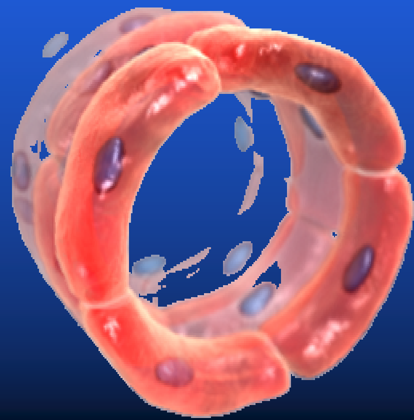
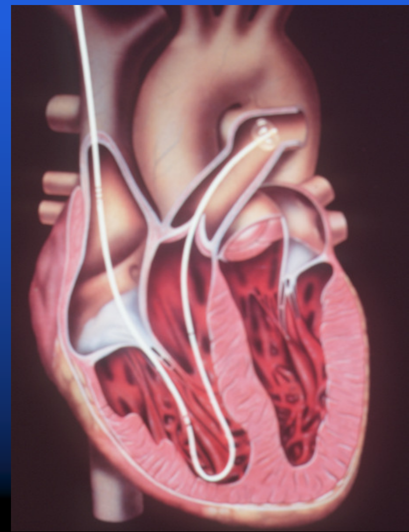


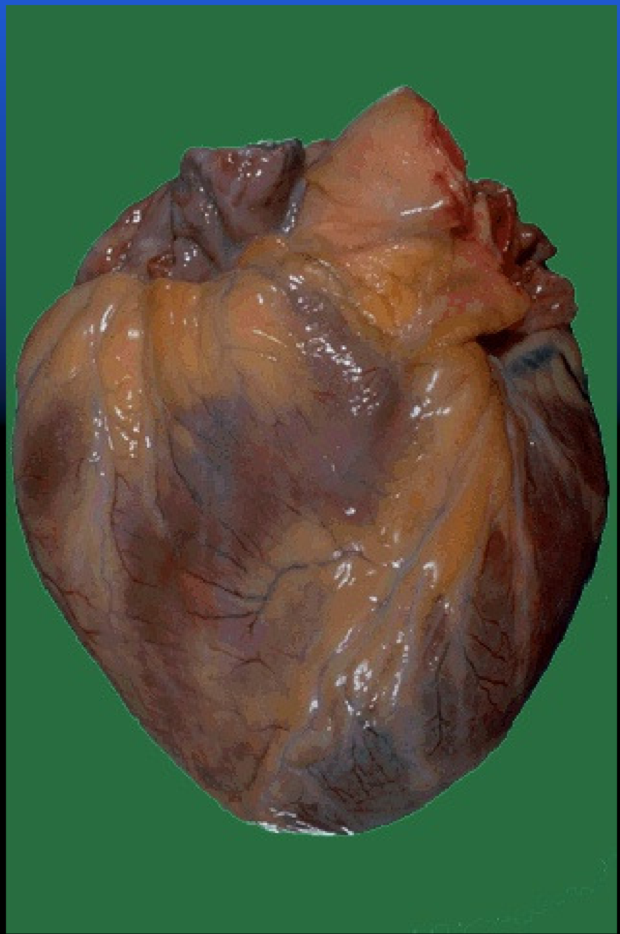


Dr. Krisada Sastravaha M.D.
Chief of Cardiology
Bhumibol Adulyadej Hospital

Cardiovascular Anatomy and Hemodynamic Monitoring

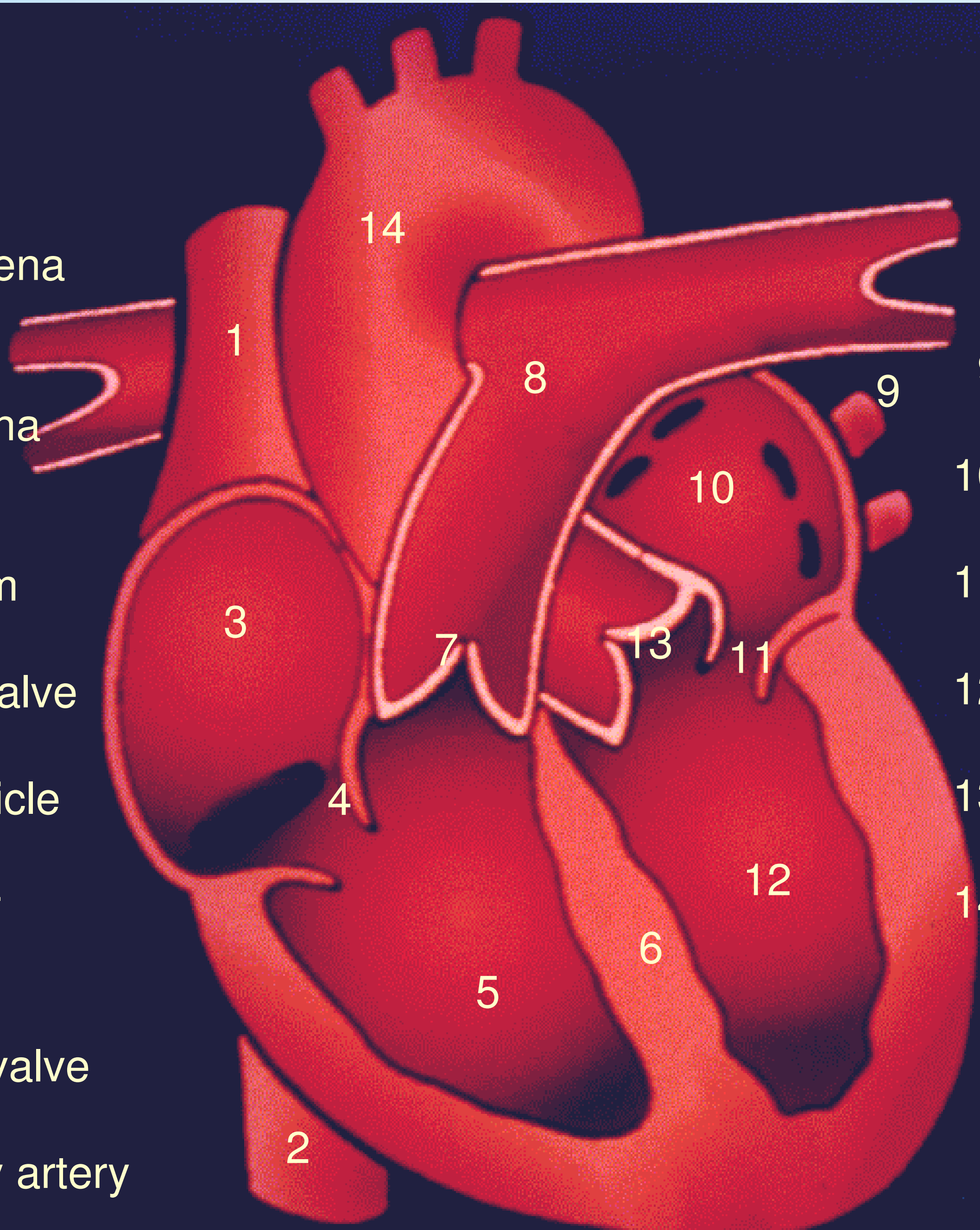
11 March 2011

Our Heart
The Supreme Pump



The Supreme Pump

- 60 - 75 beats per min.
- 3,600 beats per hour
- 86,400 beats per day
- 604,800 beats per week
- 31,449,600 beats per year
- 1,289,433,600 beats 41 years old



1 superior vena cava

2 inferior vena cava

3 right atrium

4 tricuspid valve

5 right ventricle

6 ventricular septum

7 pulmonic valve

8 pulmonary artery

9 pulmonary veins

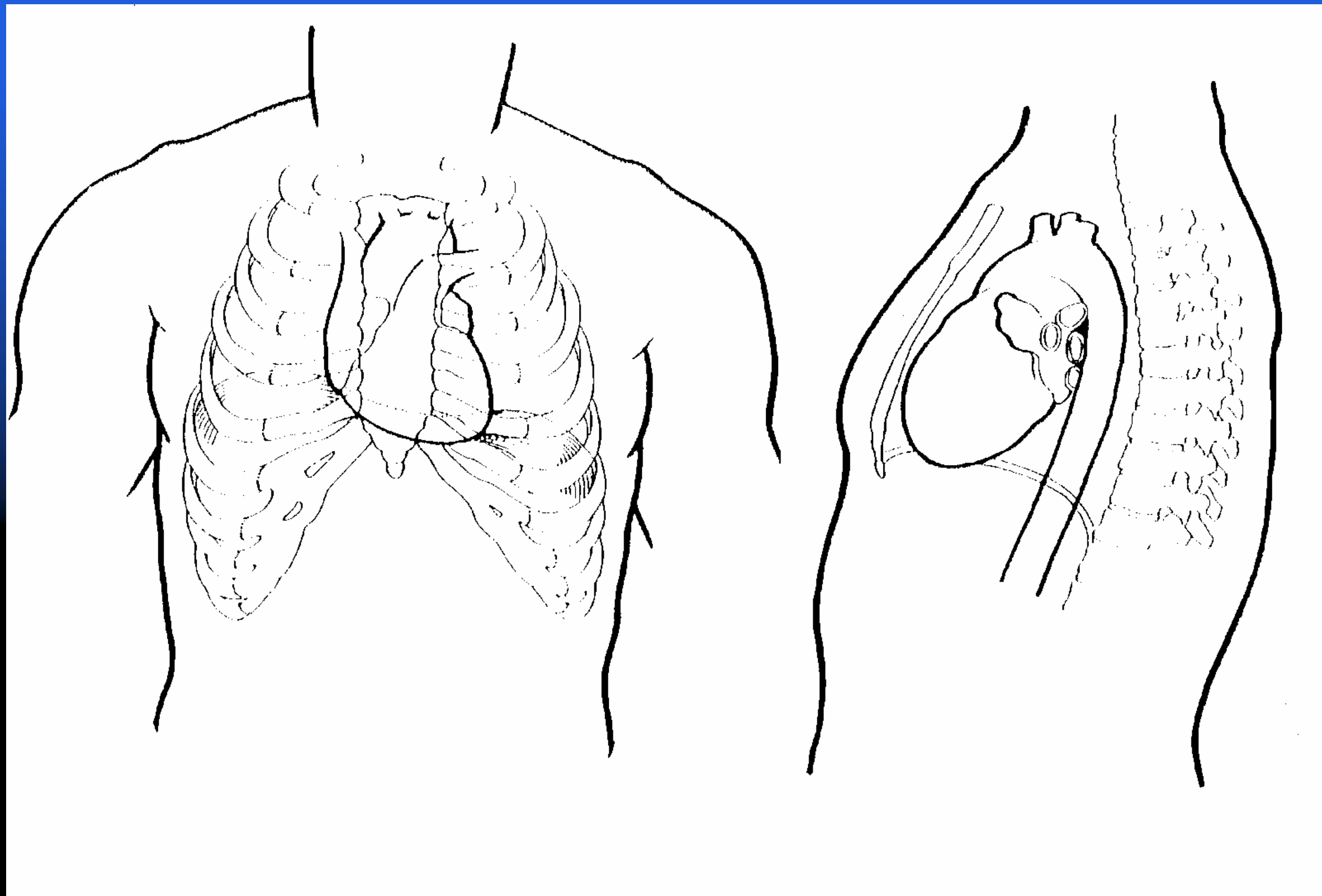
10 left atrium

11 mitral valve

12 left ventricle

13 aortic valve

14 aorta



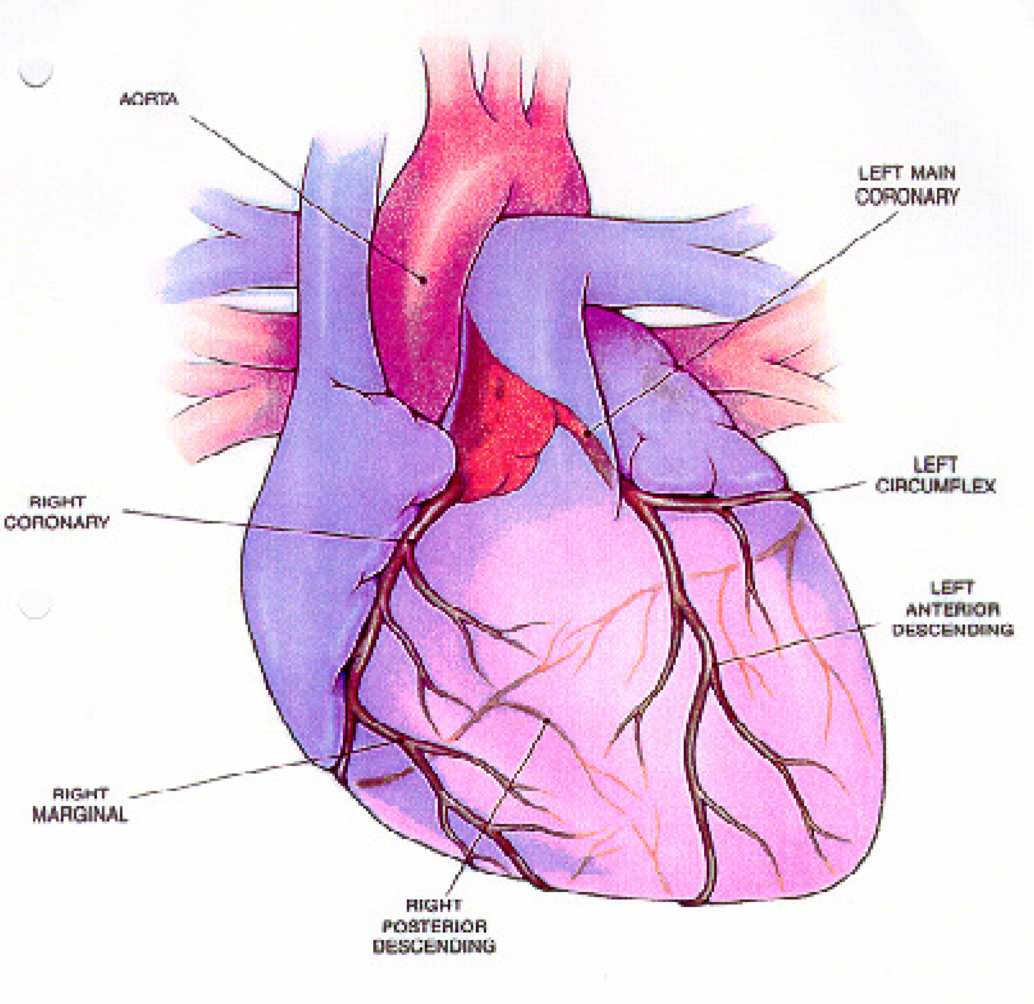
RIGHT HEART PUMP

- Superior and inferior vena cavae
- Right atrium
- Tricuspid valve
- Right ventricle
- Pulmonic valve
- Low pressure system

LEFT HEART PUMP

- Left atrium
- Mitral valve
- Left ventricle
- Aortic valve
- Aorta
- High pressure system

