REKAP DAFTAR HADIR KULIAH PAKAR & KM BLOK 14/KARDIOVASKULER

SEMESTER GASAL TAHUN AKADEMIK 2020/2021

PERIODE : 21 NOVEMBER - 4 DESEMBER 2020

NO	NAMA DOSEN	DEPARTEMEN	JLH JAM RENCANA	BLOK 14/KARDIOVASKULER				REALISASI KP
				NOVEMBER		DESEMBER		
				24	27	1	4	
1	dr. Frits R. W. Suling, SpJP(K), FIHA, FAScCC	Ilmu Peny. Dalam	12	4	4	4	-	12
2	dr. Danny E. J. Luhulima, SpPK	Pato. Klinik	4	4	-	-	-	4
3	dr. Febtusia Puspitasari, SpJP, FIHA	Ilmu Peny. Dalam	12	-	4	4	4	12
4	dr. Silphia Novelyn, M.Biomed.	Anatomi	4	-	-	4	-	4
5	dr. Chandramin, SpJP(K), FIHA, FAScCC	Ilmu Peny. Dalam	4	4	-	-	-	4
6	dr. Fajar L. Gultom, SpPA	Pato. Anatomi	4	-	-	-	4	4
TOTAL			40					40
PERSENTASI KEHADIRAN KULIAH PAKAR & KM BLOK 14			100%					

Jakarta, 6 Desember 2020

Mengetahui
Manager P2SK,

Dra. Lusia Sri Sunarti, MS

Koordinator Blok 14,

dr. Frits R.W. Suling, SpJP(K), FIHA, FAScCC

Universitas Kristen Indonesia
Fakultas Kedokteran

SURAT KEPUTUSAN
No. : 123/UKI.F5.D/HKP.3.5.6/2020
tentang

PENUGASAN TENAGA AKADEMIK DALAM MEMBERIKAN KULIAH PAKAR PIMPINAN FAKULTAS KEDOKTERAN UNIVERSITAS KRISTEN INDONESIA

MENIMBANG : Bahwa untuk kelancaran proses belajar mengajar dan meningkatkan mutu pendidikan di FKUKI diperlukan penugasan tenaga akademik FKUKI untuk memberikan Kuliah Pakar

MENGINGAT :
1. Peraturan Pemerintah No. 60 tahun 1999 tentang Pendidikan Tinggi
2. Surat Keputusan Dekan FKUKI No. 53/SK/FKUKI/11.2006 tanggal 21 November 2006 tentang Pemberlakuan Kurikulum Berbasis Kompetensi (KBK) di FKUKI
3. Surat Keputusan Rektor UKI No. 90/UKI.R/SK/SDM.8/2018 tentang pengangkatan Dekan Fakultas Kedokteran UKI
4. Surat keputusan pengangkatan sebagai tenaga akademik

MEMUTUSKAN

MENETAPKAN :
1. Penugasan dalam memberikan Kuliah Pakar :

Nama	dr. Fajar Gultom, SpPA
Departemen	Patologi Anatomi
Blok	14 (Sistem Kardiovaskular)
Judul Materi	PA Sistem Kardiovaskular
Semester	Gasal 2020/2021
Kelas	A : 0,21 SKS
	B : 0,21 SKS
SKS	0,42 SKS

2. Apabila dikemudian hari ternyata terdapat kekeliruan dalam Surat Keputusan ini akan diperbaiki sebagaimana mestinya

Asli Surat Keputusan ini disampaikan kepada yang bersangkutan untuk diketahui

Ditetapkan di : Jakarta
Pada tanggal : 10 September 2020
Dekan,

Dr. dr. Robert Hotman Sirait, Sp.An.
NIP. UKI. 031 545

Tembusan:
1. Rektor UKI
2. Wakil Dekan Bidang Akademik FKUKI

Jl. Mayjen Sutoyo no.2 Cawang - Jakarta 13630 INDONESIA Tel. 021.29362033,Tel. Langsung 021.29362038, Faks. 021.29362036 E-mail: fk-uki@uki.ac.id http://www.uki.ac.id

KardioVaskular

Fajar L. Gultom

Departemen Patologi Anatomik – FK UKI

November 2020

Gangguan dan Kelainan pada Jantung		
1	Kelainan jantung congenital (*Ventricular Septal Defect, Atrial Septal Defect, Patent Ductus Arteriosus, Tetralogy of Fallot*)	2
2	Radang pada dinding jantung (Endokarditis, Miokarditis, Perikarditis)	2
3	Syok (septik, hipovolemik, kardiogenik, neurogenik)	3B
4	Angina pektoris	3B
5	Infark miokard	3B
6	Gagal jantung akut	3B
7	Gagal jantung kronik	3A
8	*Cardiorespiratory arrest*	3b
9	Kelainan katup jantung: *Mitral stenosis, Mitral regurgitation, Aortic stenosis, Aortic regurgitation*,dan Penyakit katup jantung lainnya	2
10	Takikardi: supraventrikular, ventrikular	3B
11	Fibrilasi atrial	3A
12	Fibrilasi ventrikular	3B
13	*Atrial flutter*	3B
14	Ekstrasistol supraventrikular, ventrikular	3A
15	*Bundle Branch Block*	2
16	Aritmia lainnya	2
17	Kardiomiopati	2
18	Kor pulmonale akut	3B
19	Kor pulmonale kronik	3A

Gangguan Aorta dan Arteri		
20	Hipertensi esensial	4A
21	Hipertensi sekunder	3A
22	Hipertensi pulmoner	1
23	Penyakit Raynaud	2
24	Trombosis arteri	2
25	Koarktasio aorta	1
26	Penyakit Buerger's (*Thromboangiitis Obliterans*)	2
27	Emboli arteri	1
28	Aterosklerosis	1
29	*Subclavian steal syndrome*	1
30	Aneurisma Aorta	1
31	Aneurisma diseksi	1
32	Klaudikasio	2
33	Penyakit jantung reumatik	2

Vena* dan *Pembuluh Limfe		
34	Tromboflebitis	3A
35	Limfangitis	3A
36	Varises (primer, sekunder)	2
37	*Obstructed venous return*	2
38	Trombosis vena dalam	2
39	Emboli vena	2
40	Limfedema (primer, sekunder)	3A
41	Insufisiensi vena kronik	3A

