BOX 23-1 Streptococci and Their Diseases

***S. PYOGENES* (GROUP A)**

Pharyngitis, scarlet fever, pyoderma, erysipelas, cellulitis, necrotizing fasciitis, streptococcal toxic shock syndrome, bacteremia, rheumatic fever, glomerulonephritis

***S. AGALACTIAE* (GROUP B)**

Neonatal infections (meningitis, pneumonia, bacteremia), urinary tract infections, amnionitis, endometritis, wound infections, pneumonia, bacteremia

OTHER BETA-HEMOLYTIC STREPTOCOCCI

Pharyngitis, abscess formation, bacteremia

S. PNEUMONIAE

Pneumonia, sinusitis, otitis media, meningitis, bacteremia

VIRIDANS GROUP STREPTOCOCCI

Bacteremia, endocarditis, abscess formation, dental caries

25a

TABLE 23-1 Classification of Common Streptococcal Pathogens

SEROLOGICAL CLASSIFICATION	BIOCHEMICAL CLASSIFICATION	HEMOLYTIC PATTERNS
A	*S. pyogenes*	Beta
B	*S. agalactiae*	Beta, occasionally alpha or nonhemolytic
C	*S. anginosus, S. equisimilis*	Beta, occasionally alpha or nonhemolytic
D	*S. bovis*	Alpha, nonhemolytic; occasionally beta
F	*S. anginosus*	Beta
G	*S. anginosus*	Beta
—	*S. pneumoniae*	Alpha
Viridans group	*S. mutans* group, *S. salivarius* group, *S. sanguis* group, *S. mitis* group, *S. milleri* group	Alpha, nonhemolytic

