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Venous Tone and Venous Return

M1 – Cardiovascular/Respiratory Sequence

Louis D'Alecy, Ph.D.

Fall 2008

Friday 11/07/08, 9:00

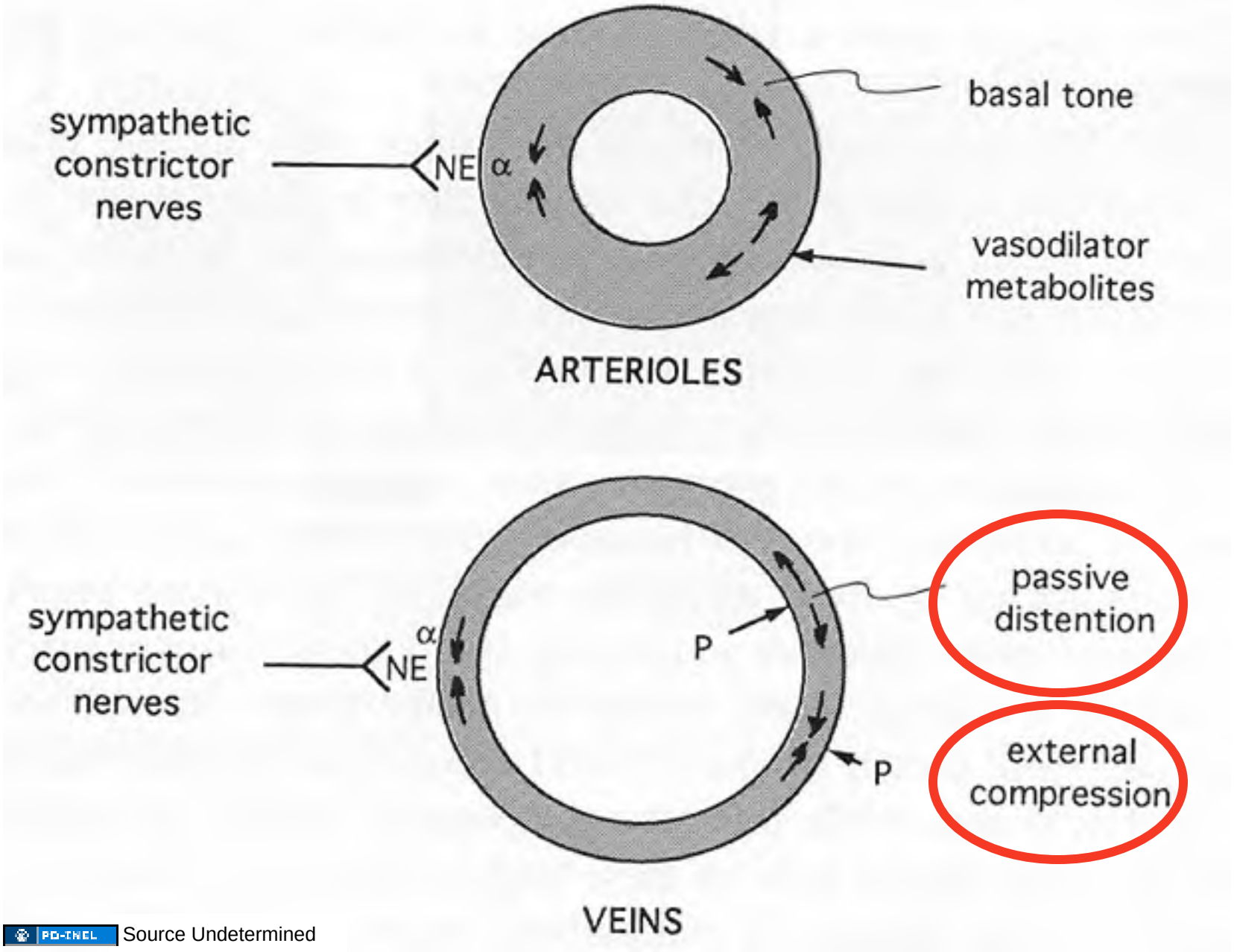
Venous Tone & Venous Return

25 slides, 50 minutes

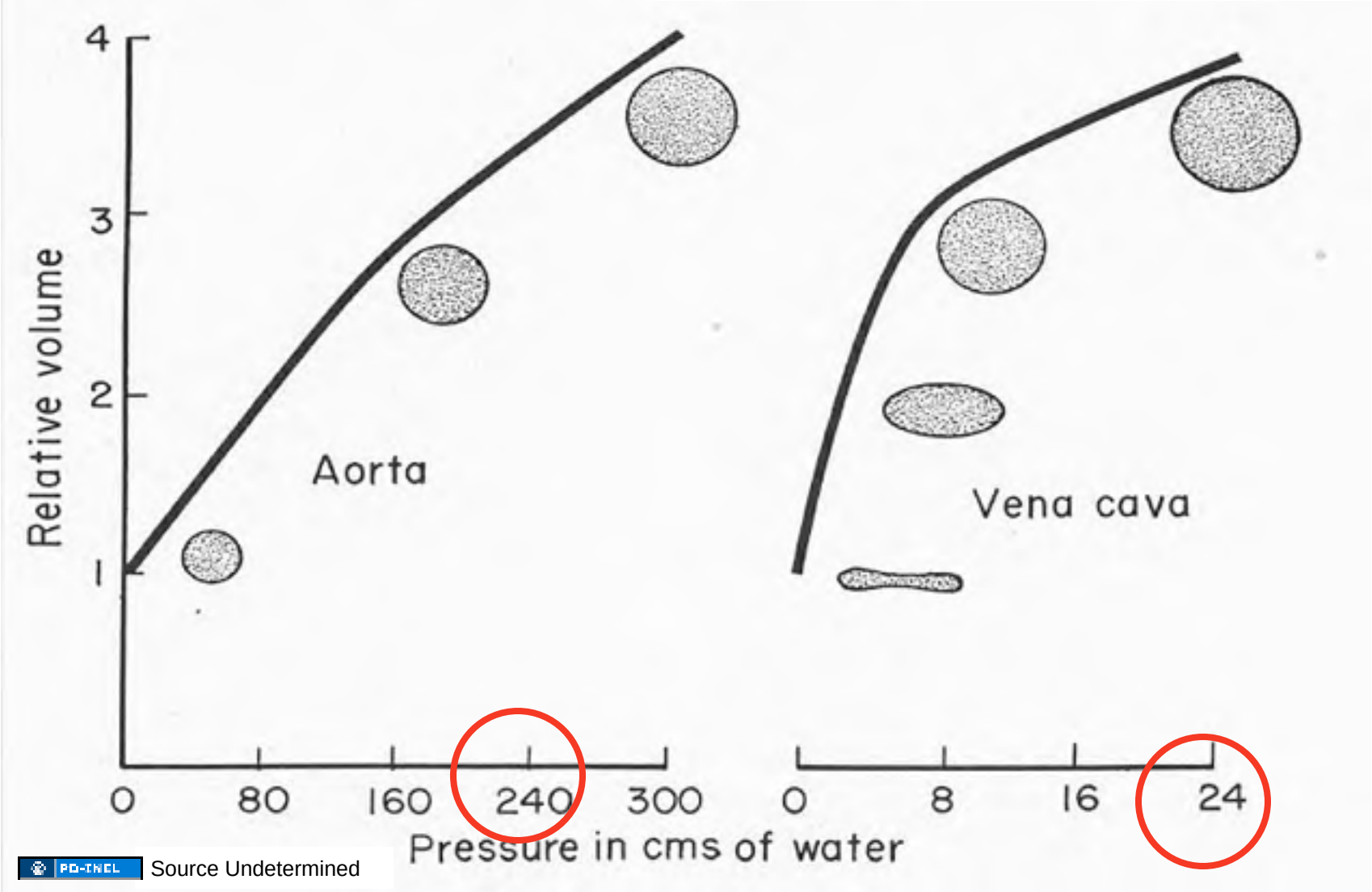
1. Venous Return vs. cardiac output
2. Family of Venous Return Curves
3. Family of Cardiac Function Curves
4. Review
   - Flow Mediated Dilation (FMD)
   - Coronary Flow Reserve