Recall..
Please help

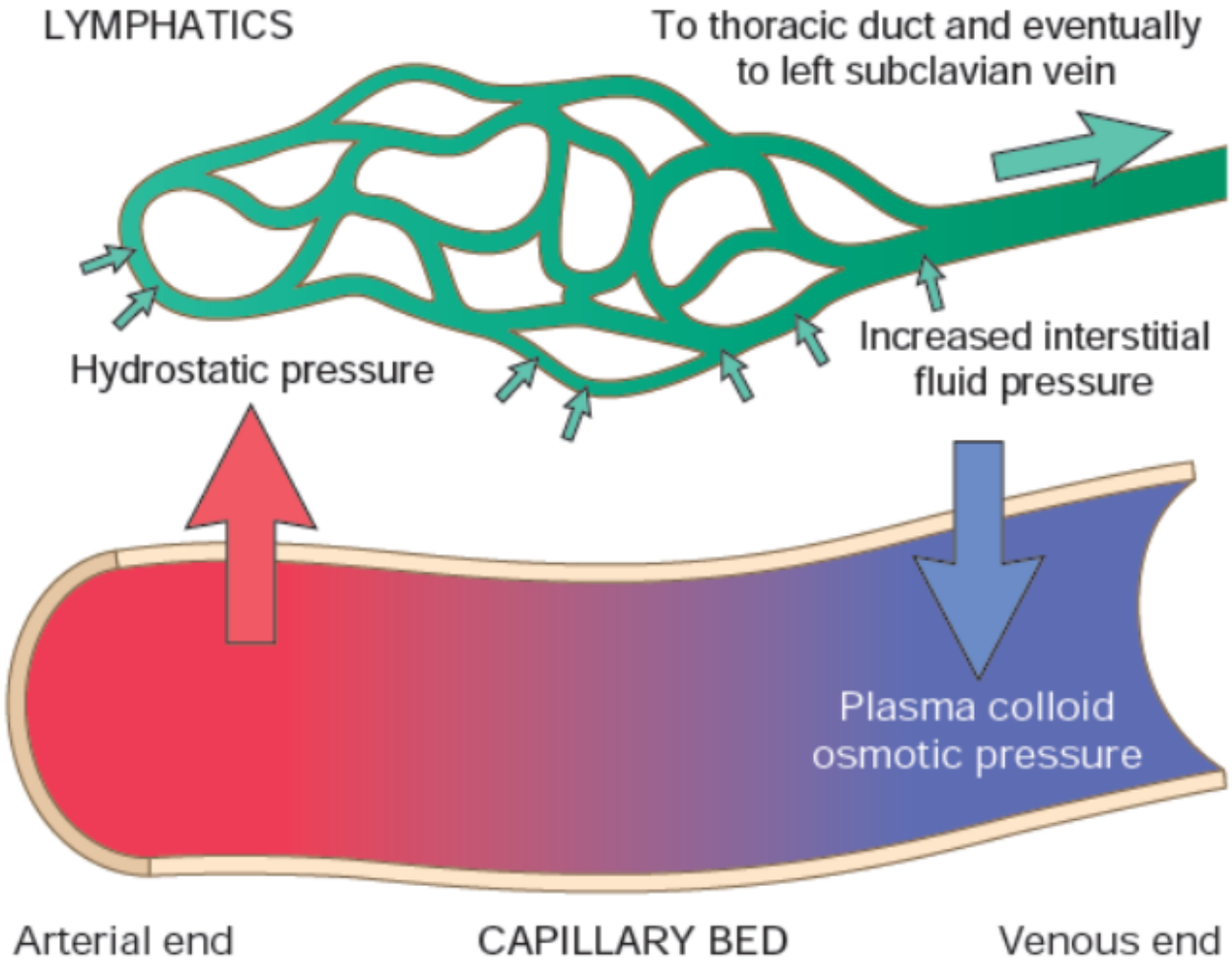


Figure 4-1 Factors influencing fluid movement across capillary walls. Normally, hydrostatic and osmotic forces are nearly balanced so that there is little net movement of fluid out of vessels. Many different pathologic disorders (Table 4-1) are associated with increases in capillary hydrostatic pressure or decreases in plasma osmotic pressure that lead to the extravasation of fluid into tissues. Lymphatic vessels remove much of the excess fluid, but if the capacity for lymphatic drainage is exceeded, tissue edema results.

Robbins, Pathologic Basis of Disease 9th ed, 2015

Table 4-1 Pathophysiologic Categories of Edema

Increased Hydrostatic Pressure
Impaired Venous Return
Congestive heart failure
Constrictive pericarditis
Ascites (liver cirrhosis)
Venous obstruction or compression
Thrombosis
External pressure (e.g., mass)
Lower extremity inactivity with prolonged dependency
Arteriolar Dilation
Heat
Neurohumoral dysregulation
Reduced Plasma Osmotic Pressure (Hypoproteinemia)
Protein-losing glomerulopathies (nephrotic syndrome)
Liver cirrhosis (ascites)
Malnutrition
Protein-losing gastroenteropathy
Lymphatic Obstruction
Inflammatory
Neoplastic
Postsurgical
Postirradiation
Sodium Retention
Excessive salt intake with renal insufficiency
Increased tubular reabsorption of sodium
Renal hypoperfusion
Increased renin-angiotensin-aldosterone secretion
Inflammation
Acute inflammation
Chronic inflammation
Angiogenesis

Modified from Leaf A, Cotran RS. Renal pathophysiology, 3rd ed. New York, Oxford University Press, 1985, p 146.

Robbins, Pathologic Basis of Disease 9th ed, 2015

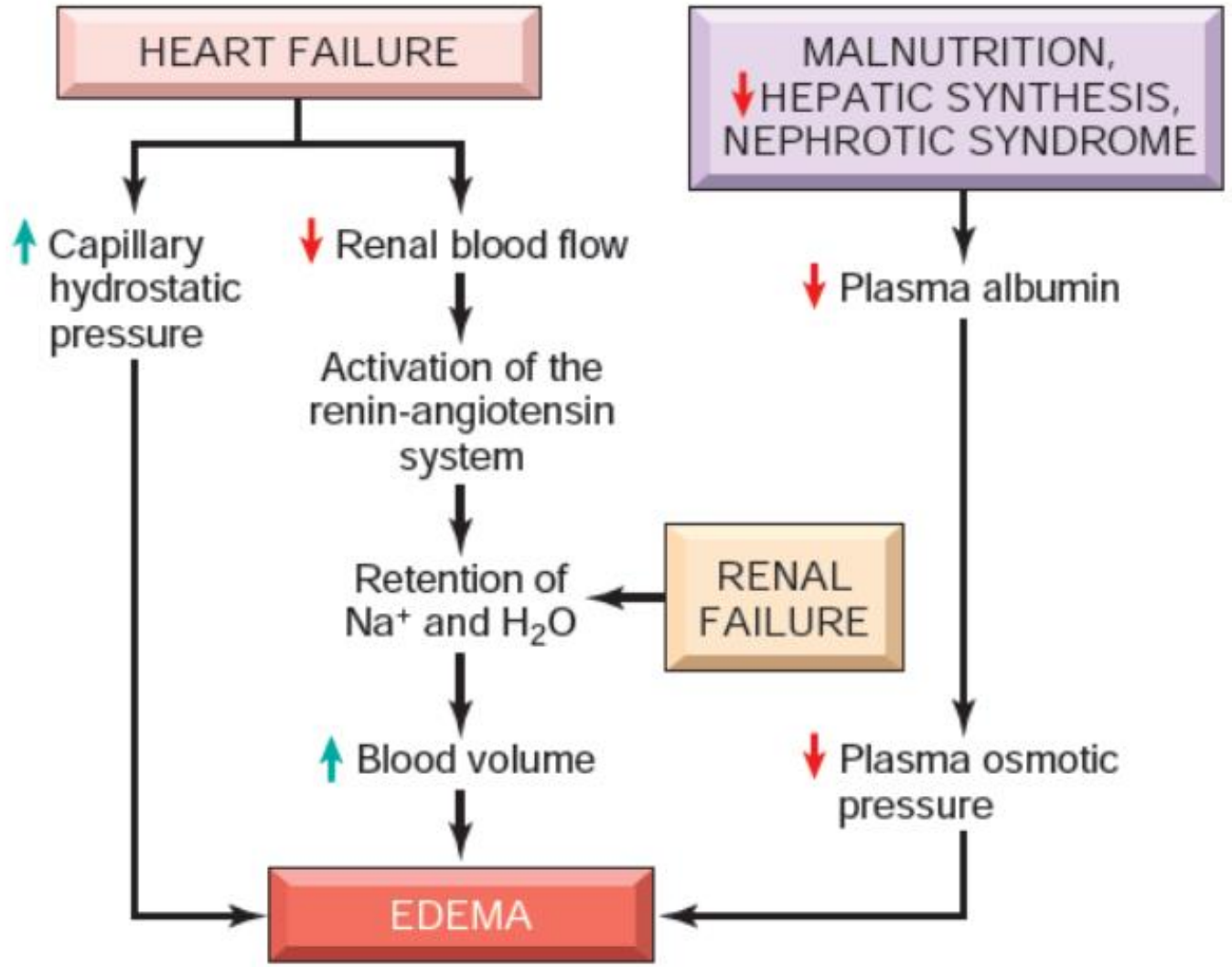


Figure 4-2 Mechanisms of systemic edema in heart failure, renal failure, malnutrition, hepatic failure, and nephrotic syndrome.

Robbins, Pathologic Basis of Disease 9th ed, 2015

Shock → definition

Shock

Shock is a life-threatening condition that occurs when the body is not getting enough blood flow. Lack of blood flow means that the cells and organs do not get enough oxygen and nutrients to function properly. Many organs can be damaged as a result. Shock requires immediate treatment and can get worse very rapidly. As many 1 in 5 people who suffer shock will die from it.

Syok

Gangguan metabolik dan hemodinamik yang ekstrem akibat kegagalan sistem sirkulasi untuk mempertahankan perfusi yang adekuat pada organ-organ vital.

Kamus Saku Kedokteran Dorlan edisi 29, 2015

Shock

- Diminished cardiac output – reduced effective circulating blood volume.
- Impairs tissue perfusion → cellular hypoxia.
  - Cardiogenic shock.
  - Hypovolemic shock.
  - Shock associated with systemic inflammation → SIRS → Sepsis shock
  - Neurogenic shock → spinal cord injury
  - Anaphylactic shock → Ig E mediated hypersensitivity rx

Table 4-3 Three Major Types of Shock

Type of Shock	Clinical Example	Principal Mechanisms
Cardiogenic	Myocardial infarction Ventricular rupture Arrhythmia Cardiac tamponade Pulmonary embolism	Failure of myocardial pump resulting from intrinsic myocardial damage, extrinsic compression, or obstruction to outflow
Hypovolemic	Fluid loss (e.g., hemorrhage, vomiting, diarrhea, burns, or trauma)	Inadequate blood or plasma volume
Shock associated with systemic inflammation	Overwhelming microbial infections (bacterial and fungal) Superantigens (e.g., toxic shock syndrome) Trauma, burns, pancreatitis	Activation of cytokine cascades; peripheral vasodilation and pooling of blood; endothelial activation/injury; leukocyte-induced damage, disseminated intravascular coagulation

Robbins, Pathologic Basis of Disease 9th ed, 2015

Phases of Shock

- Non-progressive
  Reflex compensatory mechanism – perfusion vital organ.
  Tachycardia, peripheral vasoconstriction, renal conservation of fluid.

