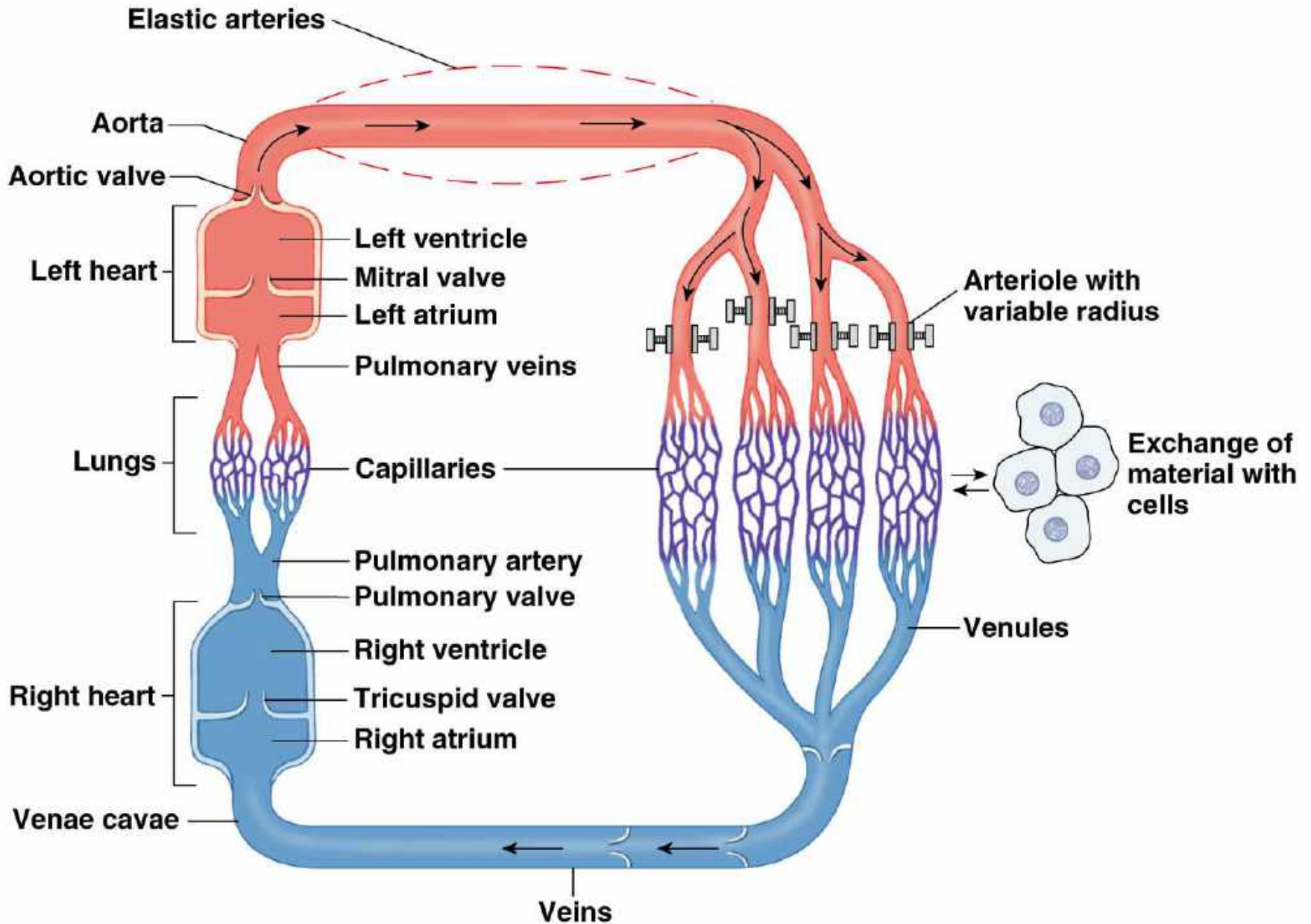


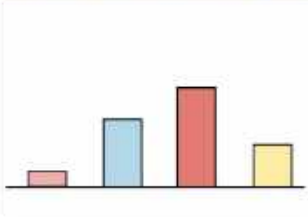
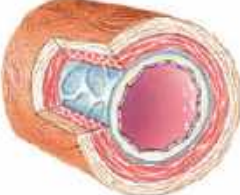
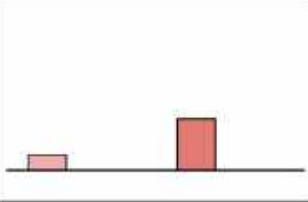
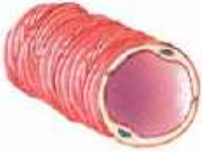




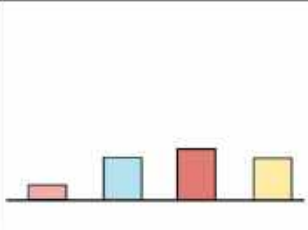

# BLOOD VESSELS





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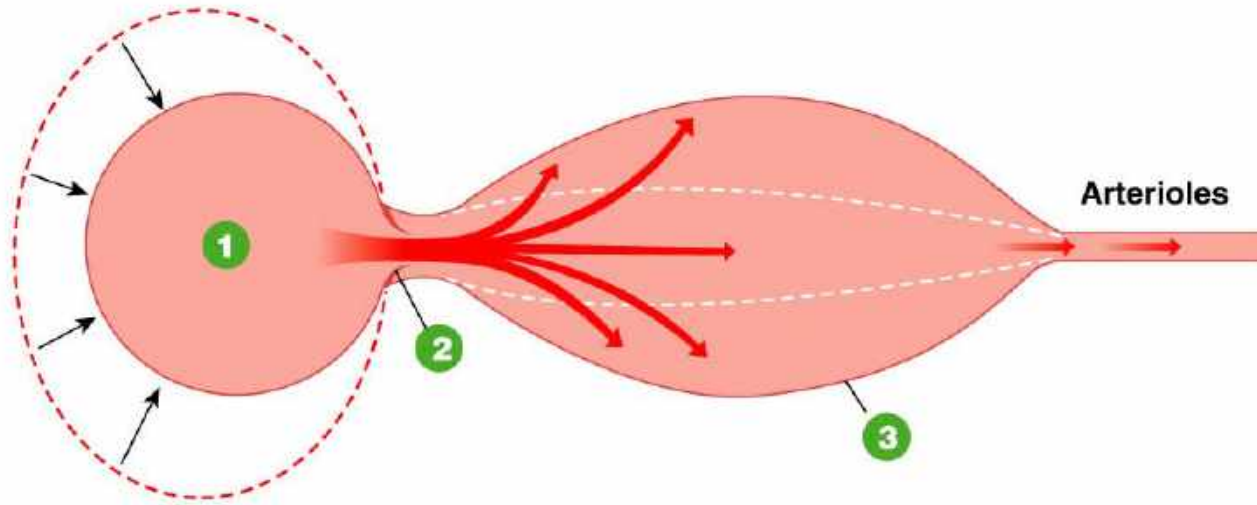
Figure 15-1

	Mean diameter	Mean wall thickness	Endothelium Elastic tissue Smooth muscle Fibrous tissue	
Artery	4.0 mm	1.0 mm		
Arteriole	30.0 μm	6.0 μm		
Capillary	8.0 μm	0.5 μm		
Venule	20.0 μm	1.0 μm		
Vein	5.0 mm	0.5 mm		

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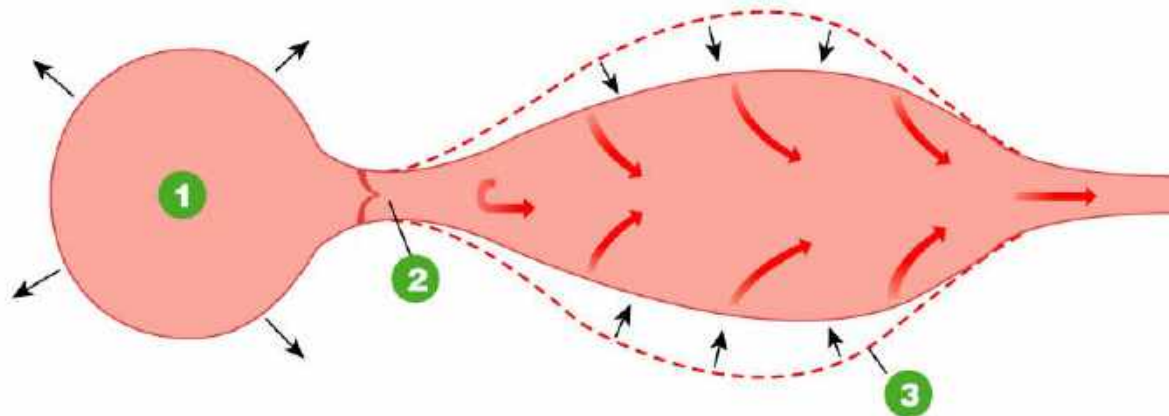
Figure 15-2

### (a) Ventricular contraction

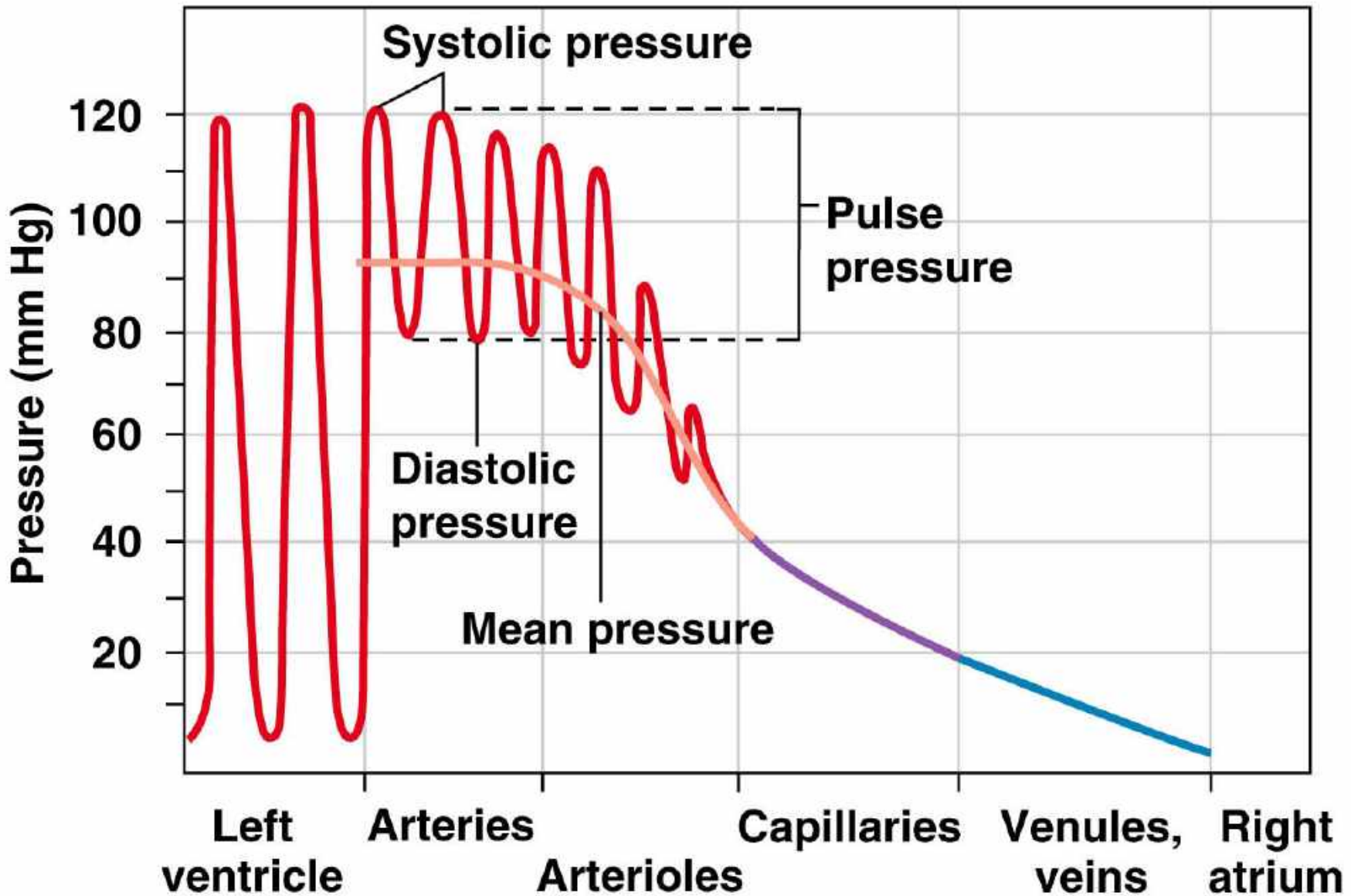


- 1 Ventricle contracts.
- 2 Semilunar valve opens.
- 3 Aorta and arteries expand and store pressure in elastic walls.

### (b) Ventricular relaxation



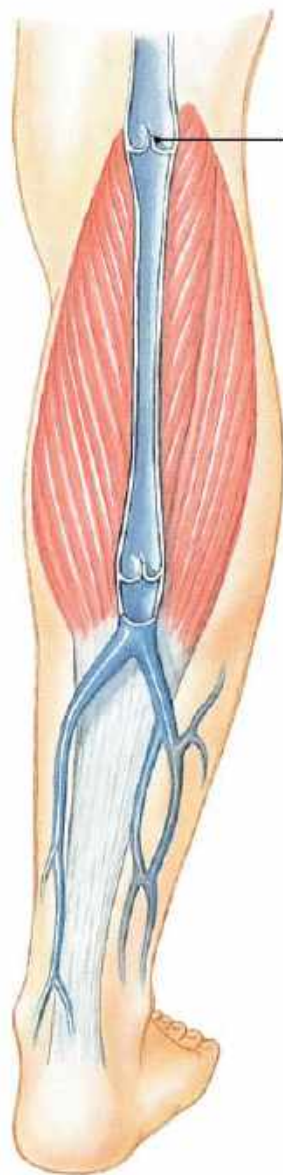
- 1 Isovolumic ventricular relaxation occurs.
- 2 Semilunar valve shuts, preventing flow back into ventricle.
- 3 Elastic recoil of arteries sends blood forward into rest of circulatory system.



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Figure 15-5

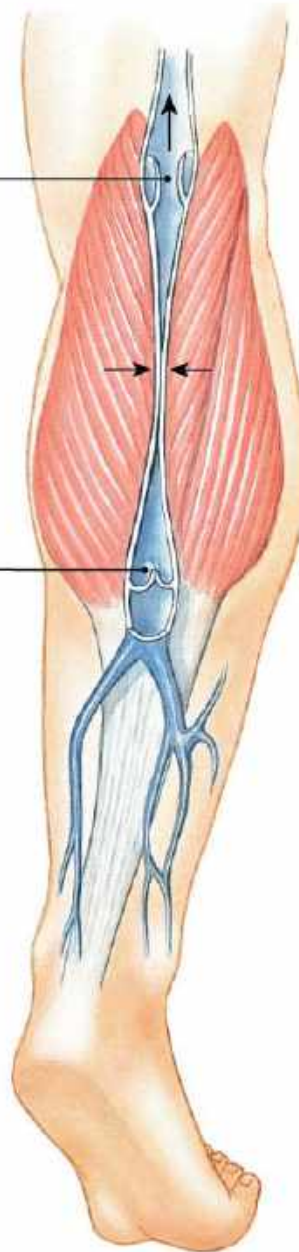
**Valves in the veins prevent backflow of blood.**