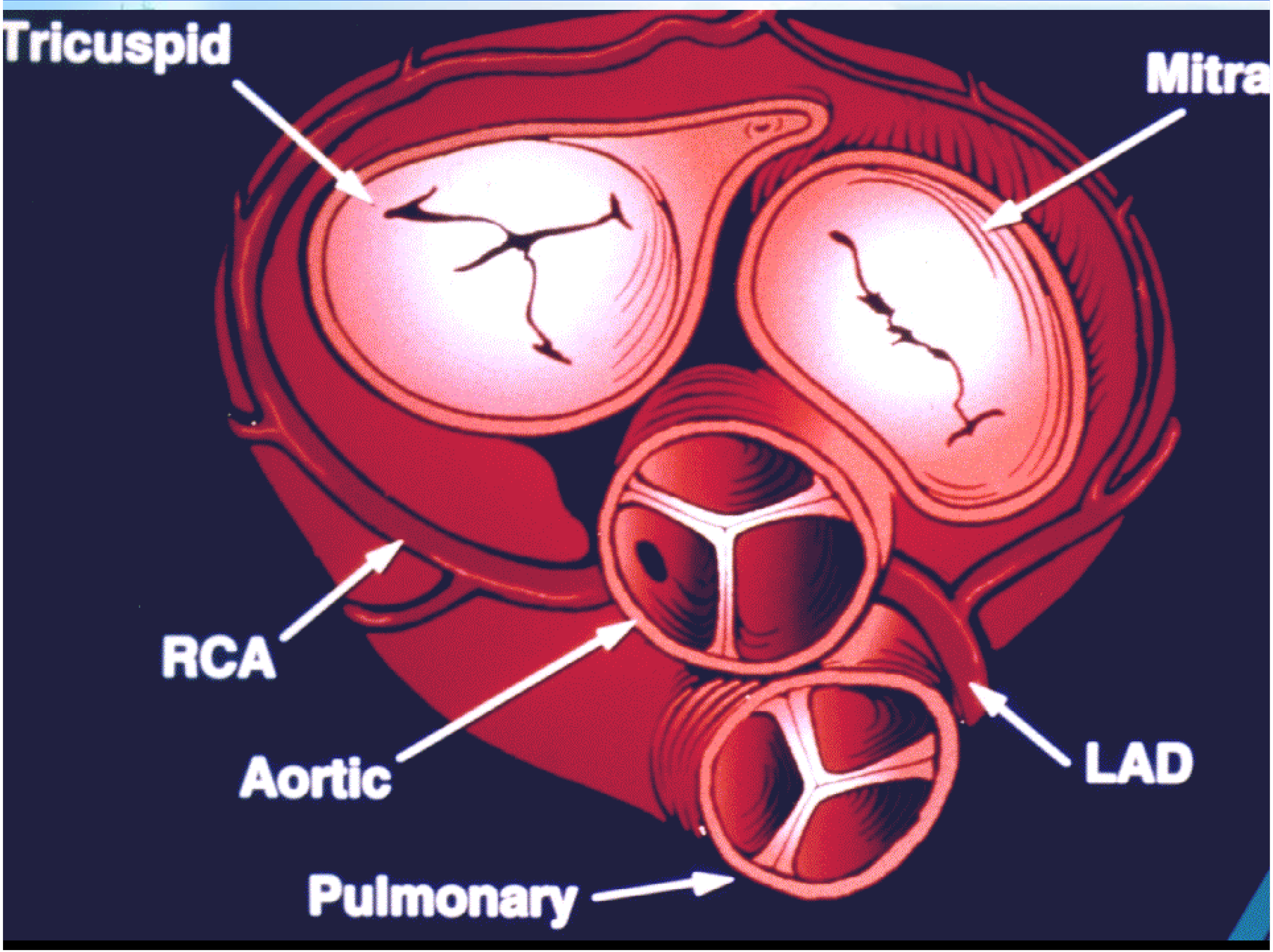
Coronary Circulation



Provide blood supply to the heart muscle

Coronary Arteries

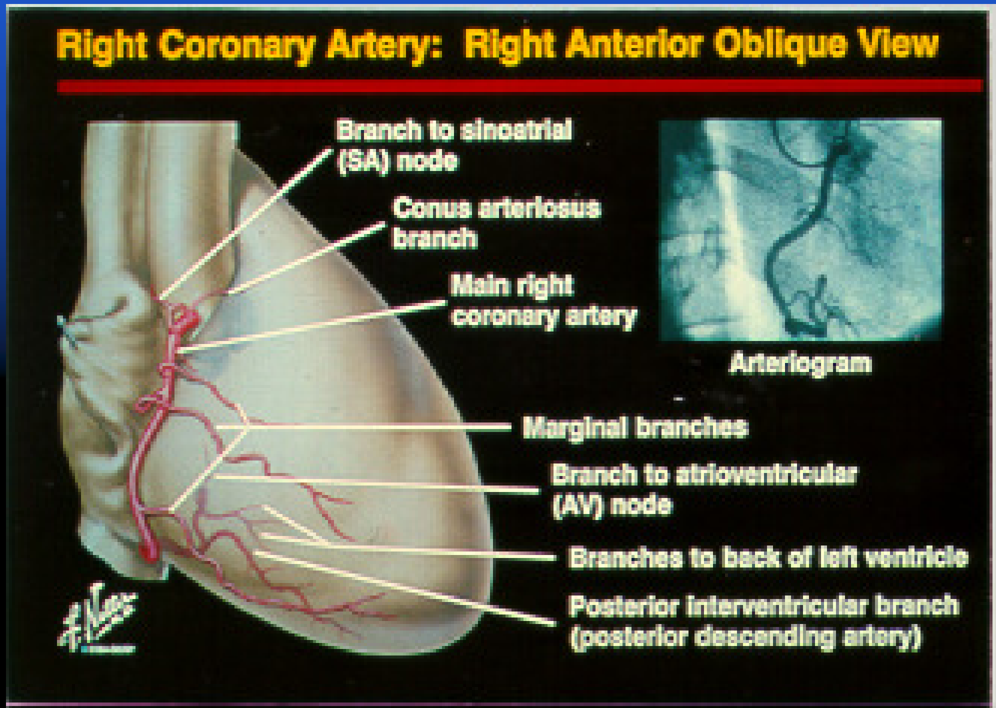
- Originate at aortic cusps
- Protected by sinus of Valsava during systole
- Provide most of coronary blood flow during diastole
- On the epicardial surface
- Send intramural branches into the myocardium


Tricuspid
Mitra
RCA
Aortic
LAD
Pulmonary

Right Coronary Artery (RCA)

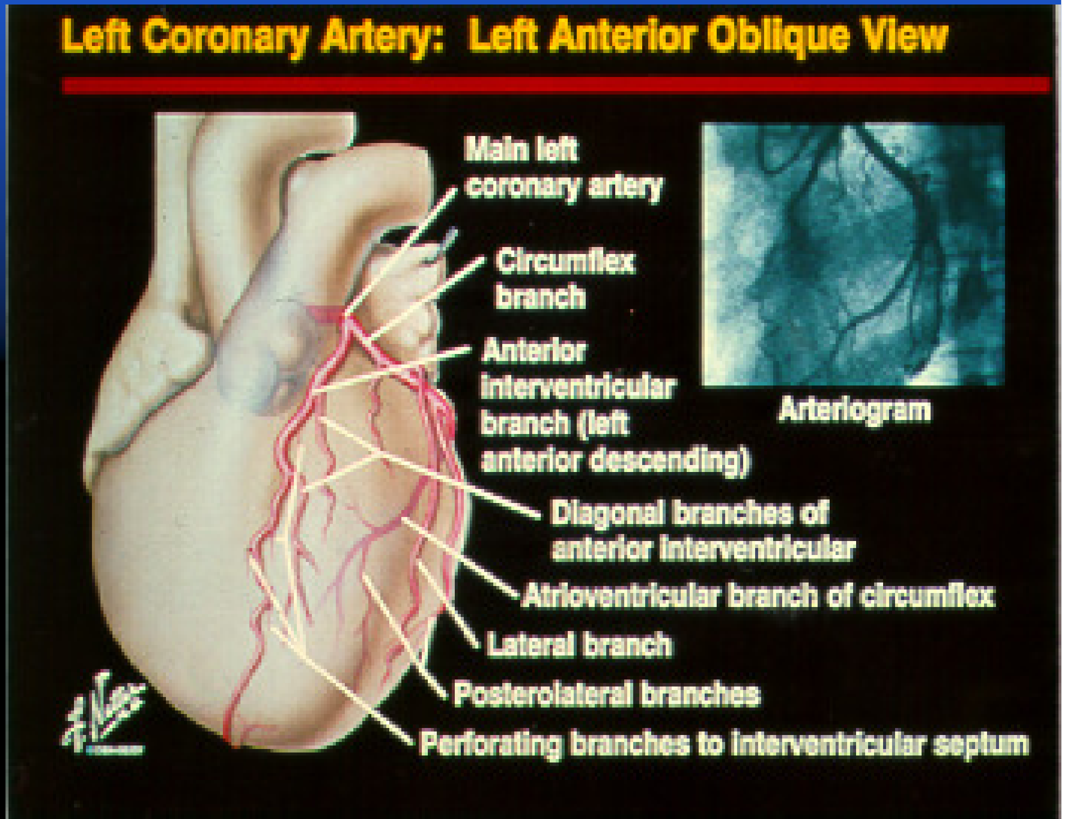
Supplies blood to:

- Right atrium
- Right ventricle
- Posterior and inferior walls of left ventricle (if dominant)



LEFT ANTERIOR DESCENDING (LAD)

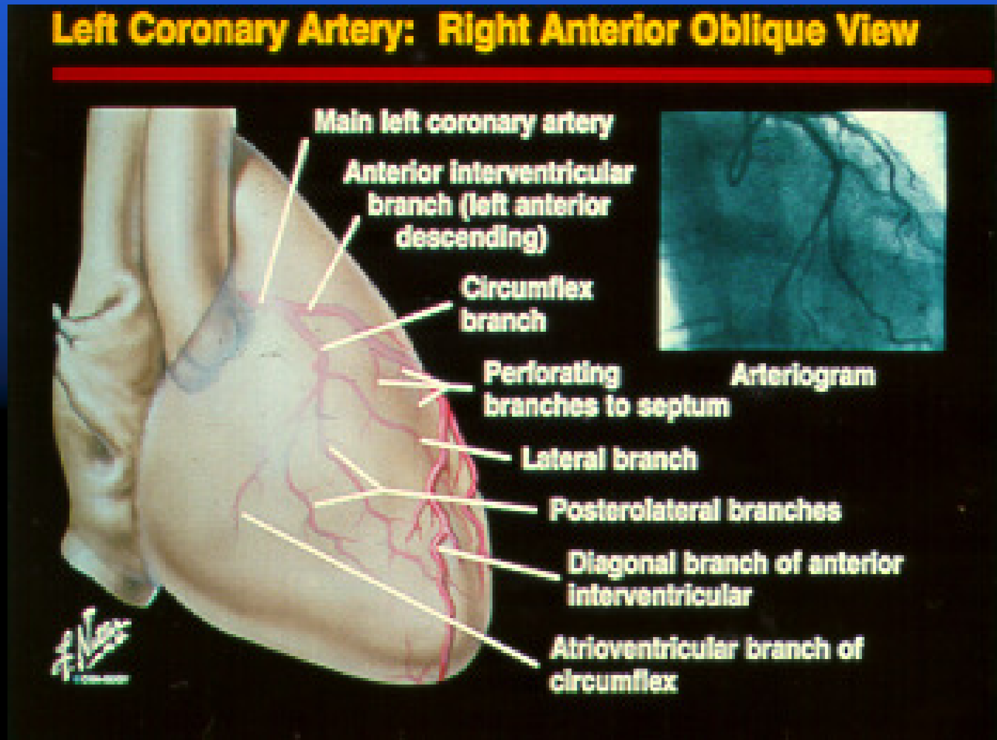
Supplies blood to the left ventricle:
- ©Anterior wall
- ©Lateral wall
- ©Apical wall
- ©Ventricular septum



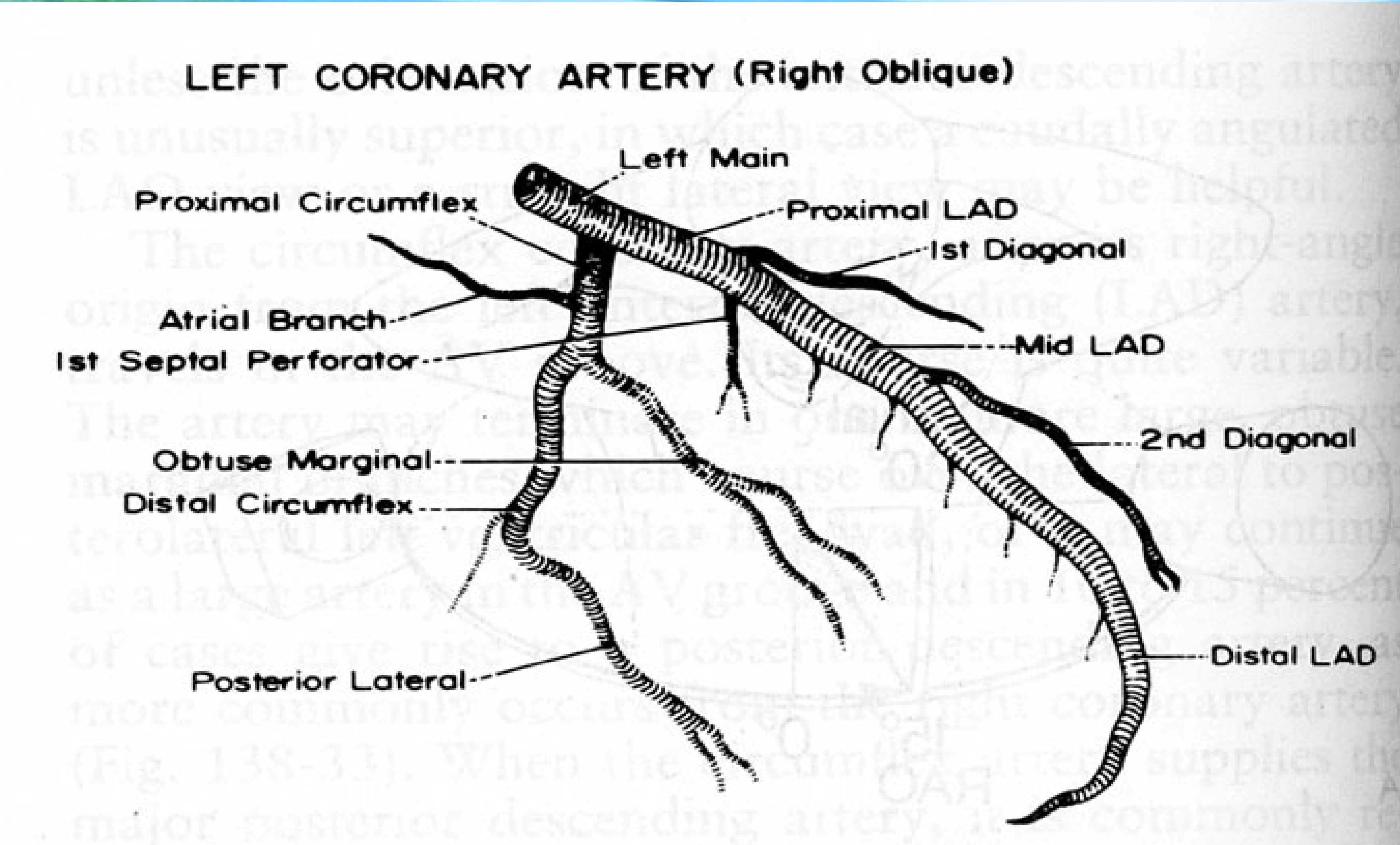
LEFT CIRCUMFLEX (LCx)

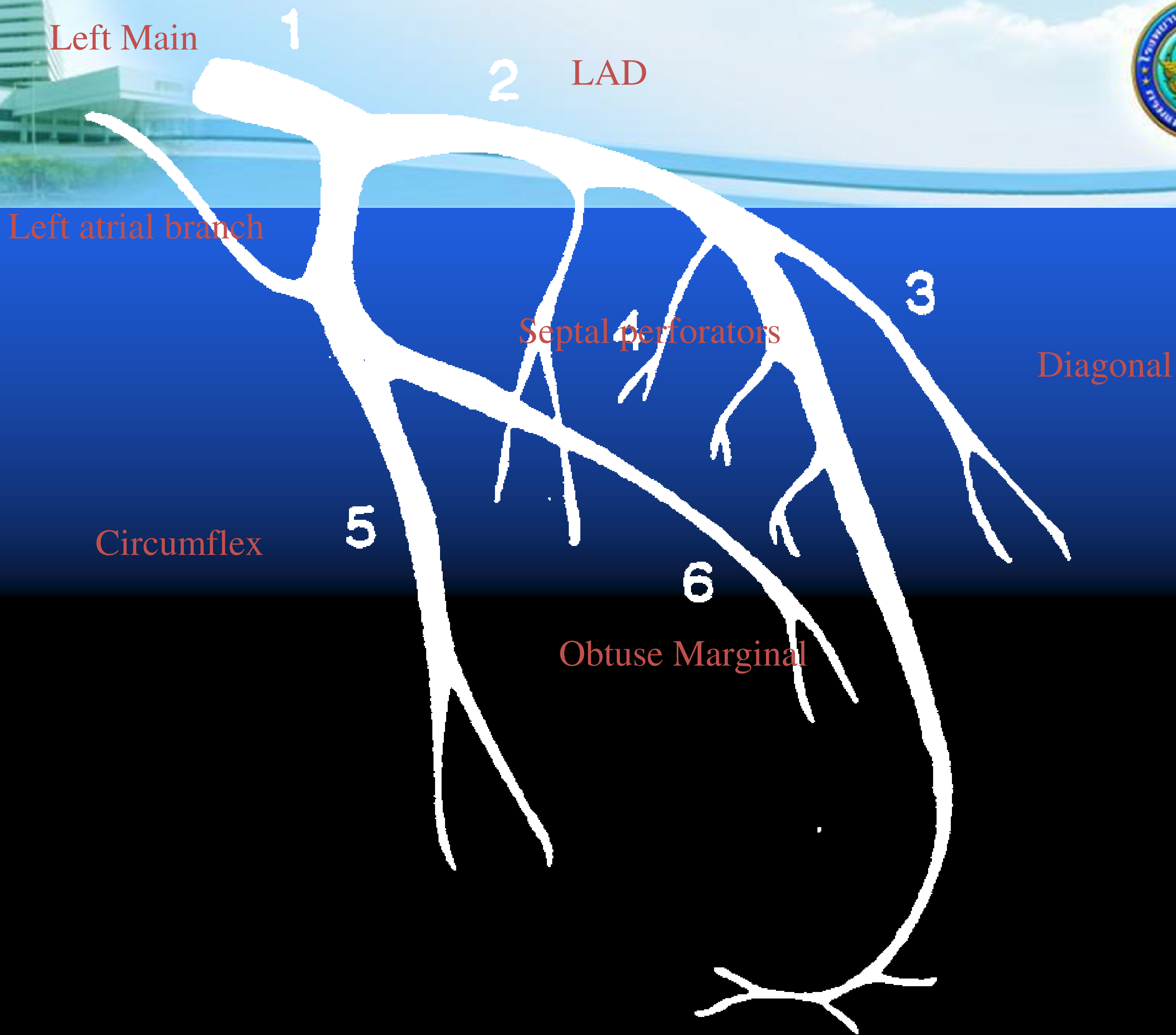
Supplies blood to:

- ♦Left atrium
- ♦Left ventricle
  - •Lateral wall
  - •Posterior wall
  - •Inferior wall (if dominant)