25b

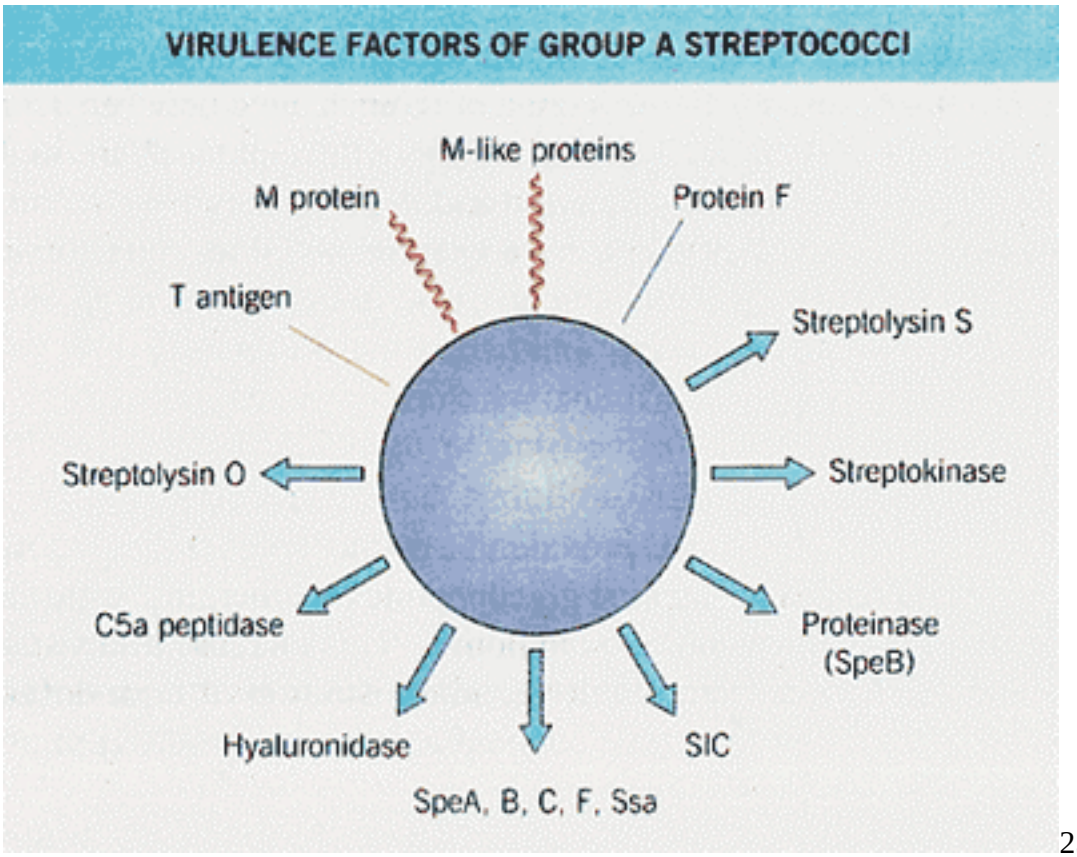
