Author(s): Louis D'Alecy, 2009

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Cardiac Muscle I

M1- Cardiovascular/Respiratory Sequence

Louis D’Alecy, Ph.D.

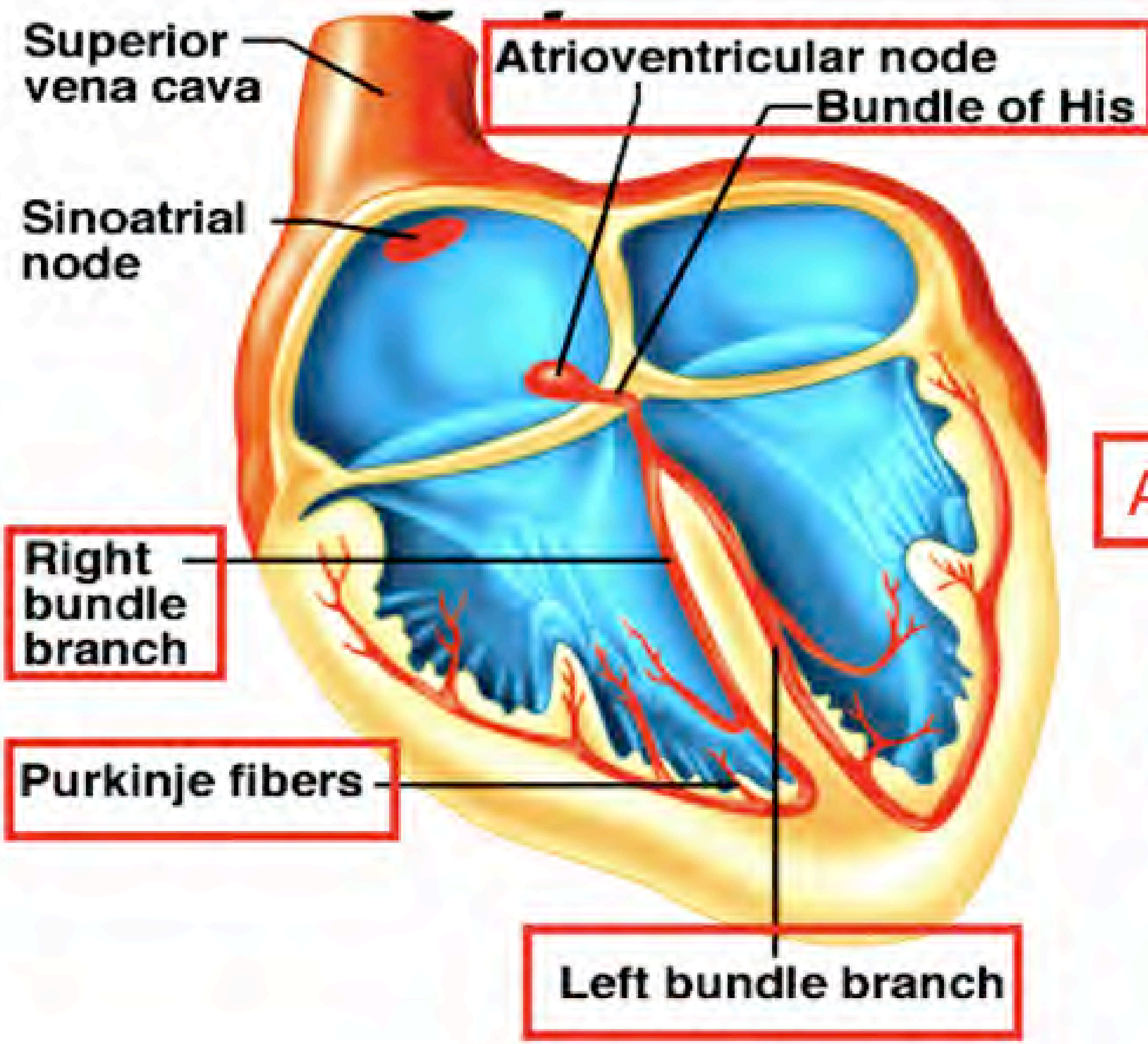
Fall 2008

Tuesday 10/28/08, 10:00
Cardiac Muscle I

19 Slides, 50 min

1. CM structure
2. CM contractile function
3. Ca^{++} induced Ca^{++} release
4. Isometric contraction
5. Isotonic contraction
6. Afterloaded contraction

General Structure = muscle !



4 Chambers
4 Valves
Syncytium
Conducting system

ALL MUSCLE

Different Conduction velocities

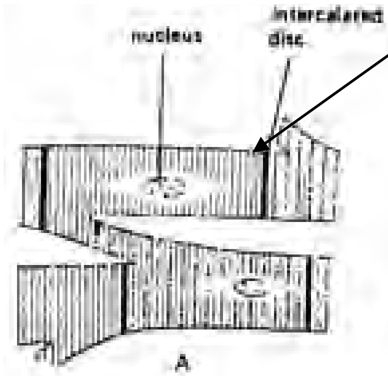
Requirements for Effective Cardiac Pumping

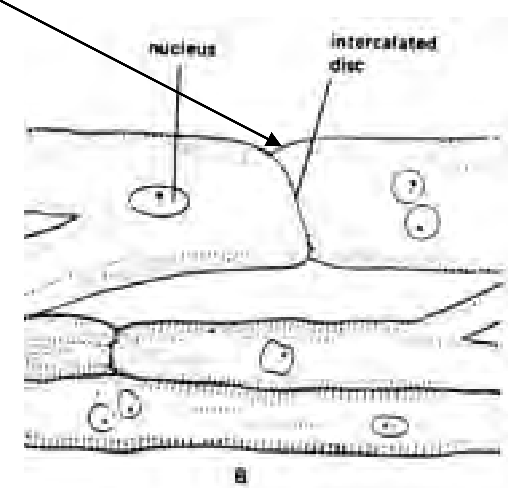
1) Synchronized	not arrhythmic
2) Valves open fully	not stenotic
3) Valves don't leak	not insufficient or regurgitant
4) Forceful	not failing
5) Must fill	Not "dry"

Intercalated Disc =

Firm mechanical connection
Low resistance electrical connection

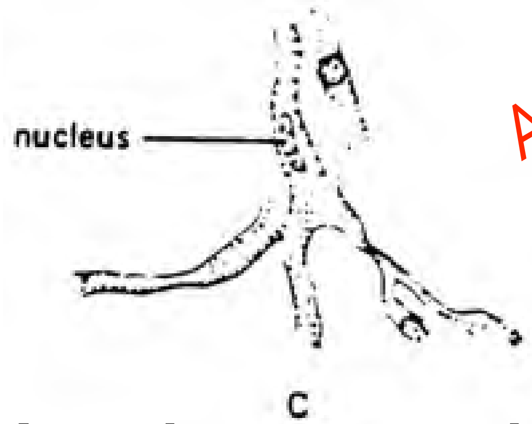
Cardiac myocyte



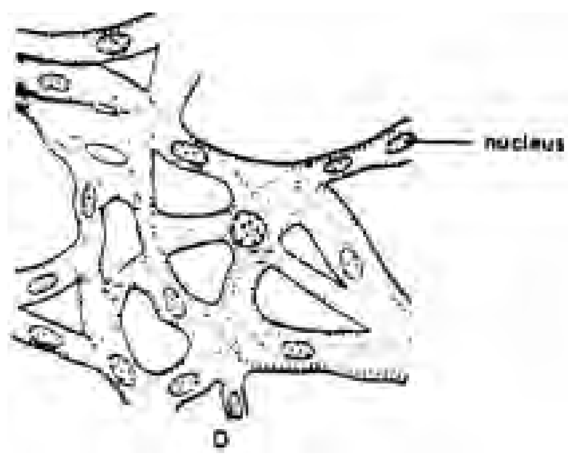


Purkinje fibers

All these are muscle cells



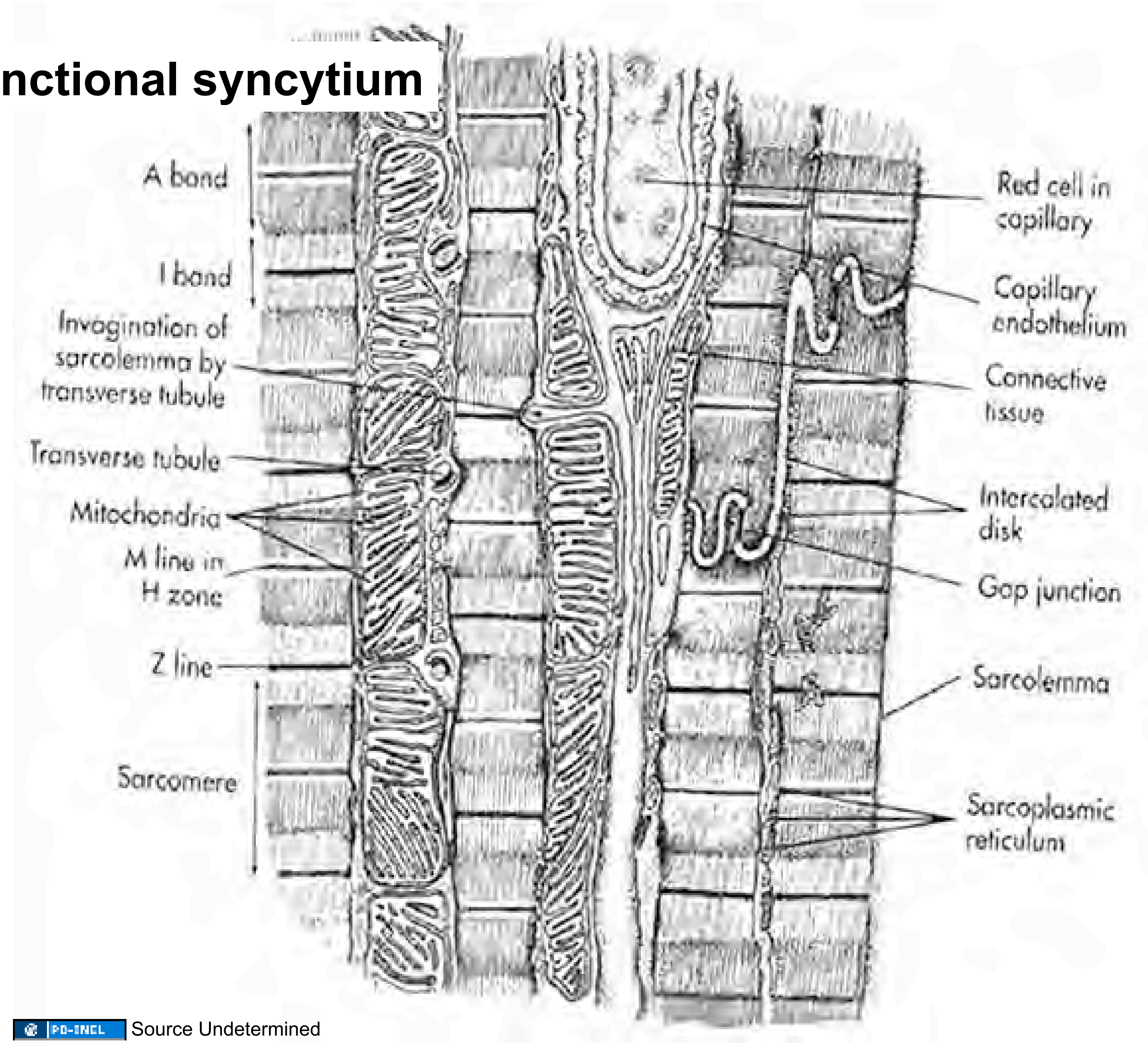
SA node - pacemaker



AV node

One heart - ~ 1 billion cells

Functional syncytium



SUMMARY OF COMPARISONS BETWEEN MUSCLE TYPES

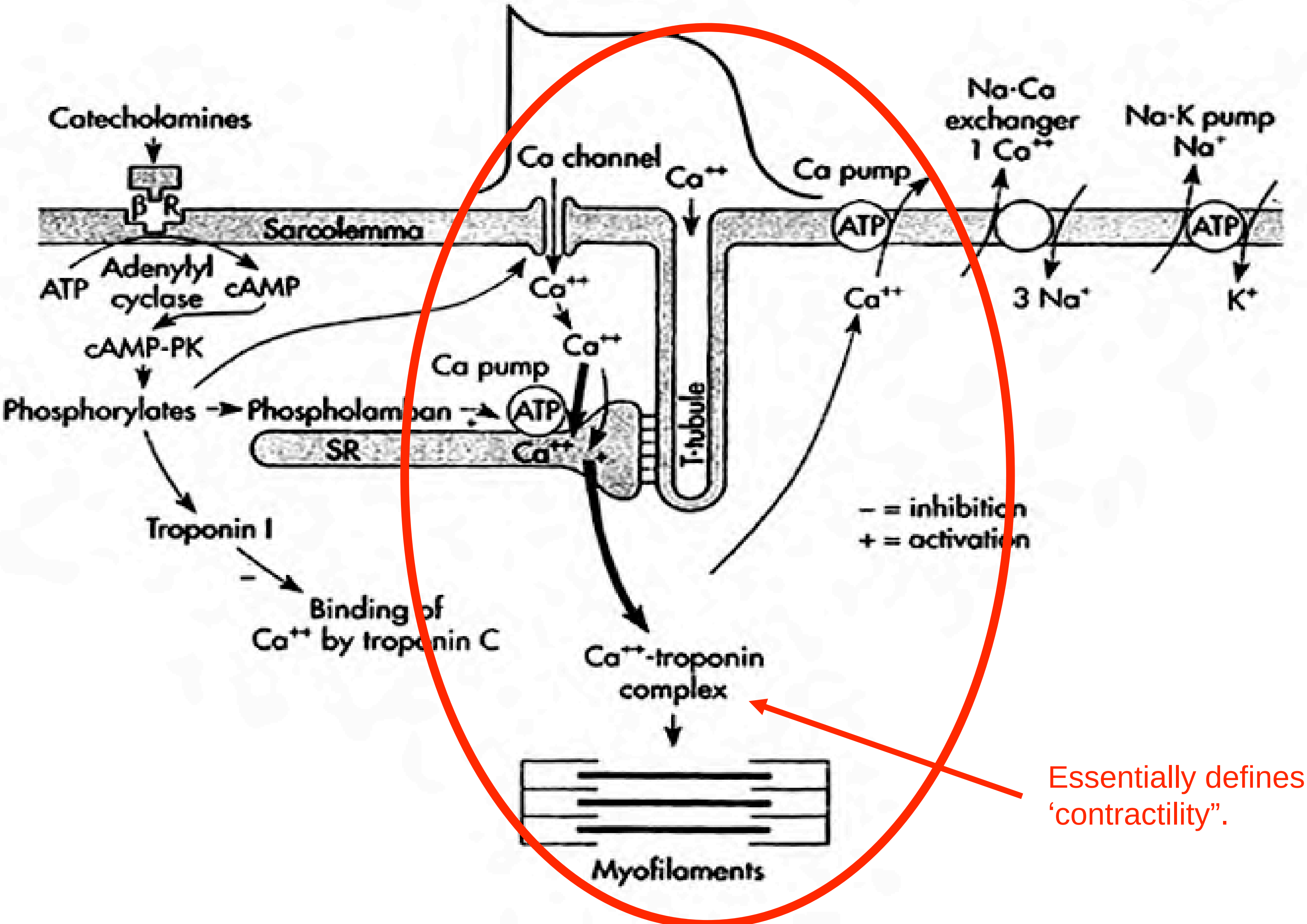
	SKELETAL	CARDIAC
Mechanism of excitation	Neuromuscular transmission	Pacemaker potentials Electrotonic depolarization via gap junctions
Electrical activity of muscle cell	Action potential spikes	Action potential plateaus
Ca^{2+} sensor	Troponin	Troponin
Excitation-contraction coupling	L-type Ca^{2+} channel (DHP receptor) in T-tubule membrane coupling to Ca^{2+} release channel (ryanodine receptor) in SR	Ca^{2+} entry via L-type Ca^{2+} channel (DHP receptor) triggers Ca^{2+}-induced Ca^{2+} release from SR
Terminates contraction	Breakdown of ACh by acetylcholinesterase	Action potential repolarization
Twitch duration	20–200 msec	200–400 msec
Regulation of force	Frequency and multifiber summation	Regulation of calcium entry
Metabolism	Oxidative, glycolytic	Oxidative

CALCIUM-INDUCED CALCIUM RELEASE

1. "Excitation" (Depolarization of plasma membrane)
2. Opening of voltage-sensitive Ca^{++} channels in transverse tubules
3. Flow of Ca^{++} into cytosol (small amount ~20%)
4. Ca^{++} binds to Ca^{++} receptors (Ryanodine receptor) on the external surface of the sarcoplasmic reticulum within the cell
5. Opening of Ca^{++} channels (large amount of calcium release ~ 80%)
6. Flow of Ca^{++} into cytosol
7. Cytosolic Ca^{++} conc. increases (10^{-7}M to ~10^{-5}M)
8. Contraction

0.1 µM to 100 µM

Calcium-Induced Calcium Release



Essentially defines 'contractility".