- Progressive
  Tissue hypoperfusion – circulatory, metabolic imbalances → lactic acidosis.

- Irreversible

Cardiovascular dysfunction

1. Pump failure
2. Flow obstruction: atherosclerotic plaque
3. Regurgitant flow: flows backward → aortic regurgitation
4. Shunted flow: defects congenital or acquired
5. Disorders of cardiac conduction: atrial/ ventricular fibrillation
6. Rupture of the heart/ major vessel: aortic dissection

Ischemic Heart Disease (IHD)

- Angina pectoris
- Myocardial infarction (MI)
- Chronic IHD with heart failure
- Sudden cardiac death

Angina Pectoris

- Paroxysmal, recurrent attack substernal/ precordial chest discomfort.
- Caused by transient (15s – 15 mnts) myocardial ischemia.
- Insufficient to induce myocyte necrosis.

- Nyeri paroksismal pada dada, seringkali menyebar ke lengan, tu. lengan kiri, akibat gangguan pasokan oksigen ke otot jantung, dipicu oleh rasa bersemangat atau kerja

(Kamus Saku Kedokteran Dorlan edisi 29, 2015)

Angina Pectoris (AP)

- Pain → ischemia-induced release of adenosine, bradikynine stimulate sympathetic and vagal afferent nerves.
- Combinations of decreased perfusion, increased demand and coronary arterial pathology.
- Silent ischemia → geriatric and diabetic patients.
- Stable, Prinzmetal and Unstable AP.

Myocardial Infarction (MI)

- “heart attack”, death of cardiac muscle due to prolonged severe ischemia.
- Virtually any age, 10% < 40 yrs, 45% < 65 yrs.
- Genetic and behavioral predisposition to **atherosclerosis**.

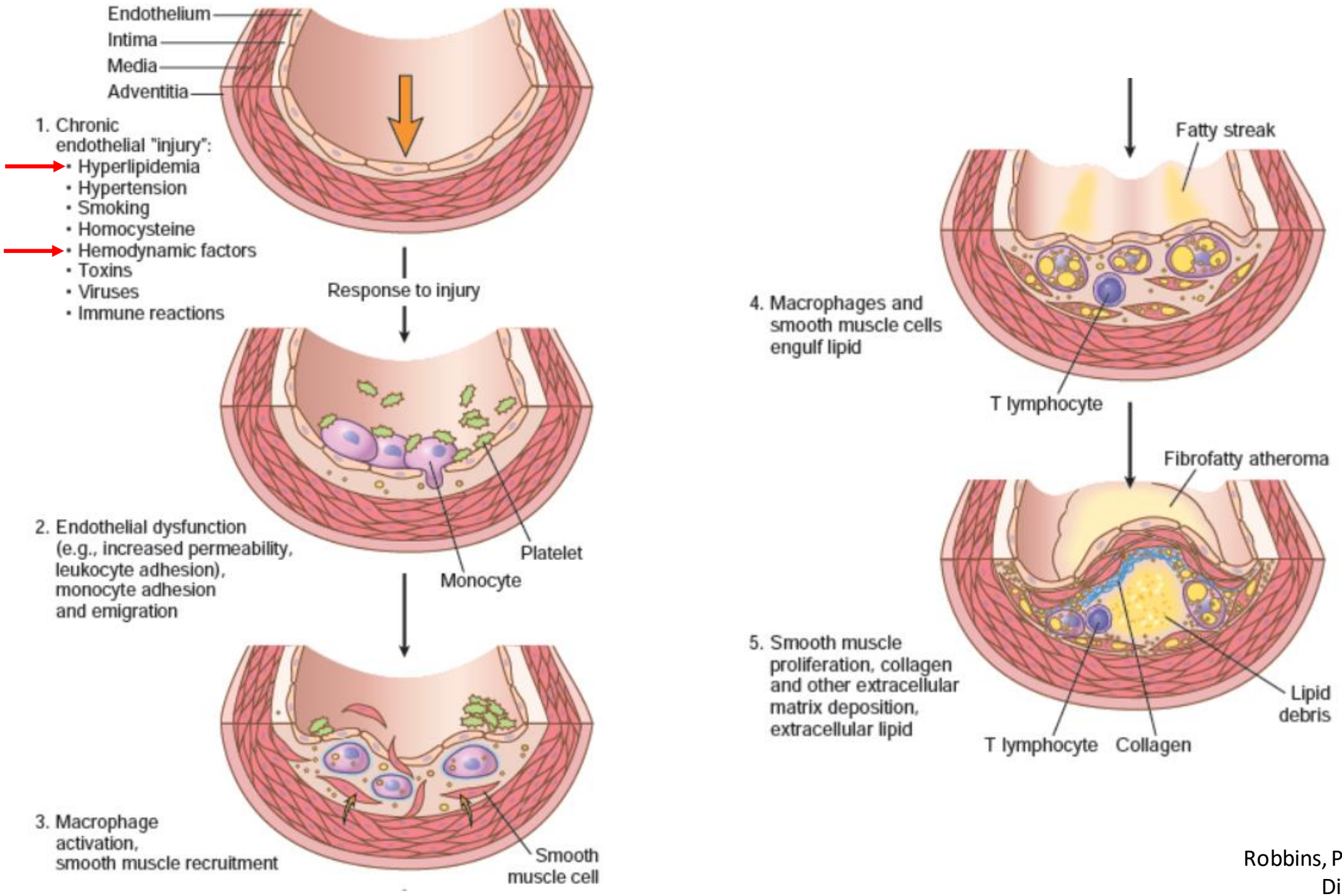
Atherosclerosis

- Satu bentuk arteriosclerosis dengan atheroma yang mengandung kolesterol, bahan lipid, dan lipofag terbentuk di dalam lapisan intima dan bagian dalam lapisan media arteri ukuran besar dan sedang.

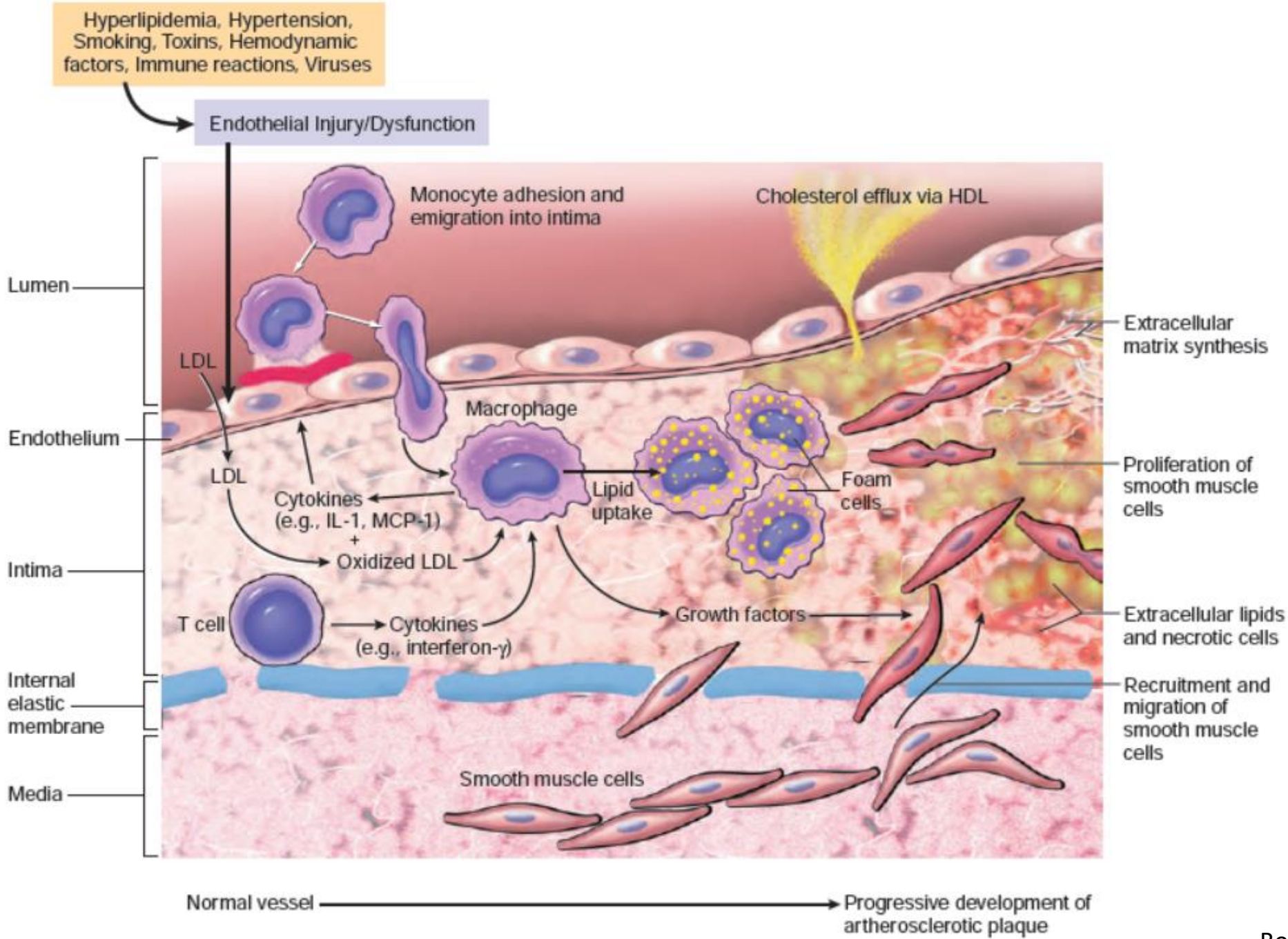
(Kamus Saku Kedokteran Dorlan edisi 29, 2015)