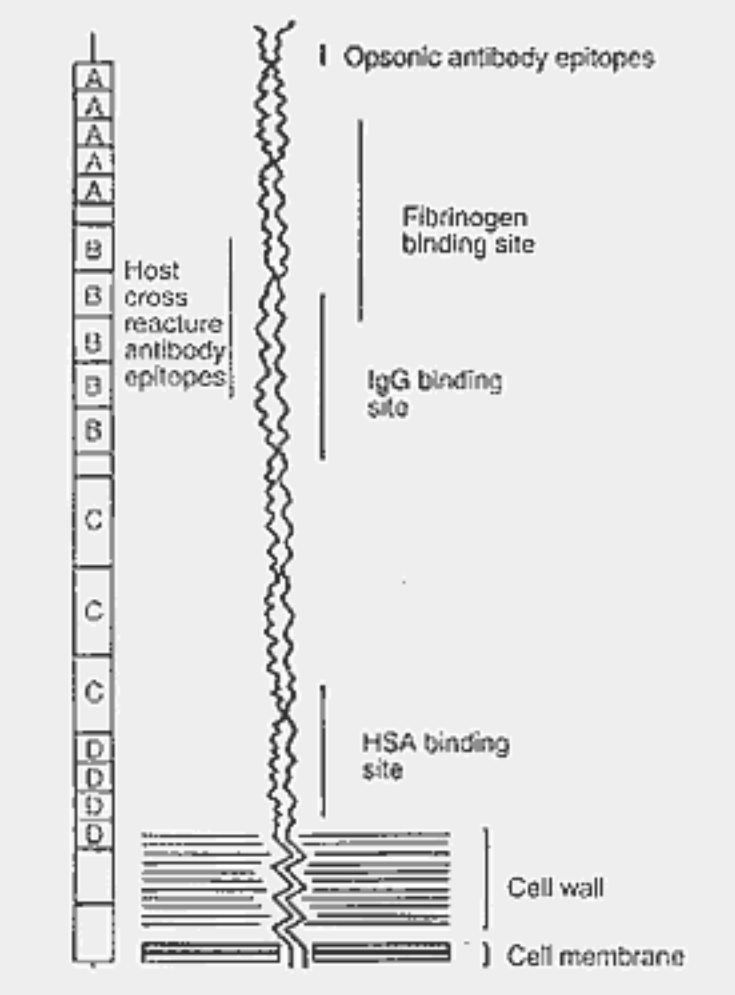
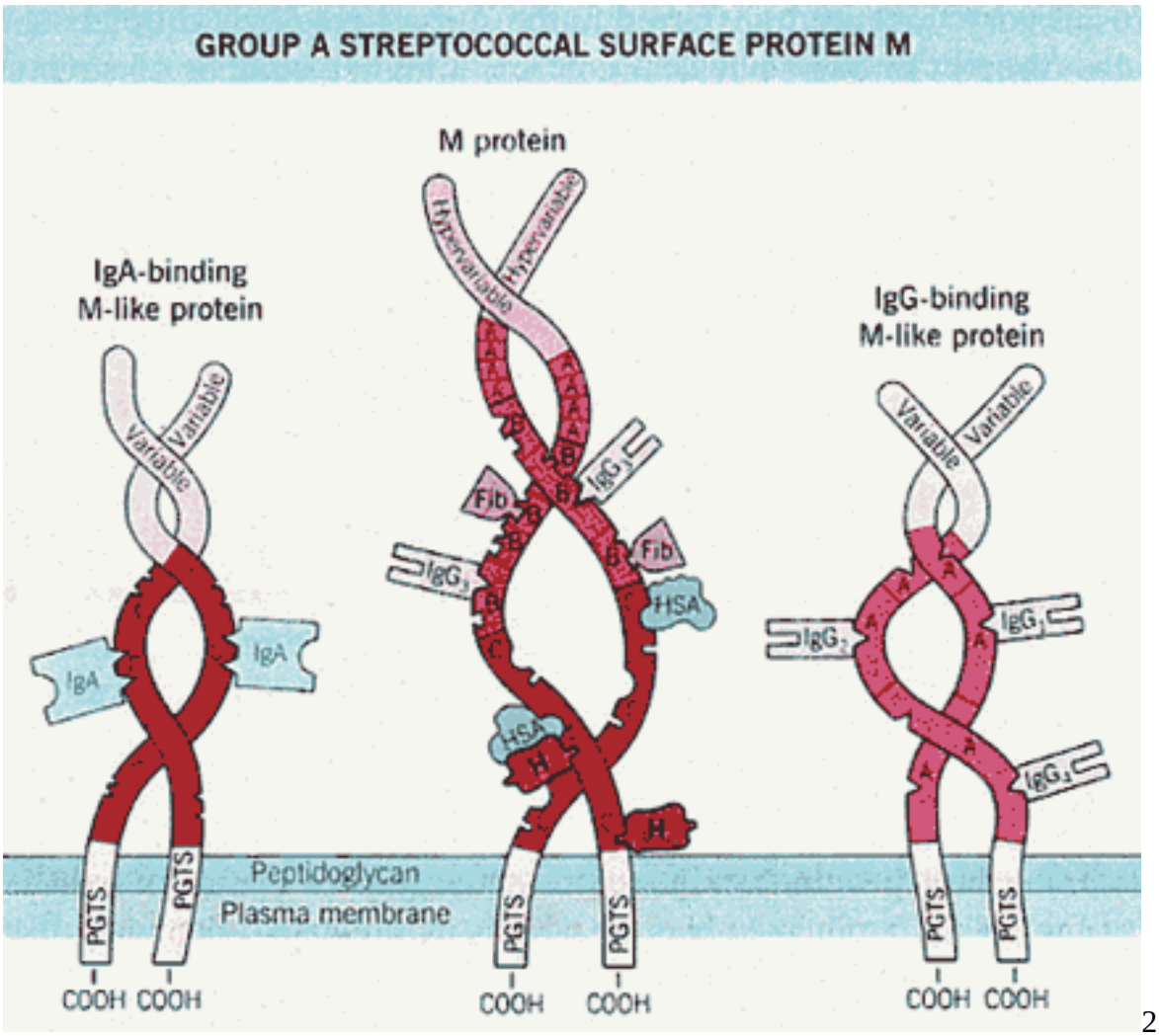

