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Coronary Blood Flow

M1 – Cardiovascular/Respiratory Sequence

Louis D'Alecy, Ph.D.

Fall 2008

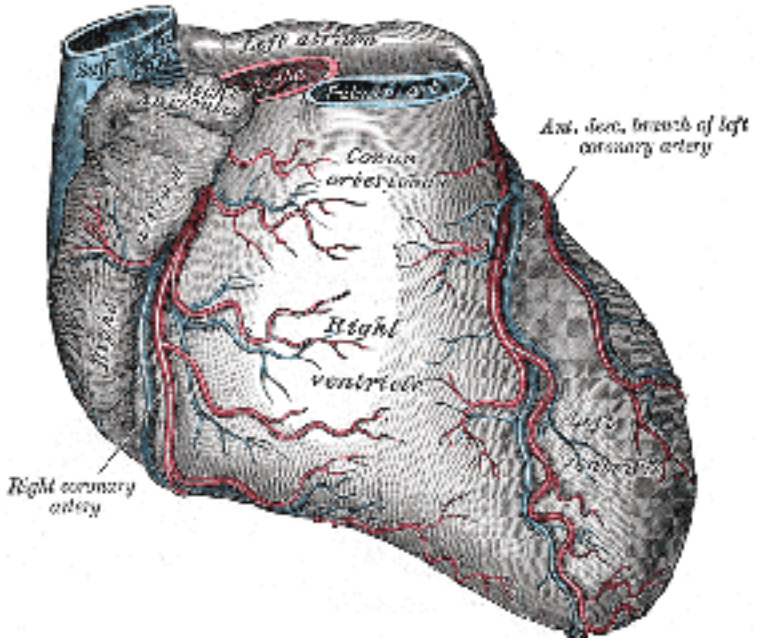
Wednesday 11/05/08, 11:00

Coronary Blood Flow

26 slides, 50 minutes

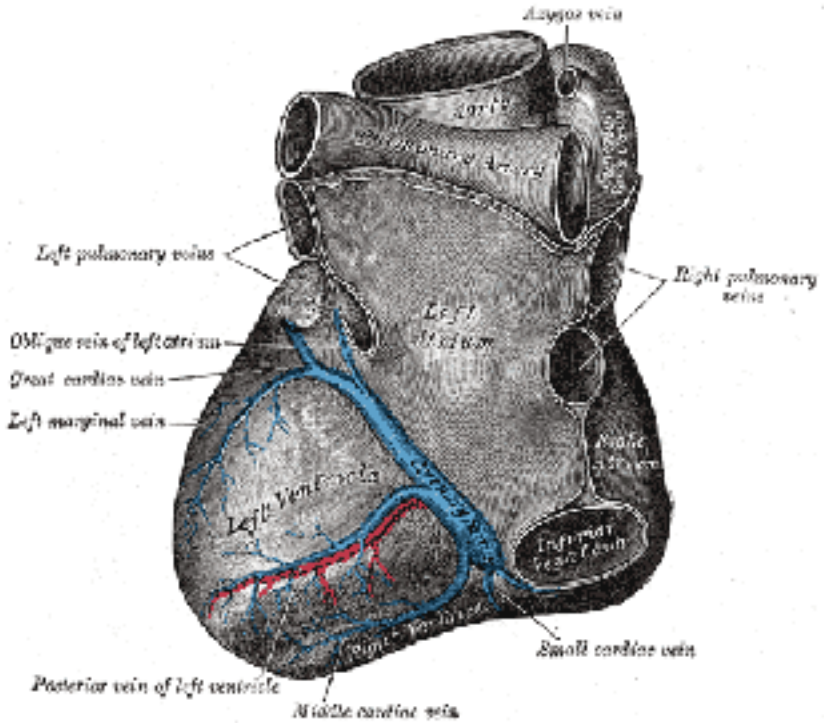
1. Vascular anatomy
2. Determinants of coronary flow
3. Flow mediated dilation
4. Ischemia
5. Coronary Flow Reserve

Surface Anatomy



PD-INCL Gray's Anatomy

Anterior view



PD-INCL Gray's Anatomy

Posterior view

Heart statistics

300g/70,000g = 0.0043 or < 0.5% Body Weight

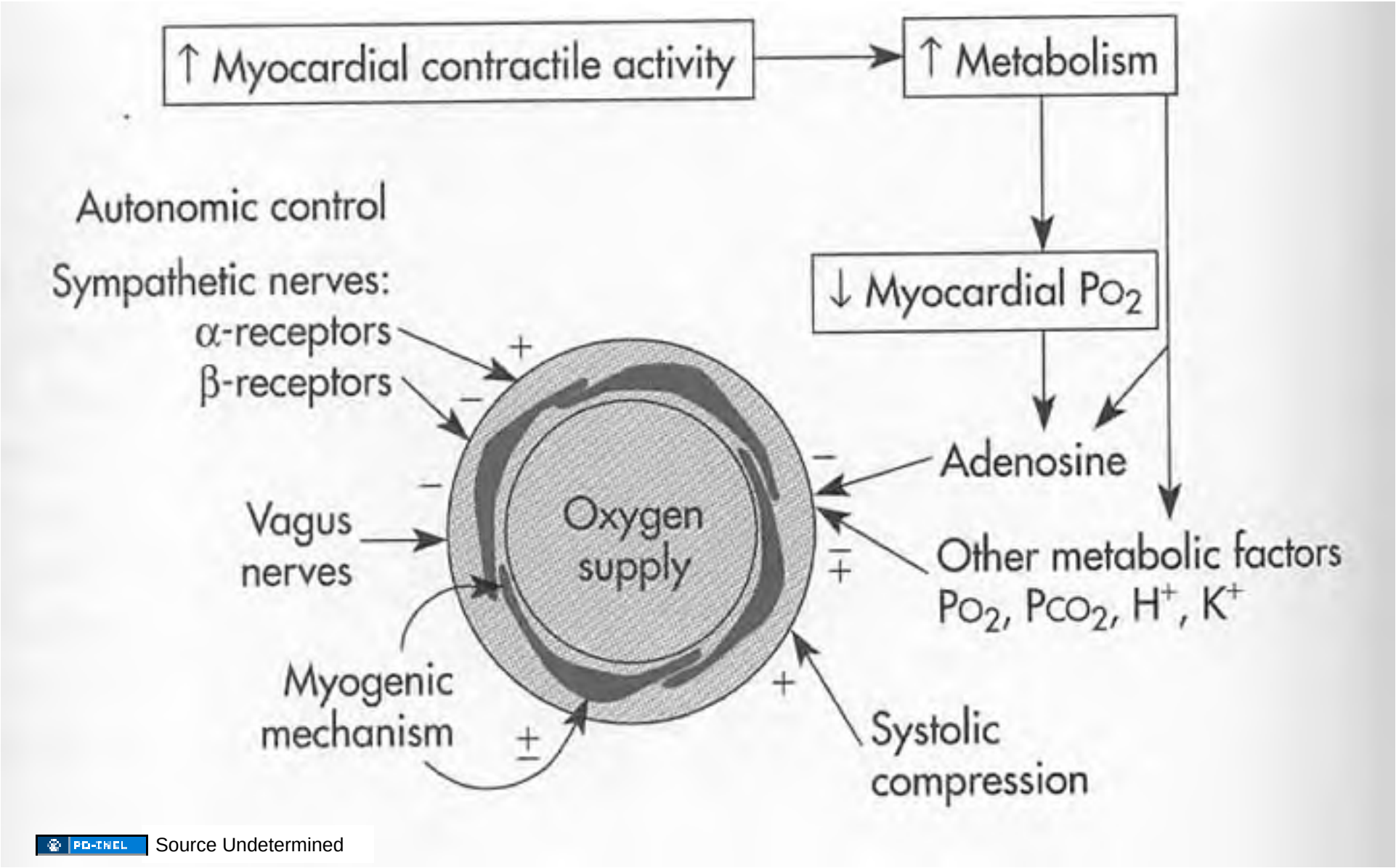
Coronary flow = 4% of cardiac output = 80 mL/mim/100g

