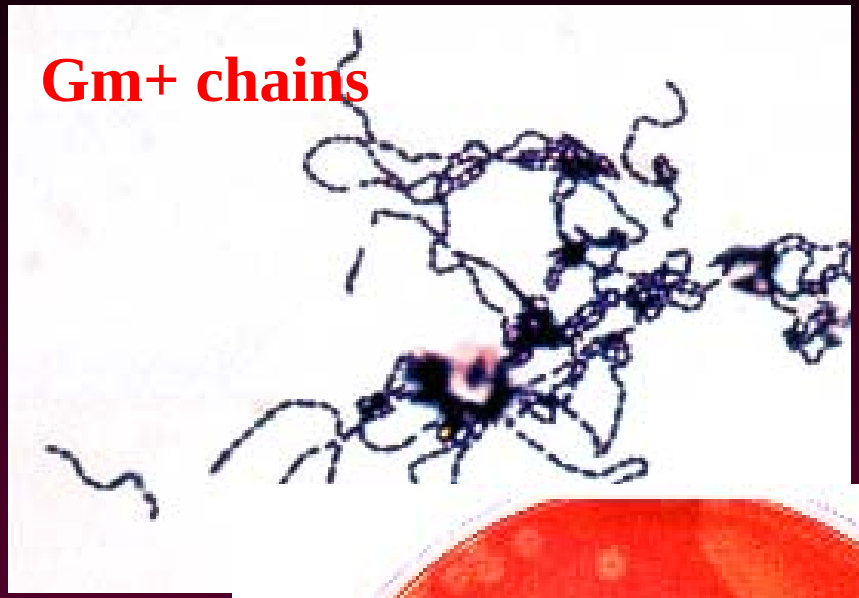
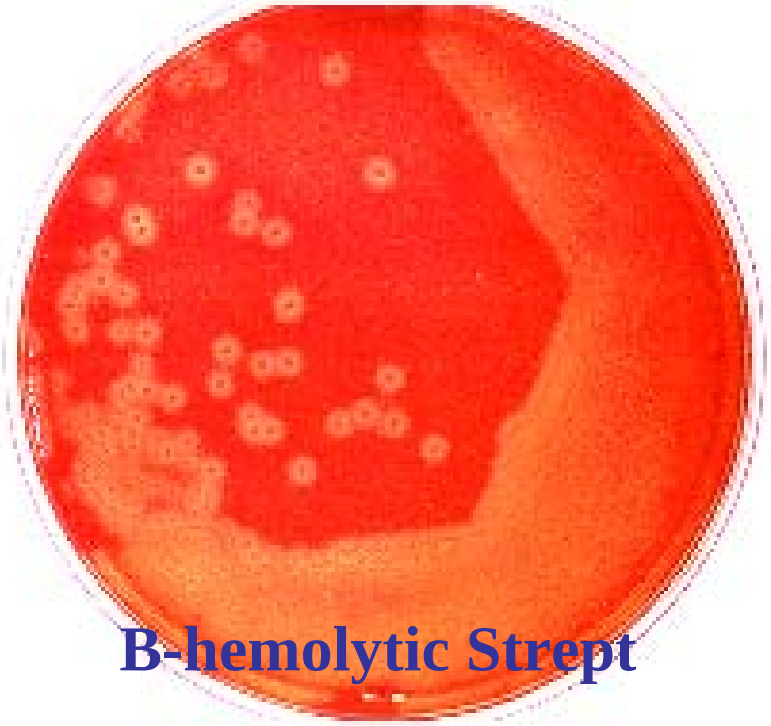
Streptococcus

Along with Staphylococcus, important disease agents found among this group

Microbiology of Strept

- Gram + cocci
  - generally in chains
- Old classification
  - by pattern of blood agar hemolysis
    - Beta-, Alpha-hemolytic Strept
- Lancefield classification
  - cell wall carbohydrate antigens
    - 20 major groups, A, B, G, etc.





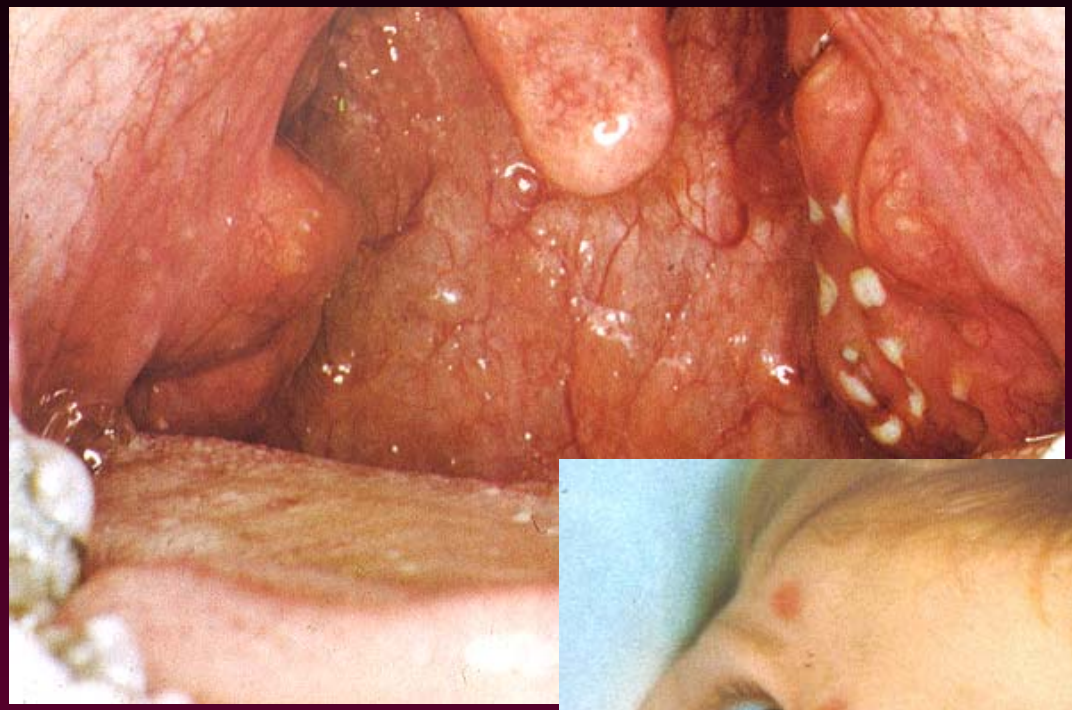
Virulence factors

- Extracellular enzymes
  - destroy host molecules
    - streptokinase
    - hyaluronidase, etc
- Cytolysins
  - Streptolysins S & O
    - act as superantigens
    - lyse RBC, WBC, etc.
- M-protein on fimbriae
  - cell attachments
  - antiphagocytic
  - autoimmune diseases (more later)
- Pyrogenic exotoxins