Reflex Influences

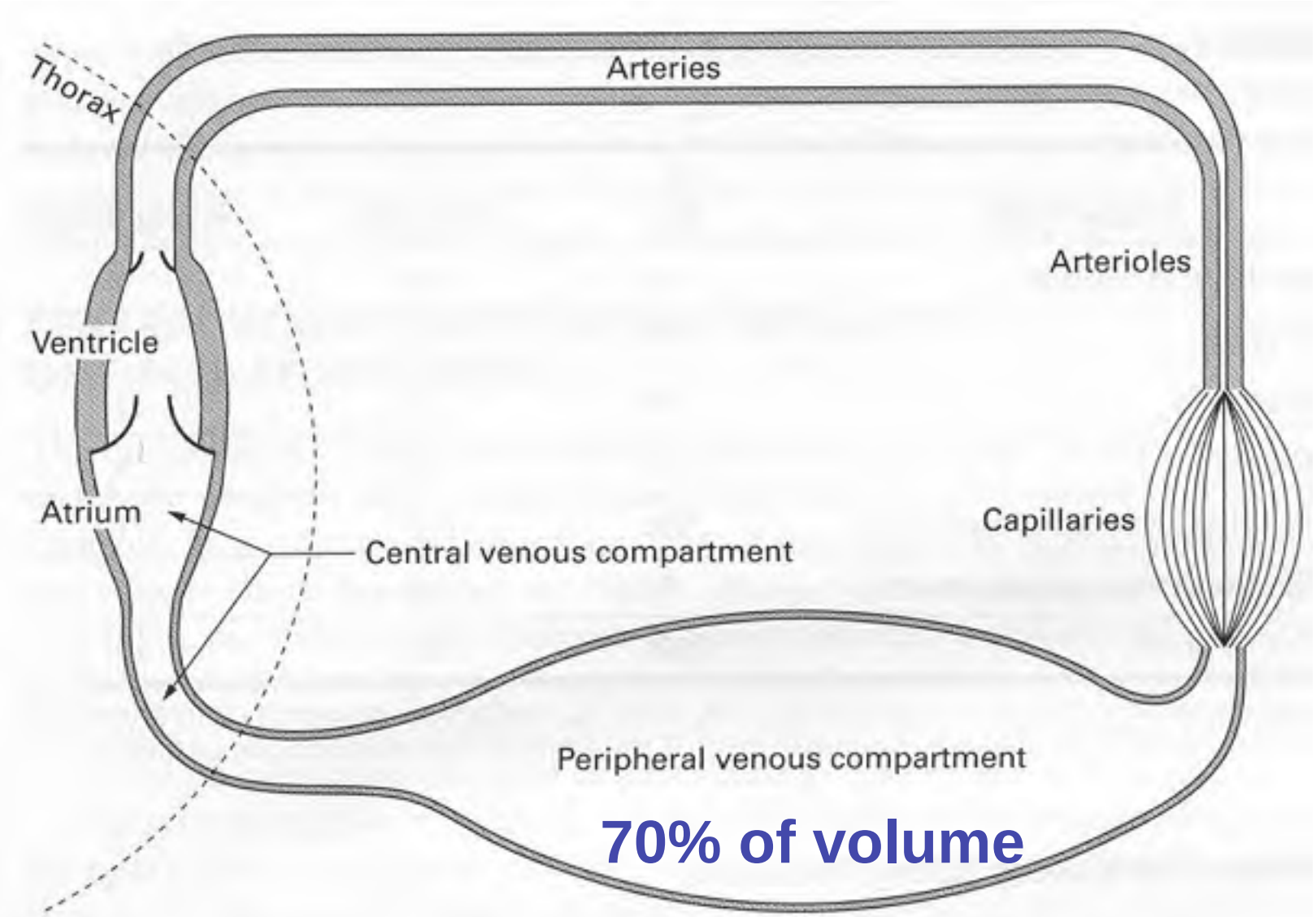
Local Influences



Slight increases in venous pressure produces large increases in venous volume.



M&H Fig 8.1 “Simplified” CVS



No right heart and no lungs -- in case you missed it!

 Mohrman and Heller. Cardiovascular Physiology. McGraw-Hill, 2006. 6th ed.

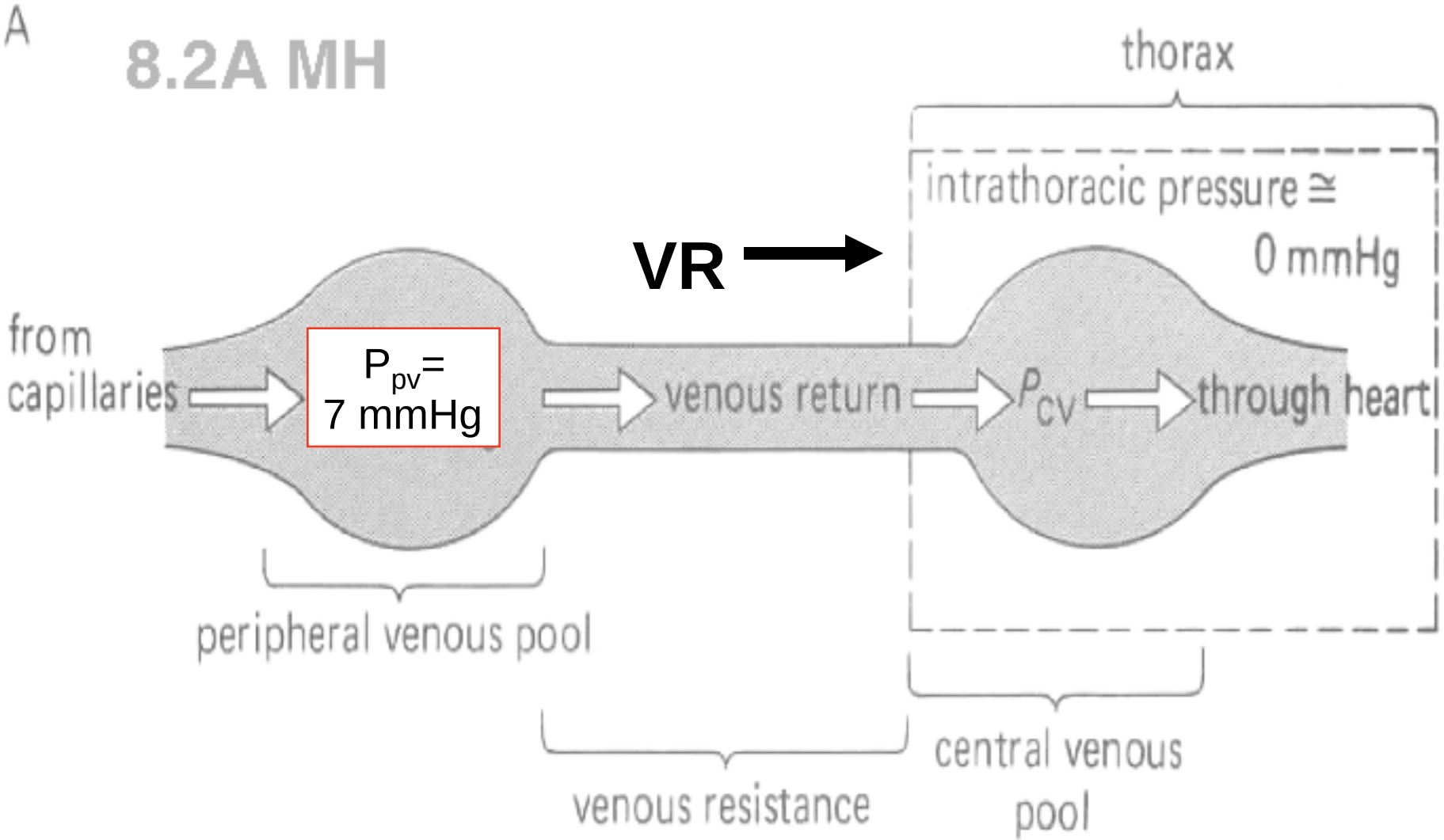
Circuit Properties

V_0= Vol @zero P, C = Compliance, R = Resistance

Compartment	V_0 mL		C mL/mmHg	R mmHg/(L/min)
Ventricle in diastole	30		24	0
Arteries	600		2	1
Arterioles	100		0	13
Capillaries	250		0	5
Peripheral venous compartment	2500	70%	110	1
Central venous compartment	80		4	0
Entire circuit	3560		140	20

*Values are for a normal, young, resting 70-kg adult. V_0, anatomical volume of compartment at zero pressure: C, compliance of compartment; R, resistance to flow through compartment.

PD-INEL Mohrman and Heller. Cardiovascular Physiology. McGraw-Hill, 2006. 6th ed.



Source: McGraw-Hill

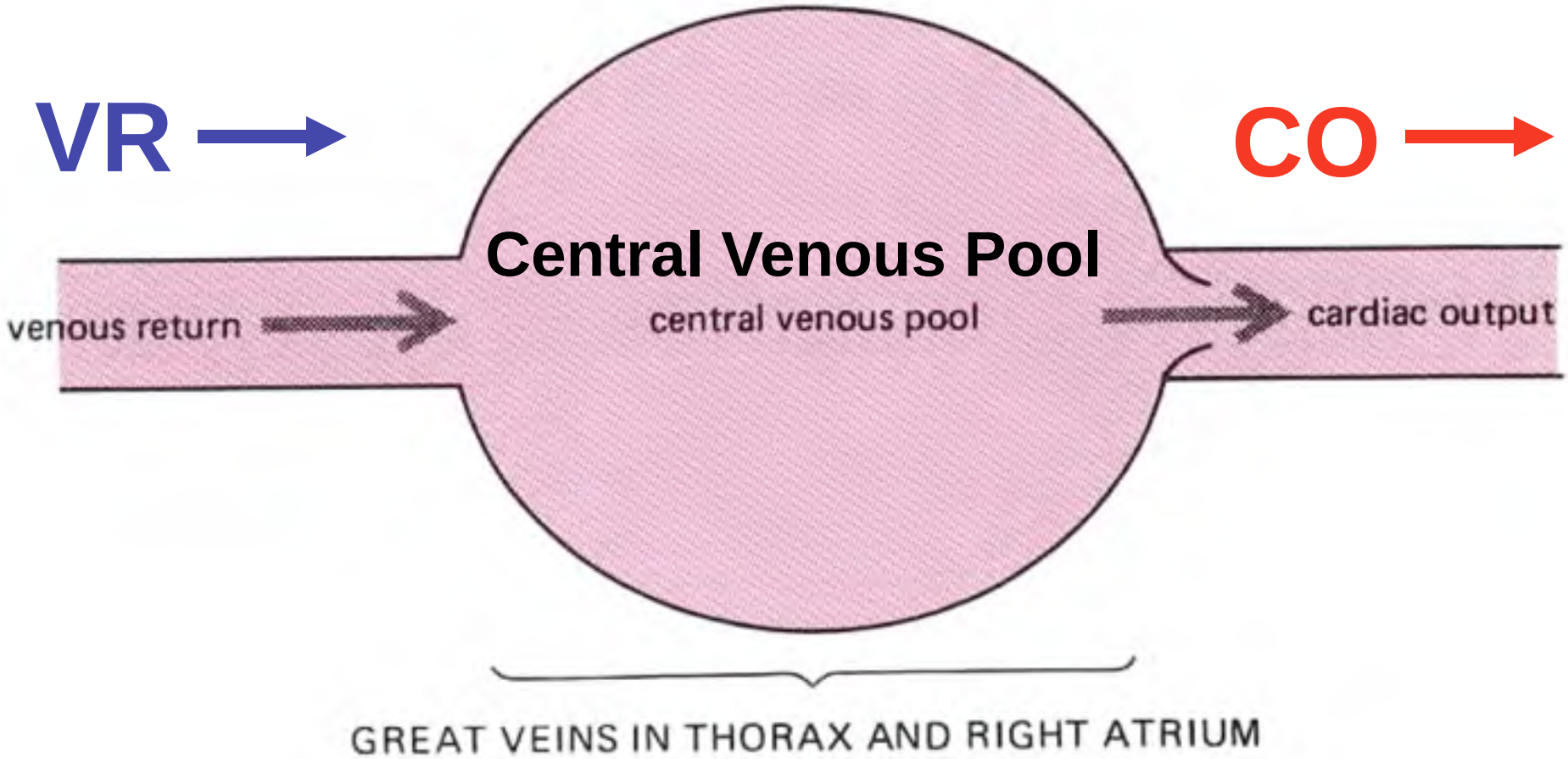
7 mmHg → 0 mmHg

Peripheral Venous Pressure — Central Venous Pressure

PD-INCL Mohrman and Heller. Cardiovascular Physiology. McGraw-Hill, 2006. 6th ed.

