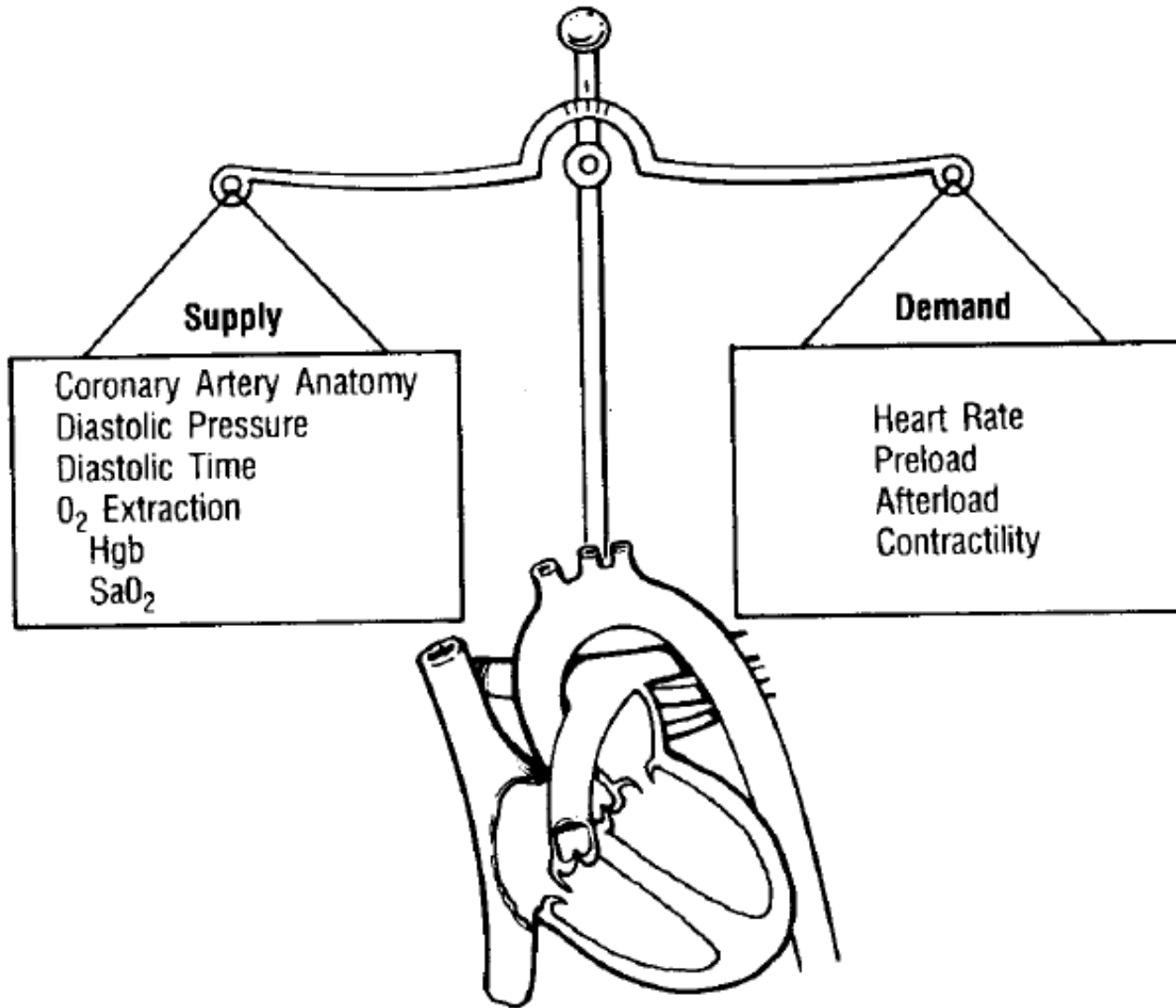


Hemodynamic dysfunction



SM COPOTOIU





Anatomy and physiology

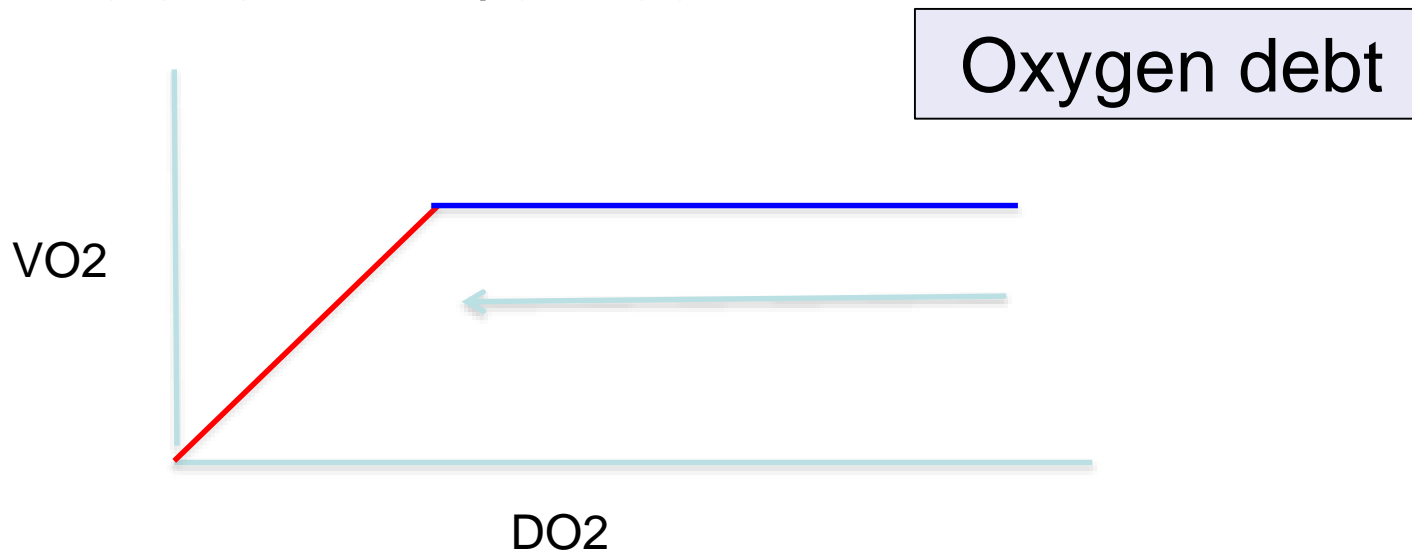
- **Supply/delivery** vs demand
- **DO₂**/min
- Oxygen content CaO₂
 - Arterial (20.1 ml/dL) & venous (15.5 ml/dL)
 - $CaO_2 = 1.38 \times Hgb \times SaO_2 + 0.0031 \times PaO_2$
- Cardiac output
- X 10 (ml/min)

