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M2 Mini Review
Summer 2008
Chapter 9 Lilly

With flash backs to Mohrman and Heller

Pathophysiology of Heart Failure

Congestive HF = CO = Demand

= ↓ CO &/or ↑ Demand

Louis G. D' Alecy, Professor of Physiology

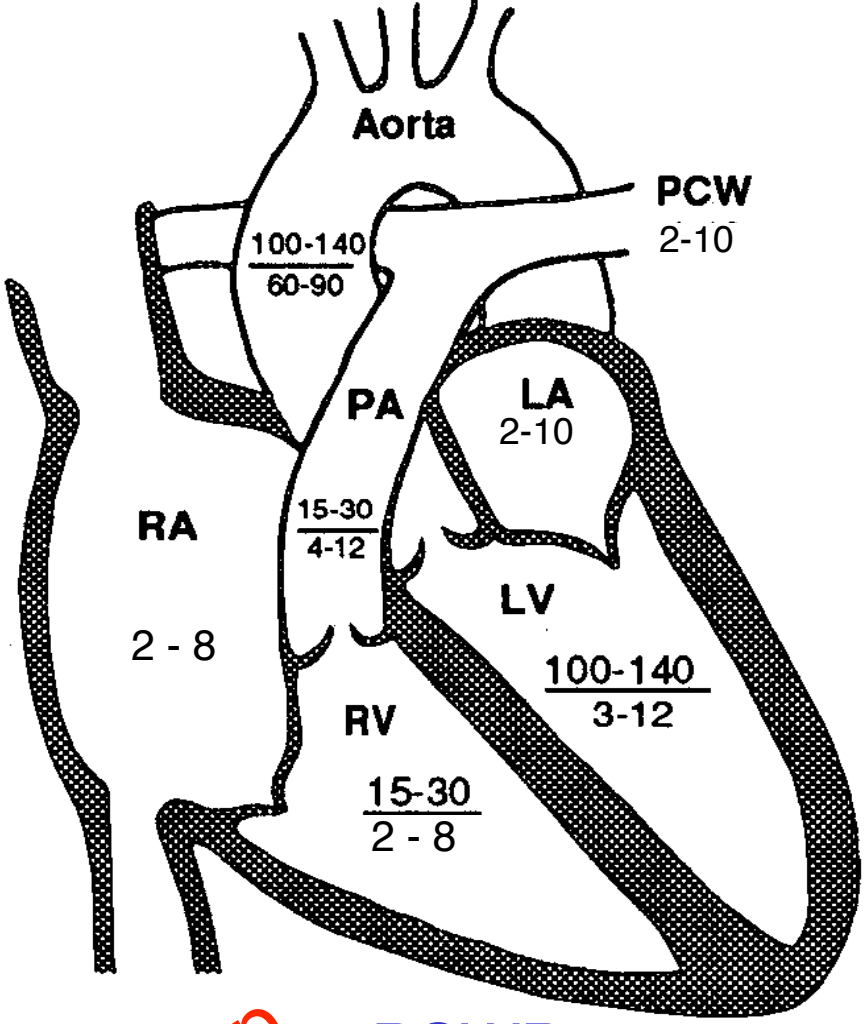
Heart Failure Outline

1) Normal Control of Stroke Volume
    a) Contractility
    b) Preload
    c) Afterload
2) Pathophysiology
    a) Systolic Dysfunction
    b) Diastolic Dysfunction
    c) Right-Sided Heart Failure
    d) Compensatory Mechanisms

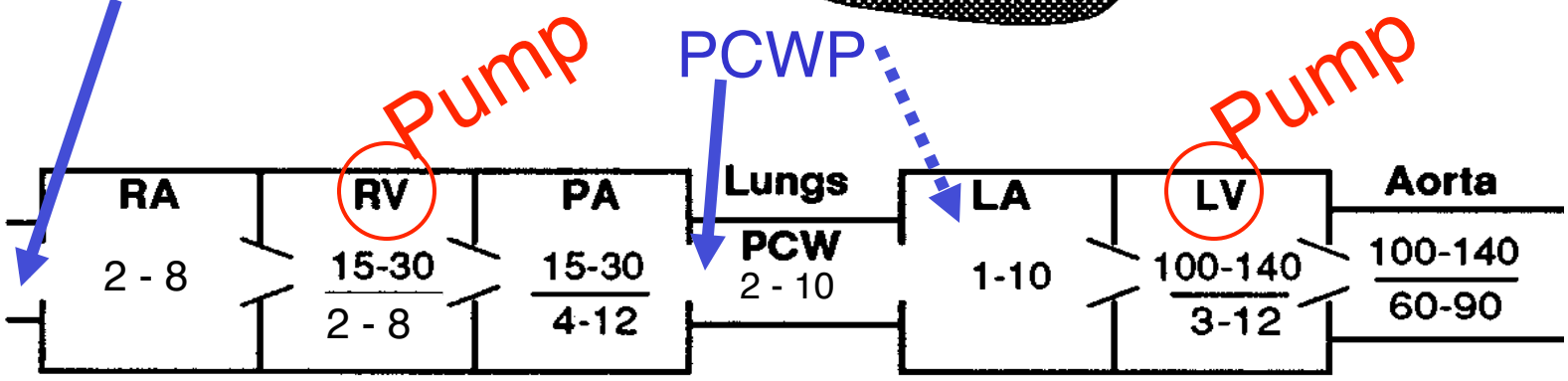
Requirements for Effective Cardiac Pumping

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"

Fig. 3.14
Lilly p 61



- 2 Pumps
- -in series
- -Interact
  - -Preload
  - -Afterload



Interaction RV & LV (e.g.)

If RV in failure inadequate blood gets to LV for adequate LV preload & LV output goes ↓.

If LV in failure inadequate blood gets removed from lungs and RV and excess afterload to RV ↓ output & ↑ pulmonary edema.

Lilly Table 9.1 Definitions

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

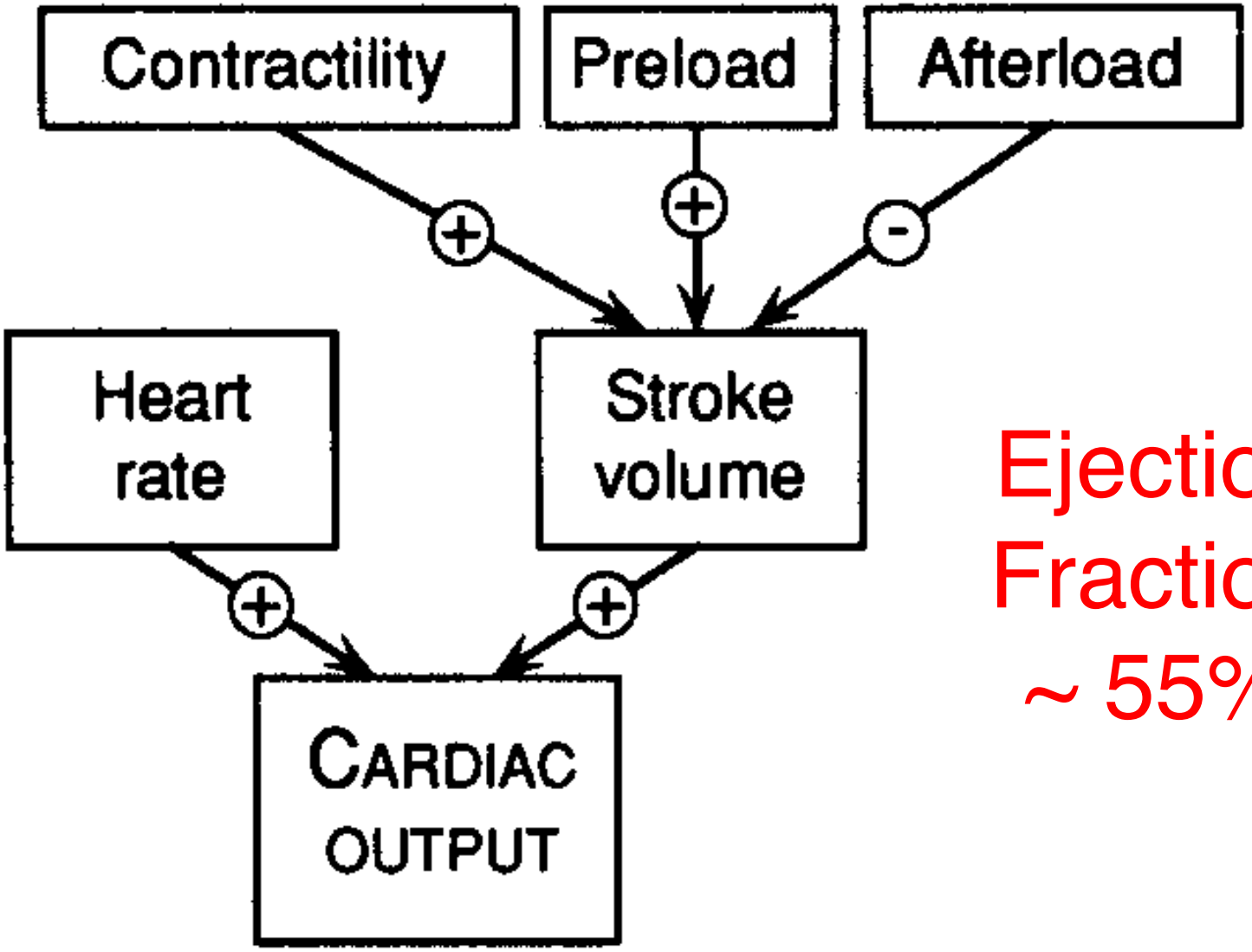
Afterload -- The ventricular wall tension during contraction; the resistance that must be overcome for the ventricle to eject its contents. Approximated by systolic ventricular or arterial pressure.