Pathogenesis

- **Endothelial injury and dysfunction**: vascular permeability >>, leucocyte adhesion, thrombosis.
- **Accumulation of lipoproteins** in the vessel wall: LDL and LDLox.
- **Monocyte adhesion to endothel**: migration into intima → transformation into macrophages and foam cells.
- **Platelet adhesion**.
- **Factor release from activated platelets, macrophages** → inducing smooth muscle recruitment.
- **Smooth muscle proliferation, extracellular matrix production and recruitment T cells**.
- **Lipid accumulation**



Robbins, Pathologic Basis of Disease 9th ed, 2015



Robbins, Pathologic Basis of Disease 9th ed, 2015

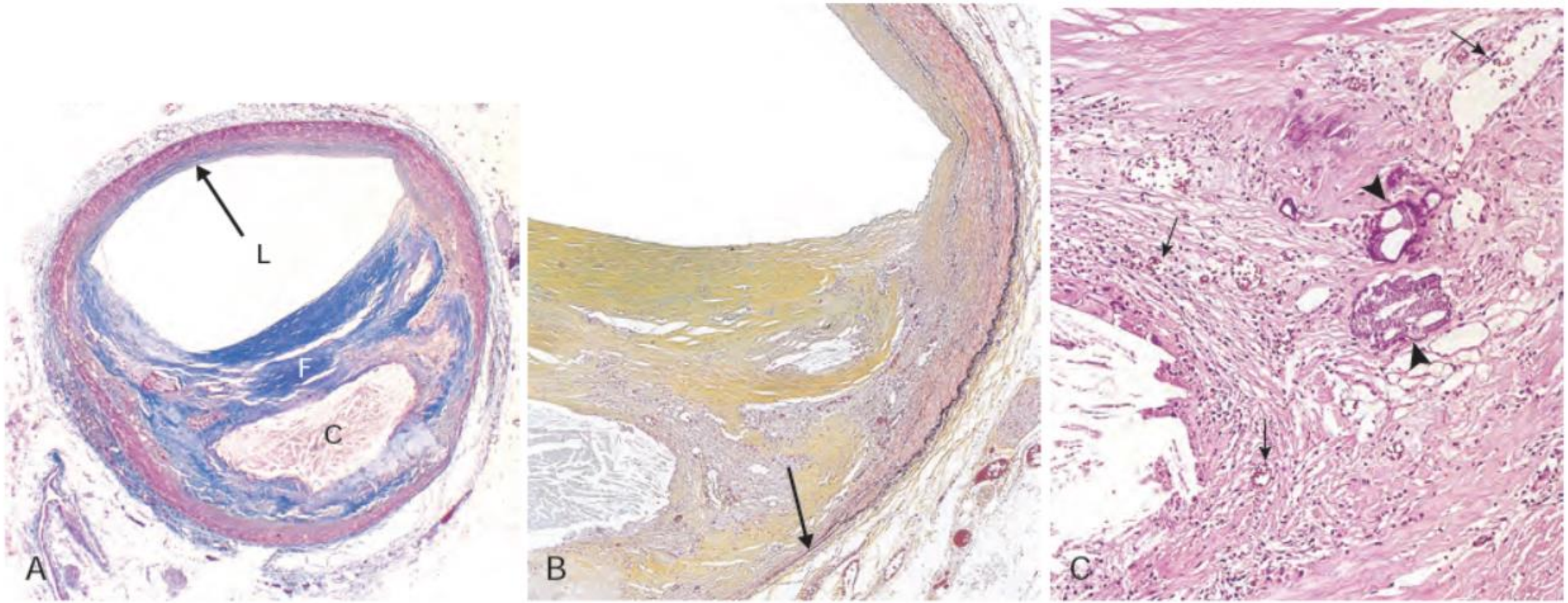


Figure 11-14 Histologic features of atheromatous plaque in the coronary artery. **A,** Overall architecture demonstrating fibrous cap (F) and a central necrotic core (C) containing cholesterol and other lipids. The lumen (L) has been moderately compromised. Note that a segment of the wall is plaque free *(arrow)*; the lesion is therefore "eccentric." In this section, collagen has been stained blue (Masson trichrome stain). **B,** Higher-power photograph of a section of the plaque shown in **A,** stained for elastin (black), demonstrating that the internal and external elastic laminae are attenuated and the media of the artery is thinned under the most advanced plaque *(arrow)*. **C**, Higher magnification photomicrograph at the junction of the fibrous cap and core, showing scattered inflammatory cells, calcification *(arrowhead)*, and neovascularization *(small arrows)*.