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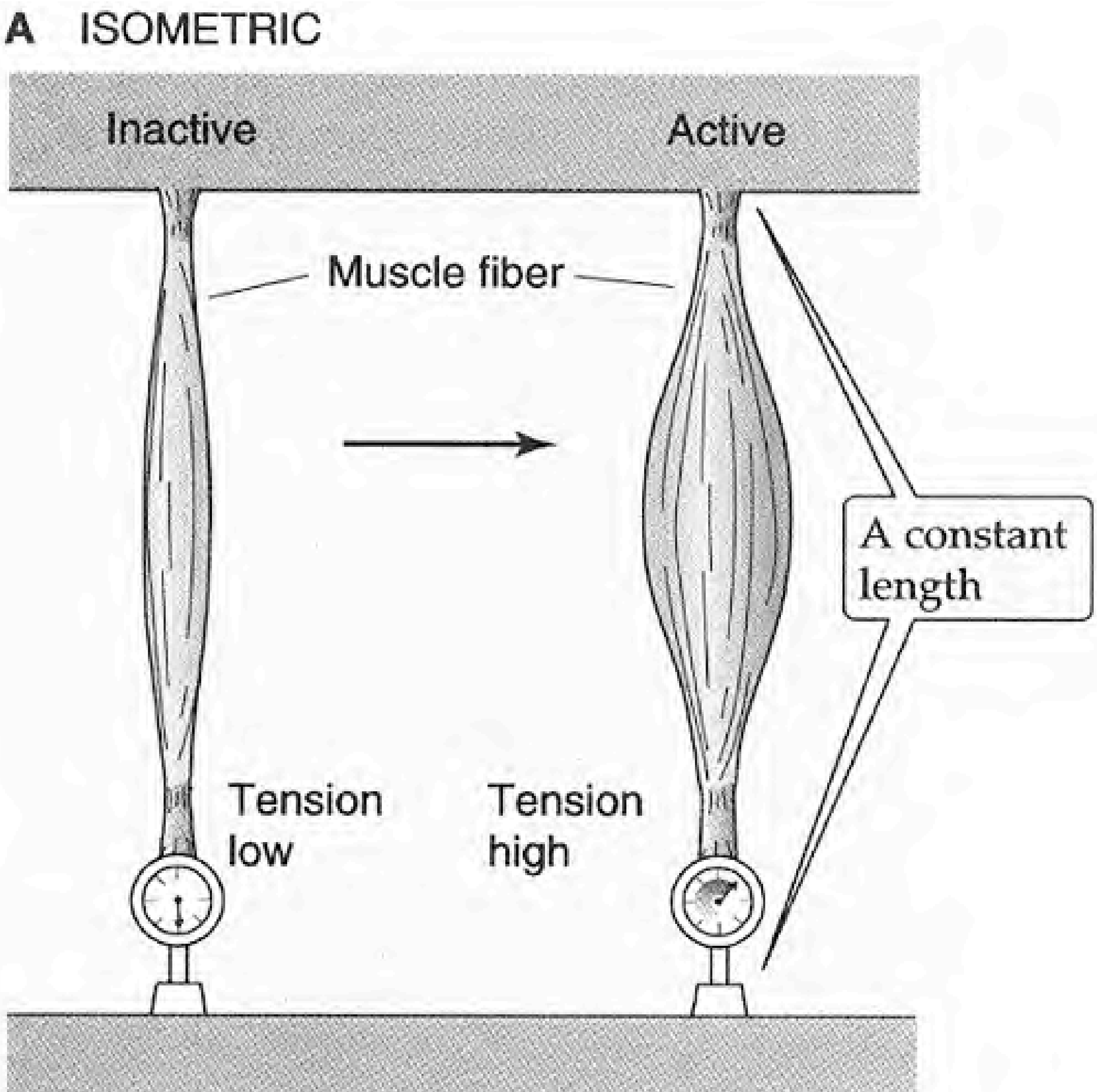
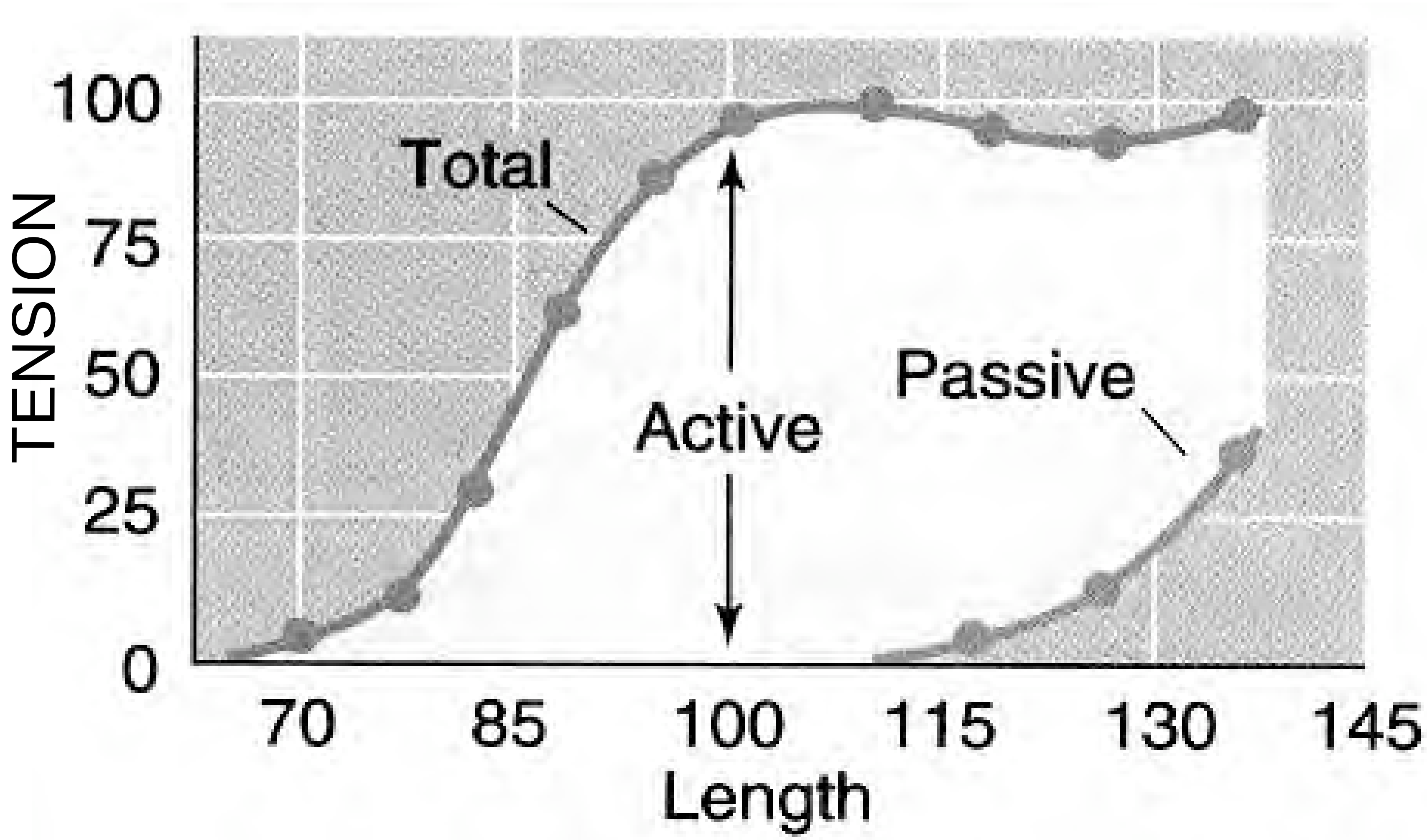

A ISOMETRIC
Inactive
Active
Muscle fiber
A constant length
Tension low
Tension high

ISOMETRIC LENGTH-TENSION



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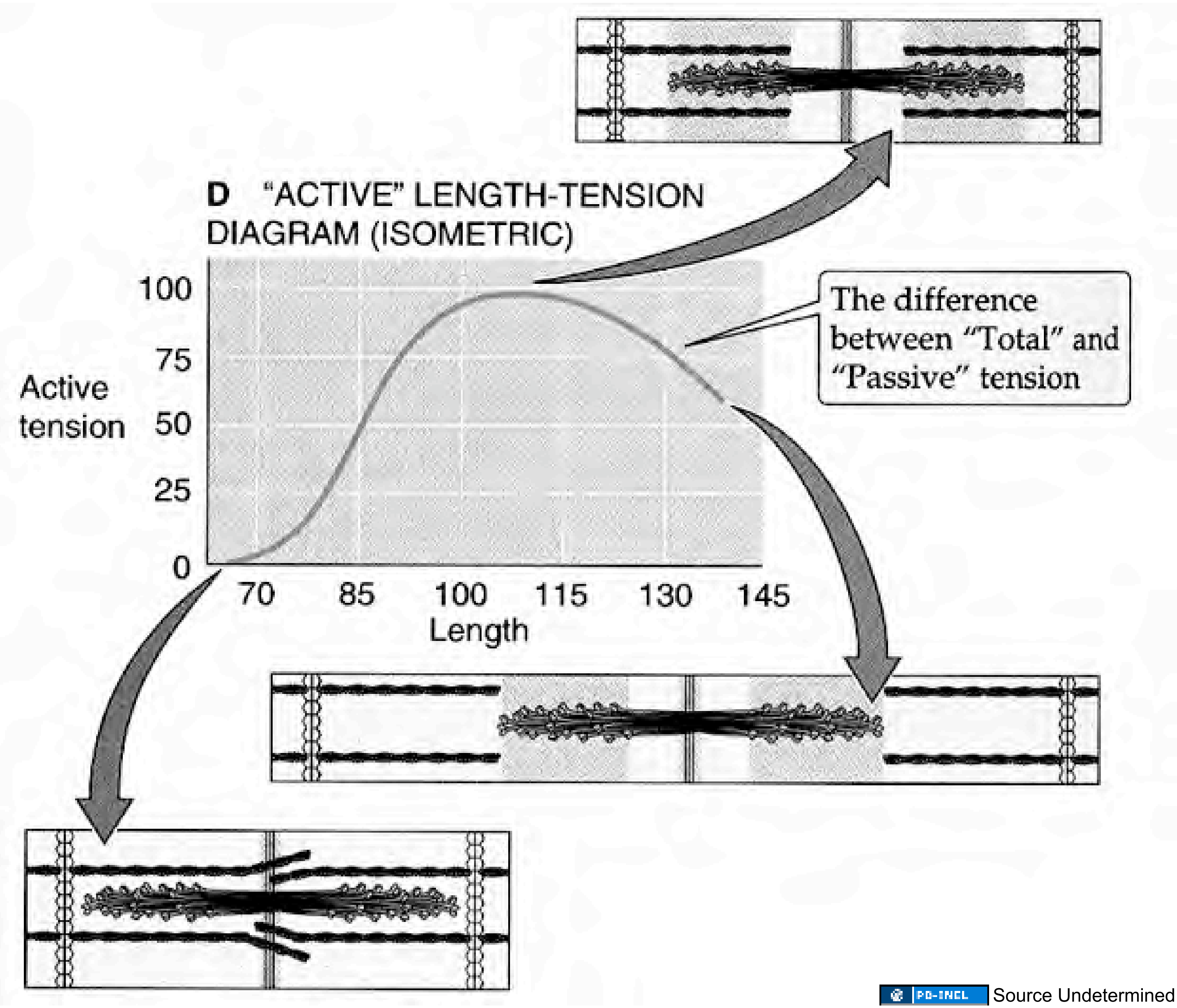
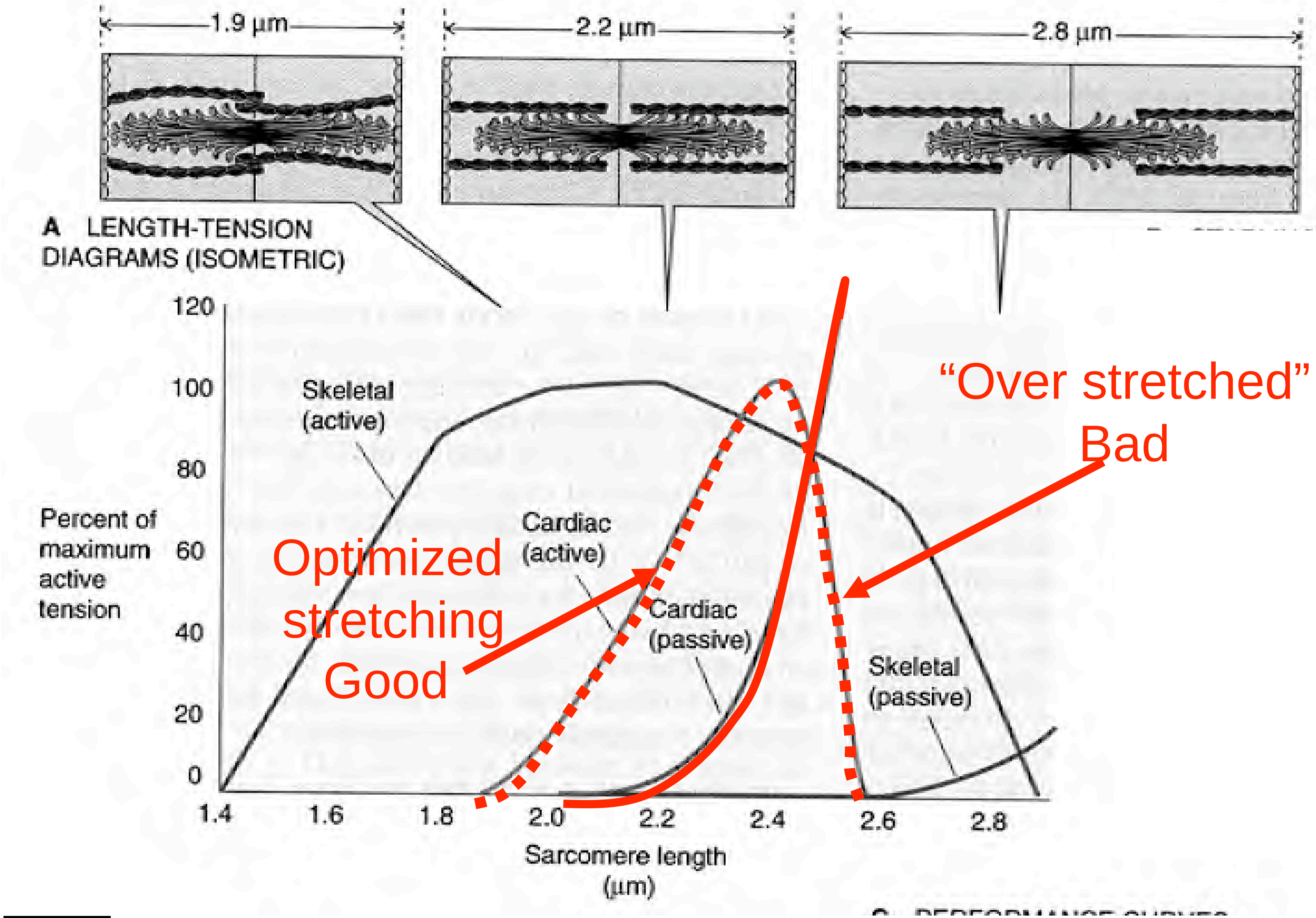
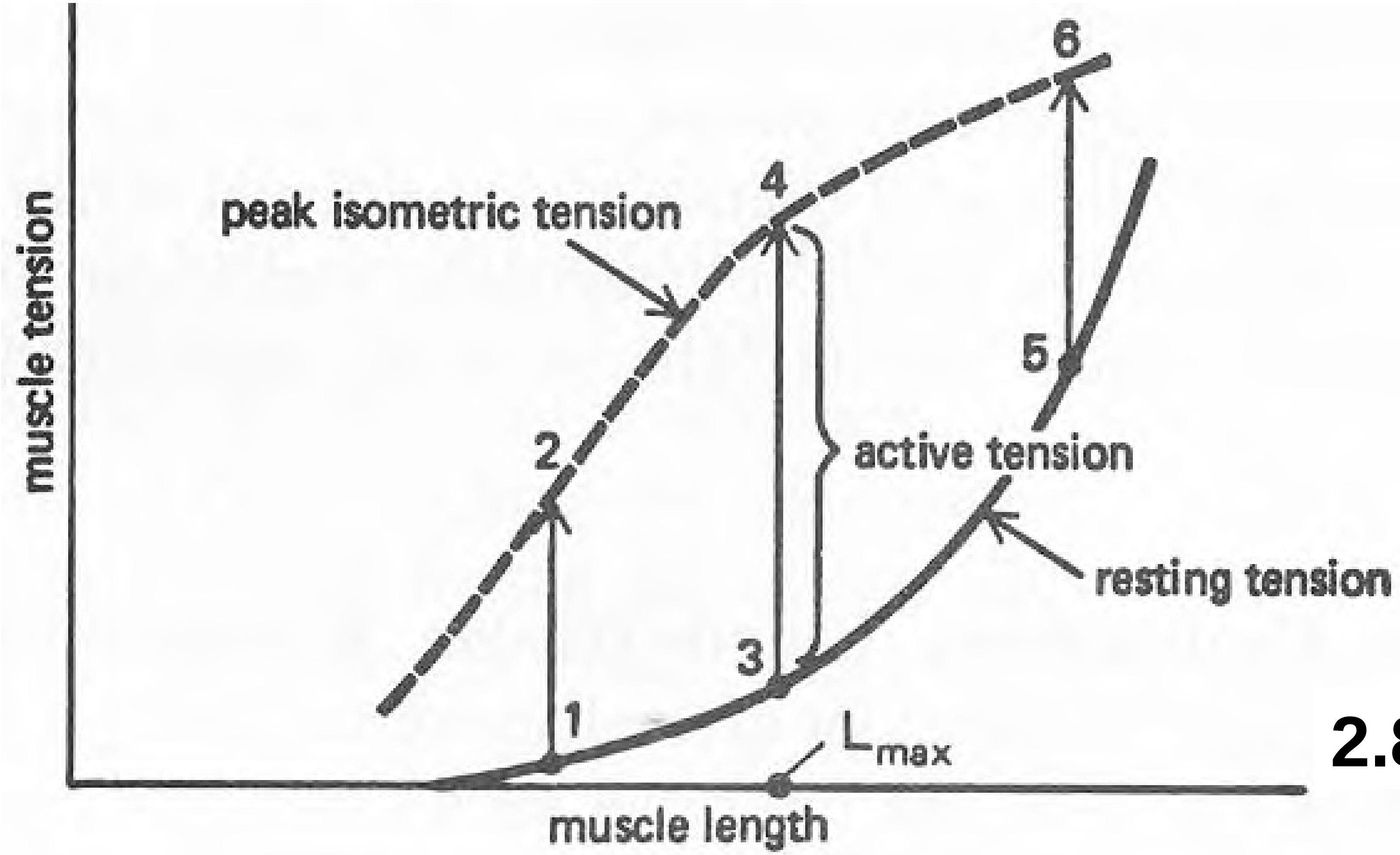