“Resting “ flow ~ 30X flow/100g tissue of skeletal muscle

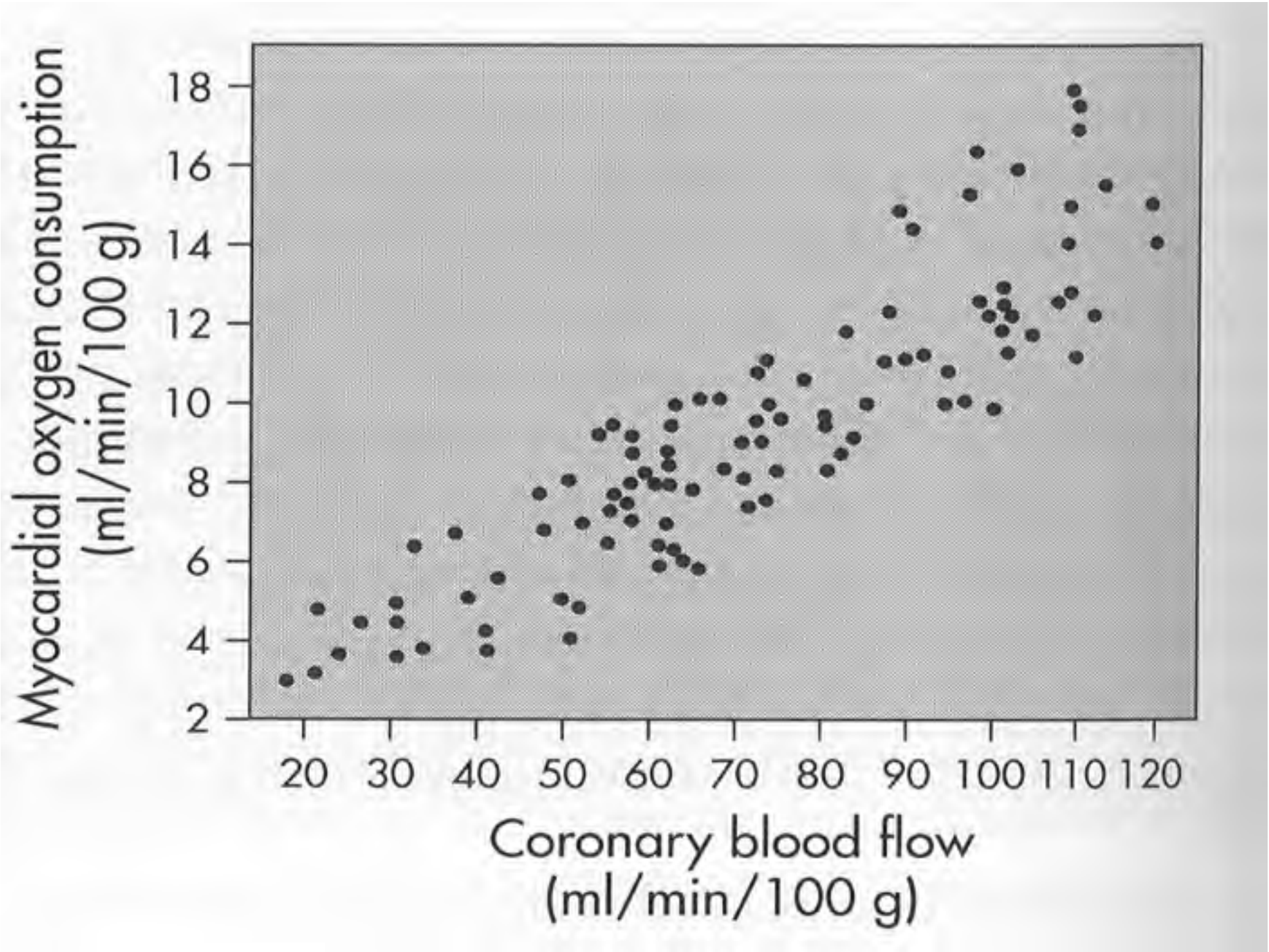
Highest oxygen extraction in body
(arterial oxygen 20 vol % to coronary sinus 8 vol %)
(typical mixed venous oxygen higher at 17 vol %)

Must increase flow to increase oxygen delivery

Vol % = mLO_2 / 100 mL blood



Cross section of coronary artery.