Robbins, Pathologic Basis of Disease 9th ed, 2015

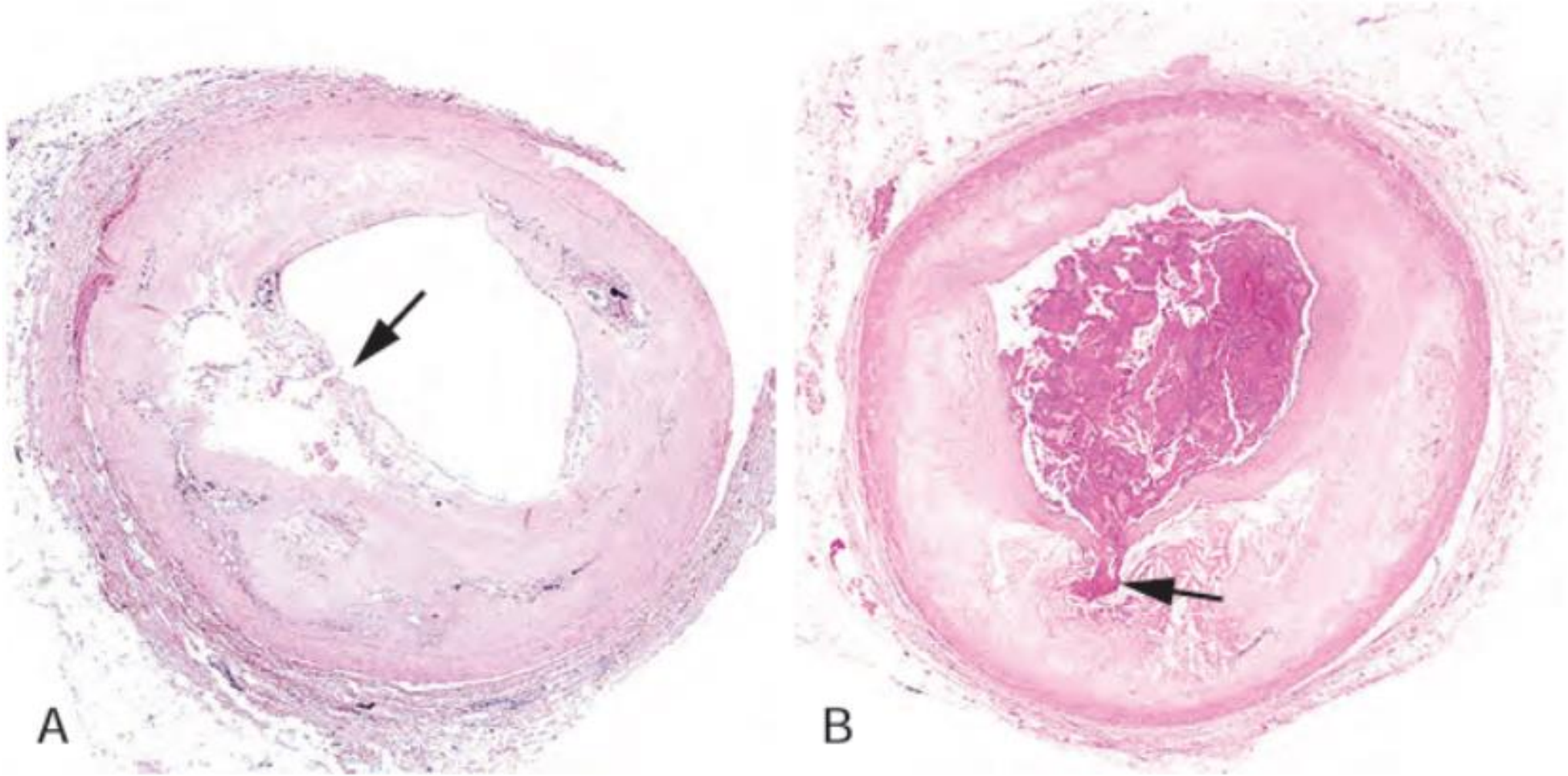


Figure 11-15 Atherosclerotic plaque rupture. **A,** Plaque rupture without superimposed thrombus, in a patient who died suddenly. **B,** Acute coronary thrombosis superimposed on an atherosclerotic plaque with focal disruption of the fibrous cap, triggering fatal myocardial infarction. In both **A** and **B,** an *arrow* points to the site of plaque rupture. (**B,** Reproduced from Schoen FJ: Interventional and Surgical Cardiovascular Pathorosclerosisology: Clinical Correlations and Basic Principles. Philadelphia, WB Saunders, 1989, p 61.)

Robbins, Pathologic Basis of Disease 9th ed, 2015

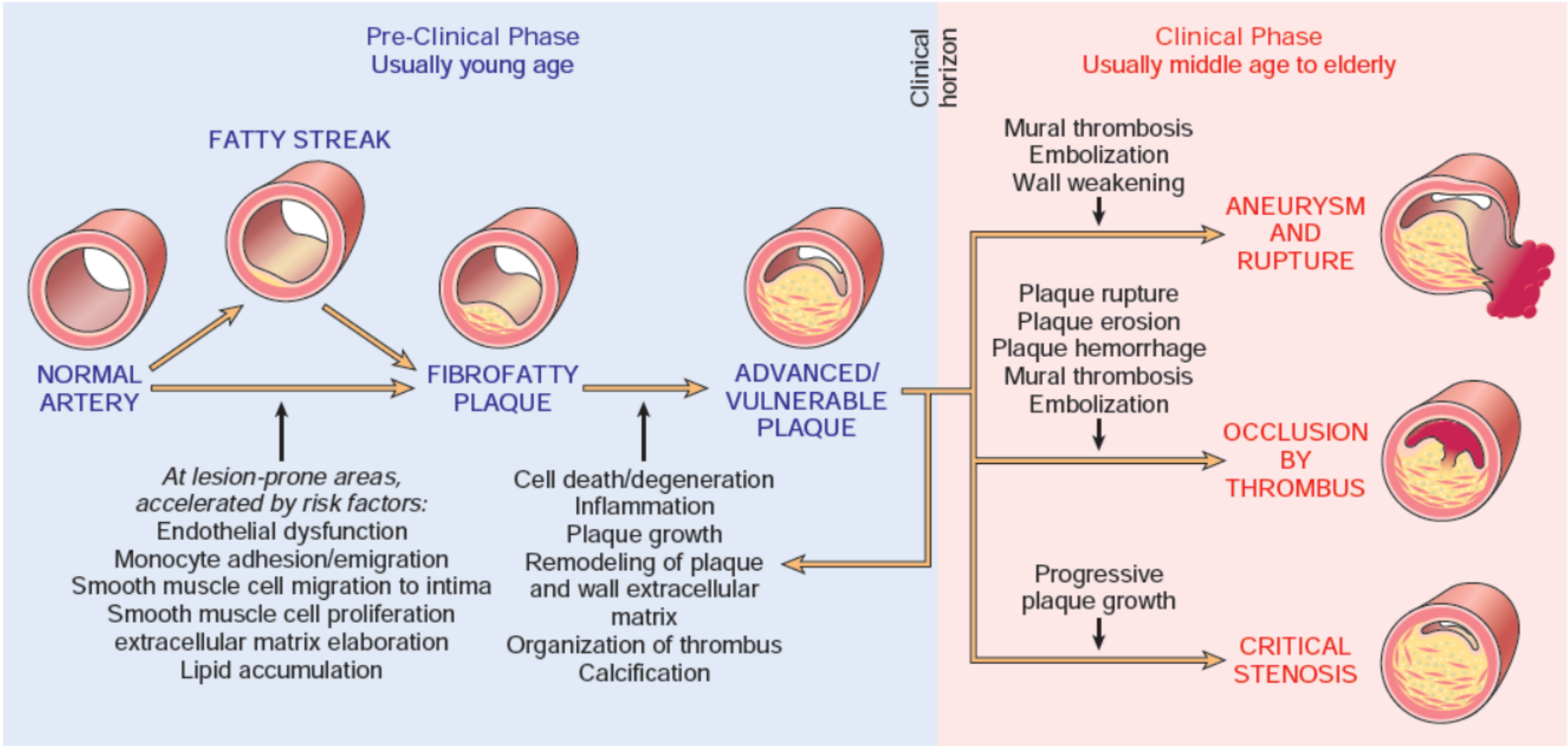


Figure 11-16 The natural history, morphologic features, main pathogenic events, and clinical complications of atherosclerosis.

Robbins, Pathologic Basis of Disease 9th ed, 2015

Myocardial Infarction (MI)

Pathogenesis:

Coronary artery occlusion

- Atheromatous plaque → acute change → intraplaque haemorrhage, erosion, ulceration, rupture/ fissure.
- Aggregate to microthrombi.
- Vasospasm stimulated by mediators.
- Tissue factor → coagulation pathway → bulk of thrombi.
- Thrombus expand completely occlude lumen.

Myocardial Infarction (MI)

Myocardial response

- Coronary arterial obstruction → blood flow << → ischemia → rapid myocardial dysfunction → myocyte death.
- Severe ischemia: blood flow ≤ 10% 20-30 mnts → irreversible damage.
- Early coronary intervention → reperfusion and salvage.