D "ACTIVE" LENGTH-TENSION DIAGRAM (ISOMETRIC)
100
75
50
25
0
Active tension
70
85
100
115
130
145
Length
The difference between "Total" and "Passive" tension


1.9 µm
2.2 µm
2.8 µm
A LENGTH-TENSION DIAGRAMS (ISOMETRIC)
120
100
80
60
40
20
0
Percent of maximum active tension
Skeletal (active)
Cardiac (active)
Cardiac (passive)
Skeletal (passive)
1.4
1.6
1.8
2.0
2.2
2.4
2.6
2.8
Sarcomere length (µm)
"Over stretched" Bad
Optimized stretching Good

Passive stretch & Isometric contraction

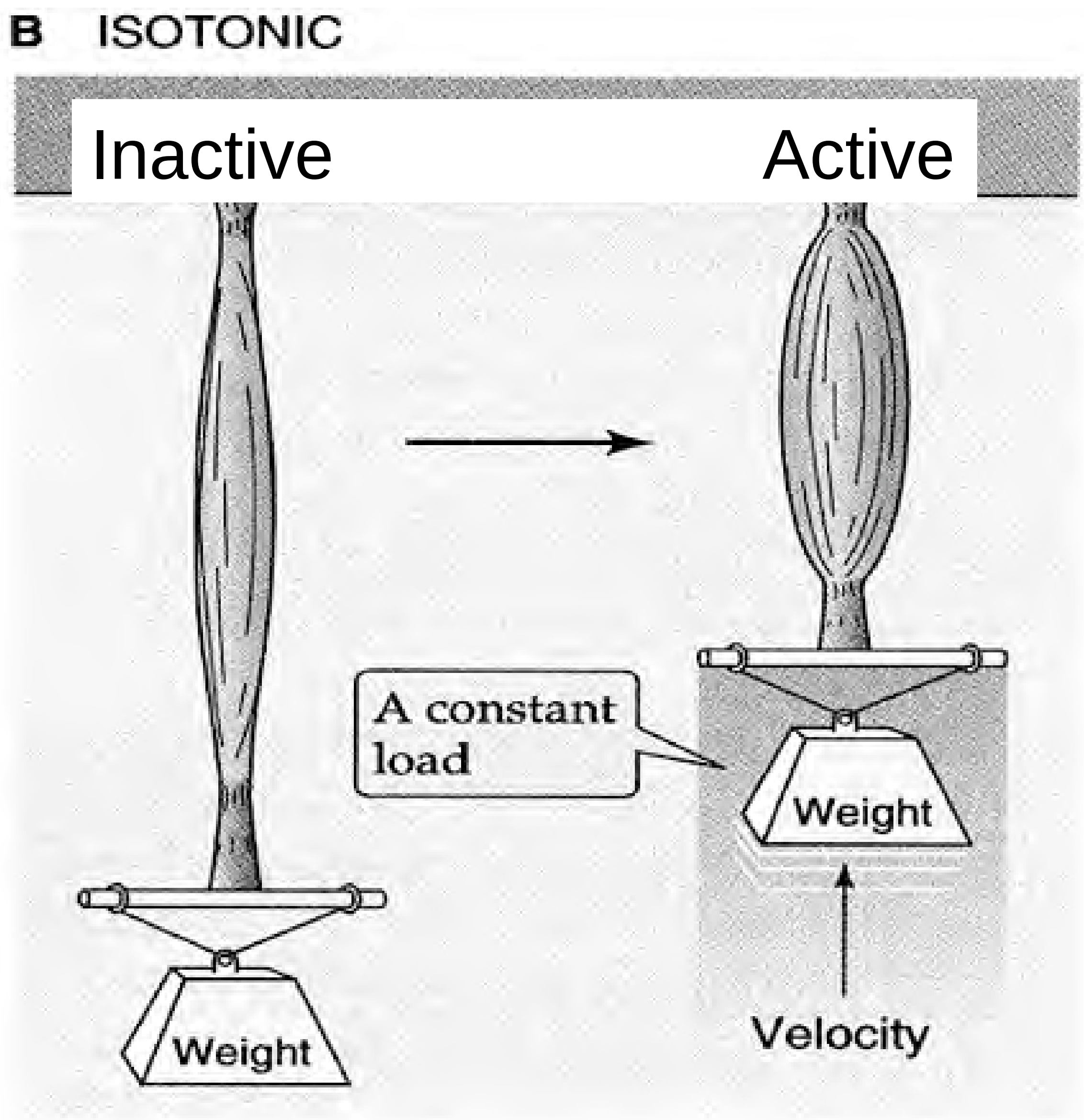


2.8 MH

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

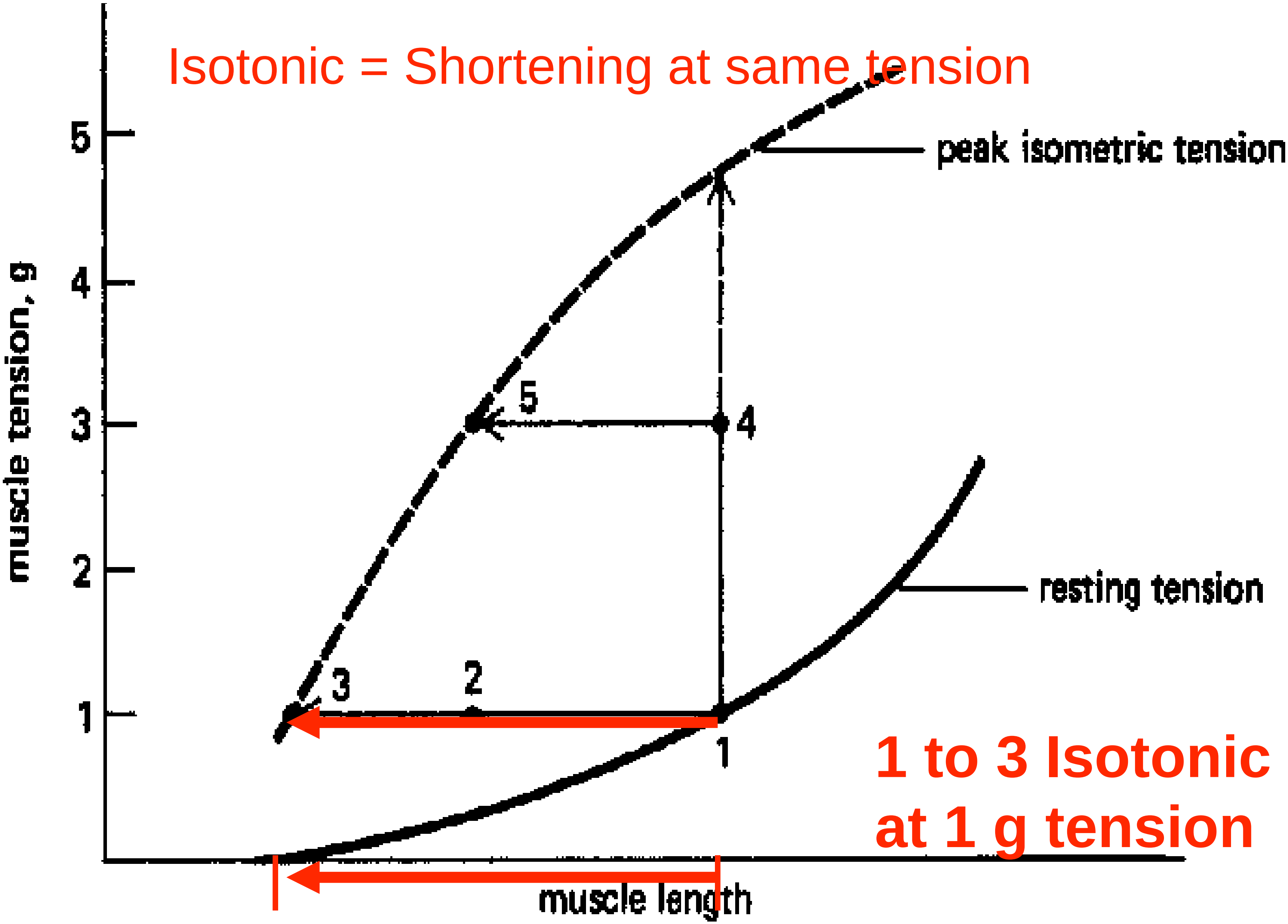
"At rest"
Diastole

Contracting
Systole



Same <u>tension (load)</u> but shorter length.