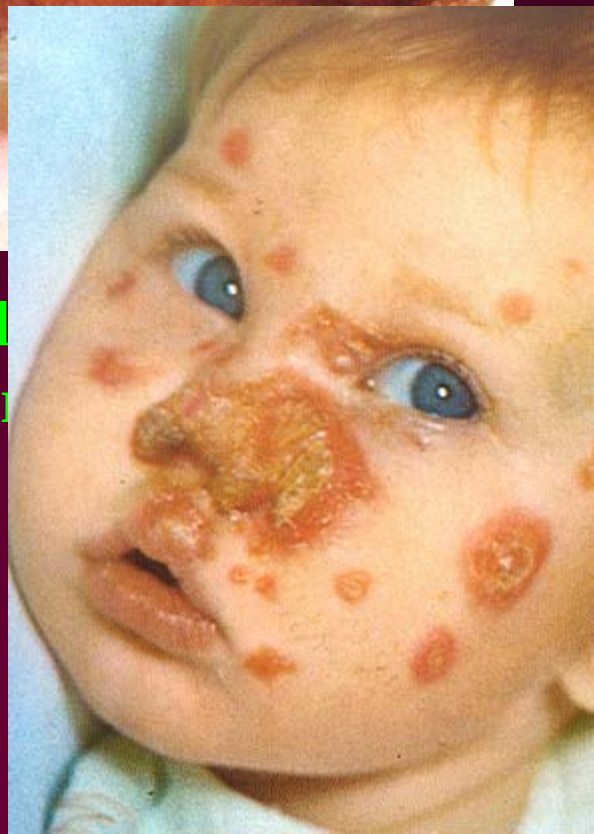
  (AKA erythrogenic toxins are phage-coded)
  - Act as superantigens
  - Fevers & rashes
  - Puerperal sepsis
  - Exotoxin A
    - Damage blood vessel causing tissue necrosis
    - Toxic-Shock-like syndrome
  - Exotoxin B
    - cysteine protease, S-S
    - tissue necrosis
    - **Seen in necrotizing fasciitis**

S. pyogenes, group A

- Strept pharyngitis
  - "Strept-throat"
- Nasal, pharyngeal, anal carriers
  - droplet transmission
  - family infections
- inflammation of throat
  - exudate covers tonsils
  - fevers, sore throat
  - malaise, headache
- Penicillin or erythromycin
  - to prevent sequelae
- Impetigo



Pharyngeal infl
exudate, "st



Impetigo

Rapid Direct Test for S. pyogenes

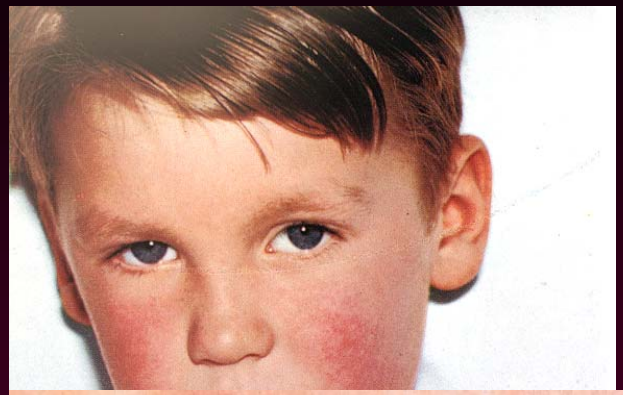
Positive reaction

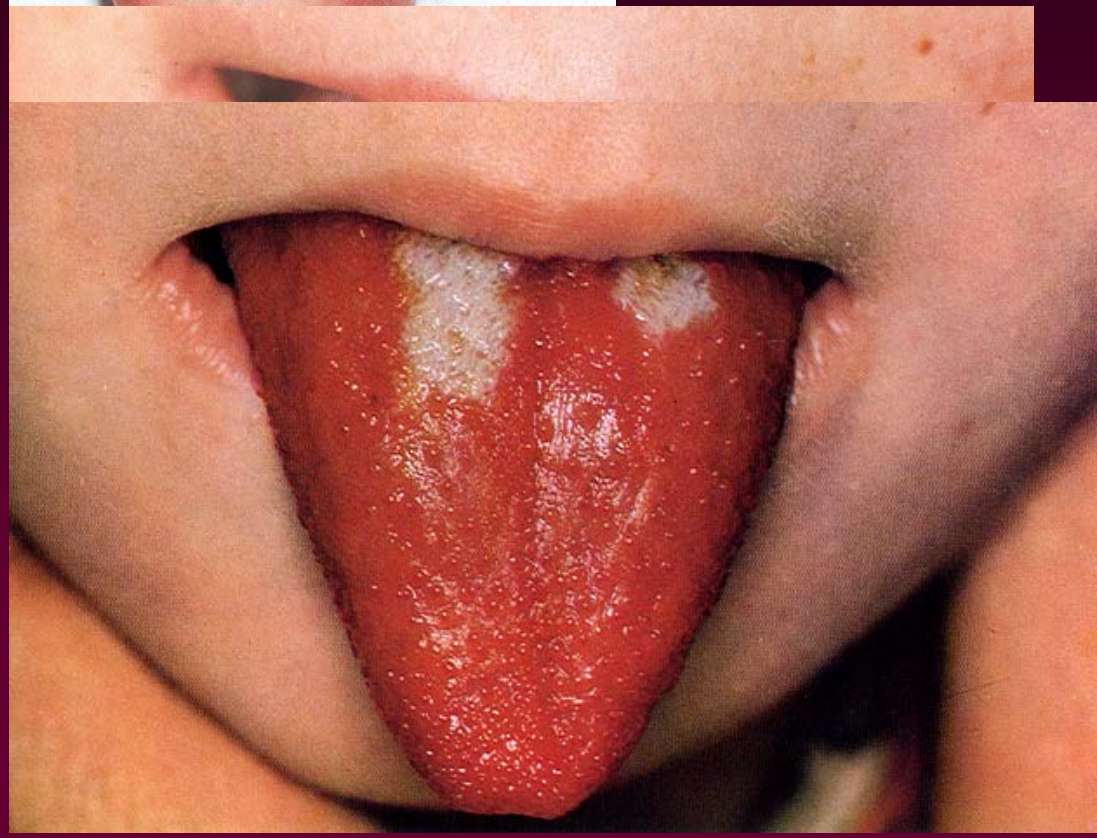
Negative reaction

Throat swab mixed with latex beads coated with MAb against *S. pyogenes*

Scarlet Fever

- Pyrogenic exotoxins
  - Erythrogenic toxin
  - lysogenic bacteriophage
- begins as strept throat
  - pyrogenic-cytotoxic
  - scarlet rash
  - strawberry tongue
  - desquamation
- Puerperal sepsis
  - “child-bed fever”
- Penicillin, erythromycin