- Consumption (VO₂)
- Arterial - venous
 - $DC \times Hgb \times 13.8 \times (SaO_2 - SvO_2)$
 - 200-250 ml/min



Anatomy and physiology

- Oxygen extraction fraction/index
 - 20-30%
 - $\frac{CaO_2 - CvO_2}{CaO_2} \times 100$
- SvO_2
 - $1 - (VO_2/DO_2) \times 10$
- Relatia ofertă/consum



Right

- Receives deoxygenated blood
- Low pressure system
- Volume pump**
- RV thin walls, crescent shape
- Biphasic coronary perfusion

Left

- Receives oxygenated blood
- High pressure system
- Pressure pump**
- LV thick walls, conical shape
- Diastolic coronary perfusion

Cardiac output

- $CO = HR \times SV$
 - HR 60-100 bpm
 - SV 60-100 ml/b
- $SV = EDV - ESV$
- $EF = (SV/EDV) \times 100$
 - right 40-60%
 - left 60-75%

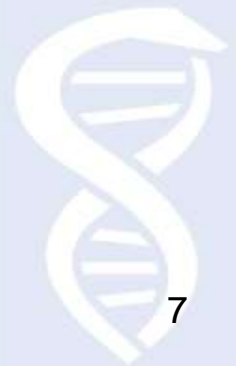


Preload

- Amount of myocardial fibre stretch at the end of diastole
- **VOLUME** vs
- **PRESSURE** (influence of compliance)
 - Left
 - LAFP
 - PAOP
 - LAP

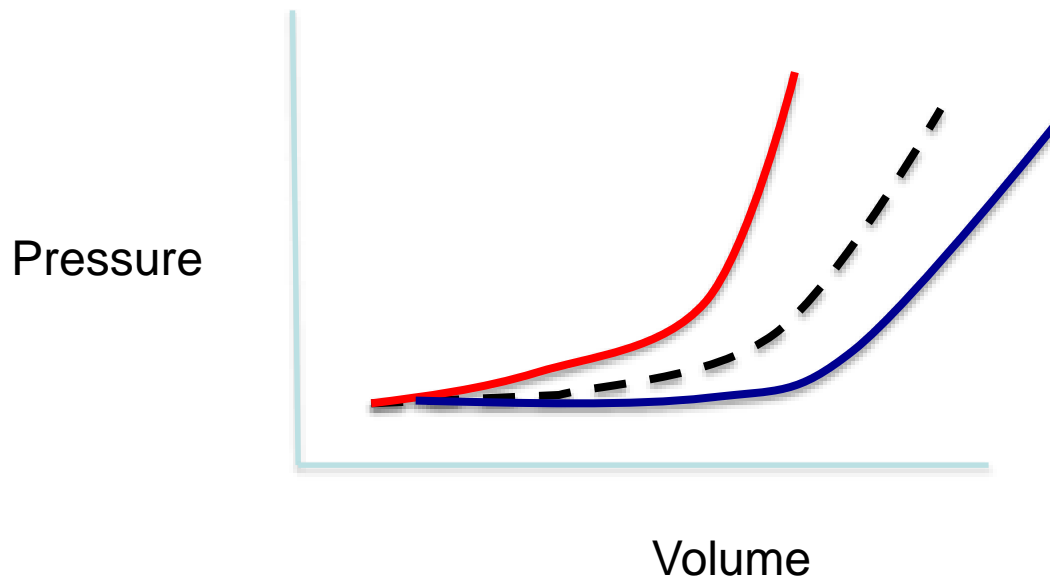
6-12 mmHg
 - Right
 - RAP
 - CVP

2-6 mmHg
- Relationship between myocardial fibre length and force of contraction



Ventricular compliance

- Relationship between volume and pressure



Ventricular compliance

Decreased

- Ischemia
- ↑ afterload
- Hypertension
- Inotropes
- Restrictive cardiomyopathies
- ↑ intrathoracic pressure
- ↑ pericardial pressure
- ↑ abdominal pressure

Increased

- Dilated cardiomyopathies
- ↓ afterload
- vasodilators



Afterload

- Tension developed by the myocardial muscle fibers during ventricular systolic ejection
- Resistance/impedance/pressure that the ventricle must overcome to eject its blood volume
- Determined by
 - SV
 - Size/wall thickness of the ventricle
 - Vascular impedance
- RVS 800-1200
- RVP < 250
- **TRANSMURAL !!!!!**

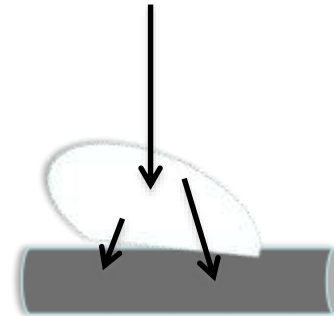
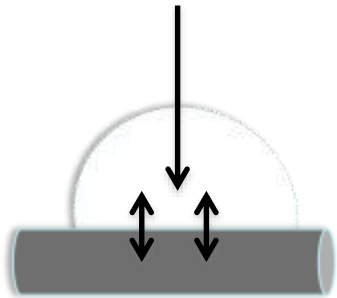


