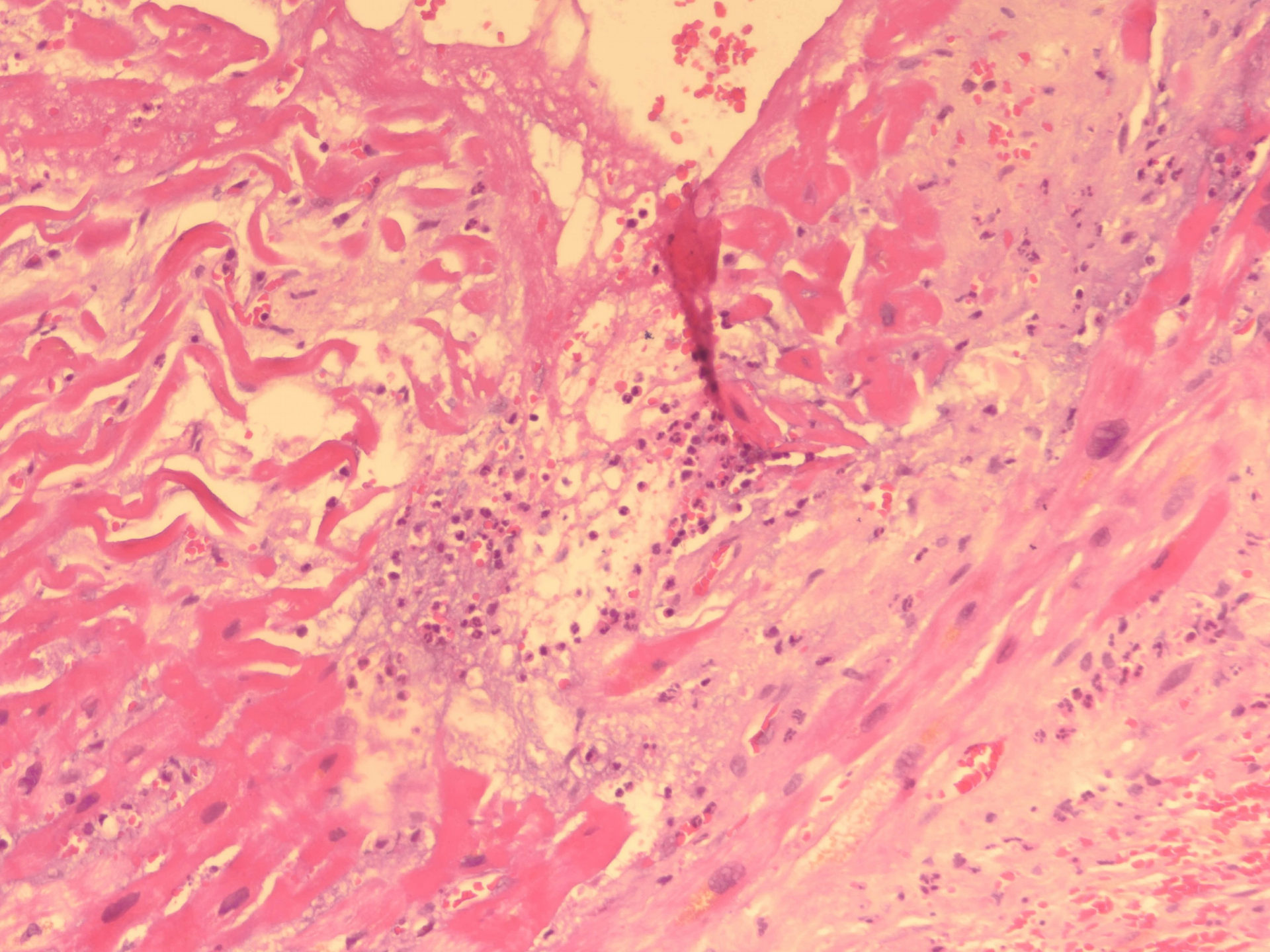
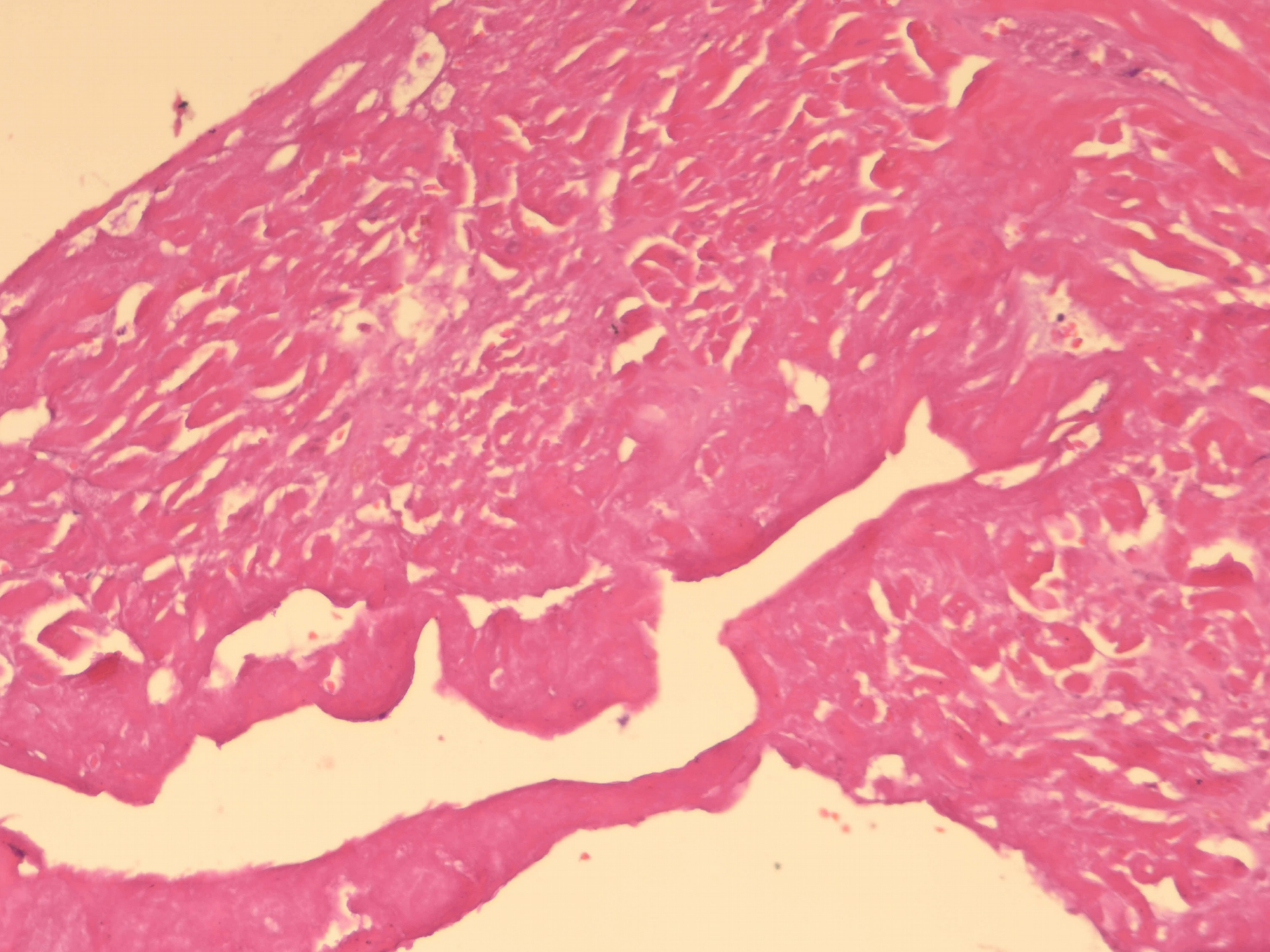
CPC1050414