Strawberry tongue, white then red

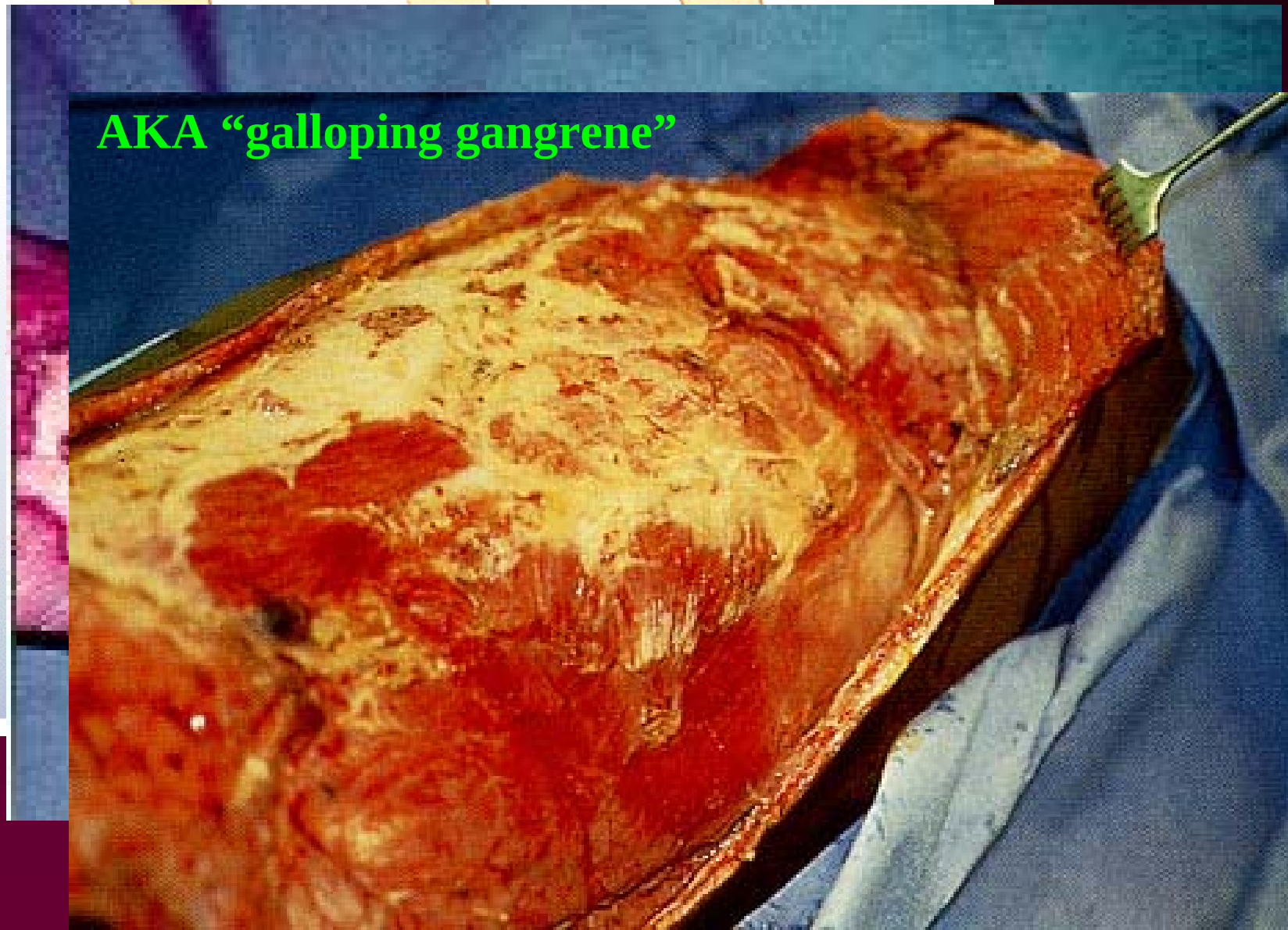
Invasive Strept Infections

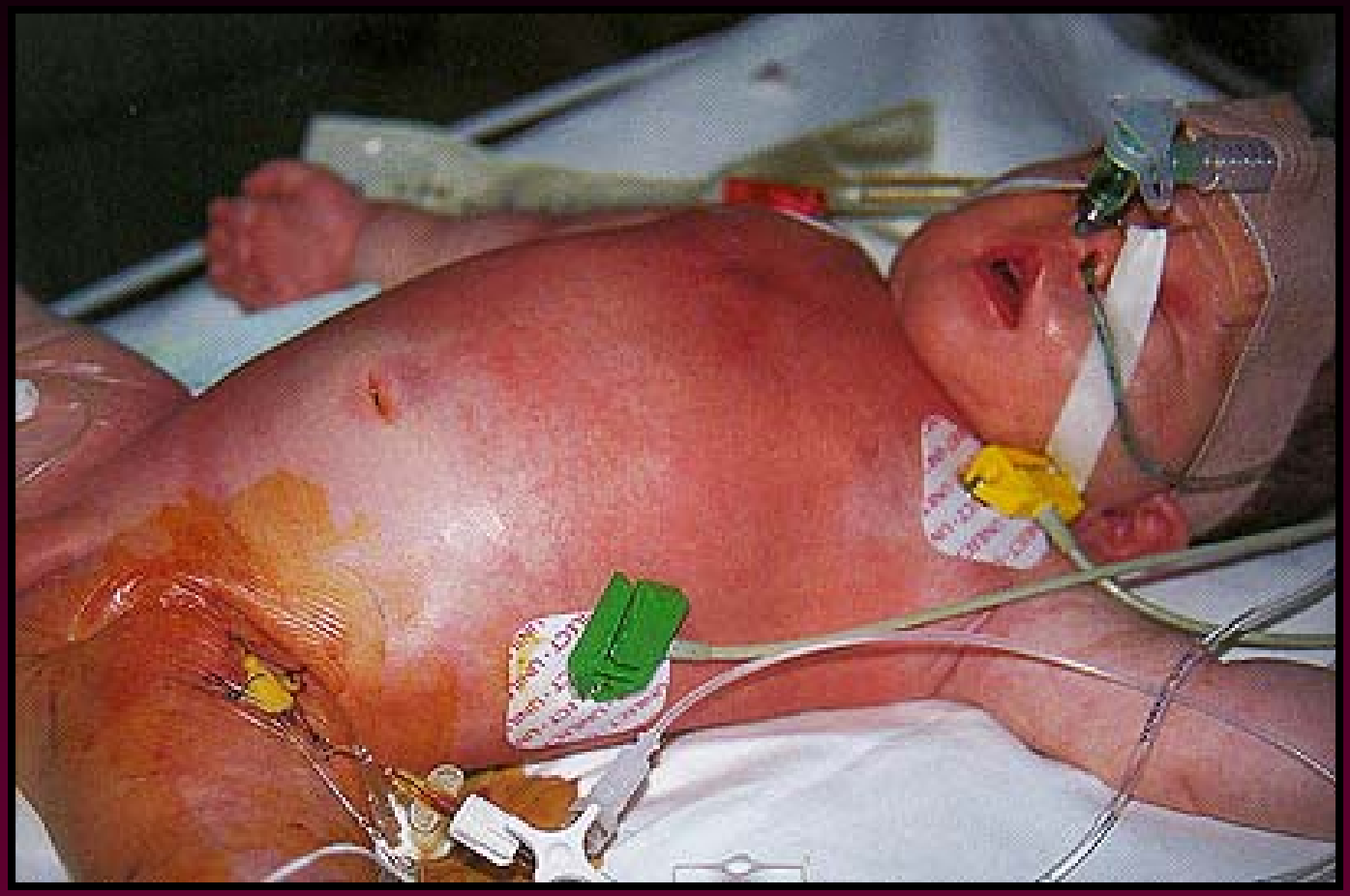
- Erysipelas
  - dermal infection
  - marked margins
  - serious infection
- Cellulitis
  - deeper tissues
    - may be septicemic
    - exotoxins A & B
    - damage blood vessels leading to necrosis
  - necrotizing fasciitis
    - “flesh-eating”
    - more severe than cellulitis
  - Toxic Shock-like Syndrome (Jim Henson)