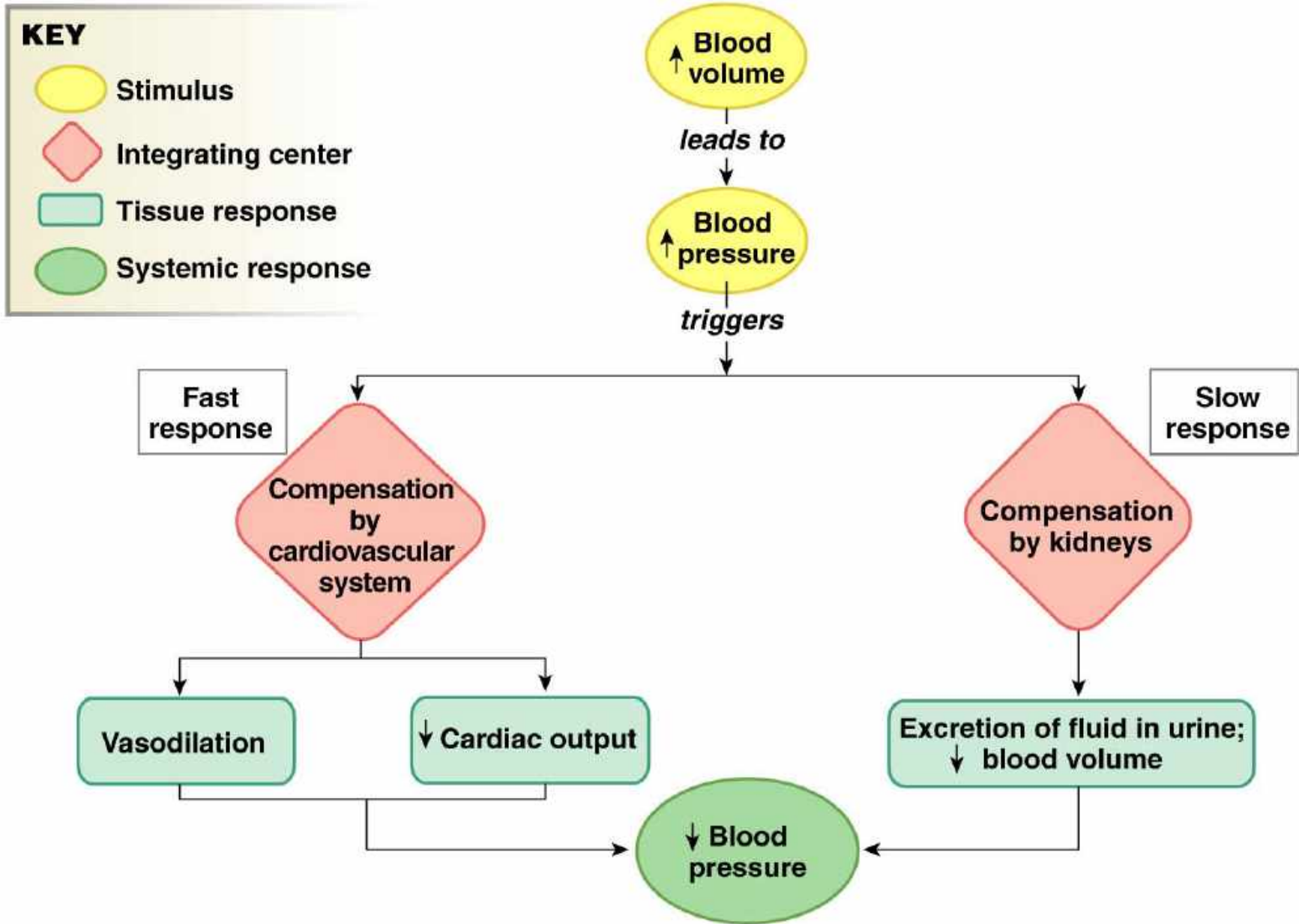
**Valve closed**

**Valve open**

**Valve closed**

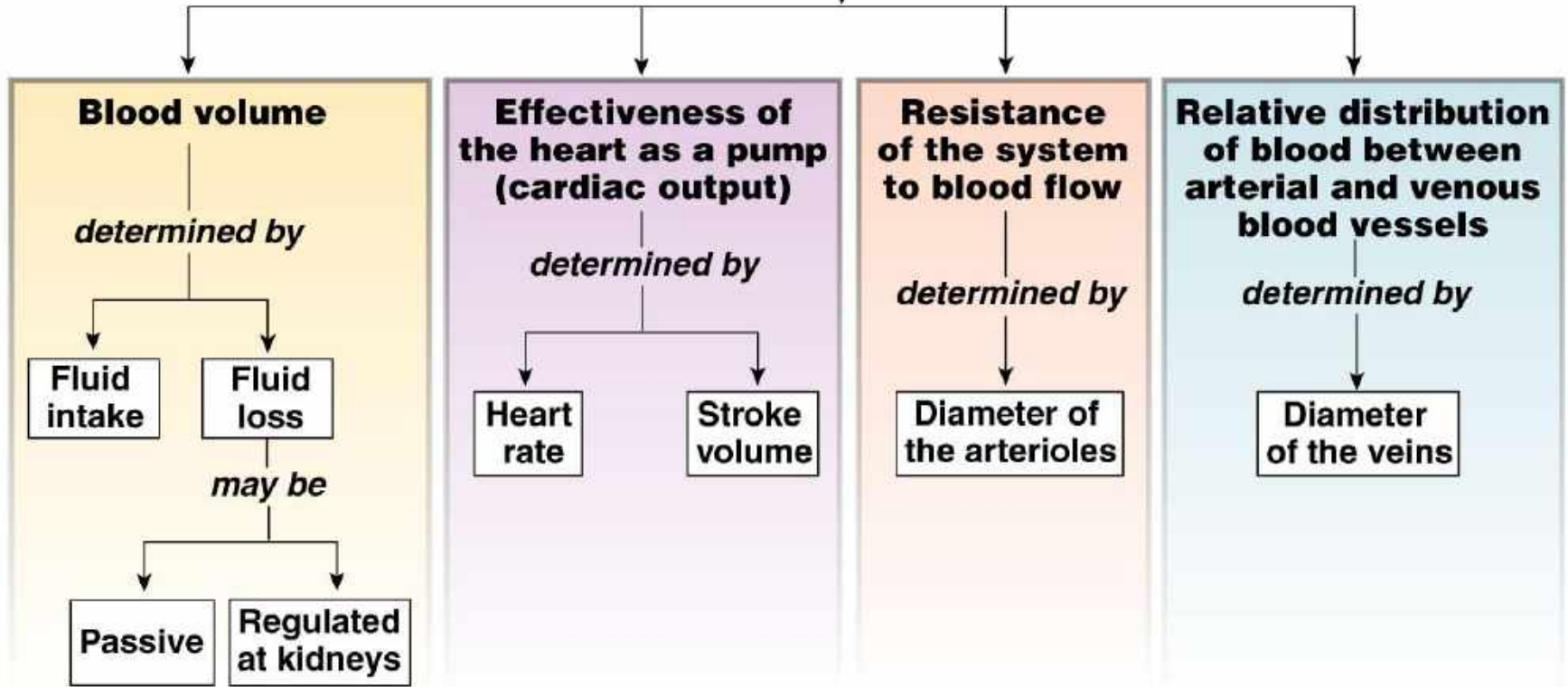


**When the skeletal muscles compress the veins, they force blood toward the heart (the skeletal muscle pump).**



**MEAN ARTERIAL BLOOD PRESSURE**

*is determined by*



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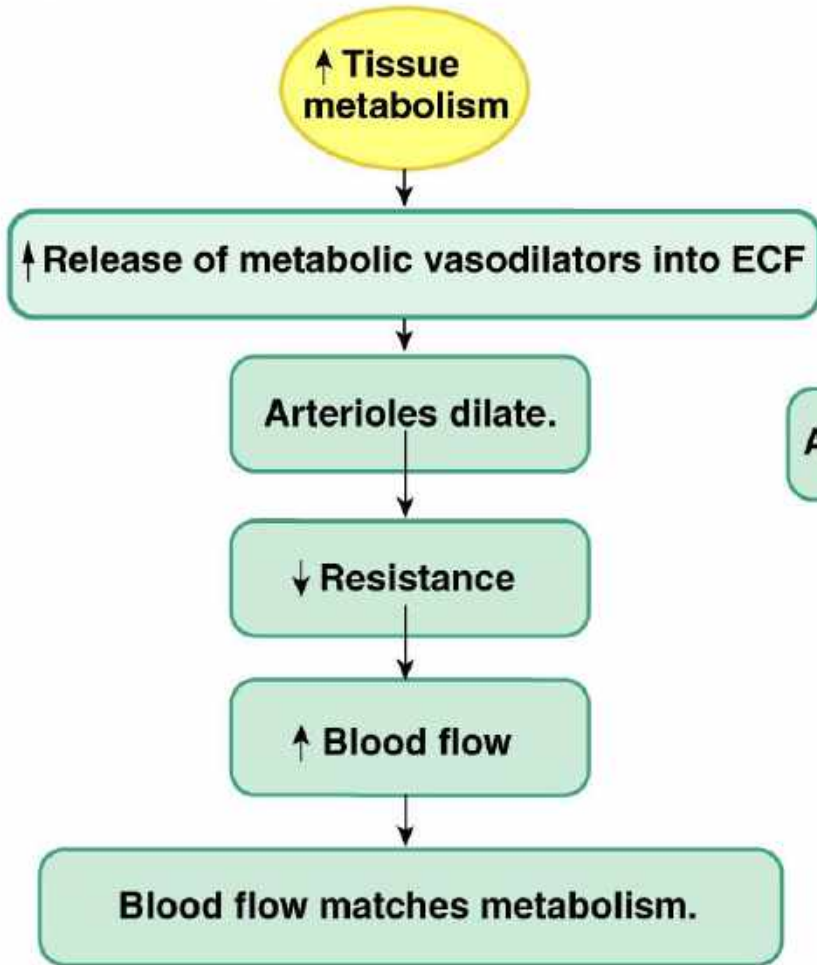
Figure 15-10



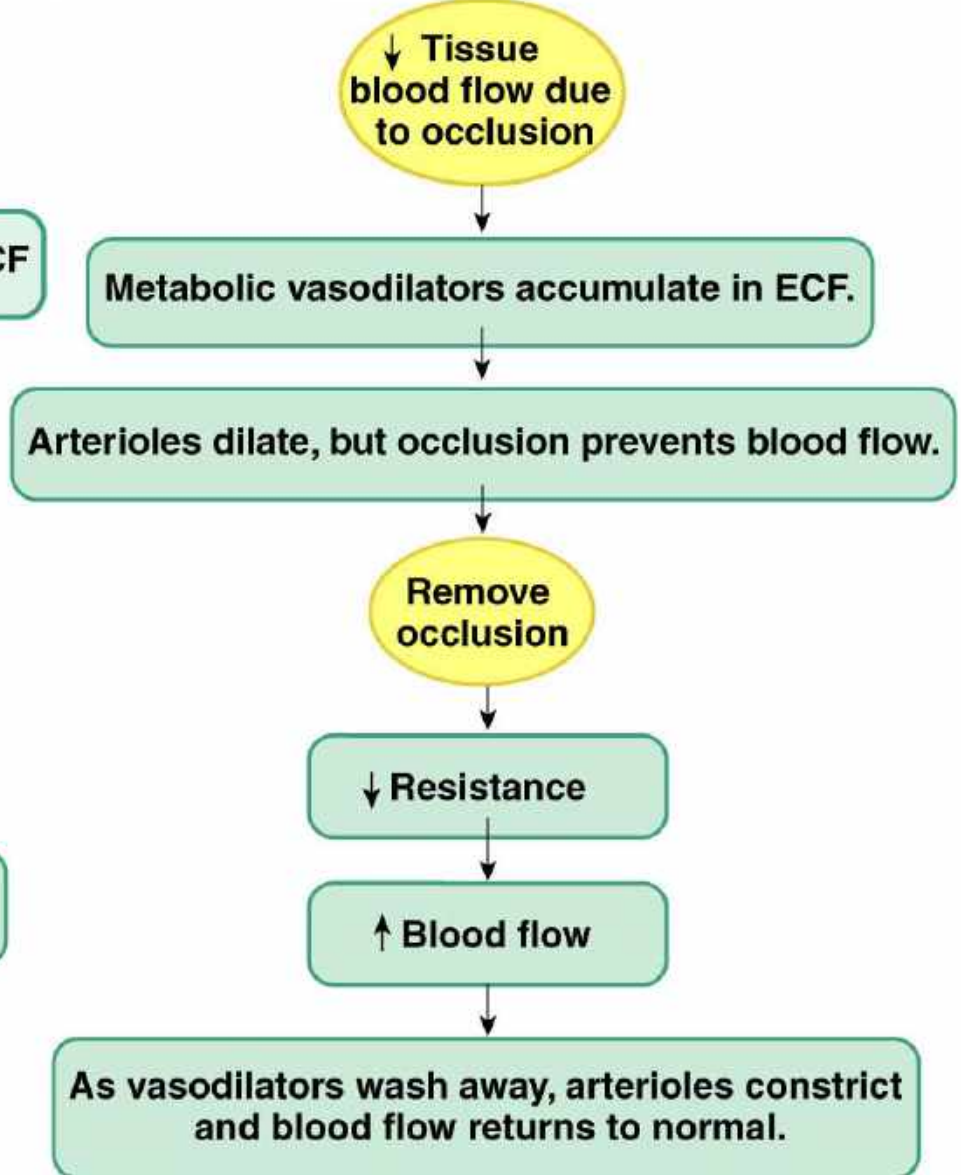
**TABLE 15-2** Chemicals Mediating Vasoconstriction and Vasodilation

CHEMICAL	PHYSIOLOGICAL ROLE	SOURCE	TYPE
<b>Vasoconstriction</b>			
Norepinephrine ( $\alpha$ -receptors)	Baroreceptor reflex	Sympathetic neurons	Neurotransmitter
Serotonin	Platelet aggregation, smooth muscle contraction	Neurons, digestive tract, platelets	Paracrine, neurotransmitter
Substance P	Pain, increase capillary permeability	Neurons, digestive tract	Paracrine, neurotransmitter
Endothelin	Paracrine mediator	Vascular endothelium	Paracrine
Vasopressin	Increase blood pressure in hemorrhage	Posterior pituitary	Neurohormone
Angiotensin II	Increase blood pressure	Plasma hormone	Hormone
<b>Vasodilation</b>			
Epinephrine ( $\beta_2$ -receptors)	Increase blood flow to skeletal muscle, heart, liver	Adrenal medulla	Neurohormone
Acetylcholine (via NO)	Erection of clitoris or penis	Parasympathetic neurons	Neurotransmitter
Vasoactive intestinal peptide	Digestive secretion, relax smooth muscle	Neurons	Neurotransmitter, neurohormone
Nitric oxide (NO)	Paracrine mediator	Endothelium	Paracrine
Bradykinin (via NO)	Increase blood flow	Multiple tissues	Paracrine
Adenosine	Increase blood flow to match metabolism	Hypoxic cells	Paracrine
$\downarrow O_2, \uparrow CO_2, \uparrow H^+, \uparrow K^+$	Increase blood flow to match metabolism	Cell metabolism	Paracrine
Histamine	Increase blood flow	Mast cells	Paracrine
Natriuretic peptides (example—ANP)	Reduce blood pressure	Atrial myocardium, brain	Hormone, neurotransmitter

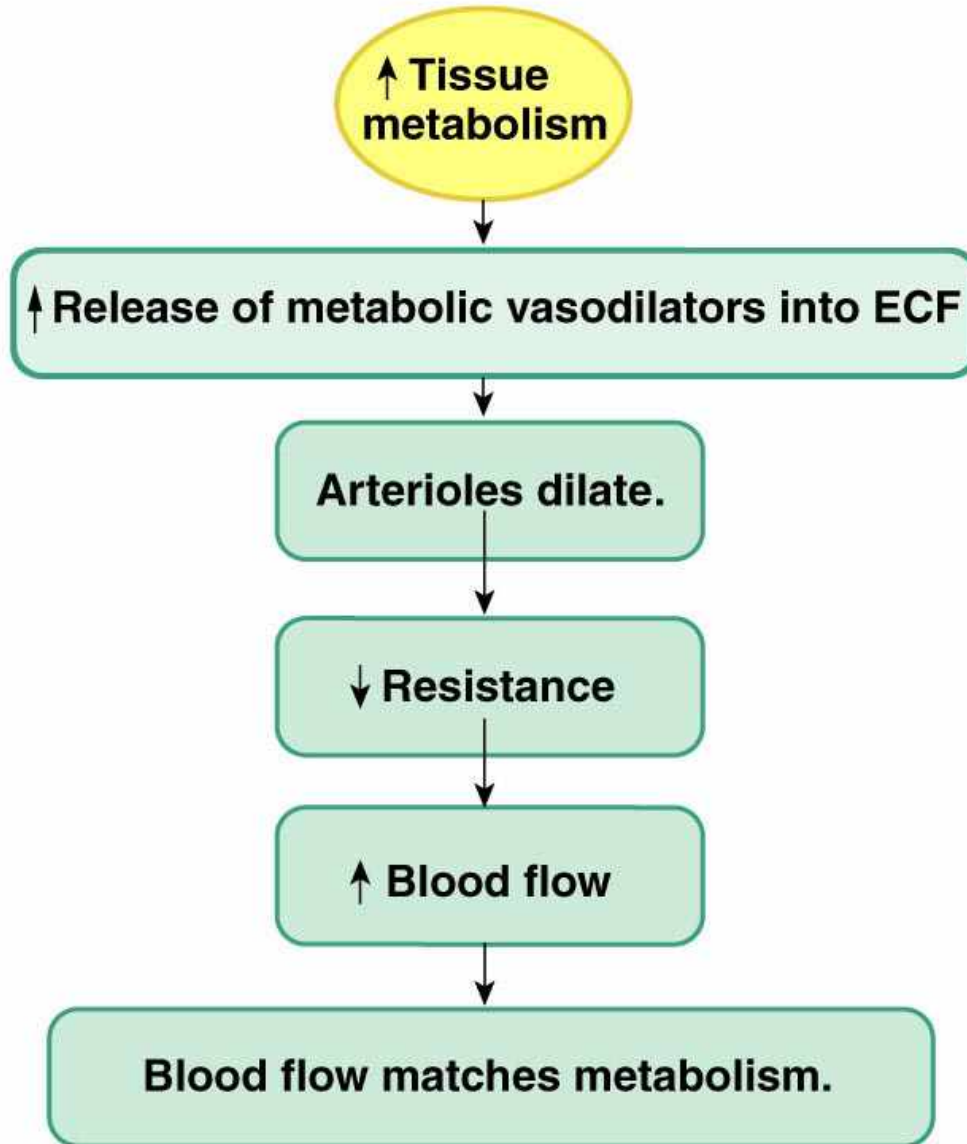
**(a) Active hyperemia**



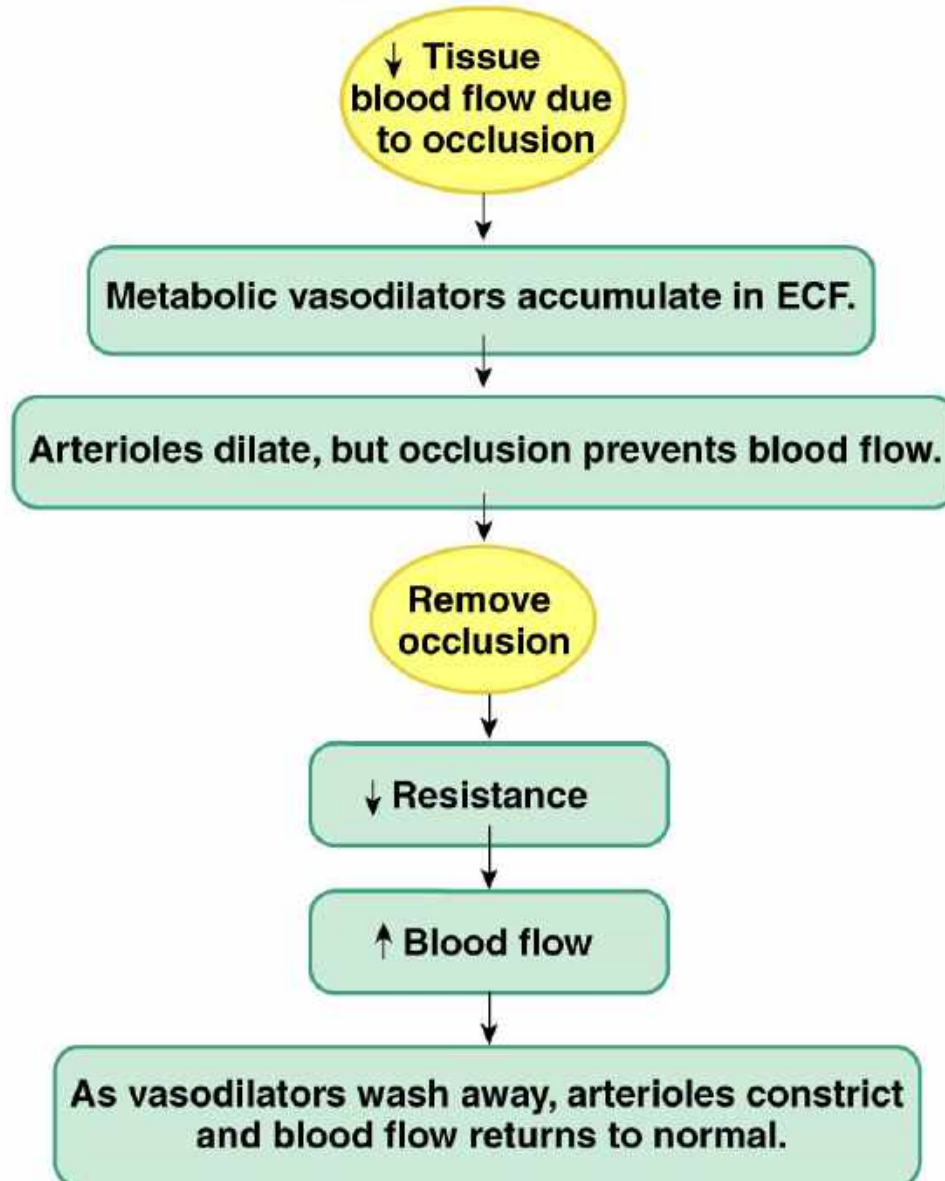
**(b) Reactive hyperemia**



### (a) Active hyperemia



## **(b) Reactive hyperemia**



Arteriole diameter is controlled by tonic release of norepinephrine.

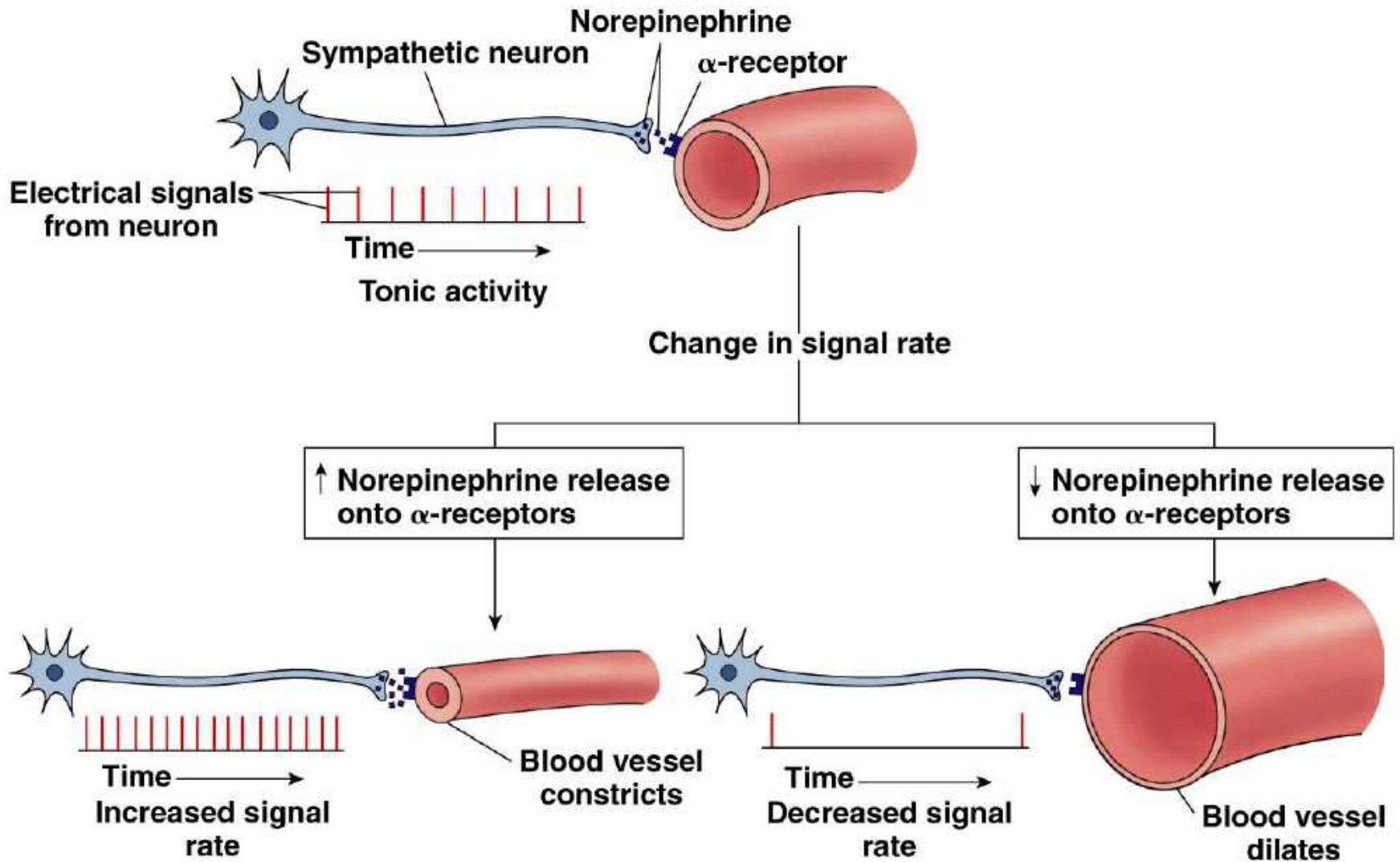
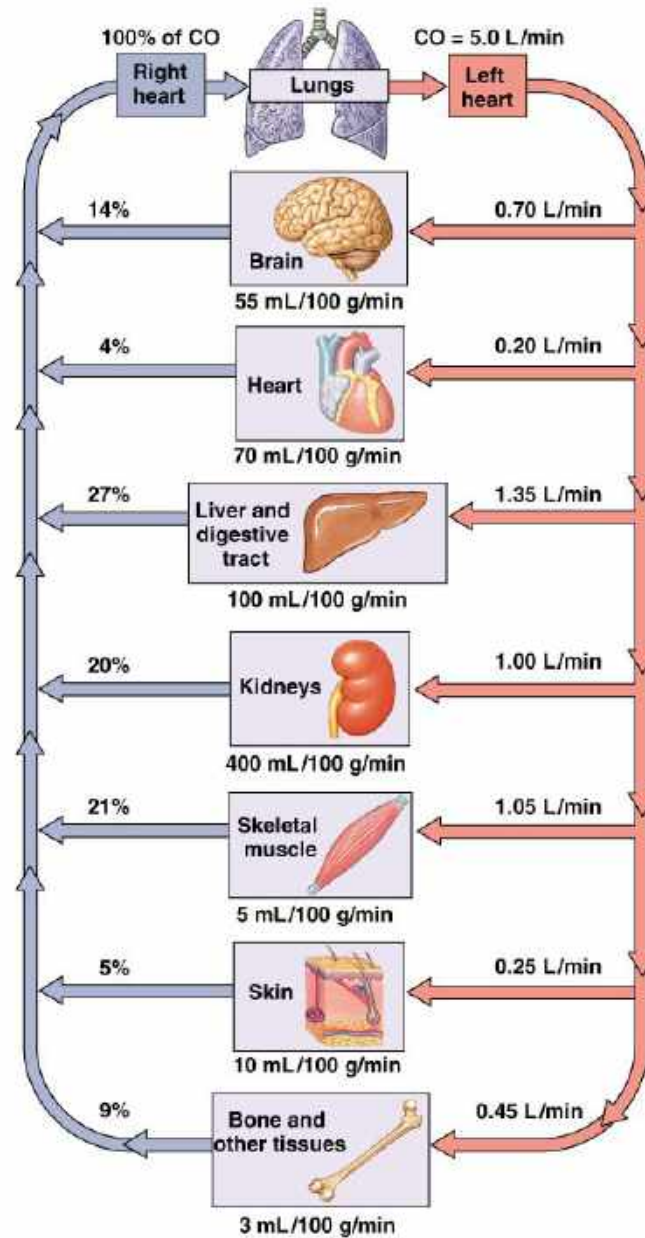