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

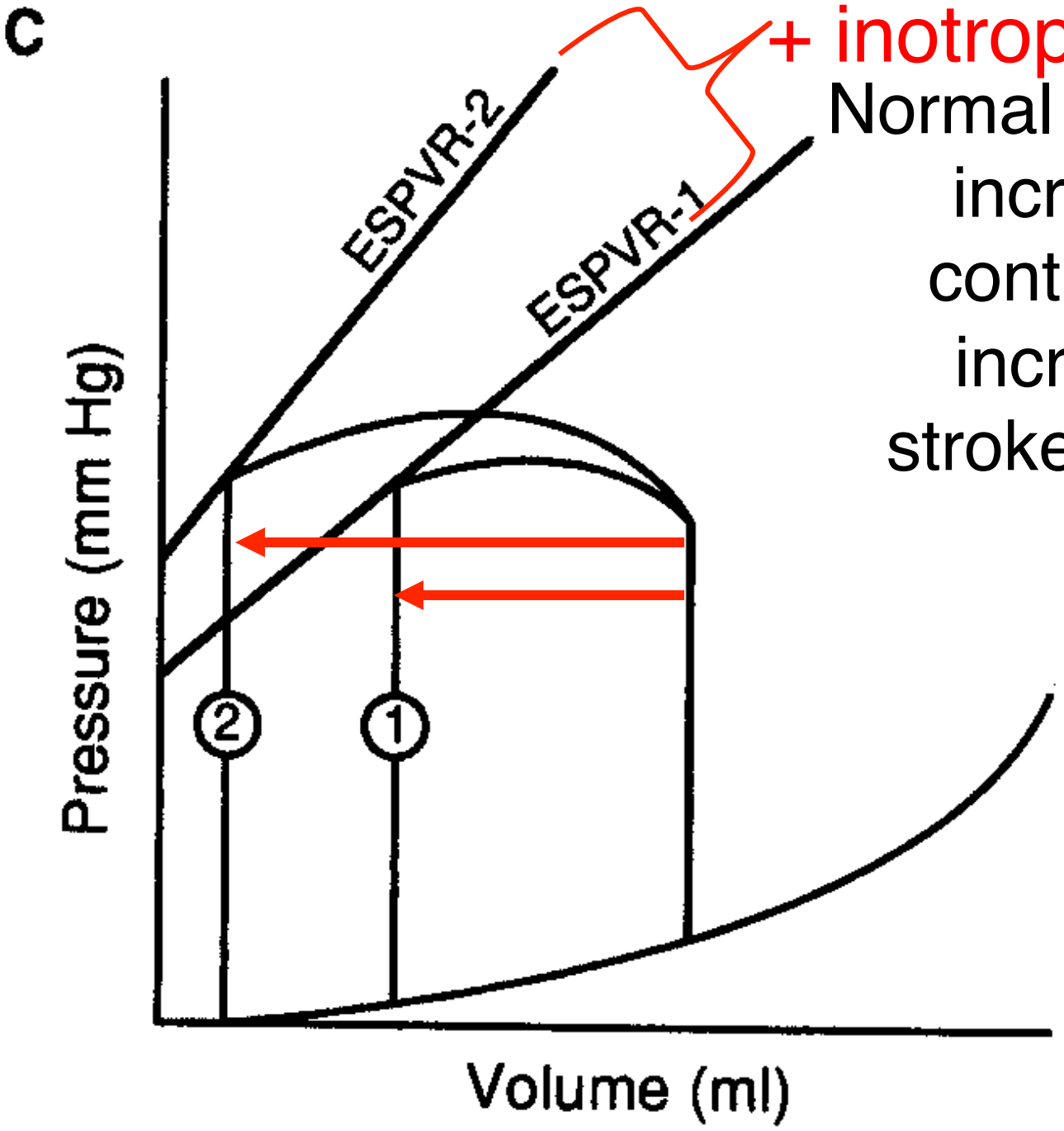
Inotropic state

End-diastolic pressure

Arterial pressure



Ejection Fraction ~ 55%



Normal response: increased contractility increases stroke volume

Fig 9.5

Decreased SV with Systolic Dysfunction

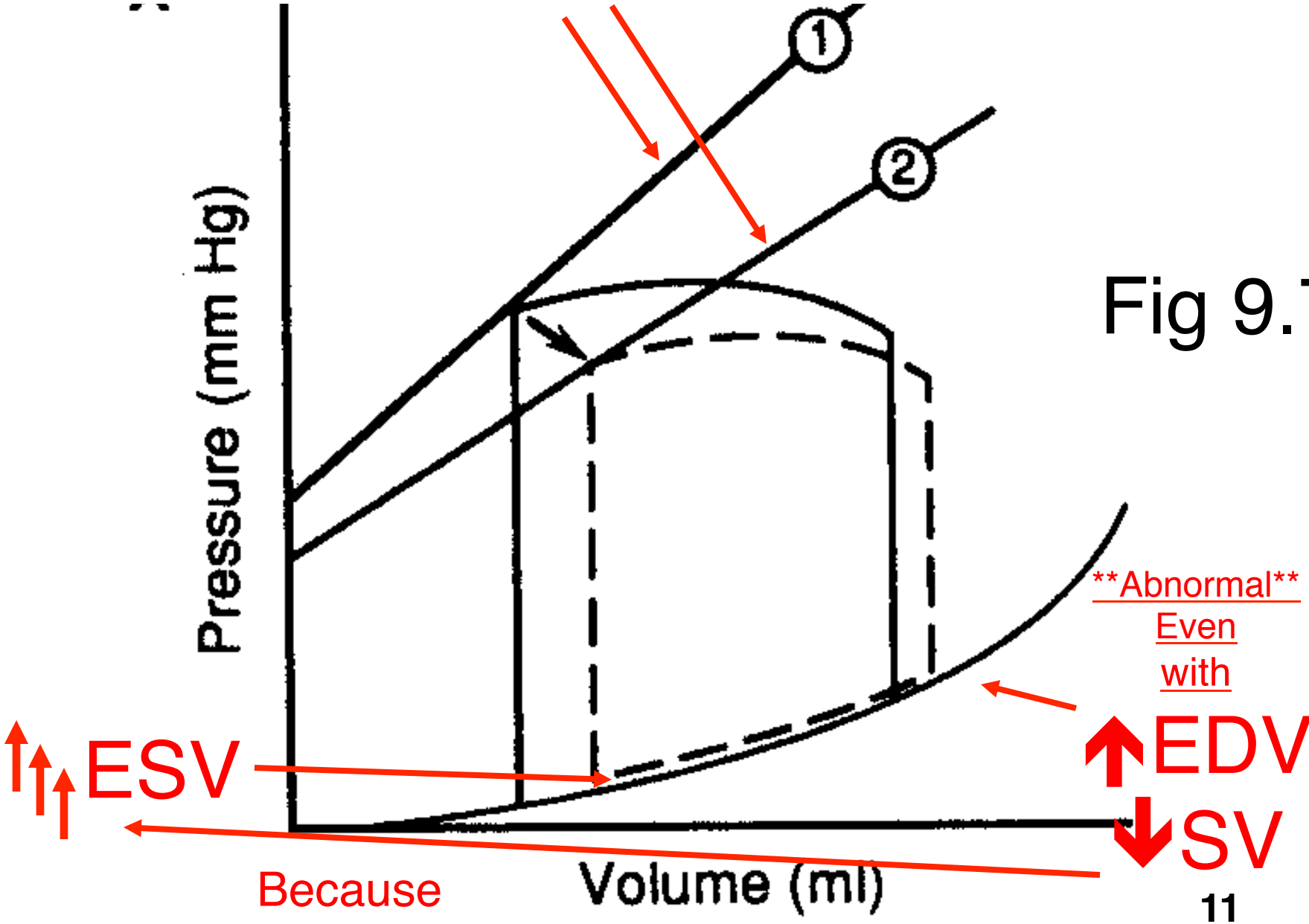


Fig 9.7

Normally:
Increased preload increases
stroke volume

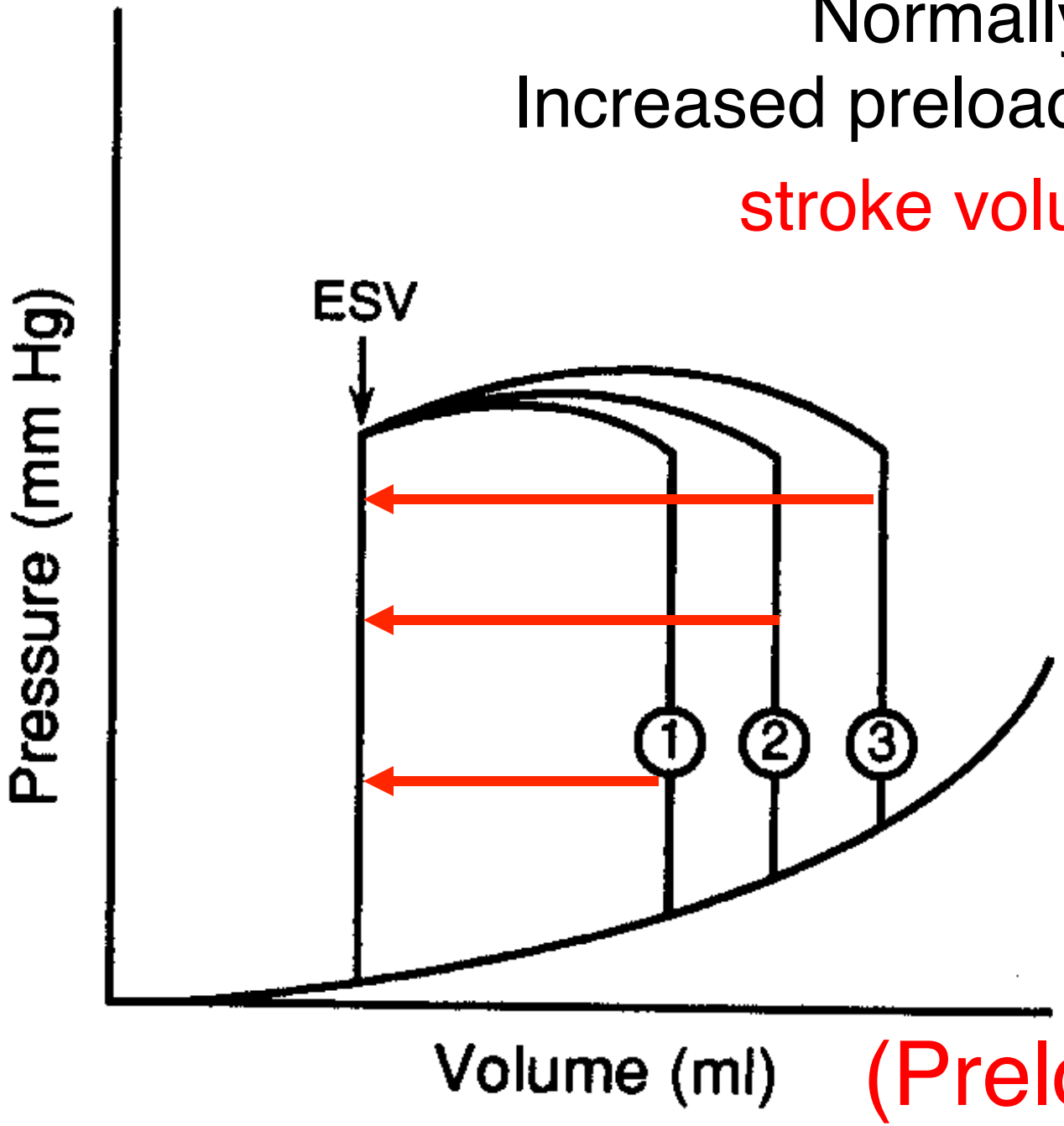
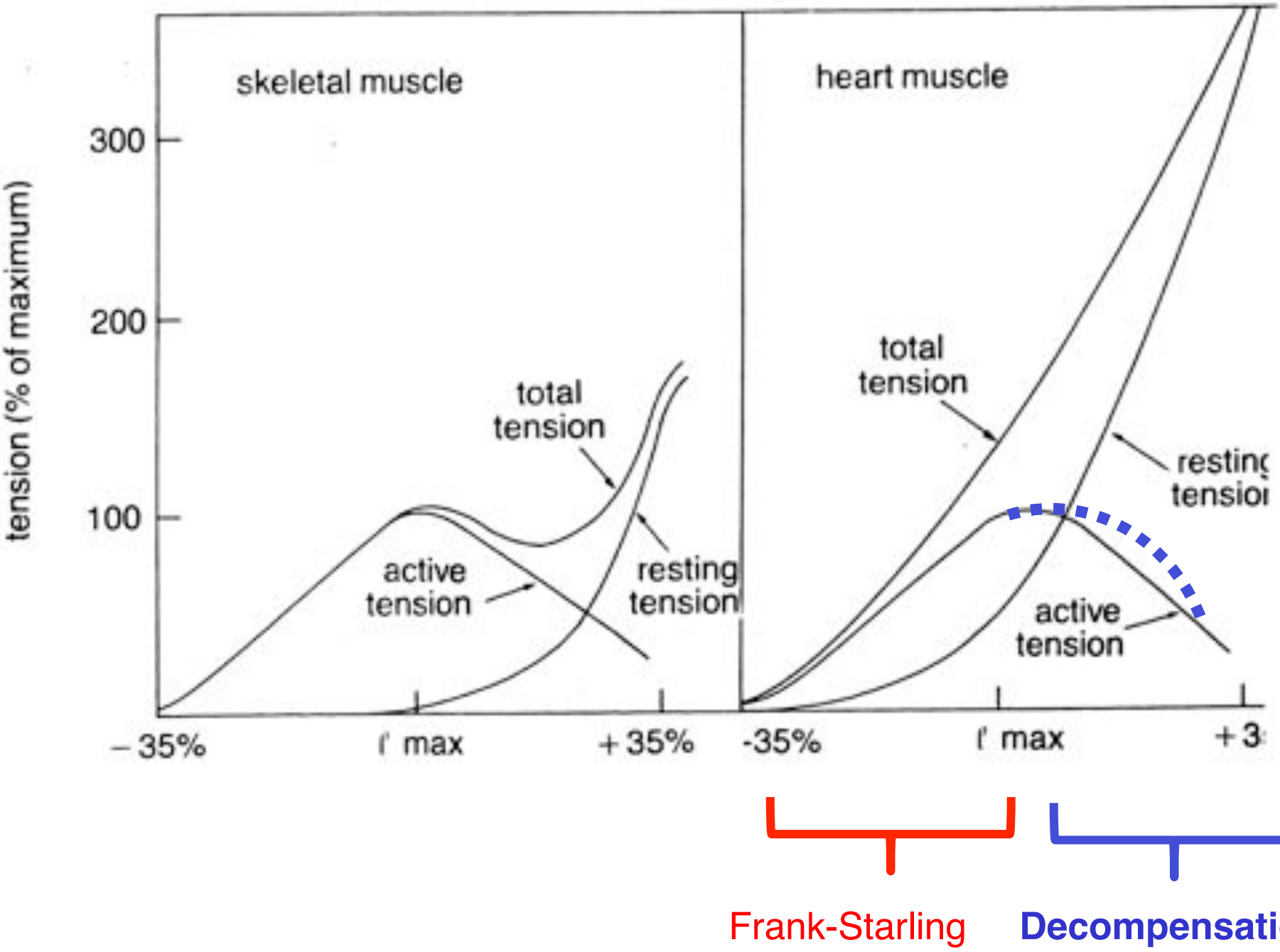


Fig 9.5

(Preload)