VIRULENCE FACTORS OF GROUP A STREPTOCOCCI
M-like proteins
M protein
Protein F
T antigen
Streptolysin S
Streptolysin O
Streptokinase
C5a peptidase
Proteinase
(SpeB)
Hyaluronidase
SIC
SpeA, B, C, F, Ssa
25c

TABLE 23-2 Group A *Streptococcus* Virulence Factors

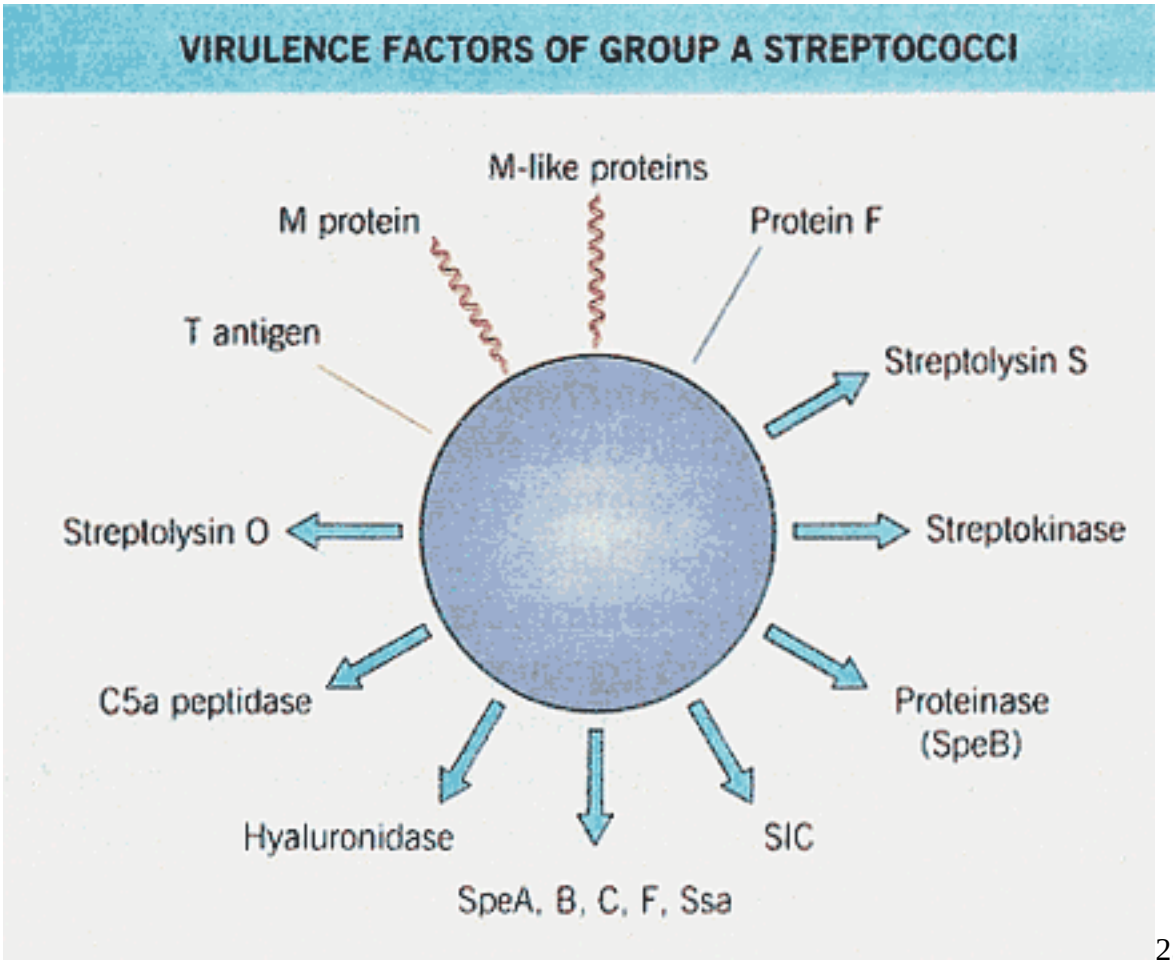
VIRULENCE FACTOR	BIOLOGICAL EFFECT
Capsule	Nonimmunogenic; antiphagocytic
M protein	Antiphagocytic; degrades C3b
M-like proteins	Binds IgM, and IgG and alpha$_2$-macroglobulin (protease inhibitor)
F protein	Mediates adherence to epithelial cells
Pyrogenic exotoxins	Mediates pyrogenicity, enhancement of delayed hypersensitivity and susceptibility to endotoxin, cytotoxicity, nonspecific mitogenicity for T cells, immunosuppression of B-cell function, and production of scarlatiniform rash
Streptolysin S	Lyses leukocytes, platelets, and erythrocytes; stimulates release of lysosomal enzymes; nonimmunogenic
Streptolysin O	Lyses leukocytes, platelets, and erythrocytes; stimulates release of lysosomal enzymes; immunogenic
Streptokinase	Lyses blood clots; facilitates spread of bacteria in tissues
DNase	Depolymerizes cell-free DNA in purulent material
C5a peptidase	Degrades C5a

DNase, Deoxyribonuclease.


A
A
A
A
A
B
B
B
B
B
C
C
C
D
D
D
D
Host cross reacture antibody epitopes
Opsonic antibody epitopes
Fibrinogen binding site
IgG binding site
HSA binding site
Cell wall
Cell membrane
25e


GROUP A STREPTOCOCCAL SURFACE PROTEIN M
M protein
IgA-binding
M-like protein
IgG-binding
M-like protein
Hypervariable
Hypervariable
Variable
Variable
Variable
Variable
IgA
IgA
Fib
Fib
HSA
HSA
Peptidoglycan
Plasma membrane
PGTS
PGTS
PGTS
PGTS
PGTS
PGTS
COOH COOH
COOH COOH
COOH COOH

25f


VIRULENCE FACTORS OF GROUP A STREPTOCOCCI
M-like proteins
M protein
Protein F
T antigen
Streptolysin S
Streptolysin O
Streptokinase
C5a peptidase
Proteinase
(SpeB)
Hyaluronidase
SIC
SpeA, B, C, F, Ssa

25g

What Is a Pathogen?