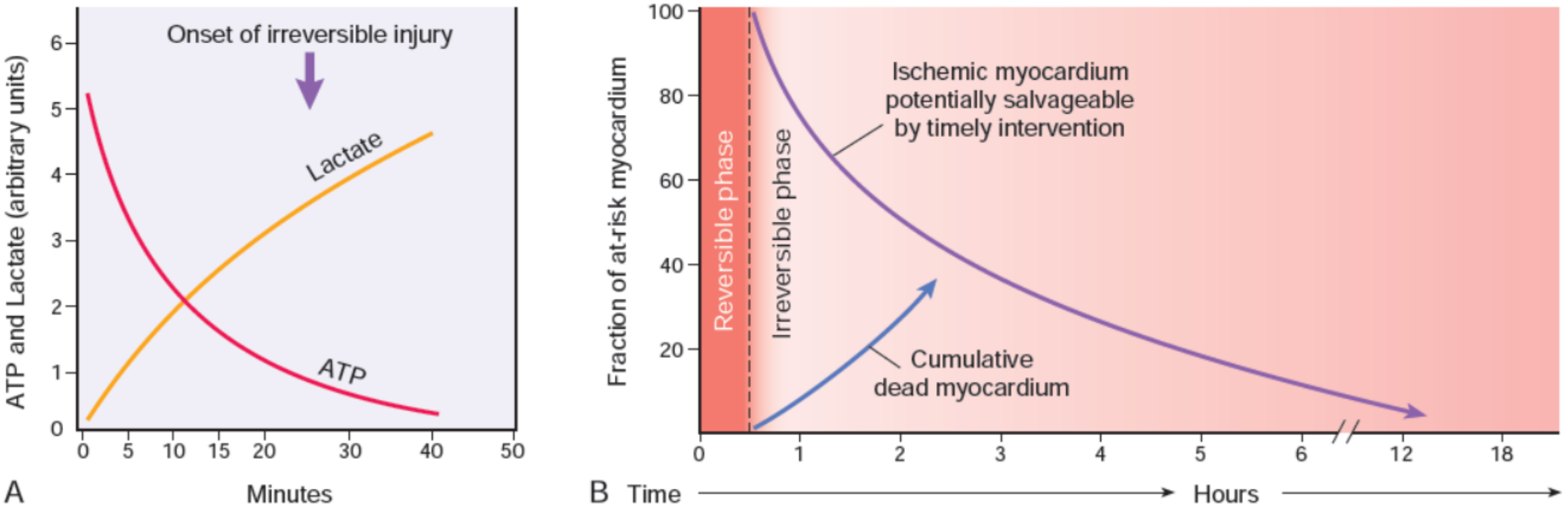
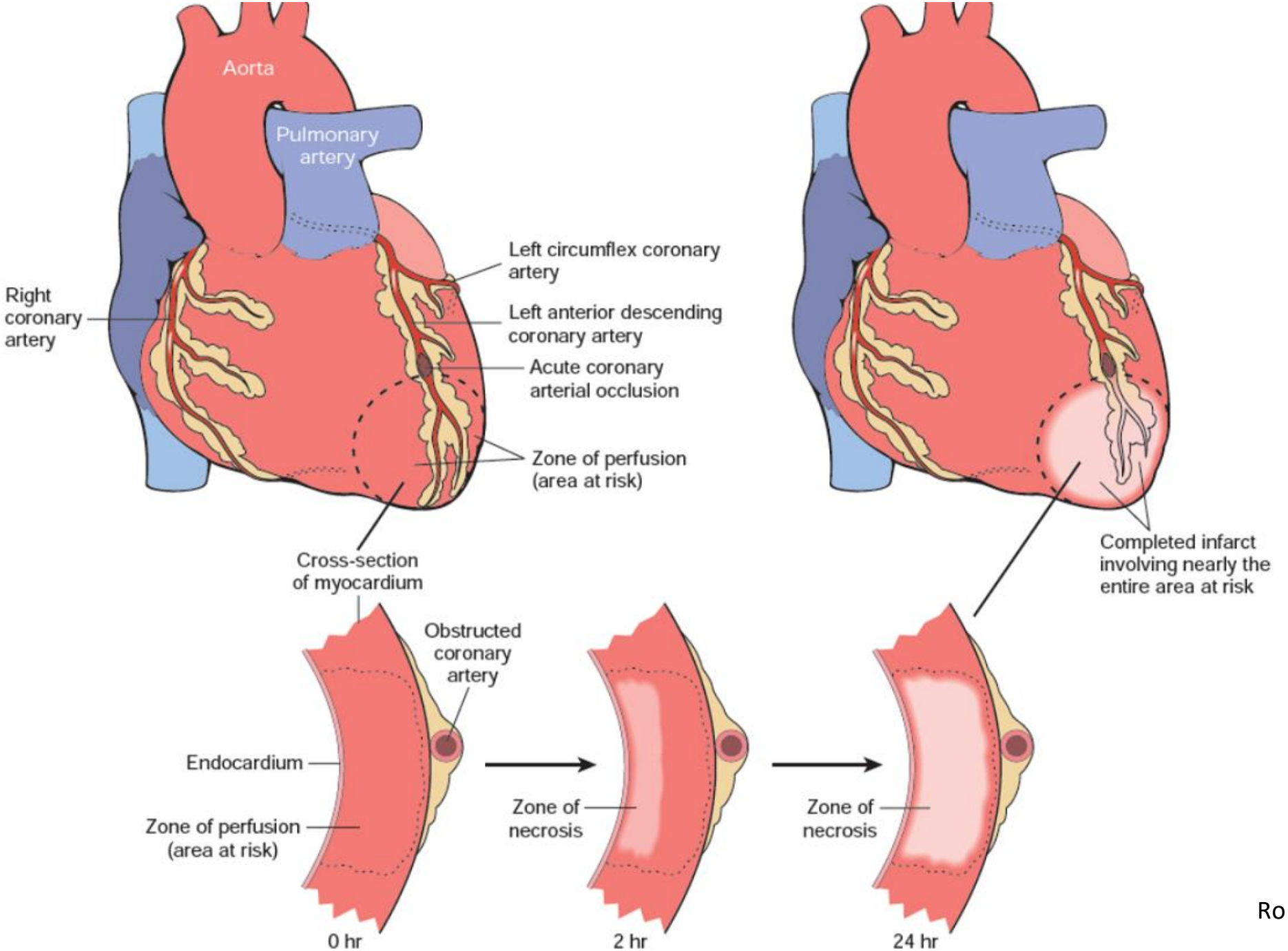


Figure 12-10 Temporal sequence of early biochemical findings and progression of necrosis after onset of severe myocardial ischemia. **A,** Early changes include loss of adenosine triphosphate (ATP) and accumulation of lactate. **B,** For approximately 30 minutes after the onset of even the most severe 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.)

Robbins, Pathologic Basis of Disease 9th ed, 2015



Robbins, Pathologic Basis of Disease 9th ed, 2015

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	

Robbins, Pathologic Basis of Disease 9th ed, 2015

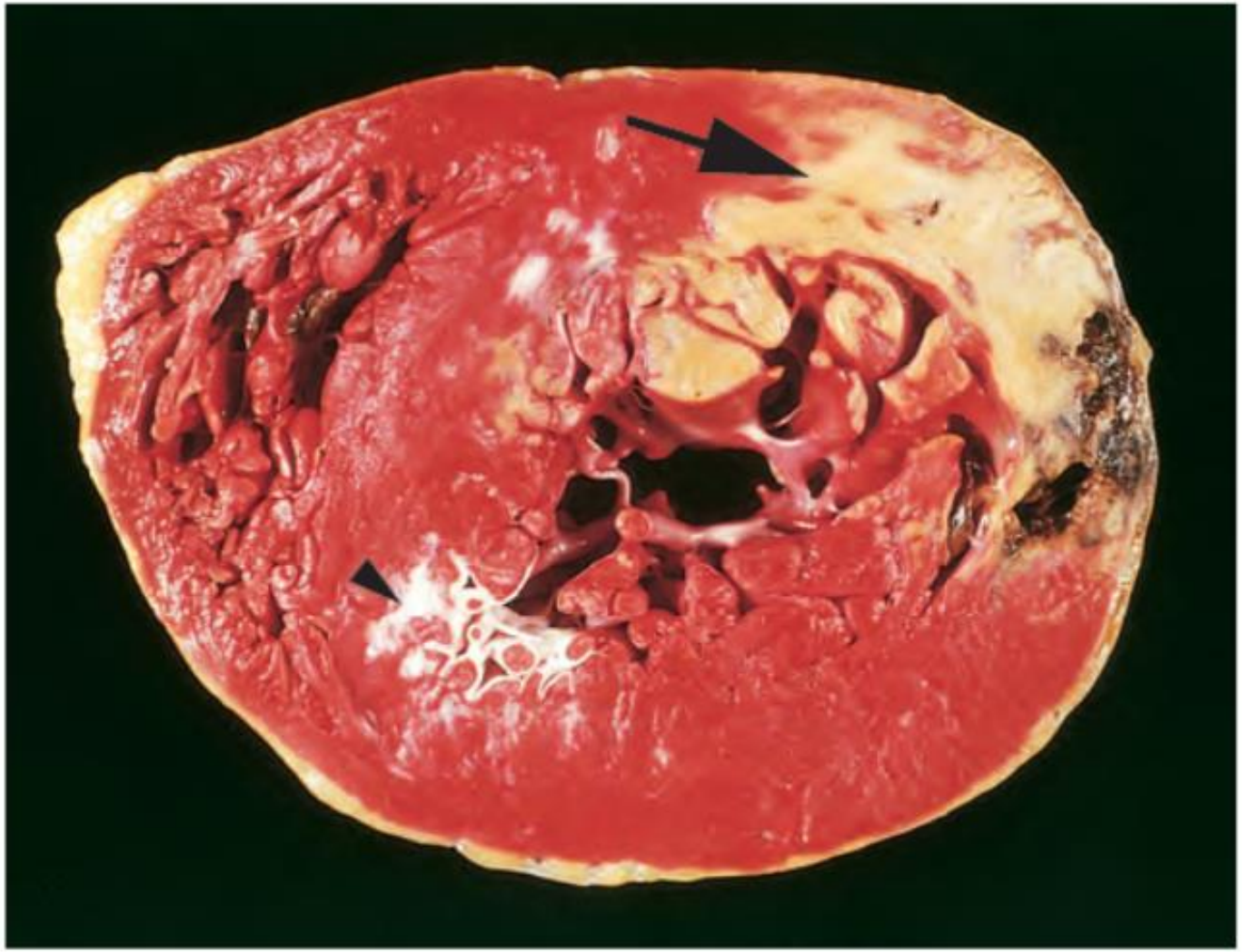


Figure 12-13 Acute myocardial infarct, predominantly of the posterolateral left ventricle, demonstrated histochemically by a lack of staining by triphenyltetrazolium chloride in areas of necrosis *(arrow)*. The staining defect is due to the lactate dehydrogenase leakage that follows cell death. Note the myocardial hemorrhage at one edge of the infarct that was associated with cardiac rupture, and the anterior scar *(arrowhead)*, indicative of old infarct. Specimen is oriented with the posterior wall at the top.