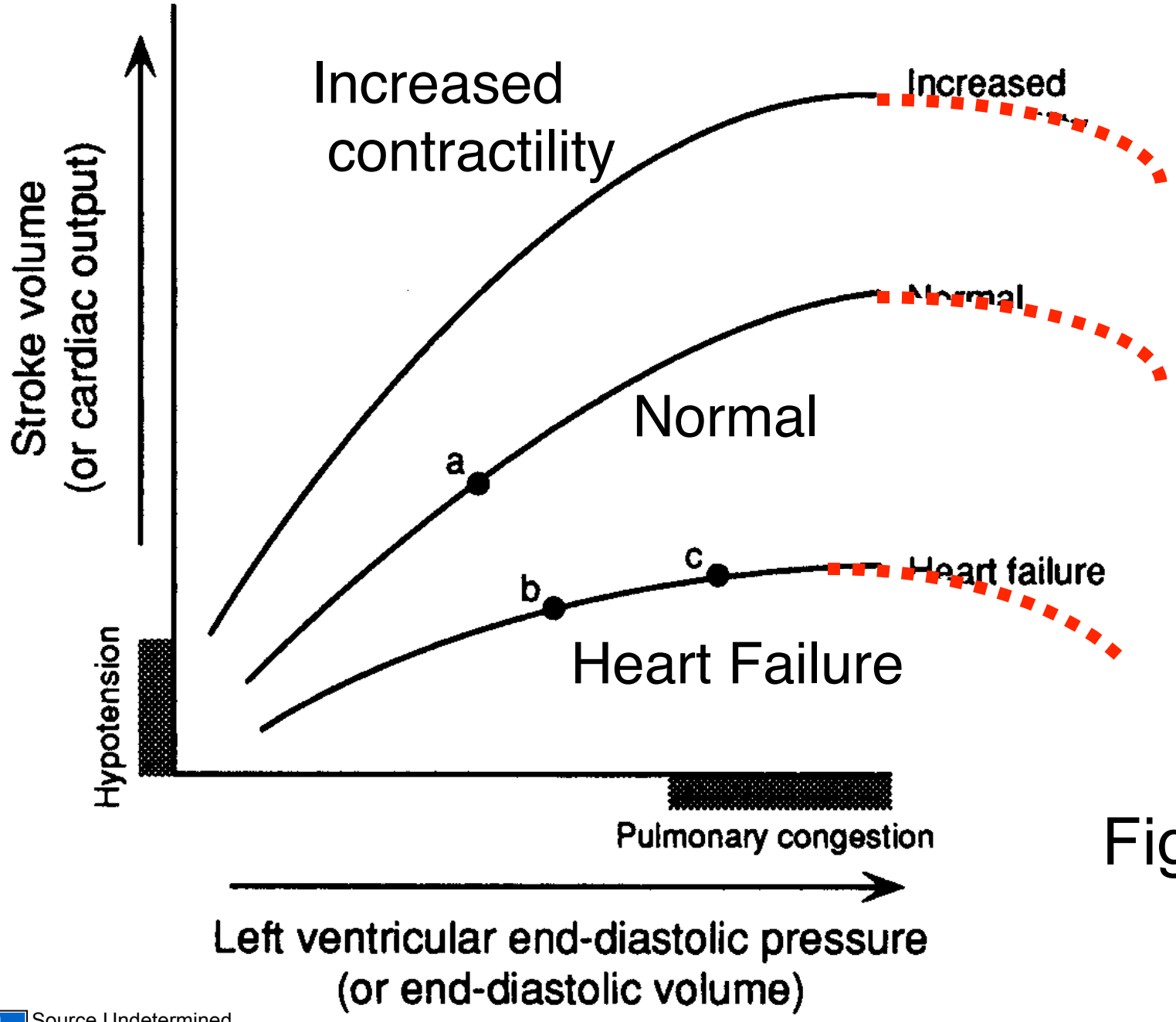
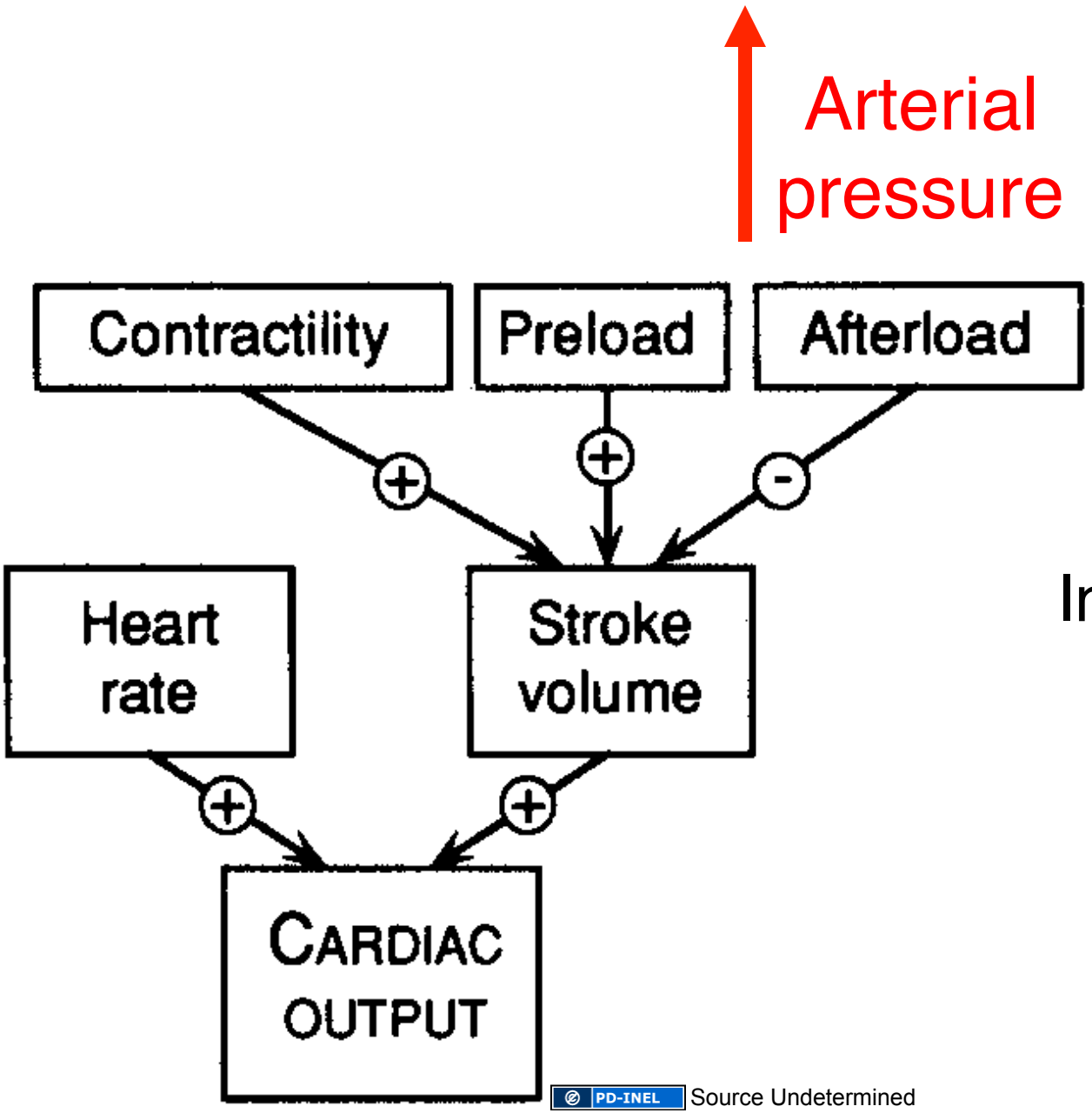