Isotonic = Shortening at same tension



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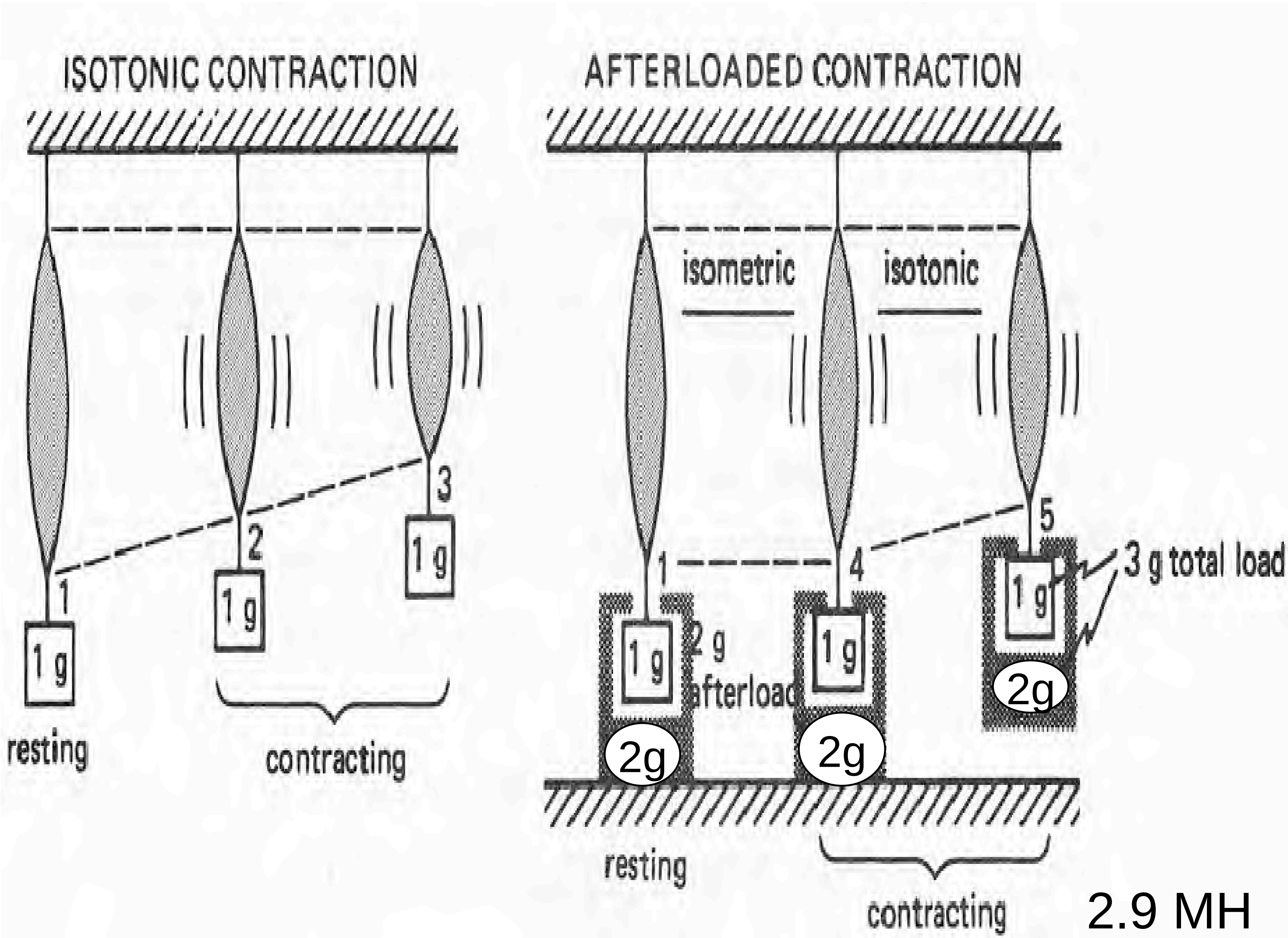

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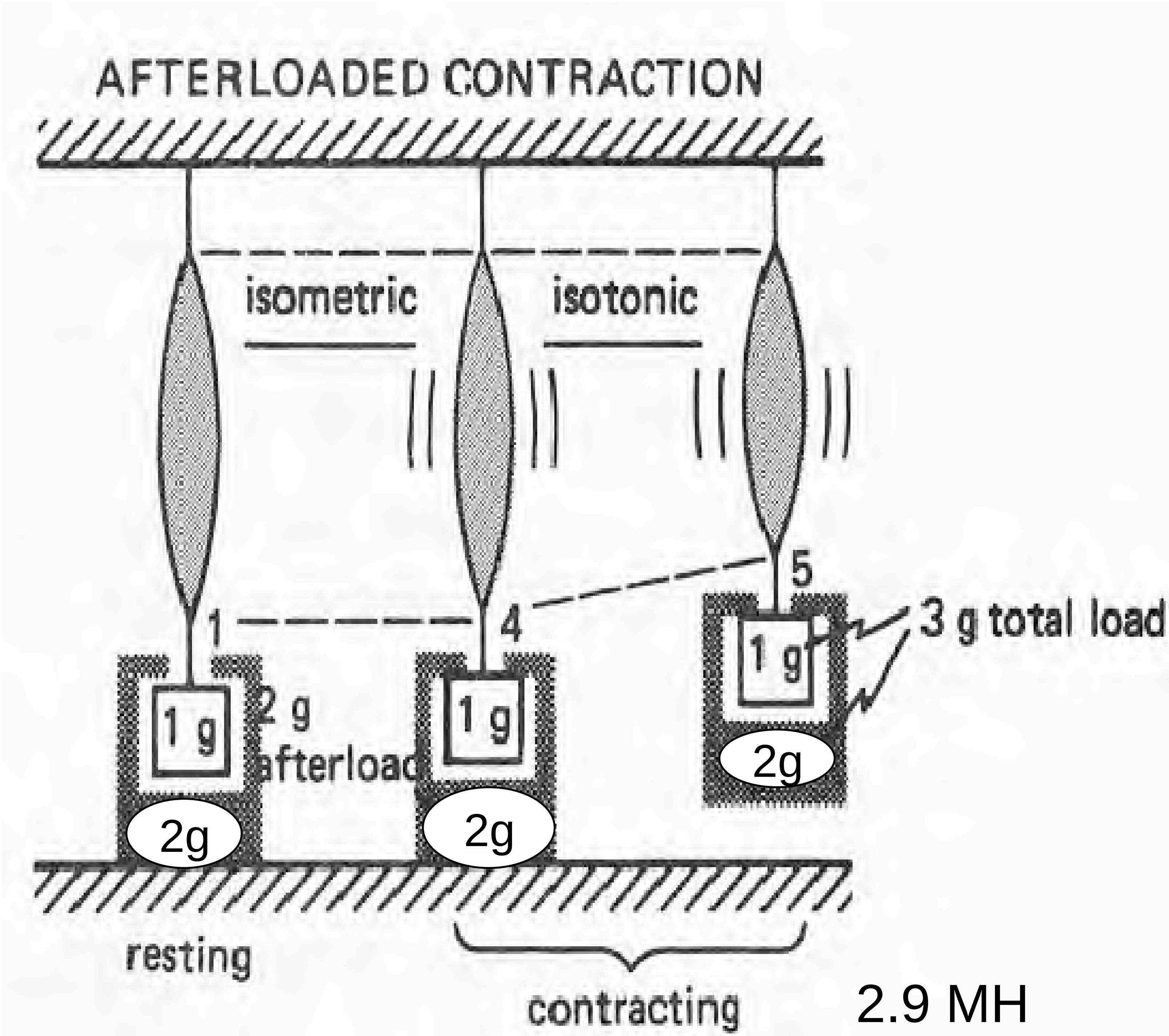
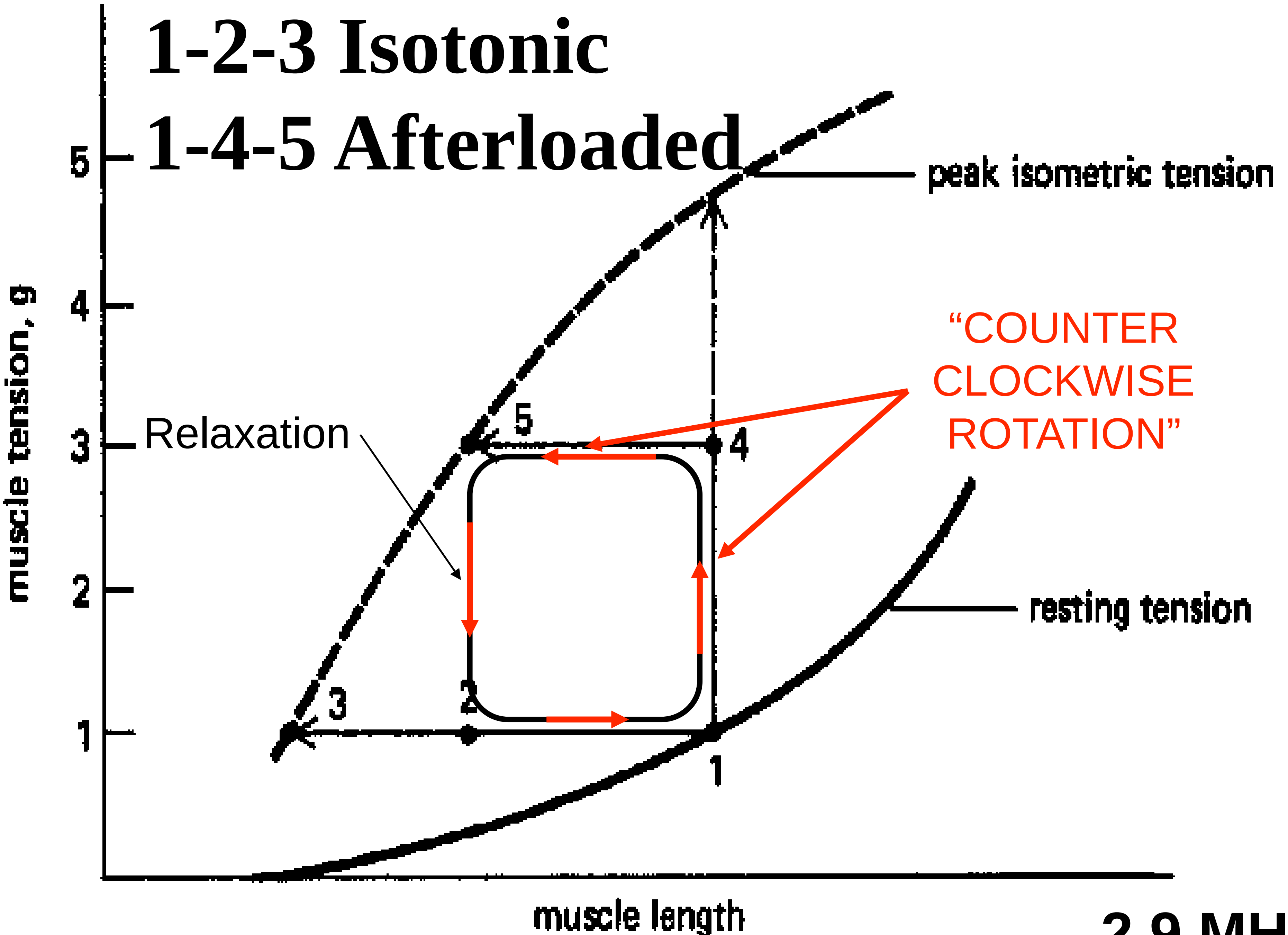


2.9 MH

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

1-2-3 Isotonic
1-4-5 Afterloaded



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2.9 MH

Terms Related to Cardiac Performance

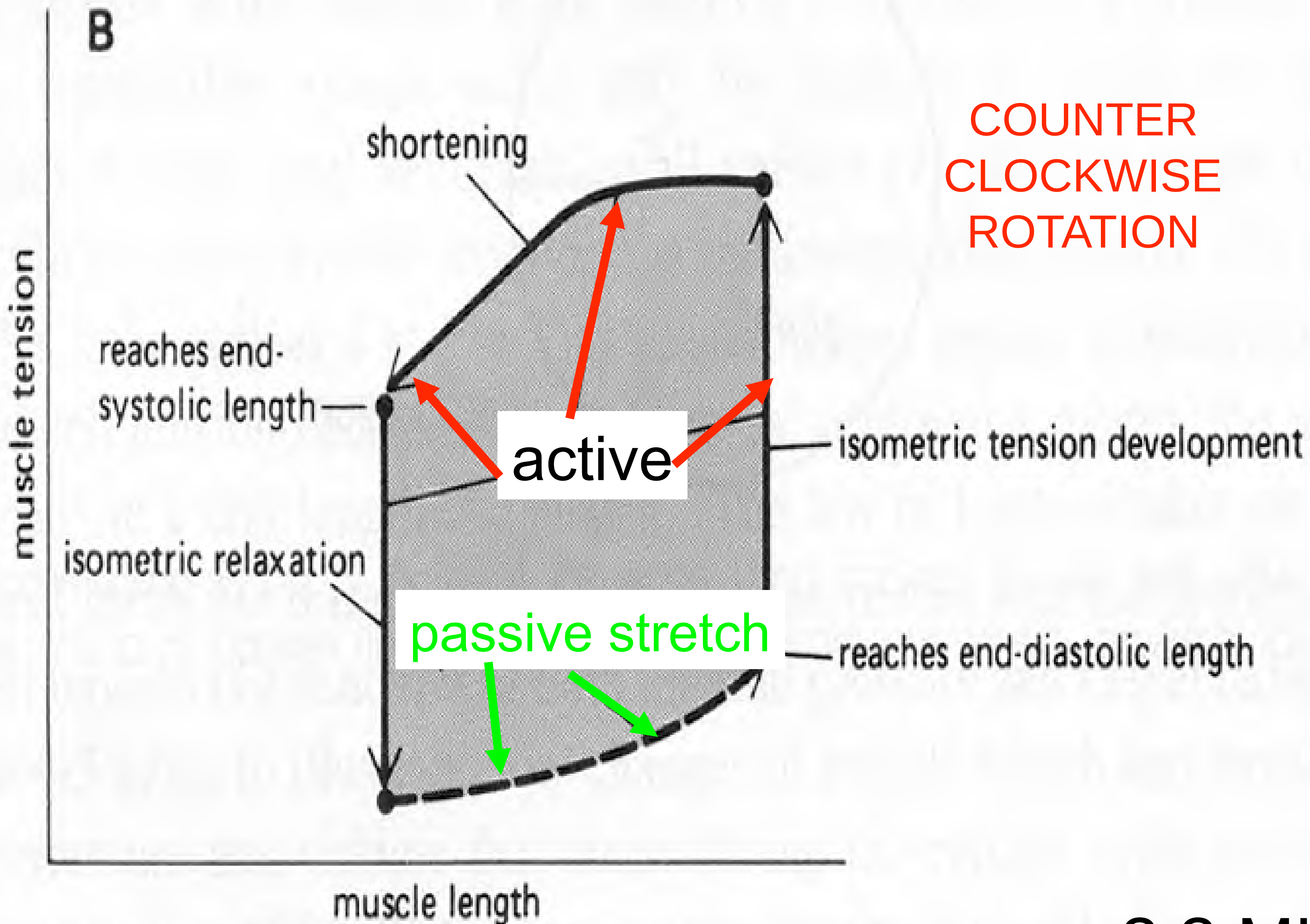
Preload - The ventricular wall tension at the end of diastole.

Afterload -- The ventricular wall tension during contraction; the resistance that must be overcome for the ventricle to eject its contents. Approximated clinically by systolic ventricular or arterial pressure.