Robbins, Pathologic Basis of Disease 9th ed, 2015

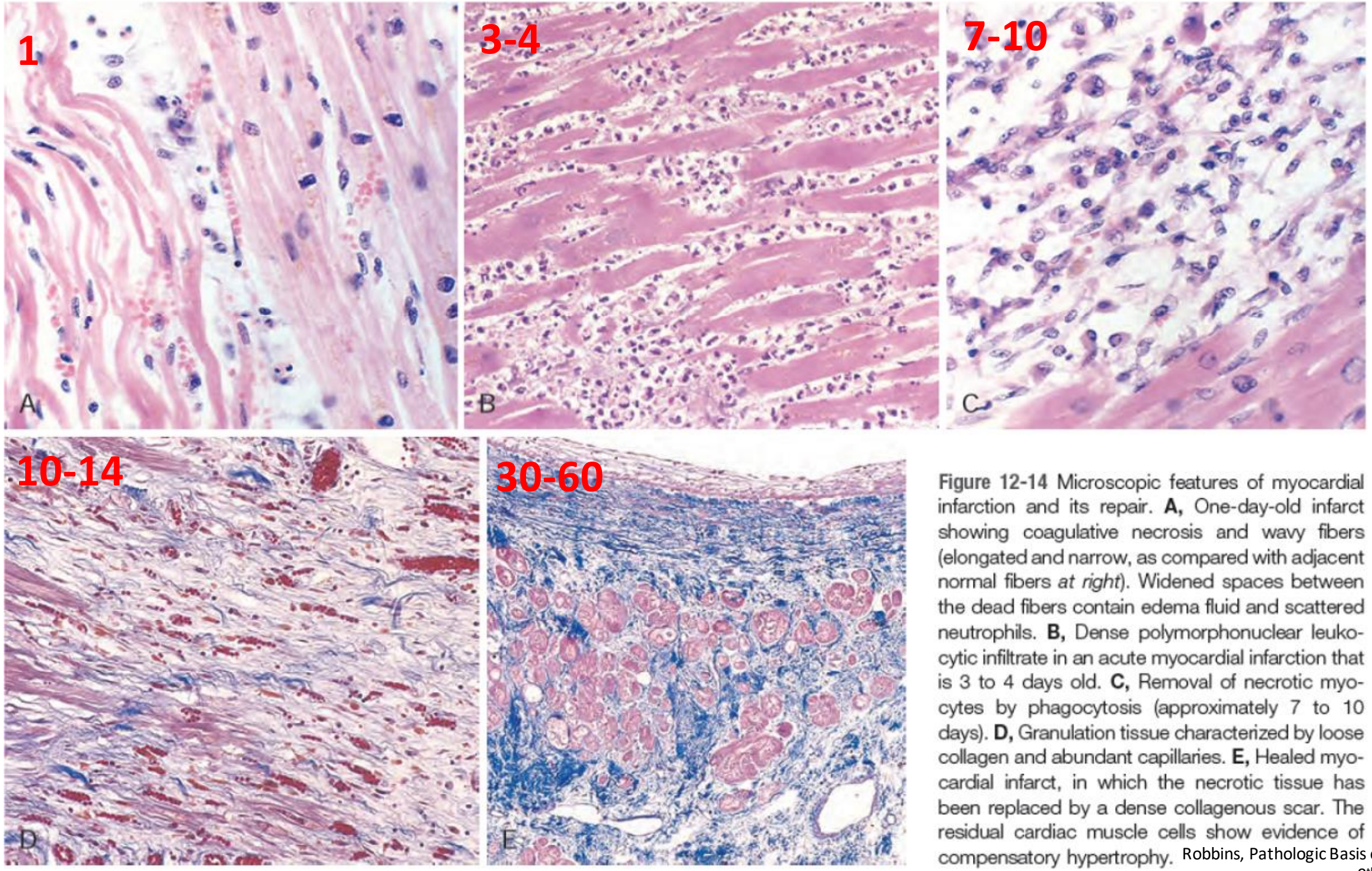


Figure 12-14 Microscopic features of myocardial infarction and its repair. **A,** One-day-old infarct showing coagulative necrosis and wavy fibers (elongated and narrow, as compared with adjacent normal fibers *at right*). Widened spaces between the dead fibers contain edema fluid and scattered neutrophils. **B,** Dense polymorphonuclear leukocytic infiltrate in an acute myocardial infarction that is 3 to 4 days old. **C,** Removal of necrotic myocytes by phagocytosis (approximately 7 to 10 days). **D,** Granulation tissue characterized by loose collagen and abundant capillaries. **E,** Healed myocardial infarct, in which the necrotic tissue has been replaced by a dense collagenous scar. The residual cardiac muscle cells show evidence of compensatory hypertrophy. Robbins, Pathologic Basis of Disease 9th ed. 2015

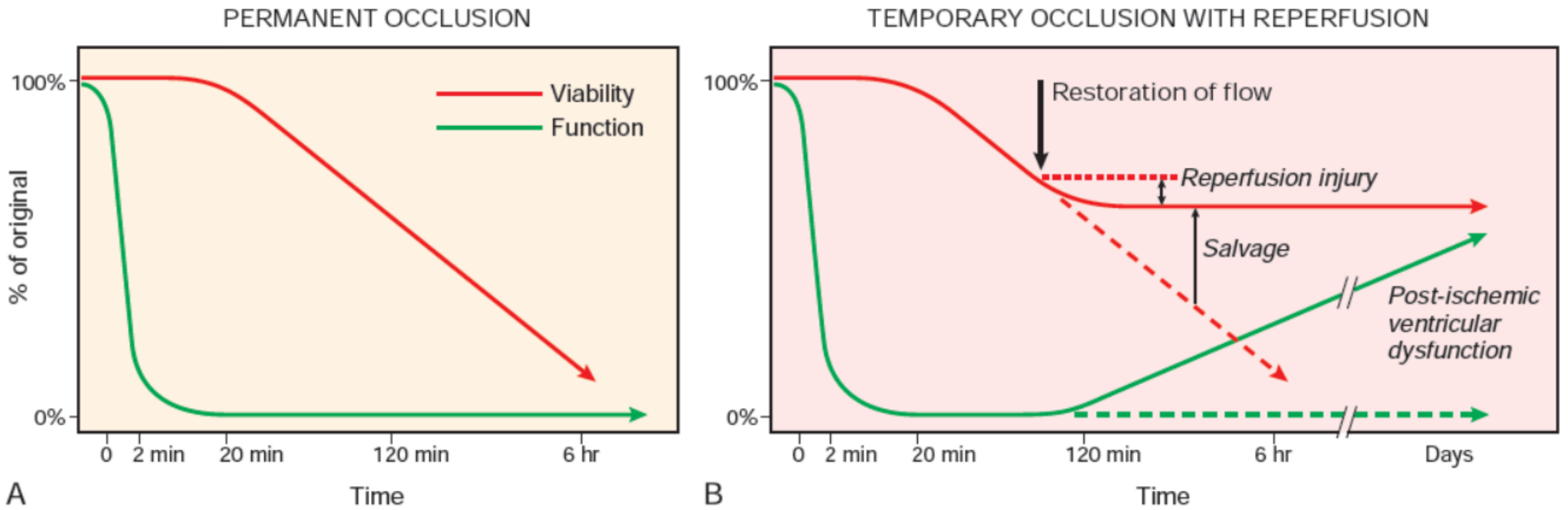


Figure 12-16 Effects of reperfusion on myocardial viability and function. Following coronary occlusion, contractile function is lost within 2 minutes and viability begins to diminish after approximately 20 minutes. If perfusion is not restored (**A**), then nearly all myocardium in the affected region suffers death. **B,** If flow is restored, then some necrosis is prevented, myocardium is salvaged, and at least some function can return. The earlier reperfusion occurs, the greater the degree of salvage. However, the process of reperfusion itself may induce some damage *(reperfusion injury)*, and return of function of salvaged myocardium may be delayed for hours to days *(postischemic ventricular dysfunction* or *stunning)*.