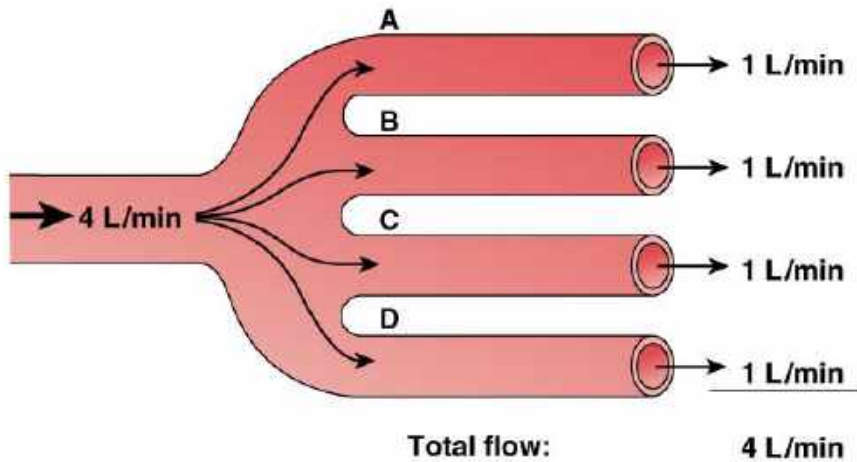
Figure 15-12



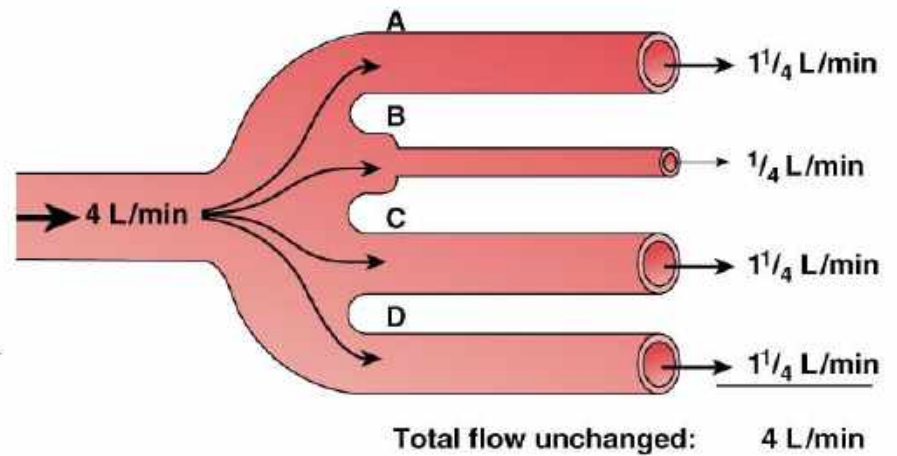
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Figure 15-13

**(a)** Blood flow through four identical vessels (A–D) is equal. Total flow into vessels equals total flow out.

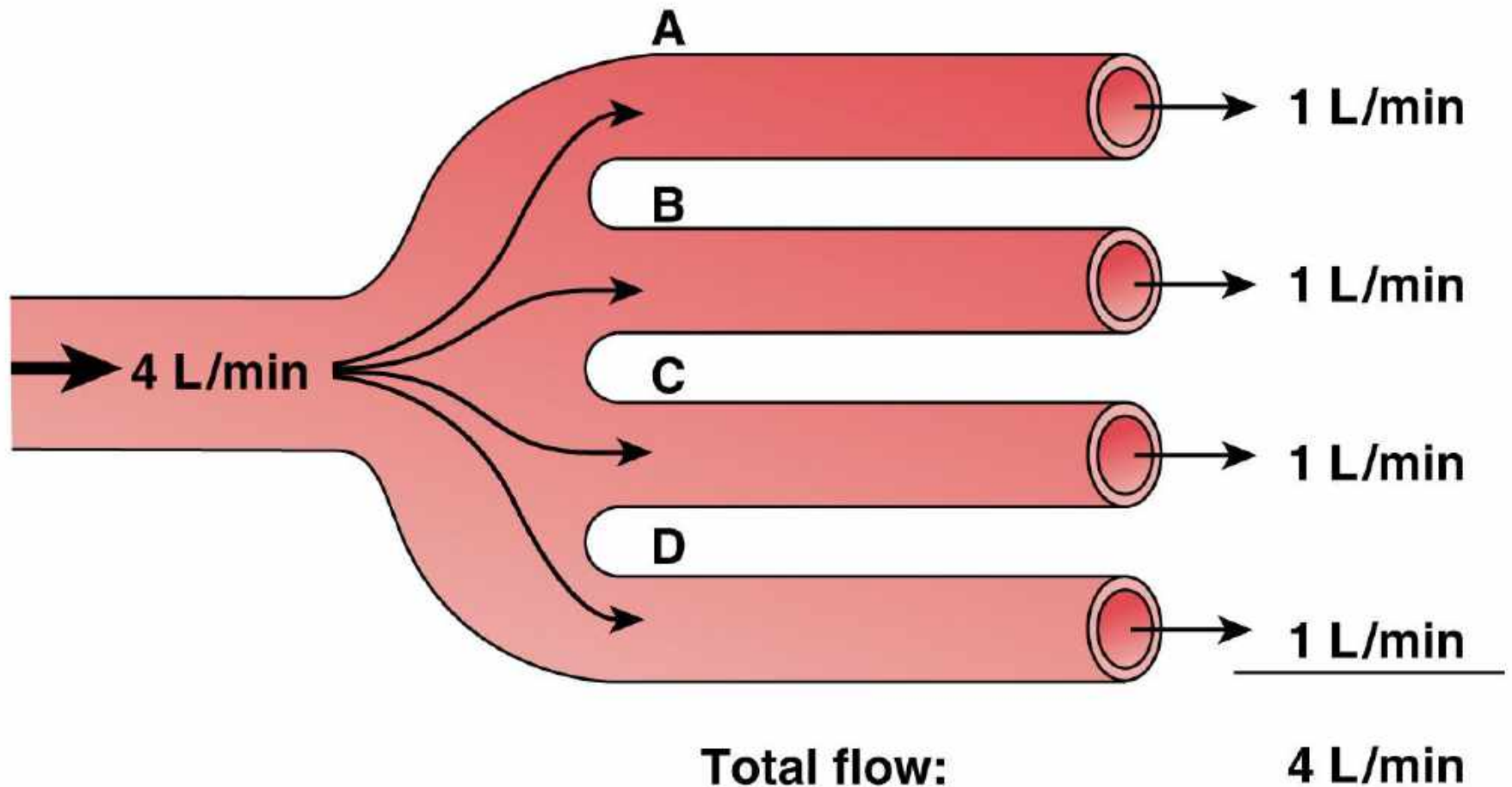


**(b)** When vessel B constricts, resistance of B increases and flow through B decreases. Flow diverted from B is divided among the lower-resistance vessels A, C, and D.



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**(a) Blood flow through four identical vessels (A–D) is equal. Total flow into vessels equals total flow out.**

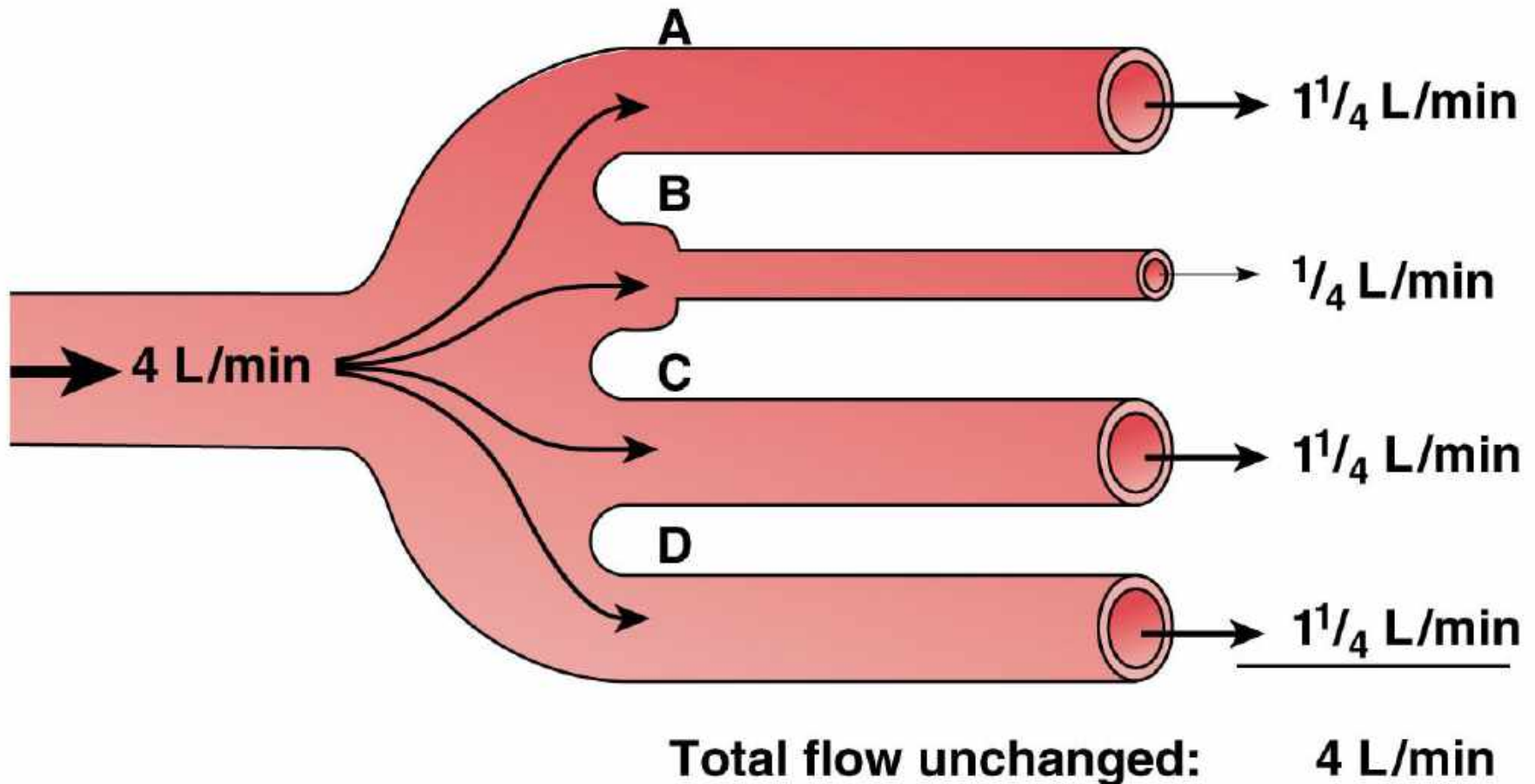


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Figure 15-14a

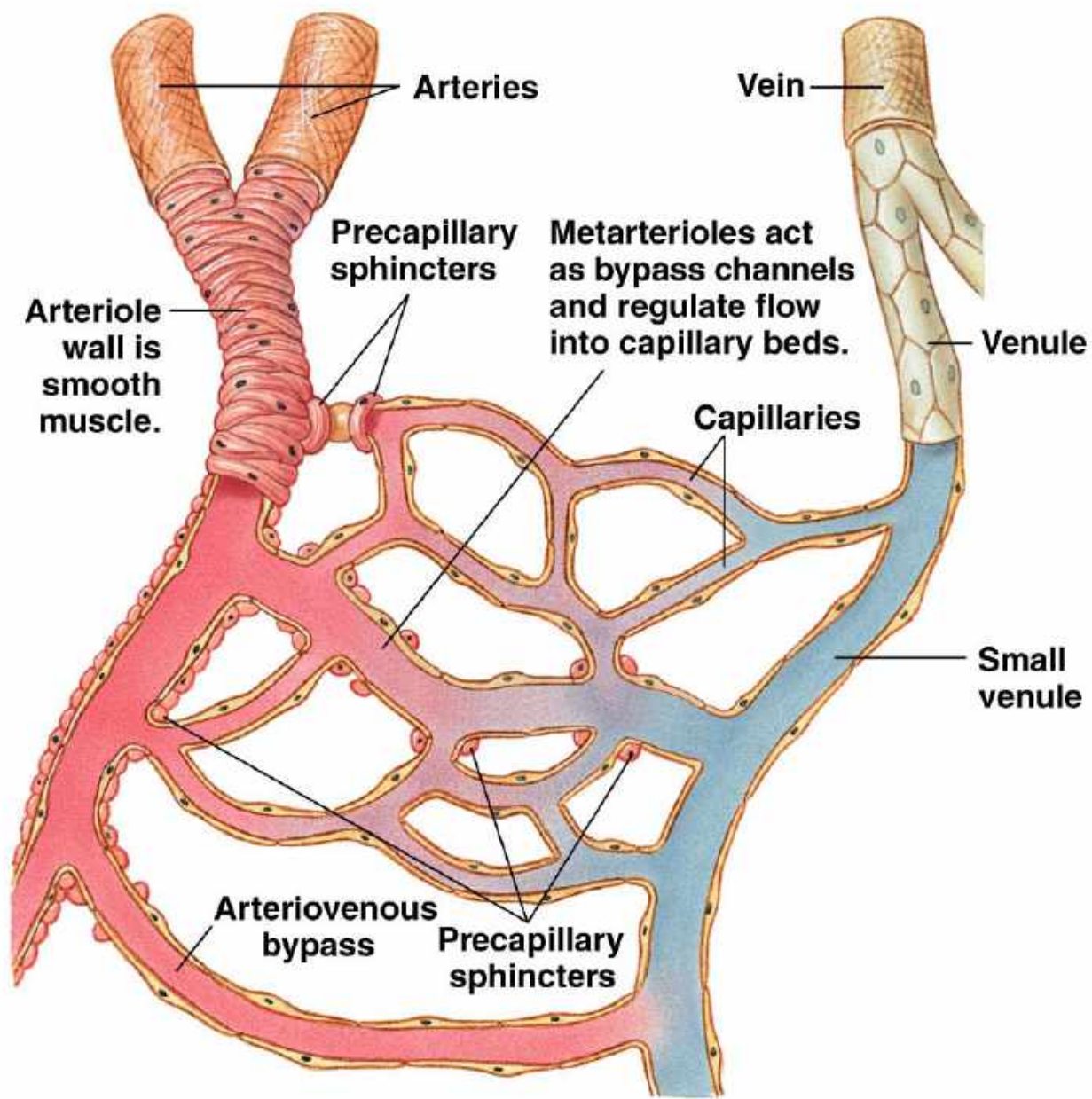


**(b)** When vessel B constricts, resistance of B increases and flow through B decreases. Flow diverted from B is divided among the lower-resistance vessels A, C, and D.



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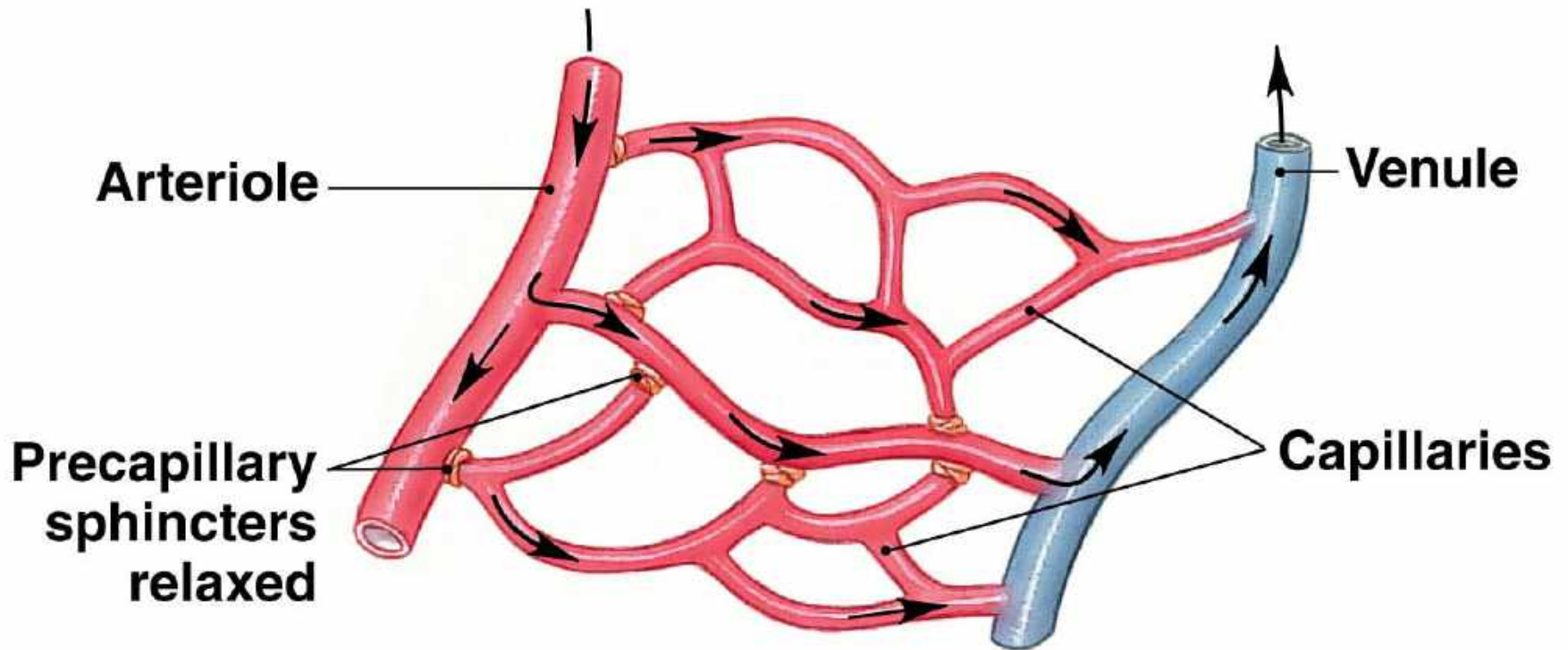
Figure 15-14b