Tuesday 10/28/08, 11:00
Cardiac Muscle II

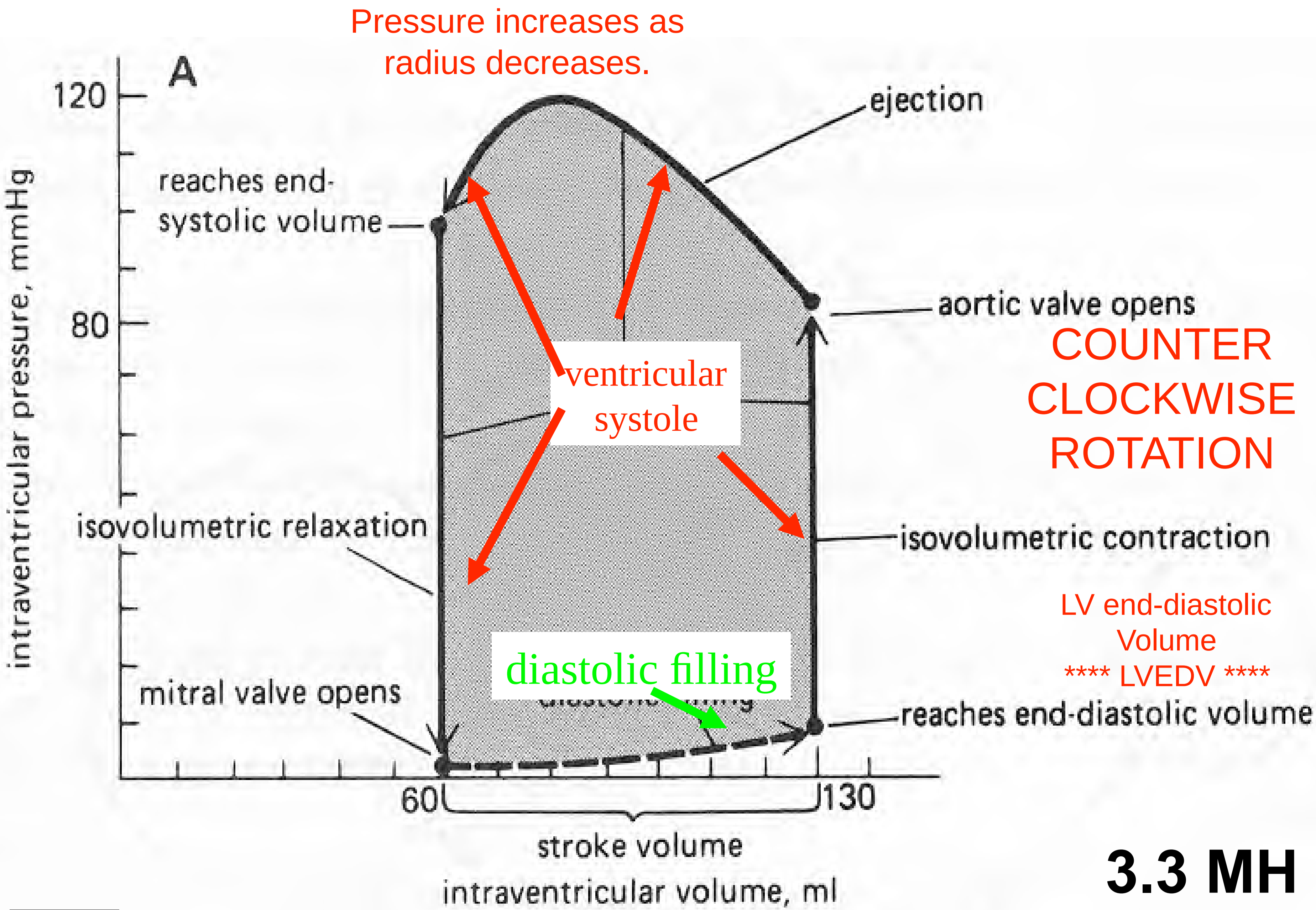
22 Slides, 50 min

1. Afterloaded contraction (length-tension)
2. Afterloaded contraction (volume-pressure)
3. LaPlace
4. Wiggers diagram
5. Stroke volume & Ejection Fraction
6. Cardiac Output
7. Right pump
8. Preload (Frank-Starling), Afterload, & Contractility



COUNTER CLOCKWISE ROTATION

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



3.3 MH

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

Ejection Fraction = 70/130 = 54%

Law of La Place

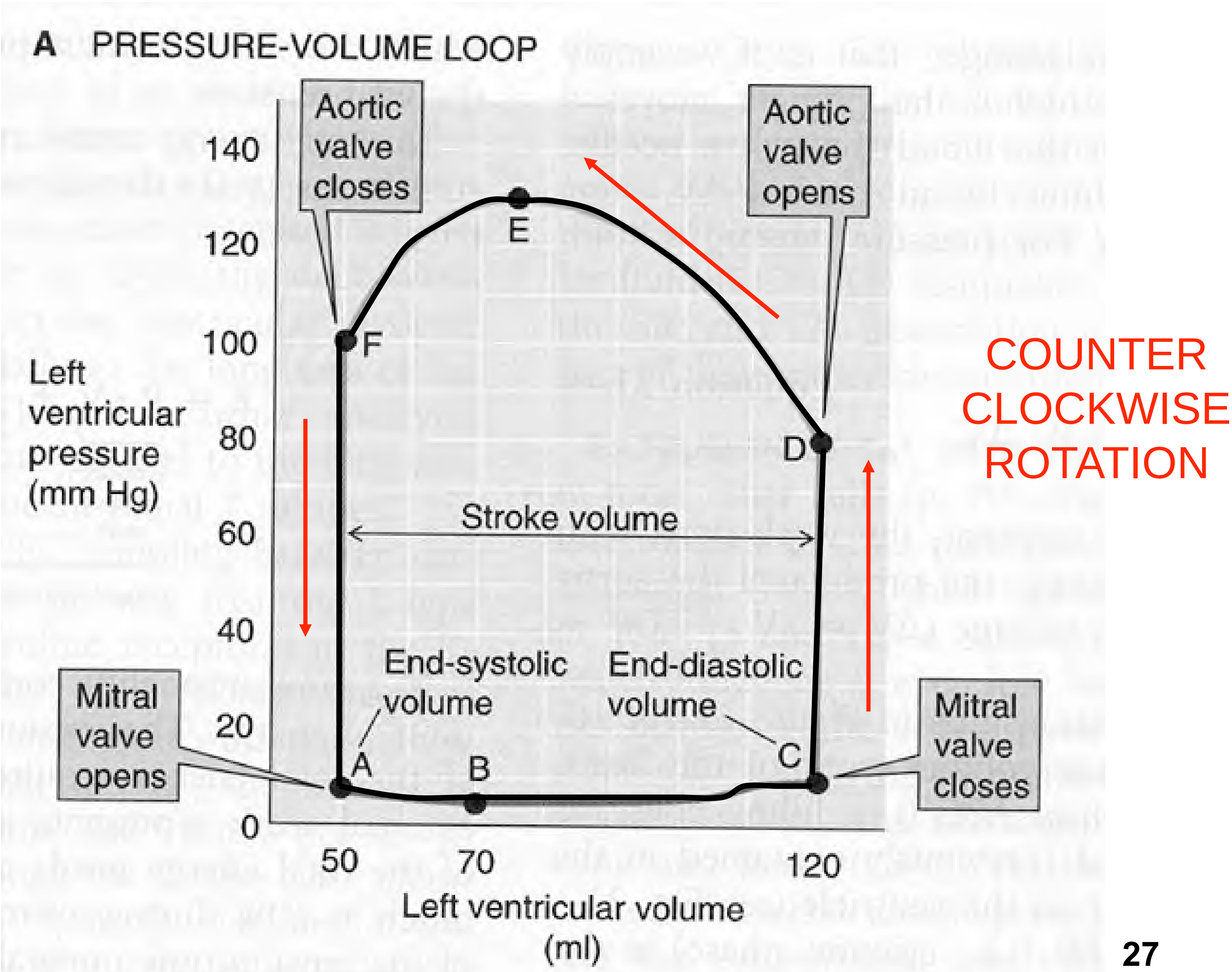
T = P x r (see page 44 of M&H)

The tension (T) in the ventricular wall depends upon both the pressure (P) in the chamber and the radius (r) of the chamber.

Thus as the ventricle gets smaller during ejection the pressure within increases

even at the same muscle tension.

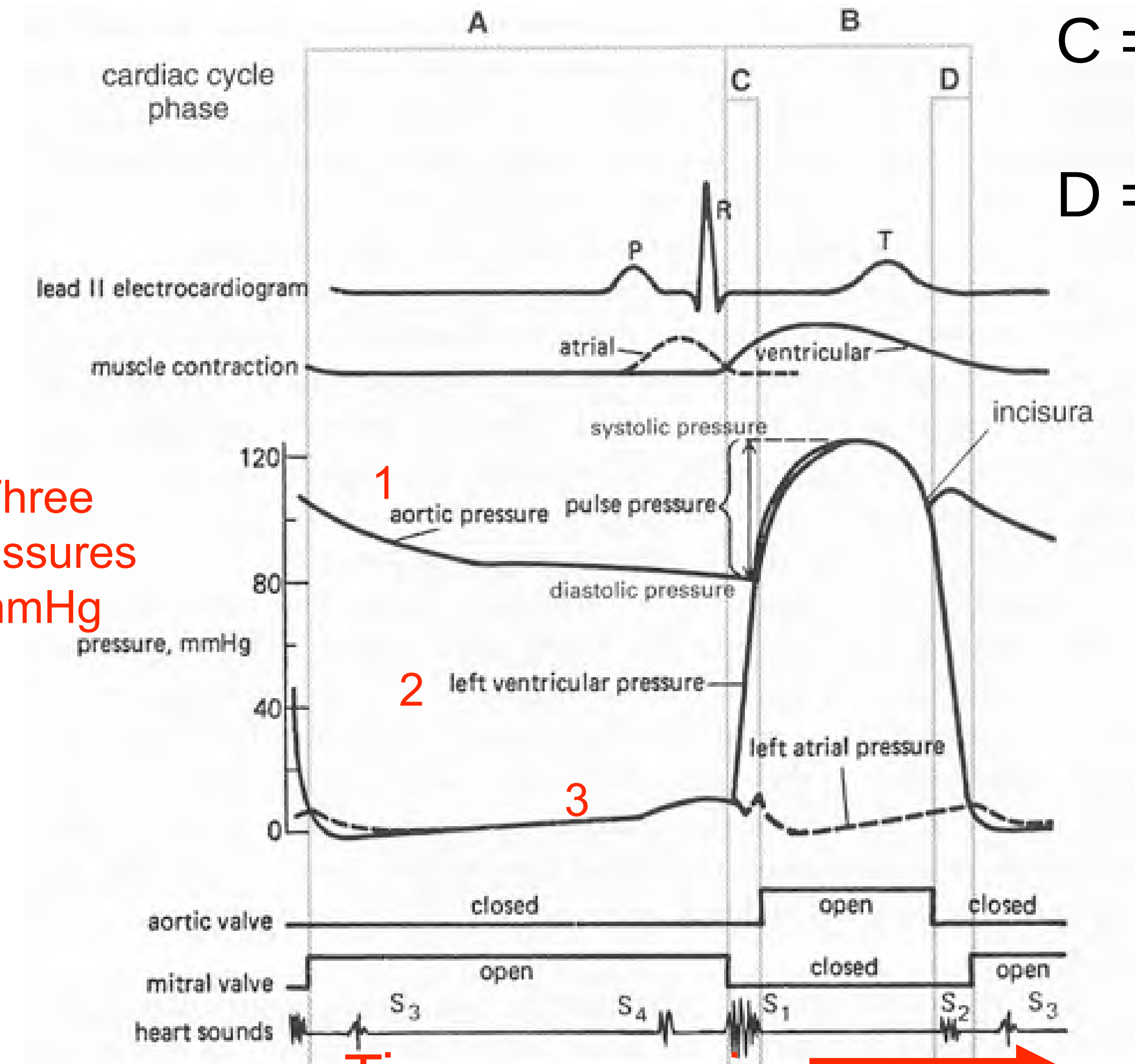
Same T = ↑P x ↓r


A PRESSURE-VOLUME LOOP
Aortic valve closes
Aortic valve opens
Mitral valve opens
Mitral valve closes
Left ventricular pressure (mm Hg)
140
120
100
80
60
40
20
0
50
70
120
Left ventricular volume (ml)
E
F
D
A
B
C
Stroke volume
End-systolic volume
End-diastolic volume
COUNTER CLOCKWISE ROTATION

Ventricular A = diastole B = ventricular systole

C = isovolumetric contraction

D = isovolumetric relaxation



Three Pressures mmHg

1
2
3

Time on x axis

Top 3.1 MH

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

Systole & Diastole

Text books vary in definitions but the more common uses of the unmodified terms “systole” and “diastole” are:

Systole is the <u>period</u> from the closing of the atrio-ventricular valve (mitral) to the closing of the aortic valve (ventricular contraction).

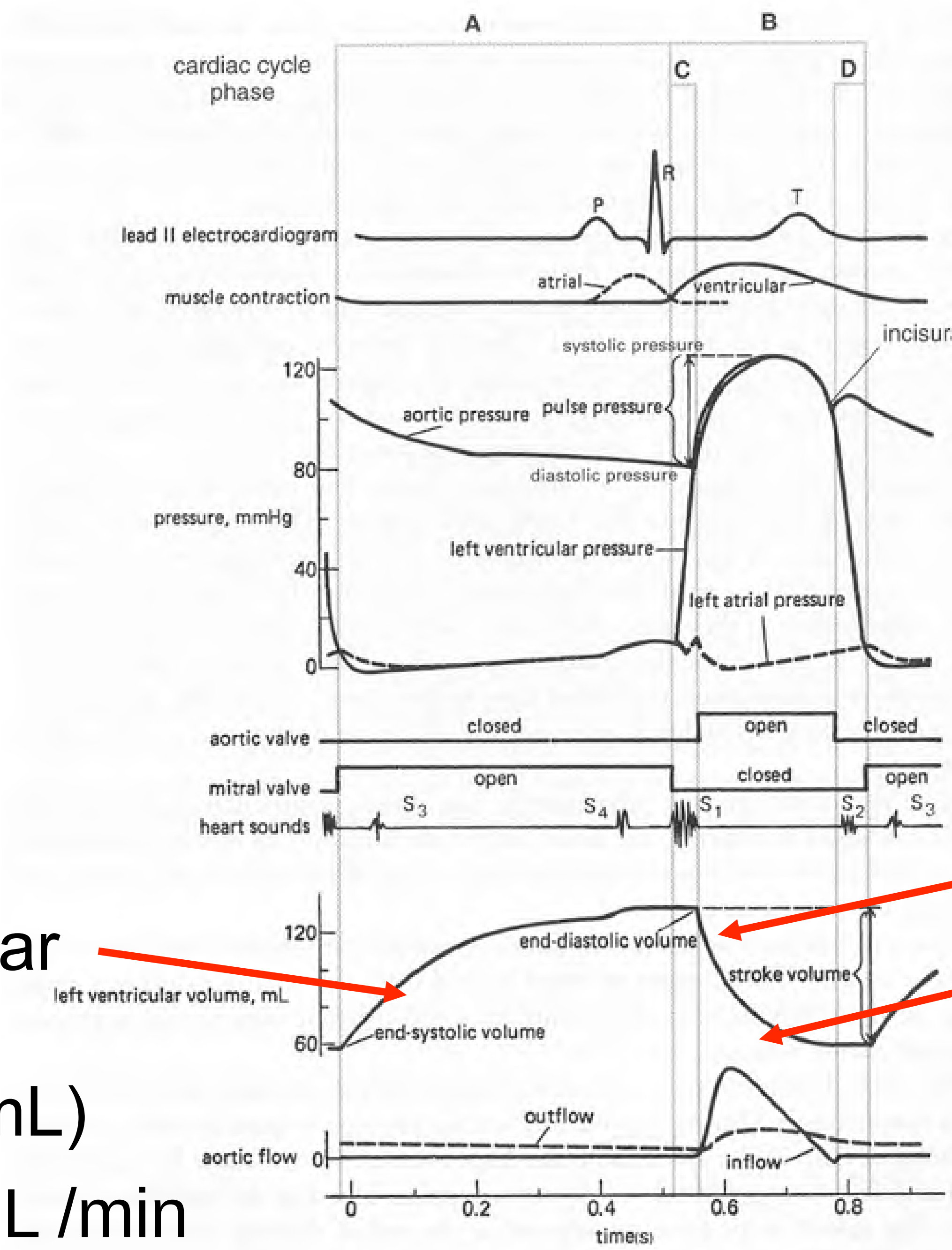
Diastole is the <u>period</u> from the closing of the aortic valve to the closing of the atrio-ventricular valve (ventricular relaxation and filling).

M & H NOTE: Your text distinguishes <u>ventricular</u> systole from <u>arterial</u> systole:

<u>Ventricular</u> systole is the period from the closing of the atrio-ventricular valve (mitral) until its opening. (Fig 3.1 M &H)

<u>Arterial</u> systole is the period from the opening of the aortic valve until its closing.