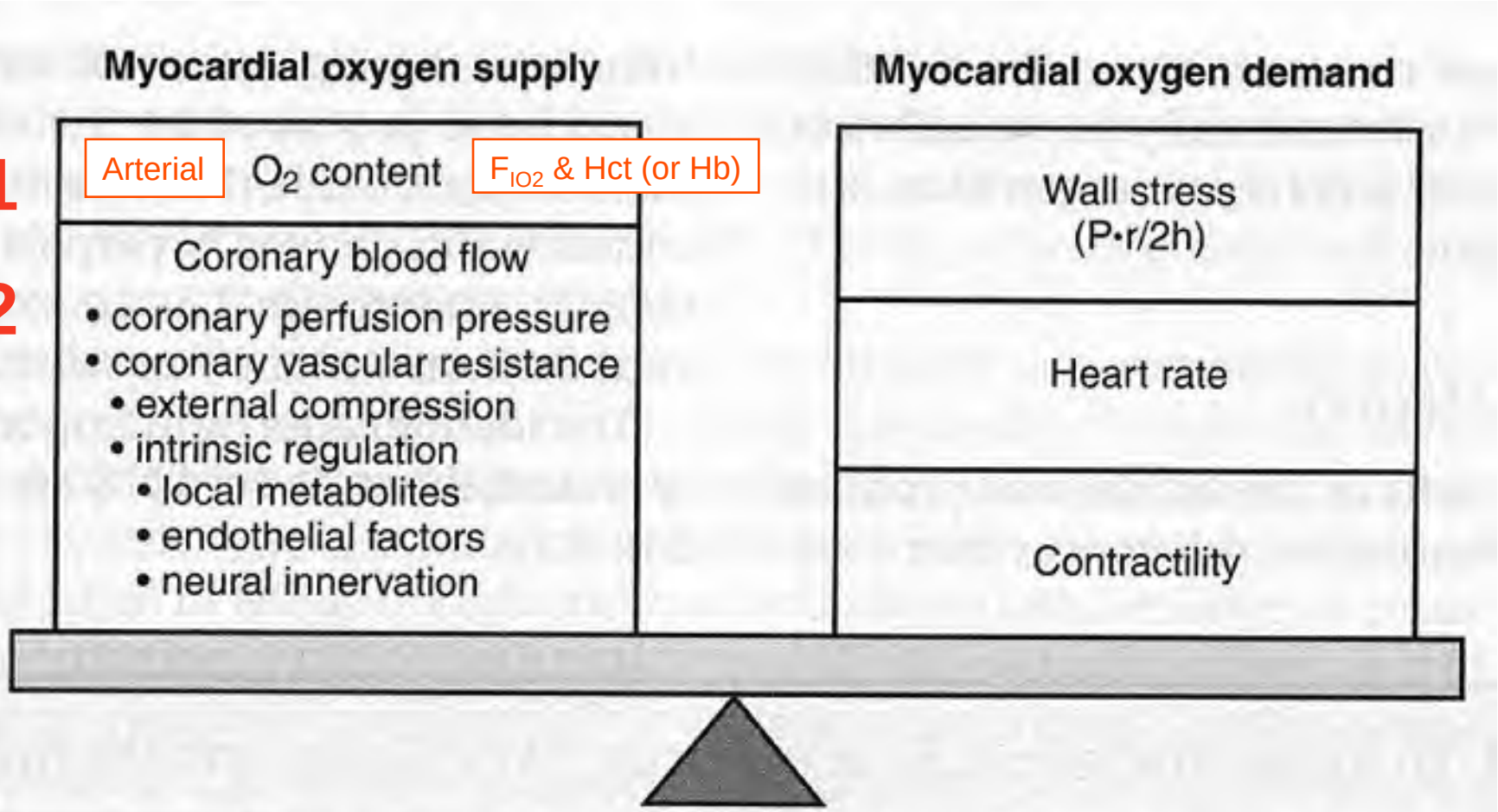


Figure 6.1. Major determinants of myocardial oxygen supply and demand.
h, ventricular wall thickness; P, ventricular pressure; r, ventricular radius.

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

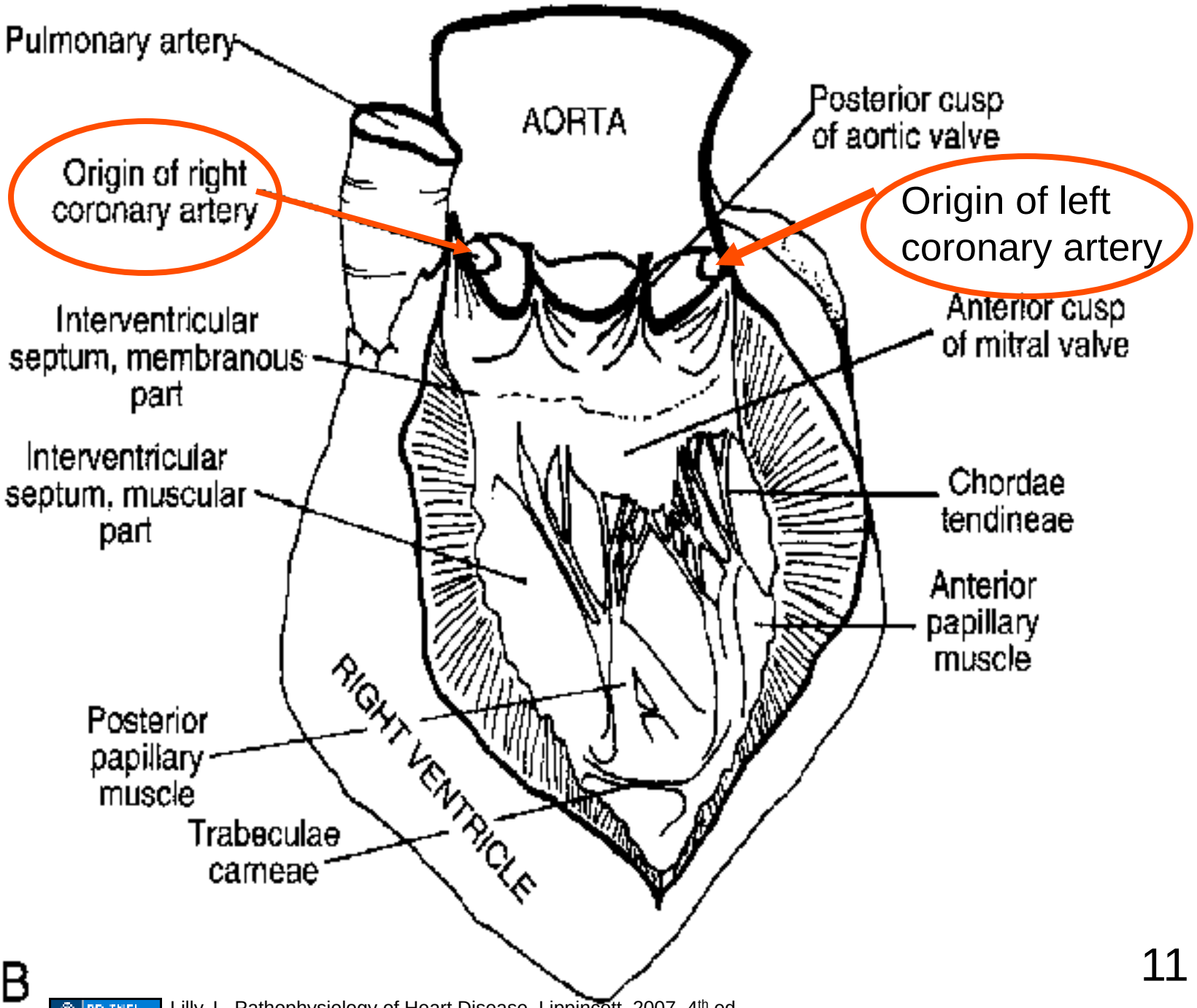
DETERMINANTS OF CORONARY BLOOD FLOW

1 DIASTOLIC PERFUSION PRESSURE ΔP

2 SYSTOLIC COMPRESSION ("Resistance")

3 METABOLIC CONTROL (Resistance)

4 NEURAL CONTROL (Resistance)



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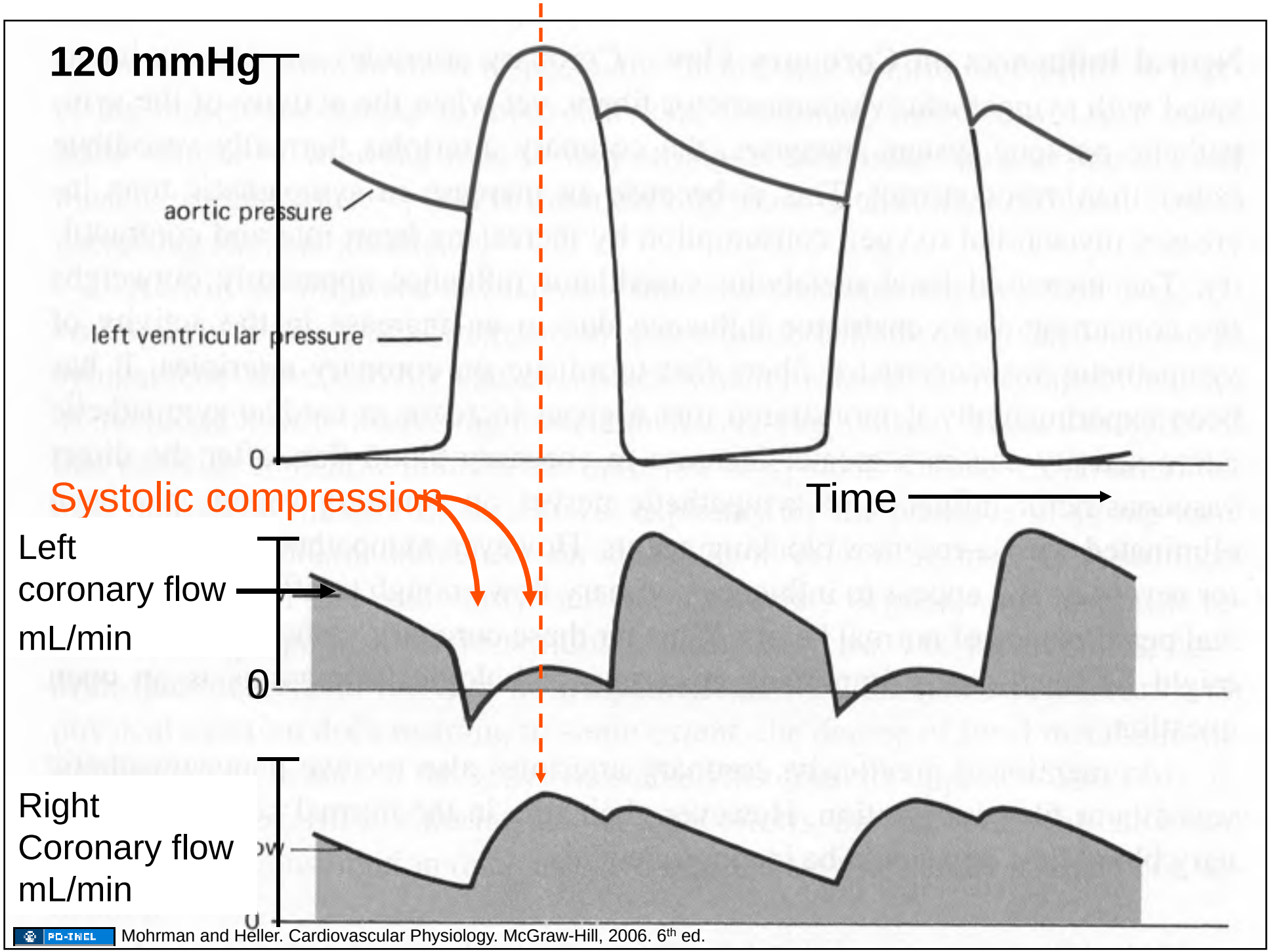