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Figure 15-3

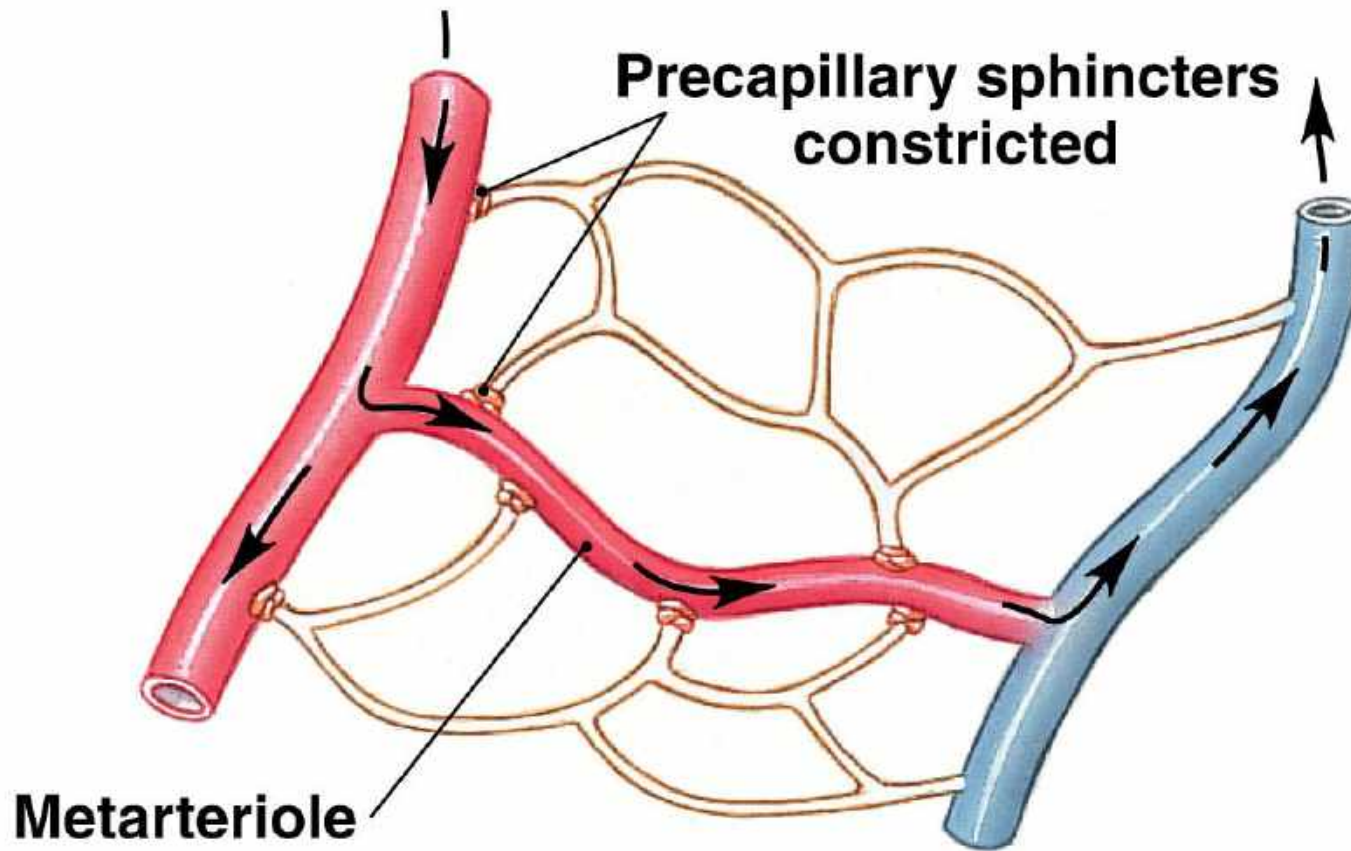
**(a) When precapillary sphincters are relaxed, blood flows through all capillaries in the bed.**



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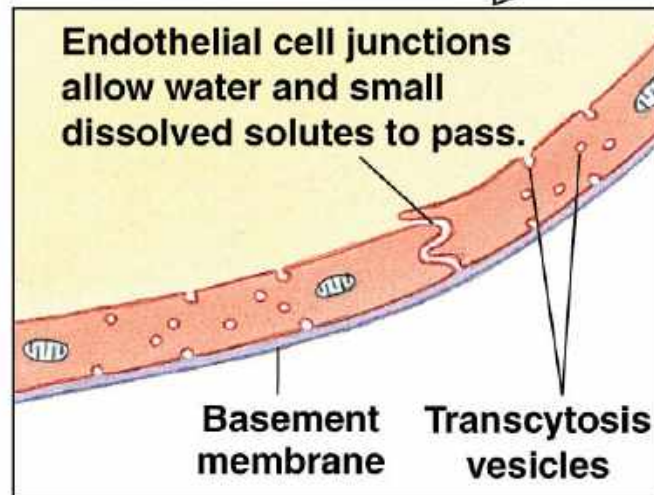
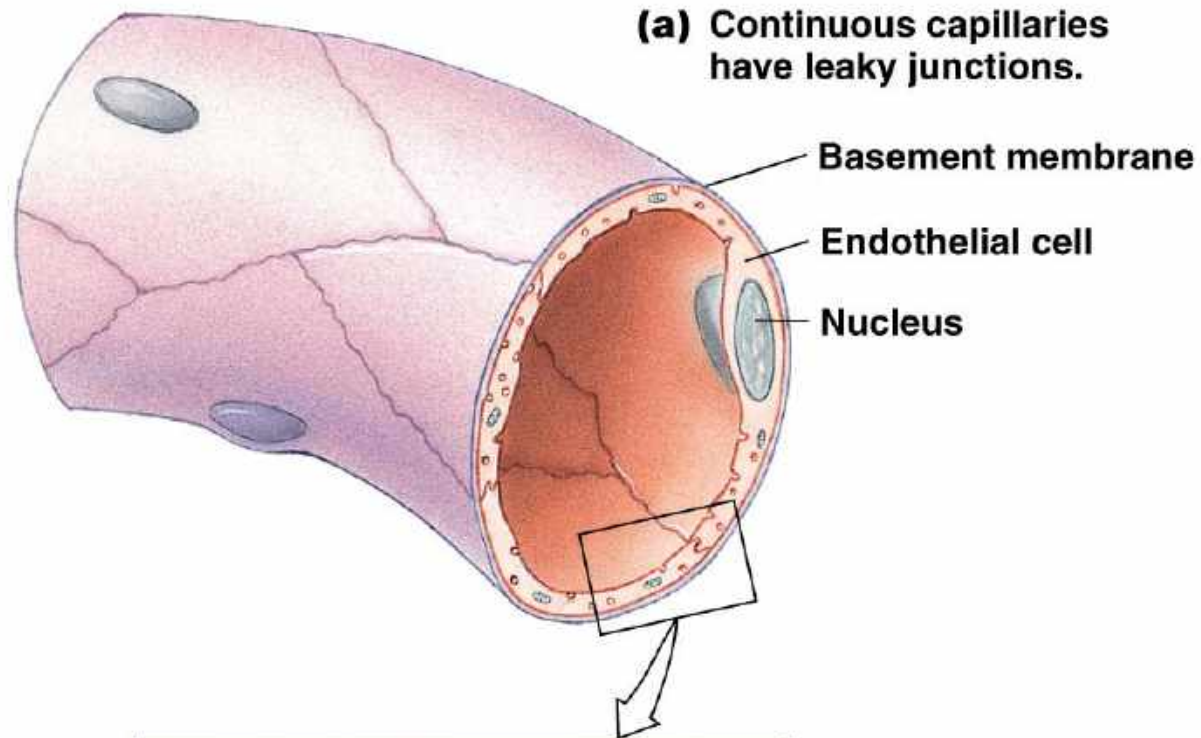
Figure 15-15a

**(b) If precapillary sphincters constrict, blood flow bypasses capillaries completely and flows through metarterioles.**

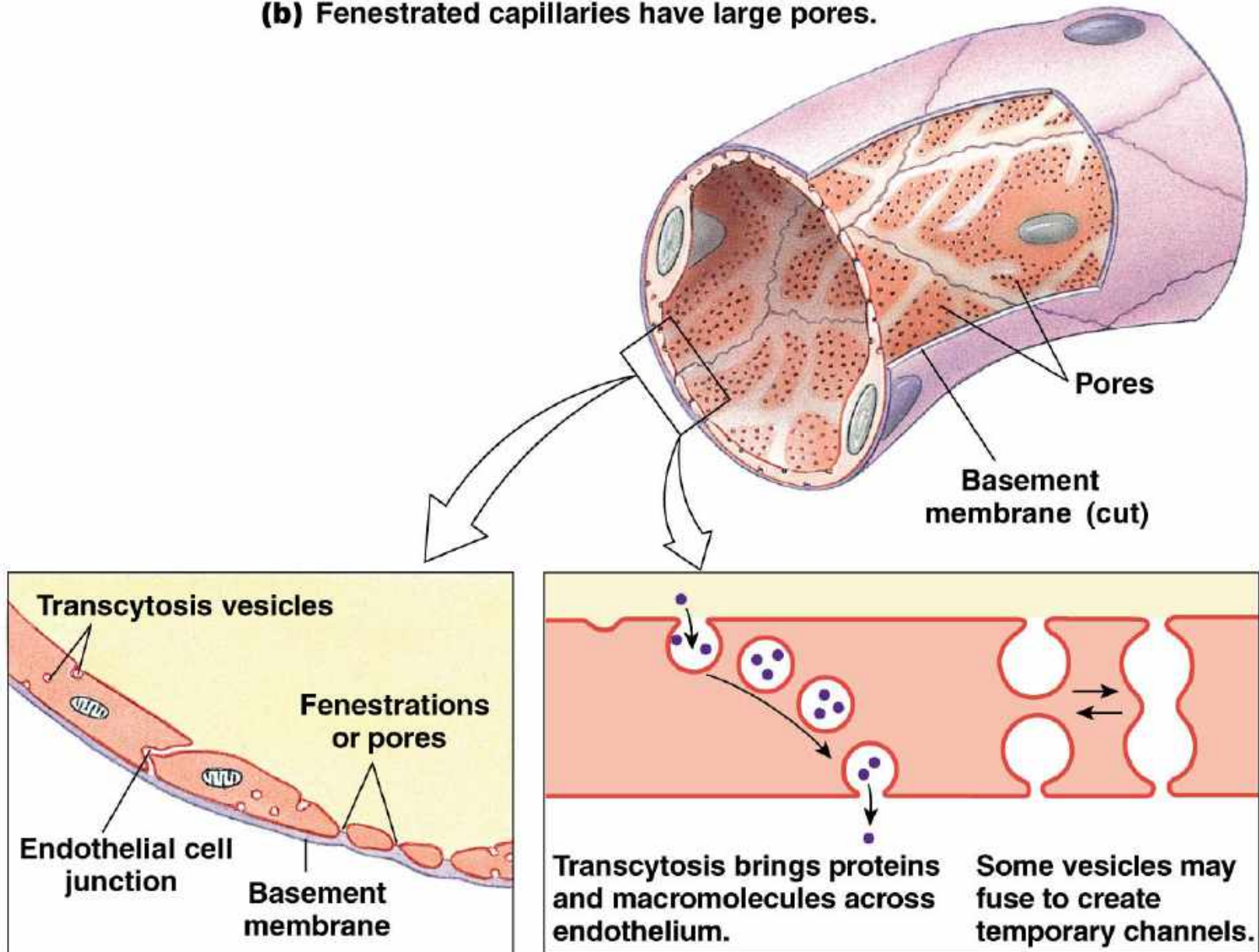


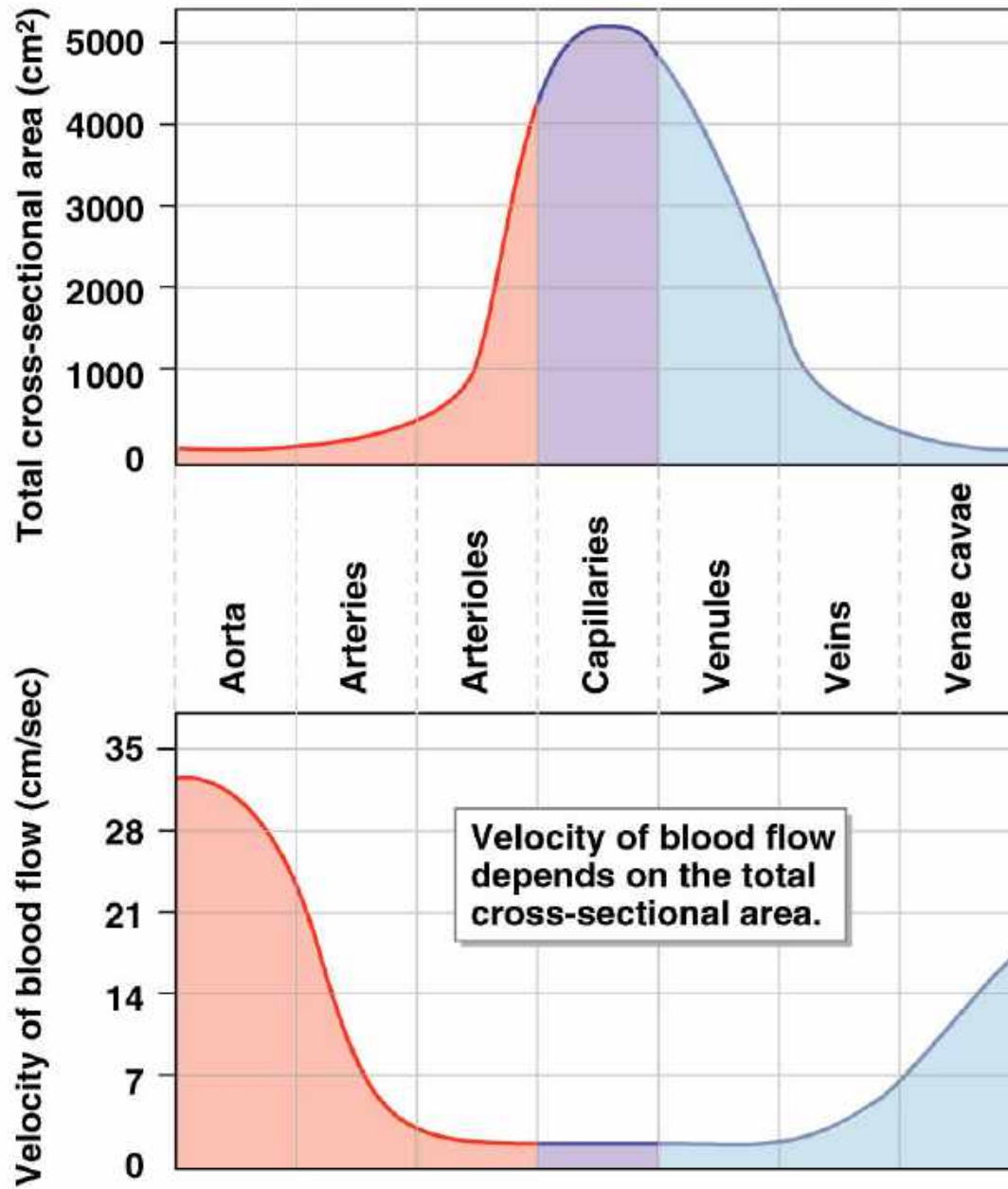
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Figure 15-15b