3.1 MH



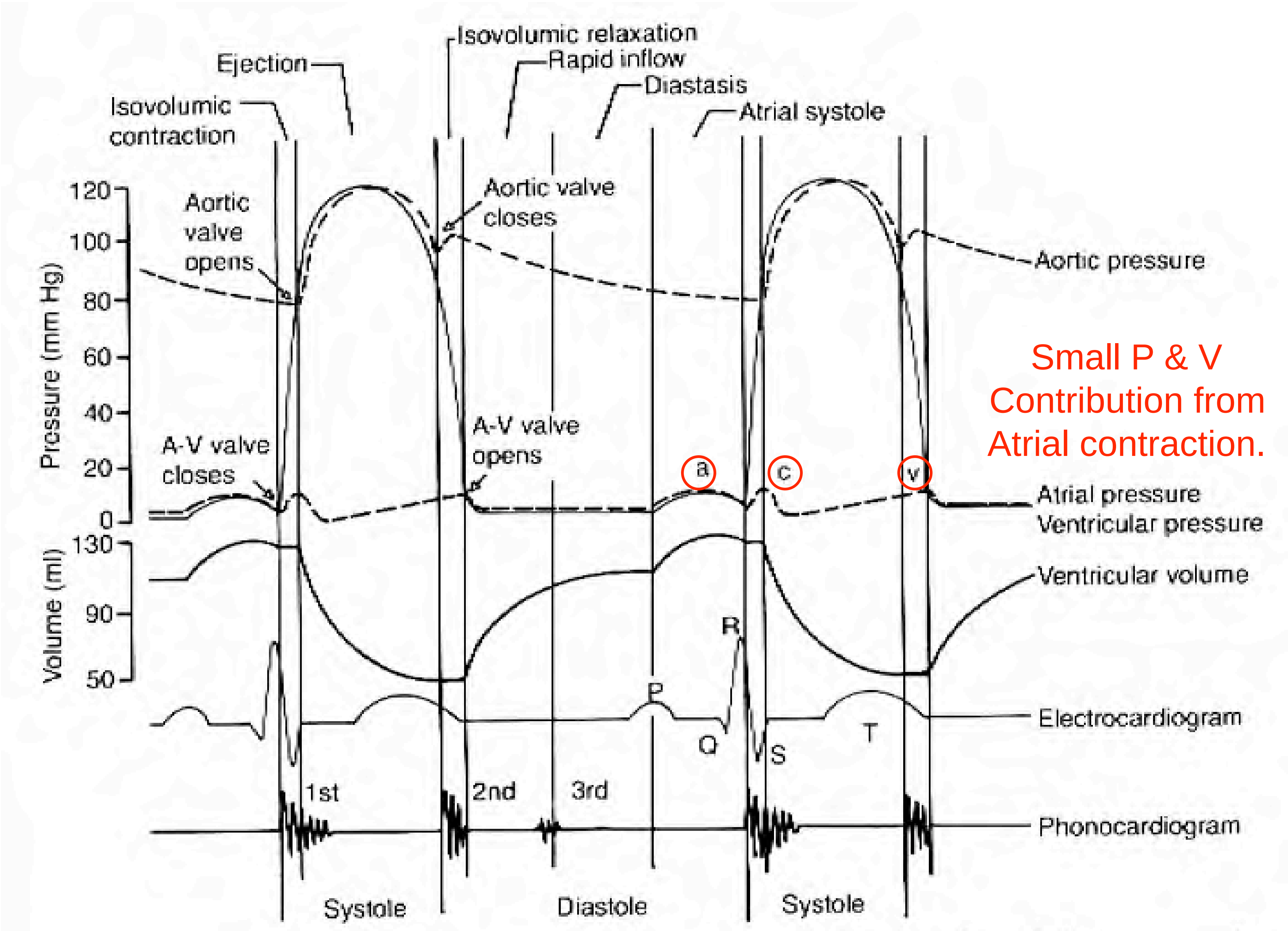
LV end-diastolic Volume ****LVEDV****

Ventricular Filling (volume mL)

Ventricular ejection

Flow mL /min

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



Small P & V Contribution from Atrial contraction.

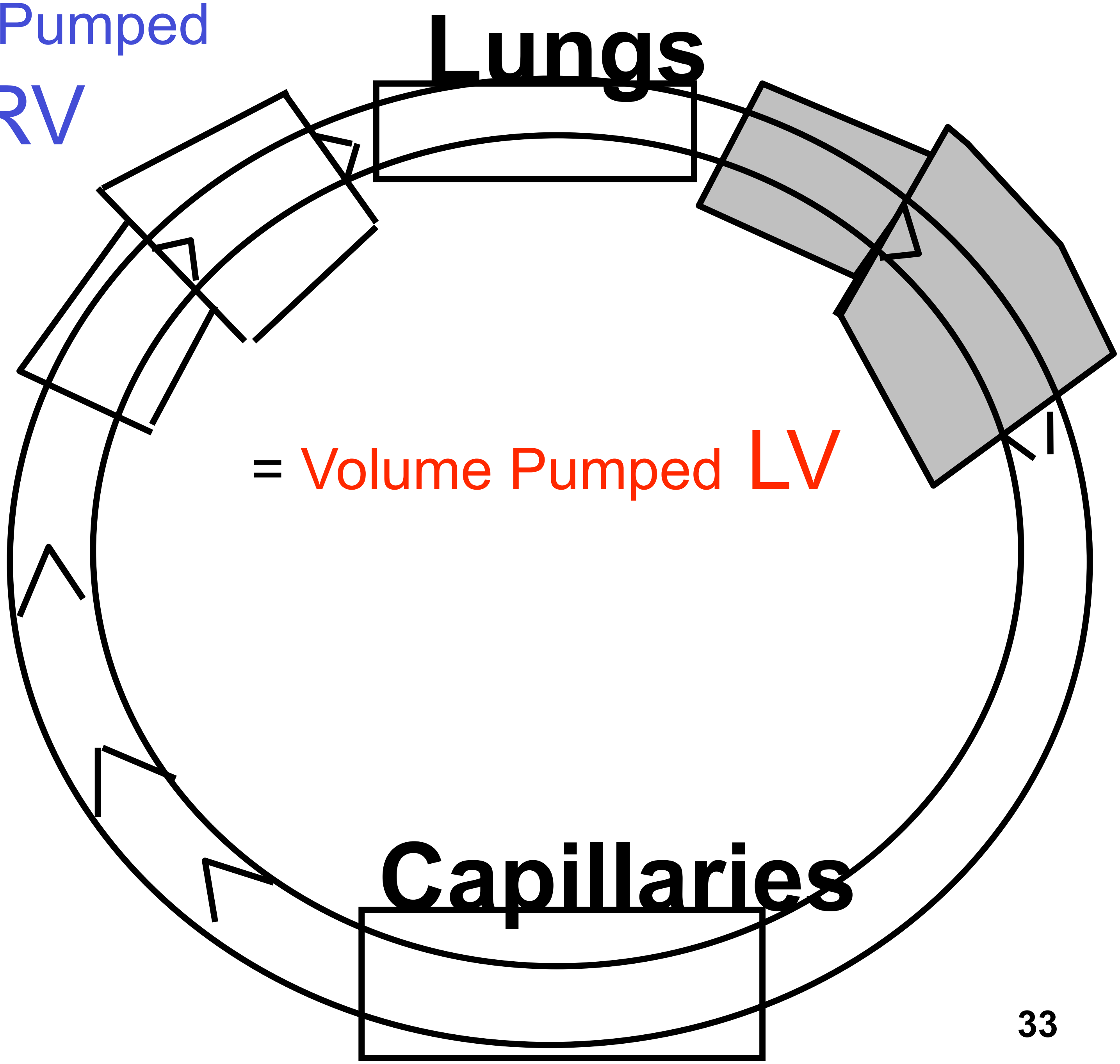
Heart is a Pressure Pump but also pumps volume/time.

Stroke Volume = volume pumped with each beat of the heart.

Heart Rate X Stroke Volume = Cardiac Output

HR	X	SV	=	CO
b/min	X	mL / b	=	mL / min

Volume Pumped RV



= Volume Pumped LV

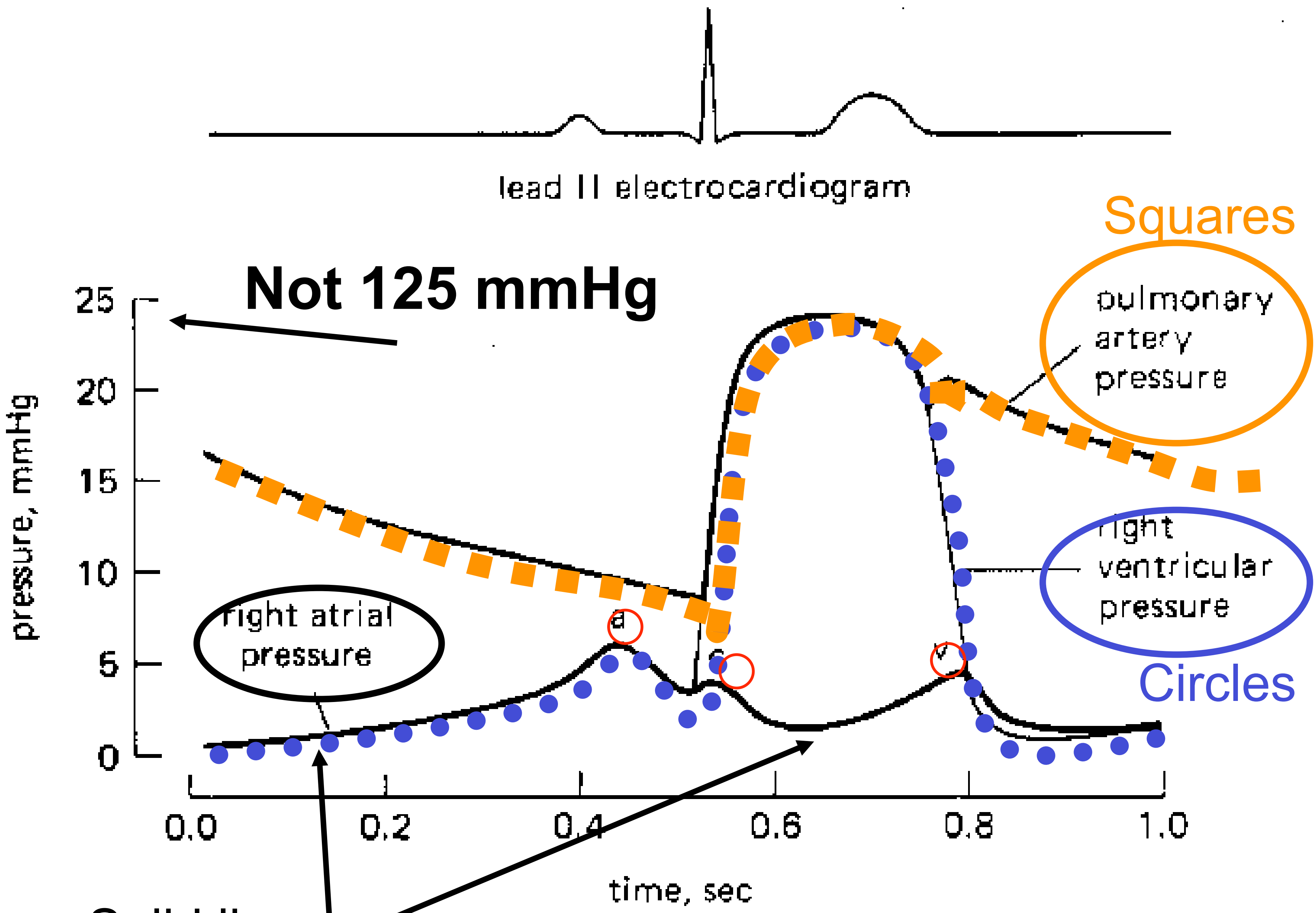
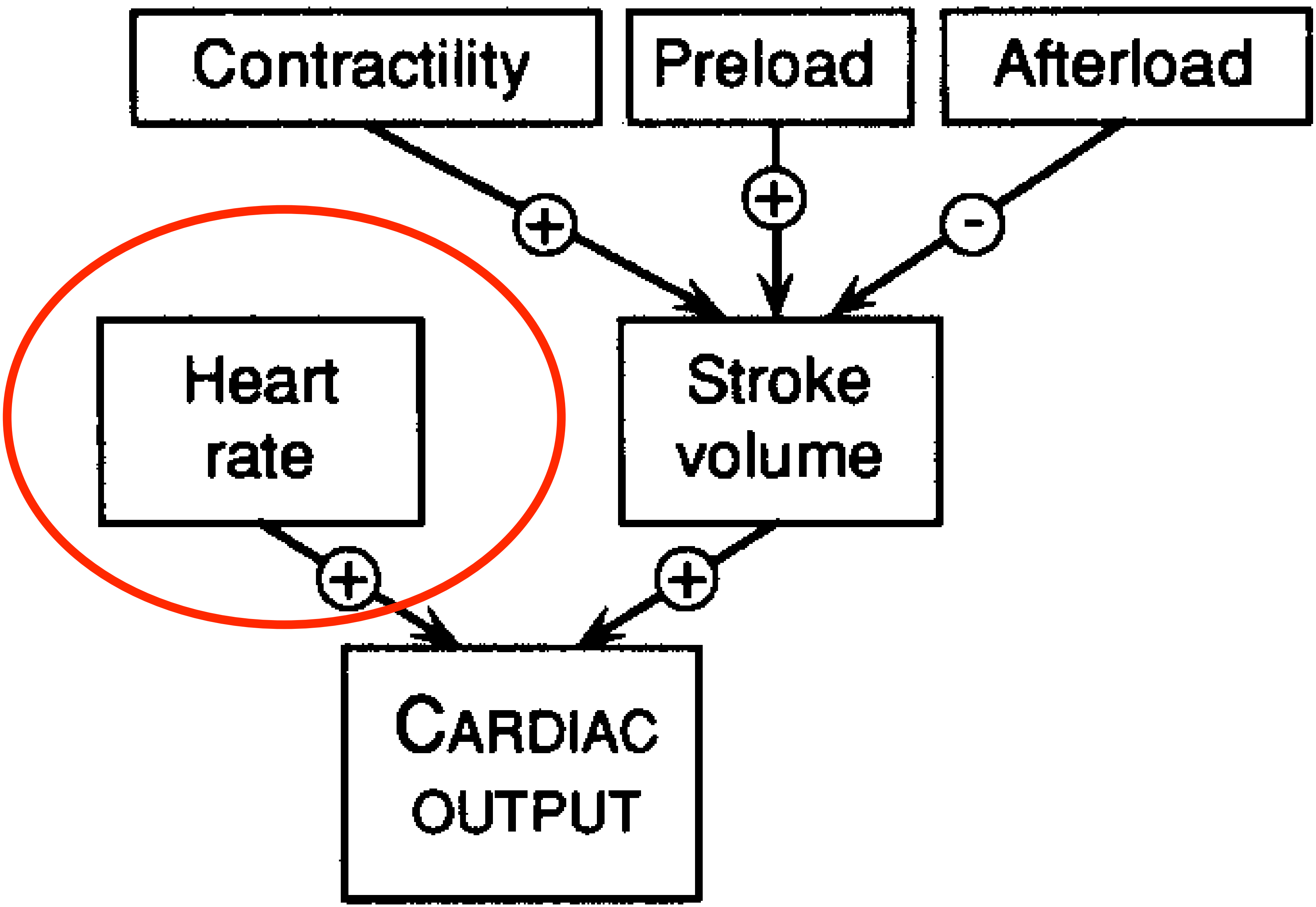