PATH 何思蓓

Microscopic findings





Patterns of Tissue Necrosis

oagulative necrosis is a form of necrosis in which the architecture of dead tissues is preserved for a span of at least some days

Major etiologies of acquired mitral and aortic valve disease

Mitral valve disease

Mitral stenosis
- Postinflammatory scarring (rheumatic)
- Calcification of mitral annulus

Mitral regurgitation
- Abnormalities of leaflets and commissures
  - Postinflammatory scarring (rheumatic)
  - Infective endocarditis
  - Floppy mitral valve
- Abnormalities of mitral apparatus
  - Rupture of papillary muscle
  - Papillary muscle dysfunction (fibrosis or ischemia)
  - Rupture of chordae tendineae
- Left ventricular enlargement (e.g., congestive cardiomyopathy)
- Calcification of mitral annulus

Ischemic Heart Disease

n imbalance between the supply (perfusion) and demand of the heart for oxygenated blood. **Ischemia** brings not only an insufficiency of oxygen, but also reduces the availability of nutrients and the removal of metabolites

athogenesis

he dominant cause of the IHD syndromes is insufficient coronary perfusion relative to myocardial demand, due to chronic, progressive **atherosclerotic** narrowing of the epicardial coronary arteries, and variable degrees of superimposed acute plaque change, thrombosis, and vasospasm.

Ischemic Heart Disease

resent as one or more of the following clinical syndromes:

- **Myocardial infarction**, the most important form of IHD, in which ischemia causes the death of heart muscle.
- Angina pectoris, in which the ischemia is of insufficient severity to cause infarction, but may be a harbinger of MI.
- Chronic IHD with heart failure.
- Sudden cardiac death.