Contractility/Inotropism

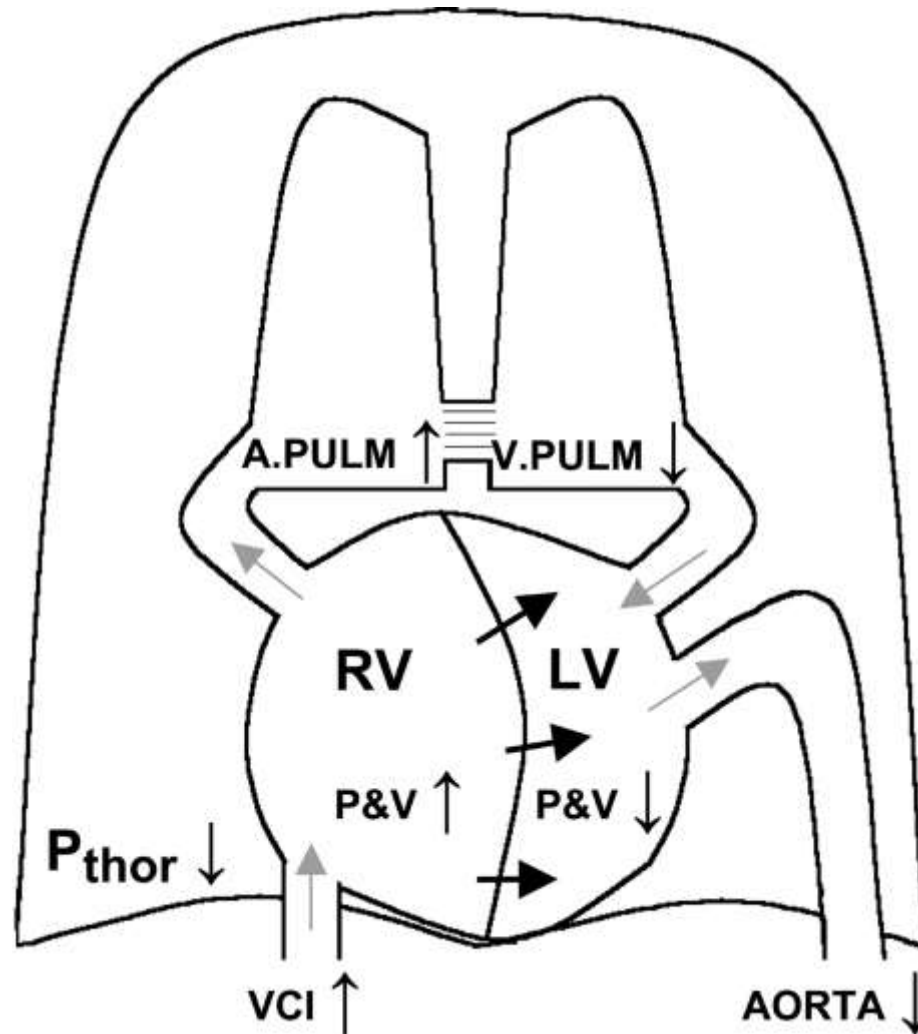
- Inherent property of the myocardial muscle fibers to shorten independent of preload and/or afterload
- It cannot be directly measured