Developing a definition of a pathogen requires looking closely at the many complicated relationships that exist among organisms

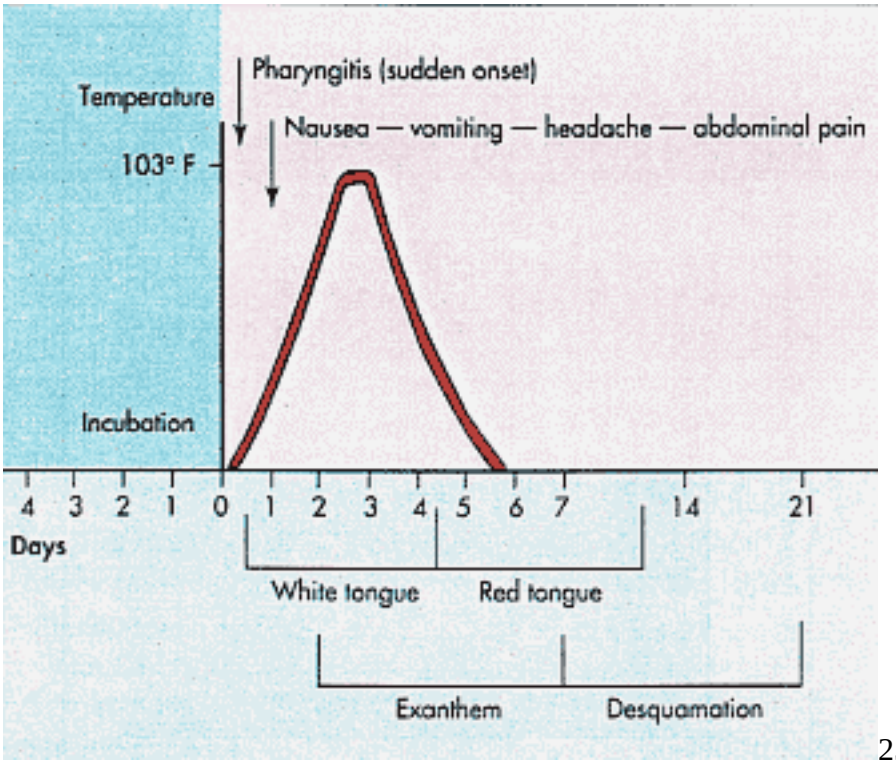
Stanley Falkow

If asked to define a pathogen, my answer is more complicated than that purely pragmatic definition. Instead, I define a pathogen as being any microorganism whose survival is dependent upon its capacity to replicate and persist on or within another species by actively breaching or destroying a cellular or humoral host barrier that ordinarily restricts or inhibits other microorganisms. This capacity to reach a unique host niche free from microbial competition and possibly safe from host defense mechanisms sets the foundation for the expression of specific determinants that permit such microbes to establish themselves within a host and to be transmitted to new susceptible hosts.

TAKEN FROM:

ASM News, Volume 63, Number 7, 1997

25h


Temperature
Pharyngitis (sudden onset)
Nausea — vomiting — headache — abdominal pain
103° F
Incubation
4 3 2 1 0 1 2 3 4 5 6 7 14 21
Days
White tongue
Red tongue
Exanthem
Desquamation
25i