PD-ThCL Mohrman and Heller. Cardiovascular Physiology. McGraw-Hill, 2006. 6th ed.

r = radius

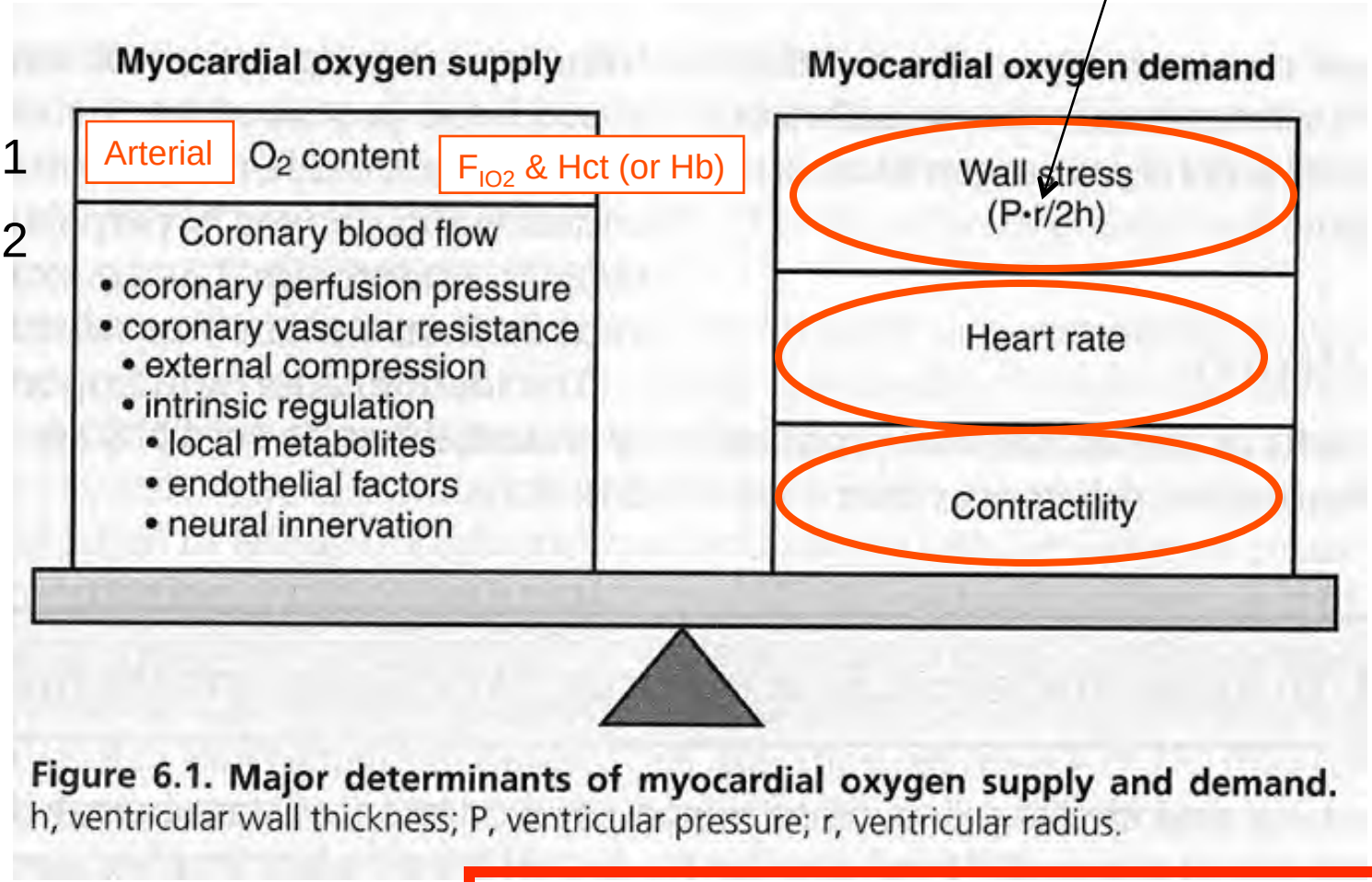

Figure 6.1. Major determinants of myocardial oxygen supply and demand. h, ventricular wall thickness; P, ventricular pressure; r, ventricular radius.

If r ↑, more tension is need for the same pressure.

Thus an enlarged heart has greater demand.

******Pressure X Rate Product**

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Law of LaPlace

$$T = P \times r$$

Tension = Press X radius

"Cost" .

$$2T = P \times 2r$$

If radius (r) ↑, more tension is need for the same pressure.

Thus an enlarged heart has greater demand (cost).