Interactions



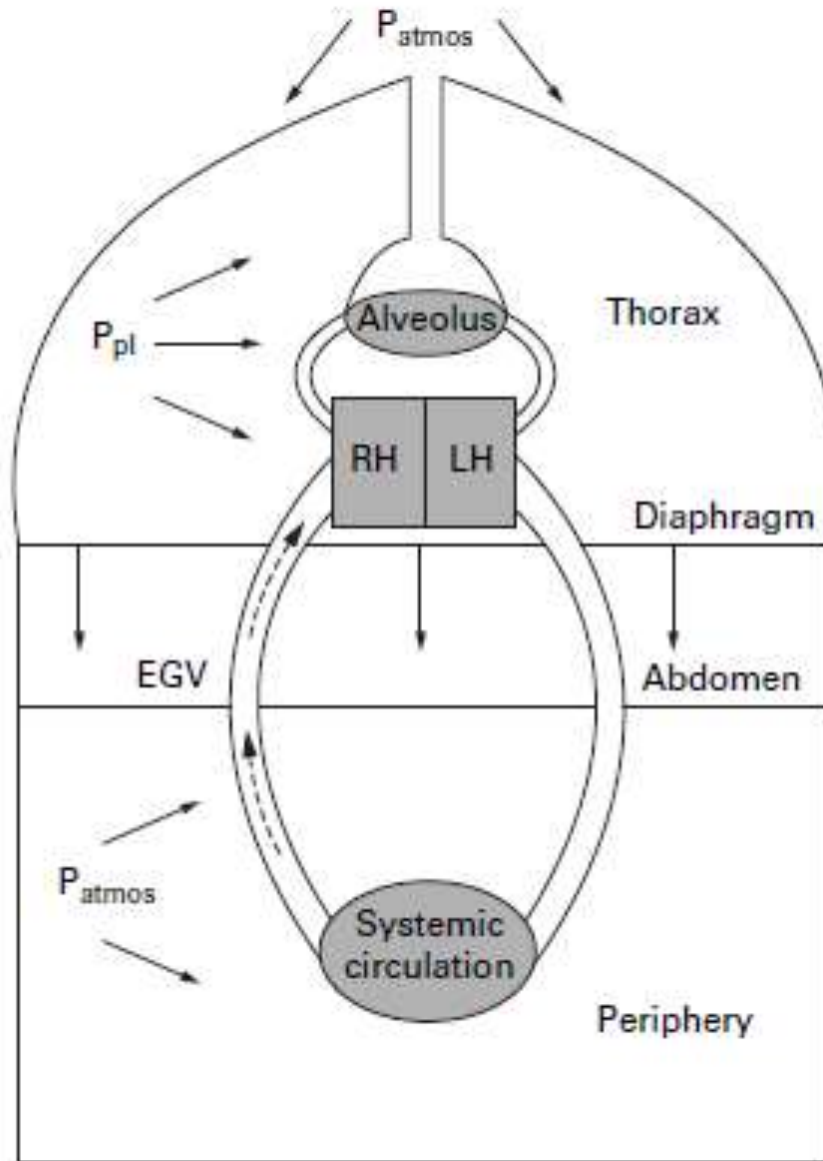
Hemodynamic events during inspiration



van den Hout R J et al. Radiology 2003;229:513-519

Radiology





Interactions – positive intrathoracic pressure

↑ CO if VIV is N (↓ afterload)

↓ CO if VIV is ↓ (inadequate filling pressure)

PRELOAD

- ↓ ITP: mobilizes blood from pulmonary veins into the RV
- ↑ ITP: RV afterload ↑ → ↓ LZ & RV filling

AFTERLOAD

- Right
 - Unchanged for N lung
 - ☞ ↑ for stiff lung
- Left
 - ☞ ↓ if filling is unchanged



Normal pressures (mmHg)

Location	Abbreviation	Mean value	Limits
Central vein	PVC/CVP	6	1-10
Right atrium	AD/RAP	4	-1,+8
Right ventricle systole	PVD/RVSP	24	15-28
Right ventricle end diastole	PDVD/RVEDP	4	0-8
Pulm a syst	PAsP	24	15-28
Pulm a diast	PAdP	10	5-16
Pulm a mean	PAP	16	10-22

Normal pressures (mmHg)

Location	Abbreviation	Mean value	Limits
Pulmonary capillary	PCWP	9	5-16
Left atrium	PAS/LAP	7	4-12
Left ventricle systole	PSVS	130	90-140
Left ventricle diastole	LVEDP	7	4-12
Brachial a syst	TAs	130	90-140
Brachial a diast	TAd/dBP	70	60-90
Brachial a mean	TAm	85	70-105

Relationship between cavitory pressures

CVP – myocardial repletion

Pulmonary vascular resistance is 6x less than systemic vascular resistance

$$1\text{mmHg} = 1,36\text{cmH}_2\text{O}$$

$$1\text{ cm H}_2\text{O} = 0,74\text{mmHg}$$

$$1\text{kPa} = 7,5\text{mmHg} = 10,2\text{cmH}_2\text{O}$$



Hemodynamic parameters

Parameter	Abbreviation	Formula	Units	Limits
Cardiac output	DC, CO	Measure	l/min	5-6
Cardiac index	IC, CI	CO/BSA	l/min/m²	2,8-4,2
Heart rate	FC, HR	Measure	bpm	60-90
Stroke volume	SV	COx100/HR	ml/beat	60-90
Stroke index	SI	SV/BSA	ml/beat/m²	45-60
Systemic vascular resistance	RSV, SVR	(TAm-PVC)x 80/DC	dynexsec/cm⁻⁵	900-1500
Pulmonary vascular resistance	RVP/PVR		dynexsec/cm⁻⁵	150-250

Monitoring good practice

1. Know what you are doing
2. Know how to do it
3. Understand what you see
4. Look out for early changes
5. Interpret
6. Notify
7. Write down: what you saw, what you did, what were the consequences
8. Treat the patient, not the monitor!



Golden rules

- CVP does not reflect RVEDV
- PAOP does not reflect R(L)VEDV
- PVC and PAOP cannot predict CO response to fluid challenge
- CO cannot be estimated on clinical examination
- CVP and PAOP cannot predict APE
- Normal MAP does not mean adequate CO