Left coronary artery



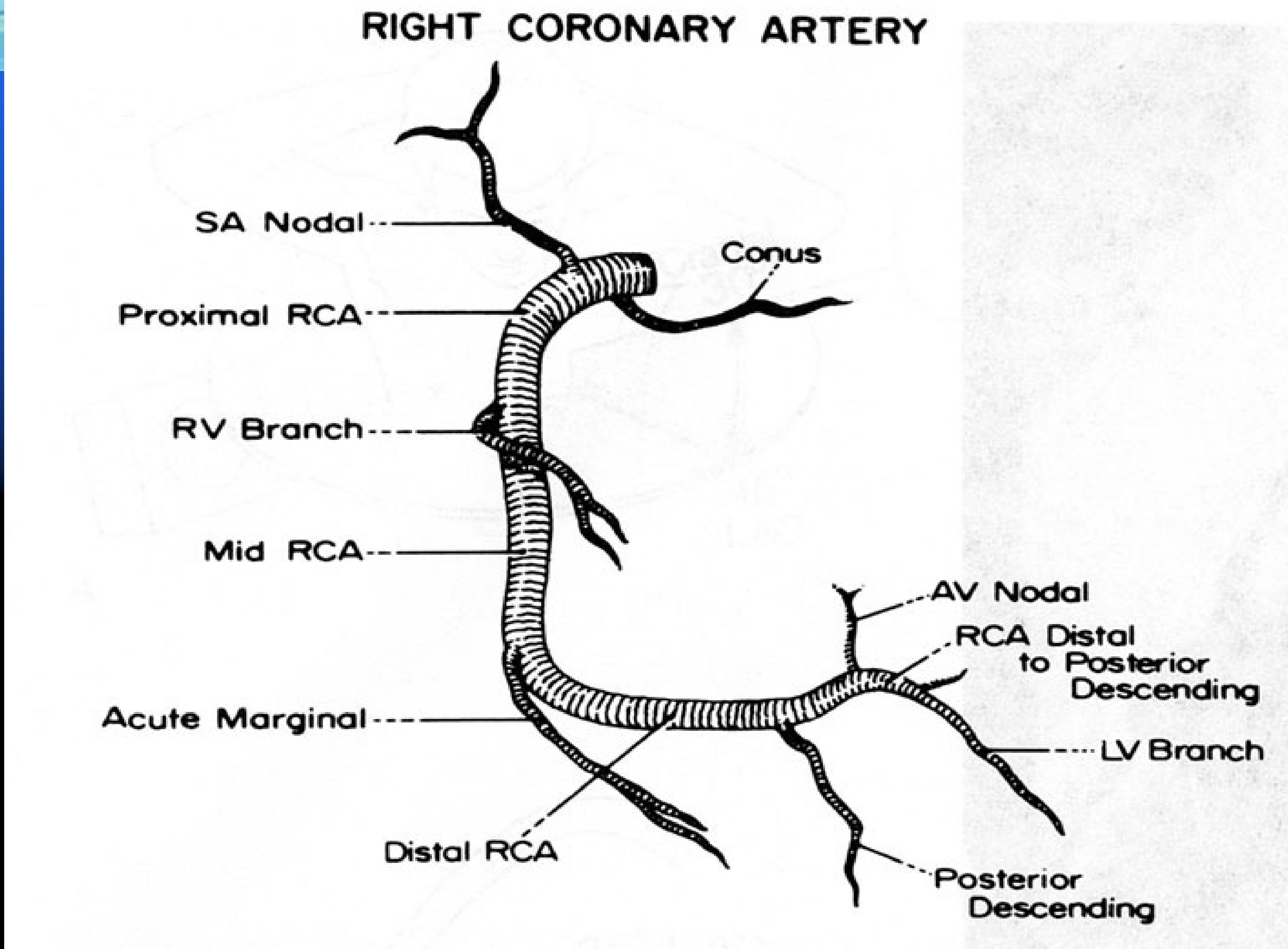

Left Main
1
2
LAD
Left atrial branch
3
Septal perforators
4
Diagonal
Circumflex
5
6
Obtuse Marginal

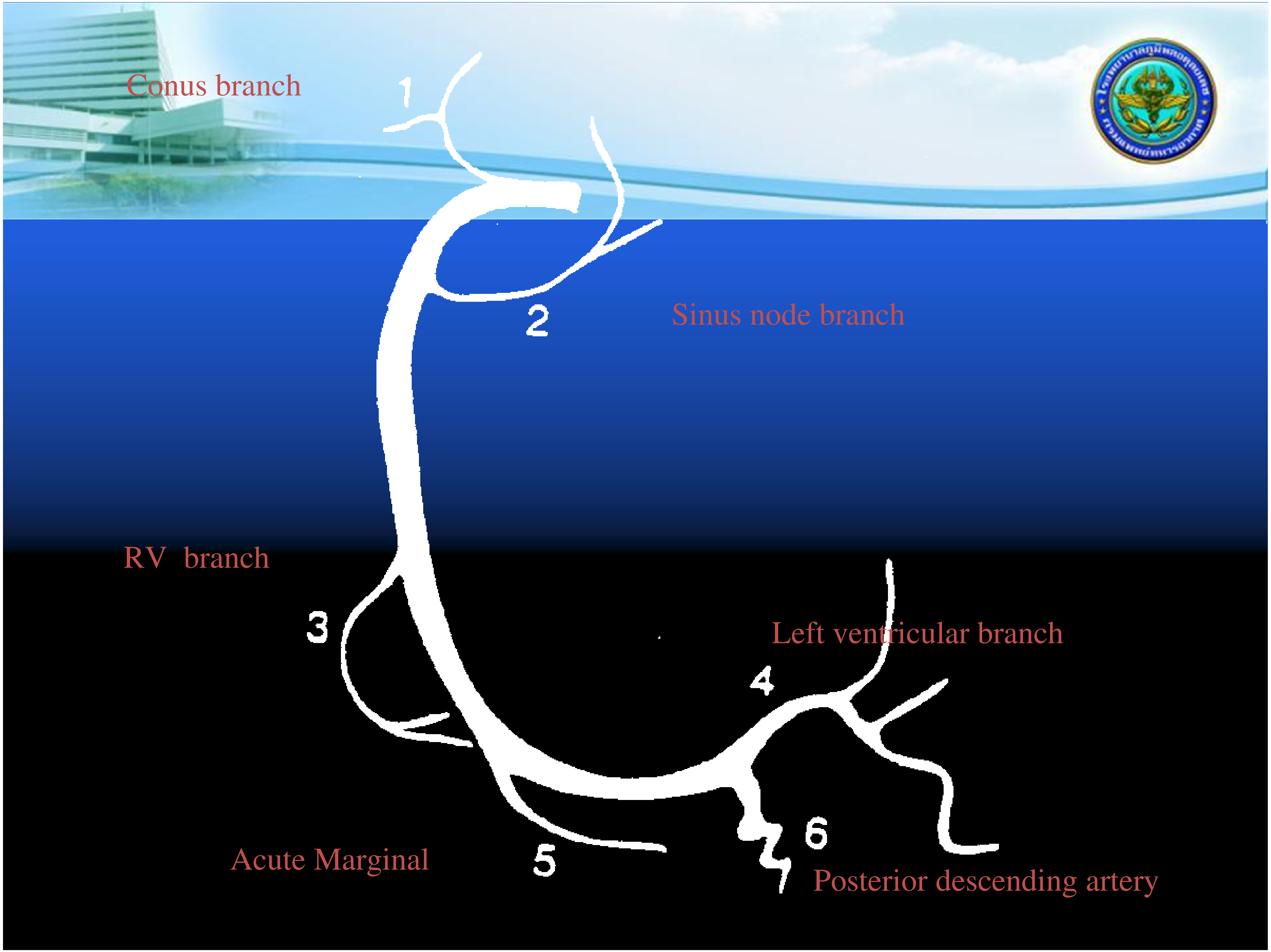
Left Coronary Artery

- LAD Supplies
  - Anterior LV
  - LV Papillary Muscle
  - Intraventricular Septum
  - Anterior surface of RV

- LCX Supplies
  - Lateral & Posterior surfaces of LA & LV

Right coronary artery

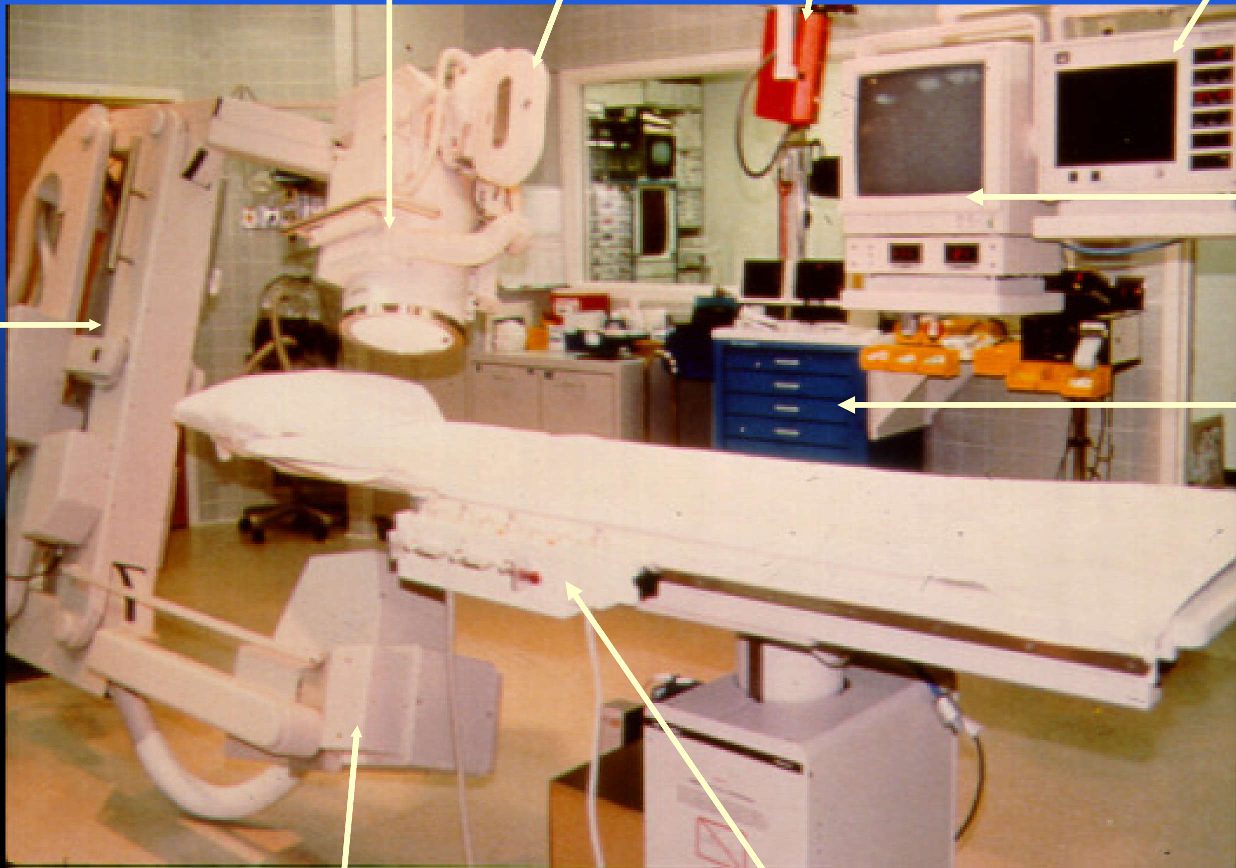




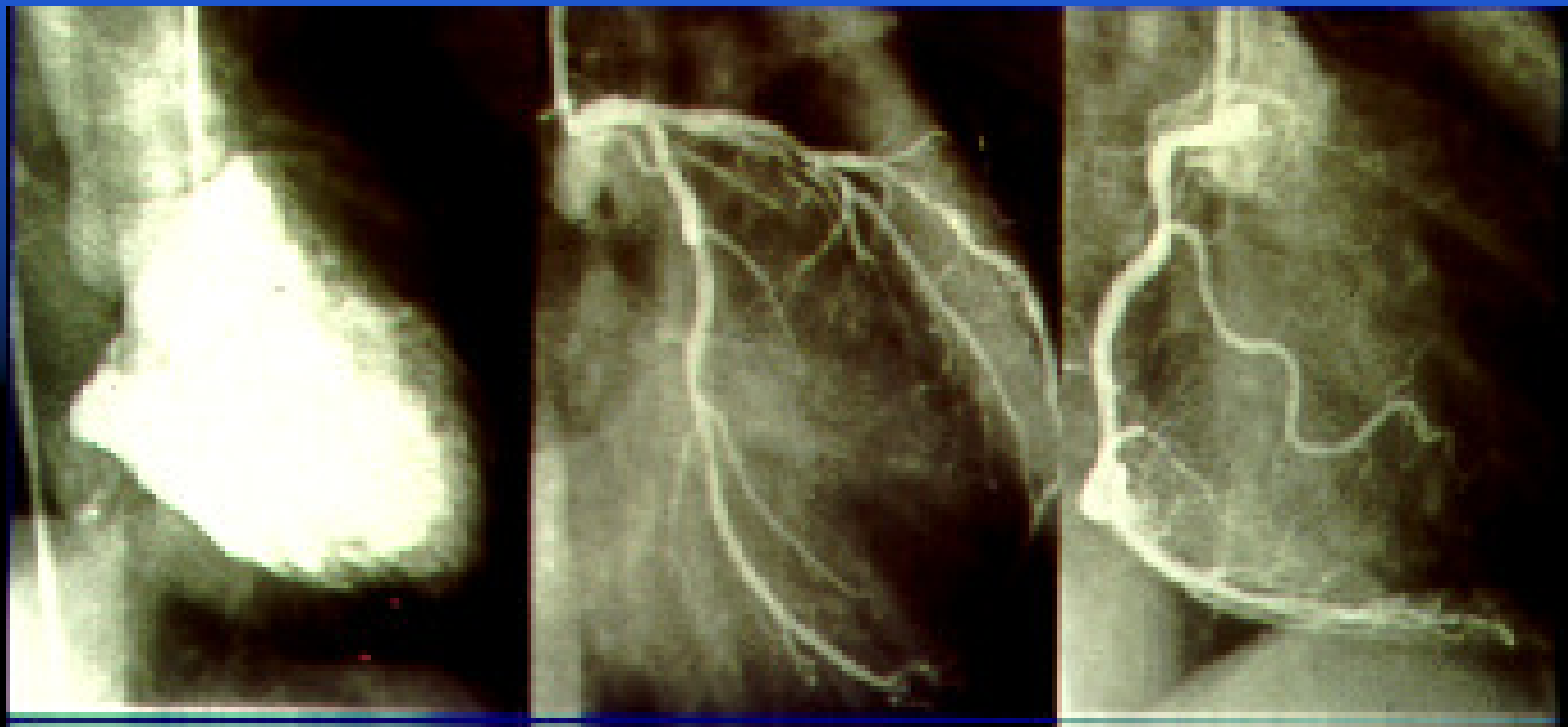
Conus branch
1
2
Sinus node branch
RV branch
3
Left ventricular branch
4
6
Acute Marginal
5
Posterior descending artery

Right Coronary Artery

- RCA Supplies
  - Right Atrium
  - SA & AV Node
  - Intra-atrial septum
  - Right Ventricle
  - RV Papillary Muscle
  - Inferior LV
  - Intraventricular septum


Cine Film
Image Intensifier
Power Injector
Hemodynamic Monitor
Image Monitor
C-arm
Crash Cart
X-Ray Tube
Control Panel

Coronary Angiography

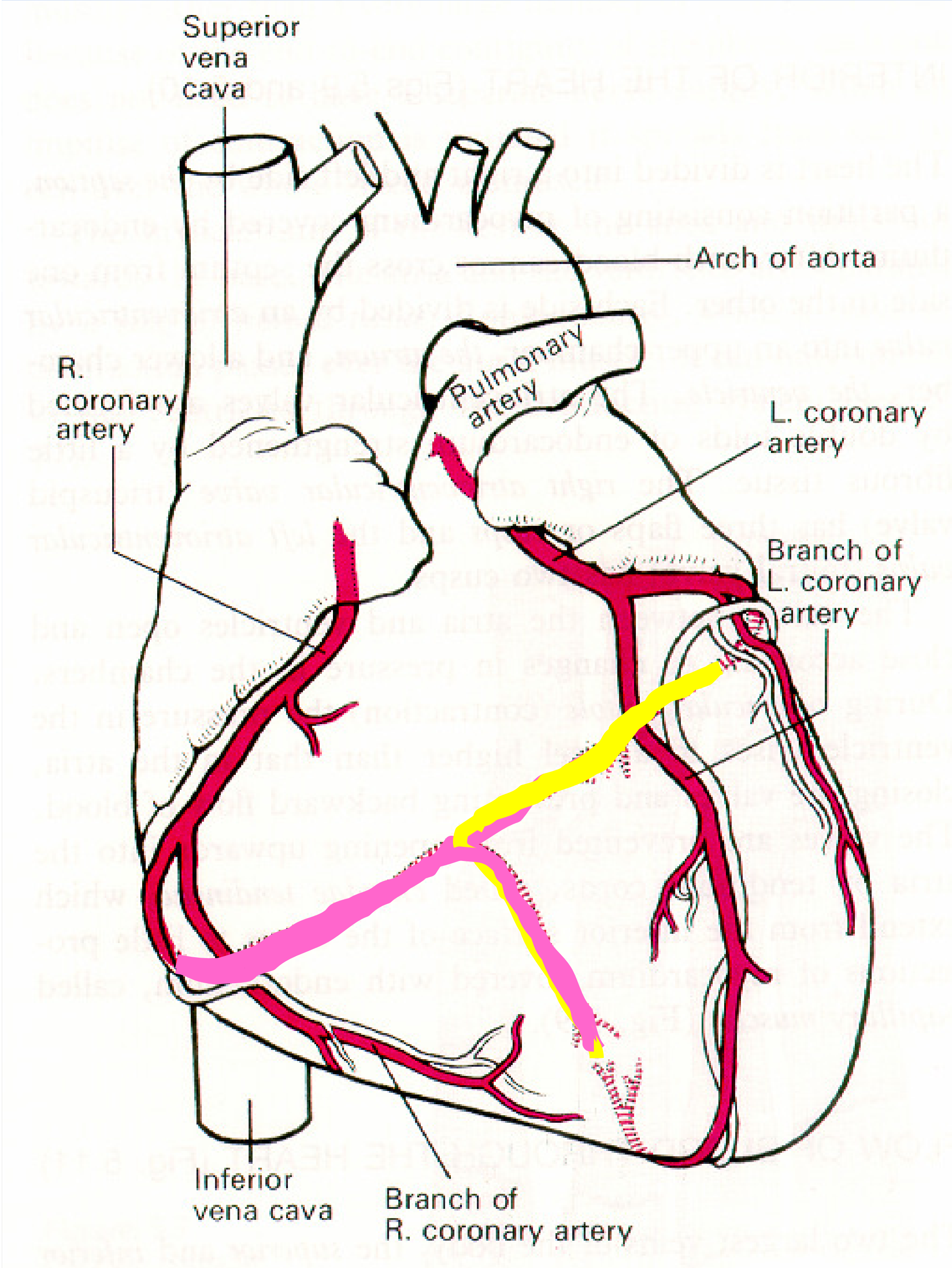


Coronary Artery Dominance

- Refers to blood supply to posterior and inferior wall of left ventricle
- Determined by the Posterior Descending (PDA) and Posterior Lateral (PLA) branches