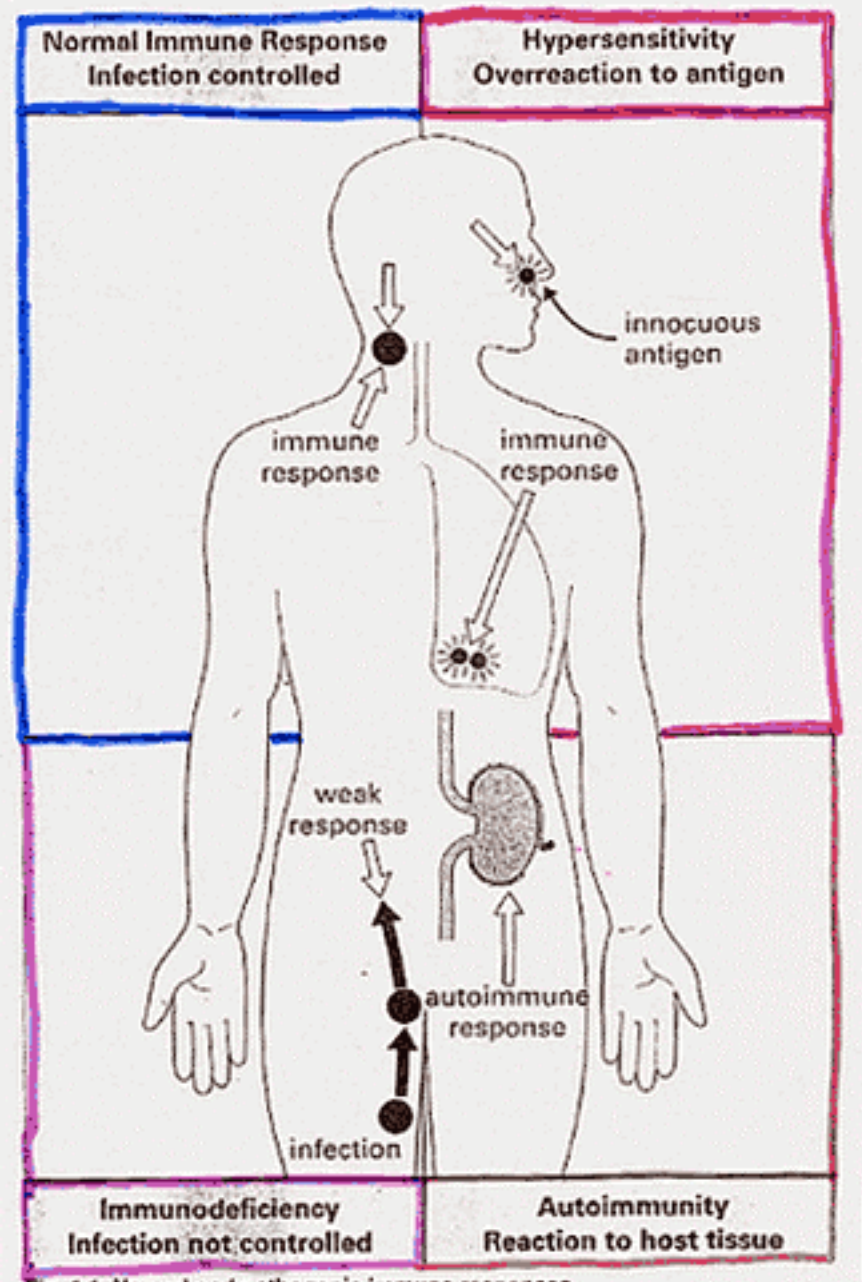


Fig. 1.1 Normal and pathogenic immune responses.

25j

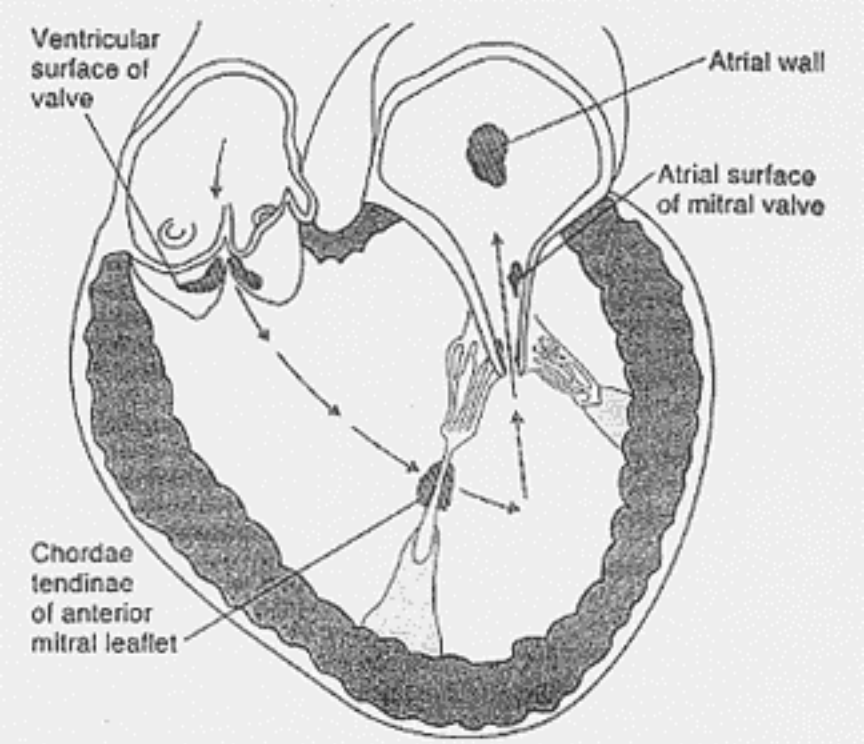


Figure 64.1. The location of endocarditic vegetations resulting from high-velocity regurgitant blood flow. The *arrows* indicate the high-velocity stream of blood. Regurgitant flow through the orifice of an incompetent aortic valve results in vegetations on the ventricular surface of the valve or on the chordae tendineae of the anterior mitral leaflet. Regurgitant flow across the incompetent mitral valve into the low-pressure left atrium allows vegetations to form on the atrial surface of the mitral valve (or at the site of jet stream impact on the atrial wall).

25k