Robbins, Pathologic Basis of Disease 9th ed, 2015

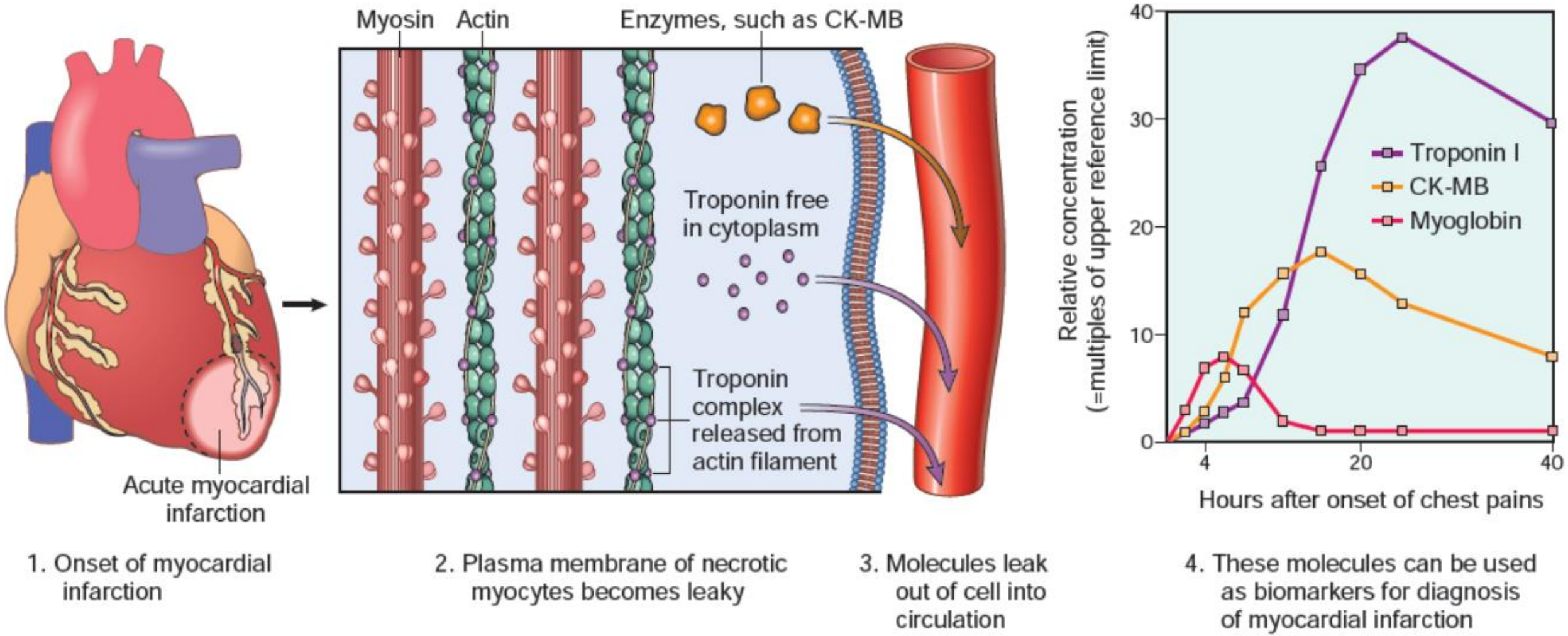


Figure 12-17 Release of myocyte proteins in myocardial infarction. Some of these proteins, for example, troponin I or troponin T, and creatine kinase, MB fraction (CK-MB) are routinely used as diagnostic biomarkers.

Robbins, Pathologic Basis of Disease 9th ed, 2015

Heart Failure (HF)

- Congestive heart failure

Heart Failure

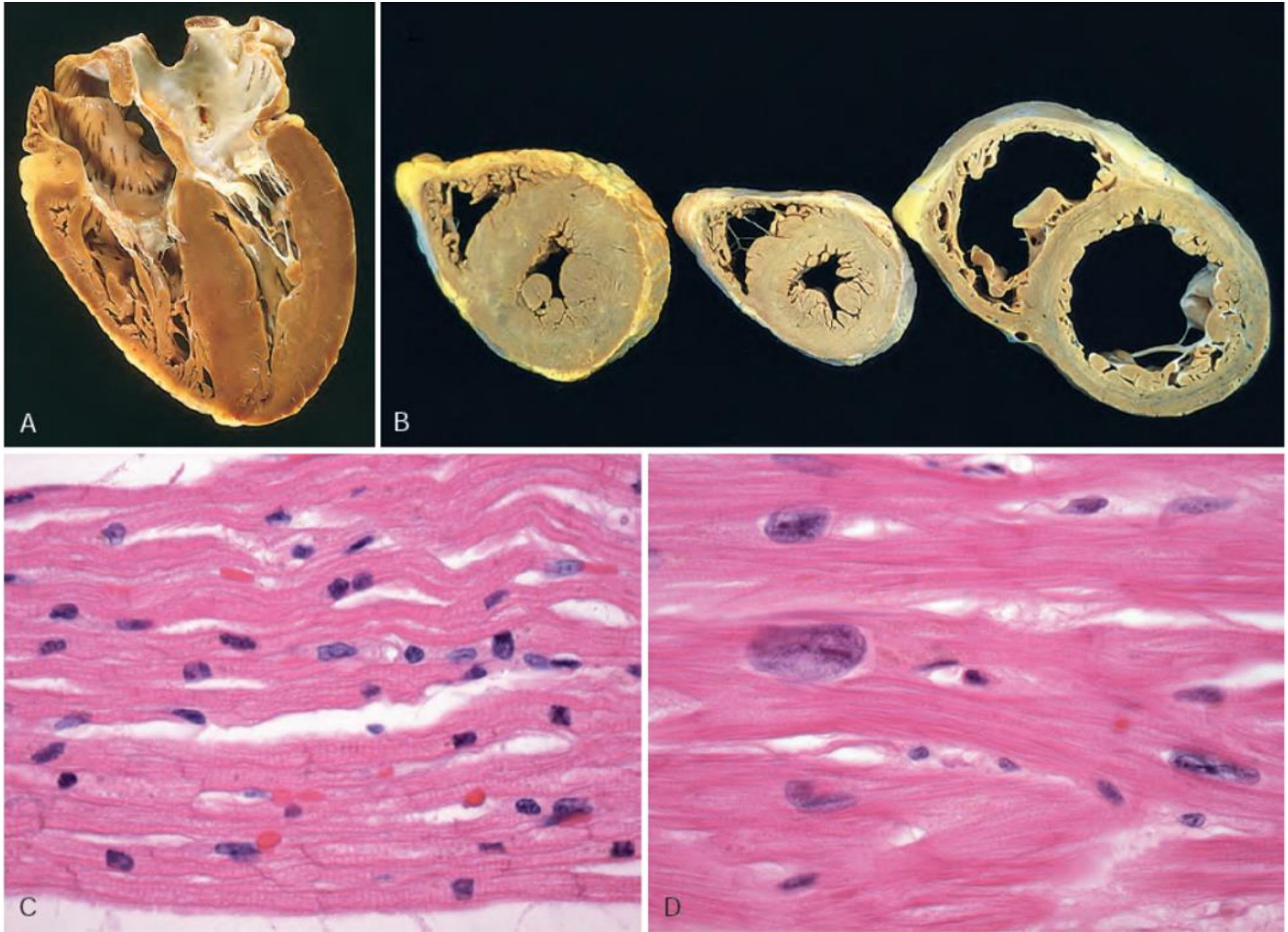
Definition

Heart failure is a condition in which the heart has lost the ability to pump enough blood to the body's tissues. With too little blood being delivered, the organs and other tissues do not receive enough oxygen and nutrients to function properly.

Heart Failure (HF)

Several mechanisms to maintain arterial pressure and organ perfusion:

- Frank – Starling mechanism
- Myocardial adaptation
- Activation of neurohumoral system
  - Release of neuroepinephrine
  - Renin – angiotensin – aldosterone system
  - Atrial natriuretic peptide



Robbins, Pathologic Basis of Disease 9th ed, 2015