Coronary Artery Dominance

- Right dominant 85%
  - RCA gives off PDA and major PLA branches
- Left dominant 8%
  - Circumflex gives off PDA and major PLA branches
- Balanced or Co-dominant 7%
  - RCA gives off PDA
  - Circumflex gives off major PLA branches

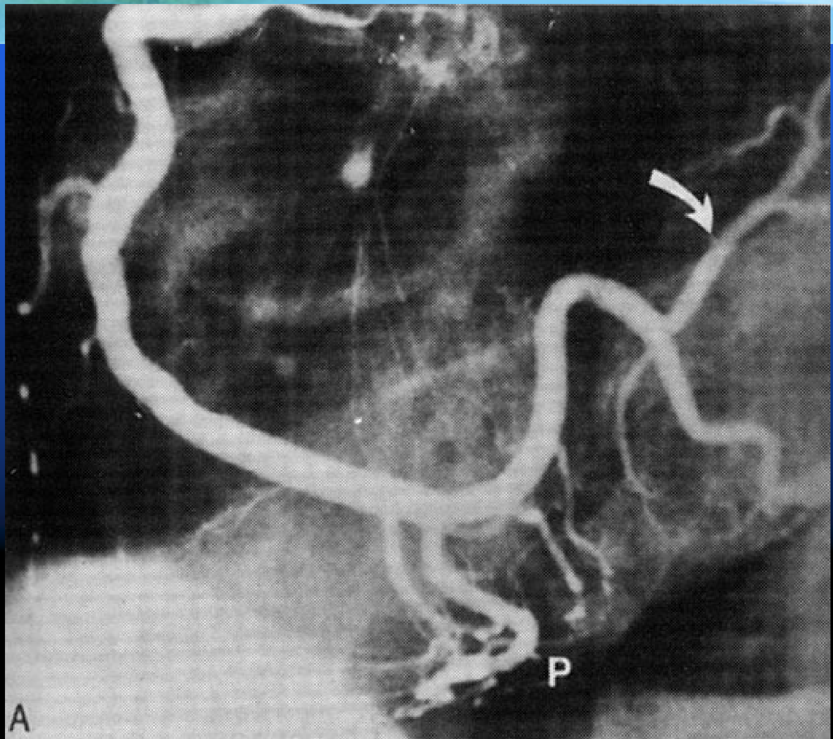


Right Dominant

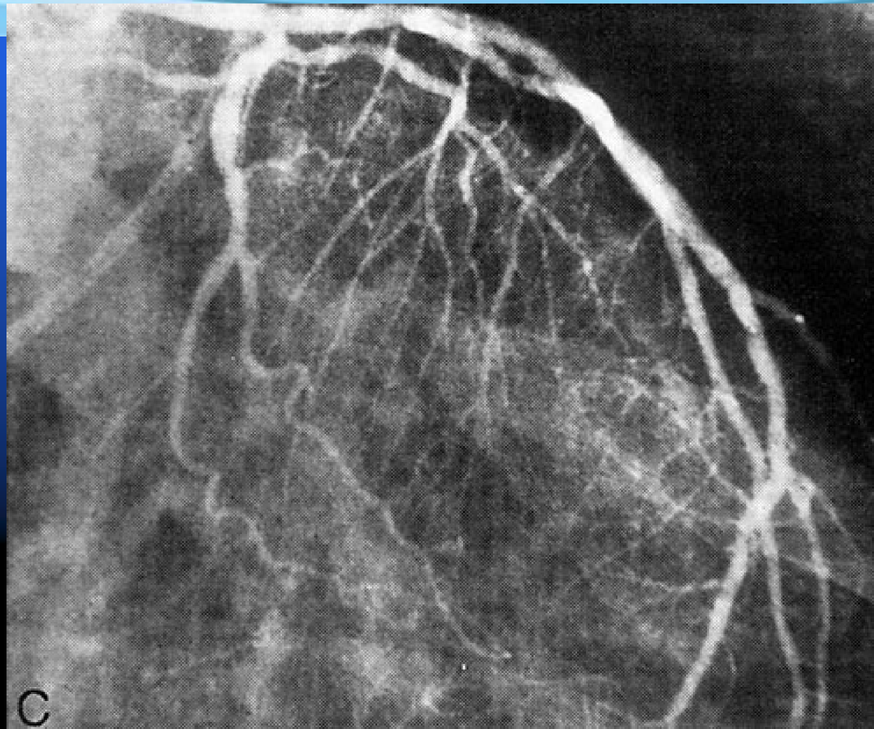
Left Dominant

Co Dominant

Right dominant

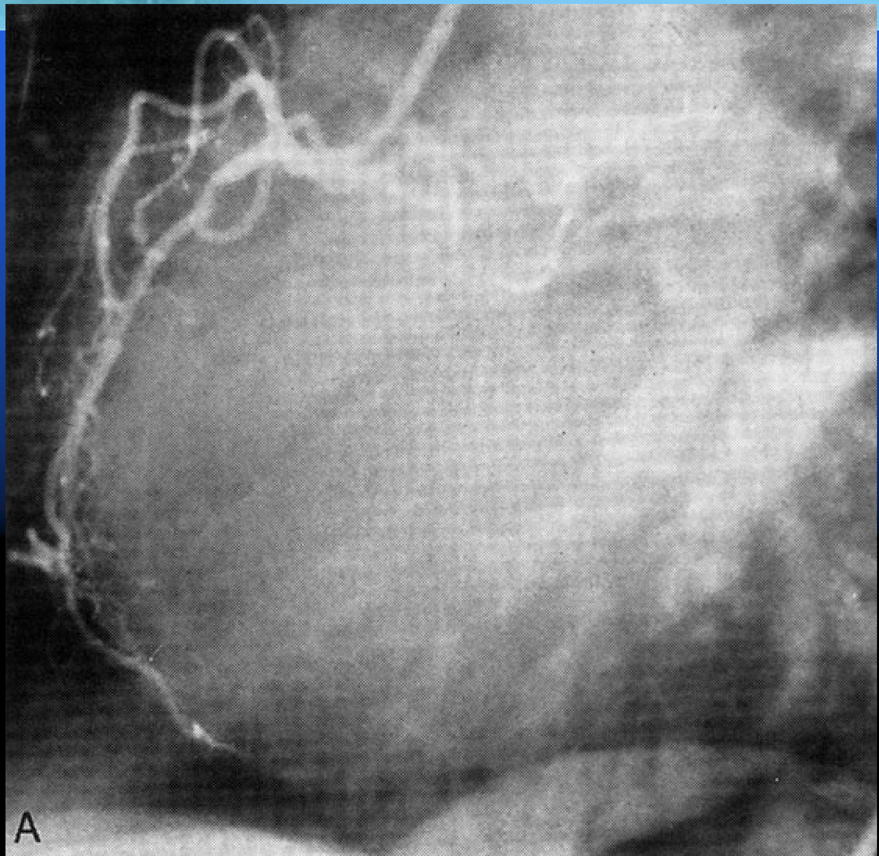


RCA

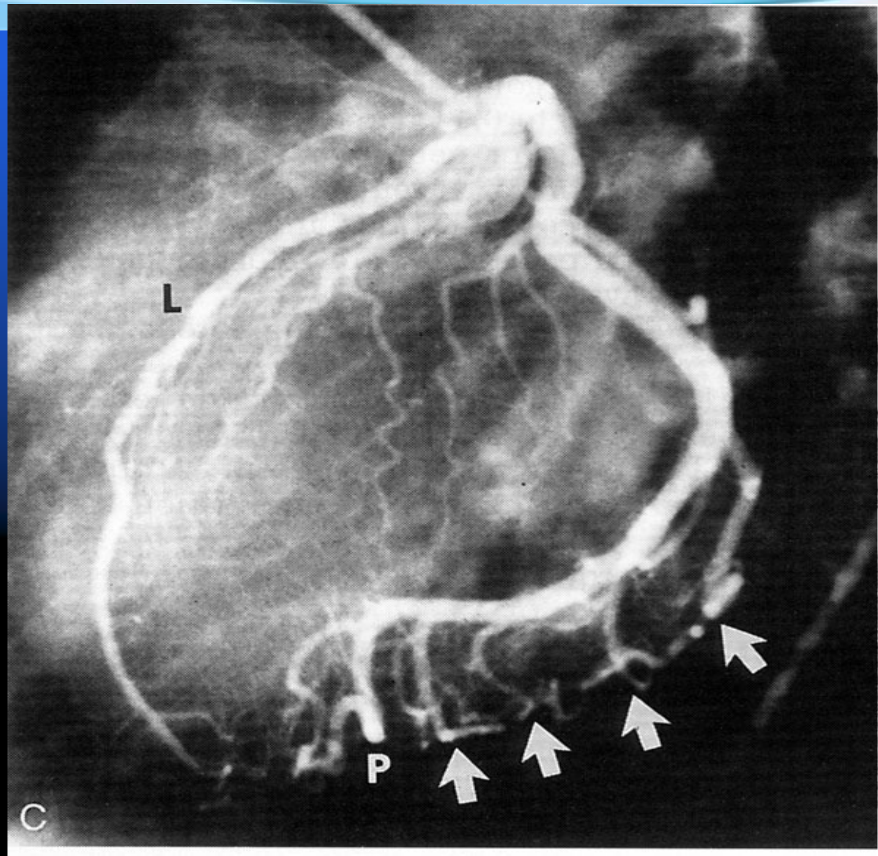


LCA

Left dominant

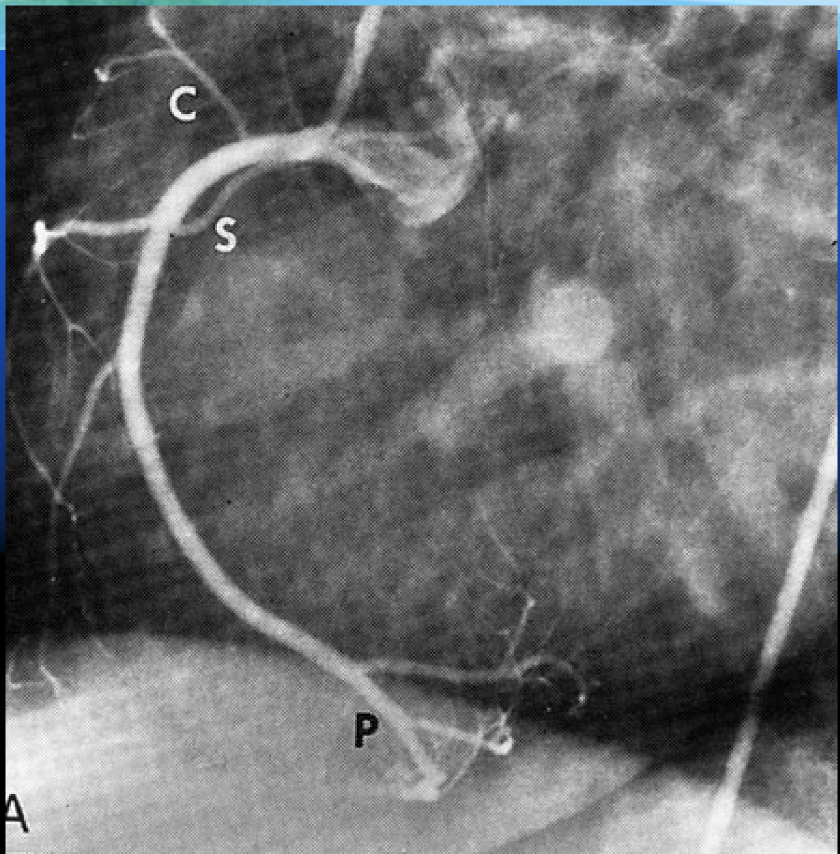


RCA

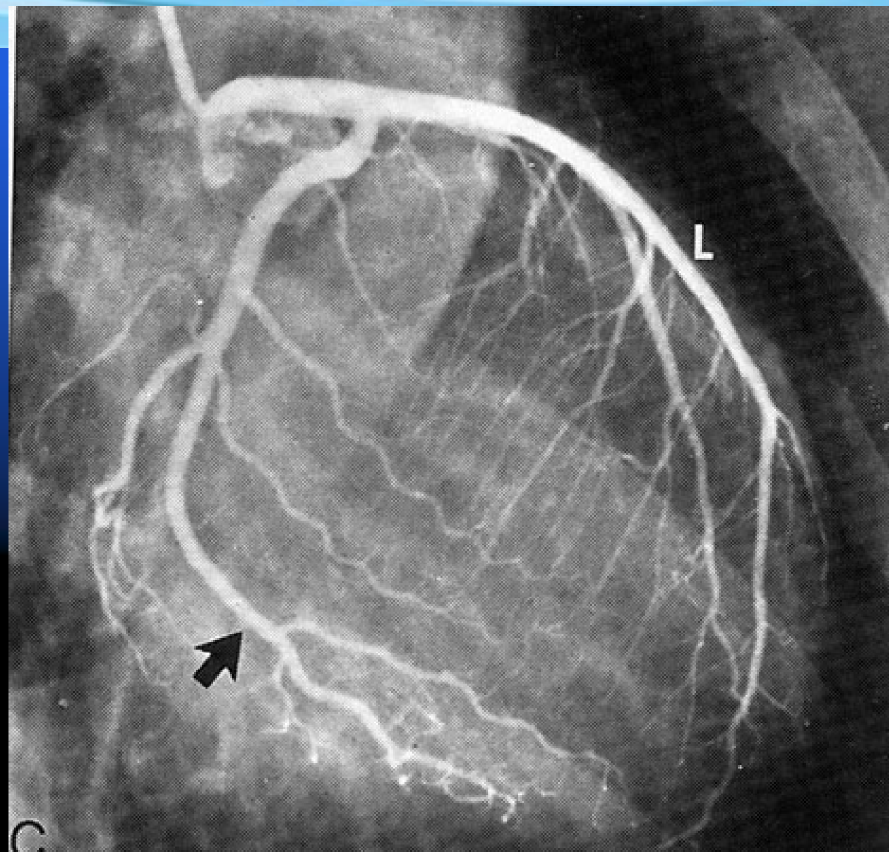


LCA

Co-dominant



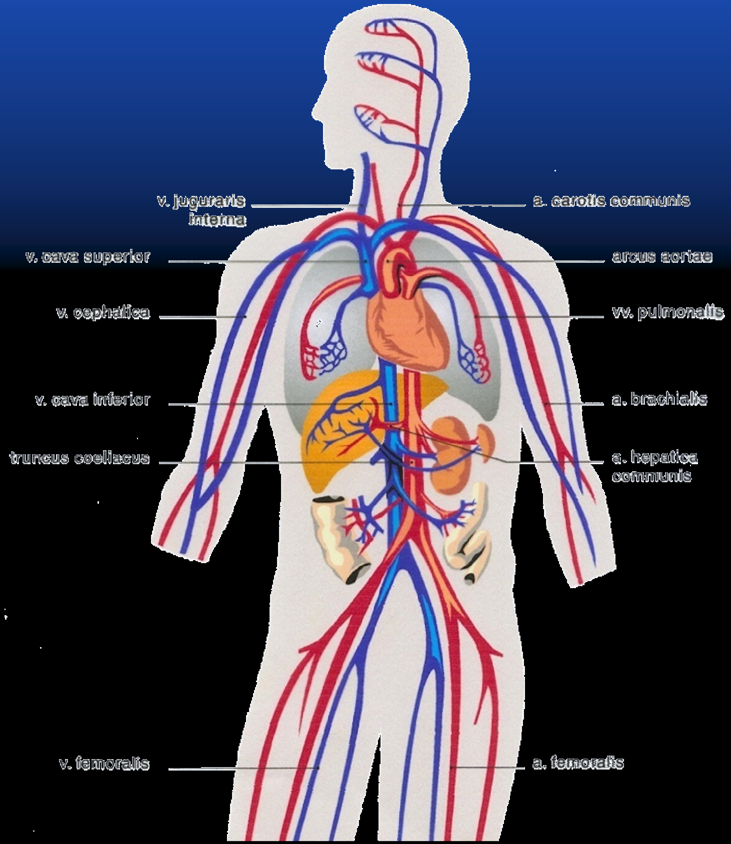
RCA

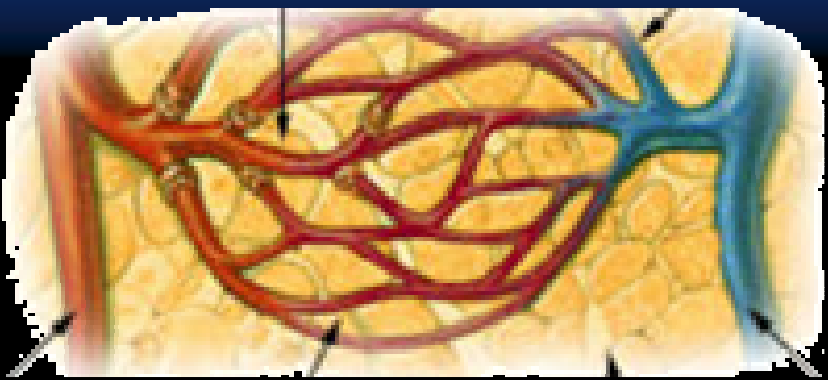


LCA

Hemodynamics

- the branch of physiology dealing with the forces involved in the circulation of the blood.