COMPLICATION FOLLOWING ACUTE MI

dysfunction

- Contractile

rupture

- Arrhythmias
- Myocardial

infarction

- Pericarditis
- Right ventricular

- Infarct extension
- Mural thrombus
- Ventricular

aneurysm

dysfunction

- Papillary muscle

heart failure

- Progressive late

Pathologic diagnosis

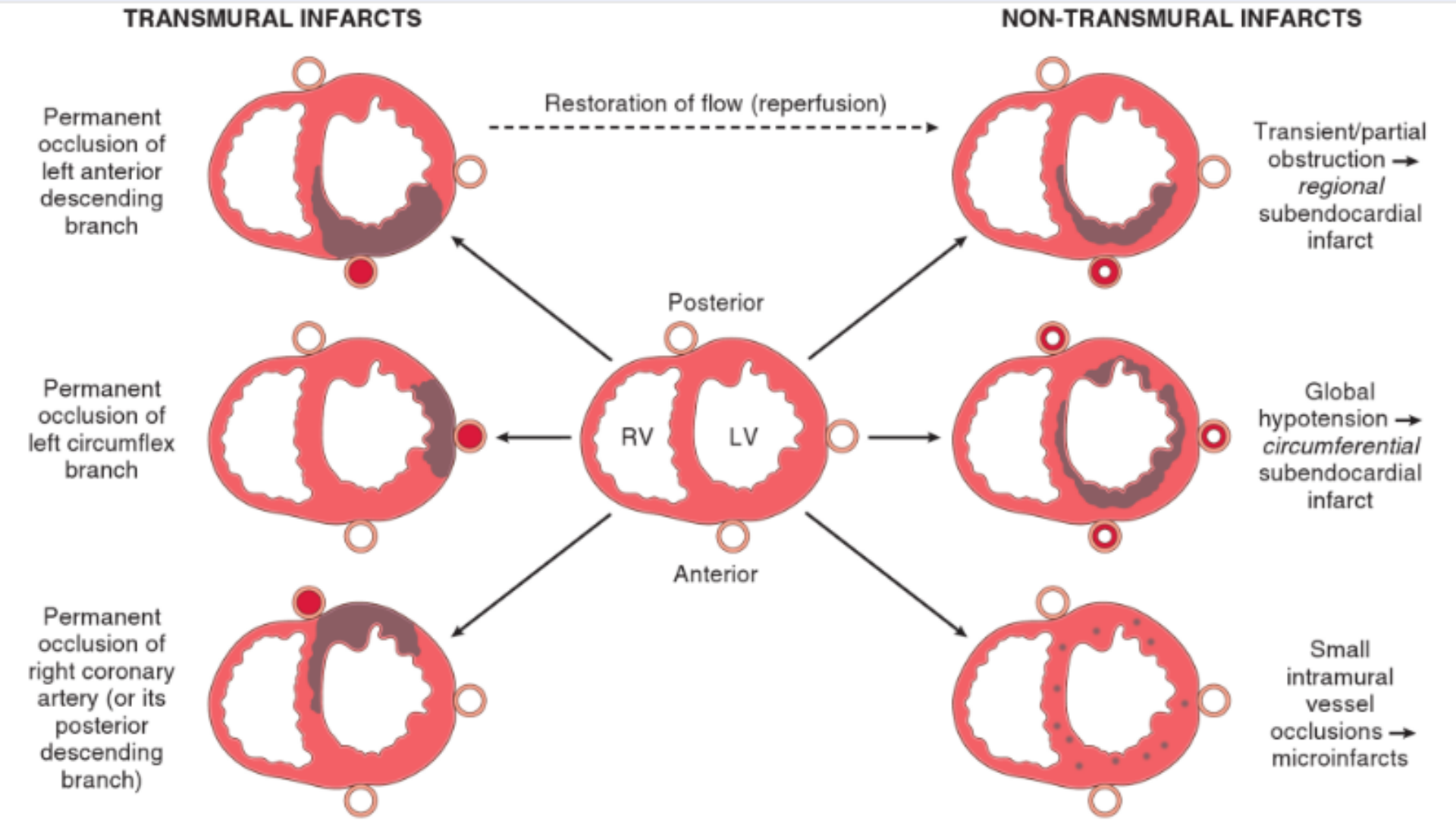
Mitral valve, MVR ---Acute papillary muscle infarction

VGHKS

Introduction

Myocardial infarction

LOCATION / SIZE



EKG

Owing to the characteristic electrocardiographic changes resulting from myocardial ischemia or necrosis in various distributions, a transmural infarct is sometimes referred to as an "ST elevation myocardial infarct" (STEMI) and a subendocardial infarct as a "non–ST elevation infarct" (NSTEMI). Depending on the extent and location of the vascular involvement, microinfarctions show nonspecific changes or can even be electrocardiographically silent.

ARTICLES

- **Papillary Muscle Rupture Following Non-ST-Elevation Myocardial Infarction: A Case Report**

  Echocardiography. 2016 Jan 29. doi: 10.1111/echo.13177

  Murad K, Missov E.

- **Partial Posteromedial Papillary Muscle Rupture Caused by Myocardial Ischemia Only without Myocardial Infarction.**

  Echocardiography. 2016 Mar 27. doi: 10.1111/echo.13223

  Tsujimoto M, Tanaka H, Matsumoto K, Inoue T, Okita Y, Hirata KI.