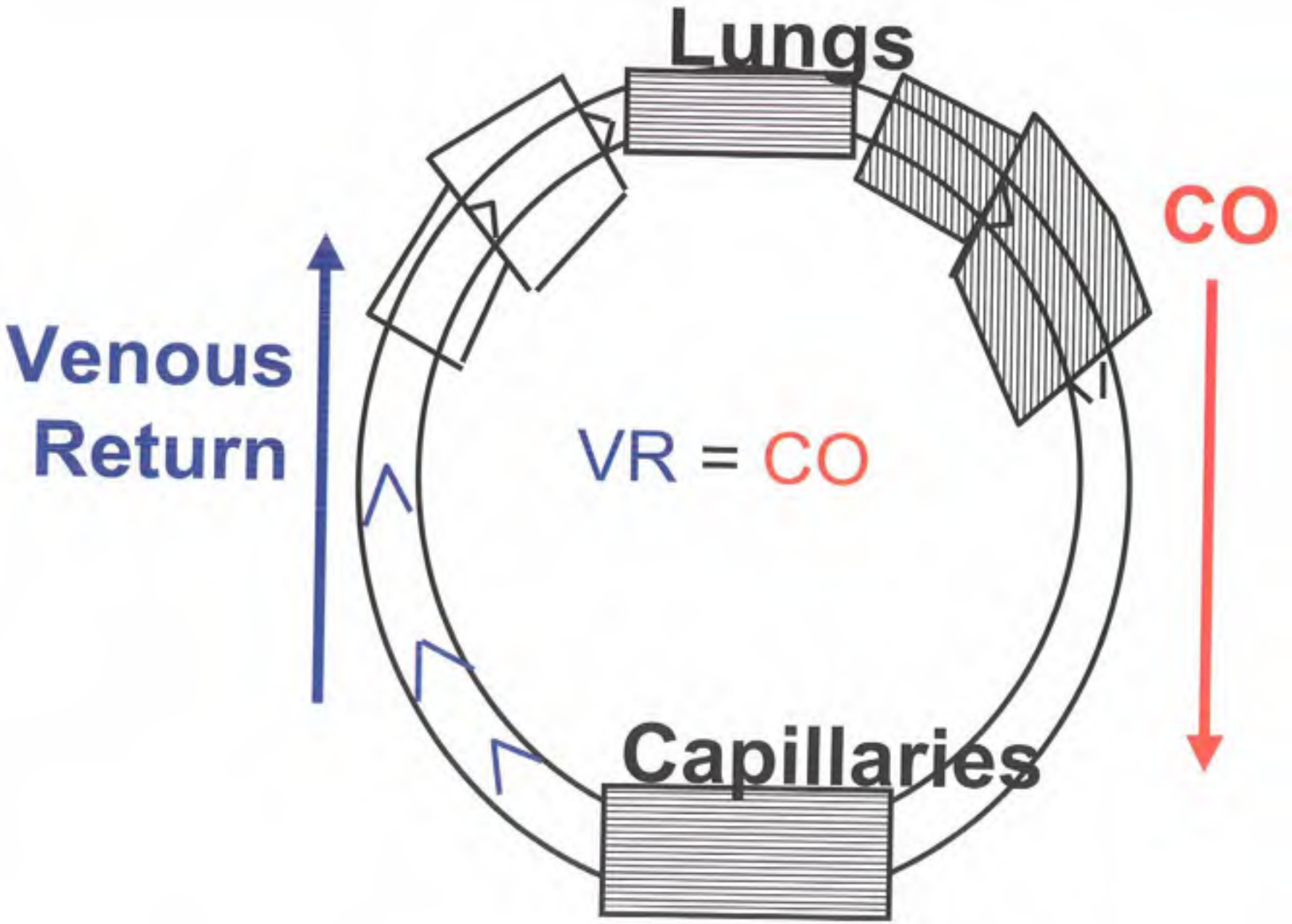
VR fills the central venous pool and

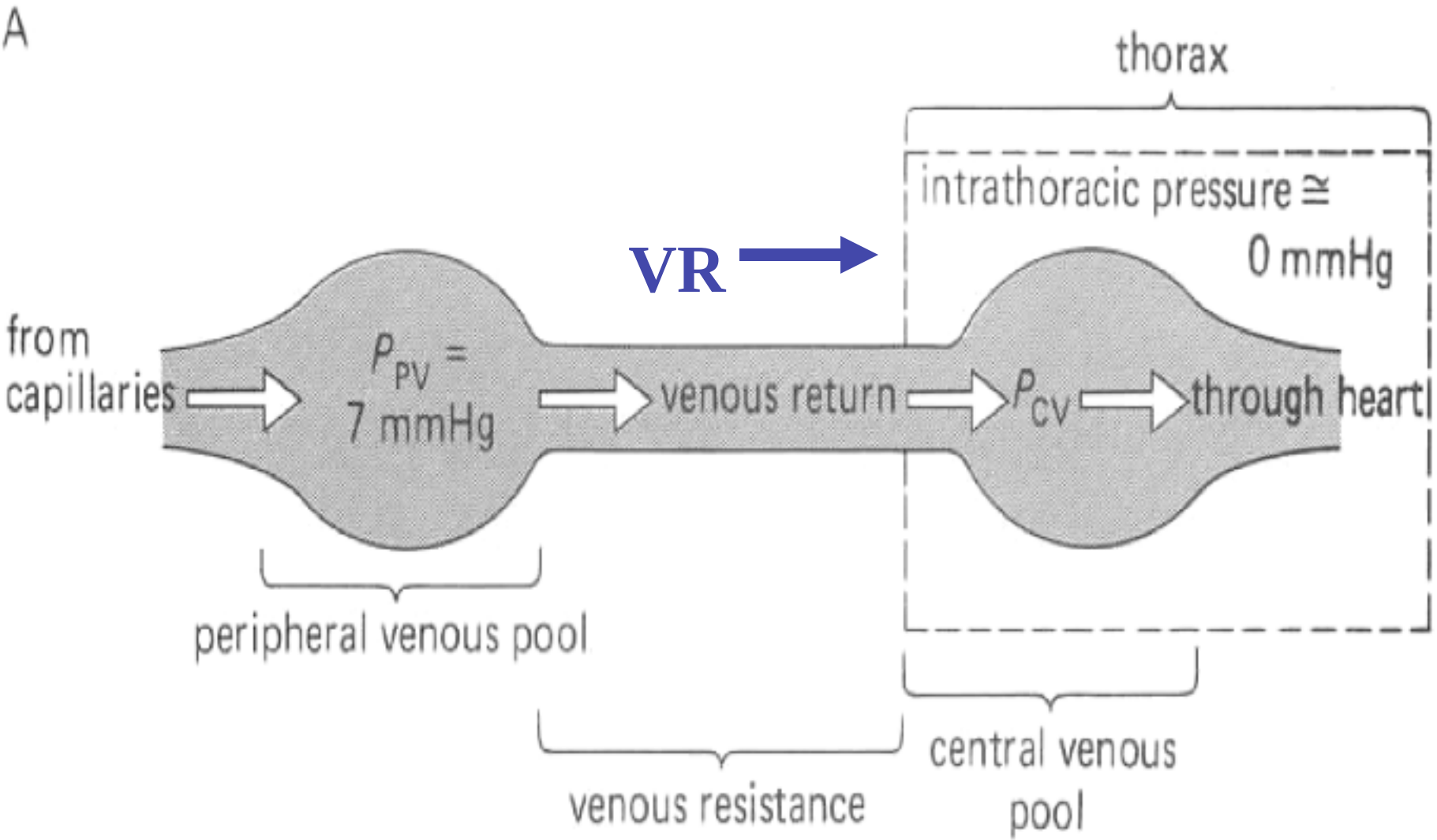
CO empties the central venous pool.



Great Veins in Thorax and Right Atrium

M&H 8.2


Lungs
CO
Venous Return
VR = CO
Capillaries



PD-ineL Mohrman and Heller. Cardiovascular Physiology. McGraw-Hill, 2006. 6th ed.