Cardiovascular Function

- Provide adequate tissue perfusion

What do we need for adequate perfusion ?

We need perfusion pressure

AND

We need volume (Cardiac Output).

Basic Hemodynamic Physic

BP = CO x SVR

CO = SV x HR

SV ~ EF / SVR and optimum EDP

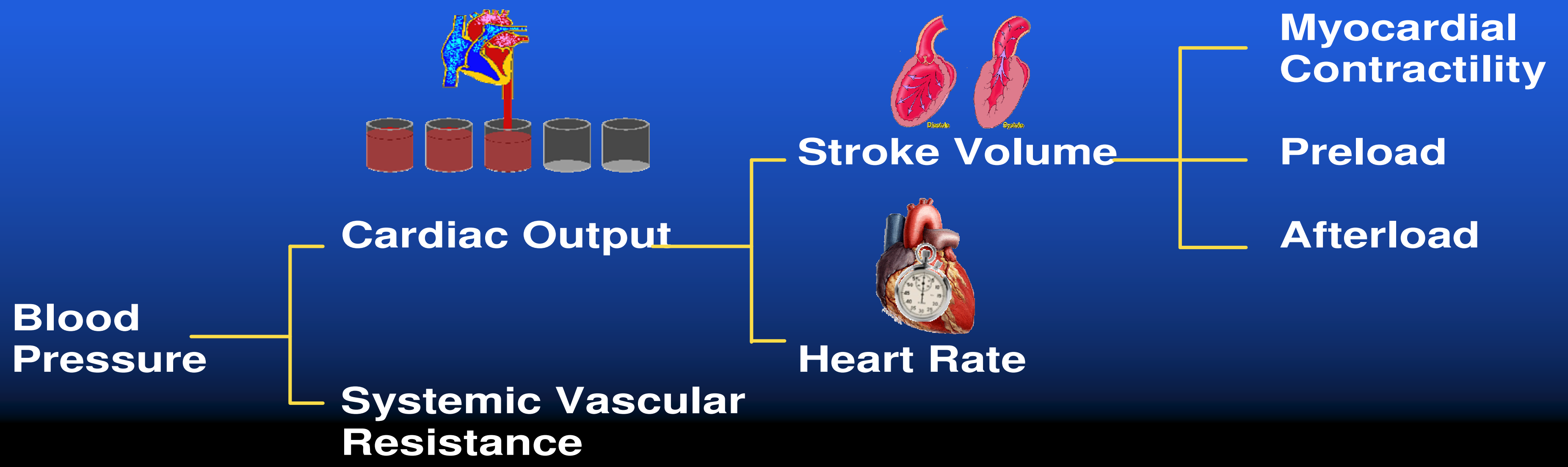
A few things to consider

- **Inadequate oxygenation**
  - Anemia
  - Hypoxemia
  - Decrease oxygen carrying capacity
- **Electrolyte and acid base disturbance**

What we want to know ?

- Adequate perfusion pressure.
  - Mean Arterial Pressure (MAP)
    - NBP
    - Arterial line
- Adequate cardiac output
  - Cardiac output measurement
    - Thermodilution
    - CCO (Continuous Cardiac Output monitoring)
    - Fick Cardiac Output

Basic Hemodynamic Physiology



- BP = CO x SVR
- CO = SV x HR
- SV ~ EF

  ~ 1/SVR

  ~ optimum EDP

BP = blood pressure
CO = cardiac output
SVR = vascular resistant
SV = stroke volume
HR = heart rate
EF = ejection fraction
EDP = end diastolic pressure

Hemodynamic Monitoring

Noninvasive Hemodynamic Monitoring:

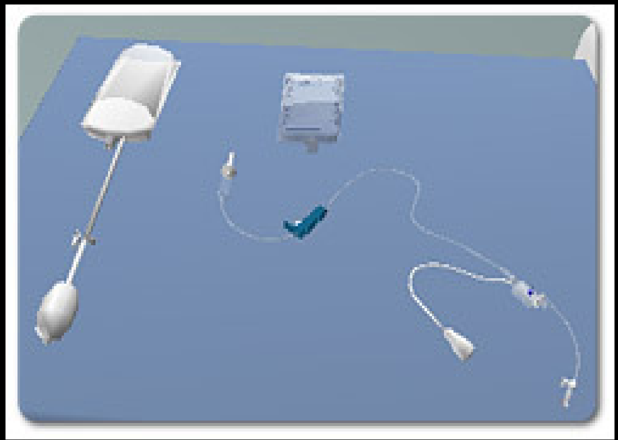
- Pulse Rate and quality
- Blood Pressure
- Skin temperature/color
- Capillary Refill
- Mentation
- Urine output

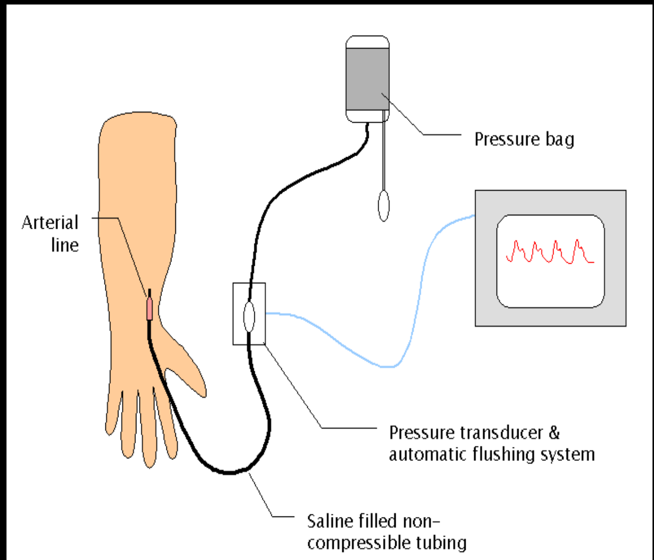
Invasive Hemodynamic Monitoring:

- Arterial catheter
- Central venous pressure
- PA catheter and CO monitoring

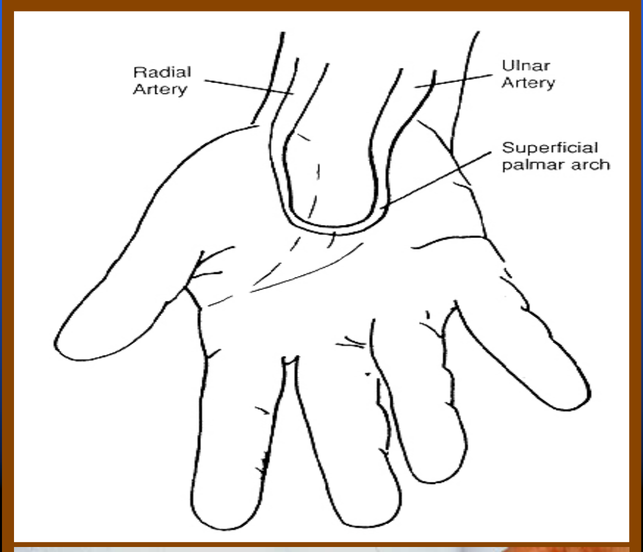
Arterial Line

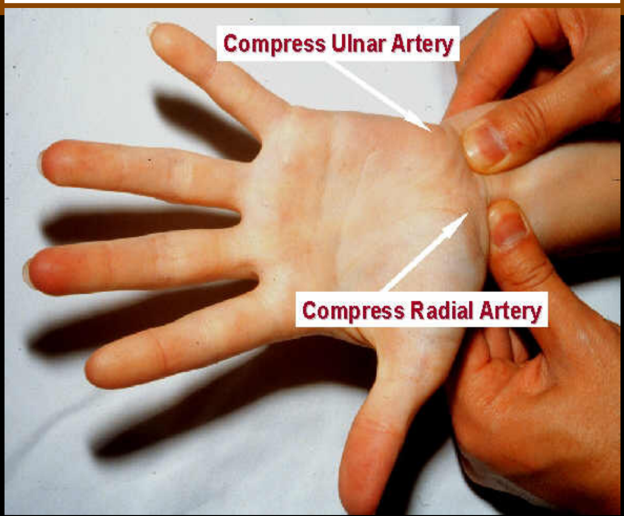
- Direct measurement of blood pressure
- most accurate technique
- continuous hemodynamic information
- blood gas measurement



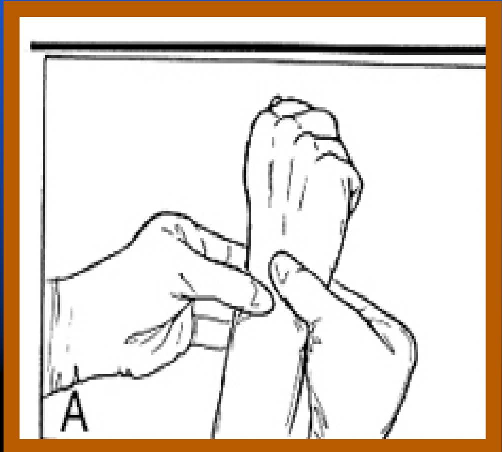


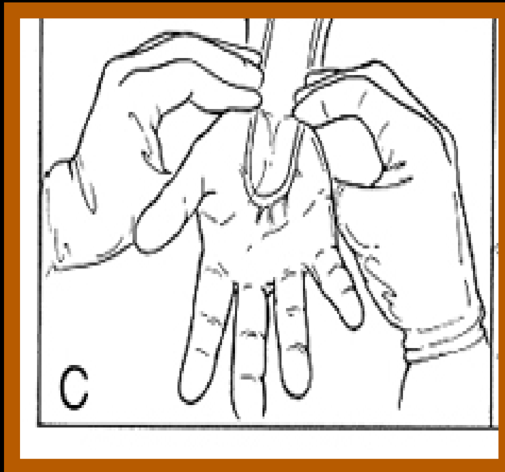
Radial Cannulation Anatomy

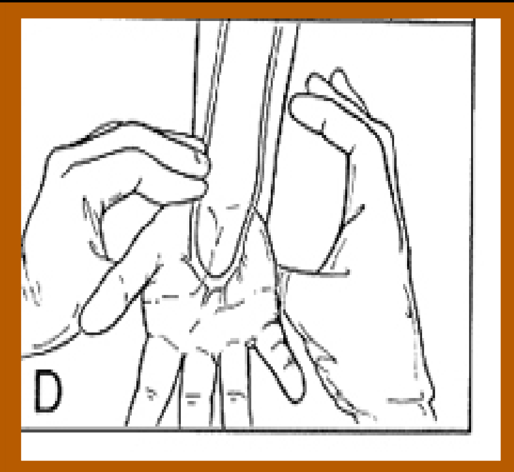




Allen's Test







Werner Forssmann

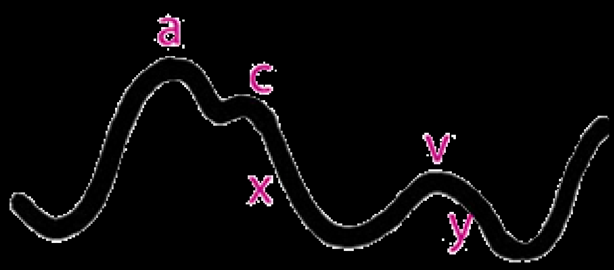
- **The Nobel Prize in Physiology or Medicine 1956**

1929

- "...develop a technique for the catheterization of the heart. This he did by inserting a cannula into his own antecubital vein, through which he passed a catheter for 65 cm and then walked to the X-ray department, where a photograph was taken of the catheter lying in his right auricle." -The Nobel Foundation 1956

Central Venous Pressure

- Central venous pressure (CVP) describes the pressure of blood in the thoracic vena cava, near the right atrium of the heart.
- CVP reflects the amount of blood returning to the heart and the ability of the heart to pump the blood into the arterial system.
- Normal Value: 2-6 mmHg



Central Venous Trace

What is Central Venous Pressure (CVP)?

Pressure in the thoracic vena cava

↓

Estimates right atrial pressure

↓

Estimates right ventricular preload

↓

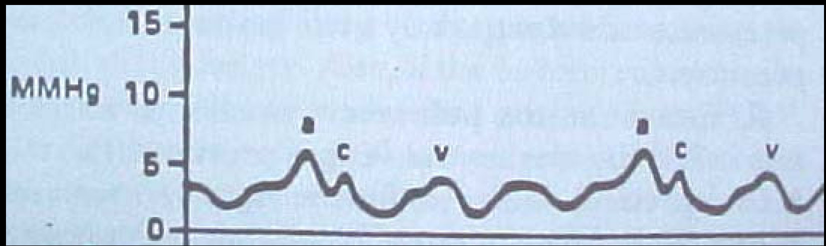
Estimates left atrial pressure

↓

Estimates left ventricular preload

CVP Values

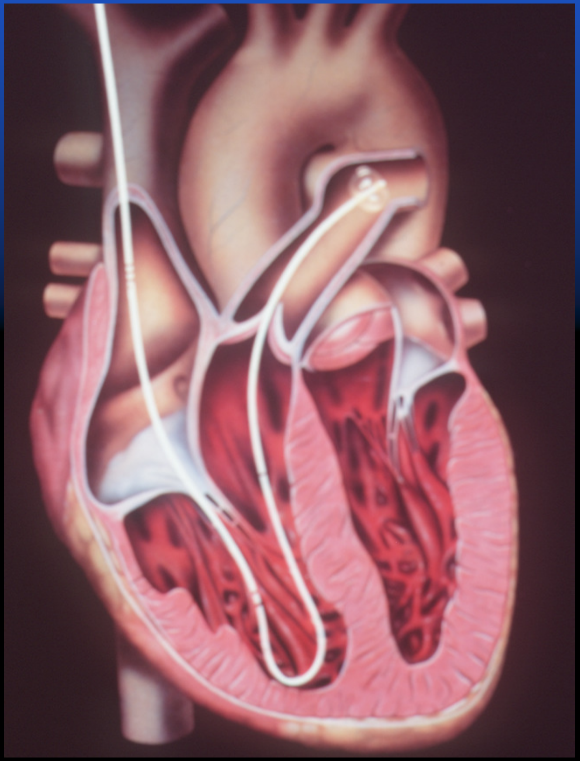
- Low CVP (<8 cmH_2O)
  - Low preload
  - Negative pressure inspiration
- High CVP (>12 cmH_2O)
  - High preload e.g. volume overload, heart failure
  - Tricuspid and/or mitral disease (both stenotic and regurgitation)
  - Right heart disease
  - Pulmonary Hypertension
  - Pericardial disease
  - Increased intrathoracic pressure



PULMONARY ARTERY CATHETER

The purpose of this catheter is to:

- Measure the patient's hemodynamic status and evaluate the hemodynamic treatments.



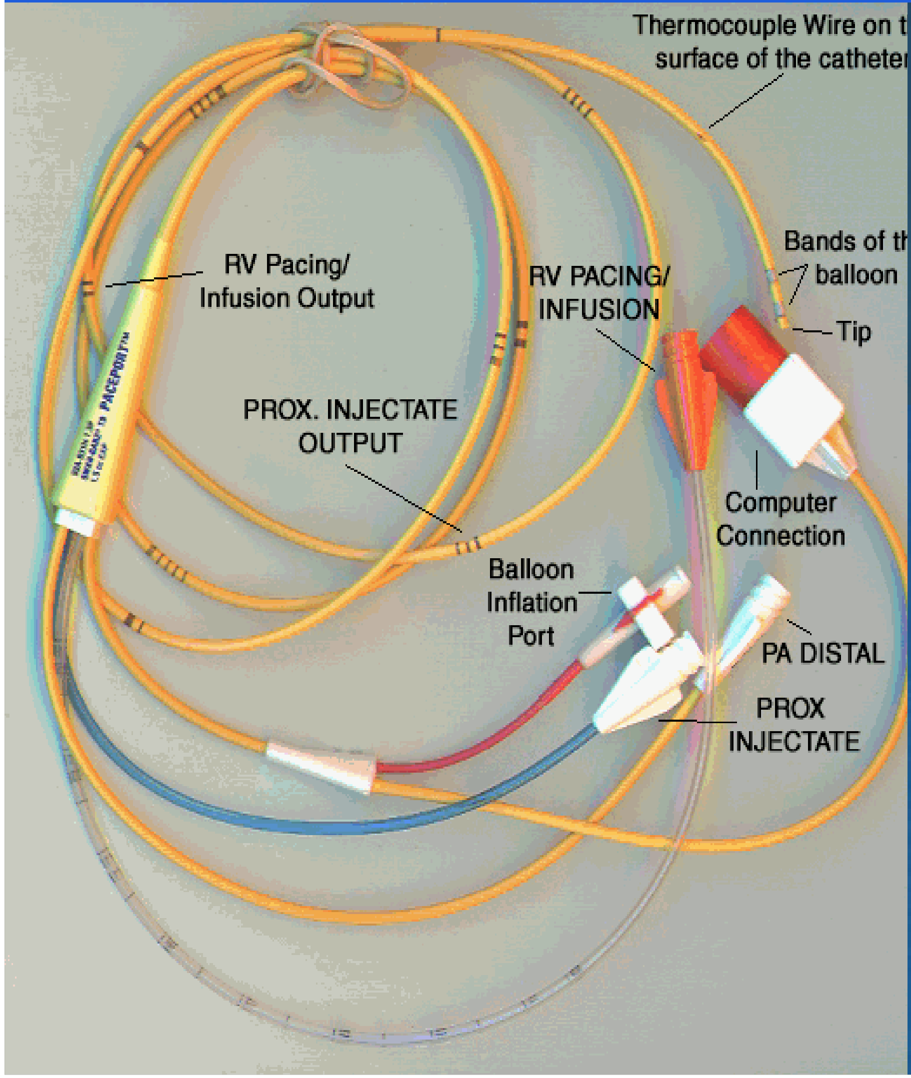
What is being measure ?

