively lobular.

H. Influenzae is important in COPD exacerbations

- H. Influenzae is identified by Robbins as an important bacterial cause of acute COPD exacerbations.
- Nonencapsulated strains often occupy the respiratory mucosal surface.
- Clinical host context should guide how an isolate is interpreted.

Moraxella catarrhalis has age-associated importance

Older adults

Recognized cause of
bacterial pneumonia

Patients with COPD

Important cause of acute
exacerbations

Children

Among frequent organisms
in otitis media

Three organisms link the lung and middle ear

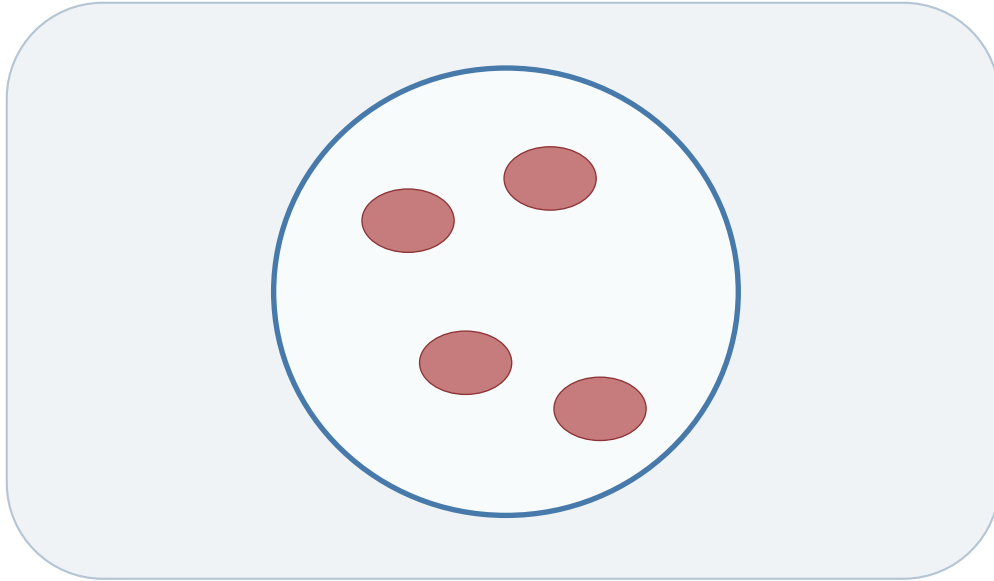
Common bacterial organisms	Clinical intersection
S. Pneumoniae	Community-acquired pneumonia; Otitis media
H. Influenzae	Bronchopneumonia; Otitis media; COPD
M. Catarrhalis	Pneumonia in older adults; Otitis media; COPD

Staphylococcus aureus often follows viral infection

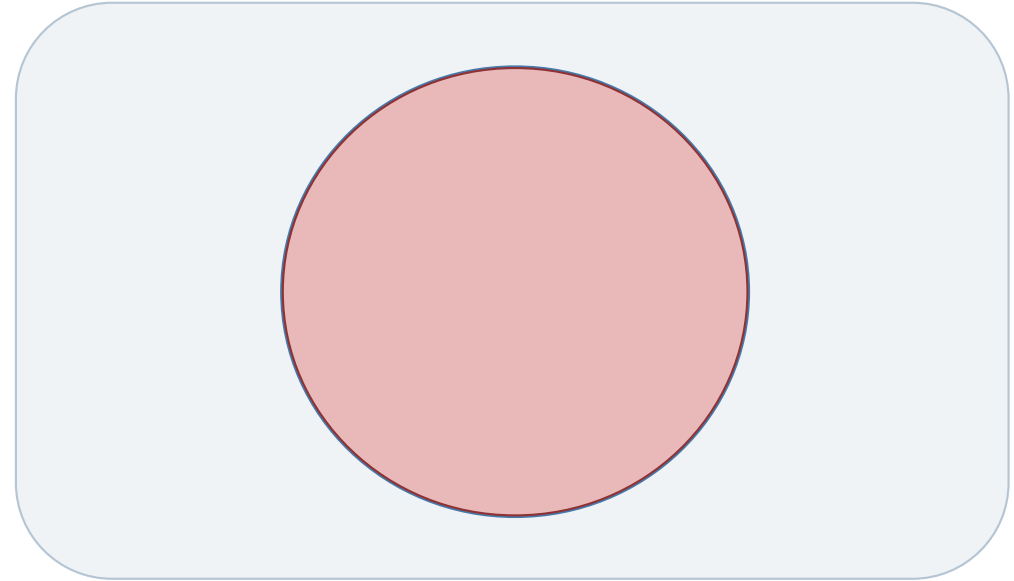
- S. Aureus may cause bacterial pneumonia after influenza or measles.
- Complications emphasized in the source include pulmonary abscess and empyema.
- It is also an important hospital-acquired organism.

Bronchopneumonia and lobar pneumonia differ by spread

Patchy bronchopneumonia



Confluent lobar pneumonia



Schematic distribution only · not actual histology

Bronchopneumonia: Patchy foci of consolidation, often centered on small airways. · Lobar pneumonia: Consolidation spans a large portion of a lobe or an entire lobe.

Bacterial pneumonia patterns can overlap

Bronchopneumonia

Patchy foci may merge into a confluent opacity

Lobar pneumonia

Same organism may cause a different distribution in another host

Lobar pneumonia has a classic four-stage sequence

- 1 Congestion: Vascular engorgement and alveolar edema
- 2 Red hepatization: RBCs, neutrophils and fibrin fill alveoli
- 3 Gray hepatization: RBC breakdown; Fibrinosuppurative exudate remains
- 4 **Resolution: Enzymatic clearing / macrophage removal**

Anatomic spread can produce serious complications

Abscess

Local necrosis and cavitory suppuration

Empyema

Fibrinosuppurative infection of pleural space

Bacteremia

Distant infection:
Endocarditis, meningitis or abscesses

Case: Recurrent pyogenic pneumonia

Patient with repeated bacterial lower-respiratory infections and deficient antibody production.

- Question: Which pulmonary immune functions are most directly weakened?
- Consider opsonization, complement activation and phagocytic clearance.

Case interpretation: Humoral deficiency

Deficient IgG/IgM weakens opsonization and classical complement activation.

- Pyogenic organisms become harder to clear from distal air spaces.
- Recurrent bacterial infection fits the immune phenotype.

Case: Fever after influenza

Adult has an influenza-like illness, followed by renewed high fever and productive cough.

- Question: What pathophysiologic sequence makes secondary bacterial infection plausible?
- Link viral epithelial damage to loss of local airway defenses.

Case interpretation: Bacterial superinfection

Respiratory viruses disrupt epithelial defense and mucociliary clearance.

- Secondary bacterial invasion triggers neutrophil-rich alveolar exudate.
- The acute bacterial process may produce new consolidation.

Synthesis: Three decisions organize pneumonia

1

Where is infection established?

2

Which host defenses are compromised?

3

Which clinical syndrome constrains the likely pathogens?

4

What pattern of tissue damage and complication is developing?

References and source boundaries

- Robbins, Cotran & Kumar. Pathologic Basis of Disease. 11th ed. (2026). Chapter 15, pp. 643–646.
- Specific framework: Fig. 15.27 (lung defenses), Table 15.7 (syndromes), adjacent bacterial morphology discussion.
- The original chapter PDF and diagnostic figures were not provided; clinical images have not been reproduced or independently reviewed.