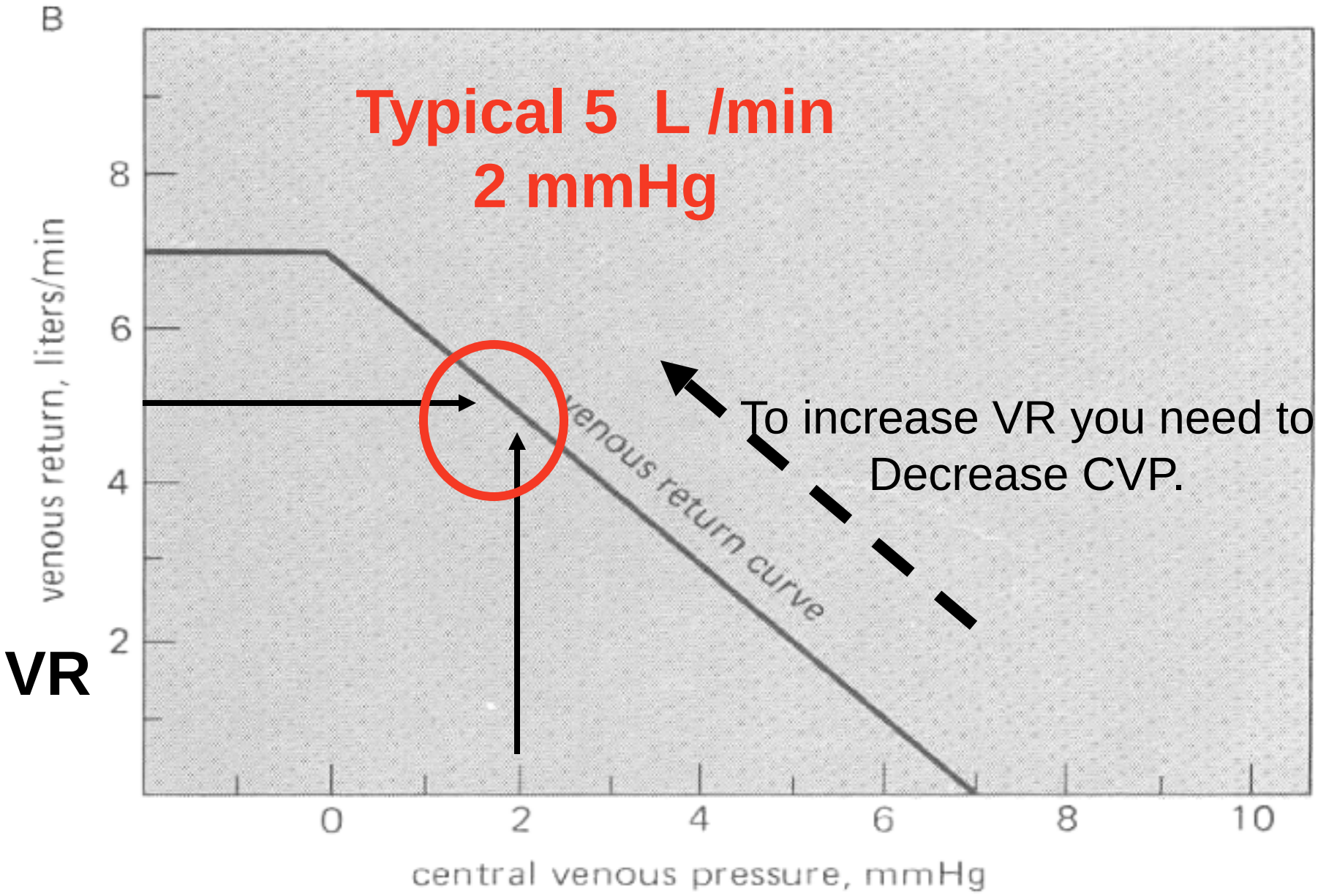
7 mmHg → 0 to 2 mmHg

Posture?

Muscle move?

Ventilation??

Volume

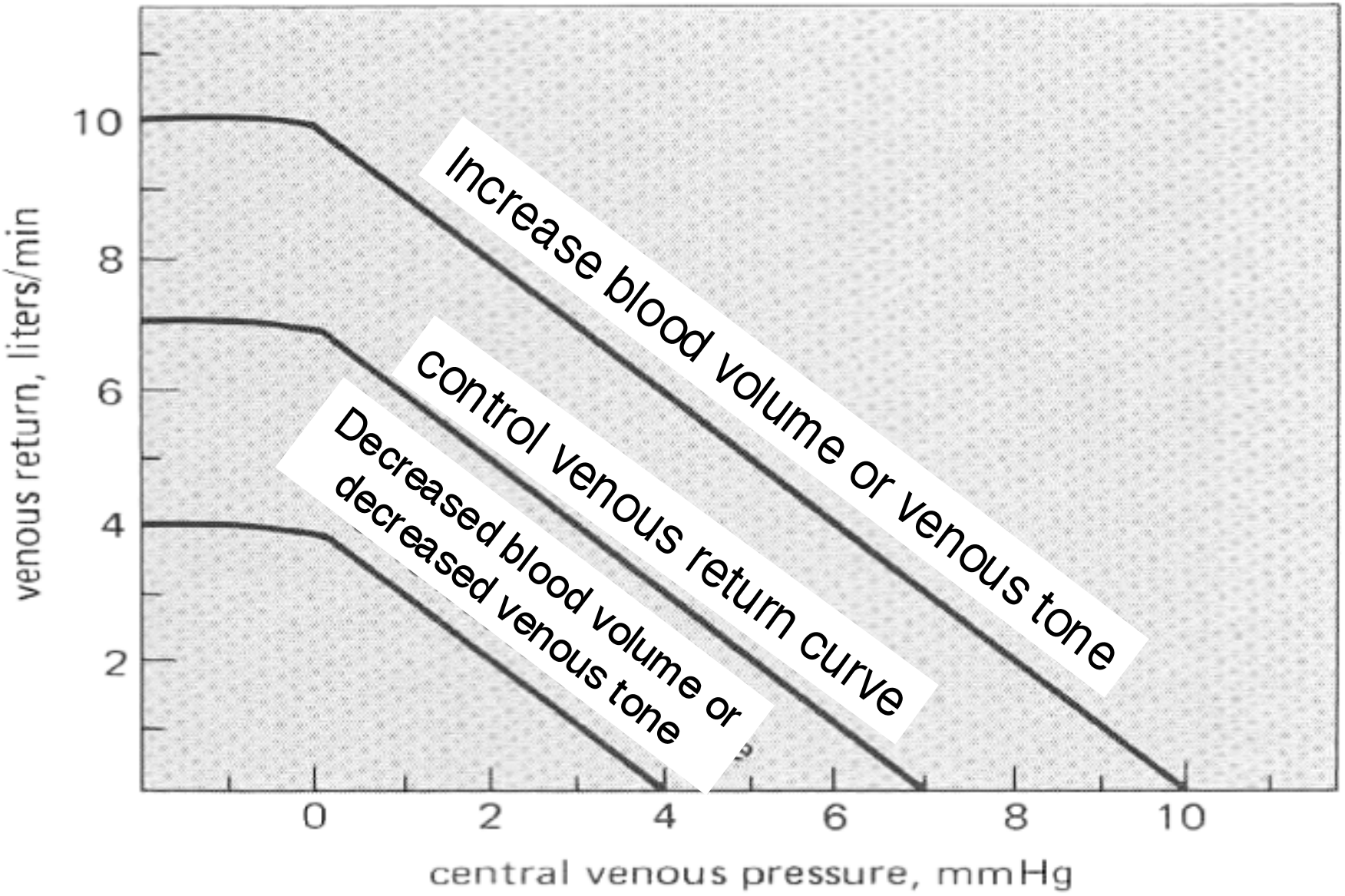


VR

CVP, mmHg

8.3 MH

 Mohrman and Heller. Cardiovascular Physiology. McGraw-Hill, 2006. 6th ed.