Tissue Blood Flow and Tissue Vascular Resistance

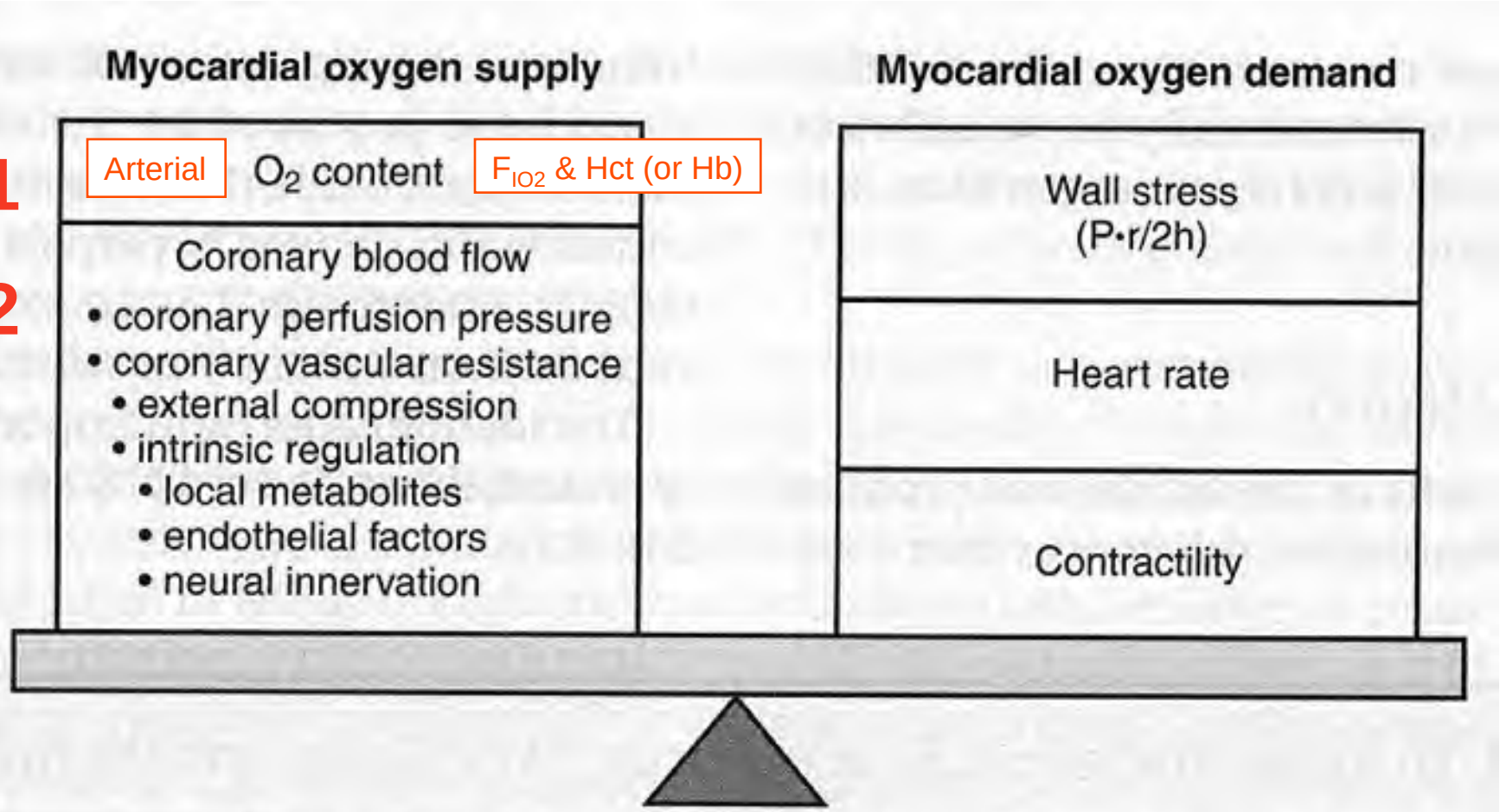
(*Assume Perfusion Pressure is Constant ***)**

- **Vasoconstriction**
- **$\Rightarrow \Downarrow r \Rightarrow \Uparrow R_{tissue} \Rightarrow \Downarrow F_{tissue}$**

- **Vasodilation**

$\Rightarrow \Uparrow r \Rightarrow \Downarrow R_{tissue} \Rightarrow \Uparrow F_{tissue}$

$$F_{tissue} = \frac{\text{Perfusion Pressure}}{R_{tissue}}$$



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Isolated Vascular Effects

1. **Sympathetic alpha adrenergic vasoconstriction α_1**

2. **Sympathetic beta adrenergic vasodilation**

   β_1 (evidence for innervated VSM)

   β_2 non-innervated VSM

3. **Parasympathetic cholinergic vasodilation**

BUT HOW DOES IT WORK IN VIVO ????

Parasympathetic Activation

Stimulate parasympathetic to heart >> Ach >>SA node >> ↓↓ HR >>↓↓ metabolism >> ↓↓ Coronary Blood flow

BUT

PACE heart (i.e. fixed heart rate) >> no change in HR >> no change metabolism ---------- Therefore Stimulate parasympathetic to paced heart >> >> Ach vasodilation >> ↑↑ coronary blood flow !!

BUT HOW DOES IT WORK IN VIVO ????

Sympathetic Activation

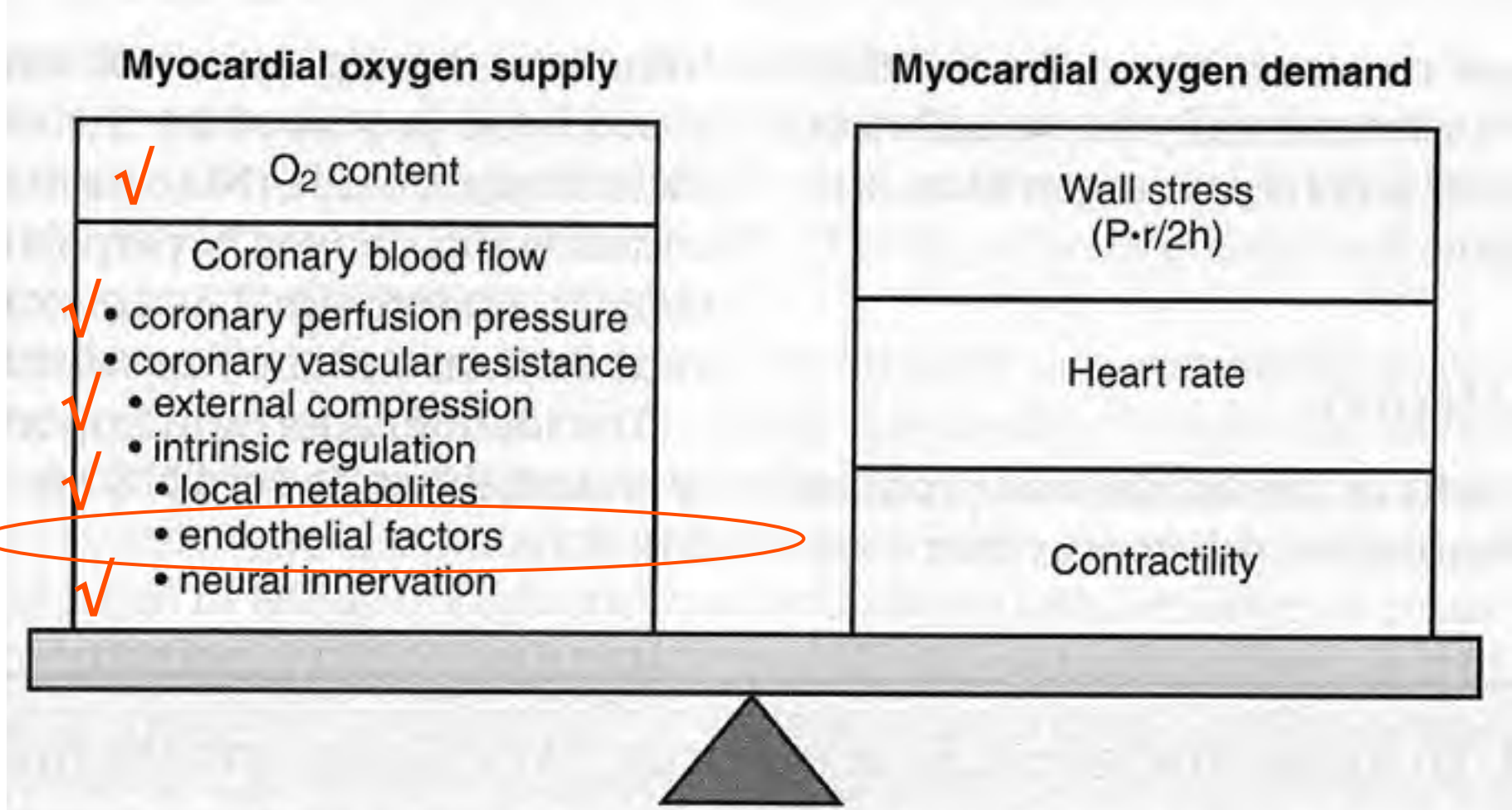
Stimulate sympathetic nerves to heart >> ↑↑ Norepi >> >> ↑↑ inotropism >> ↑↑ metabolism ++ >> ↑↑ HR >> ↑↑ Coronary Blood flow

BUT

Block $\beta_{1\&2}$ receptors and Stimulate sympathetics to heart >> ↑↑ Norepi (stress) >> no change in HR >> >> no change metabolism >> >> no change in inotropism >> potential for >> ↓↓ Coronary Blood flow by "unmasked" α_1 adrenergic vasoconstriction

Can Metabolic control still dominate??