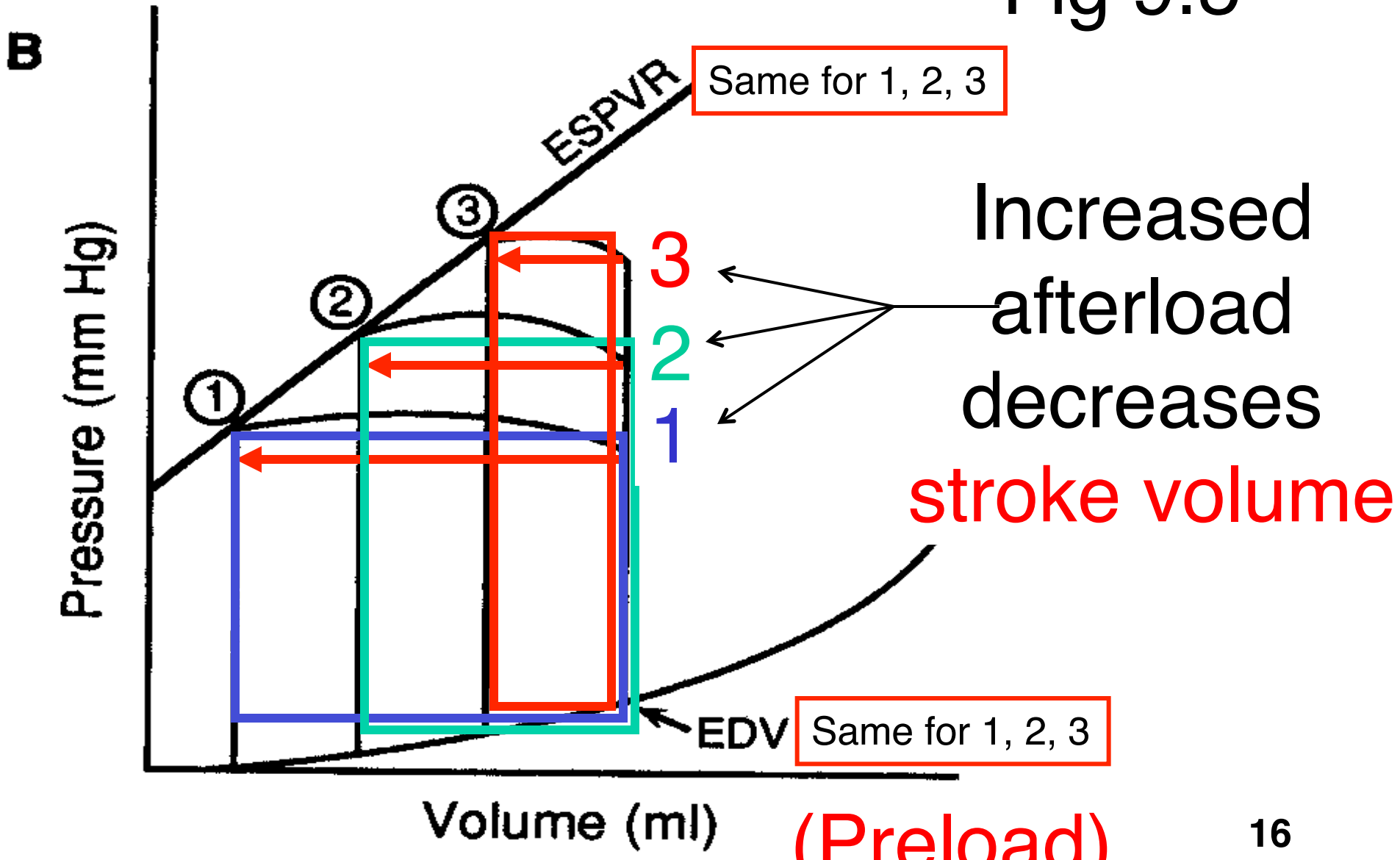
Fig 9.3

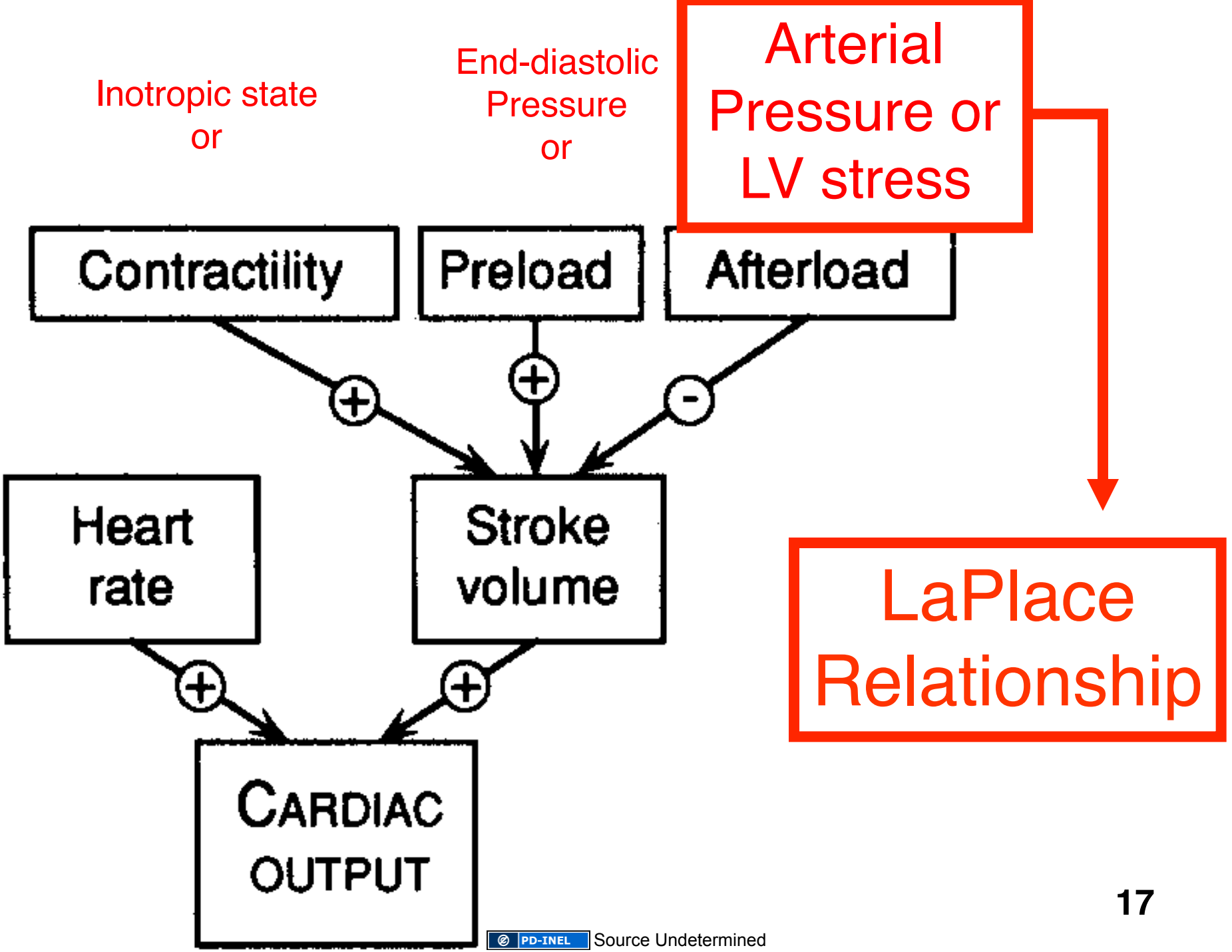


Increased afterload decreases

stroke volume

Fig 9.5





systolic ejection. Wall stress (σ), like pressure, is expressed as force per unit area, and for the left ventricle, may be estimated from the LaPlace relation for a hollow sphere:

stress $$\sigma = \frac{P \cdot r}{2h}$$

Pressure X radius
2 X thickness

in which P is ventricular pressure, r is ventricular chamber radius, and h is ventricular wall thickness. In general, a useful mea-

Hypertrophy:
not beat to beat.

Page 229 Lilly

From **Surgery: Scientific Principles and Practice**
Ed. By Greenfield, Mulholland, Oldham, Zelenock,and Lillemoe

Laplace Law

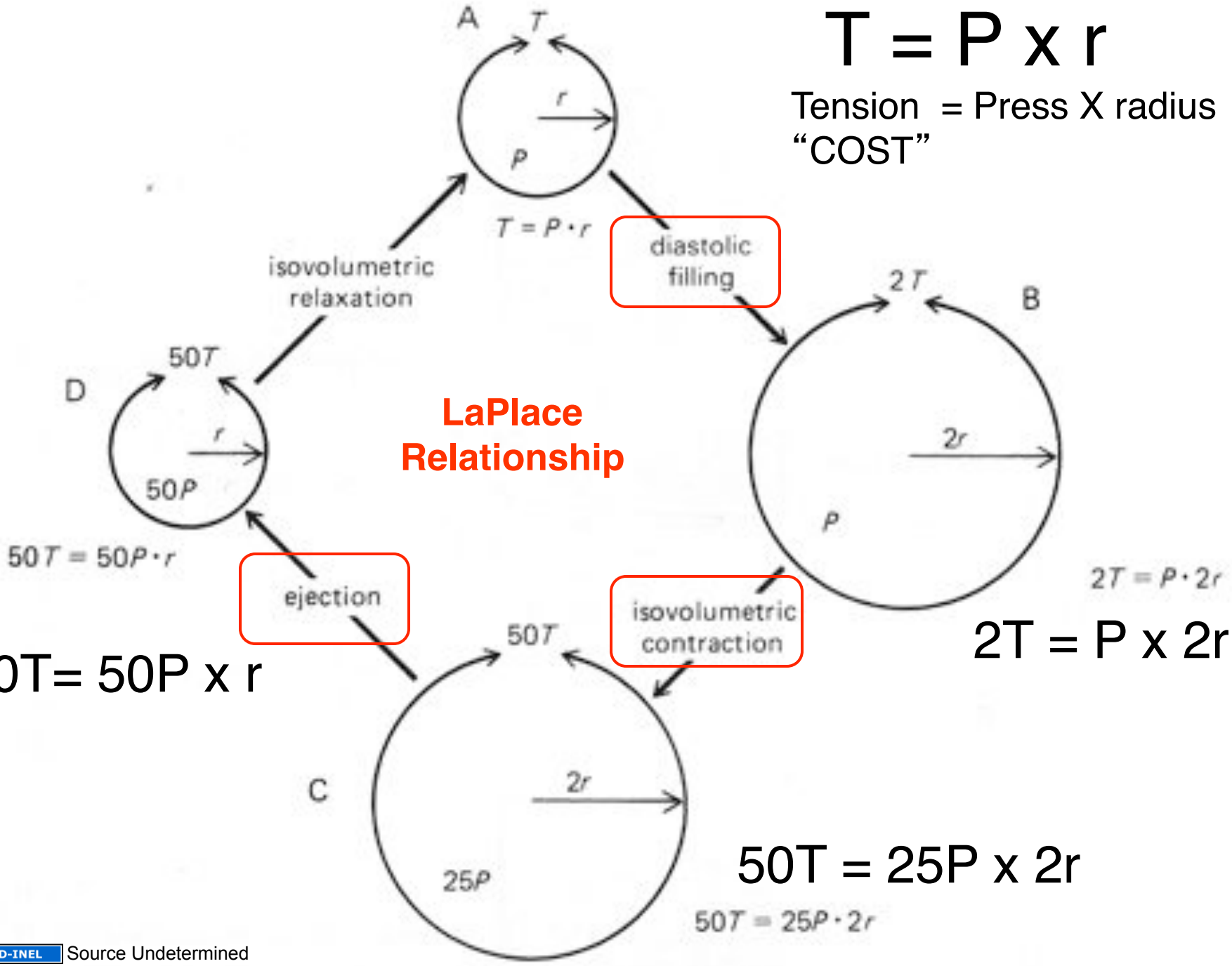
$$CWS = \frac{(Pb)}{h}\left(1 - \frac{b^2}{2a^2} - \frac{h}{2b} + \frac{h}{8a^2}\right)$$

where:

CWS = circumferential wall stress in dynes/cm^2 $\times$ 10^3;
P = left ventricular pressure in dynes/cm^2; a and b
= major and minor semiaxes, respectively, in cm;
h = left ventricular wall thickness in cm

T = P x r

Tension = Press X radius
"COST"



LaPlace Relationship

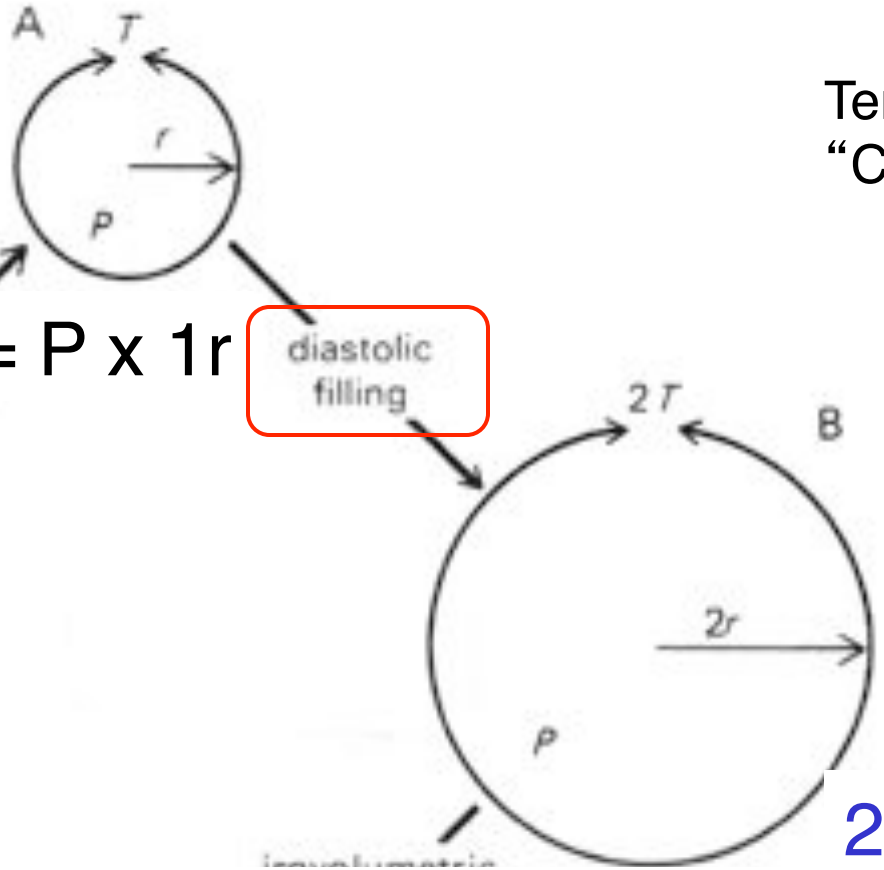
50T= 50P x r

2T = P x 2r

50T = 25P x 2r

T = P x r

Tension = Press X radius
"COST"



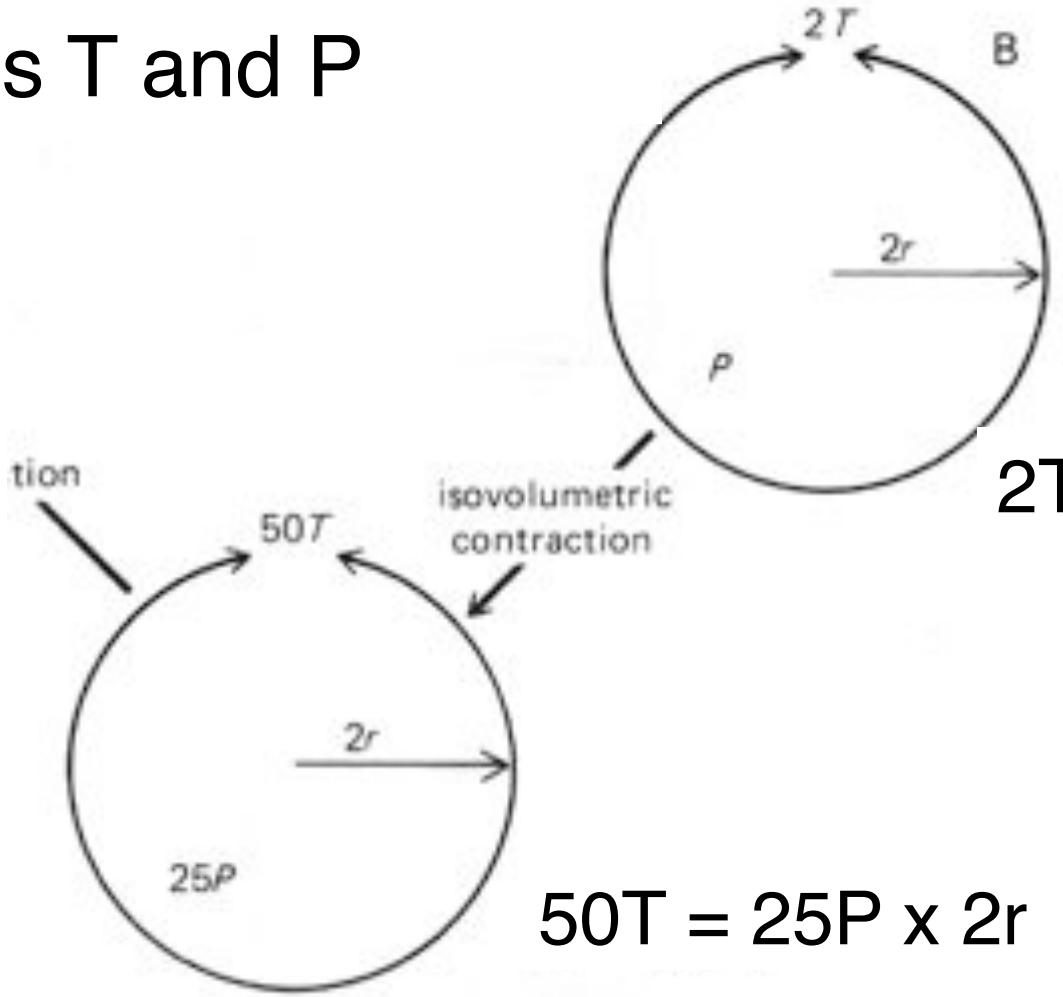
PCWP or LVEDP

As ventricle fills during diastole
the volume increases, tension doubles,
with little increase in pressure