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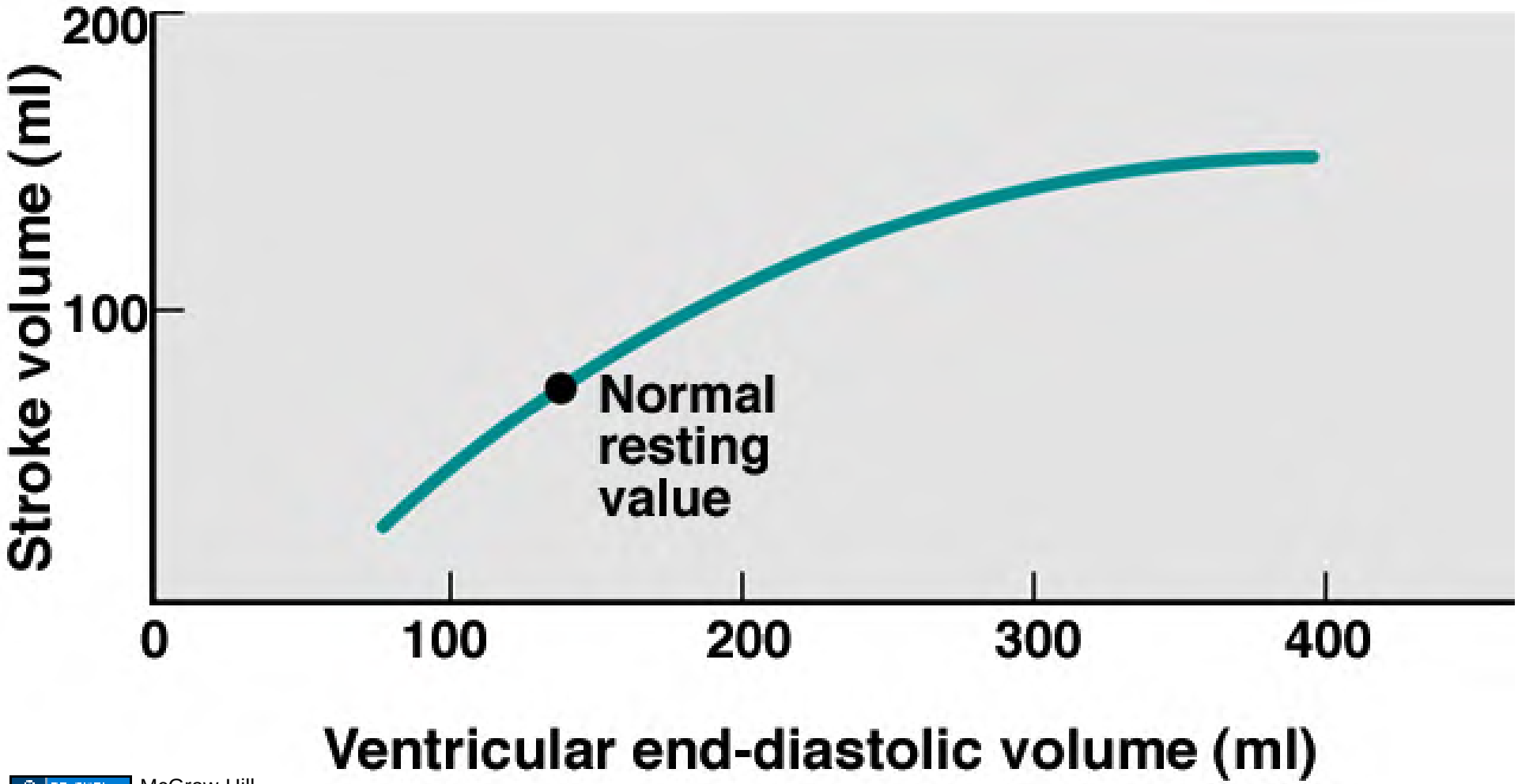
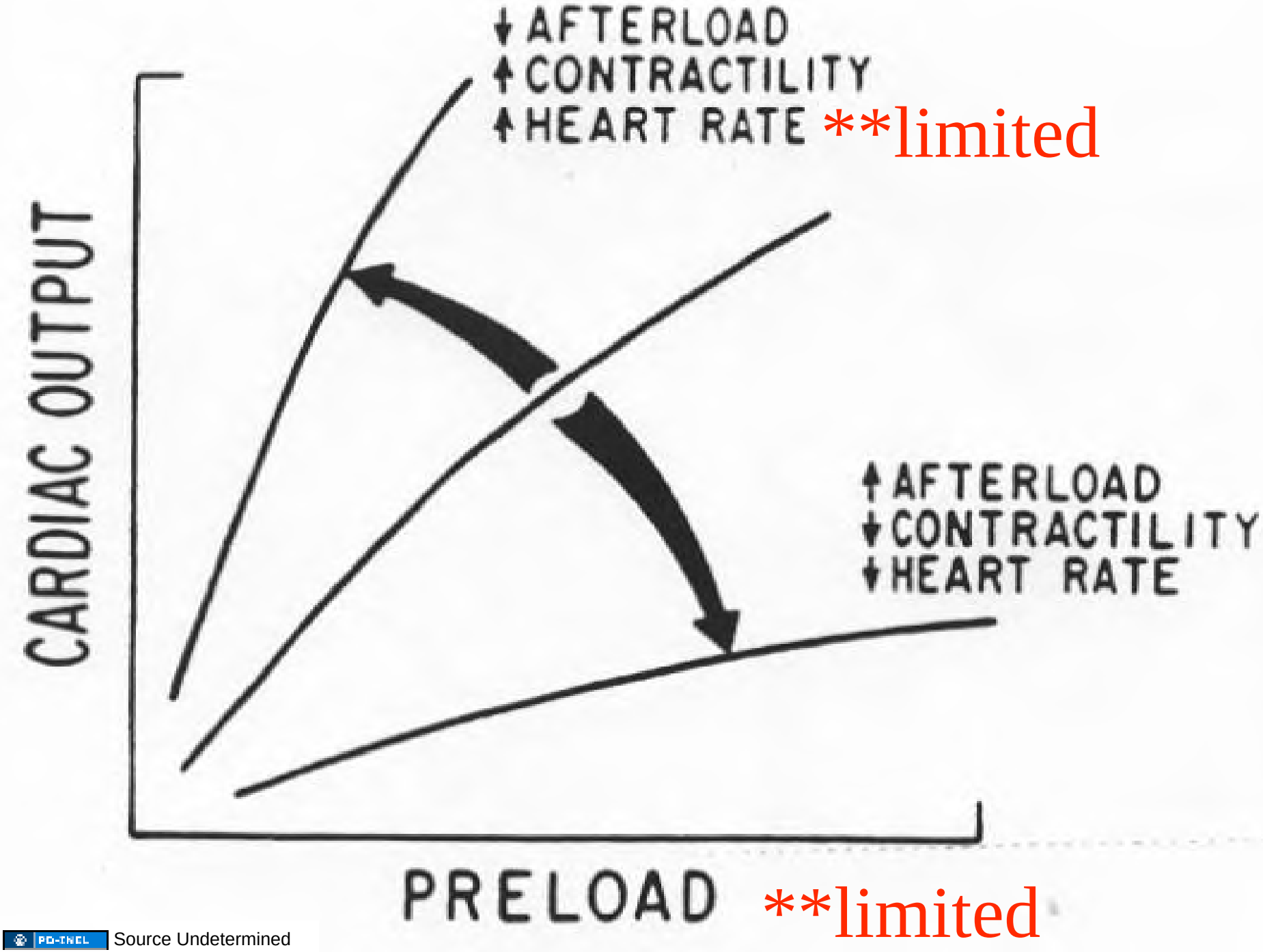
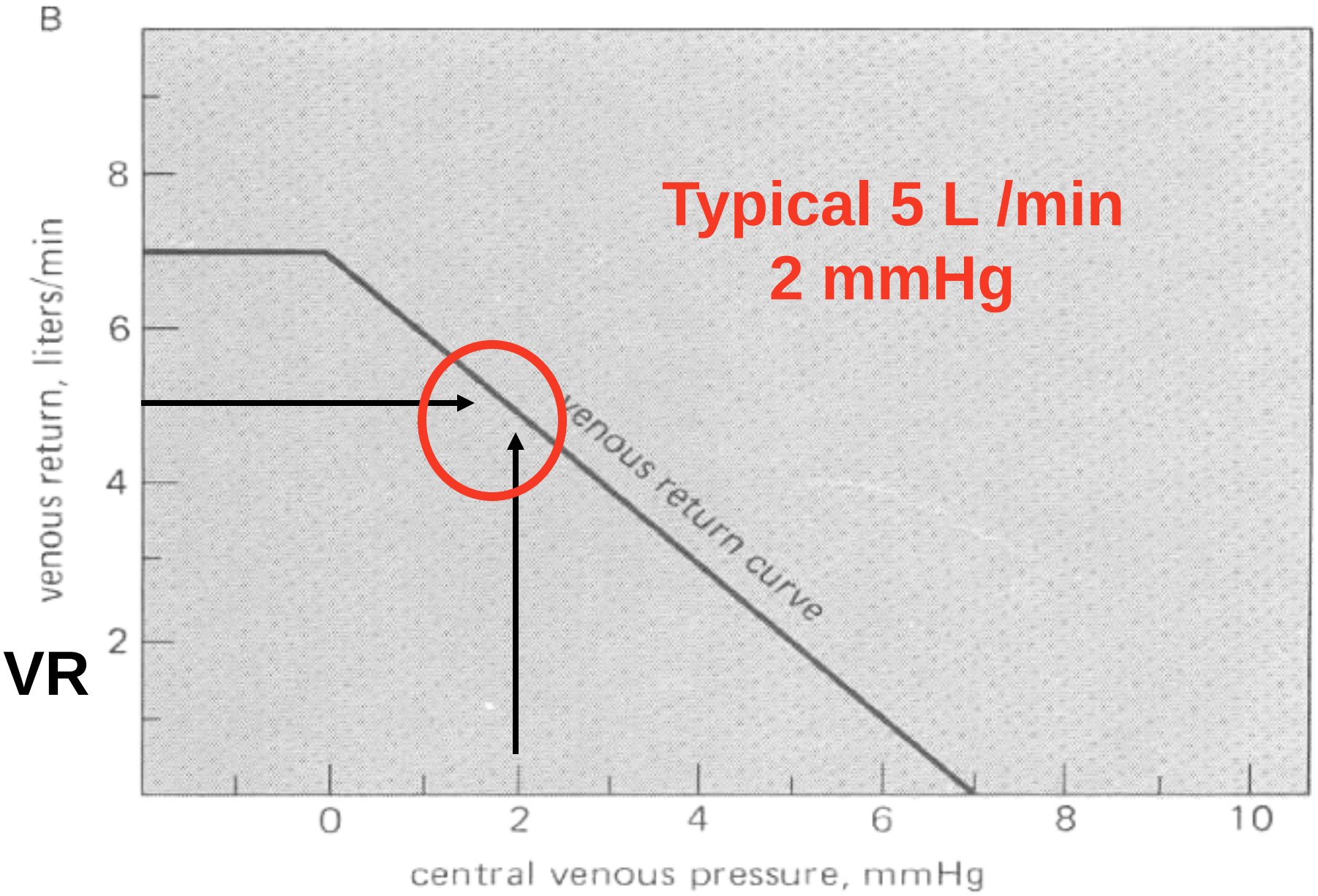
 Mohrman and Heller. Cardiovascular Physiology. McGraw-Hill, 2006. 6th ed.