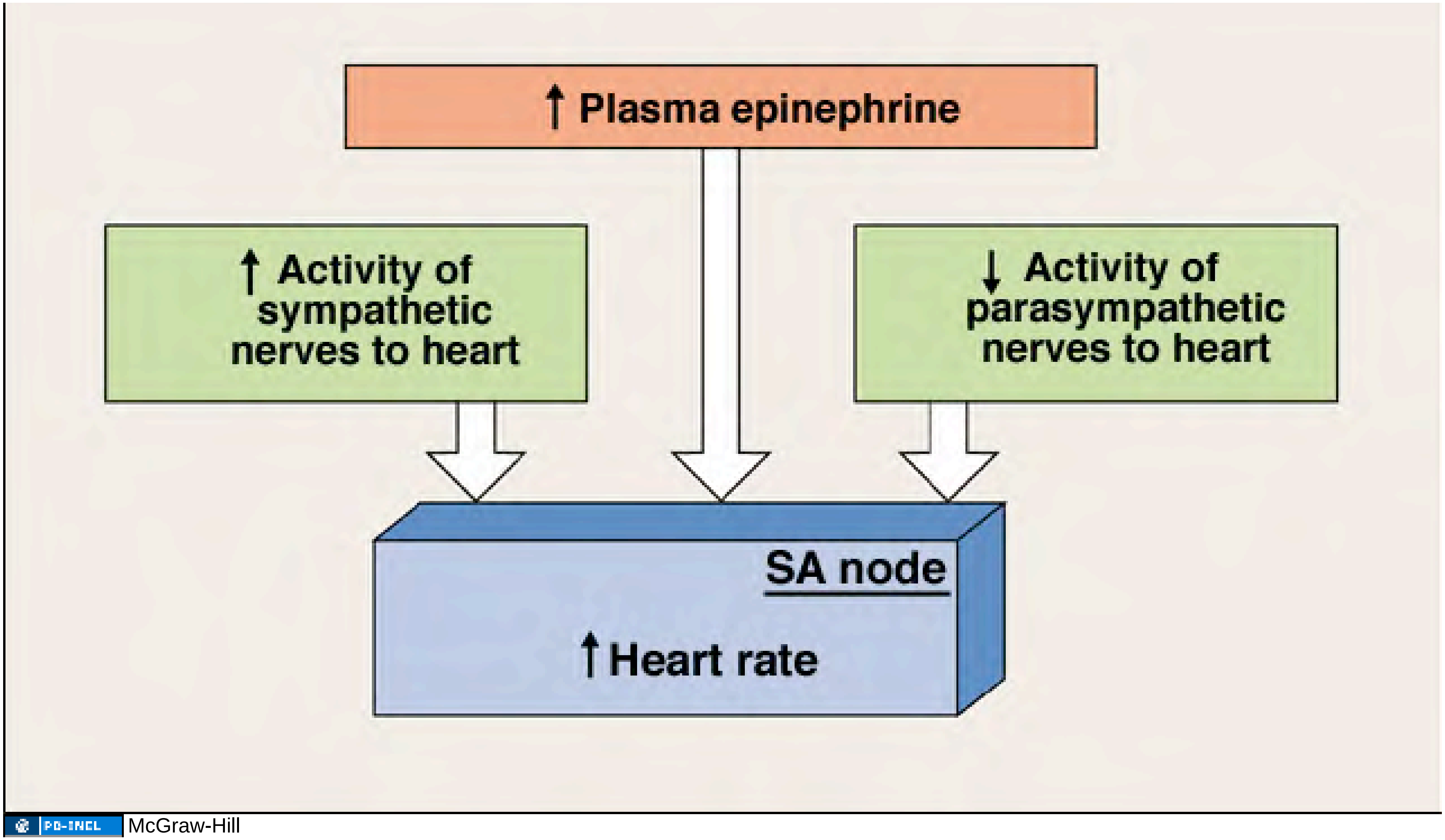
Figure 3.2 Cardiac cycle—right heart.

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


Contractility
Preload
Afterload
+
+
-
Heart rate
Stroke volume
+
+
CARDIAC OUTPUT

Factors influencing heart rate

+ and (-) CHRONOTROPIC EFFECTS

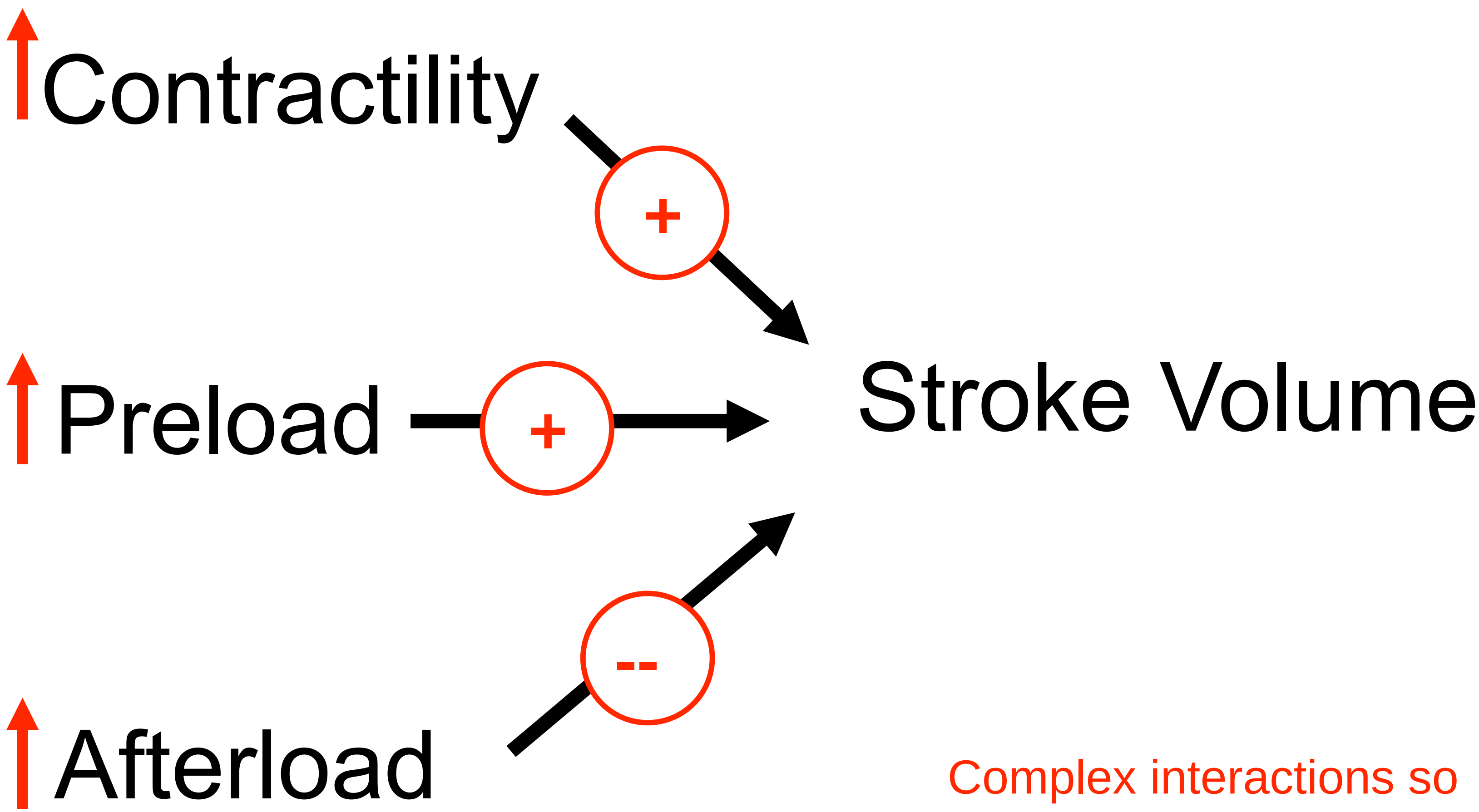


Terms Related to Cardiac Performance

Preload - The ventricular wall tension at the end of diastole.

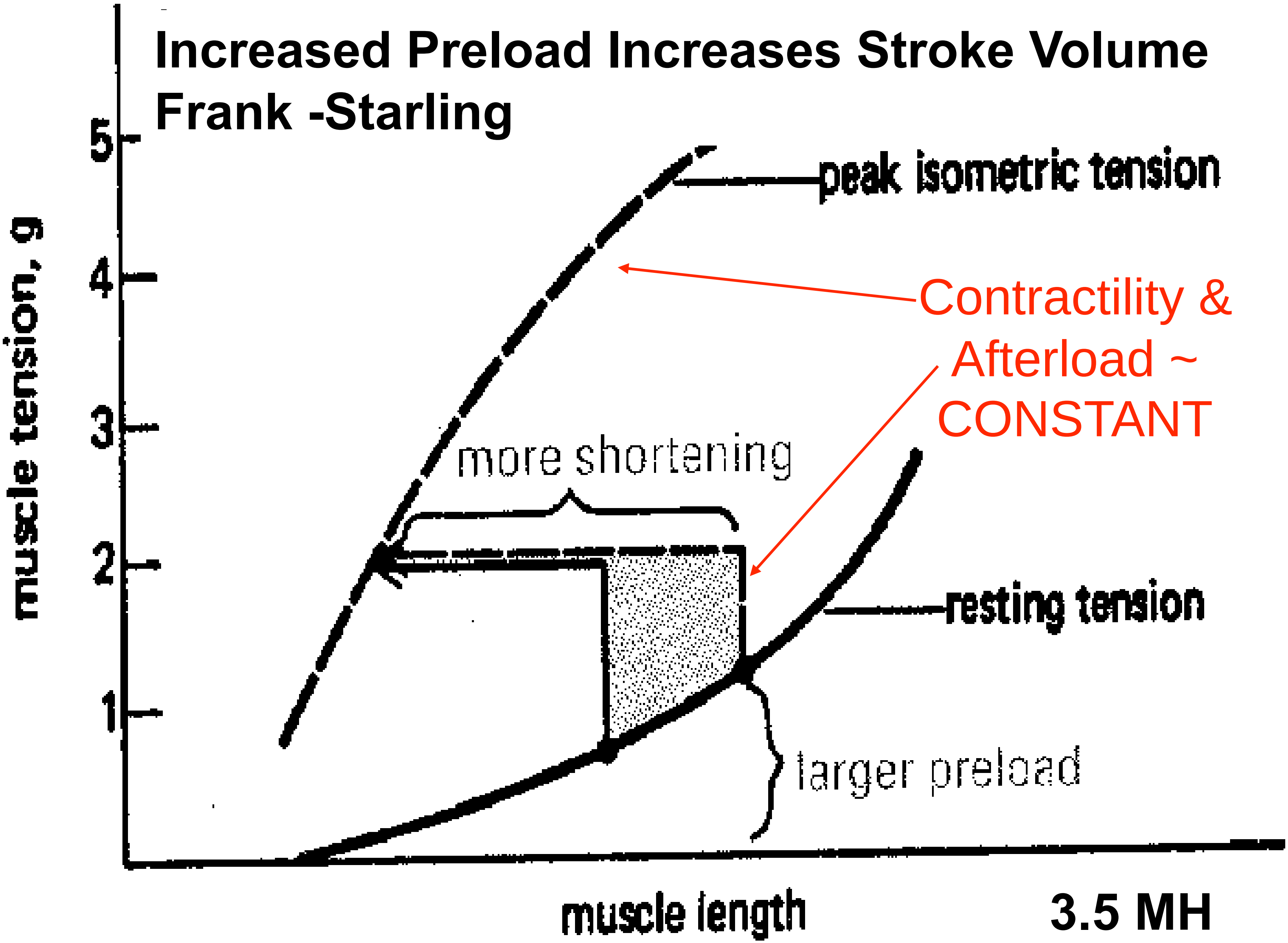
Afterload -- The ventricular wall tension during contraction; the resistance that must be overcome for the ventricle to eject its contents. Approximated by systolic ventricular or arterial pressure.

Contractility -- Property of heart muscle that accounts for changes in strength of contraction independent of preload and afterload.



Complex interactions so
we will treat each separately
with others held constant.

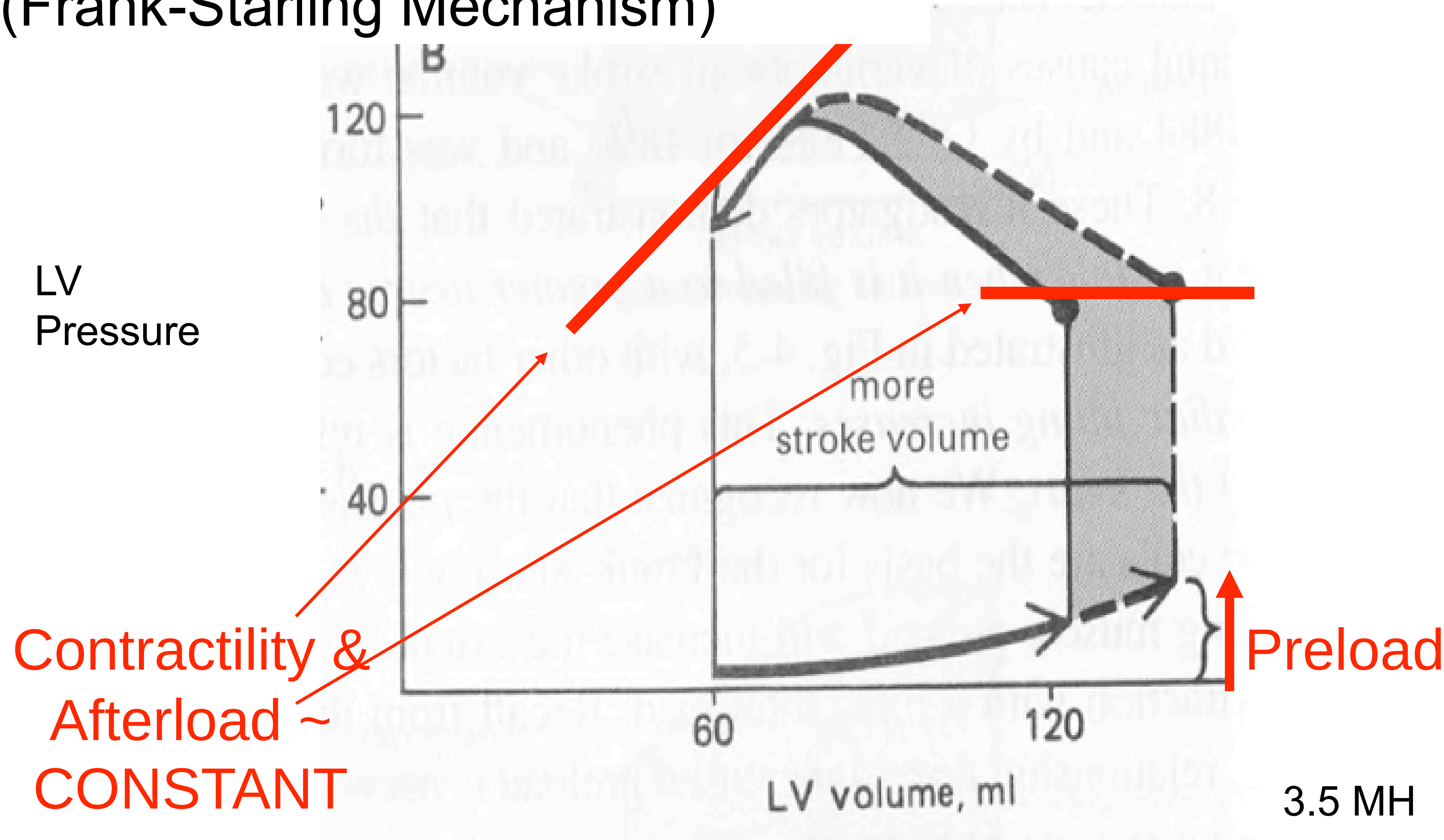
Increased Preload Increases Stroke Volume
Frank -Starling