T = P x r

Tension = Press X radius
"COST"

Isovolumetric contraction
Increases T and P



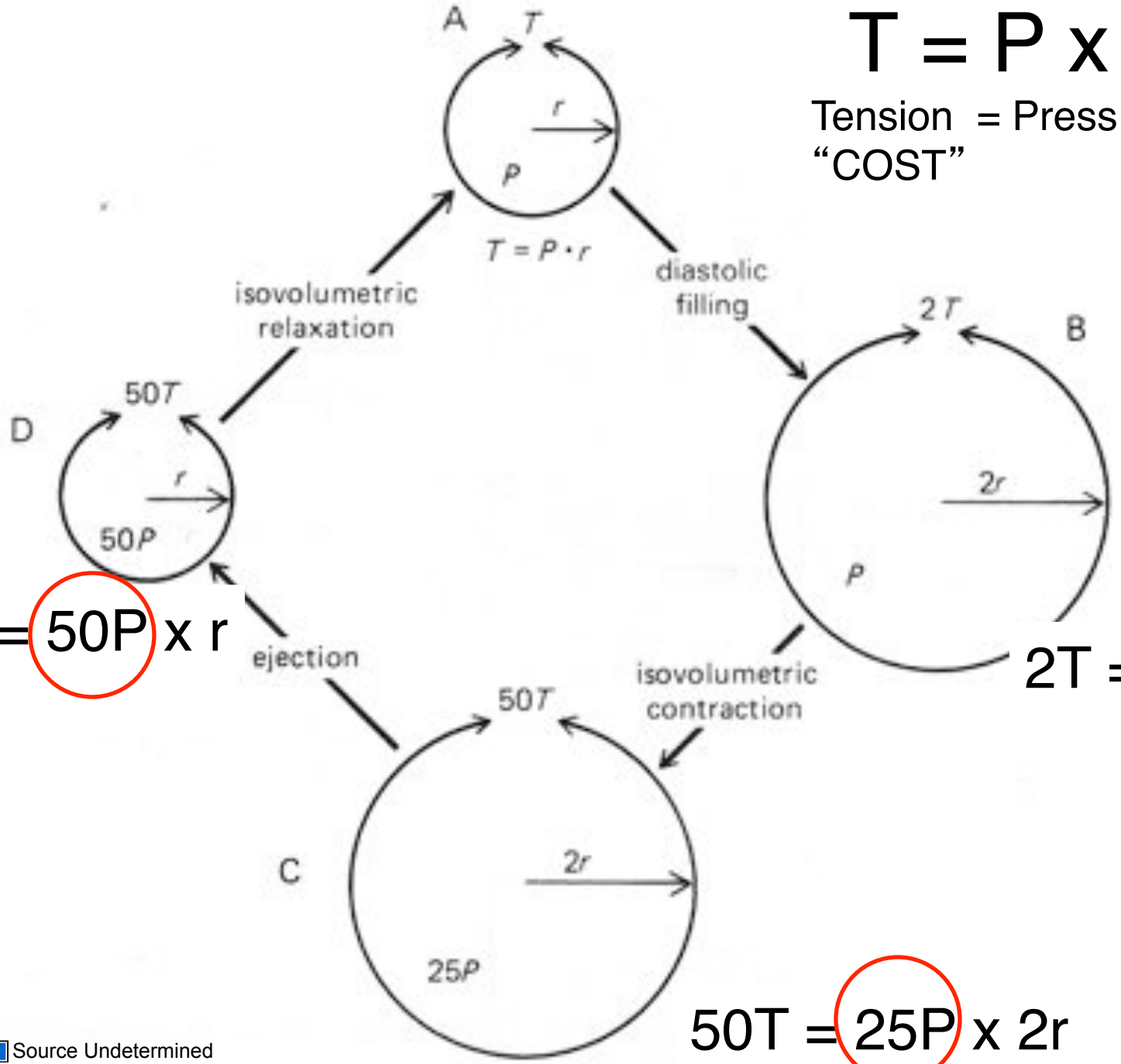
2T = P x 2r

"iso"

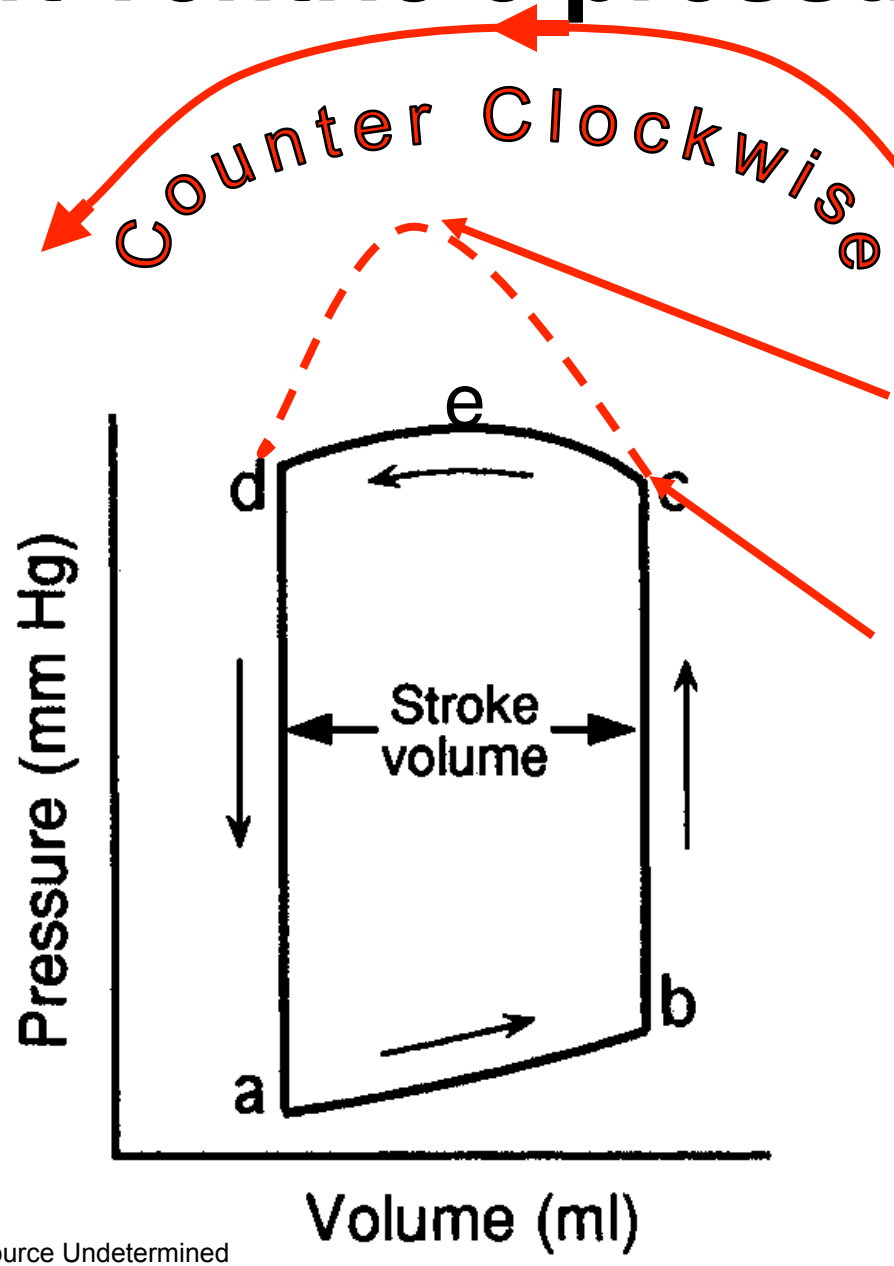
50T = 25P x 2r

$T = P \times r$

Tension = Press X radius
"COST"



Left ventricle pressure-volume loop



50P (Systolic)
LaPlace
25P (Diastolic)

Fig. 9.4

Pathophysiology of HF

CO fails to meet demand because:

1) SYSTOLIC DYSFUNCTION
   a) Impaired ventricular contractile function
   b) Increased afterload
2) DIASTOLIC DYSFUNCTION
   a) Impaired ventricular filling
3) COMPENSATORY MECHANISMS
   a) Frank-Starling
   b) Hypertrophy
   c) Neurohumoral