Ventricular function curve





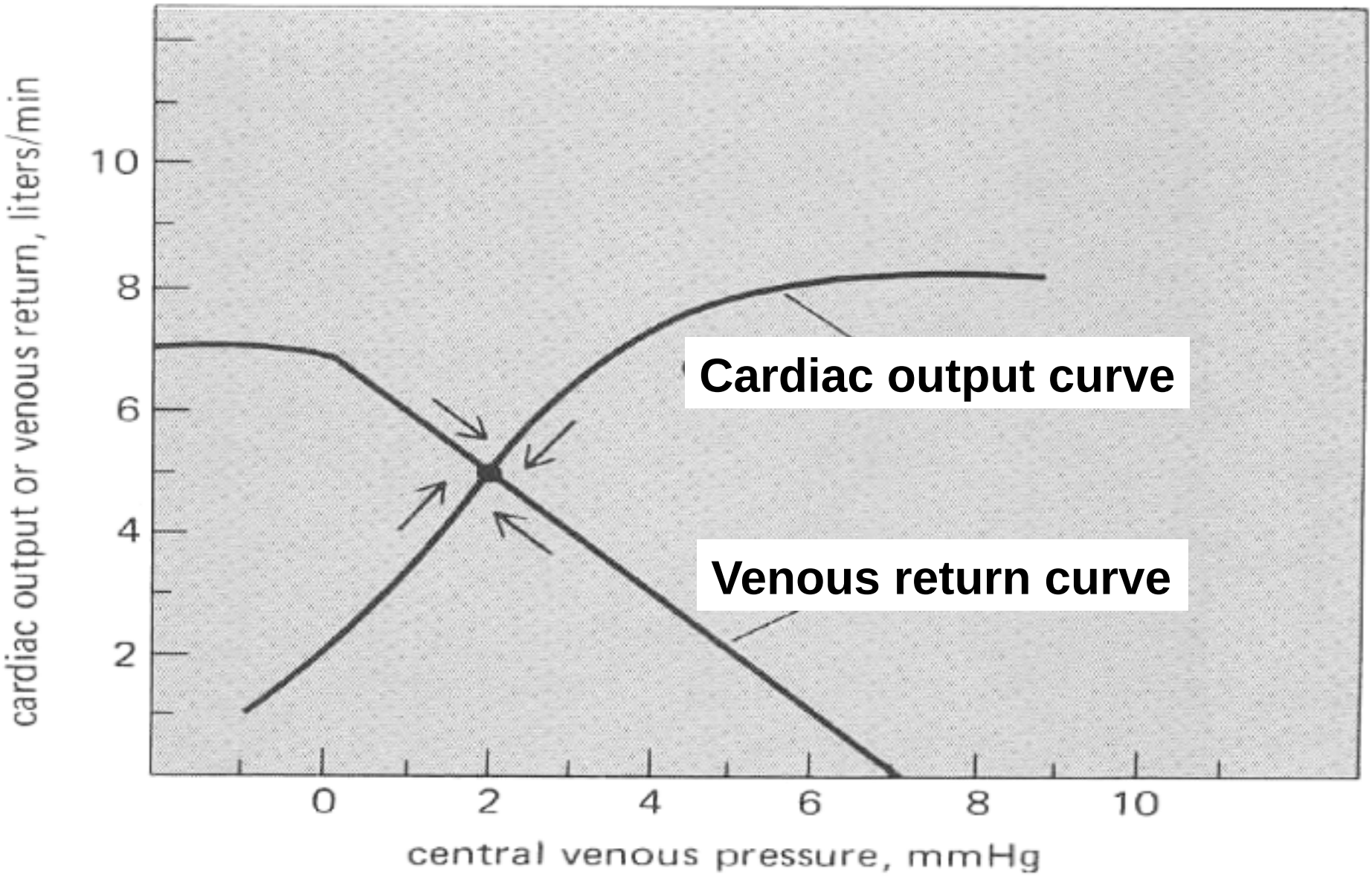


VR

CVP, mmHg

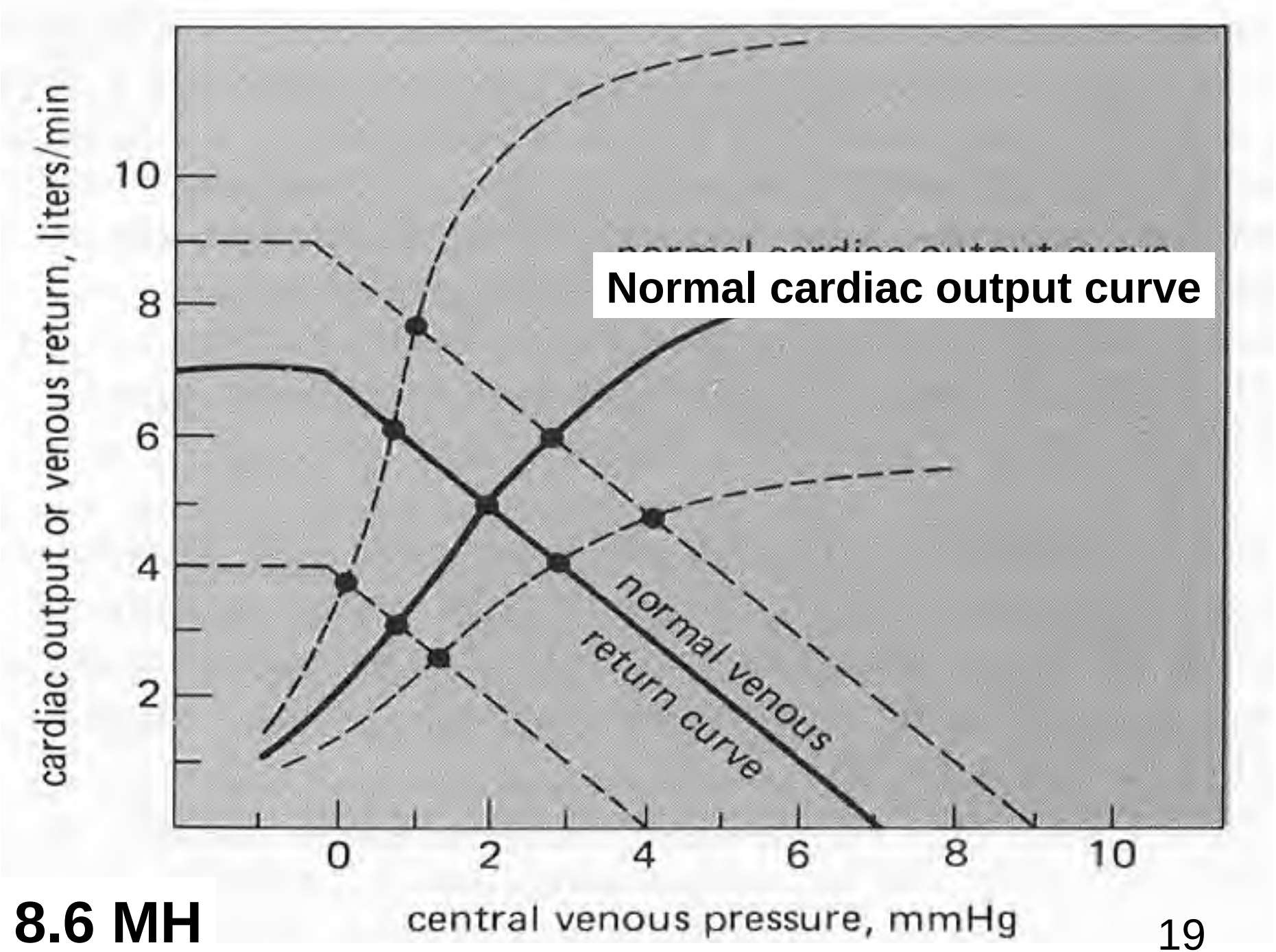
8.2B MH

 Mohrman and Heller. Cardiovascular Physiology. McGraw-Hill, 2006. 6th ed.



8.5 MH

 Mohrman and Heller. Cardiovascular Physiology. McGraw-Hill, 2006. 6th ed.

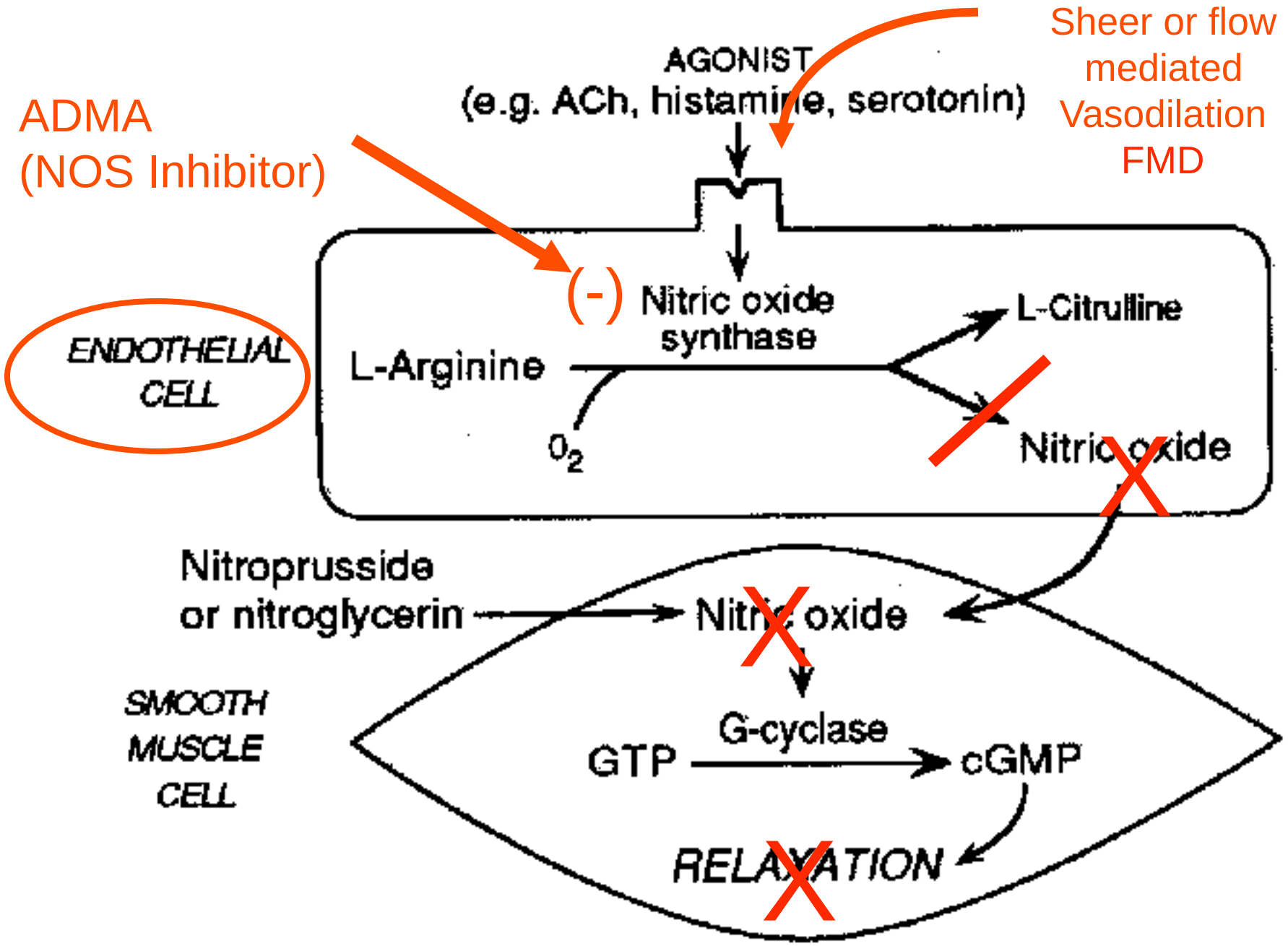


8.6 MH

 Mohrman and Heller. Cardiovascular Physiology. McGraw-Hill, 2006. 6th ed.