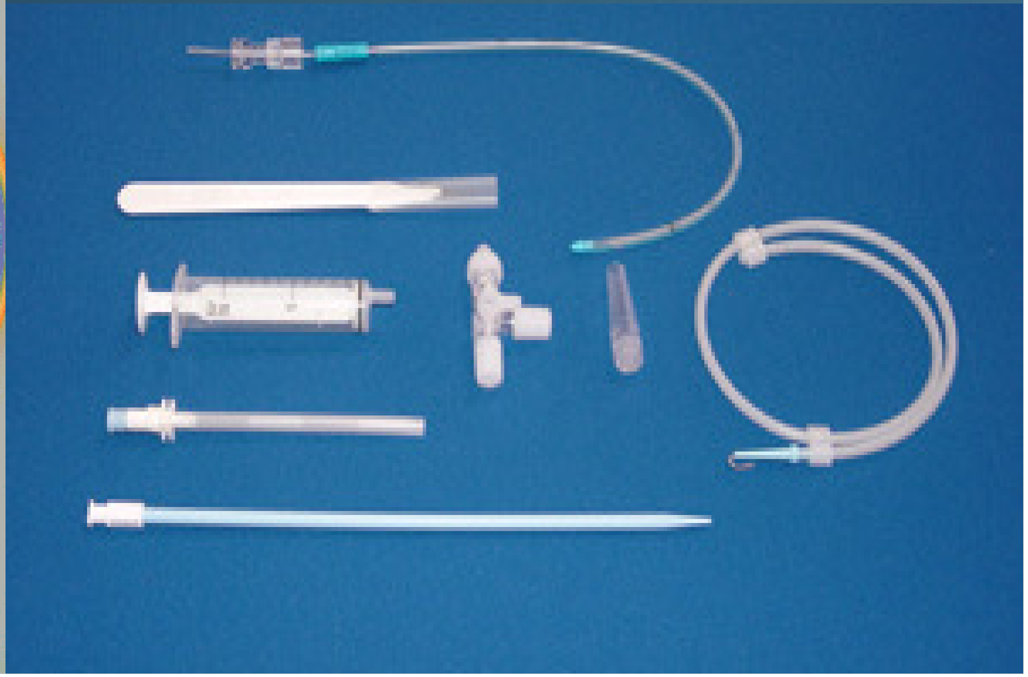
- Central venous pressure
- Pulmonary artery pressure
- Pulmonary capillary wedge pressure
- Cardiac output

And also:

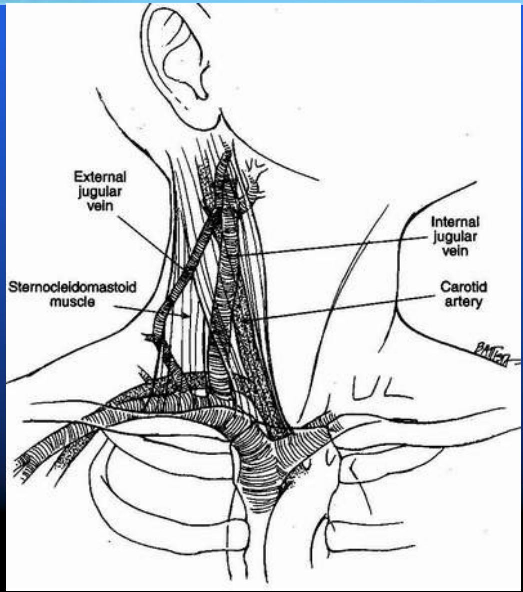
- Mixed venous oxygen saturation
- Derived data such as stroke volume and vascular resistant

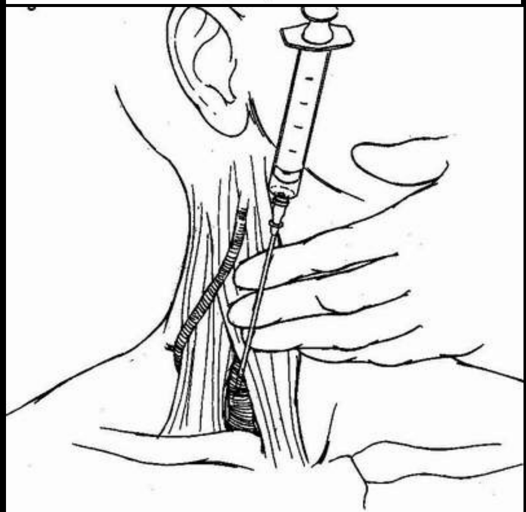
Equipments


Thermocouple Wire on t
surface of the catheter
RV Pacing/
Infusion Output
RV PACING/
INFUSION
Bands of th
balloon
Tip
PROX. INJECTATE
OUTPUT
Computer
Connection
Balloon
Inflation
Port
PA DISTAL
PROX
INJECTATE



Technique





- Supine or Trendelenburg with face turn toward left side
- Modified Seldinger
- Point needle 45° outward from midline and 45° tilt up.
- Preferable 8Fr vascular sheath but 7Fr is acceptable.

PA Insertion Waves

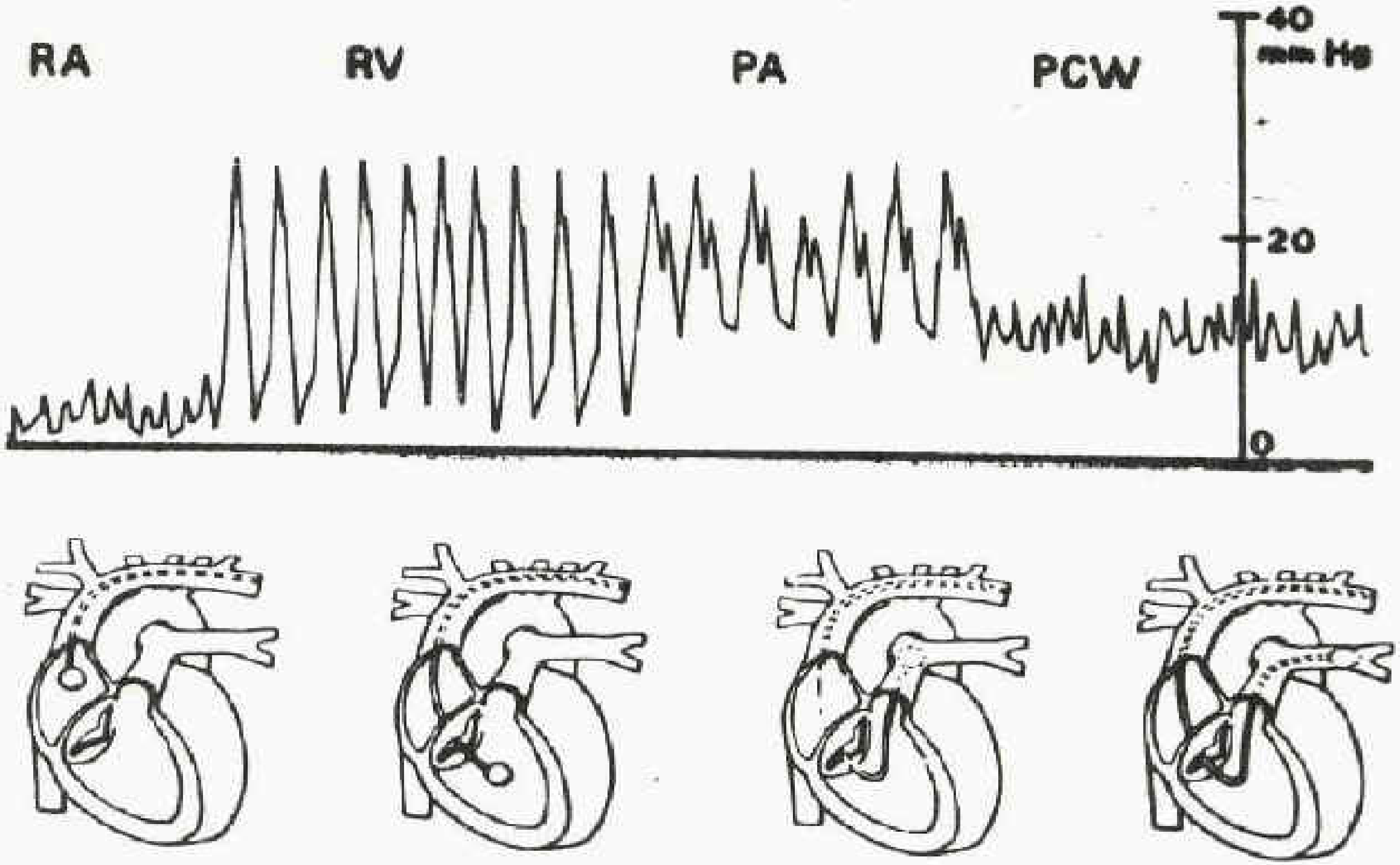
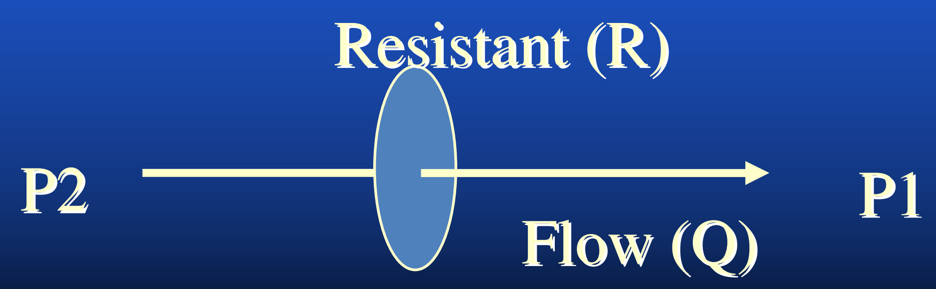


Fig. 1. Characteristic intracardiac pressure wave forms during passage through the heart

Hemodynamic data

- Pressure data:
  - CVP, PA pressure, PCWP
- Direct hemodynamic data
  - CO, SVO_2
- Calculated hemodynamic data
  - SVR, PVR



$$(P2-P1) = Q \times R$$

R	= SVR (systemic vascular resistant)
Q	= CO (Cardiac output)
P2	= MAP (mean arterial pressure)
P1	= CVP (central venous pressure)

Calculate hemodynamic data

- **Cardiac Index (CI)**
  - = CO (l/min) / BSA (m^2)
- **Stroke volume Index (SVI)**
  - = CI / HR
- **Systemic vascular resistant (SVR)**
  - = (MAP – CVP) x 80 / CO
- **Systemic vascular resistance index (SVRI)**
  - = (MAP – CVP) x 80 / CI
- **Pulmonary vascular resistant (PVR)**
  - = (MPAP – PCWP) x 80 / CO
- **Pulmonary vascular resistance index (PVRI)**
  - = (MPAP – PCWP) x 80 / CI

Normal Value

CVP	1-6	mmHg
PCWP	6-12	mmHg
CO	4-6	l/min
CI	2.4-4.0	$l/min/m^2$
SVI	40-70	$ml/beat/m^2$
SVR	950-1500	$dynes.sec/cm^{-5}$
SVRI	1600-2400	$dynes.sec.m^2/cm^{-5}$
PVR	120-240	$dynes.sec/cm^{-5}$
PVRI	200-400	$dynes.sec.m^2/cm^{-5}$

Hemodynamic profiles in shock

Hypovolemic	Cardiogenic	Vasogenic
low PCWP	high PCWP	low PCWP
low CI	low CI	high CI
high SVRI	high SVRI	low SVRI

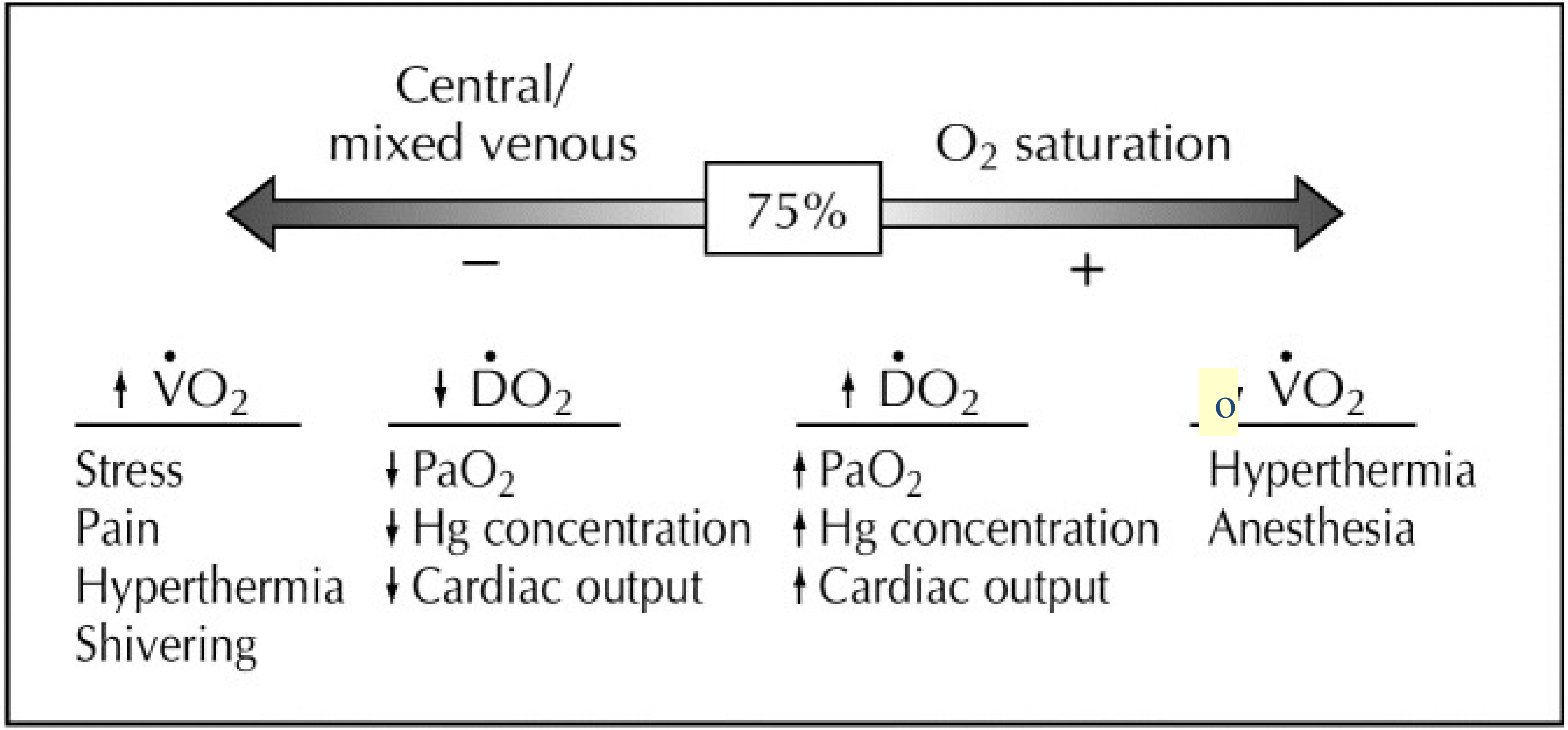
Cardiogenic shock

CI < 2.2 $l/min/m^2$
PCWP > 18 mmHg
MAP < 65 mmHg
SVR > 1800 $dynes.sec/cm^{-5}$

Mixed Venous O_2 Saturation (SvO_2)

- Oxygen Saturation of Venous hemoglobin in the central vein (Pulmonary artery)
- Amount of oxygen left over after the body removes what it needs
- Represents the balance between oxygen delivery and consumption
- Normal is 75 %