Necrotizing fasciitis

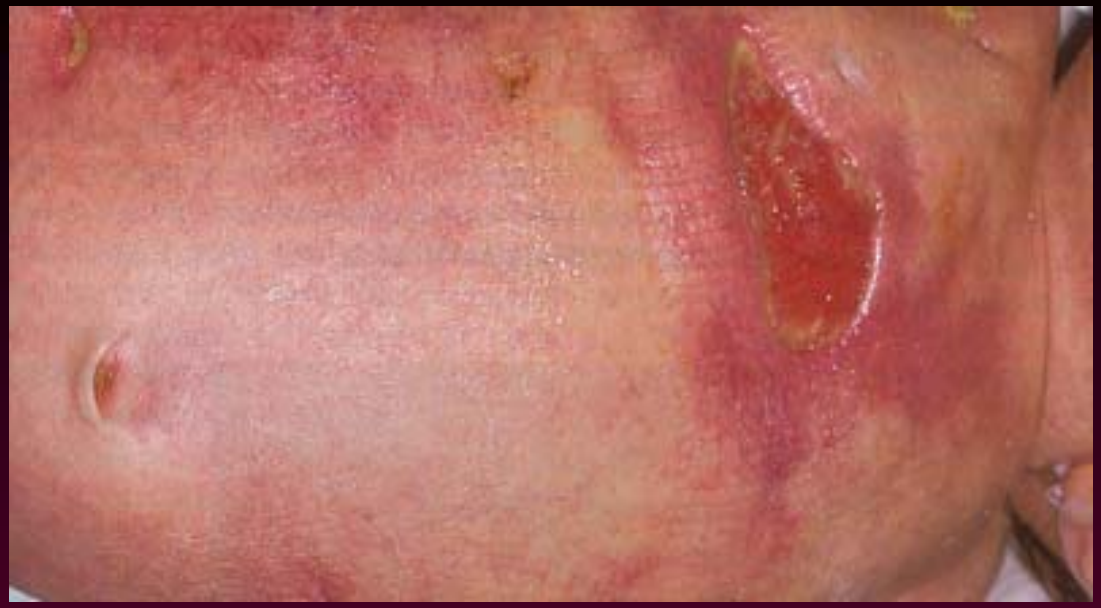
Foundations of Microbiology, 2nd ed., by Talaro & Talaro, ©1996 Times Mirror Higher Education Group, Inc.

Streptococcus pyogenes. Figure MF18.2 (T)