**(b) Fenestrated capillaries have large pores.**



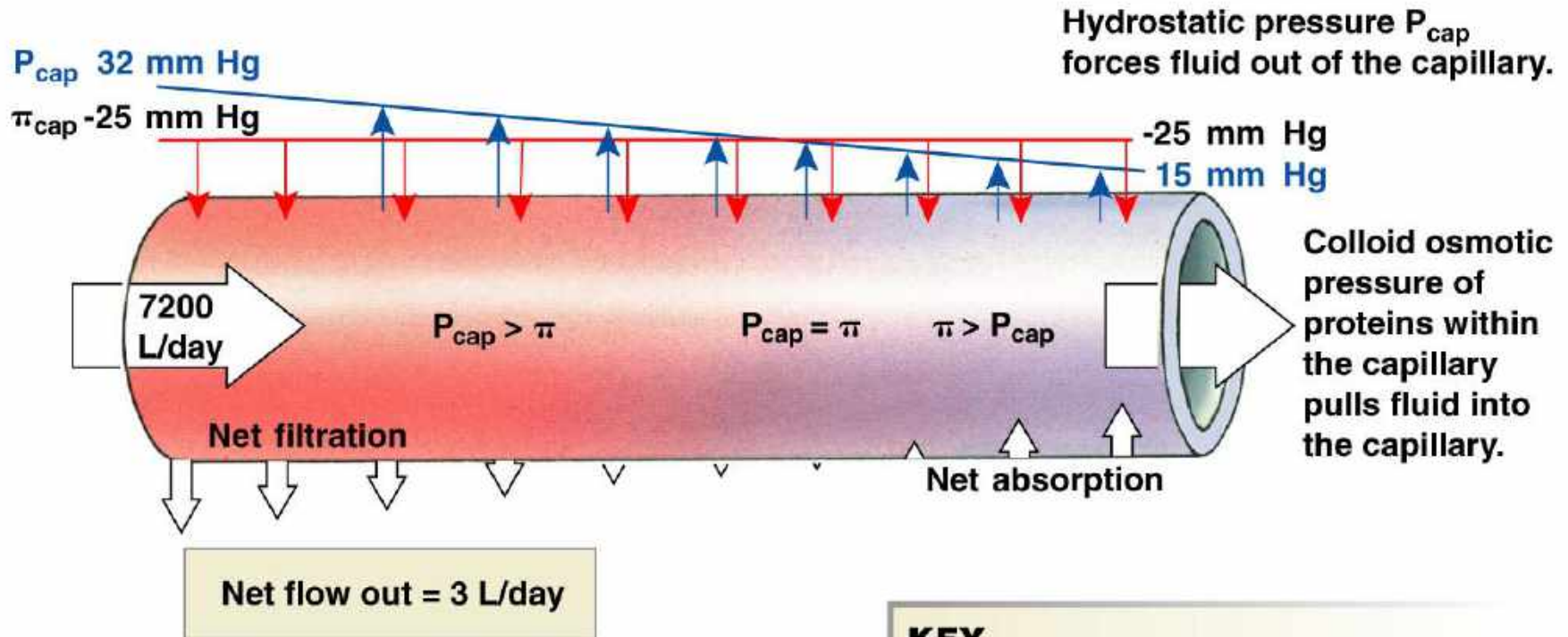


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Figure 15-17

### (a) Filtration in systemic capillaries

Net pressure = hydrostatic pressure – colloid osmotic pressure

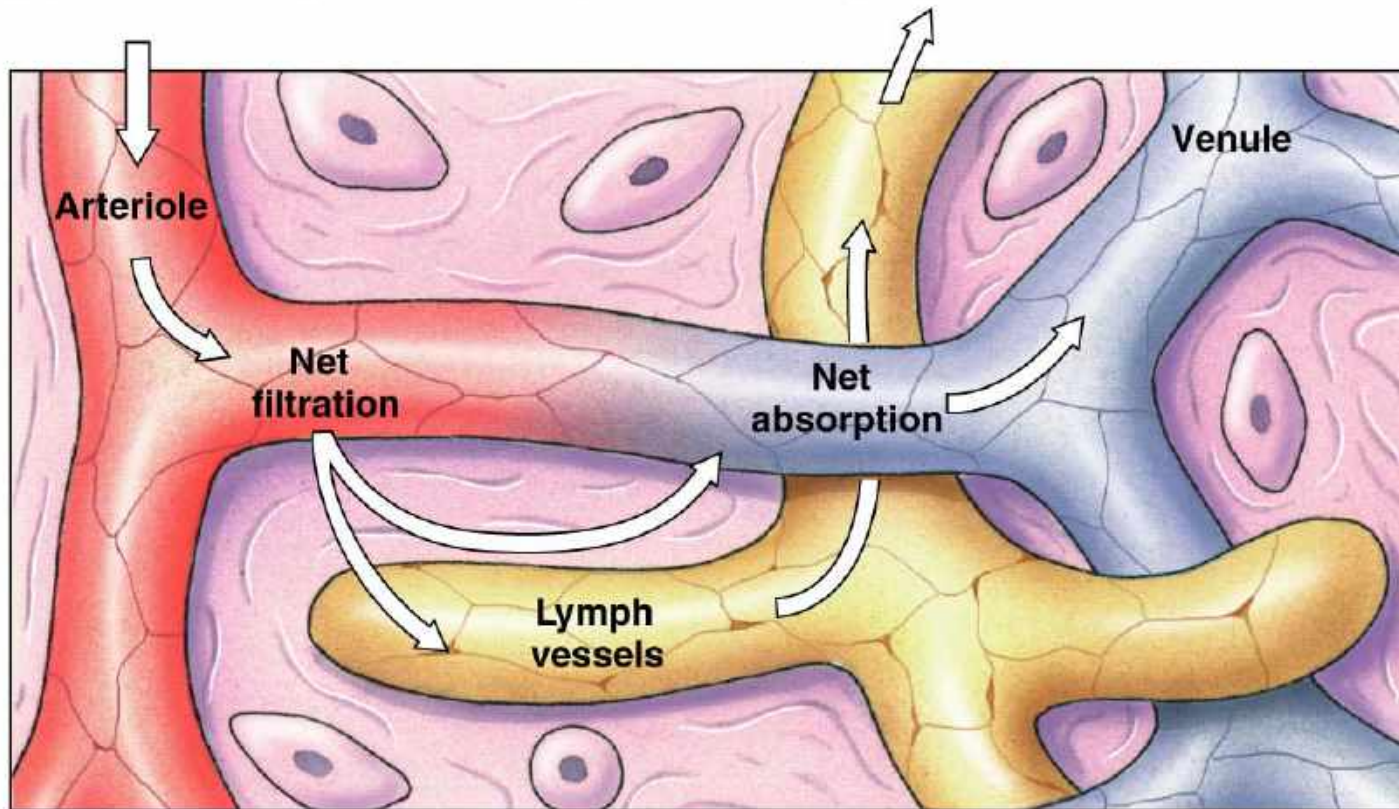


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Figure 15-18a



**(b) Relationship between capillaries and lymph vessels**

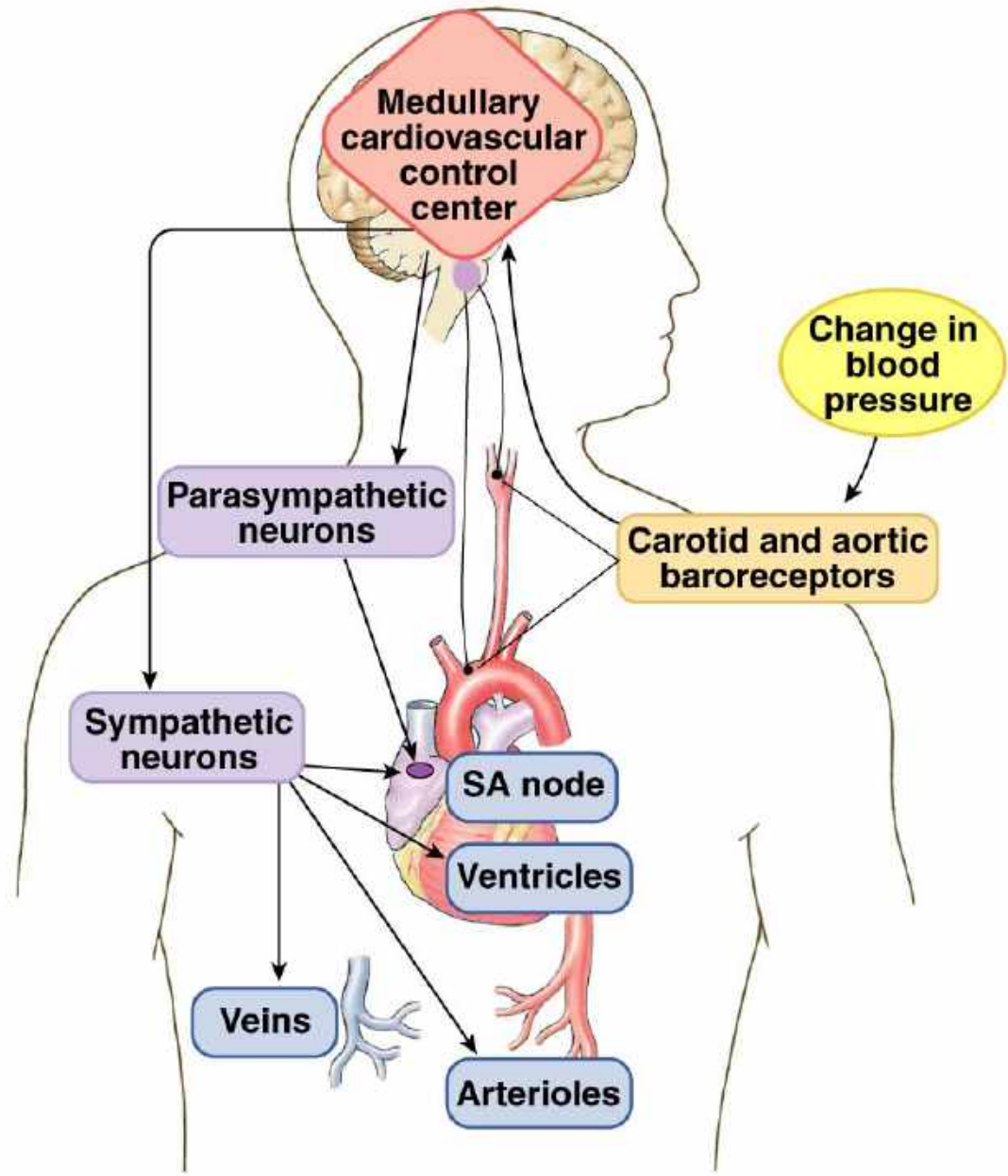
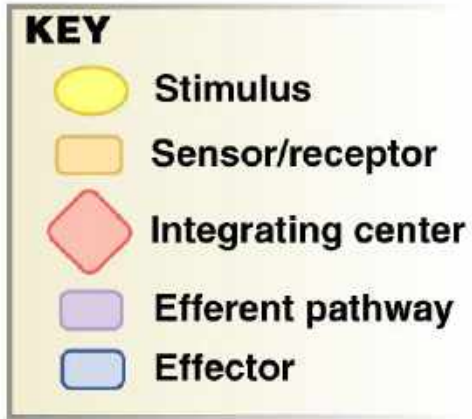


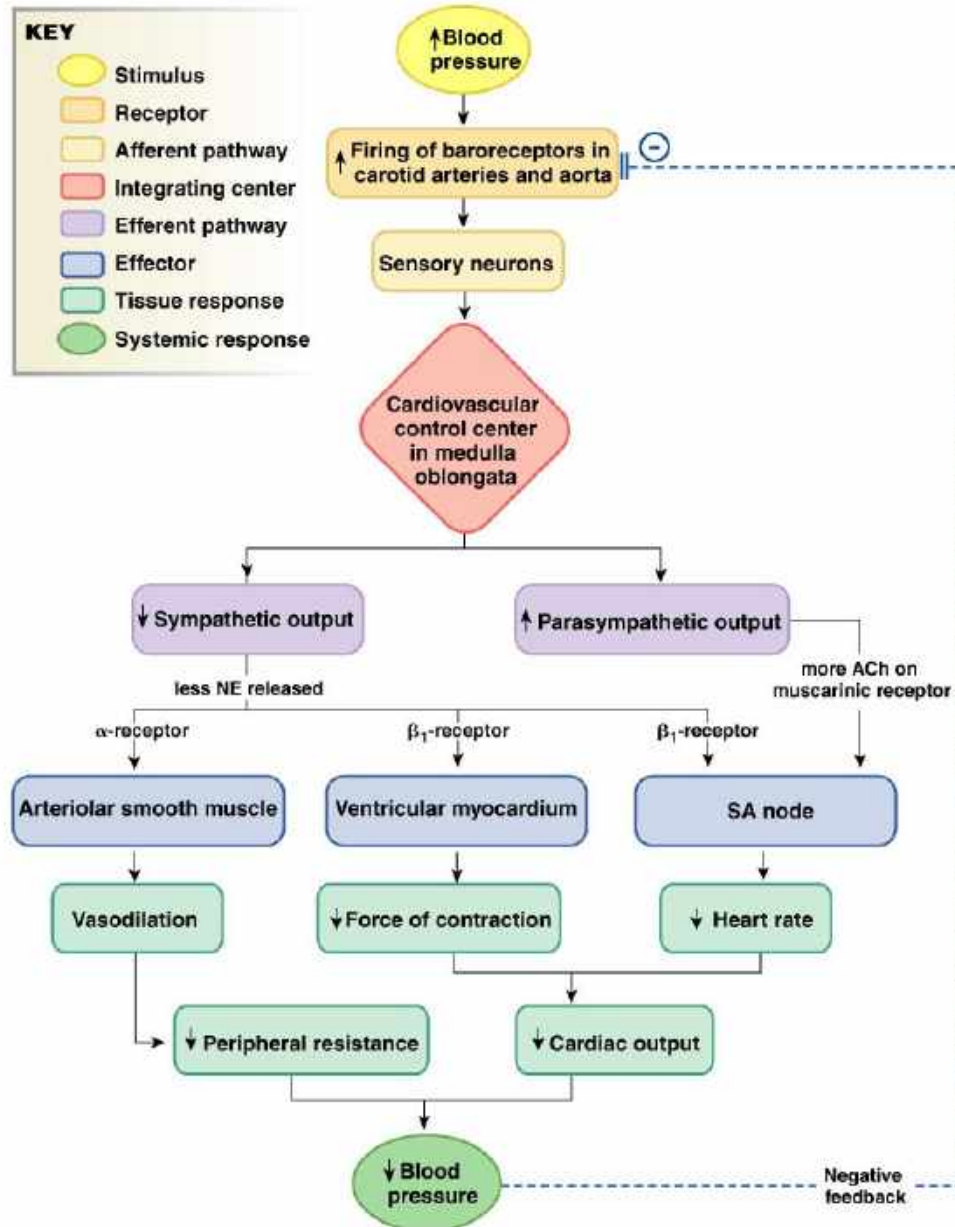
The excess water and solutes that filter out of the capillary are picked up by the lymph vessels and returned to the circulation.

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Figure 15-18b

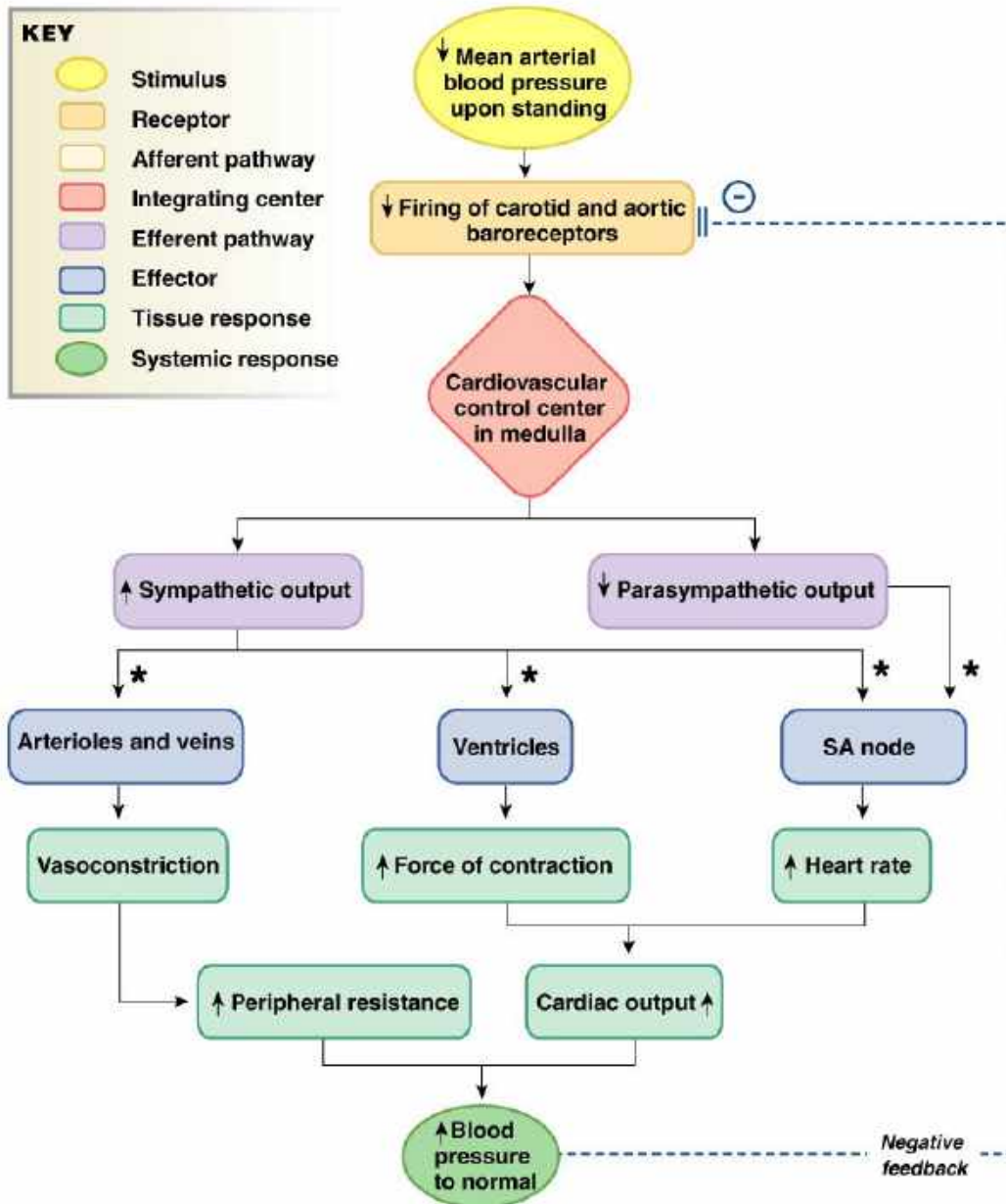


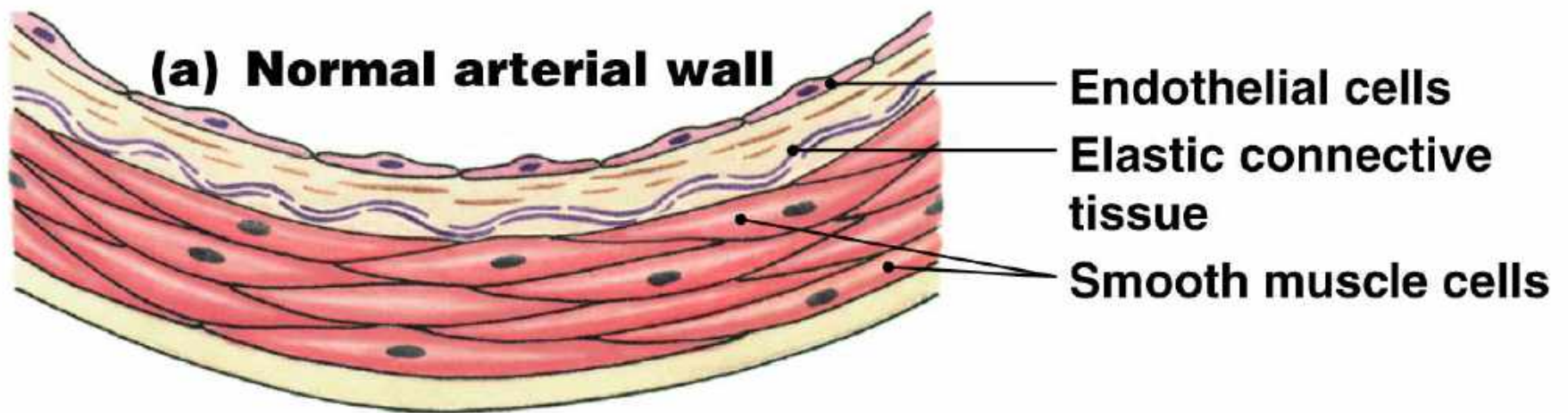




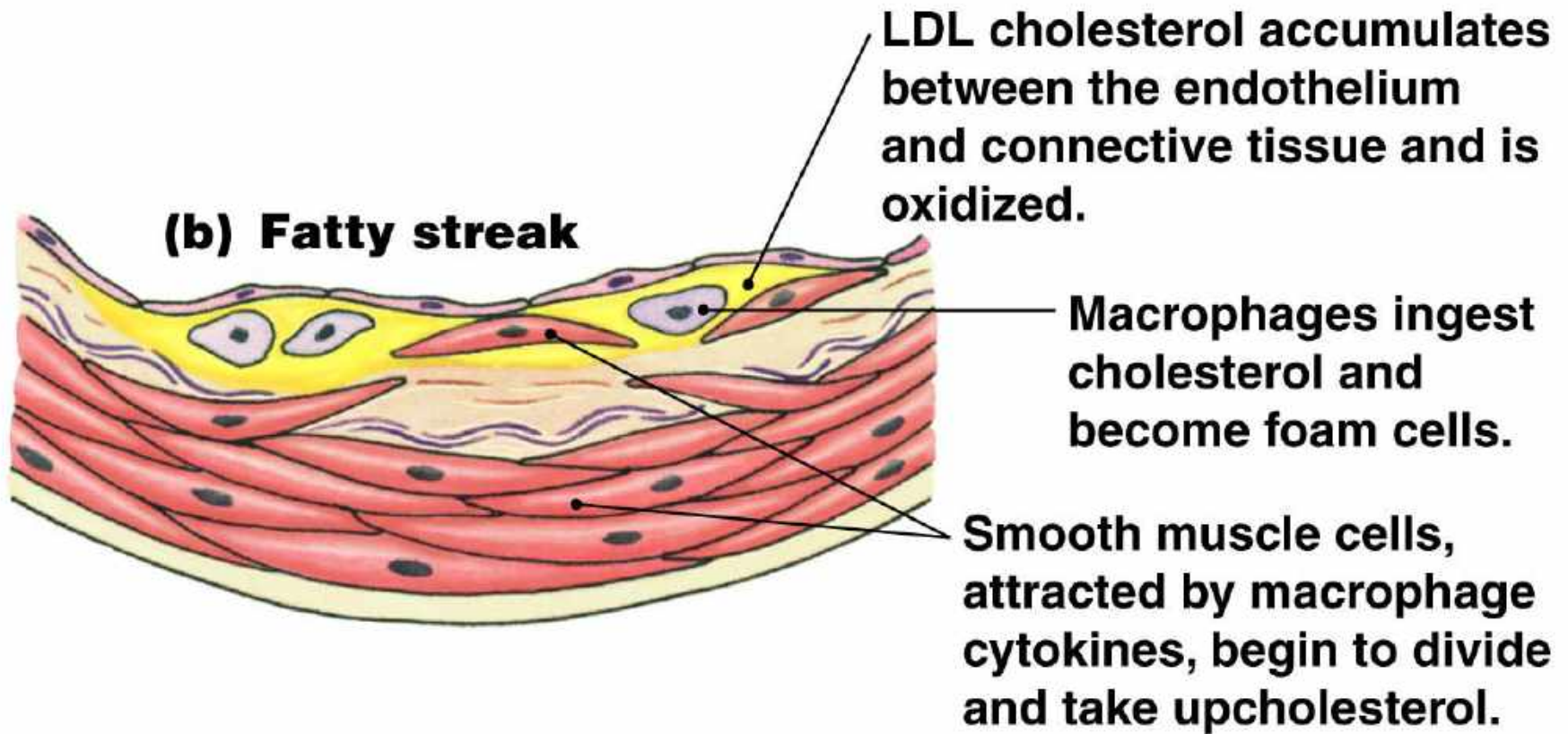
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Figure 15-22



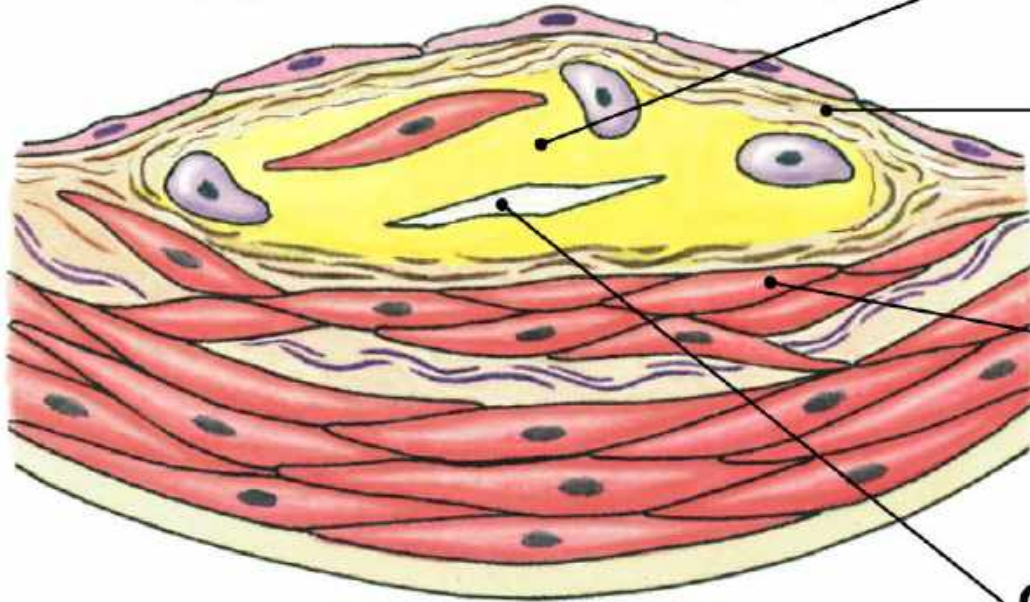


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**(c) Stable fibrous plaque**



**A lipid core accumulates beneath the endothelium.**

**Fibrous scar tissue cap forms to wall off the lipid core.**

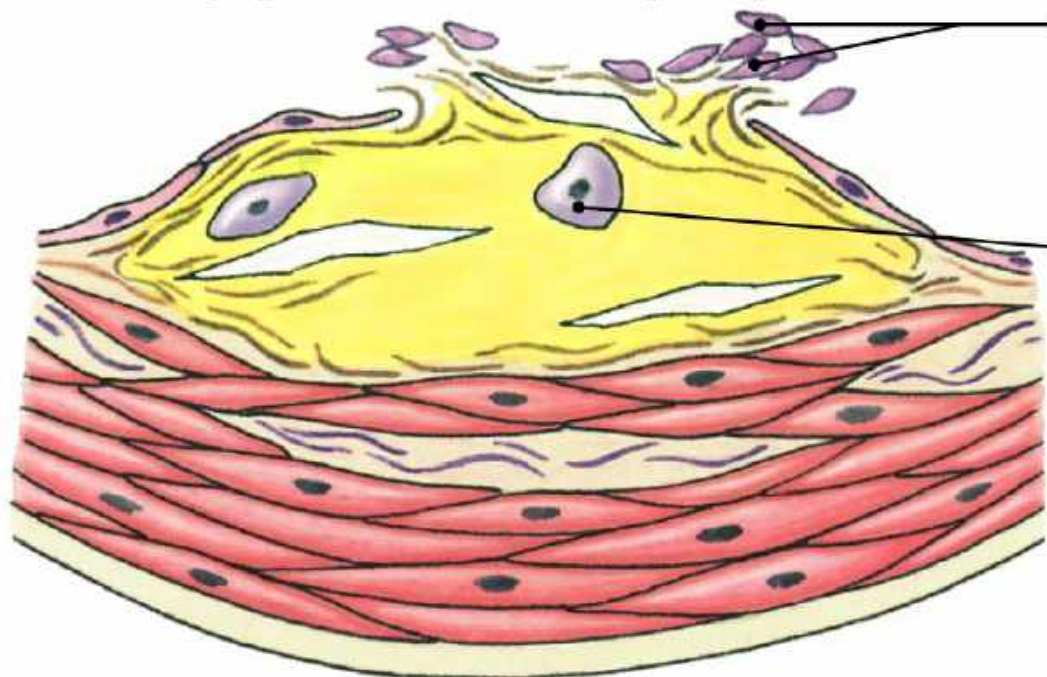
**Smooth muscle cells divide and contribute to thickening of the intima.**