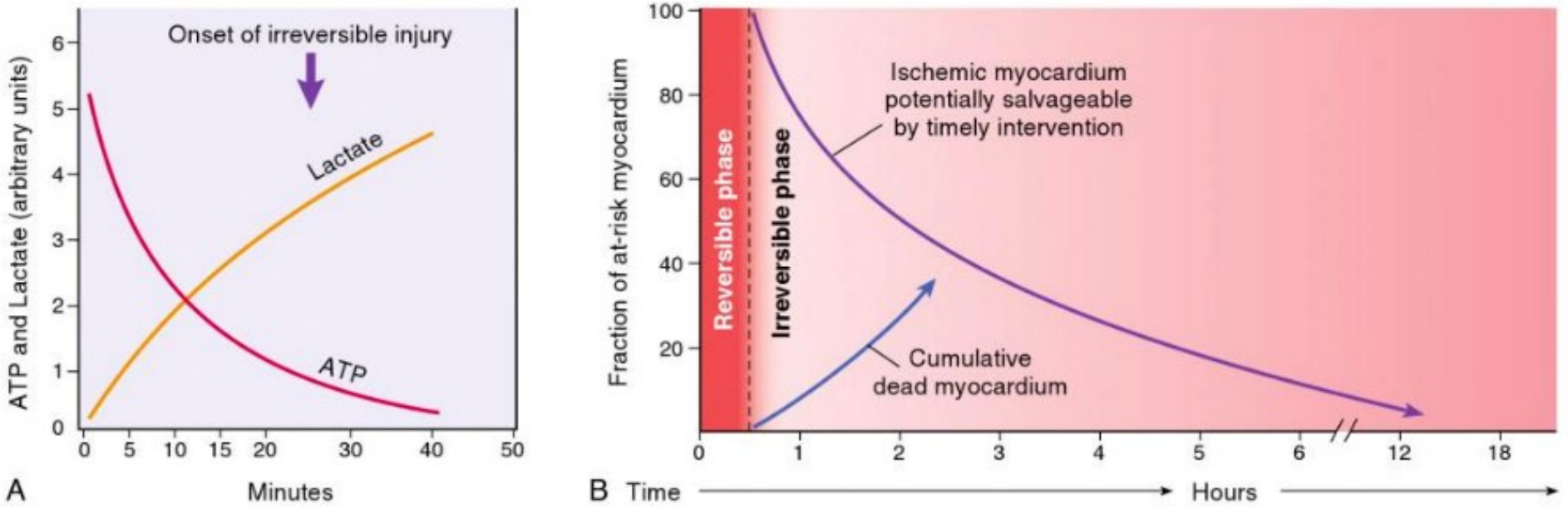


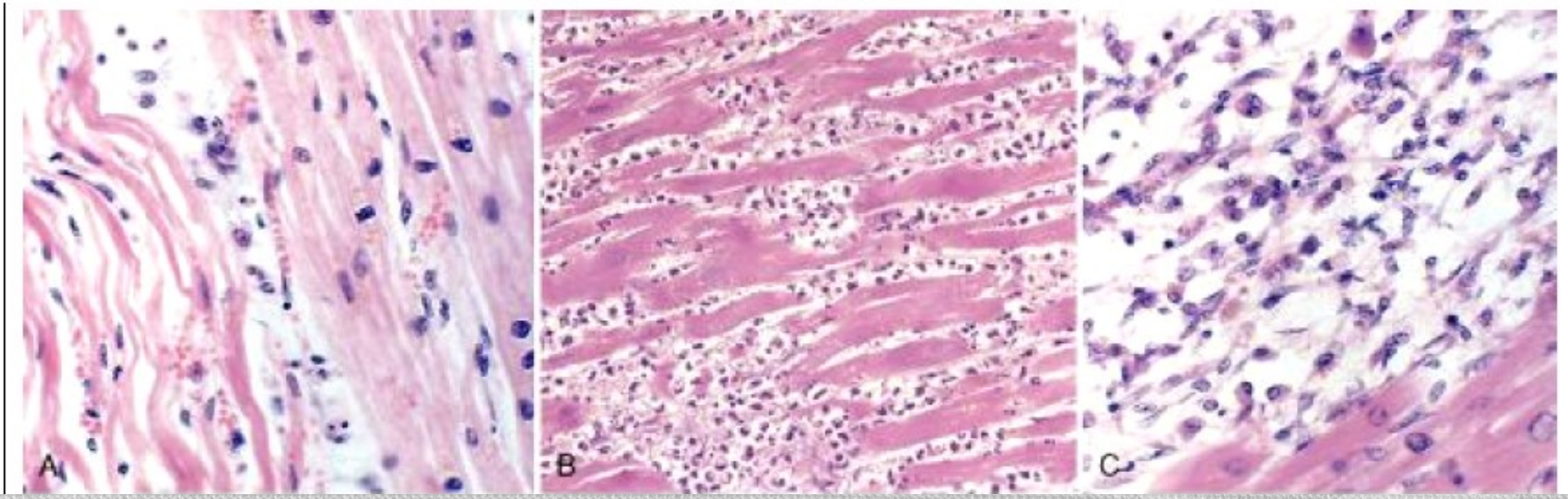
Figure 12-10 Temporal sequence of early biochemical findings and progression of necrosis after onset of severe myocardial ischemia. **A,** Early changes include loss of adenosine triphosphate (ATP) and accumulation of lactate. **B,** For approximately 30 minutes after the onset of even the most severe ischemia, myocardial injury is potentially reversible. Thereafter, progressive loss of viability occurs that is complete by 6 to 12 hours. The benefits of reperfusion are greatest when it is achieved early, and are progressively lost when reperfusion is delayed. (Modified with permission from Antman E: Acute myocardial infarction. In Braunwald E, et al [eds]: Heart Disease: A Textbook of Cardiovascular Medicine, 6th ed. Philadelphia, WB Saunders, 2001, pp 1114-1231.)

DIFFIRENT DAYS

Table 12-5 Evolution of Morphologic Changes in Myocardial Infarction

Time	Gross Features	Light Microscope	Electron Microscope
Reversible Injury			
0-½ hr	None	None	Relaxation of myofibrils; glycogen loss; mitochondrial swelling
Irreversible Injury			
½-4 hr	None	Usually none; variable waviness of fibers at border	Sarcolemmal disruption; mitochondrial amorphous densities
4-12 hr	Dark mottling (occasional)	Early coagulation necrosis; edema; hemorrhage	
12-24 hr	Dark mottling	Ongoing coagulation necrosis; pyknosis of nuclei; myocyte hypereosinophilia; marginal contraction band necrosis; early neutrophilic infiltrate	
1-3 days	Mottling with yellow-tan infarct center	Coagulation necrosis, with loss of nuclei and striations; brisk interstitial infiltrate of neutrophils	
3-7 days	Hyperemic border; central yellow-tan softening	Beginning disintegration of dead myofibers, with dying neutrophils; early phagocytosis of dead cells by macrophages at infarct border	
7-10 days	Maximally yellow-tan and soft, with depressed red-tan margins	Well-developed phagocytosis of dead cells; granulation tissue at margins	
10-14 days	Red-gray depressed infarct borders	Well-established granulation tissue with new blood vessels and collagen deposition	
2-8 wk	Gray-white scar, progressive from border toward core of infarct	Increased collagen deposition, with decreased cellularity	
>2 mo	Scarring complete	Dense collagenous scar	

Microscopic features of myocardial infarction and its repair 1)