SvO_2



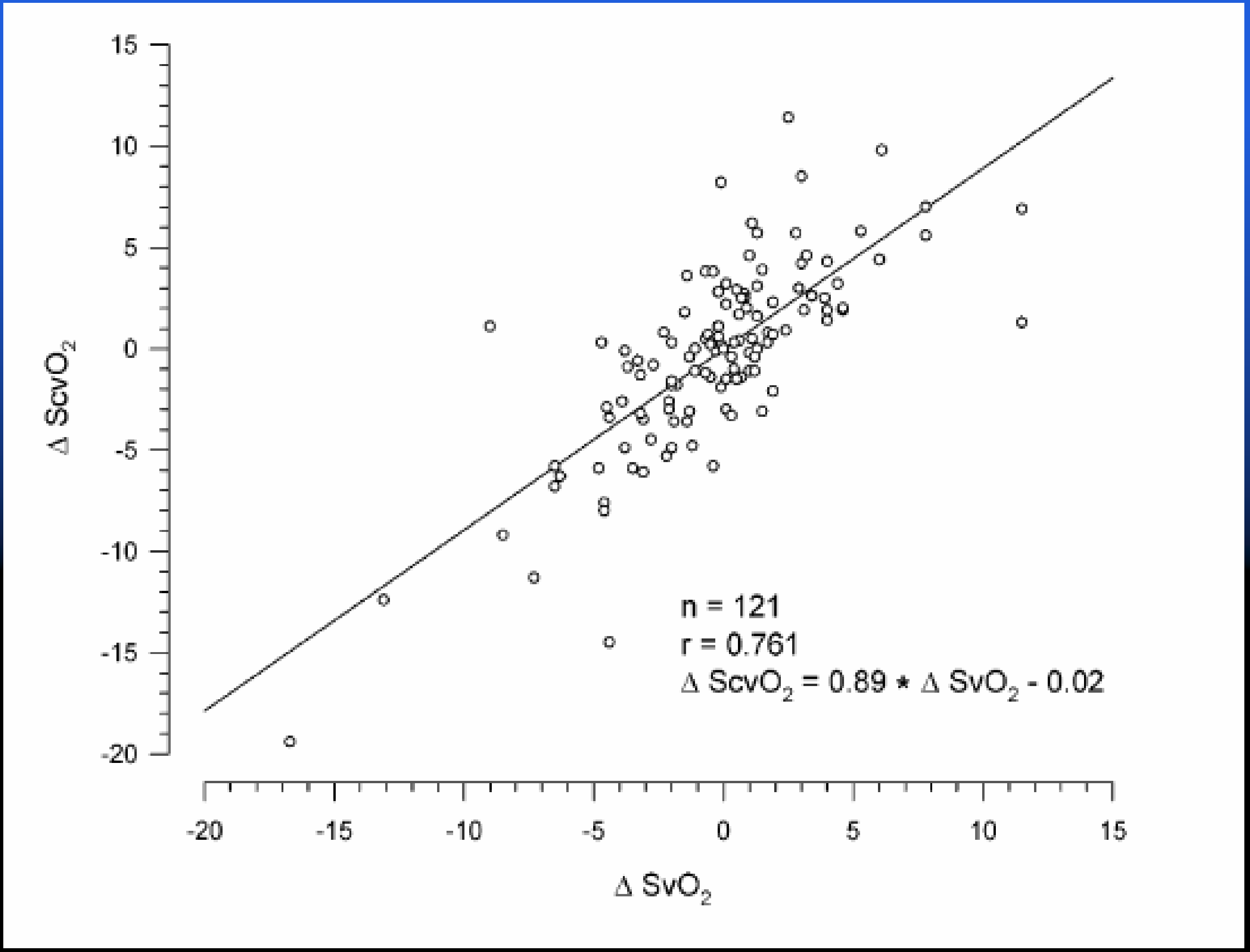
Rivers et al, 2001

SvO_2 and $ScvO_2$

- SvO_2 reflex cardiac output if arterial oxygen saturation and oxygen consumption remain the same
- $ScvO_2$ has good correlation with SvO_2 and can be used to represent SvO_2
- **Thinks to remember**
  - In septic shock: oxygen consumption can be lower than normal due to inabilities to use oxygen from cellular dysfunction and toxin
  - So high SvO_2 in septic shock does not necessary mean high CO but implies adequate CO to provide O_2 to tissue at that time

Comparison of venous O_2 saturation in pulmonary artery and in Superior vena cava

Δ $ScvO_2$



Δ SvO_2

Reinhart et al. Intensive Care Med (2004) 30:1572–1578

Physiological Truth

- There is no such thing as a
  "Normal Cardiac Output"
- Cardiac output is either
  - Adequate to meet the metabolic demands
  - Inadequate to meet the metabolic demands
- All Hypotension are disease
  - Impair cardiac output
  - Abnormal systemic resistance
  - Or BOTH

Pressure Volume Relation or loop

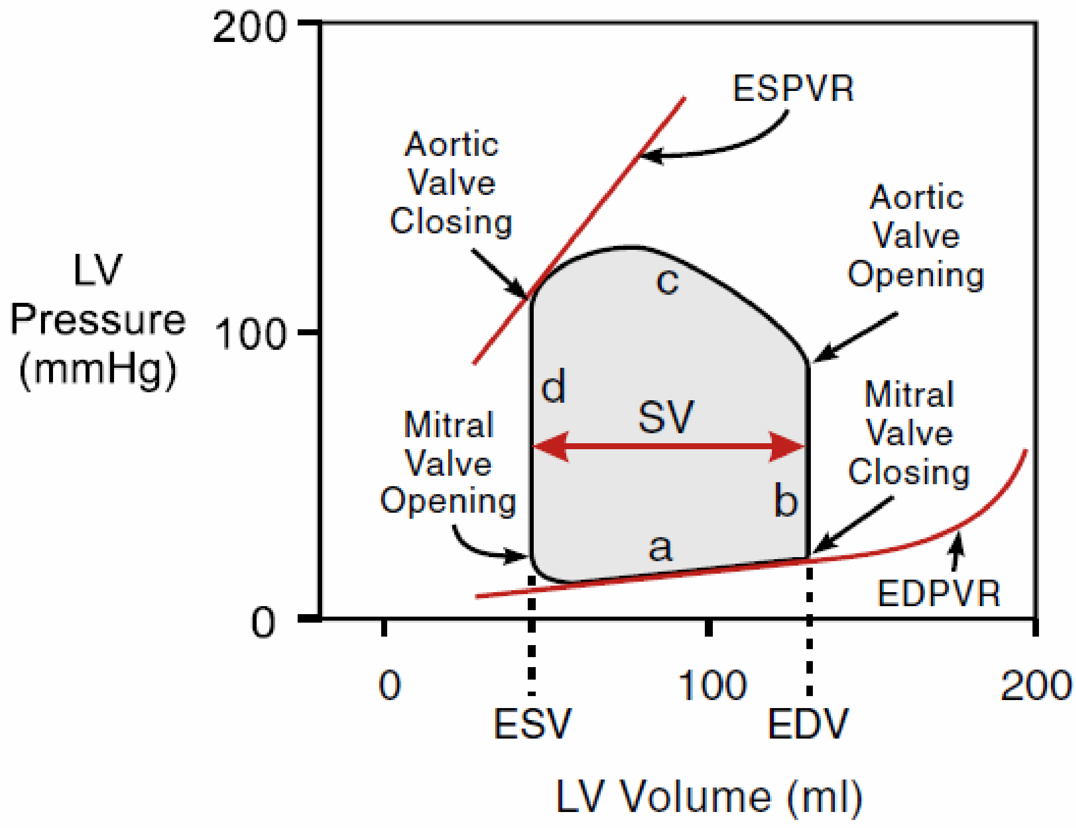
ESPVR- End-systolic pressure-volume relationship

EDPVR- End-diastolic pressure-volume relationship

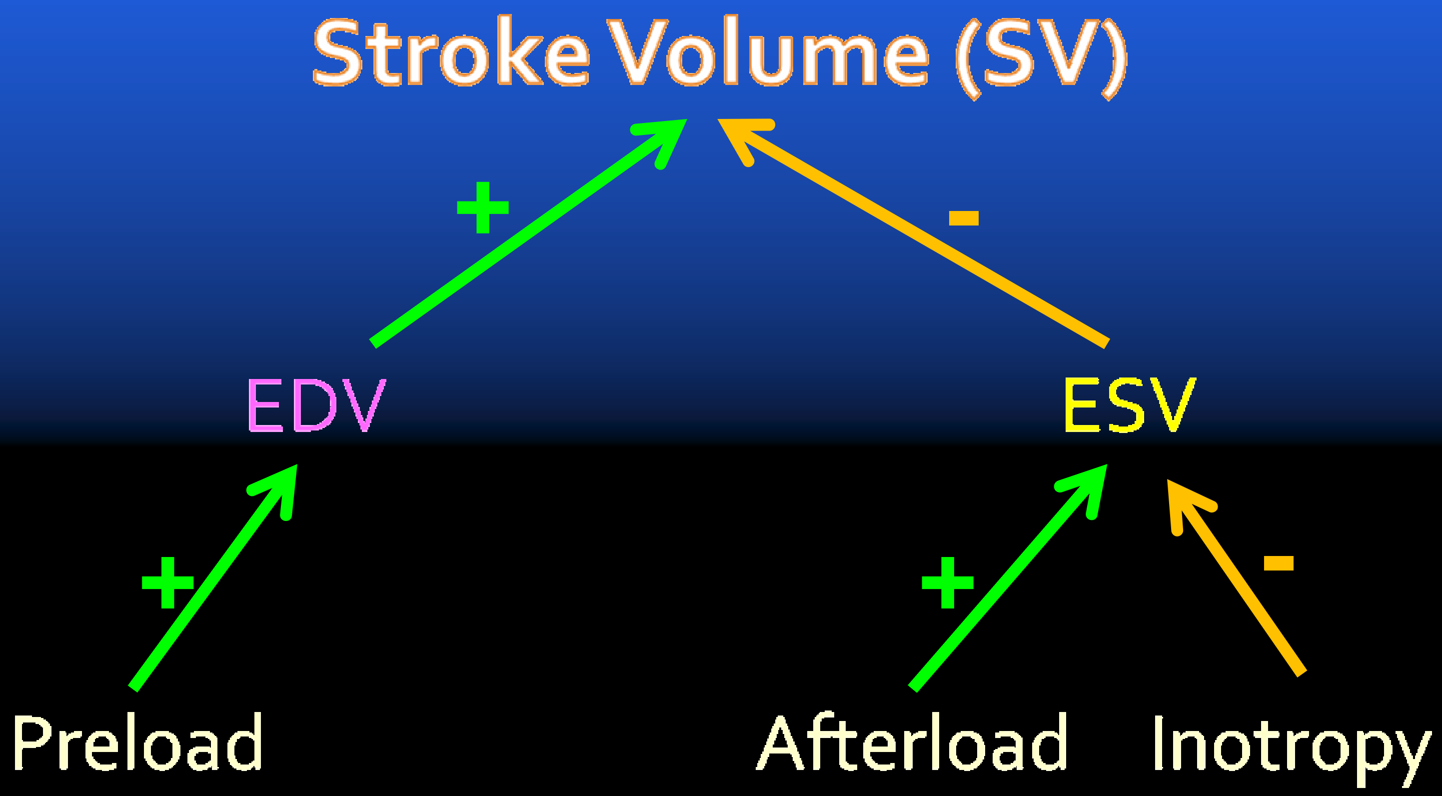
ESV- End-systolic volume

EDV- End-diastolic volume

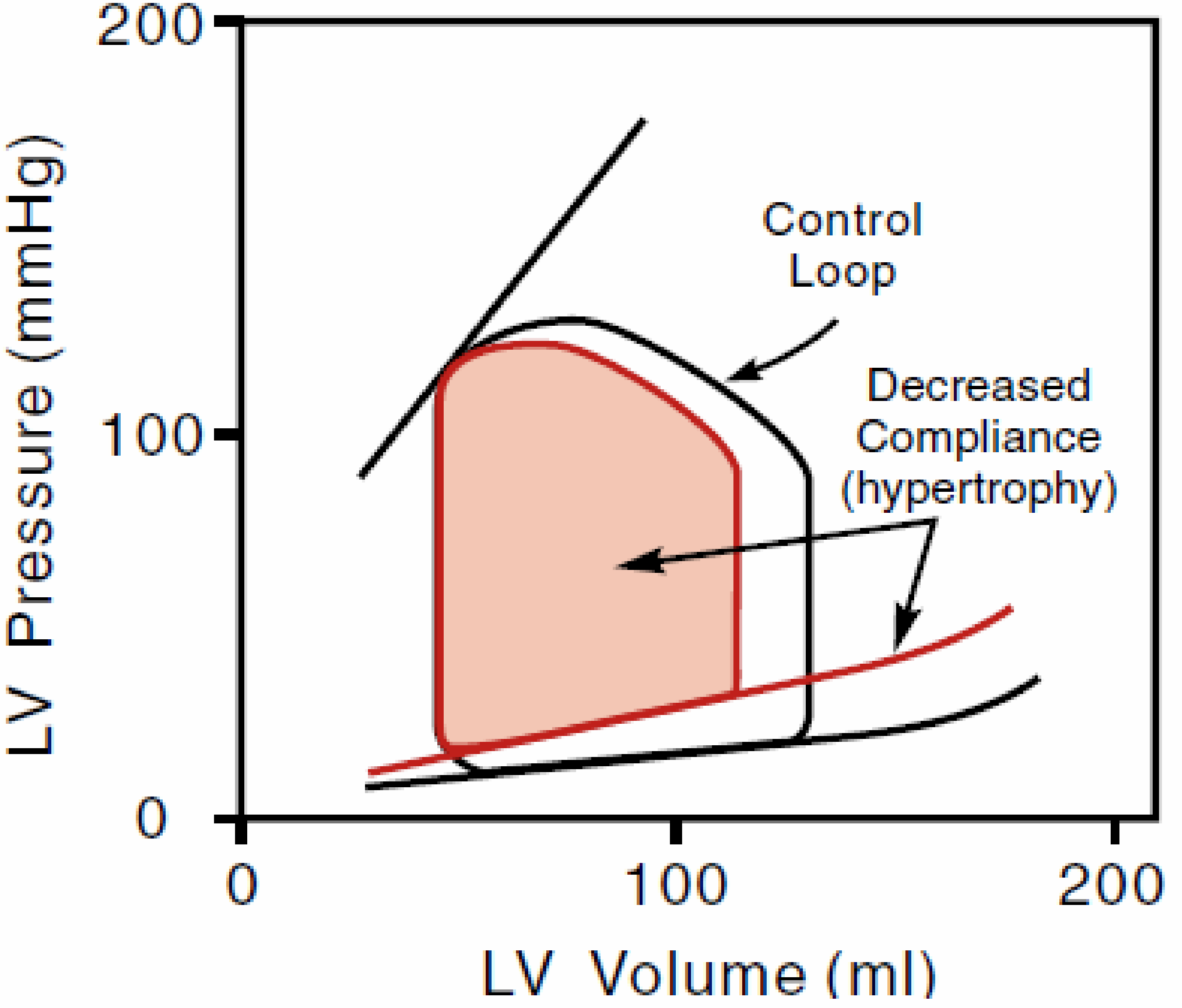
SV- Stroke volume



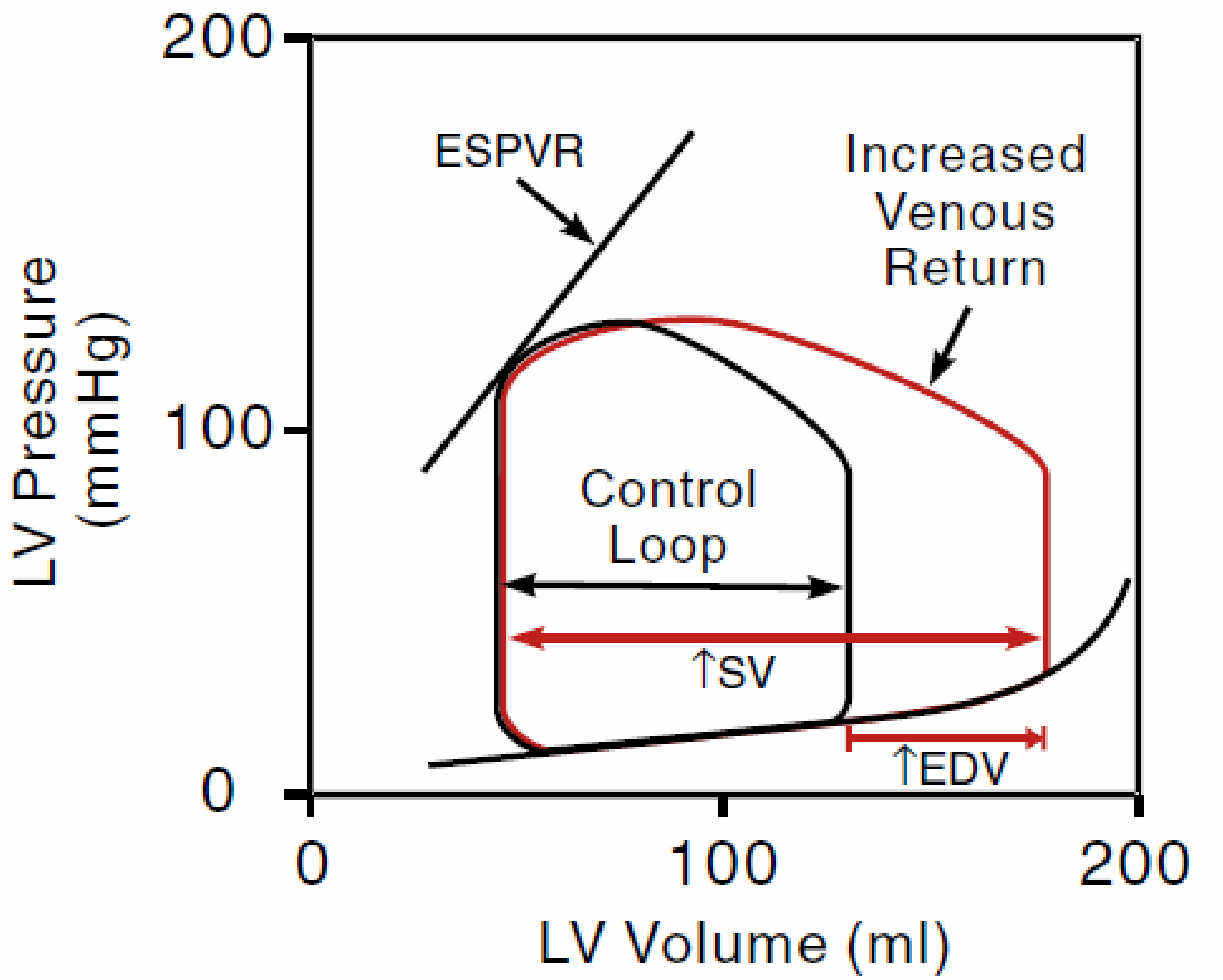
Effect of preload, afterload and contraction