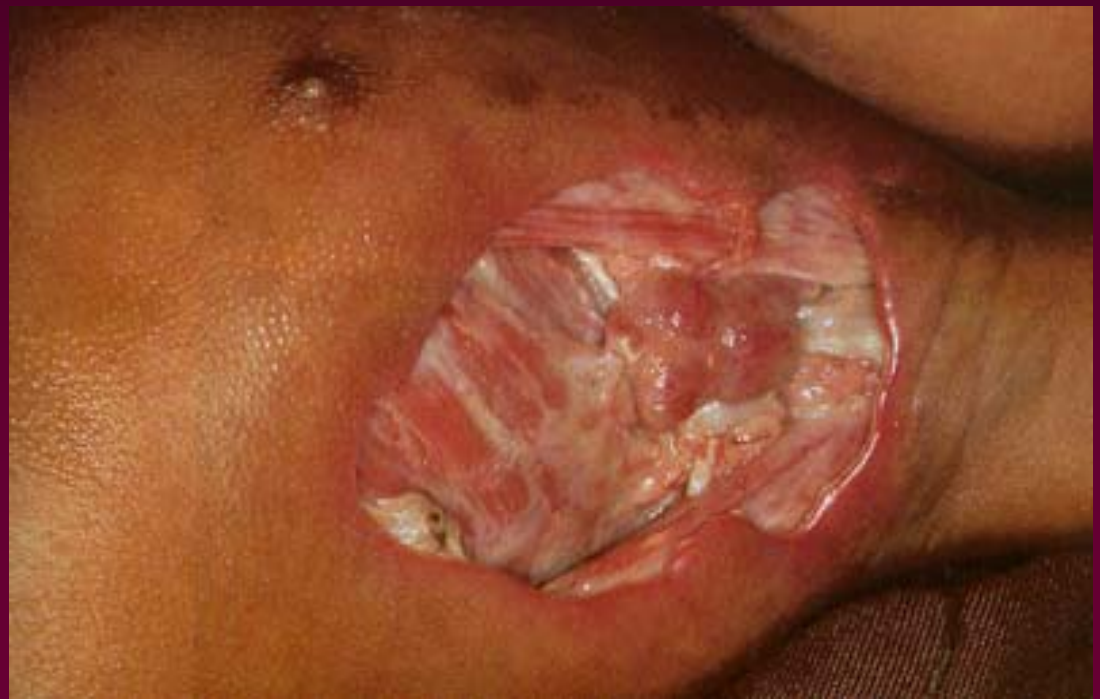


Necrotizing fasciitis



Before treatment

Necrotizing fasciitis

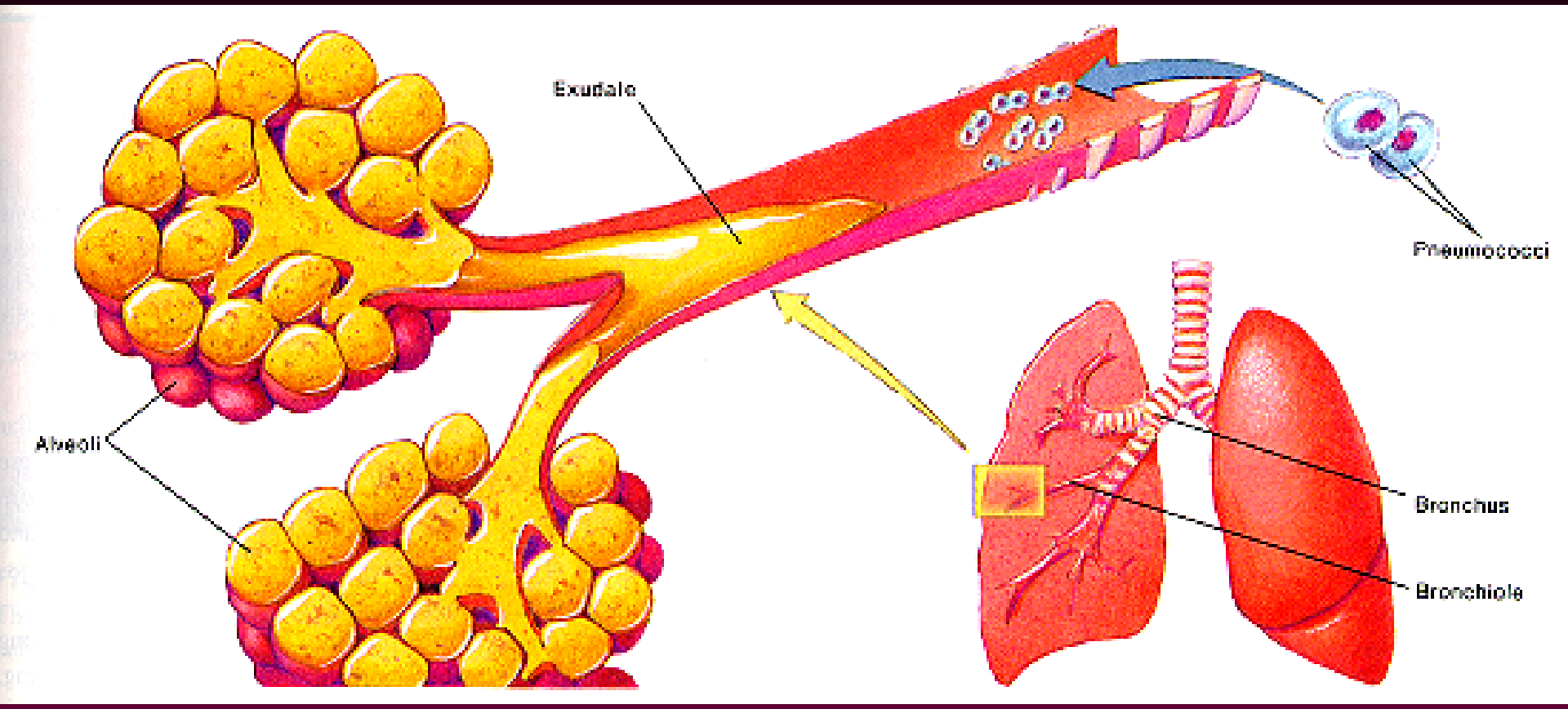


After debulking

Streptococcus pneumoniae

- Oval diplococcus, bullet shaped
- One of the most significant human pathogens (isolated by Pasteur)
  - Leading cause of death before antibiotics, still among top 10.
  - Causes 60-70% of all bacterial pneumonias.
  - Causes the majority of otitis media cases in young children, some of which progress to meningitis
  - Pathogenesis tied to large polysaccharide capsule, and host inflammation.
  - IgA protease, pneumonlysin & ↓ Oxidative burst
  - classical & alternative complement activation

Pneumococcal pneumonia:



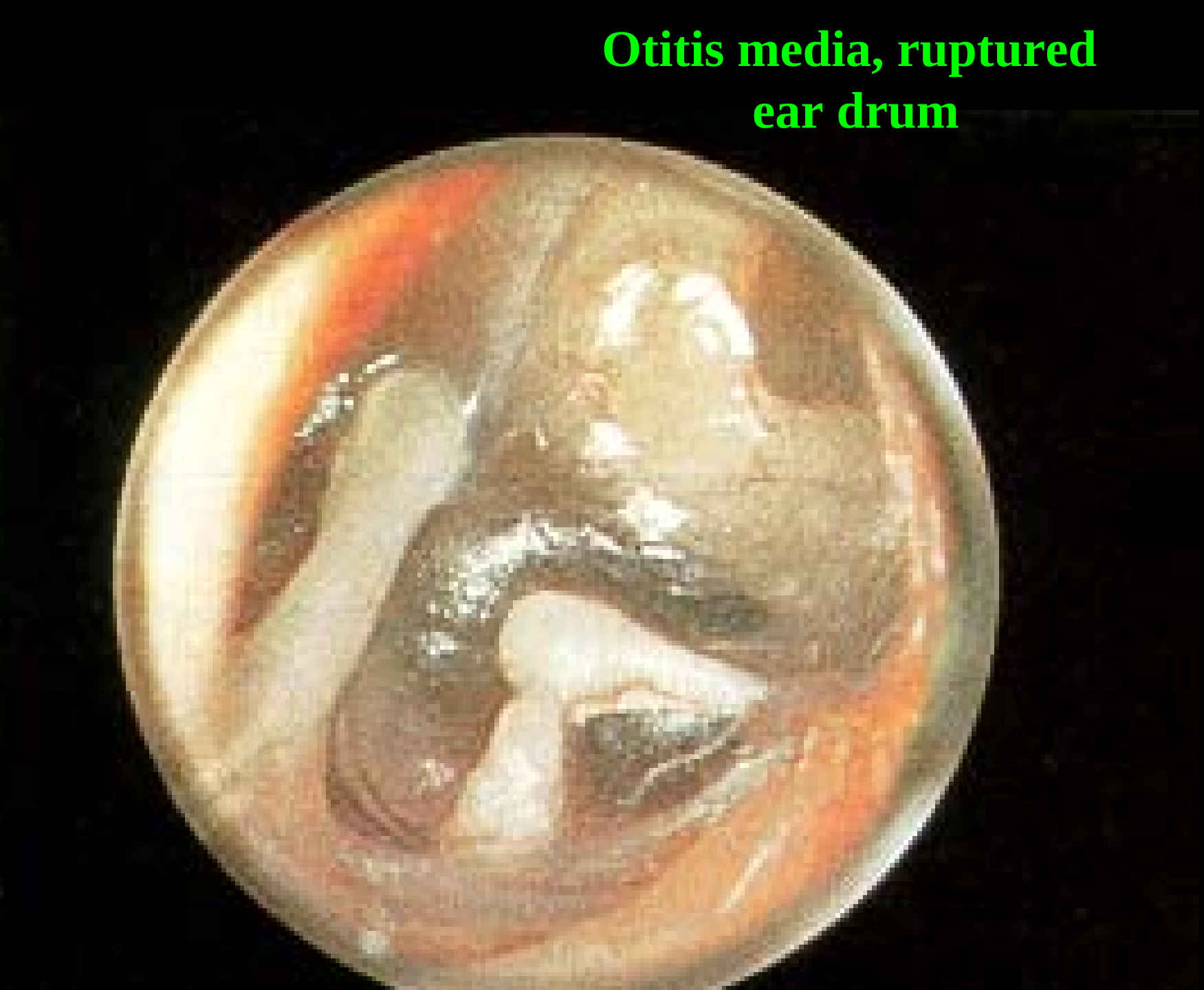
--As pneumococci grow, they induce an acute inflammatory response.

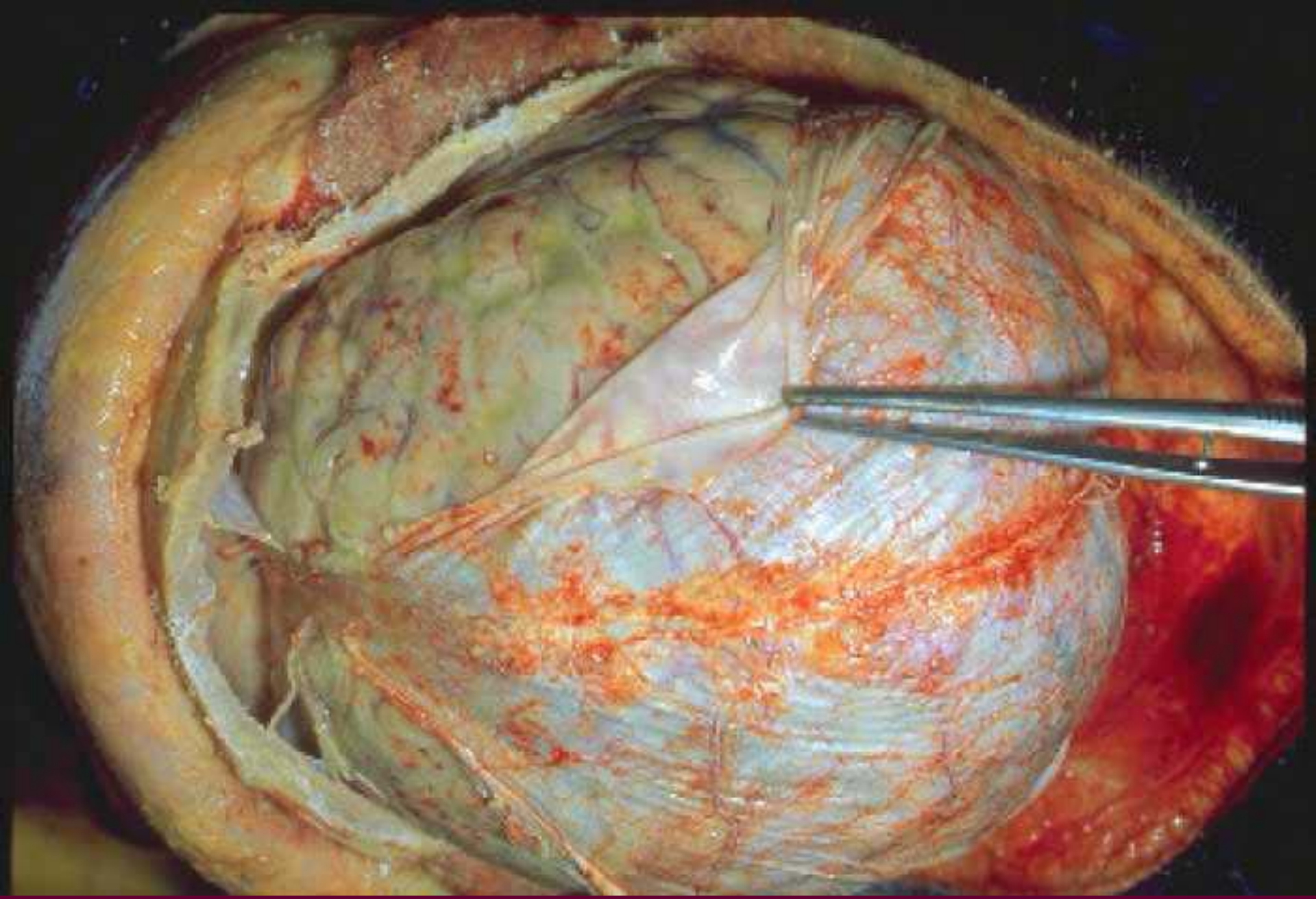
--Exudate fills the lungs with fluid

Transmission & Epidemiology of Pneumonia

- **In most cases, the source is endogenous, but aerosol transmission is common.**
  - **Lungs, ears, brain, mostly**
- **Most common predisposing factor are: damage of respiratory tract by viral infection, toxic gases, alcohol abuse, chronic renal disease, diabetes, etc..**
- **Advanced age and immunodeficiency also predisposing factors**
- **Treatment with penicillin or erythromycin**
- **Vaccine is available for population at risk**