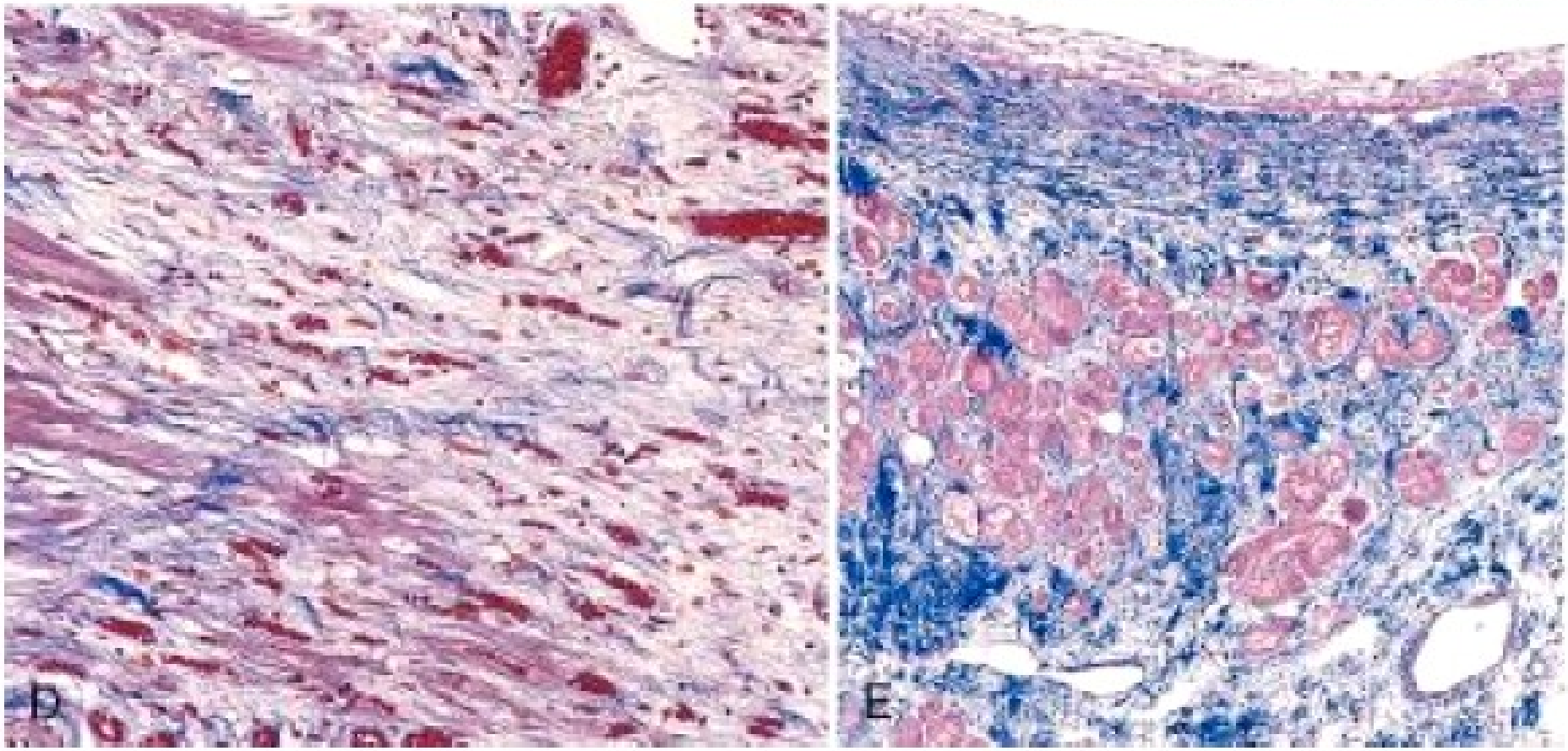


A, One-day-old infarct showing coagulative necrosis and wavy fibers (elongated and narrow, as compared with adjacent normal fibers *at right*). Widened spaces between the dead fibers contain edema fluid and scattered neutrophils.

B, Dense polymorphonuclear leukocytic infiltrate in area of acute myocardial infarction of 3 to 4 days' duration.

C, Nearly complete removal of necrotic myocytes by phagocytosis (approximately 7 to 10 days).

Microscopic features of myocardial infarction and its repair 2)



D, Granulation tissue characterized by loose collagen and abundant capillaries.

E, Well-healed myocardial infarct with replacement of the necrotic fibers by dense collagenous scar. A few residual cardiac muscle cells are present.

Infarct Modification by Reperfusion

he most effective way to “rescue” ischemic myocardium threatened by infarction is to **restore myocardial blood flow** as rapidly as possible

ay also trigger deleterious complications, including arrhythmias,

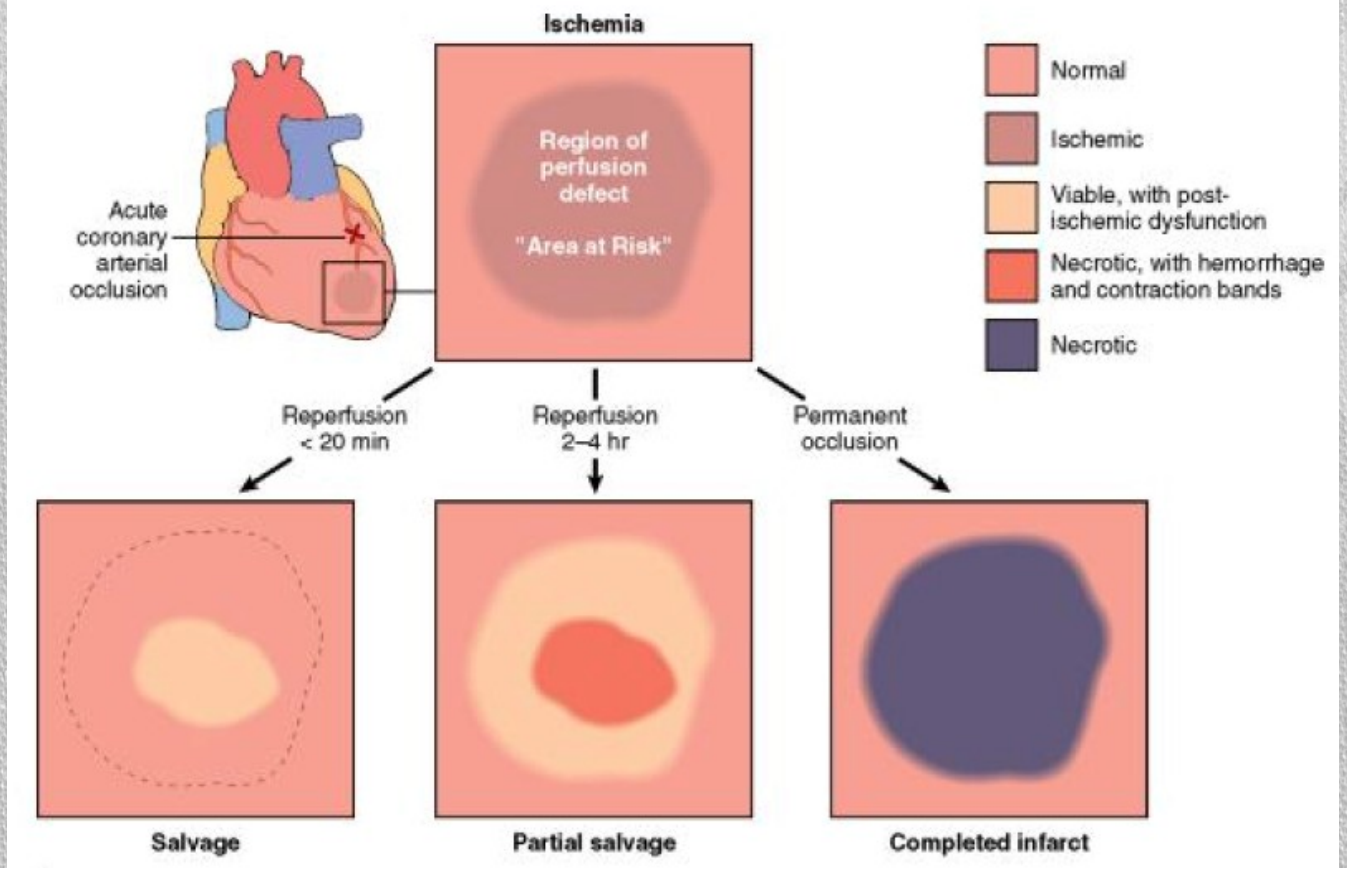
myocardial hemorrhage with contraction bands, irreversible cell