**Calcifications are deposited within the plaque.**

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**(d) Vulnerable plaque**



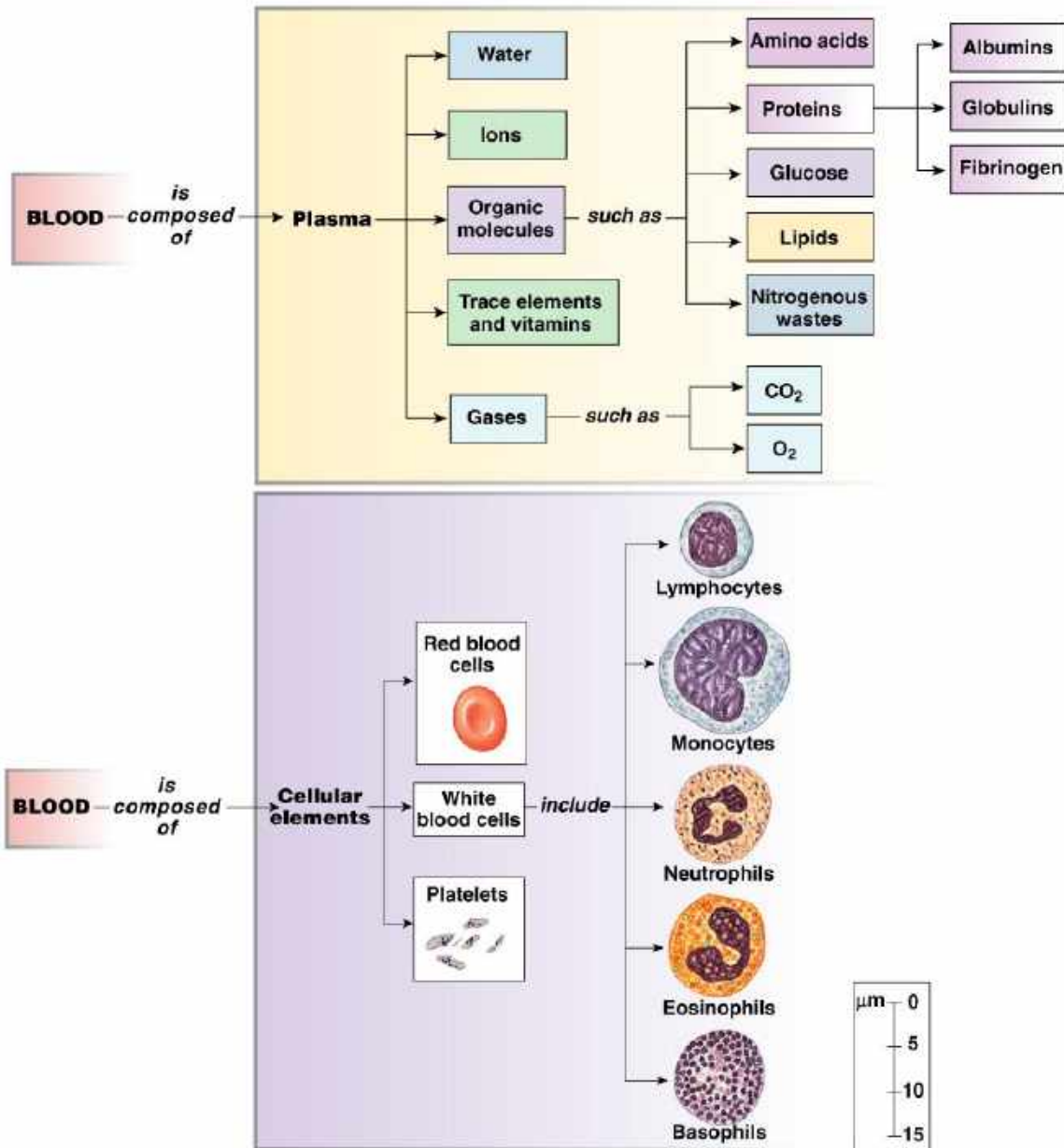
**Platelets that are exposed to collagen activate and initiate a blood clot.**

**Macrophages may release enzymes that dissolve collagen and convert stable plaques to unstable plaques.**

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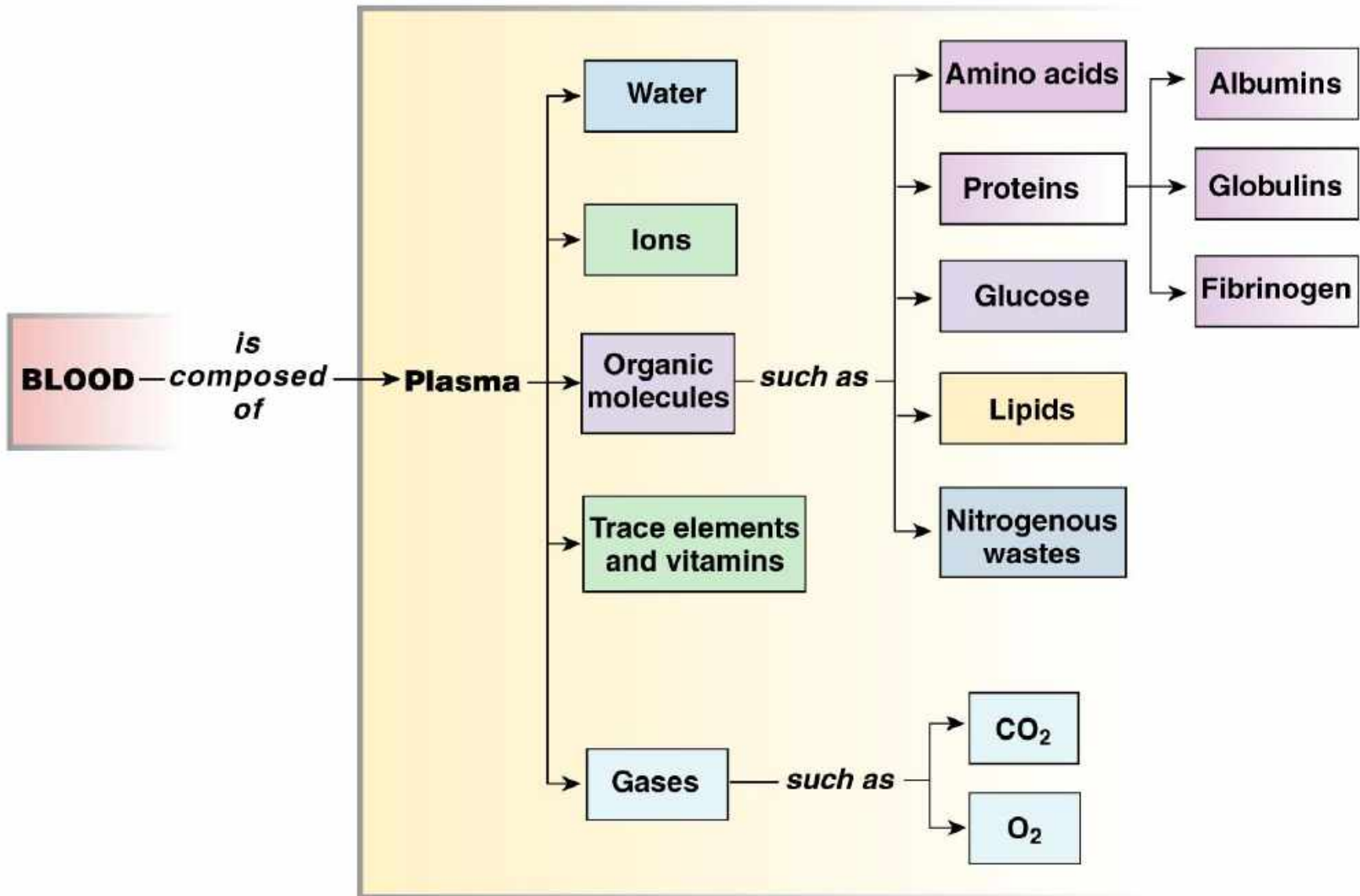
**BLOOD**

The word "BLOOD" is written in a bold, dark red, sans-serif font. The letters are thick and have a slight shadow or reflection effect below them, making them appear to float above a surface. The background is plain white.



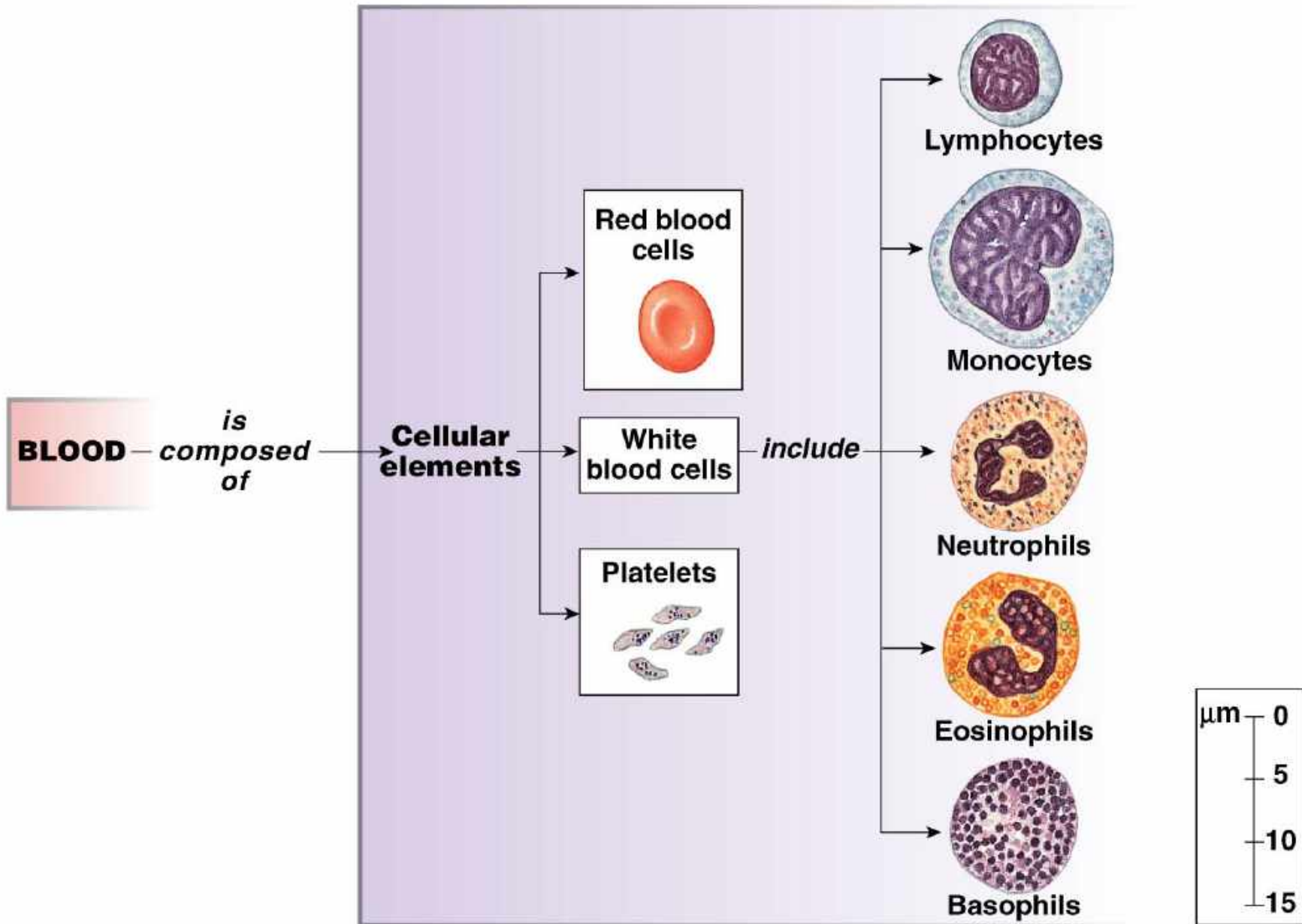
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Figure 16-1 - Overview (1 of 3)



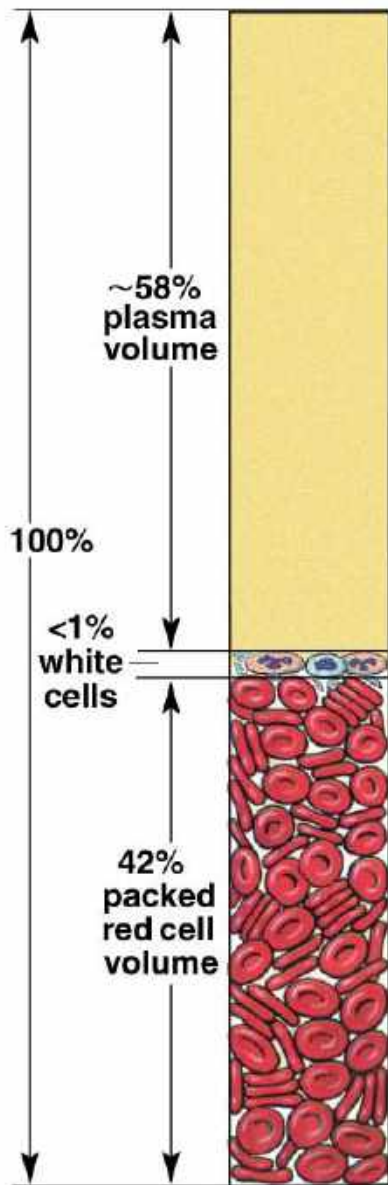
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Figure 16-1 (2 of 3)



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Figure 16-1 – Overview (3 of 3)

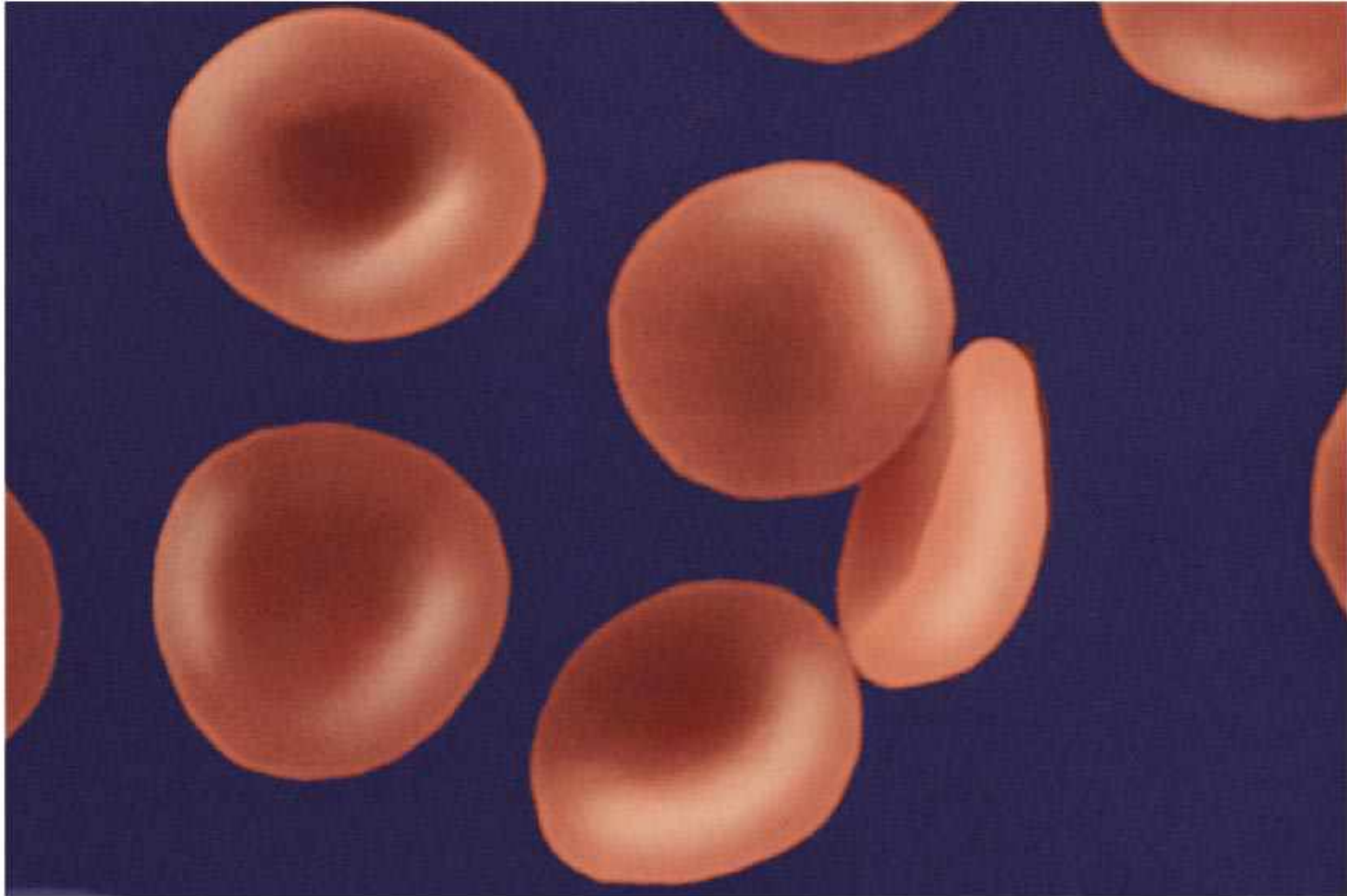


	Males	Females
Hematocrit	40–54%	37–47%
Hemoglobin (g Hb/dL* blood)	14–17	12–16
Red cell count (cells/ $\mu$ L)	$4.5\text{--}6.5 \times 10^6$	$3.9\text{--}5.6 \times 10^6$
Total white cell count (cells/ $\mu$ L)	$4\text{--}11 \times 10^3$	$4\text{--}11 \times 10^3$
Differential white cell count		
Neutrophils	50-70%	50-70%
Eosinophils	1-4%	1-4%
Basophils	<1%	<1%
Lymphocytes	20–40%	20–40%
Monocytes	2–8%	2–8%
Platelets (per $\mu$ L)	$200\text{--}500 \times 10^3$	$200\text{--}500 \times 10^3$
* 1 deciliter (dL) = 100 mL		

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Figure 16-3

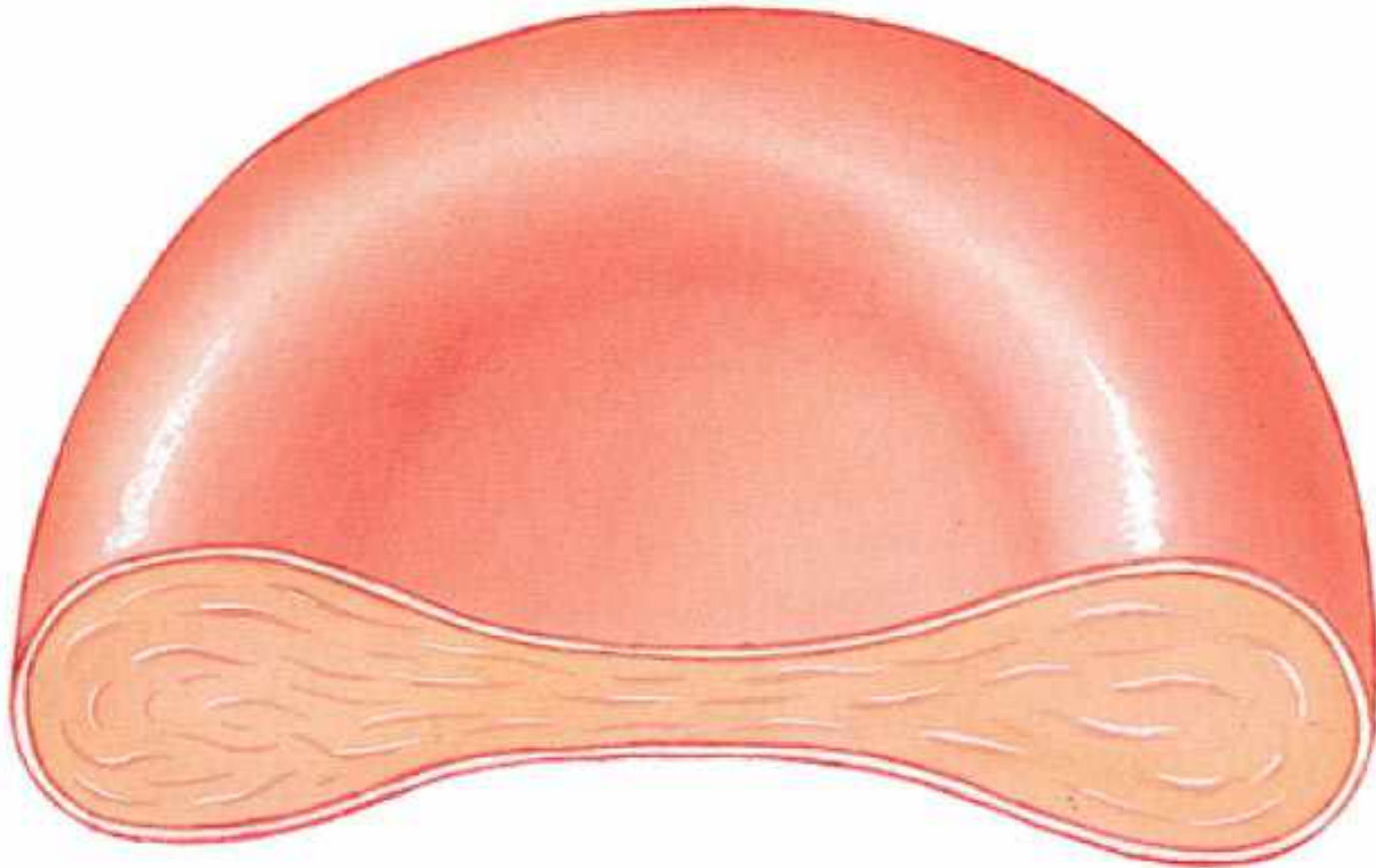
**(a) SEM shows biconcave disk shape of RBCs.**