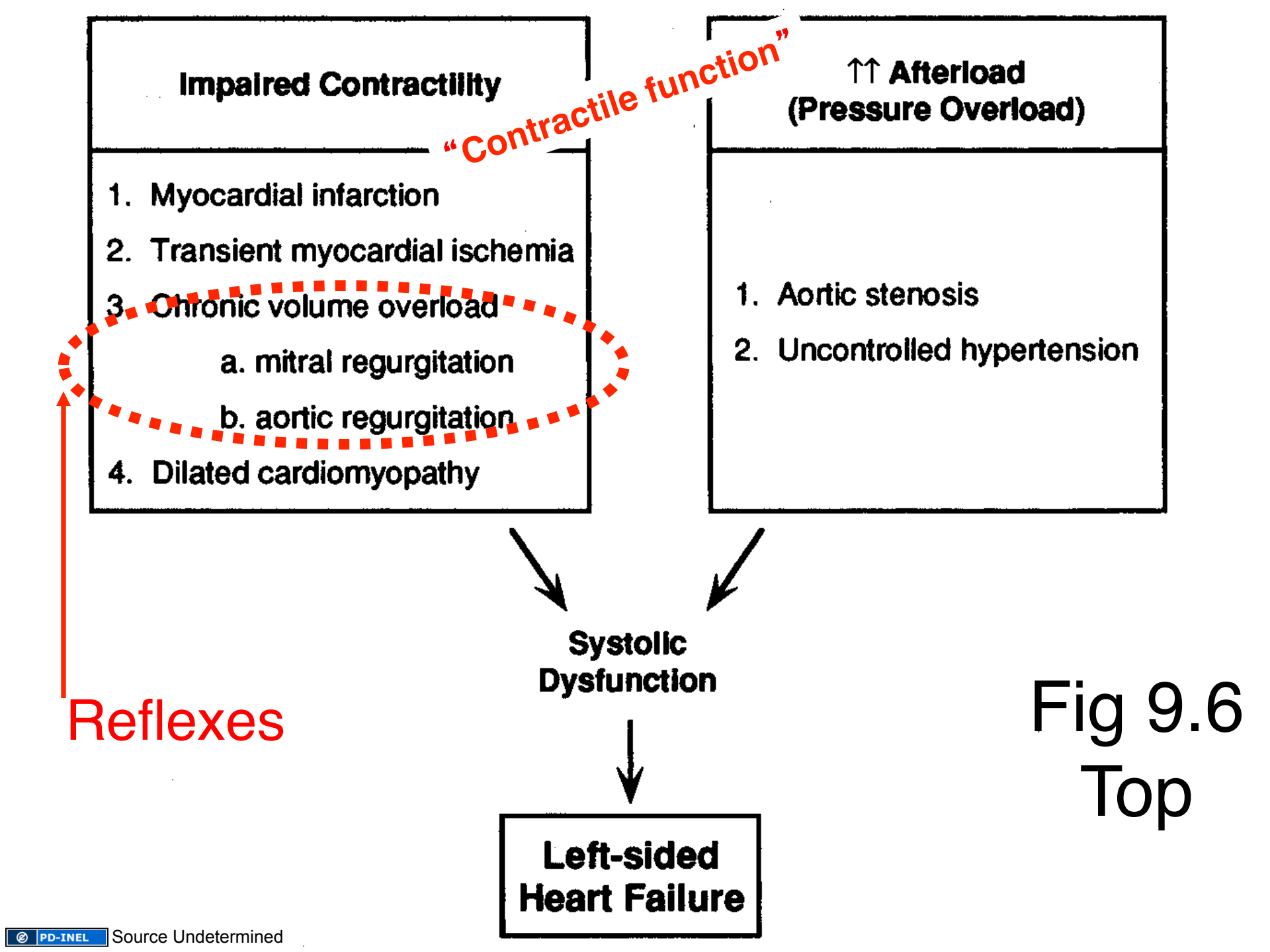


Fig 9.6
Top

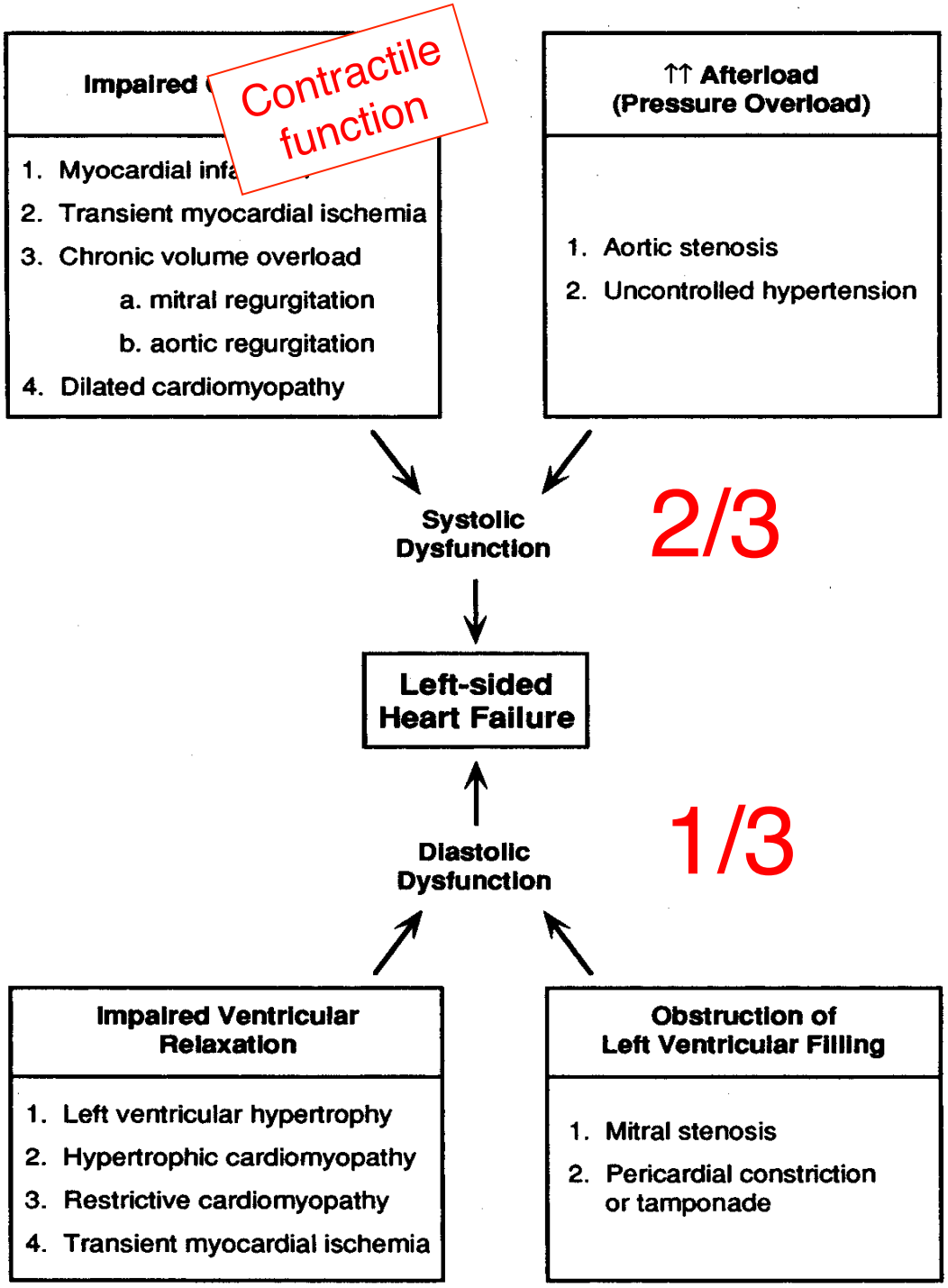
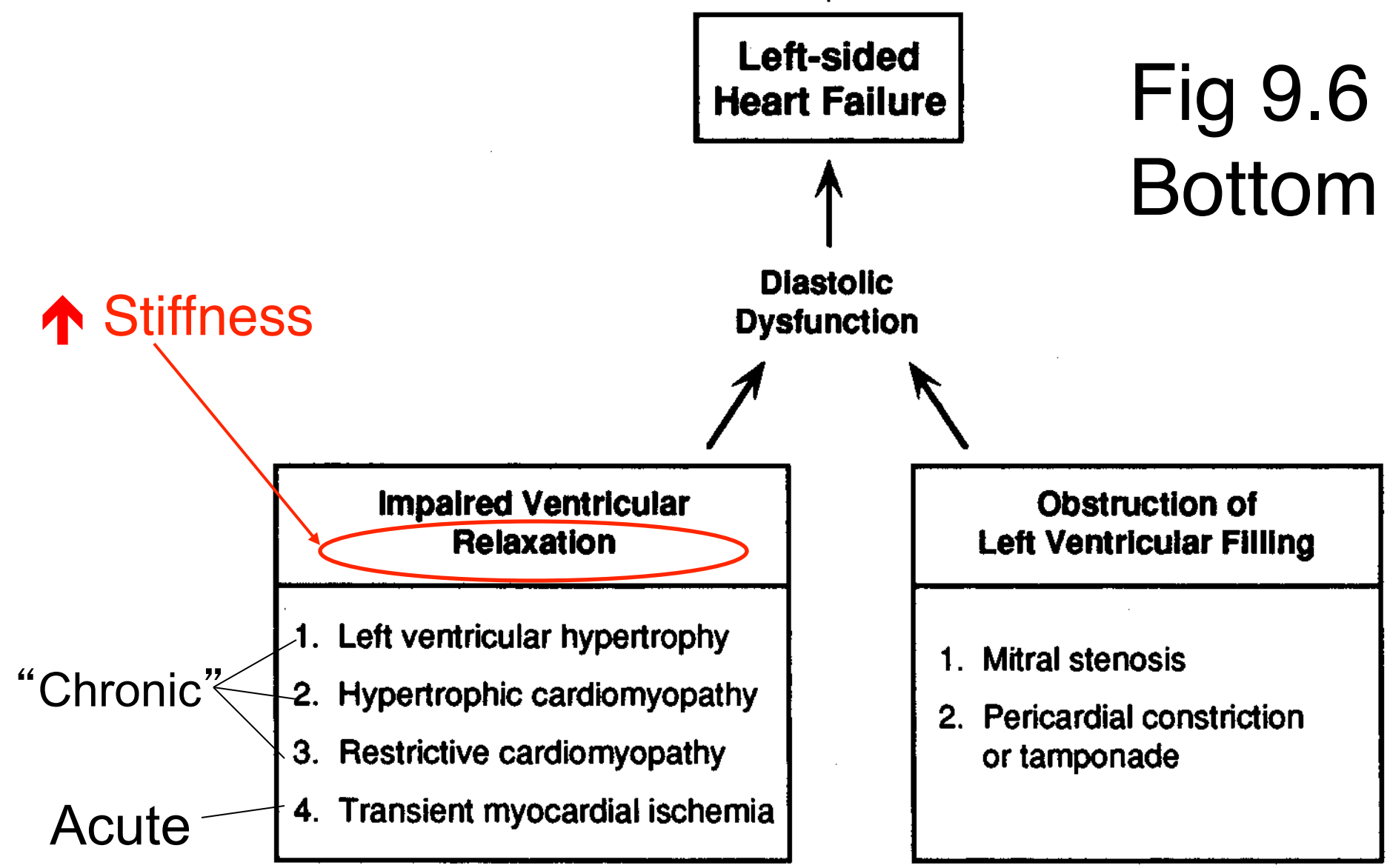


Fig. 9.6

Fig 9.6 Bottom



Decreased SV with Diastolic Dysfunction

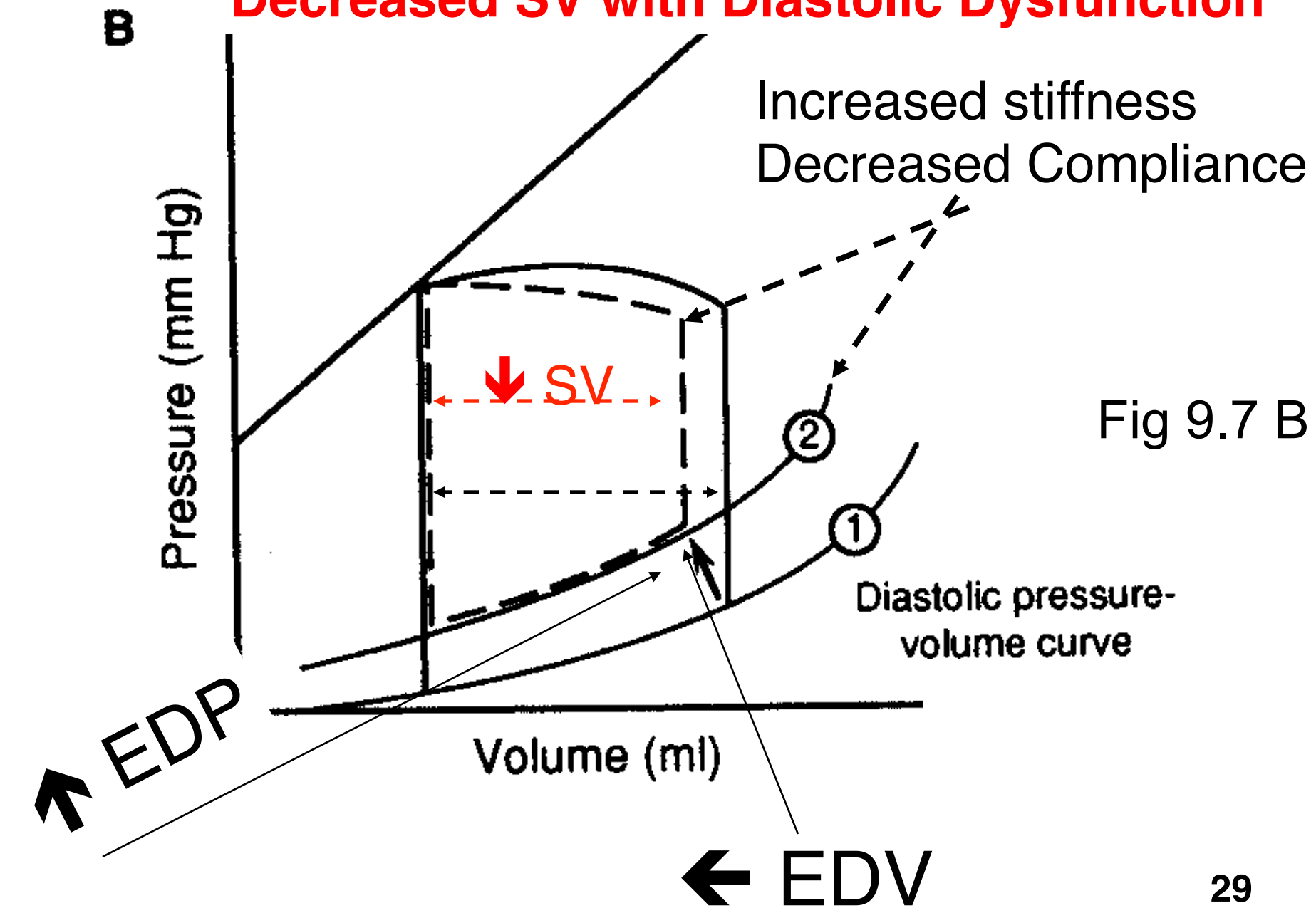
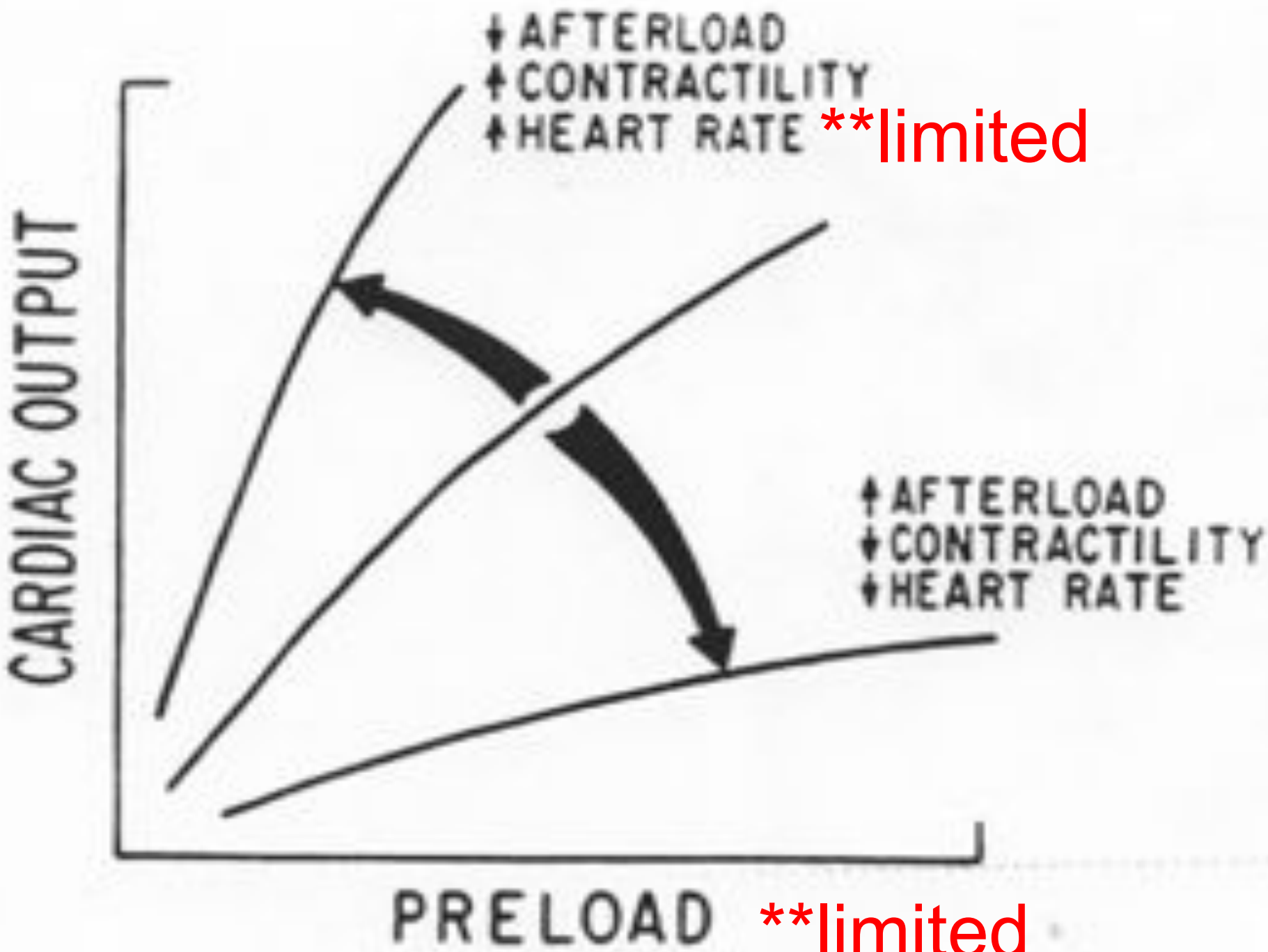