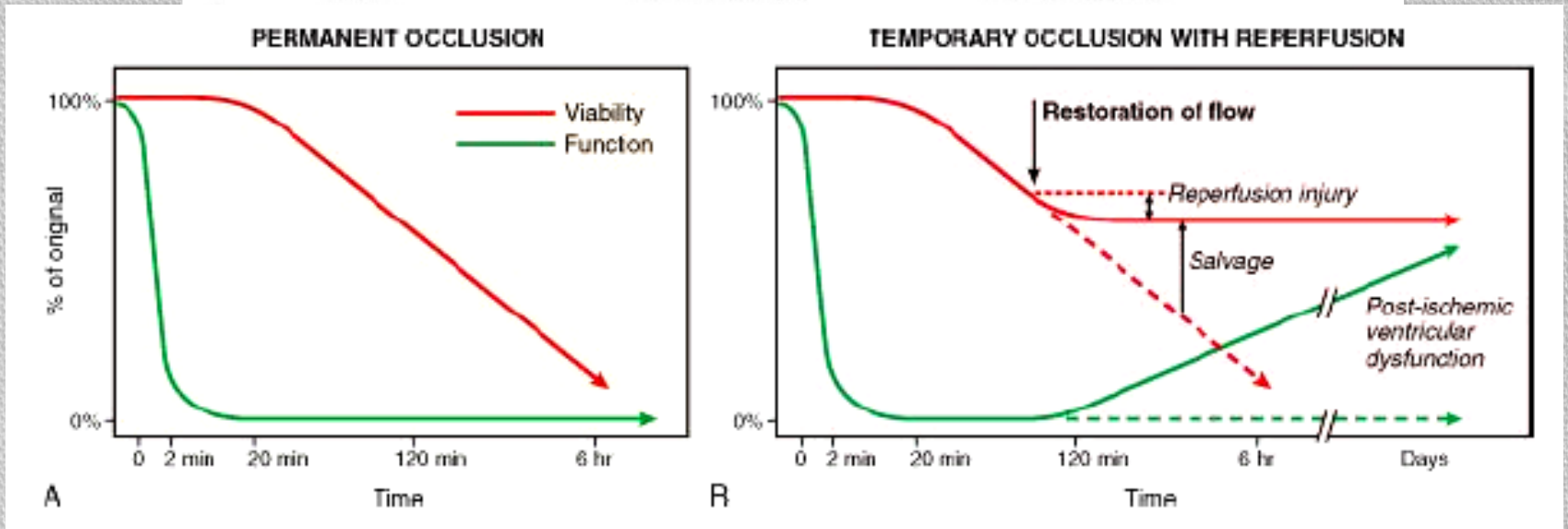
damage superimposed on the original ischemic injury *(reperfusion*

injury), microvascular injury, and prolonged ischemic dysfunction

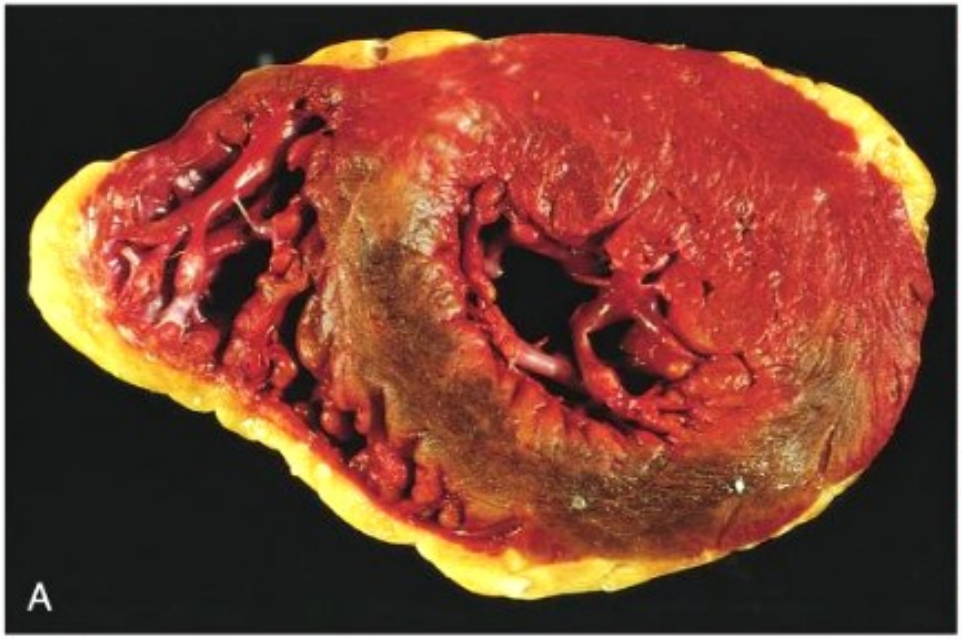
(myocardial stunning)