Flow Mediated Dilation (FMD) & Coronary Stenosis



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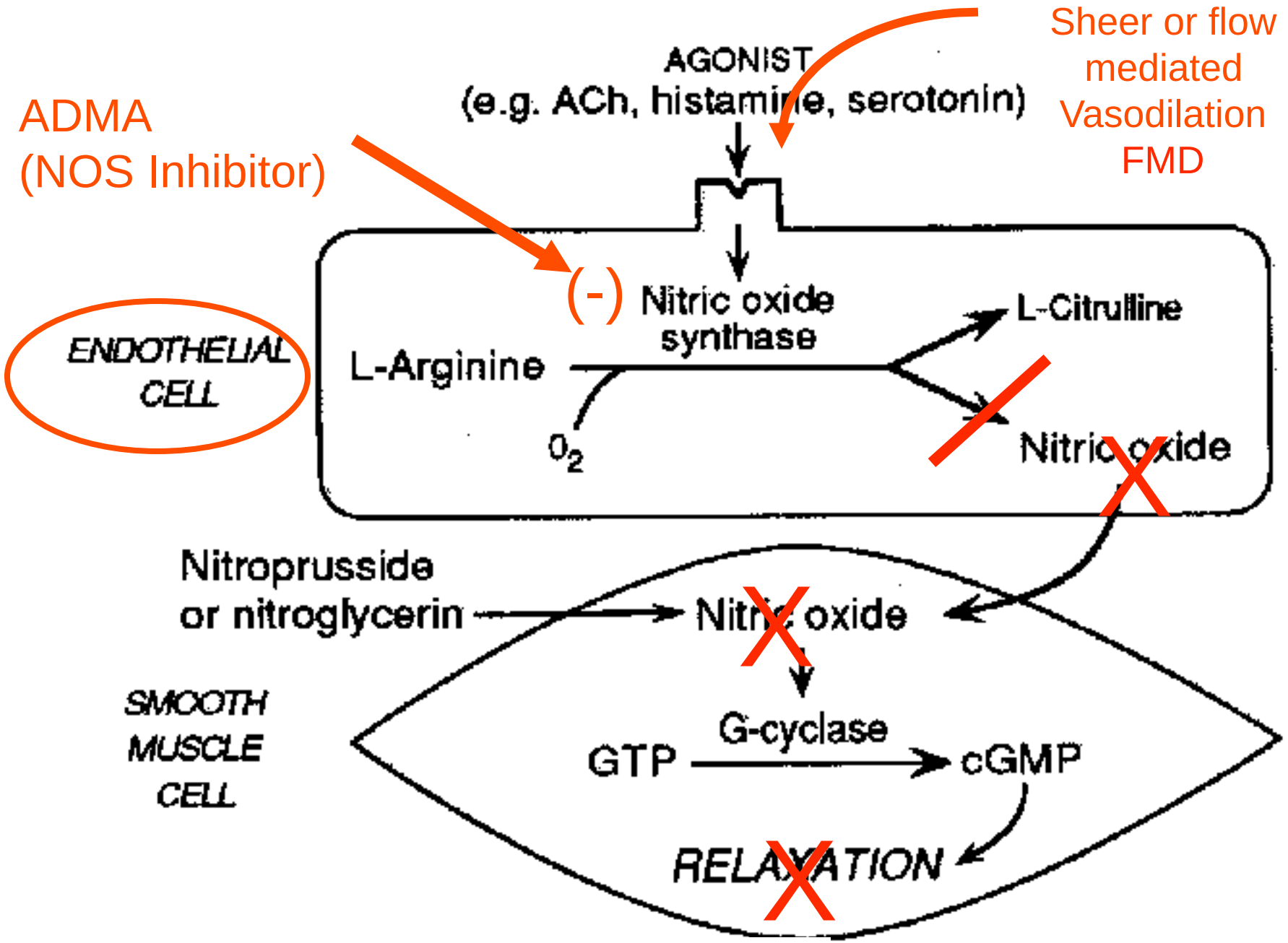
FMD = Flow Mediated Dilation



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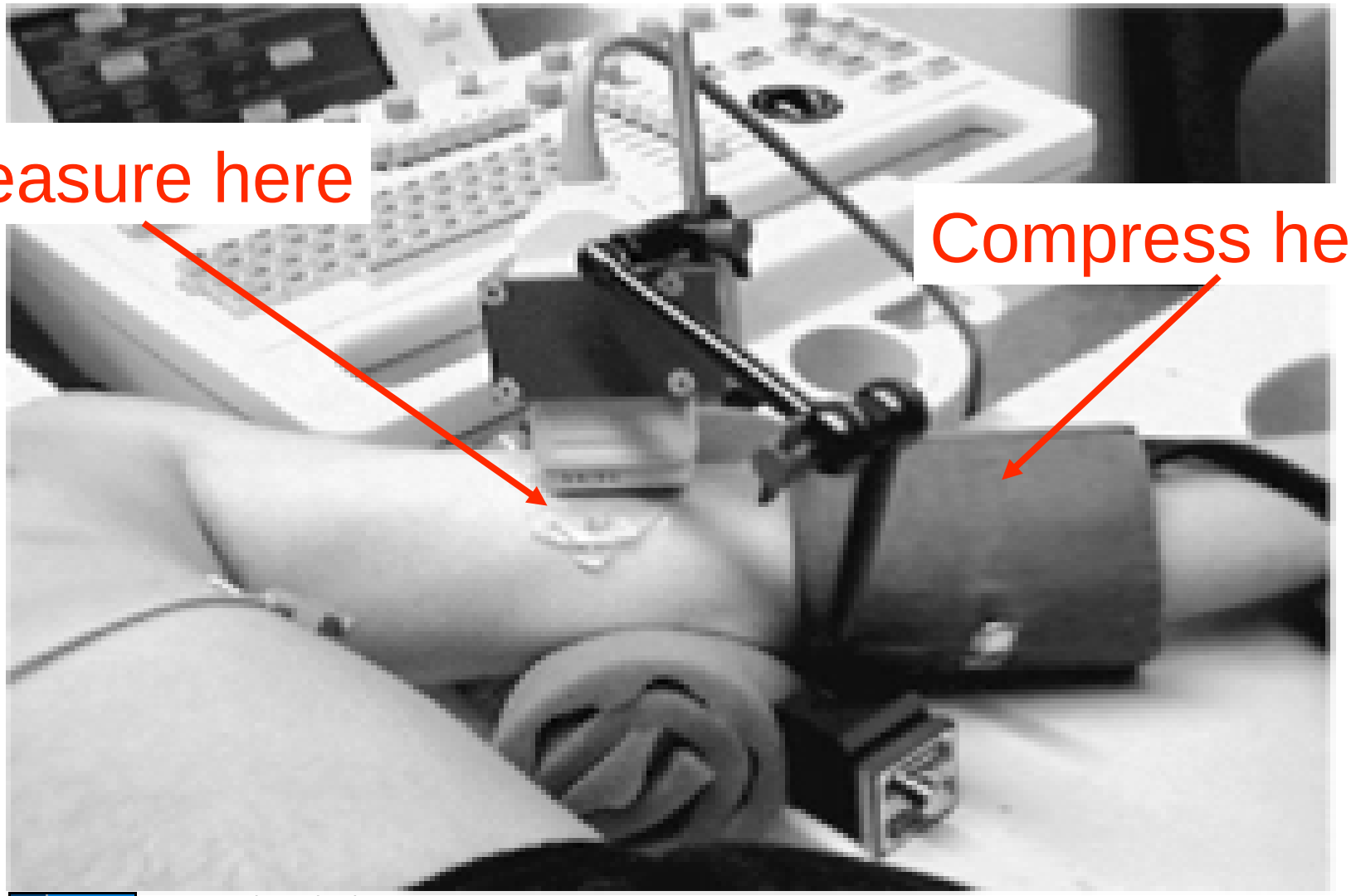
FMD Setup of Arm, ultrasound, & cuff on fore arm