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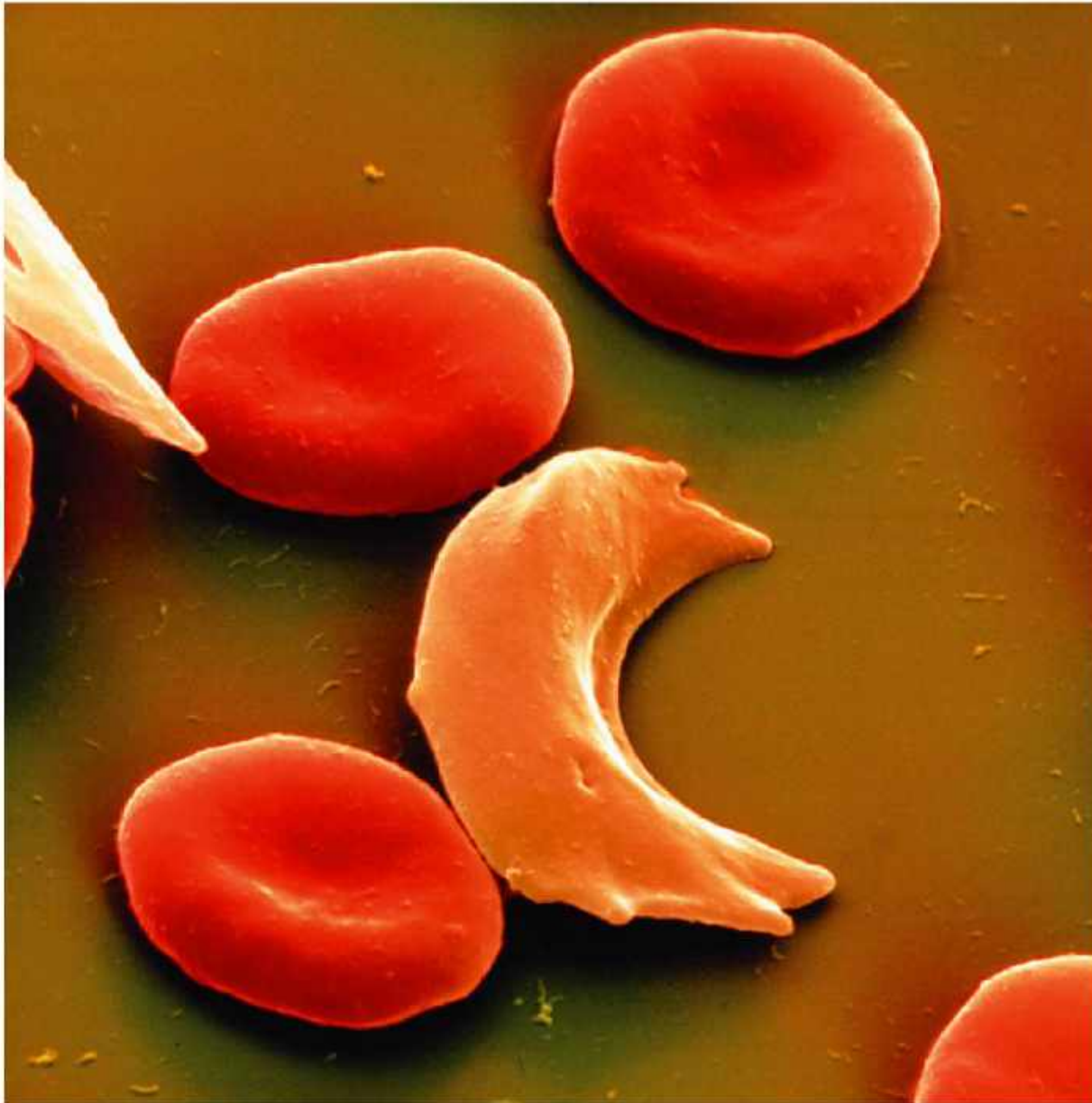
Figure 16-5a

## **(b) Cross-section of RBC**



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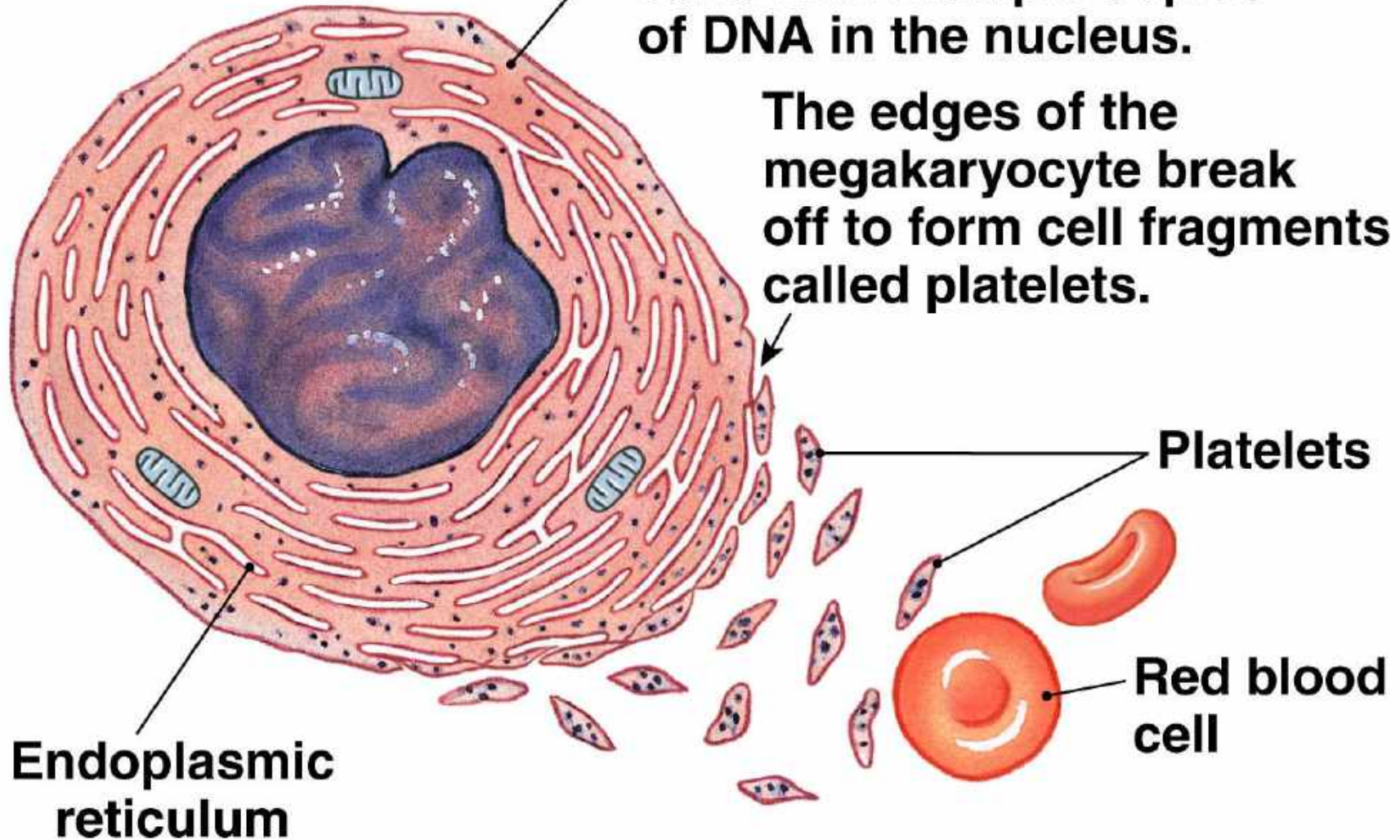
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Figure 16-8

**(a)**

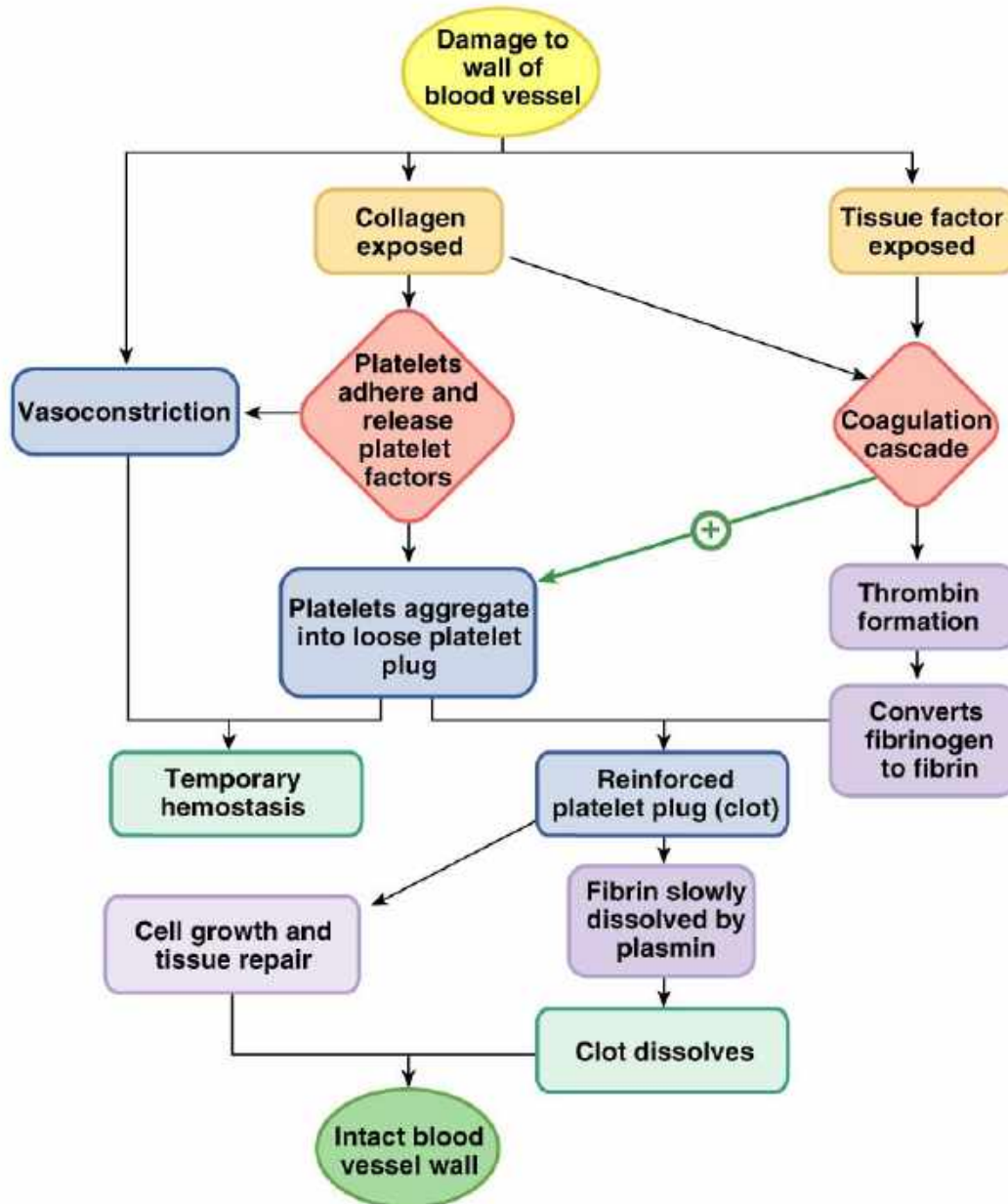
**Megakaryocytes are giant cells with multiple copies of DNA in the nucleus.**

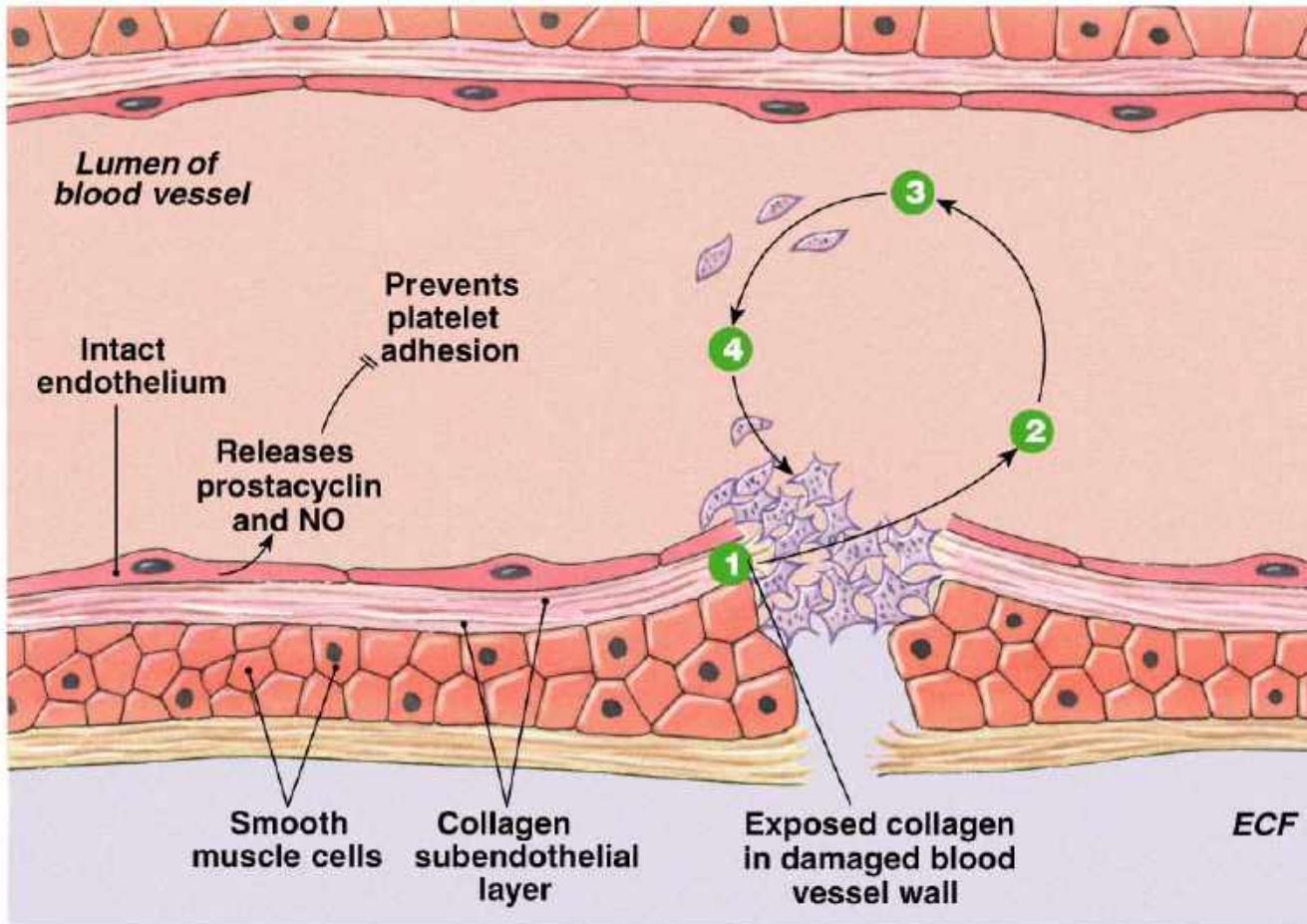
**The edges of the megakaryocyte break off to form cell fragments called platelets.**



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Figure 16-9a

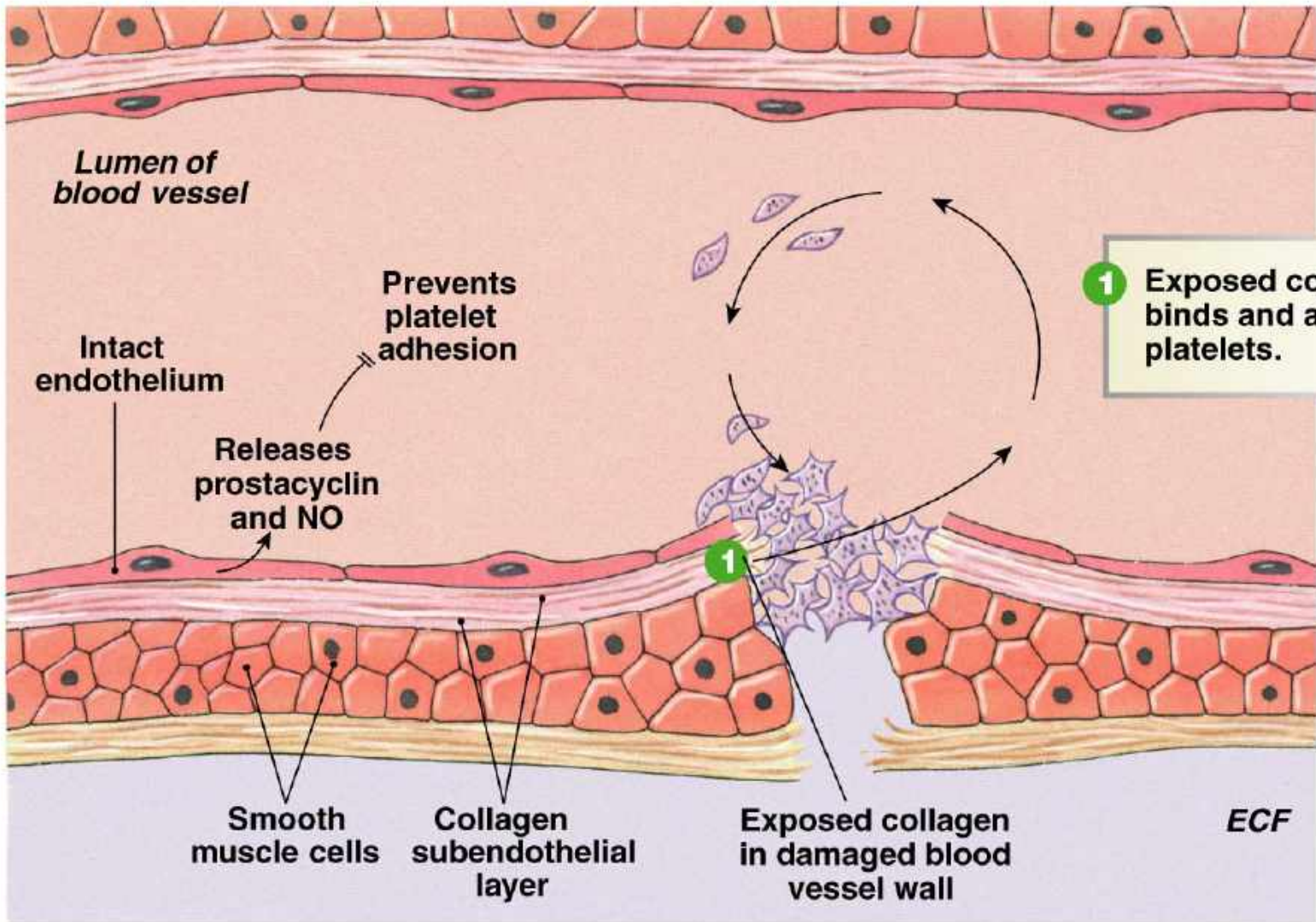




- 1 Exposed collagen binds and activates platelets.
- 2 Platelet factors are released.
- 3 Factors attract more platelets.
- 4 Platelets aggregate into platelet plug.