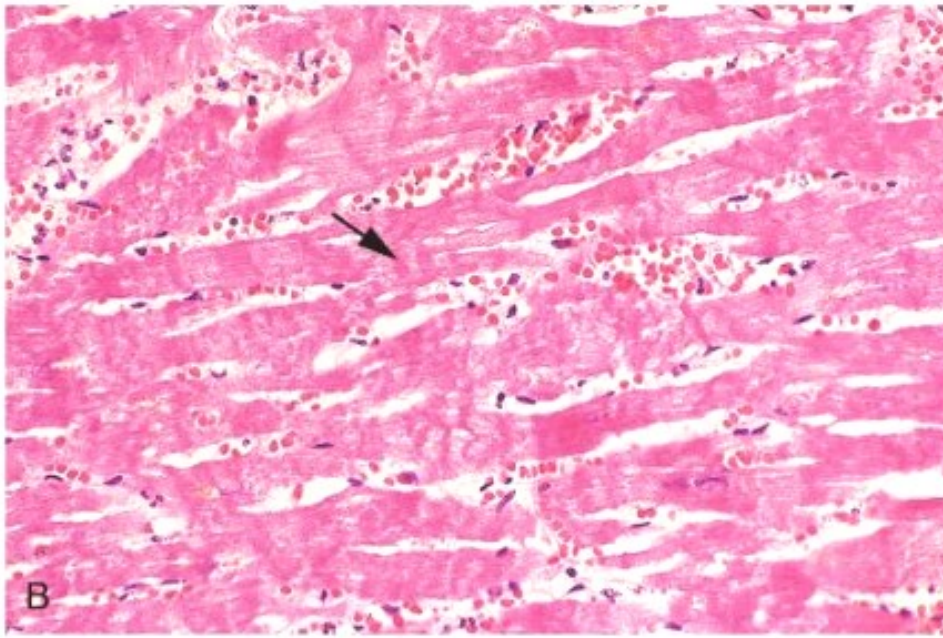
REPE


Ischemia
Acute coronary arterial occlusion
Region of perfusion defect
"Area at Risk"
Normal
Ischemic
Viable, with post-ischemic dysfunction
Necrotic, with hemorrhage and contraction bands
Necrotic
Reperfusion < 20 min
Reperfusion 2–4 hr
Permanent occlusion
Salvage
Partial salvage
Completed infarct


PERMANENT OCCLUSION
100%
0%
% of original
Viability
Function
0
2 min
20 min
120 min
6 hr
Time
A
TEMPORARY OCCLUSION WITH REPERFUSION
100%
0%
Restoration of flow
Reperfusion injury
Salvage
Post-ischemic ventricular dysfunction
0
2 min
20 min
120 min
6 hr
Days
Time
B

REPERFUSION INJURY (IMAGE)