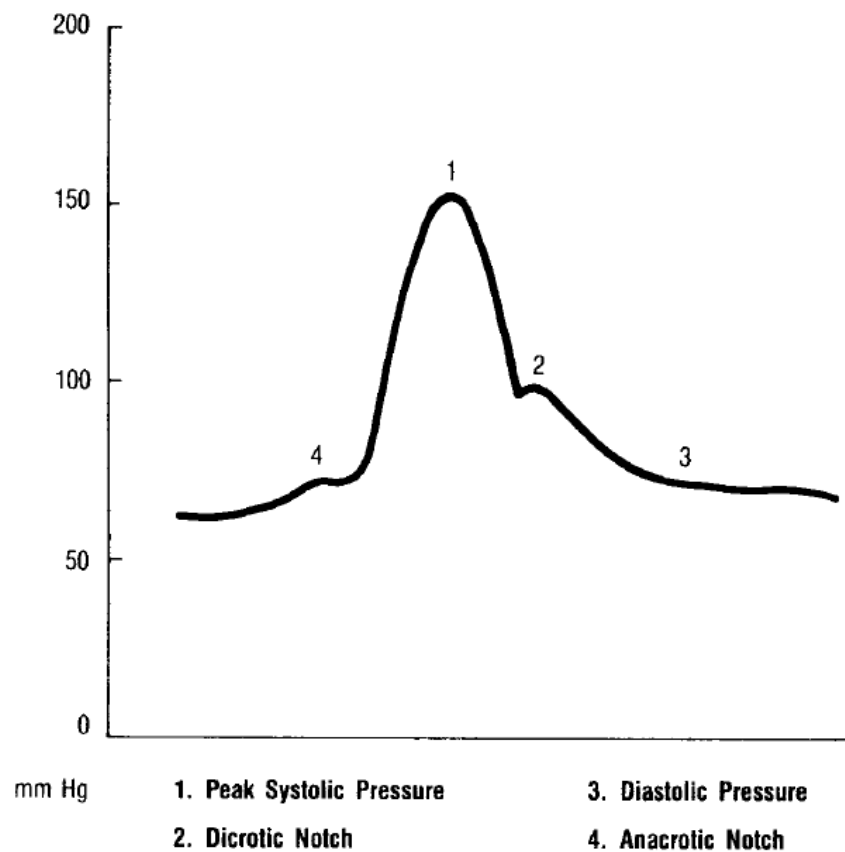
Golden rules

- Normal calculated oxygen delivery is no guarantee of adequate tissue perfusion
- SvO_2 normal values are not always equivalent to an adequate tissue perfusion
- Changes in oxygen consumption as a response to altered oxygen delivery (calculated) are not always synonymous to an existent oxygen debt
- PAOP is no substitute for pressure in pulmonary capillaries



Arterial catheterization





Central venous catheterization

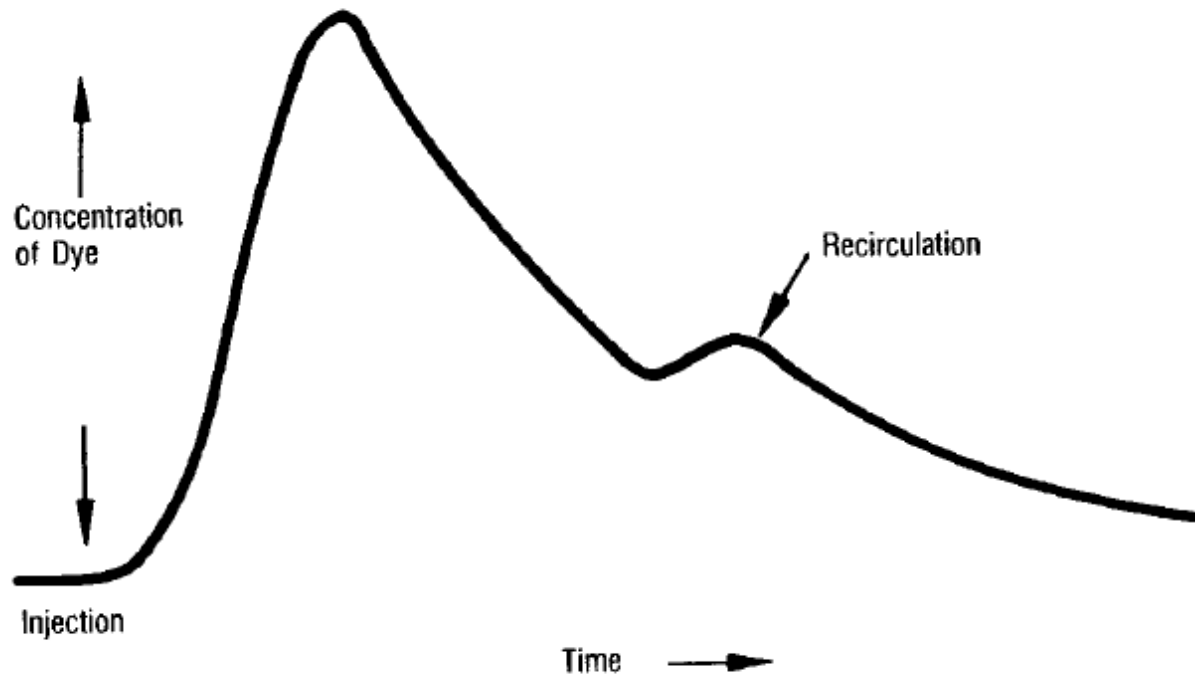
Internal jugular vein



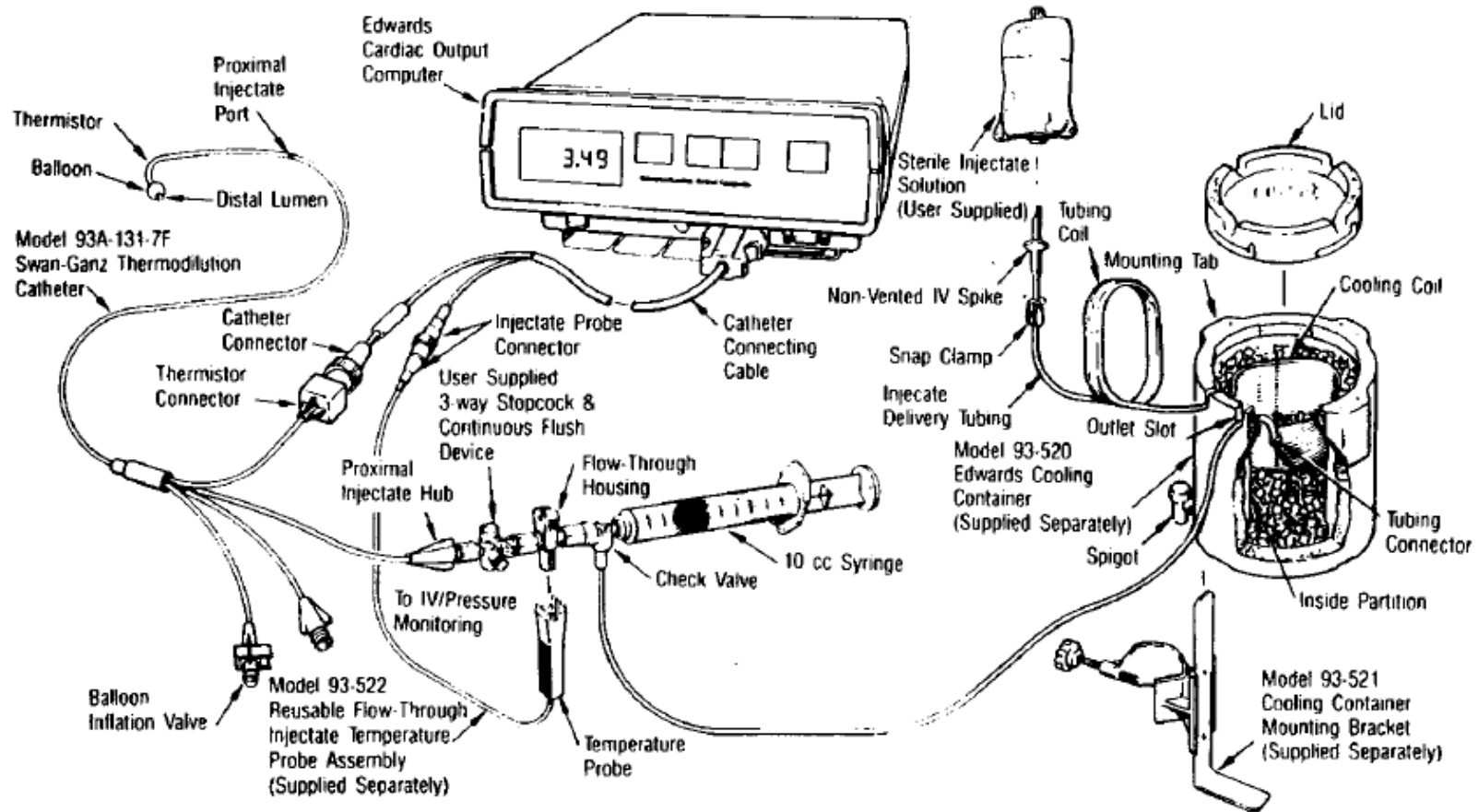
Central venous catheterization Subclavian vein