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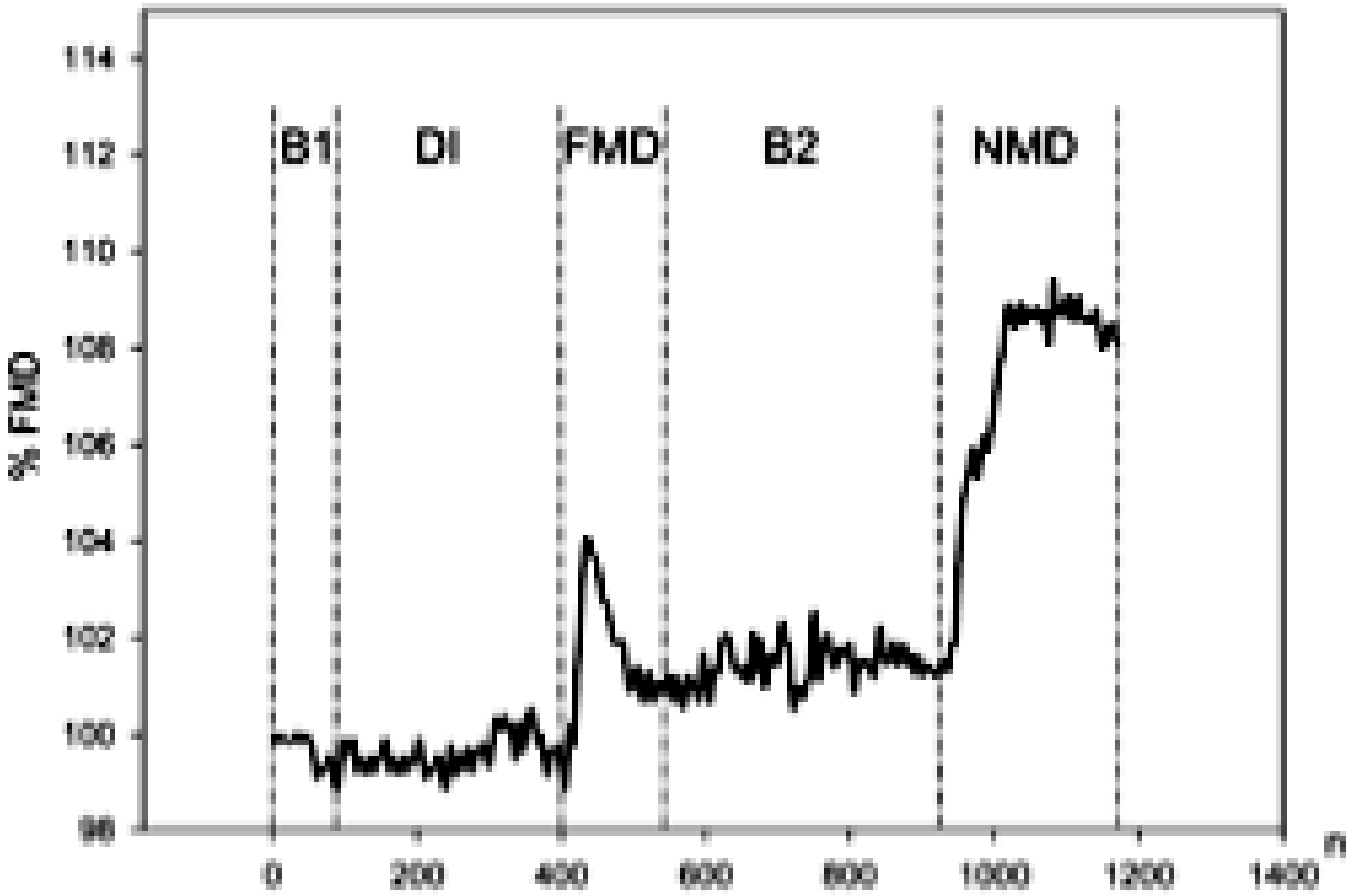
FMD = flow mediated dilation

NMD = nitroglycerine (Max)mediated dilation



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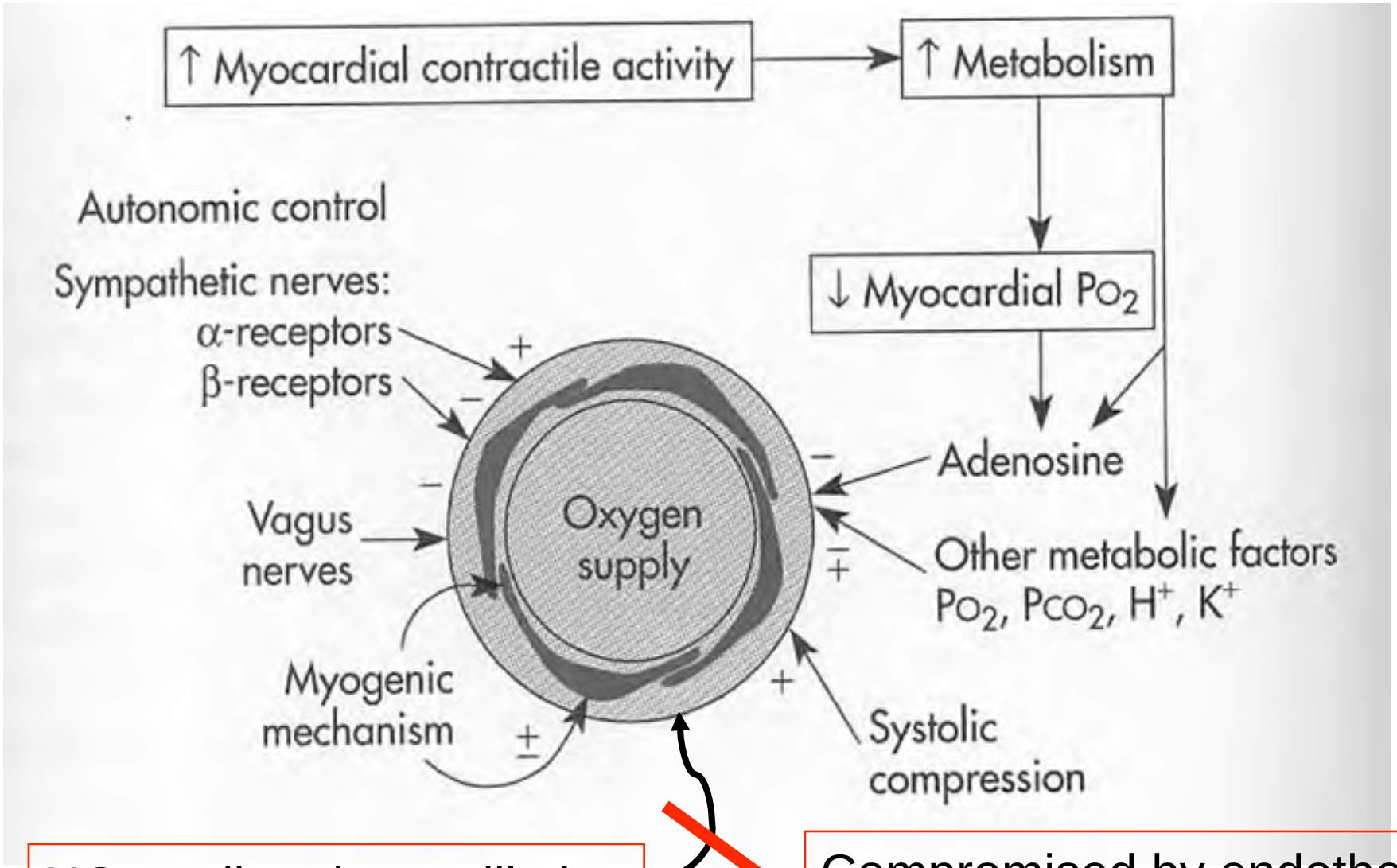