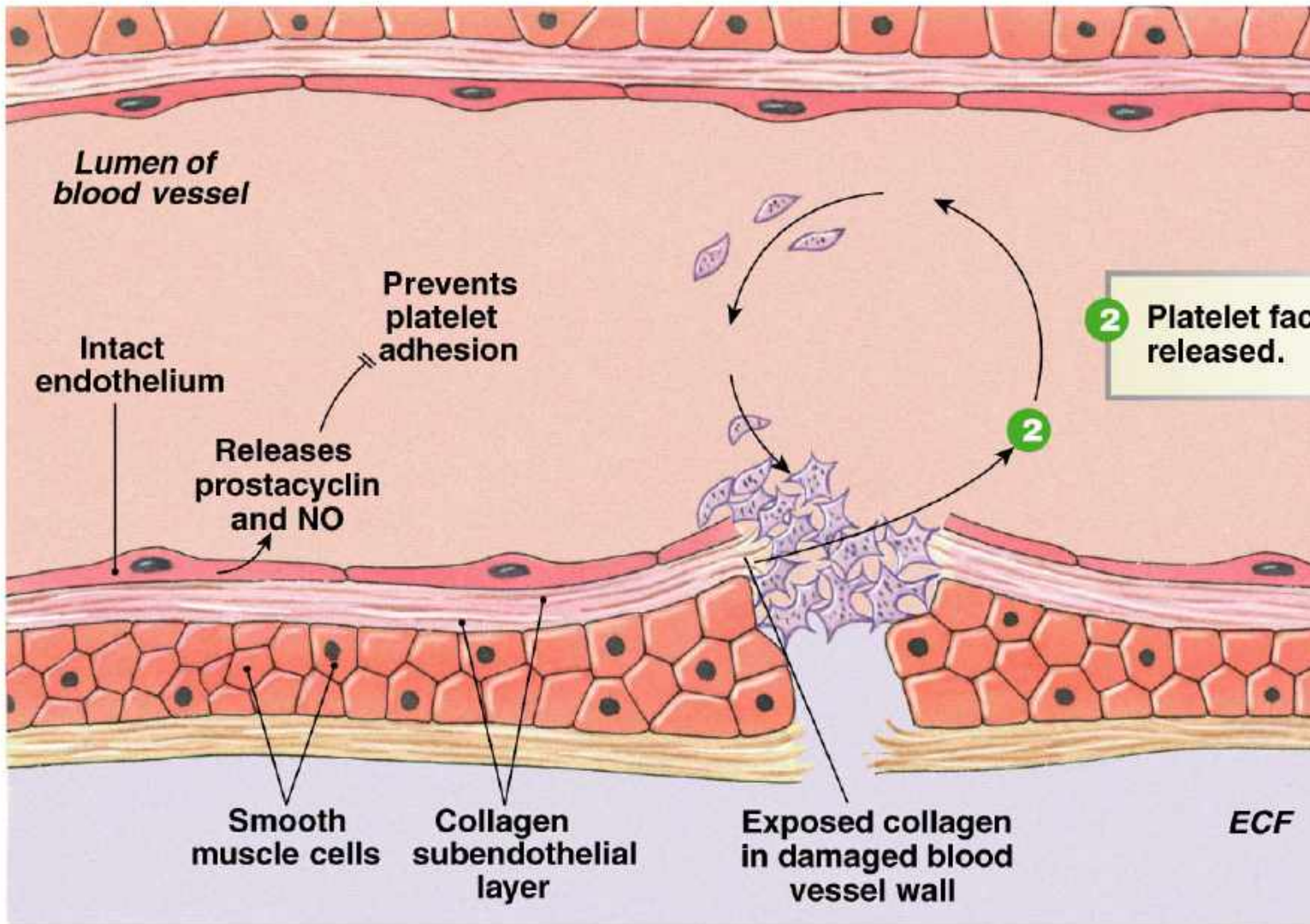
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Figure 16-11 - Overview



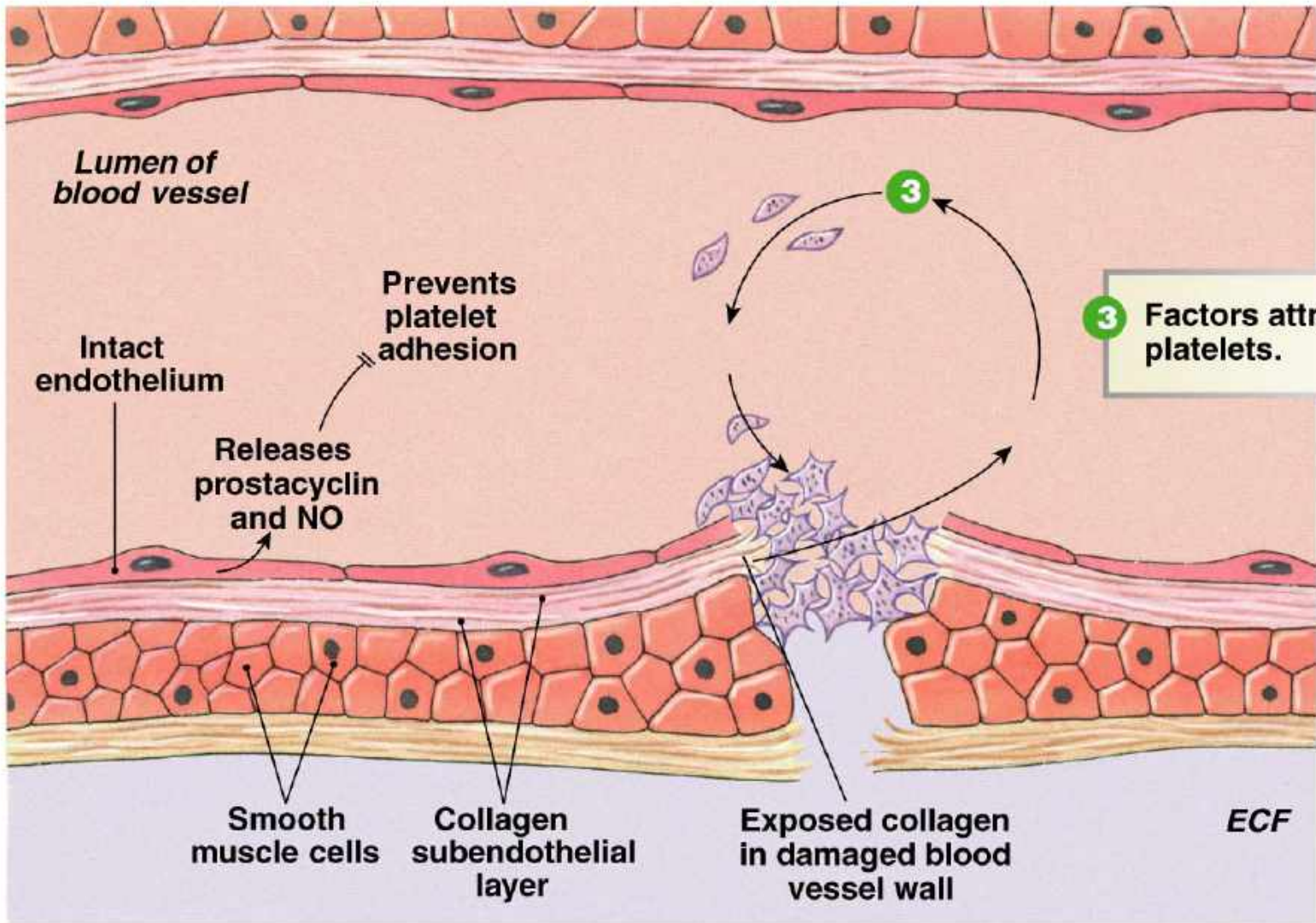
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Figure 16-11, step 1



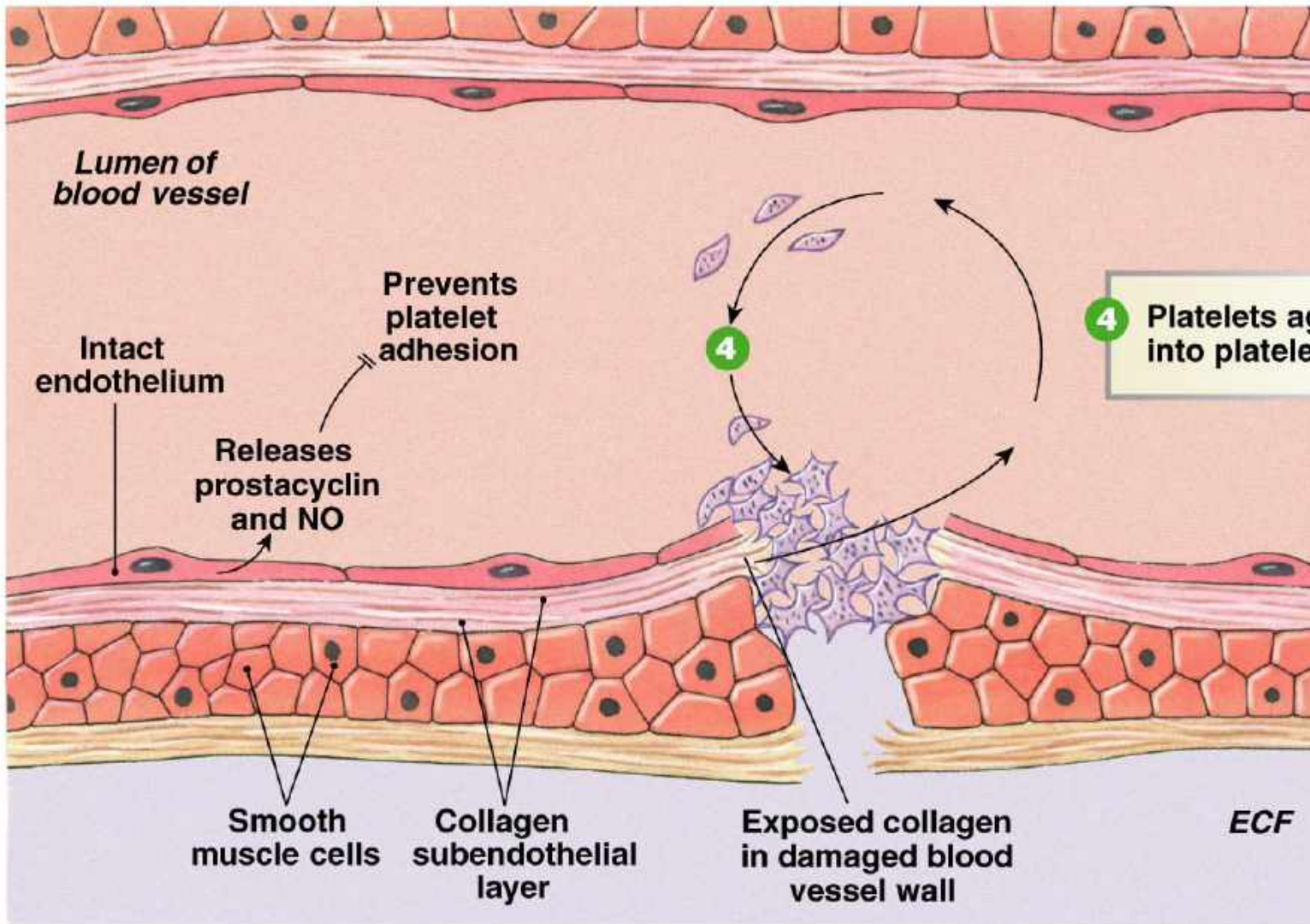
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Figure 16-11, step 2



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Figure 16-11, step 3



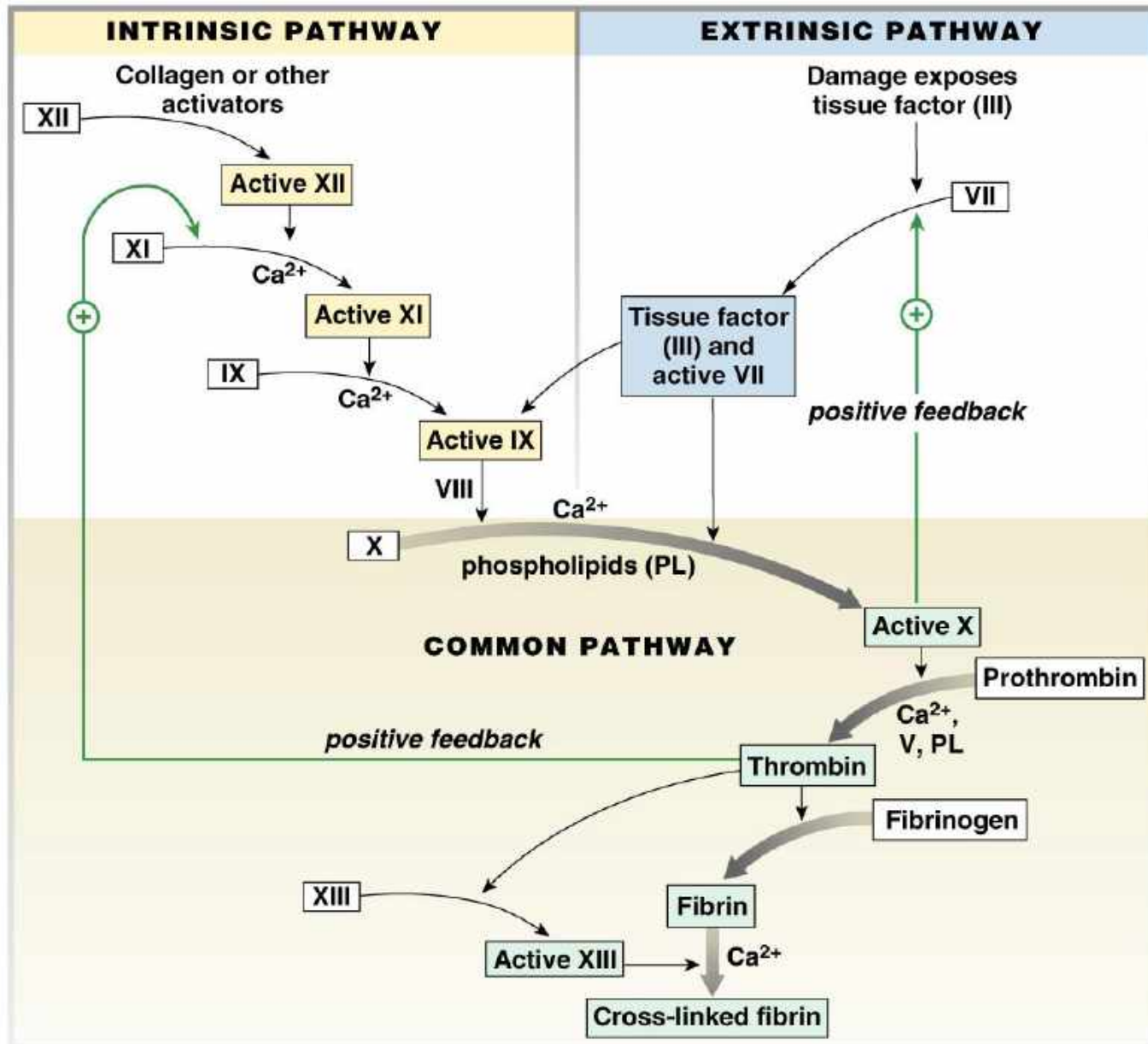
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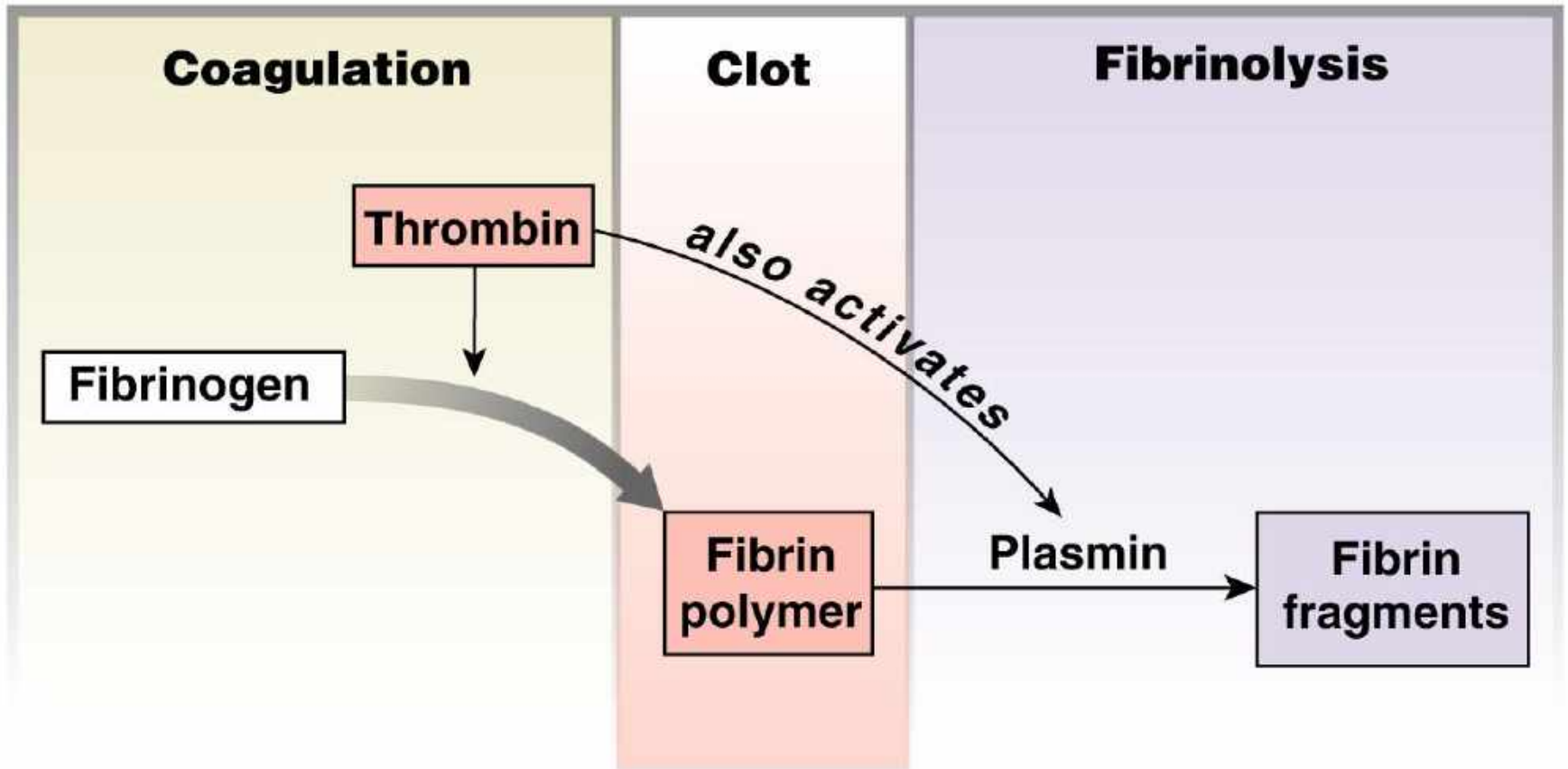
Figure 16-11, step 4



**TABLE 16-4** Factors Involved in Platelet Function

CHEMICAL FACTOR	SOURCE	ACTIVATED BY OR RELEASED IN RESPONSE TO	ROLE IN PLATELET PLUG FORMATION	OTHER ROLES AND COMMENTS
Collagen	Subendothelial extracellular matrix	Injury exposes platelets to collagen	Binds platelets to begin platelet plug	—
von Willebrand factor (vWF)	Endothelium, megakaryocytes	Exposure to collagen	Links platelets to collagen	Deficiency or defect causes prolonged bleeding
Serotonin	Secretory vesicles of platelets	Platelet activation	Platelet aggregation	Vasoconstrictor
Adenosine diphosphate (ADP)	Platelet mitochondria	Platelet activation, thrombin	Platelet aggregation	—
Platelet-activating factor (PAF)	Platelets, neutrophils, monocytes	Platelet activation	Platelet aggregation	Plays role in inflammation; increases capillary permeability
Thromboxane A <sub>2</sub>	Phospholipids in platelet membranes	Platelet activating factor	Platelet aggregation	Vasoconstrictor; eicosanoid
Platelet-derived growth factor (PDGF)	Platelets	Platelet activation	—	Promotes wound healing by attracting fibroblasts and smooth muscle cells





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Figure 16-13

**TABLE 16-5** Factors Involved in Coagulation

CHEMICAL FACTOR	SOURCE	ACTIVATED BY OR RELEASED IN RESPONSE TO	ROLE IN COAGULATION	OTHER ROLES AND COMMENTS
Collagen	Subendothelial extracellular matrix	Injury that exposes collagen to plasma clotting factors	Starts intrinsic pathway	NA
von Willebrand factor (vWF)	Endothelium, megakaryocytes	Exposure to collagen	Regulates level of factor VIII	Deficiency or defect causes prolonged bleeding
Kininogen and kallikrein	Liver and plasma	Cofactors normally present in plasma pathway	Cofactors for contact activation of intrinsic pathway	Mediate inflammatory response; enhance fibrinolysis
Tissue factor (tissue thromboplastin or factor III)	Most cells except platelets	Damage to tissue	Starts extrinsic pathway	NA
Prothrombin and thrombin (factor II)	Liver and plasma	Platelet lipids, Ca <sup>2+</sup> , and factor V	Fibrin production	NA
Fibrinogen and fibrin (factor I)	Liver and plasma	Thrombin	Form insoluble fibers that stabilize platelet plug	NA
Fibrin-stabilizing factor (XIII)	Liver, megakaryocytes	Platelets	Cross-links fibrin polymers to make stable mesh	NA
Ca <sup>2+</sup> (factor IV)	Plasma ions	NA	Required for several steps of coagulation cascade	Never a limiting factor
Vitamin K	Diet	NA	Needed for synthesis of factors II, VII, IX, X	NA

**TABLE 16-6** Endogenous Factors Involved in Fibrinolysis and Anticoagulation

CHEMICAL FACTOR	SOURCE	ACTIVATED BY OR RELEASED IN RESPONSE TO	ROLE IN ANTICOAGULATION OR FIBRINOLYSIS	OTHER ROLES AND COMMENTS
Plasminogen and plasmin	Liver and plasma	t-PA and thrombin	Dissolves fibrin and fibrinogen	NA
Tissue plasminogen activator (t-PA)	Many tissues	Normally present; levels increase with stress, protein C	Activates plasminogen	Recombinant t-PA used clinically to dissolve clots
Antithrombin III	Liver and plasma	NA	Anticoagulant; blocks factors IX, X, XI, XII, thrombin, kallikrein	Facilitated by heparin; no effect on thrombin despite name
Prostacyclin (prostaglandin I <sub>2</sub> or PGI <sub>2</sub> )	Endothelial cells	NA	Blocks platelet aggregation	Vasodilator

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