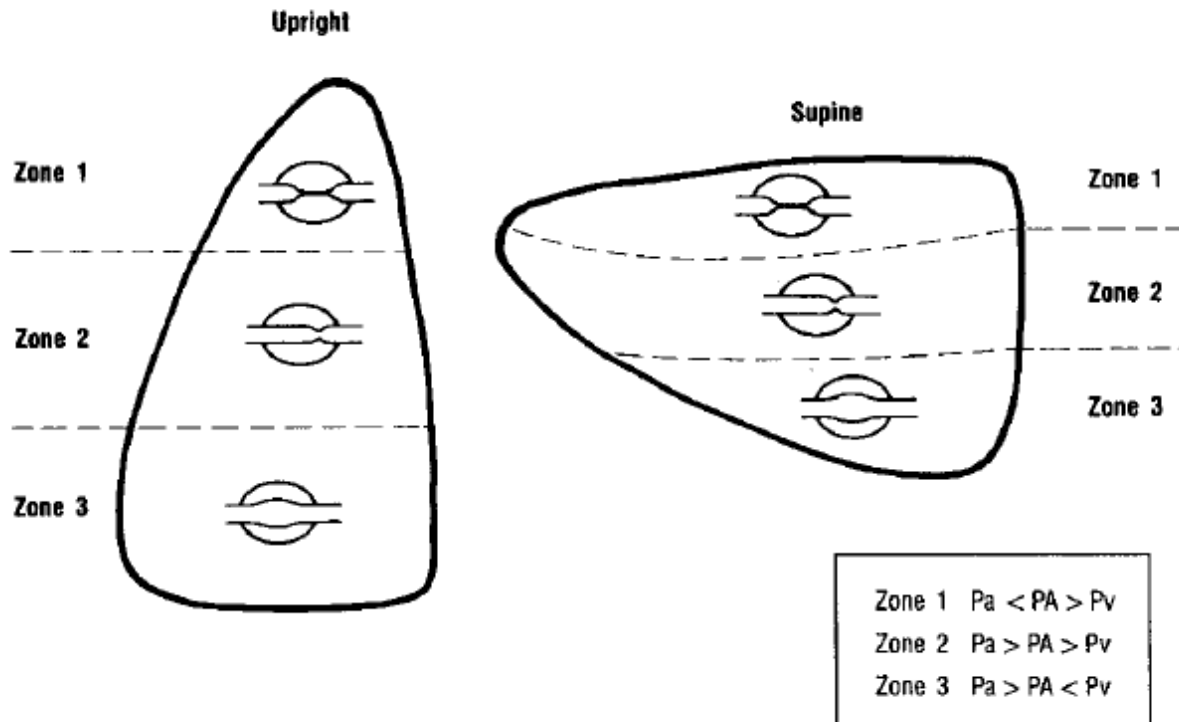
Dilution



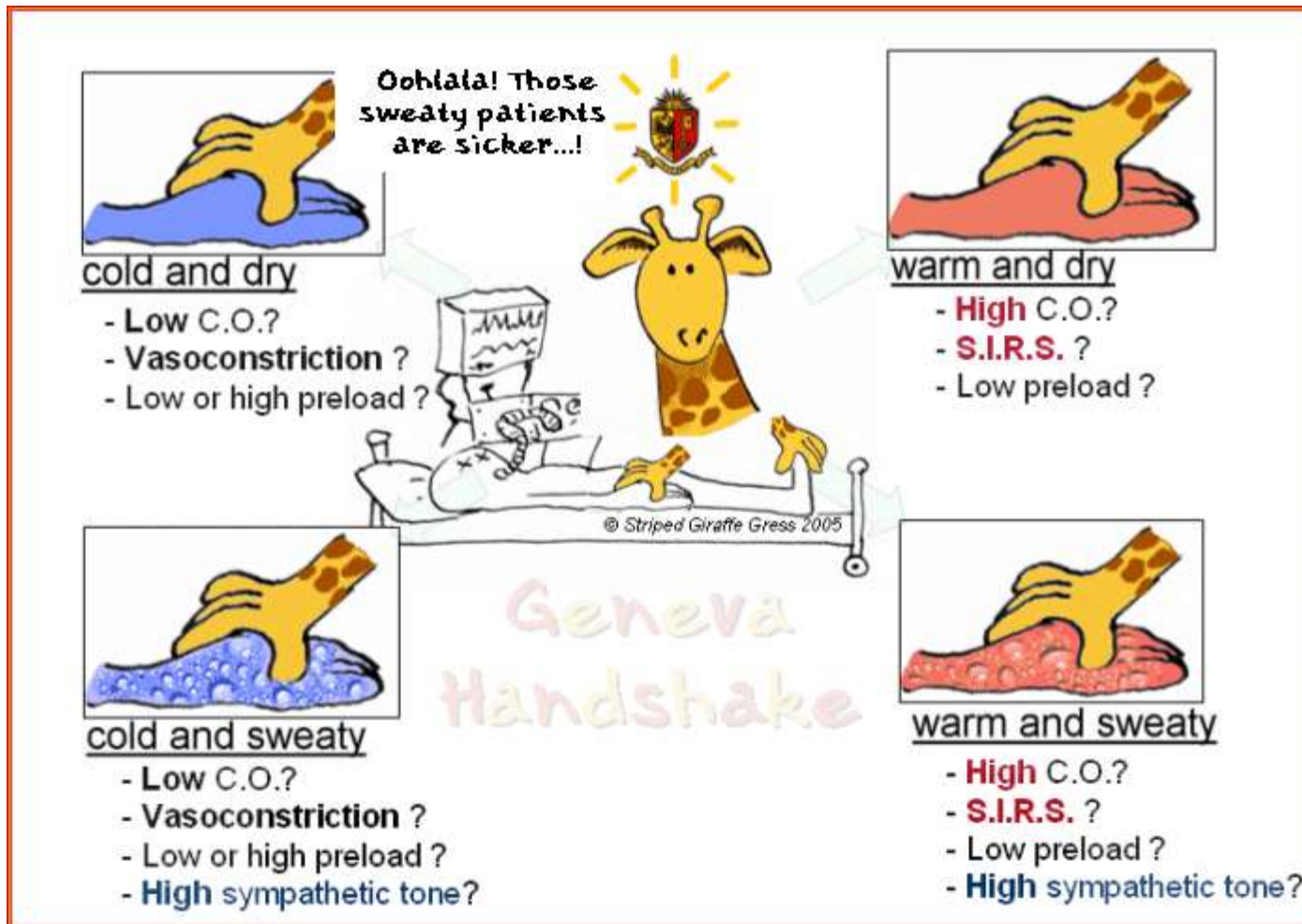
System



Lung zone placement

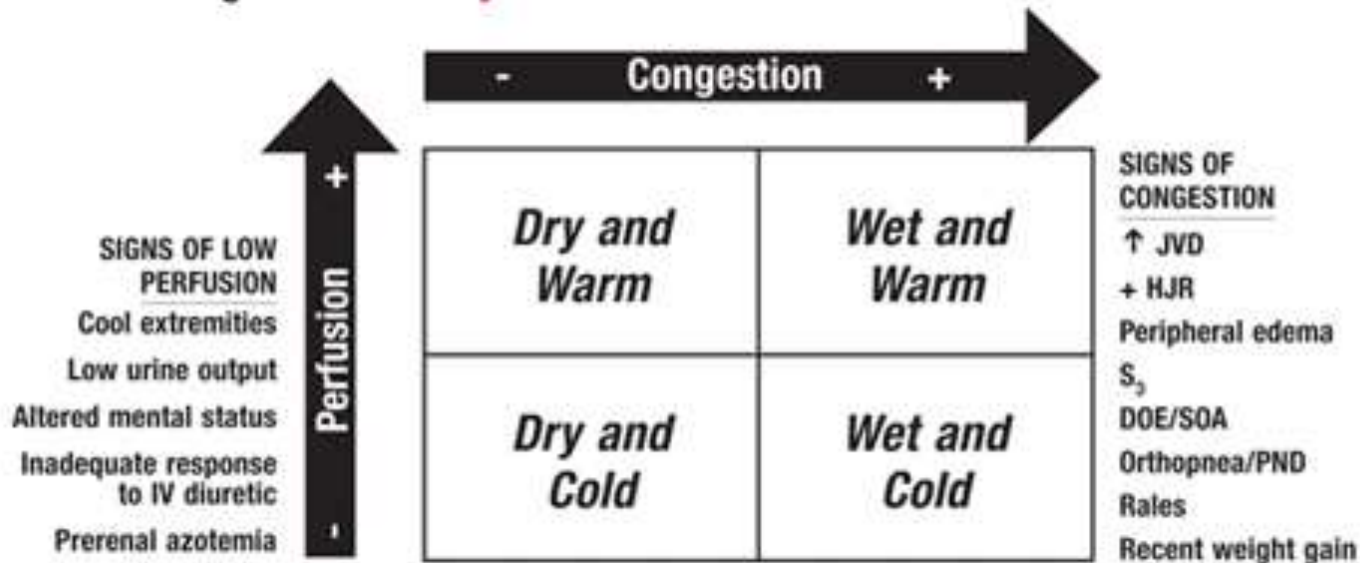


Clinical evaluation



Heart failure

Figure 1. Hemodynamic/Clinical State in Acute Heart Failure



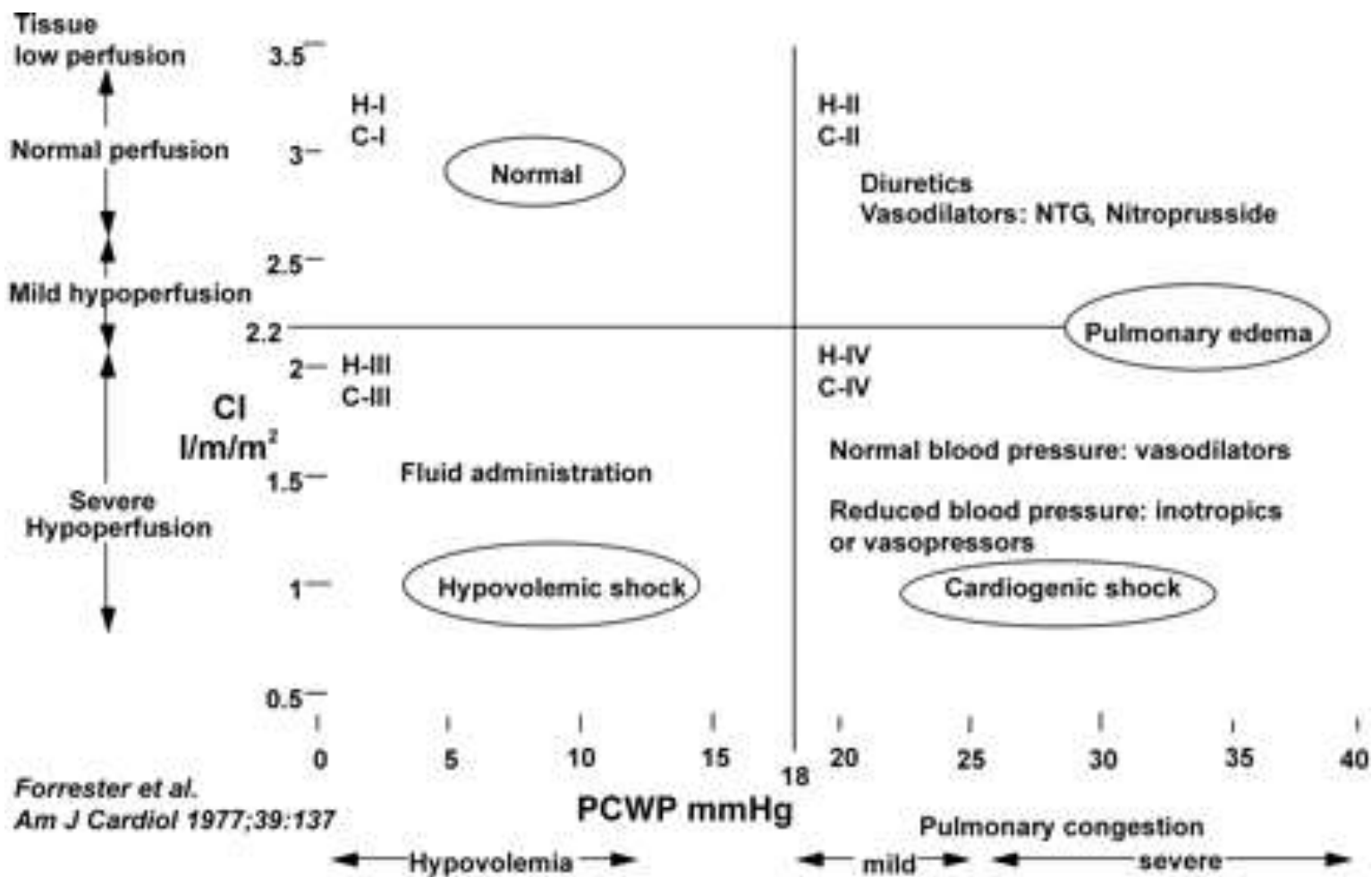
↑: increased; +: positive; -: negative; DOE: dyspnea on exertion; HJR: hepatojugular reflux; JVD: jugular venous distention; PND: paroxysmal nocturnal dyspnea; S₃: ventricular filling murmur; SOA: shortness of air.

Source: References 10, 11.



Forrester classification

Hemodynamic subsets



Forrester classification

Therapy and outcome