Otitis media, ruptured ear drum



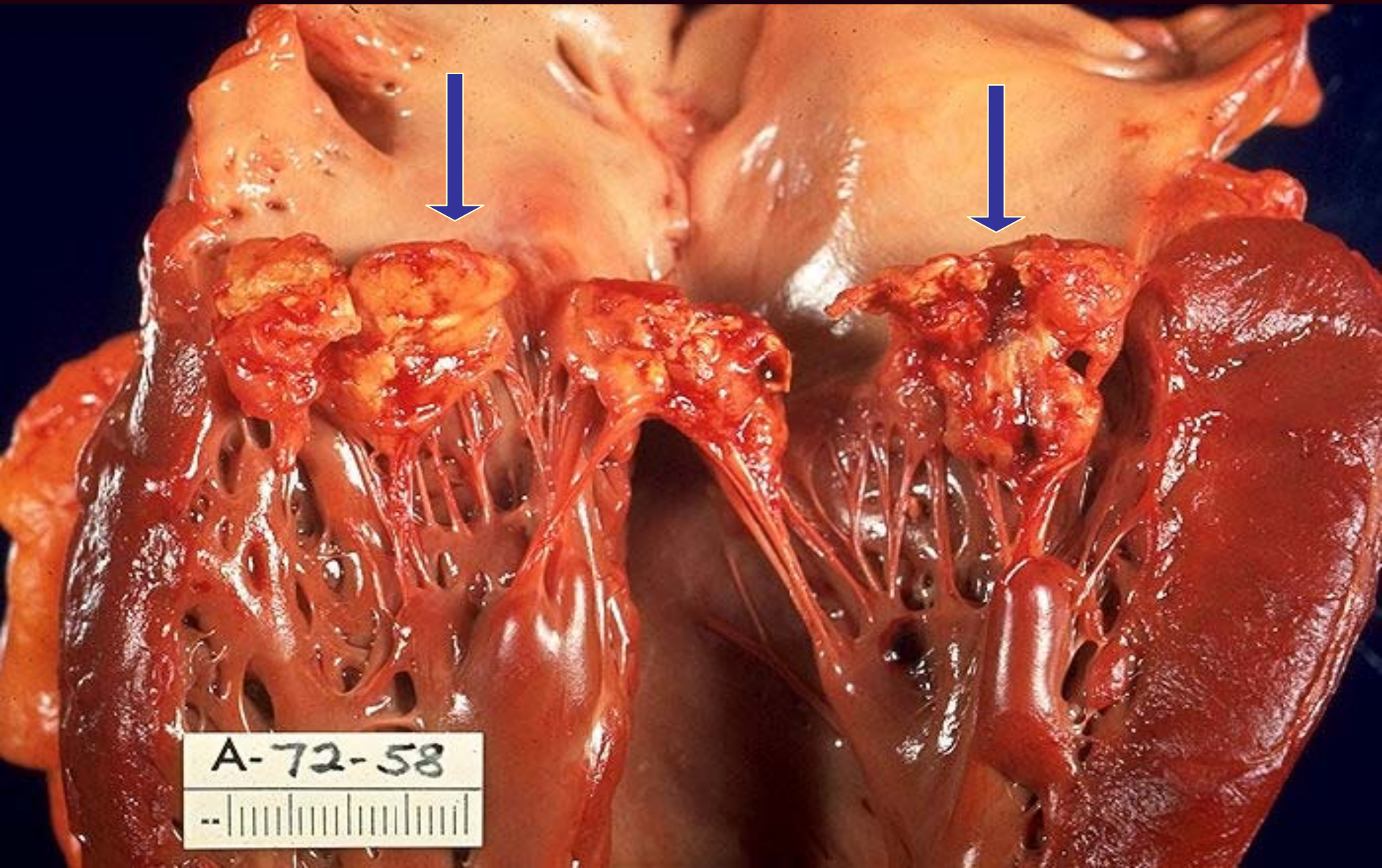


Strept Pneumococcal meningitis

Other Disease-Causing Strep

- Viridans Group, (normal flora, skin & throat)
  - Large and complex group of Alpha-hemolytic strep that are not characterized by Lancefield typing.
  - Cause 70% of all cases of **bacterial endocarditis**. Once these organisms gain access to the blood, they can localize in the heart at locations that have been previously damaged by disease or injury.
  - Colonization of these areas leads to the formation of deformed tissue masses called "vegetations", which continue to increase in size
    - especially in rheumatic hearts
    - causes "heart murmurs"
- Important cause of dental caries; *S. mutans* forms dextrans, the matrix of dental plaque
  - Also produces acids that demineralize tooth enamel

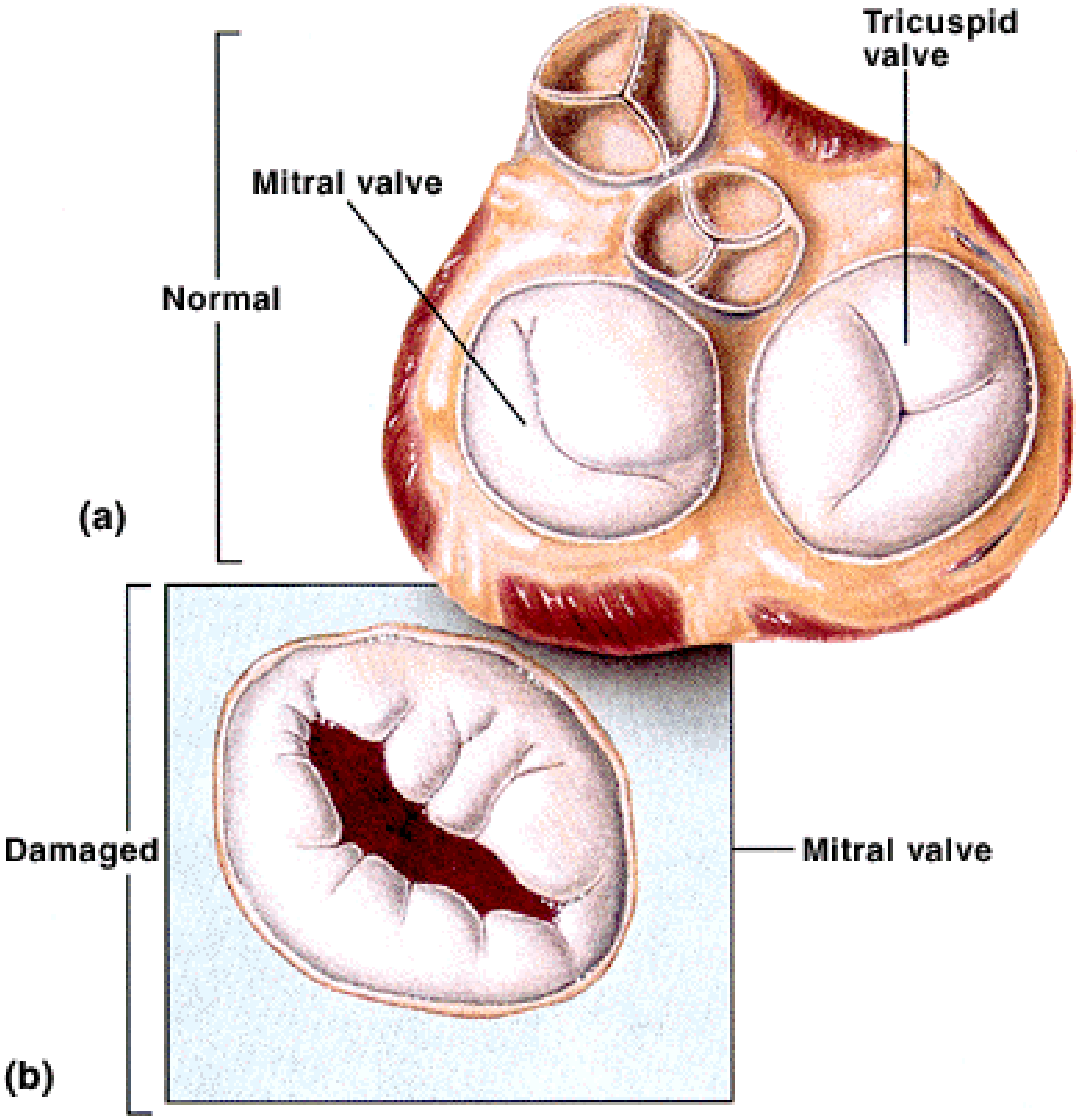
Subacute endocarditis, note vegetations on cardiac mitral valve