Fig 9.7 B

Right-Sided Heart Failure

TABLE 9.2. Examples of Conditions That Cause Right-Sided Heart Failure

Cardiac causes
Left-sided heart failure
Pulmonic valve stenosis
Right ventricular infarction
Parenchymal pulmonary disease
Chronic obstructive pulmonary disease
Interstitial lung disease (e.g., sarcoidosis)
Adult respiratory distress syndrome
Chronic lung infection or bronchiectasis
Pulmonary vascular disease
Pulmonary embolism
Primary pulmonary hypertension



Heart Failure

COMPENSATORY MECHANISMS

or failing compensatory mechanisms!

Frank-Starling
Hypertrophy
Neurohumoral

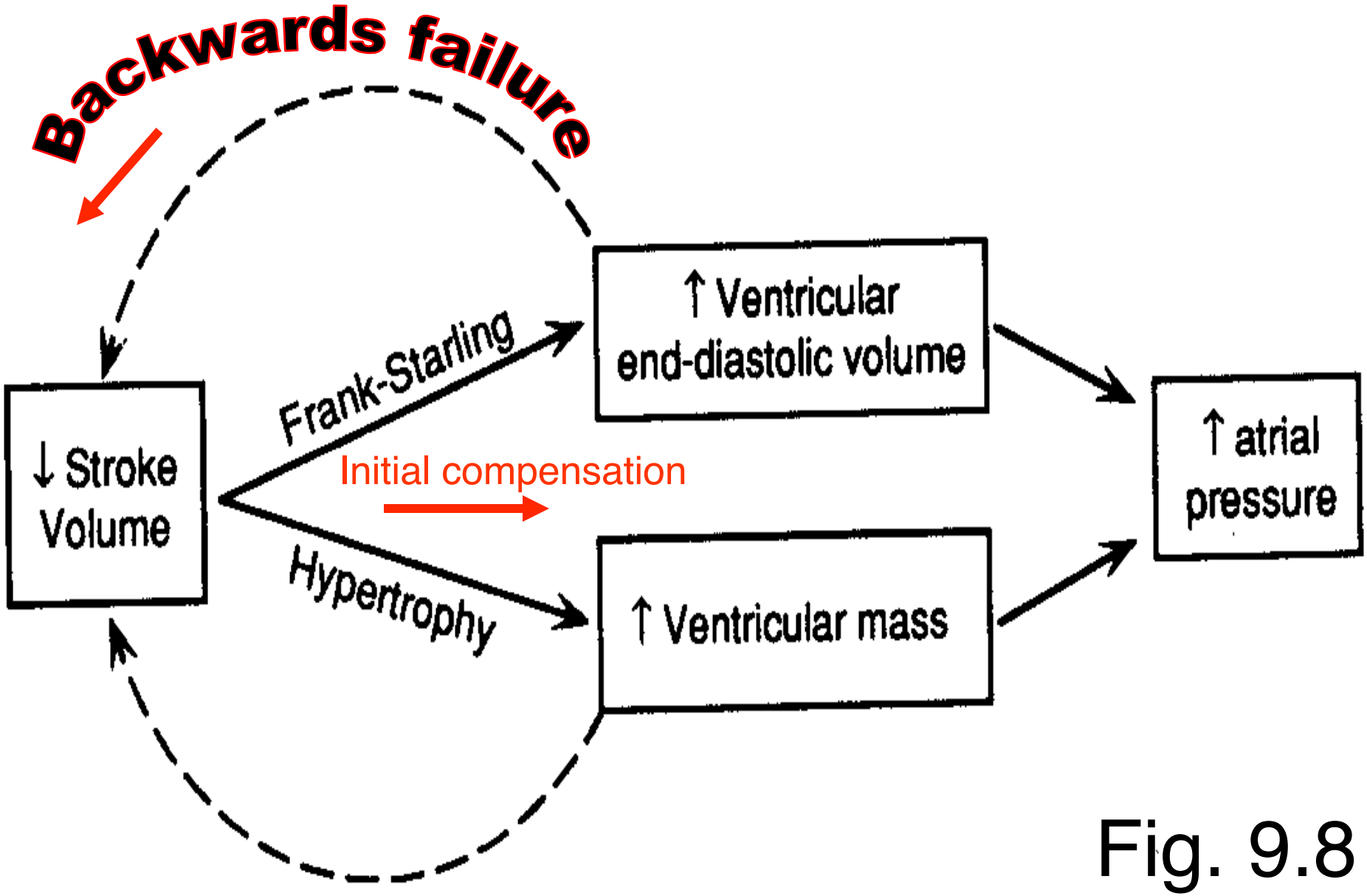


Fig. 9.8

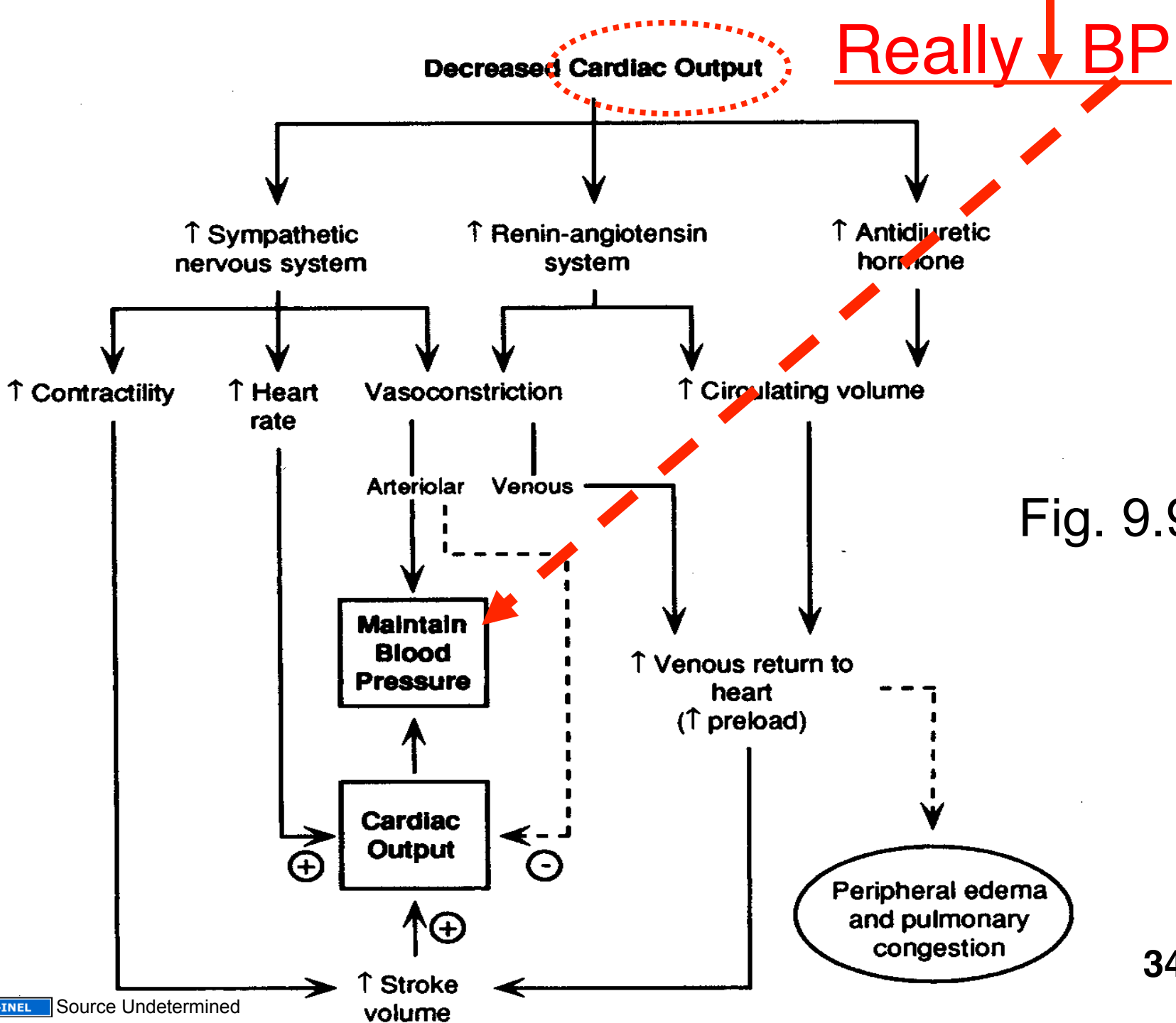
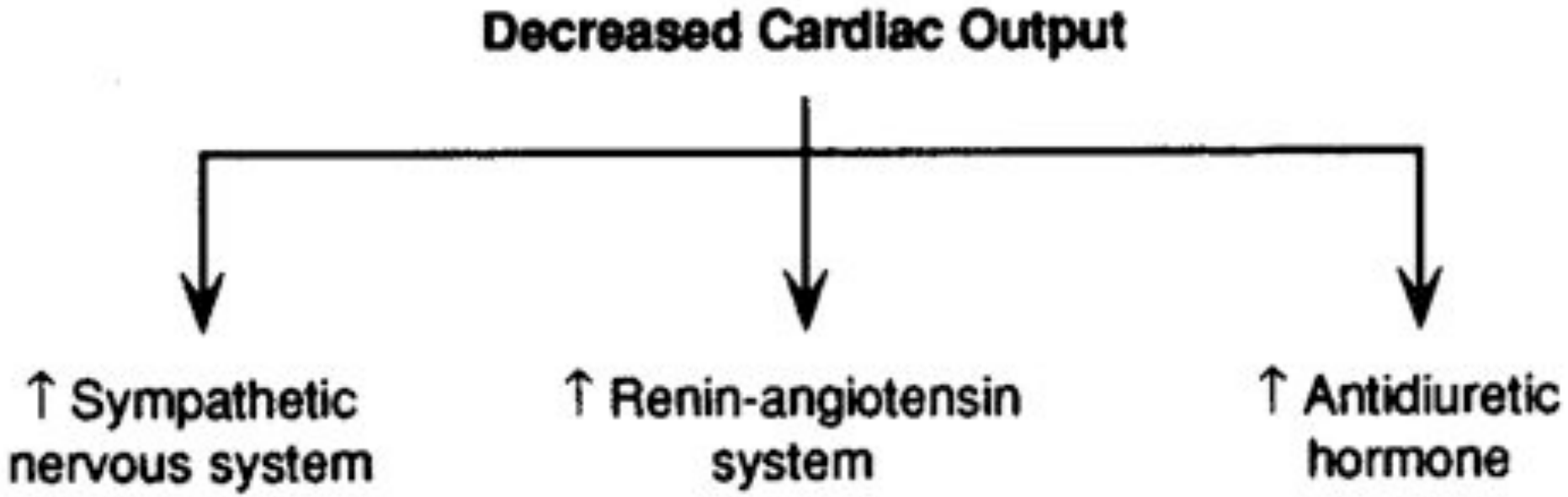
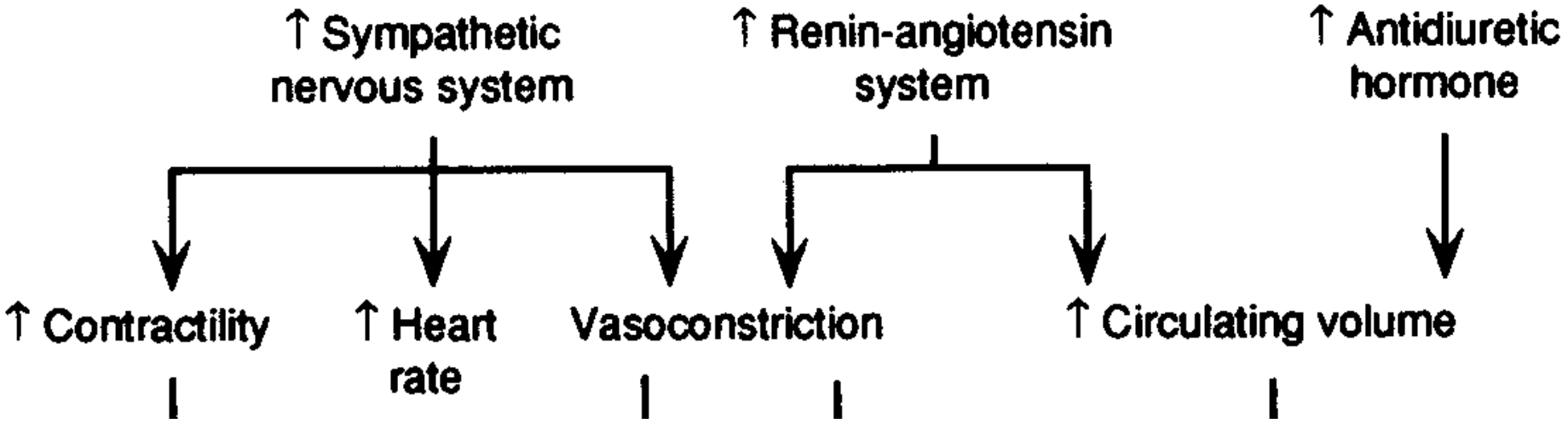