NO mediated vasodilation

Compromised by endothelial dysfunction (?ADMA?)

Source Undetermined

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 !!!!!!**

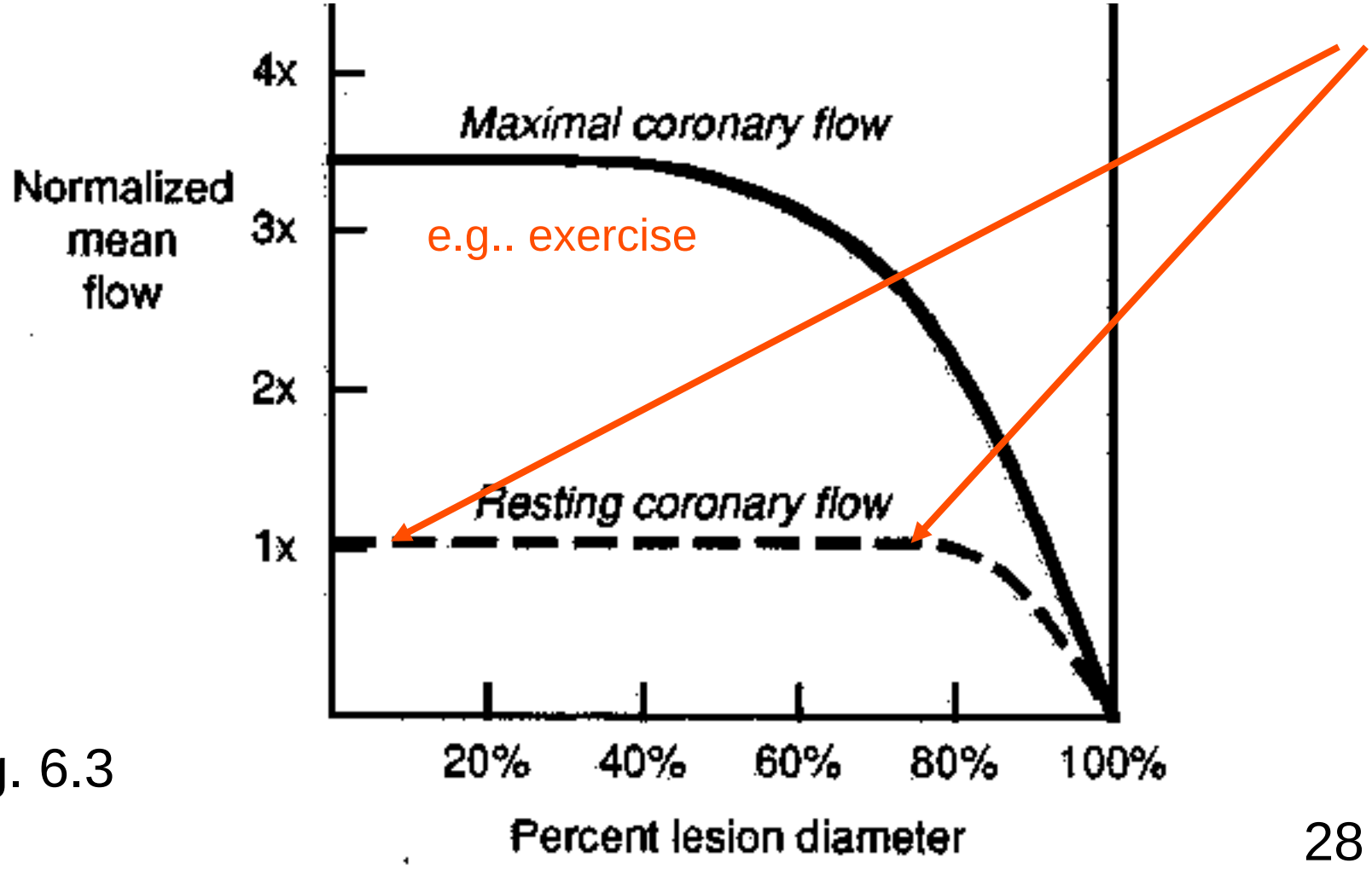
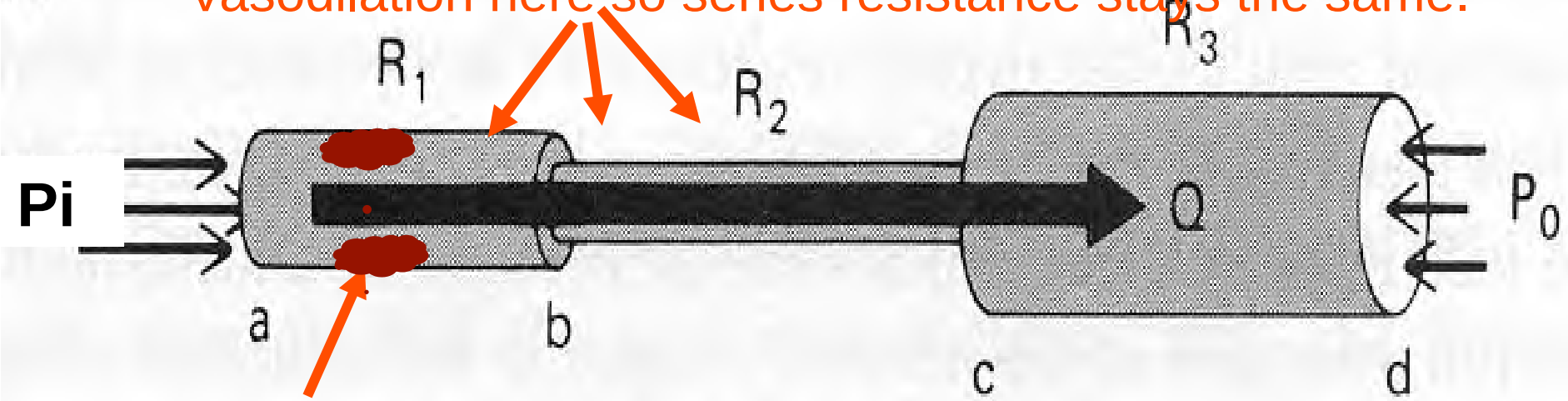


Fig. 6.3

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

Series Resistance Network

Compensatory
Vasodilation here so series resistance stays the same.



Lesion here

$$R_s = R_1 + R_2 + R_3$$

$$\Delta P = P_i - P_0$$

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

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

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