	Indications	PCW	Therapy	Mortality
SUBSET I No Failure	Cardiac Index Greater than 2.2 L/MIN/M ²	Less than 18 mmHg	Sedate	3%
SUBSET II Pulmonary Congestion	Cardiac Index Greater than 2.2 L/MIN/M ²	Greater than 18 mmHg	Normal Blood Pressure: Diuretics Elevated Blood Pressure: Vasodilators	9%
SUBSET III Peripheral Hypoperfusion	Cardiac Index Less than 2.2 L/MIN/M ²	Less than 18 mmHg	Elevated Heart Rate: Add Volume Depressed Heart Rate: Pacing	23%
SUBSET IV Congestion & Hypoperfusion	Cardiac Index Less than 2.2 L/MIN/M ²	Greater than 18 mmHg	Depressed Blood Pressure: Inotropes Normal Blood Pressure: Vasodilators	51%



Inotropic therapy

- USA
 - Low output syndrome
 - Left ventricular systolic dysfunction
 - Systolic blood pressure < 90 mmHg despite adequate filling pressure
- Europe
 - BP < 100 mmHg



Inotropic therapy

EPINEPHRINE



Inotropic therapy

NOREPINEPHRINE



Inotropic therapy

DOPAMINE



Inotropic therapy

DOBUTAMINE



Inotropic therapy

DOPEXAMINE



Inotropic therapy

ISOPROTERENOL (ISOPRENALINE)



Inotropic therapy

PHOSPHODIESTERASE INHIBITORS



Inotropic therapy

CALCIUM SENSITIZERS



Inotropic therapy

On trial

- **Cardiac myosin activators**
 - ↑ ATPase activity
 - Inotrop effect
- **Istaroxime**
 - Inhibits the Na/K ATPase
 - Inotrop & lusitrop effects