Fig. 9.9



Decreased CO ..decreased MAP…Baroreceptor Reflex !!!

Baroreceptor Reflex



+ Ino + Chron VC + Veno C Fluid retention

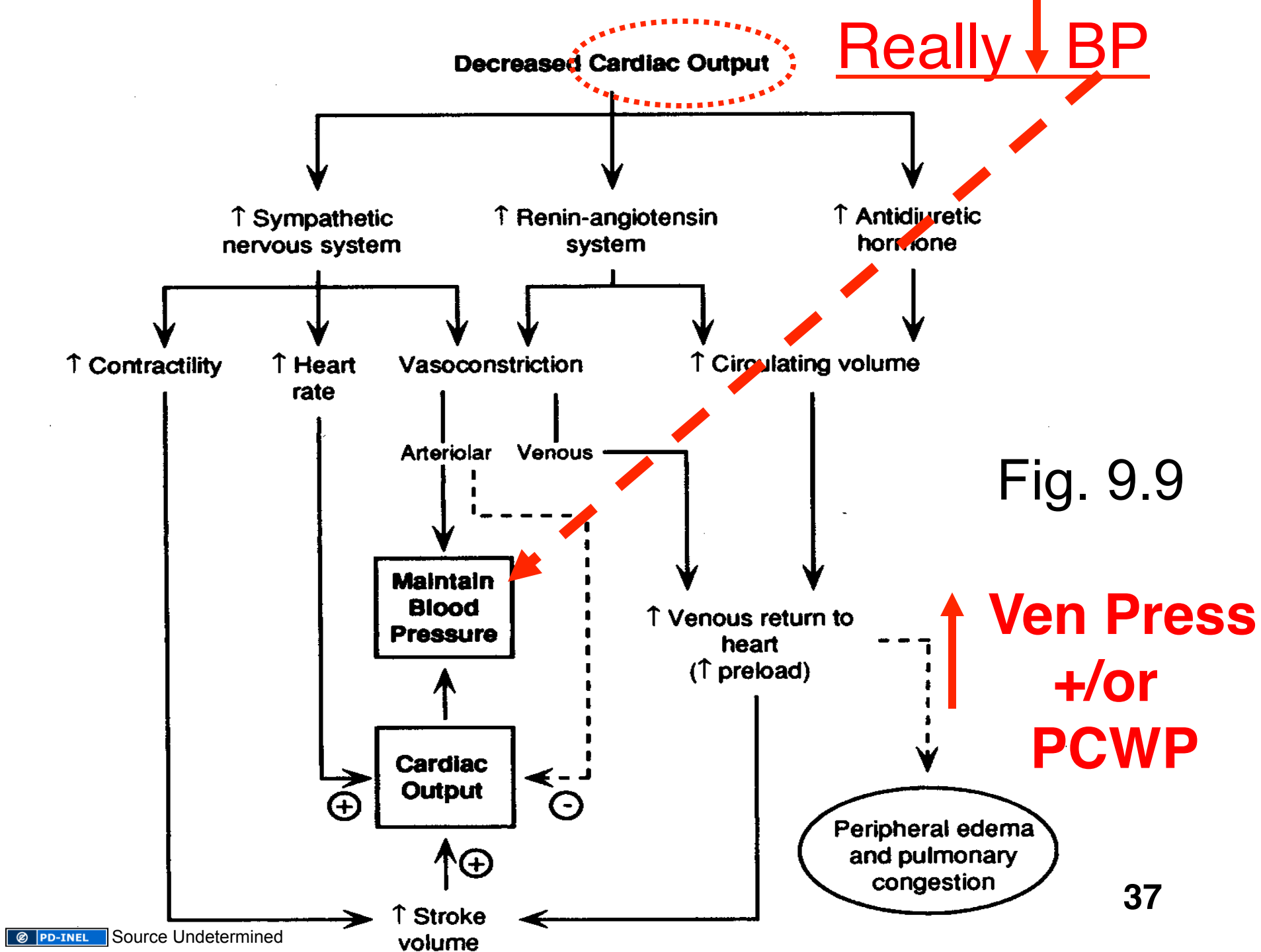


Fig. 9.9

TABLE 9-4 COMPARISON OF CARDIOVASCULAR FUNCTION IN A NORMAL PERSON AND A PATIENT WITH MODERATE-TO-SEVERE CONGESTIVE HEART FAILURE (CHF) AT REST AND AT MAXIMAL (MAX) EXERCISE

	CO (LITERS/MIN)	HR (BEATS/MIN)	SV (ML)	MAP (MM HG)	VO_2 (ML O_2/MIN)	A-VO_2 (ML O_2/100 ML)
Normal (Rest)	5.6	70	80	95	220	4.0
Normal (Max)	18.0	170	106	120	2500	13.9
CHF (Rest)	4.0	80	50	90	220	5.5
CHF (Max)	6.0	120	50	85	780	13.0

CO, cardiac output; HR, heart rate; SV, stroke volume; MAP, mean arterial pressure; VO_2, whole-body oxygen consumption; A-VO_2, arterial-venous oxygen difference. VO_2 is calculated from the product of CO and A-VO_2, after the units for CO are converted to mL/min and the units for A-VO_2 are converted to mL O_2/mL blood.

TABLE 9.3. Factors that may Precipitate Symptoms in Compensated Heart Failure

Increased metabolic demands

- Fever
- Infection
- Anemia
- Tachycardia
- Hyperthyroidism
- Pregnancy

Things that require an increase in cardiac output.

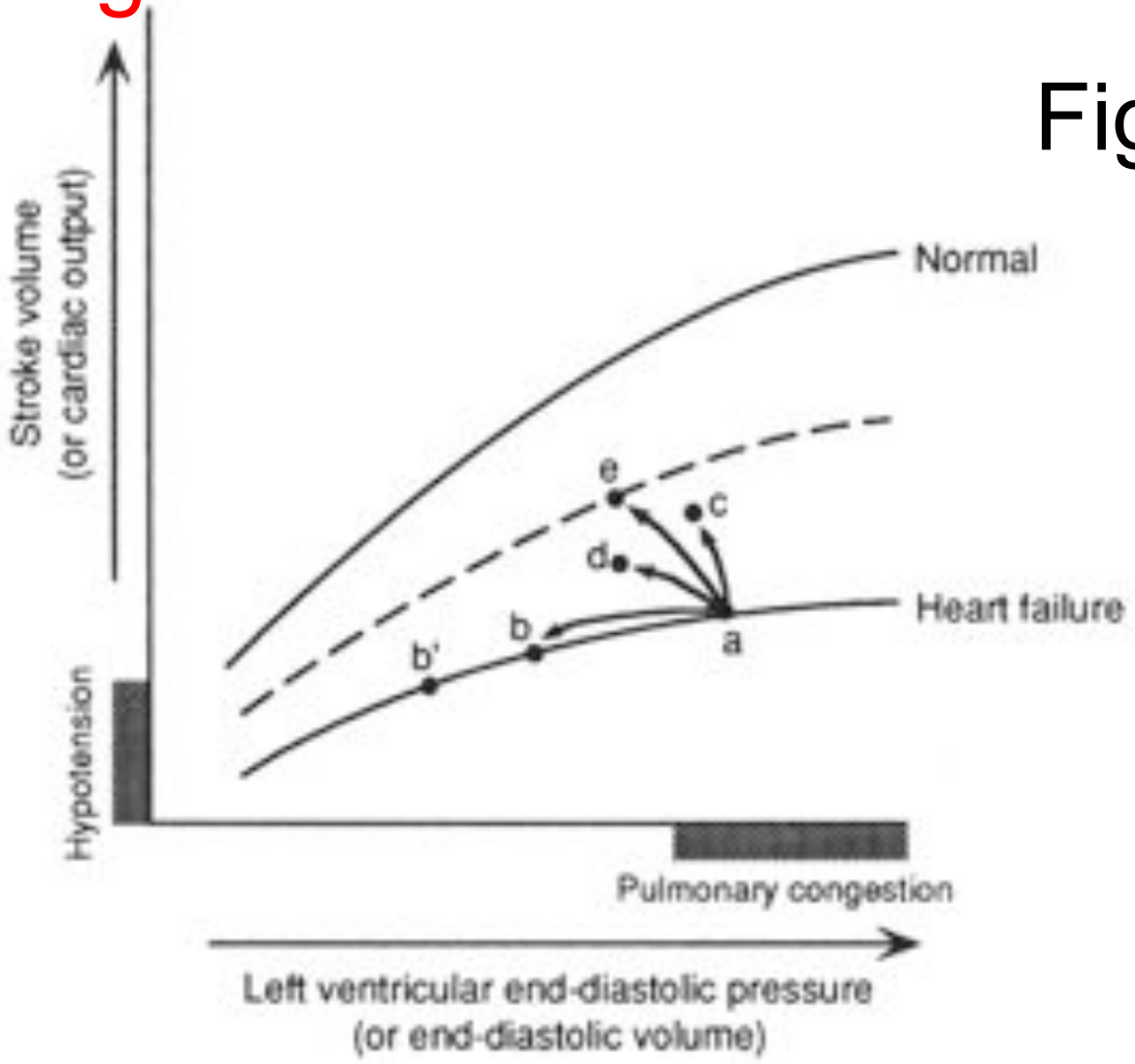
TABLE 9.3. Factors that may Precipitate Symptoms in Compensated Heart Failure

- Increased circulating volume (increased preload)
  - Excessive sodium content in diet
  - Excessive fluid administration
  - Renal failure
- Conditions that increase afterload
  - Uncontrolled hypertension
  - Pulmonary embolism (increased right ventricular afterload)
- Conditions that impair contractility
  - Negative inotropic medications
  - Myocardial ischemia or infarction
  - Ethanol ingestion
- Failure to take prescribed heart failure medications
- Excessively slow heart rate

e.g.
Beta blk.
Isoflurane
Thiopental

Coming Attractions: what to do !

Fig 9.10



HF-Evidence Based Therapies

1) ACE inhibitors

2) ARB's (angiotensin receptor blockers)

3) Beta-blockers

4) Aldosterone antagonists

5) Anticoagulants for Atrial fibrillation

6) Implantable cardioverter (ICD)

7) Cardiac resynchronization (CRT)

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