3.5 MH

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

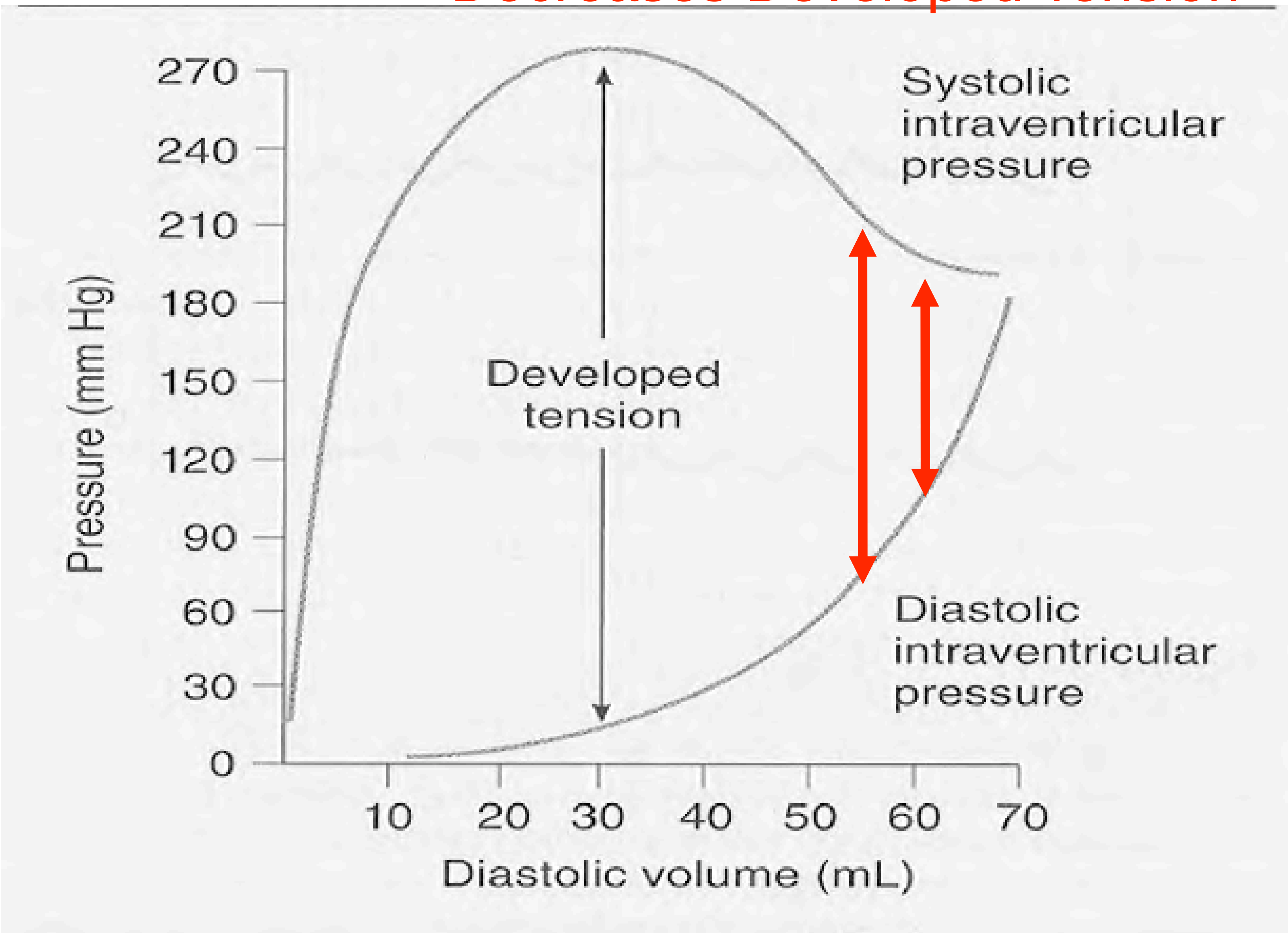
Increased Preload ~ Increases SV (Frank-Starling Mechanism)



3.5 MH

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

However ! Excessive Diastolic Volume or Pressure Decreases Developed Tension



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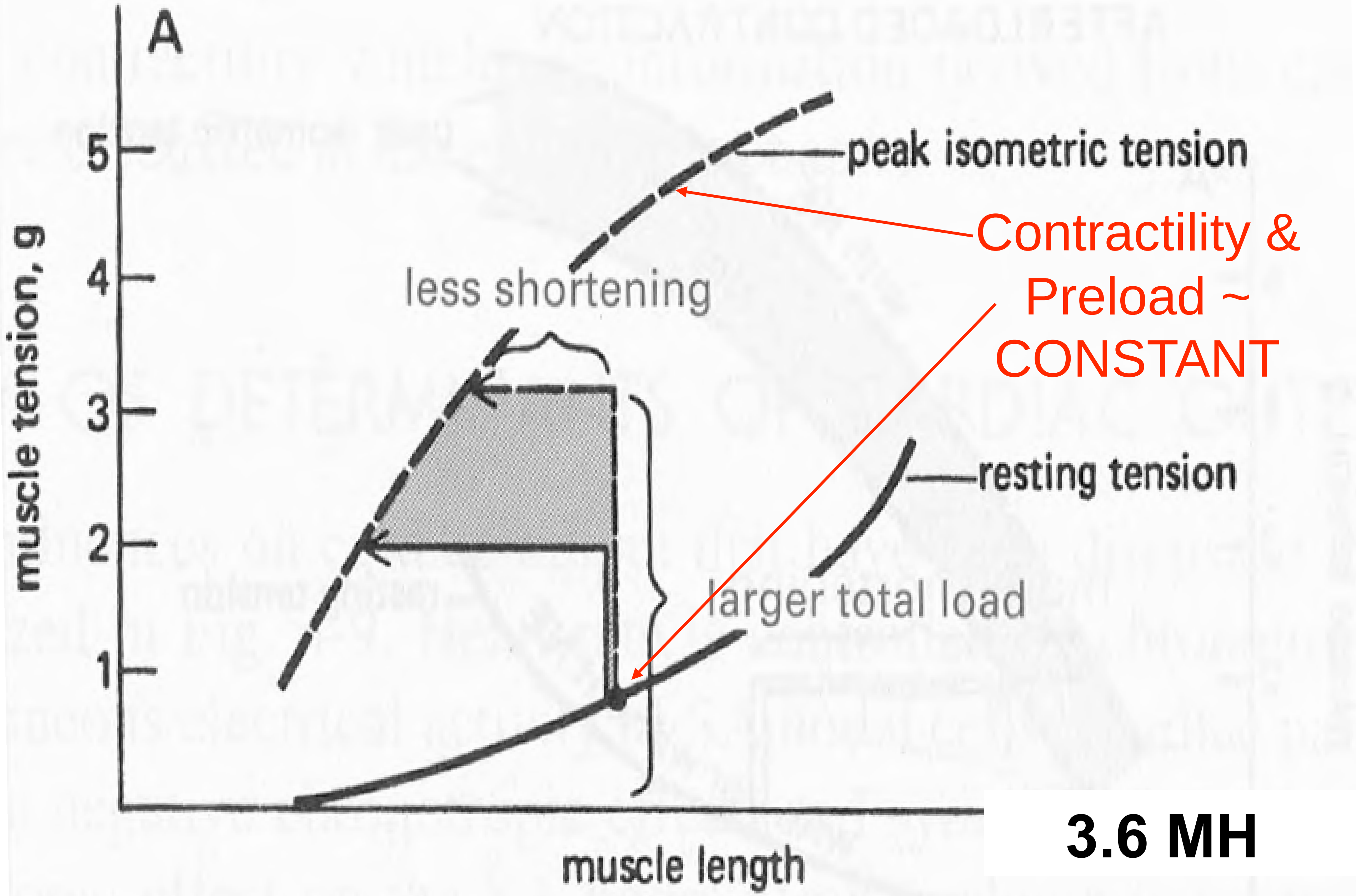
Increased Afterload Decreases SV



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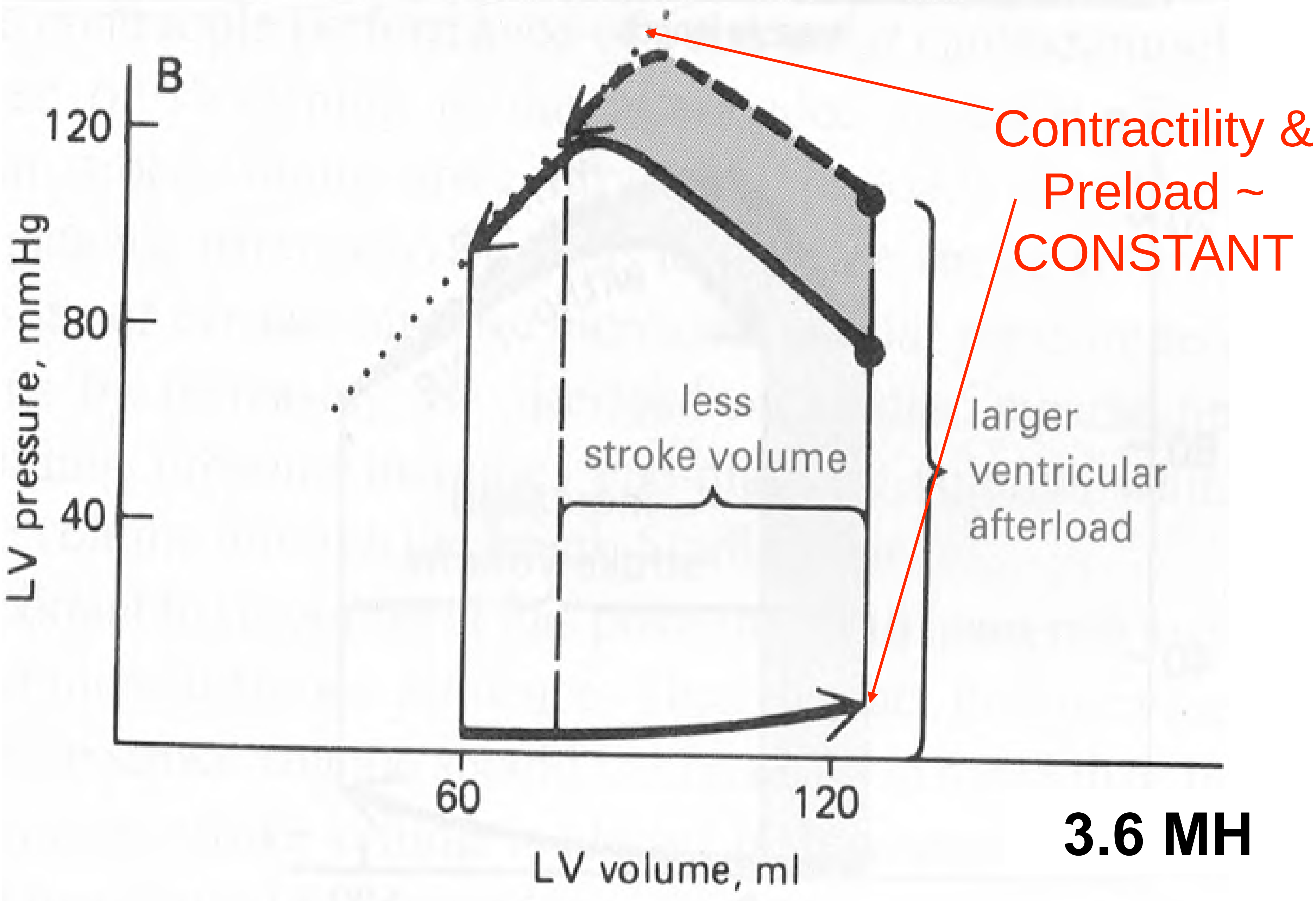
Increased Afterload Decreases SV



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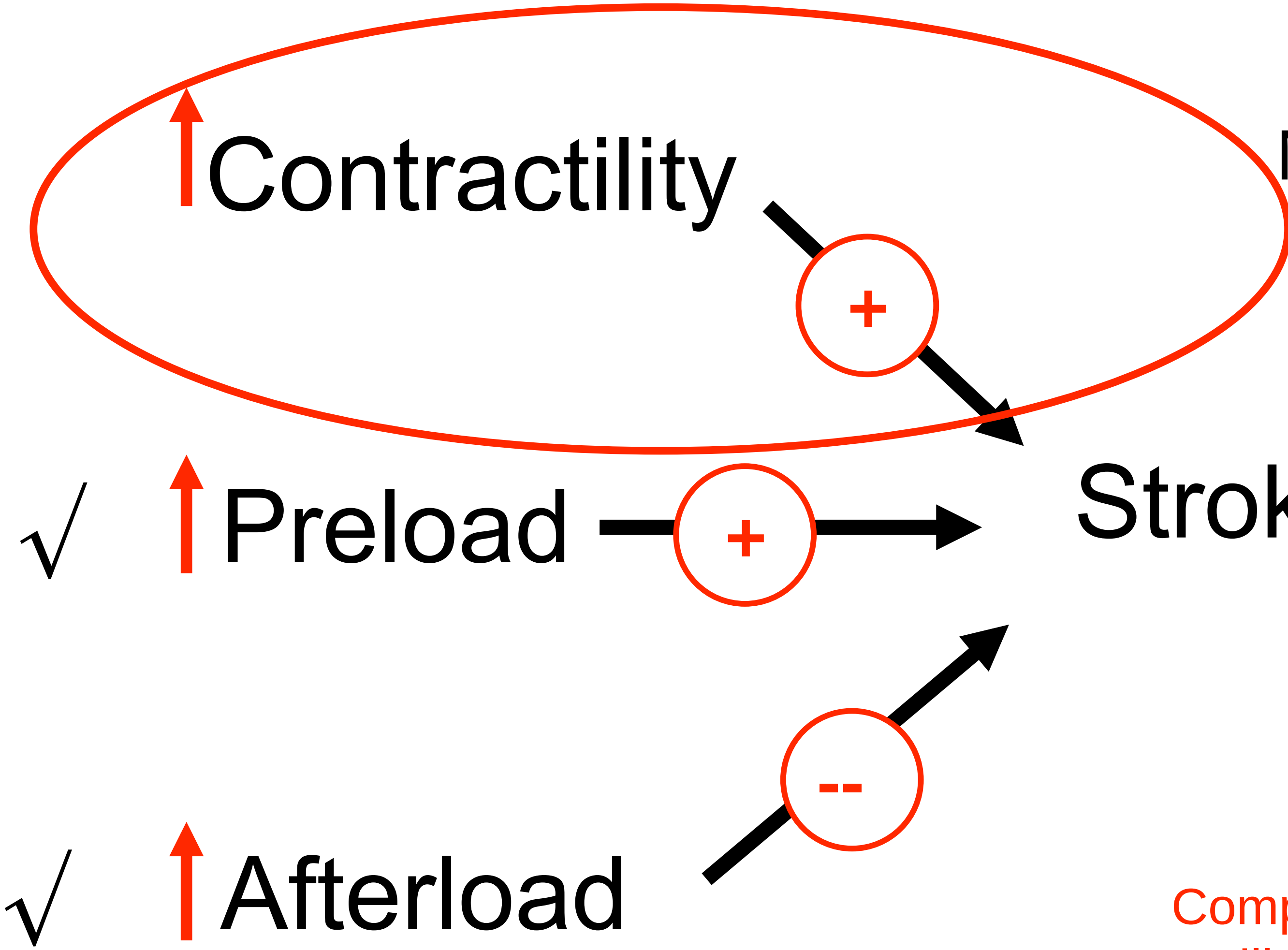

Next hour.

Complex interactions so
we will treat each separately
with others held constant.

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