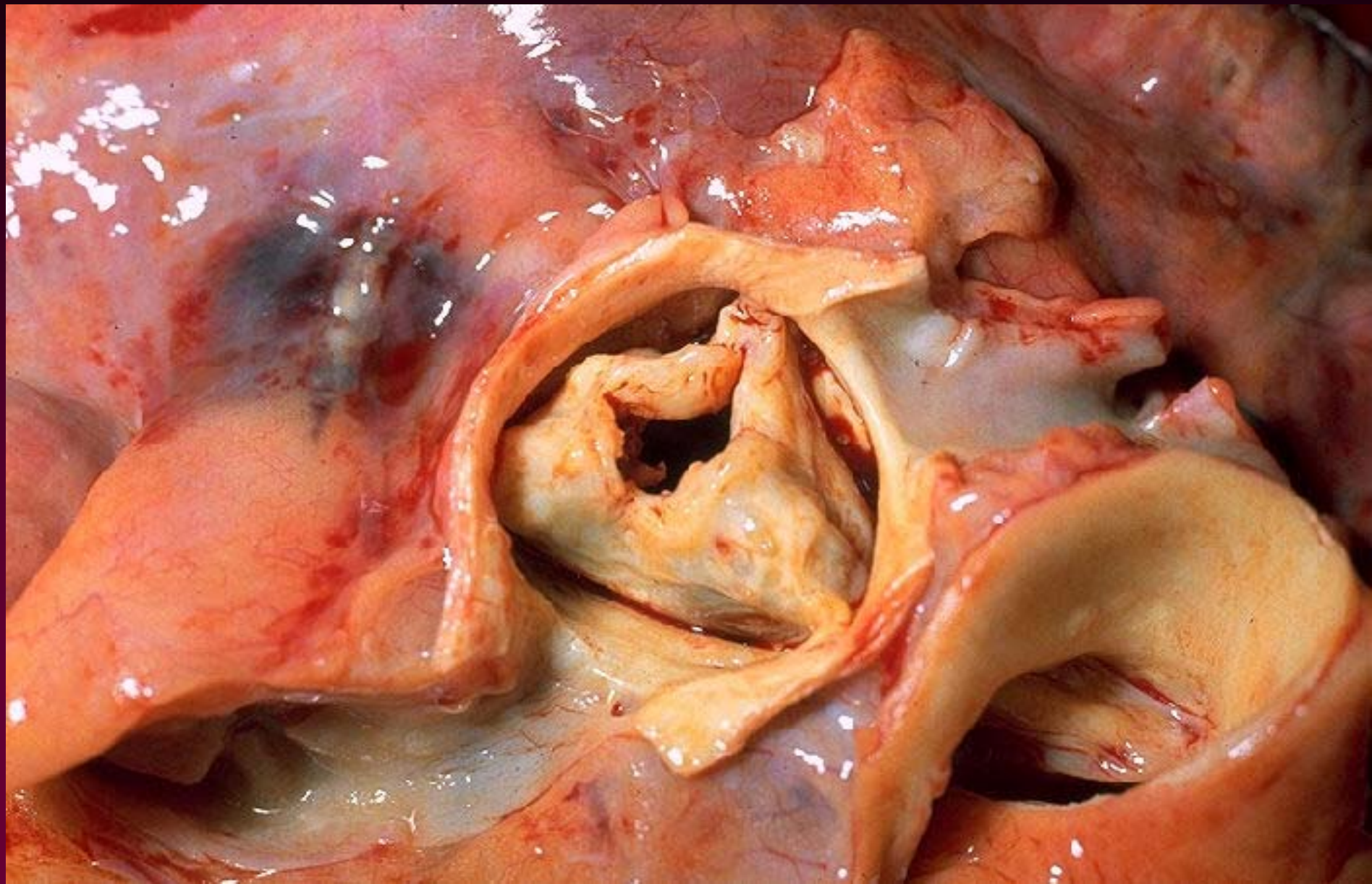
Post-Streptococcal Sequelae:

- **Rheumatic Fever (RF)**
  - Symptoms include carditis, abnormal EKG, painful arthritis, fever, etc. Severe carditis can result in permanent damage to heart valves (murmurs). Responsible for 15,000 deaths/year in U.S.
- **Acute Glomerulonephritis (AGN)**
  - Kidney damage may be caused by cross-reacting antibodies (Type II) or deposition of immune complexes (Type III), or toxins like SLO & SLS. Can be self-limiting or fatal
  - Often leads to kidney transplants
- **Role of Strept-"M" proteins**
  - cross-reacting with heart & kidney cell antigens

The Cardiac Complications of Rheumatic Fever. Figure 18.13 (T)



Rheumatic heart, aortic valve stenosis, fused leaflets



Rheumatic heart, thickened mitral valve, hypertrophied left ventricle