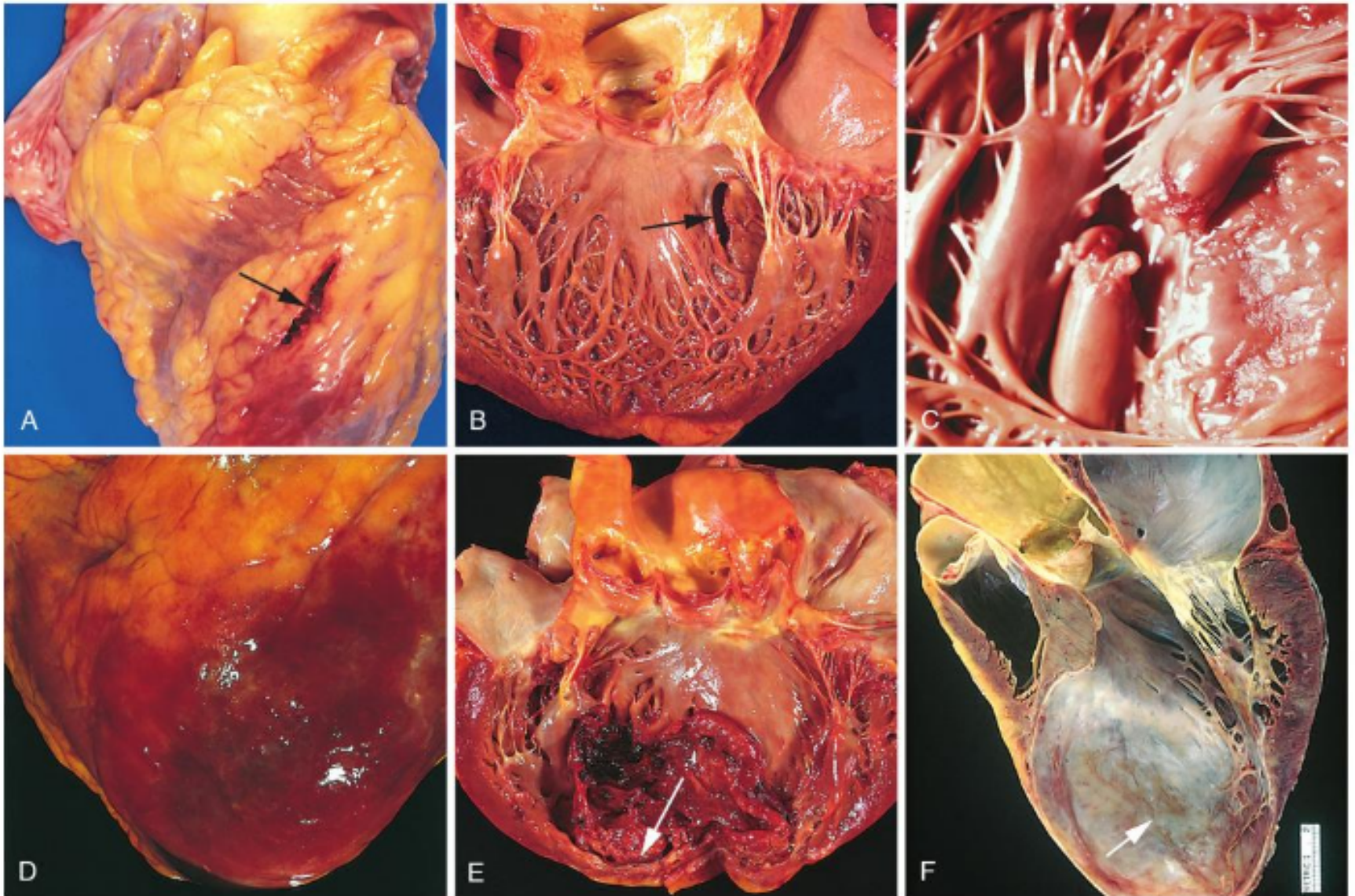




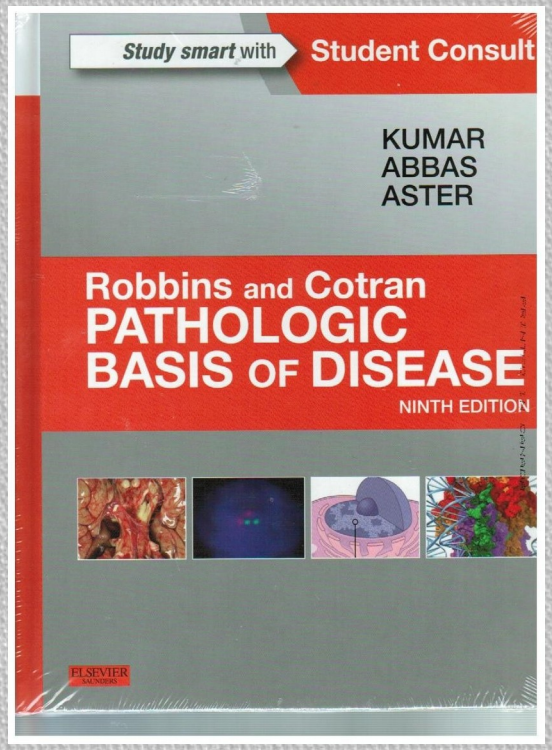
A), Large, densely hemorrhagic, anterior wall acute myocardial infarction in a patient with left anterior descending artery thrombus treated with streptokinase, a fibrinolytic agent (triphenyl tetrazolium chloride–stained heart slice). Specimen oriented with posterior wall at top.

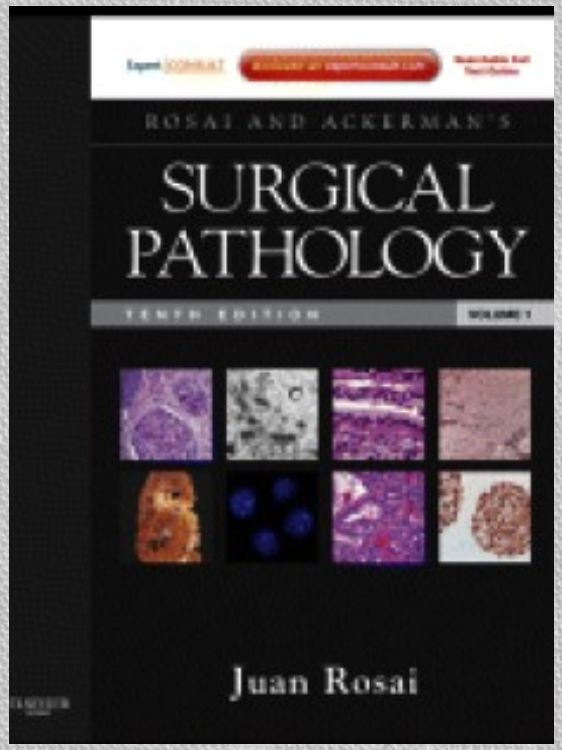
B), Myocardial necrosis with hemorrhage and contraction bands, visible as dark bands spanning some myofibers *(arrow)*. This is the characteristic appearance of markedly ischemic myocardium that has been reperfused.

COMPLICATION FOLLOWING ACUTE MI



Reference


Study smart with
Student Consult
KUMAR
ABBAS
ASTER
Robbins and Cotran
PATHOLOGIC
BASIS OF DISEASE
NINTH EDITION
ELSEVIER


ROSAI AND ACKERMAN'S
SURGICAL
PATHOLOGY
Juan Rosai

ure 12-18 Complications of myocardial infarction. **A,** Anterior myocardial rupture in an acute infarct *(arrow).* **B,** Rupture of the ventricular septum *(arrow).* Complete rupture of a necrotic papillary muscle. **D,** Fibrinous pericarditis, showing a dark, roughened epicardial surface overlying an acute infarct. **E,** Early pansion of anteroapical infarct with wall thinning *(arrow)* and mural thrombus. **F,** Large apical left ventricular aneurysm. The left ventricle is on the right in s apical four-chamber view of the heart. (**A-E,** Reproduced with permission from Schoen FJ: Interventional and Surgical Cardiovascular Pathology: Clinical rrelations and Basic Principles. Philadelphia, WB Saunders, 1989; **F,** Courtesy William D. Edwards, MD, Mayo Clinic, Rochester, Minn.)