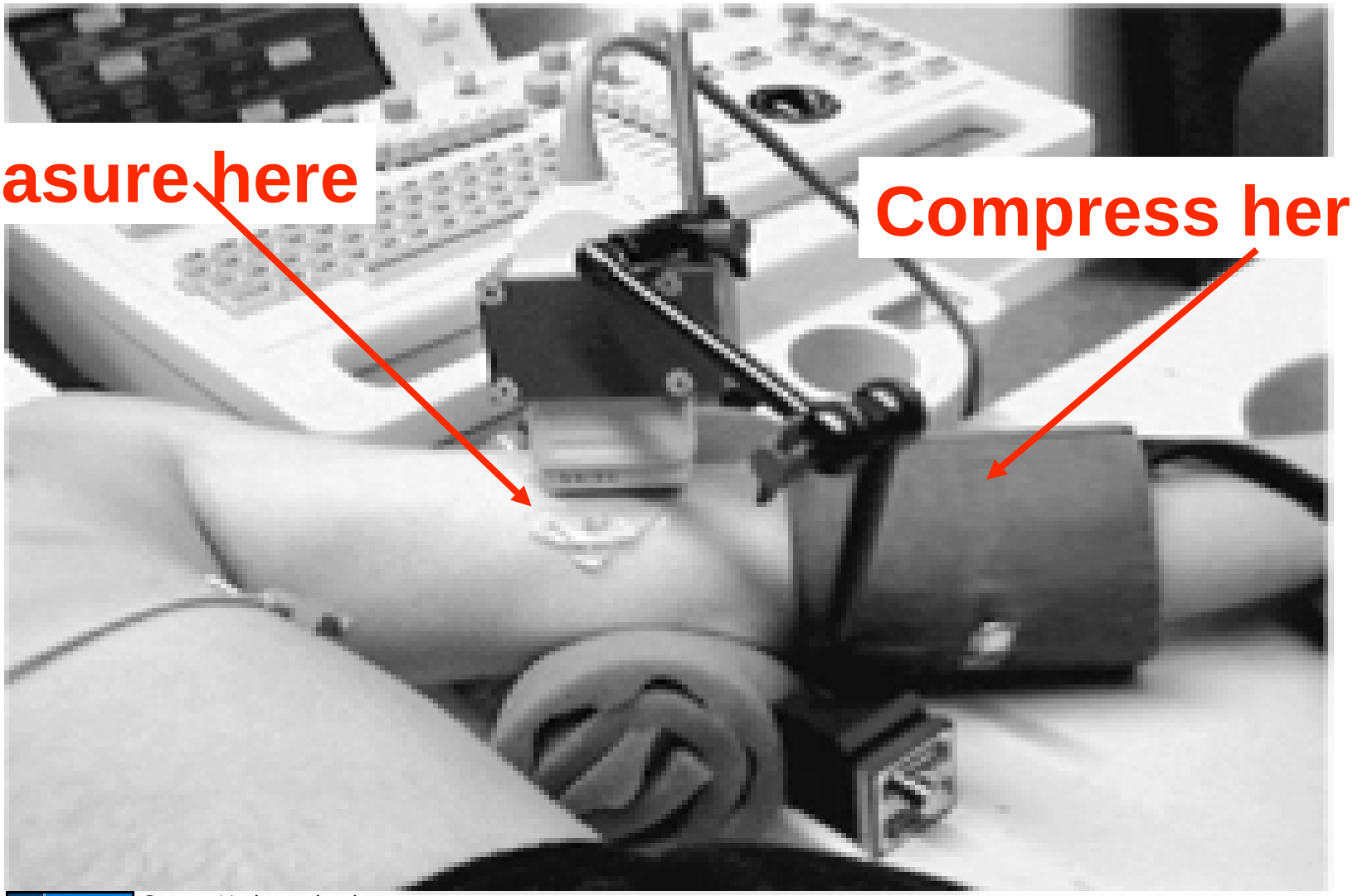
Review??

Flow Mediated Dilation (FMD) & Coronary Stenosis



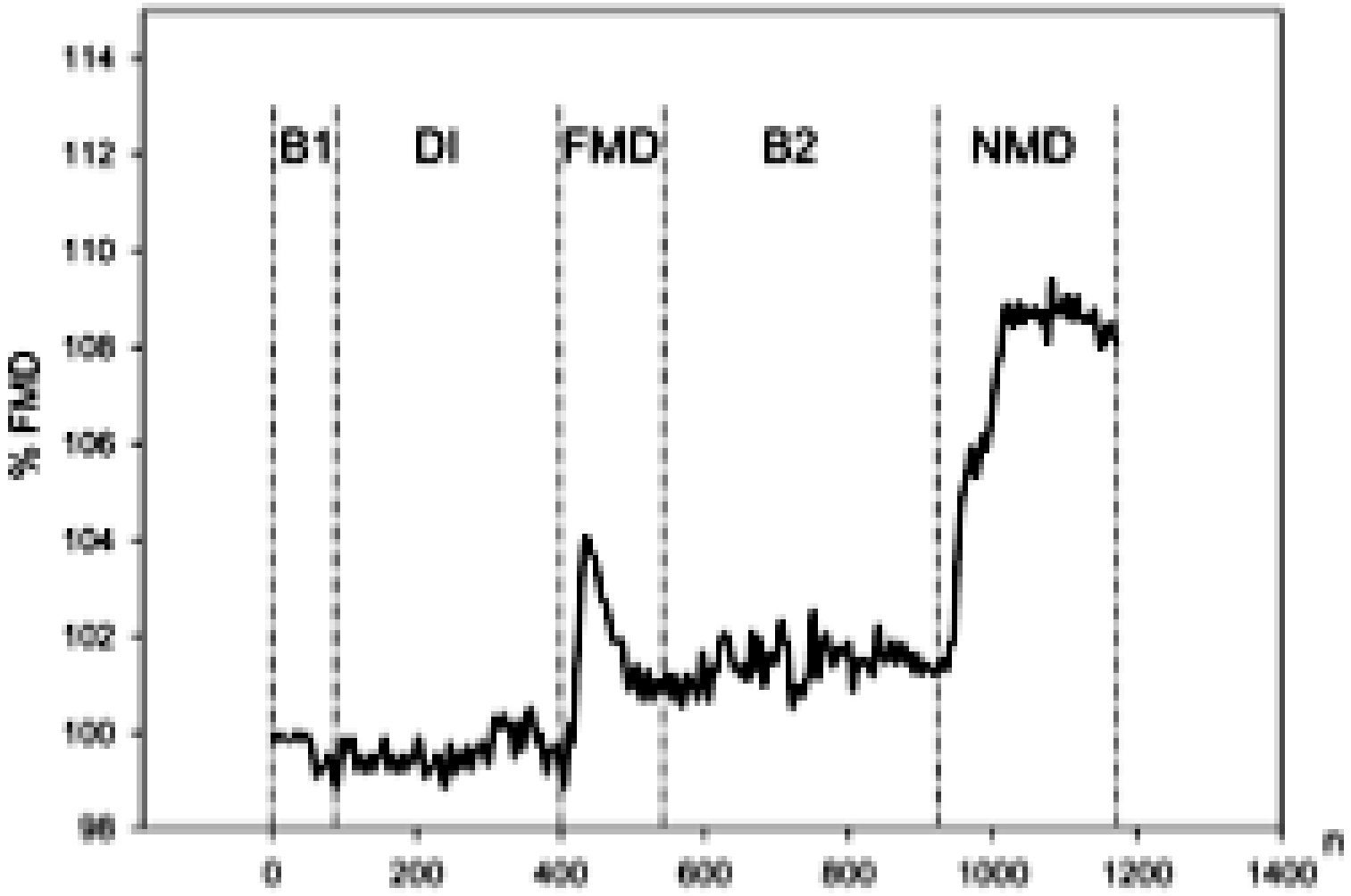
 Lilly, L. Pathophysiology of Heart Disease. Lippincott, 2007. 4th ed.

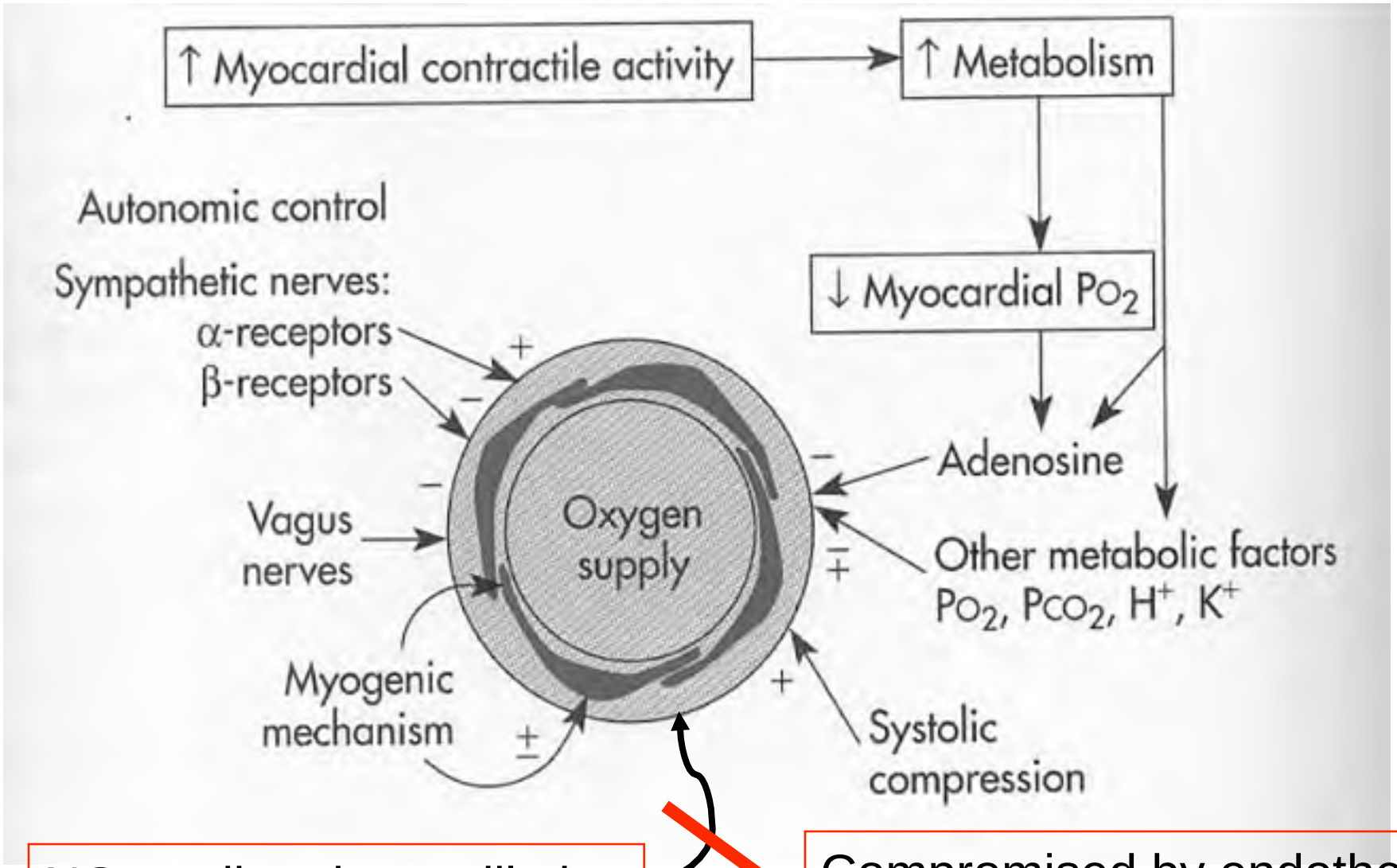
FMD Setup of Arm, ultrasound, & cuff on fore arm



PD-INEL Source Undetermined

FMD = flow mediated dilation
NMD = nitroglycerine (Max)mediated dilation





NO mediated vasodilation

Compromised by endothelial dysfunction (?ADMA?)

lated that in normal individuals, the relaxation effect of EDRF-NO outweighs the direct α-adrenergic constrictor effect of catecholamines on arterial smooth muscle, such that vasodilatation results. However, in patients with dysfunctional endothelium (e.g., atherosclerosis), an *impaired release of endothelial vasodilators* leaves the direct catecholamine effect unopposed, such that relative *vasoconstriction* occurs instead. The resultant decrease in coronary blood flow and myocardial oxygen supply contributes to ischemia. Of note, in patients with risk factors

Ischemia

-blood flow to a tissue or organ that is inadequate to maintain function.

- i.e. myocardial ischemia (MI)

With the same perfusion pressure, the same measured flow means the overall (series) resistance is the same regardless of a focal lesion!
BUT * You have used up vasodilator reserve !!!!!!**

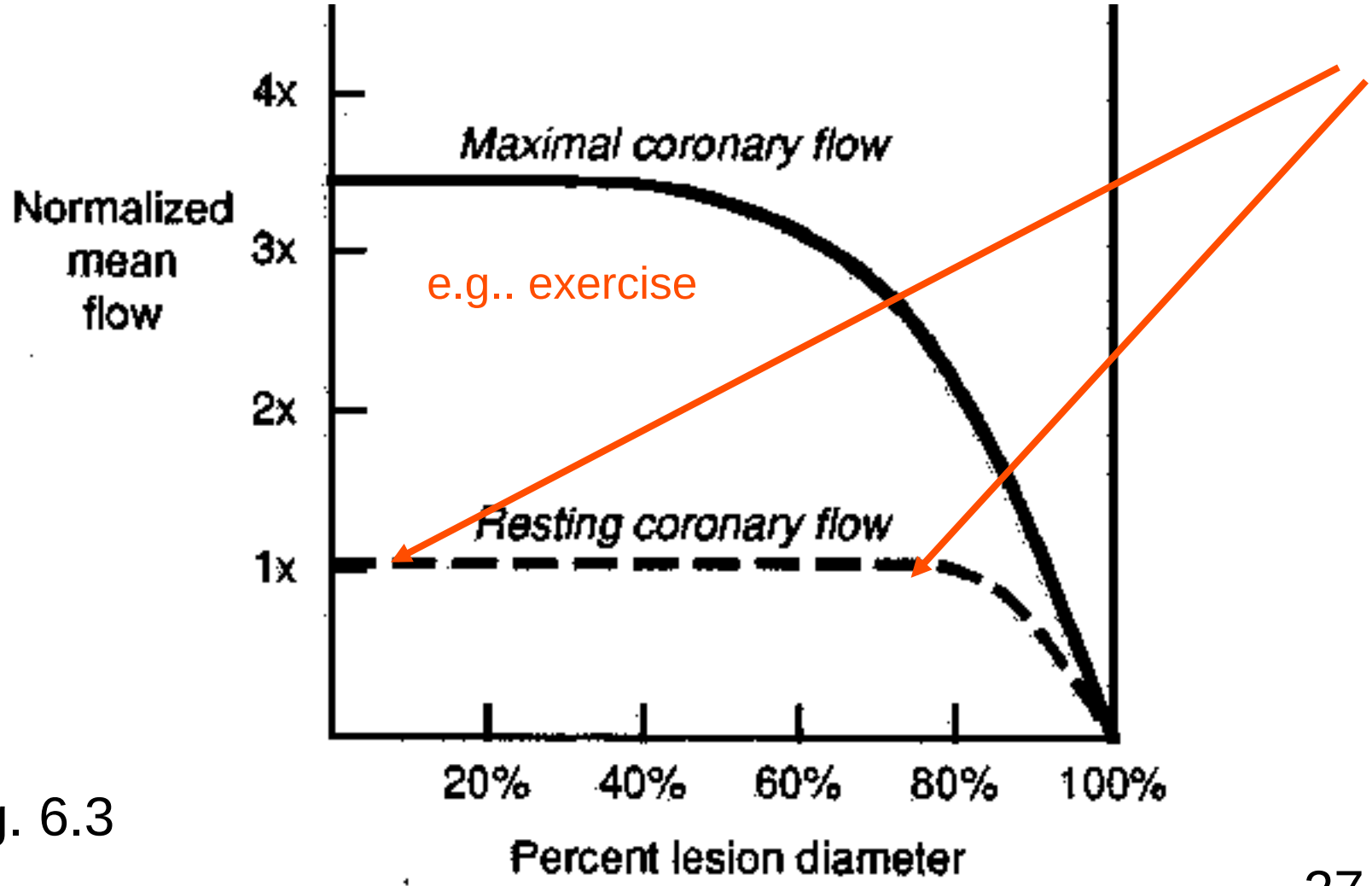
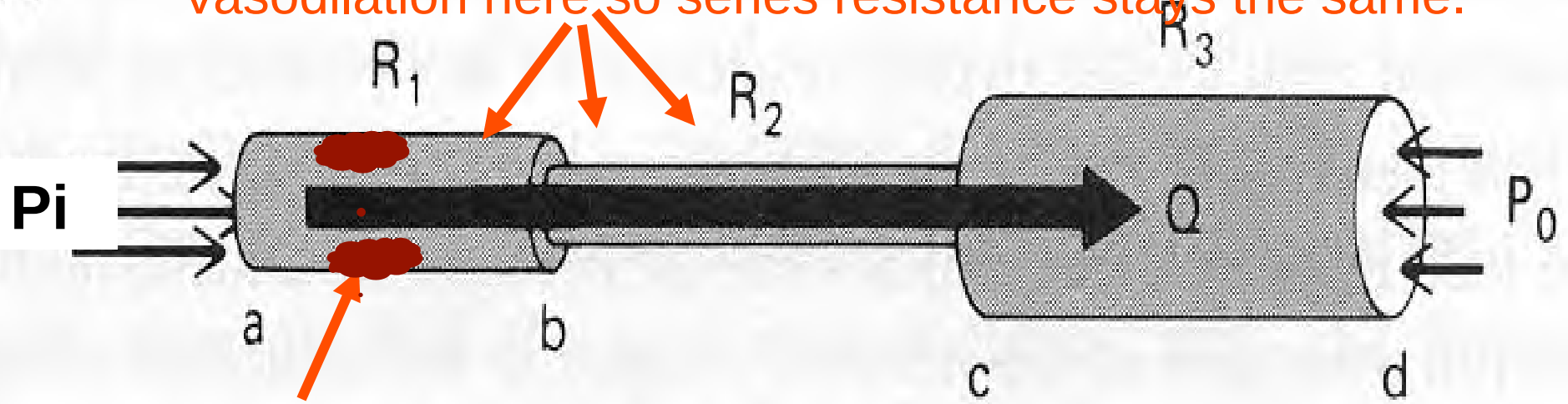


Fig. 6.3

Series Resistance Network

Compensatory
Vasodilation here so series resistance stays the same.



Lesion here

$$R_s = R_1 + R_2 + R_3$$

$$\Delta P = P_i - P_0$$

$$\dot{Q} = \Delta P / R_s$$

6.3 MH

Mohrman and Heller. Cardiovascular Physiology. McGraw-Hill, 2006. 6th ed.

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