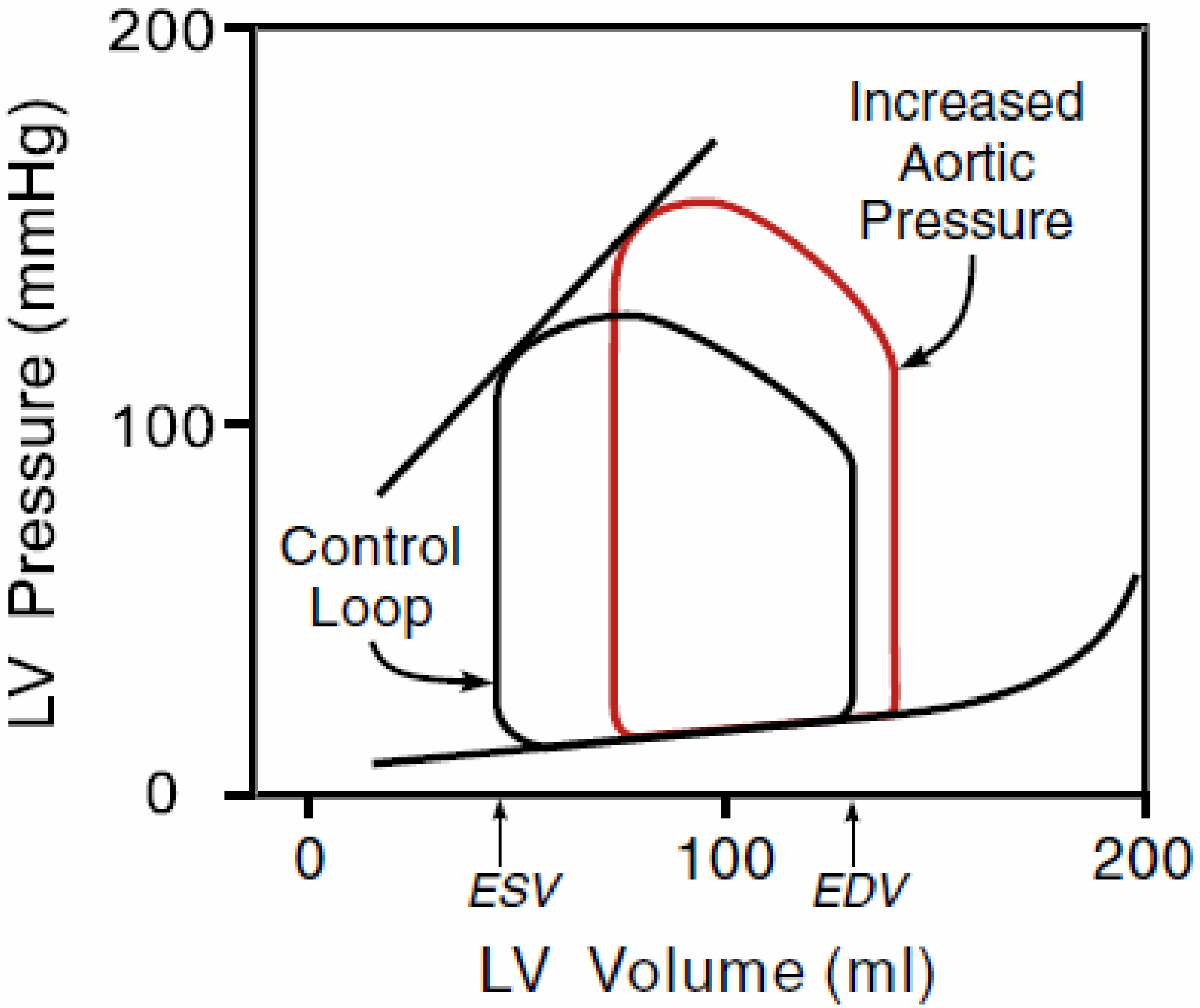
Pressure-Volume Loop in Hypertrophy



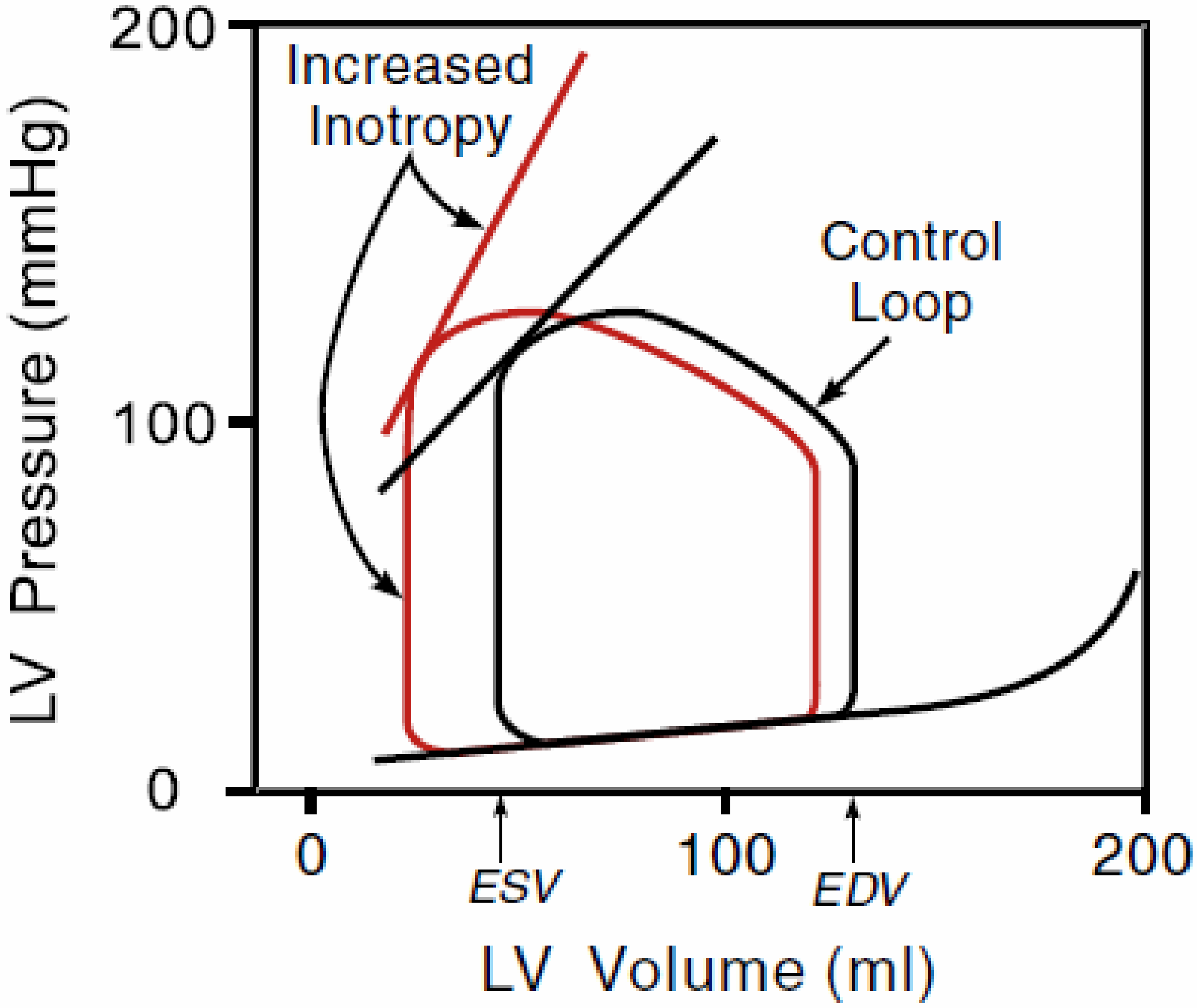
Pressure-Volume Loop in increase Preload



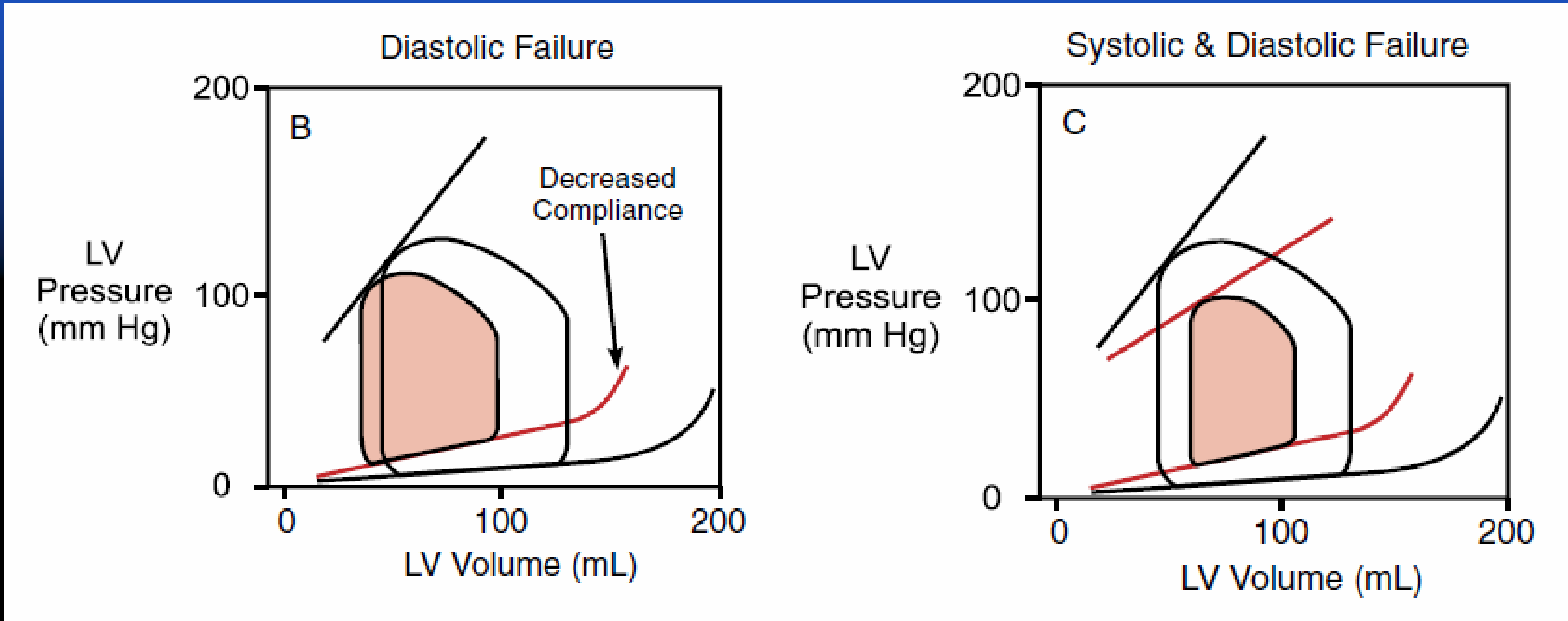
Pressure-Volume Loop in Afterload



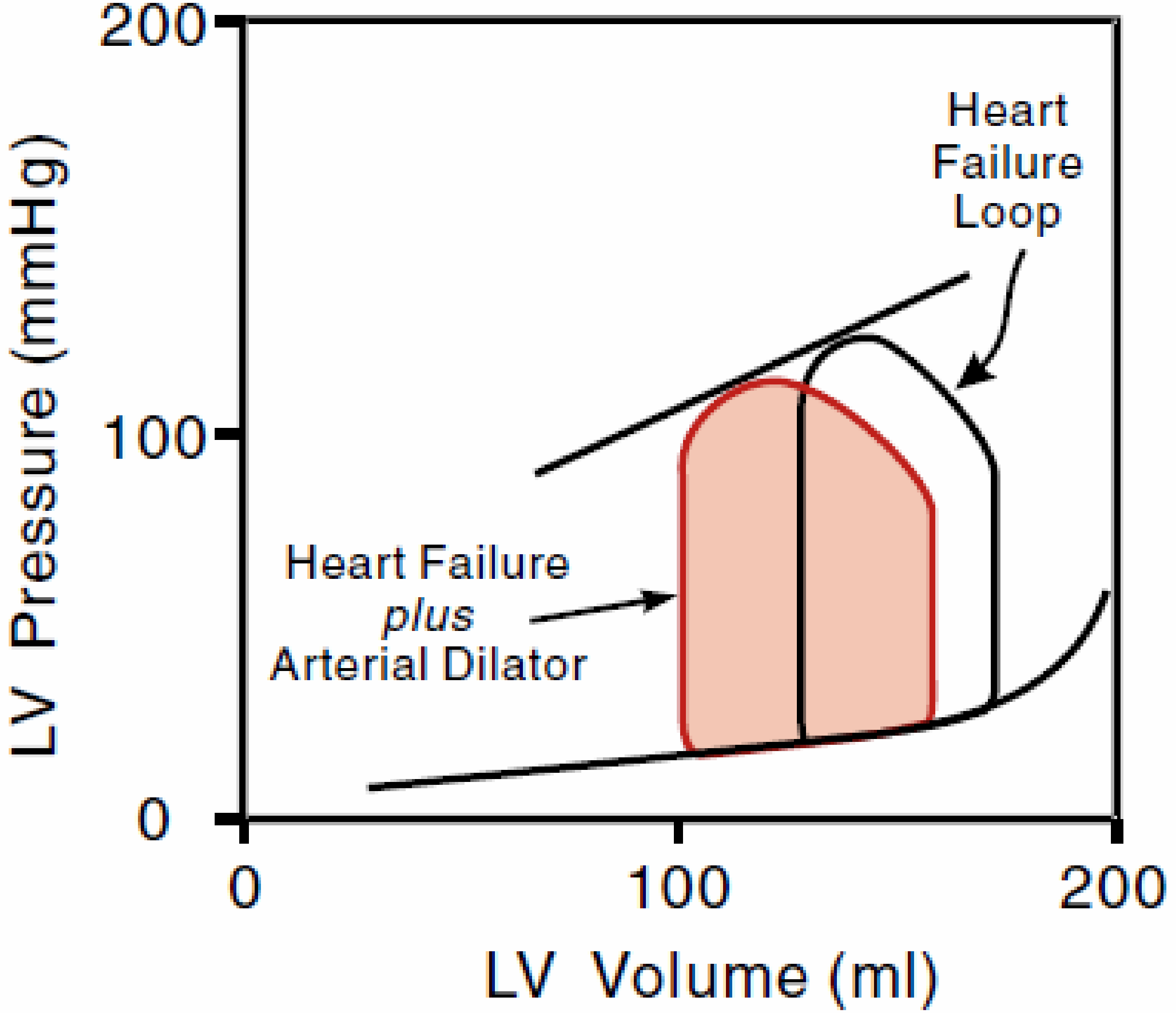
Pressure-Volume Loop in increase inotropy



Pressure-Volume Loop in Heart Failure



Pressure-Volume Loop in CHF with vasodilator



Effects of Inotropic Agents and Vasodilators

Drug	Receptor	CO	SVR	Dose Range
Epinephrine	α_1 ,β_1, (β_2)	↑ ↑	↑	0.01 – 0.5
Norepinephrine	α_1, β_1	0 - ↑	↑ ↑ ↑	0.01 – 0.5
Dopamine	β_1, DR, (α)	↑	↑	2 -15 (20)
Dobutamine	β_1, β_2	↑ ↑	↑	2 – 15 (20)
Dopexamine	β_1, β_2, DR	↑ ↑	0 - ↑	0.9 - 5
Vasopressin	Angiotensin III	0 - ↓	↑ ↑ ↑	5 - 20
Amrinone	PDI	↑ ↑	↓ ↓	5 -10

(µg/kg/min)

Effects of Inotropic Agents and Vasodilators

Drug	CO	SVR	Dose Range
Nifedipine	0 - ↑	↓	0.5 - 10
Nitroglycerin	0 - ↑	↓	3 - 5
Nitroprusside	0 - ↑	↓	0.5 - 5
Prostacyclin	↑	↓	10 - 40

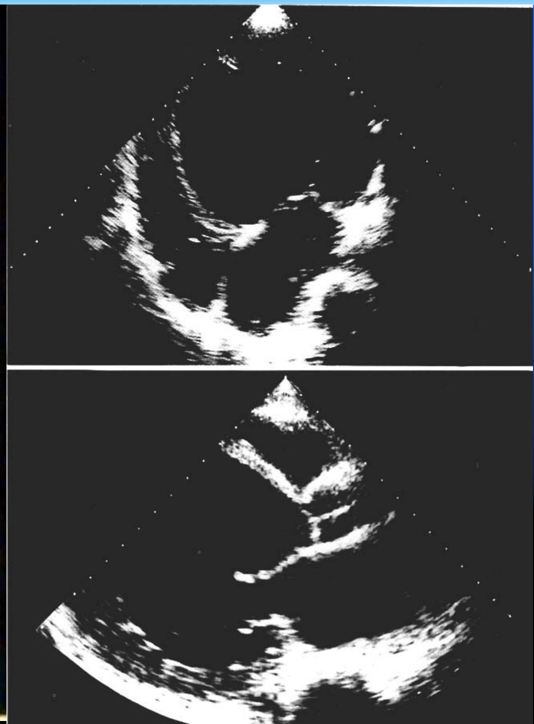
(µg/kg/min)

Real Patient

- The 56 years old man dilated cardiomyopathy presented with epigastric discomfort, drowsiness and confusion. He stopped taking all of his medications for 1 week because he ran out of his medication.
- PE showed poor perfusion with BP 70/50 HR 110 JVP up to ear at upright position, cardiomegaly with S3 and MR, pulmonary and hepatic congestion and bilateral lower extremities edema.

Investigation

CXR



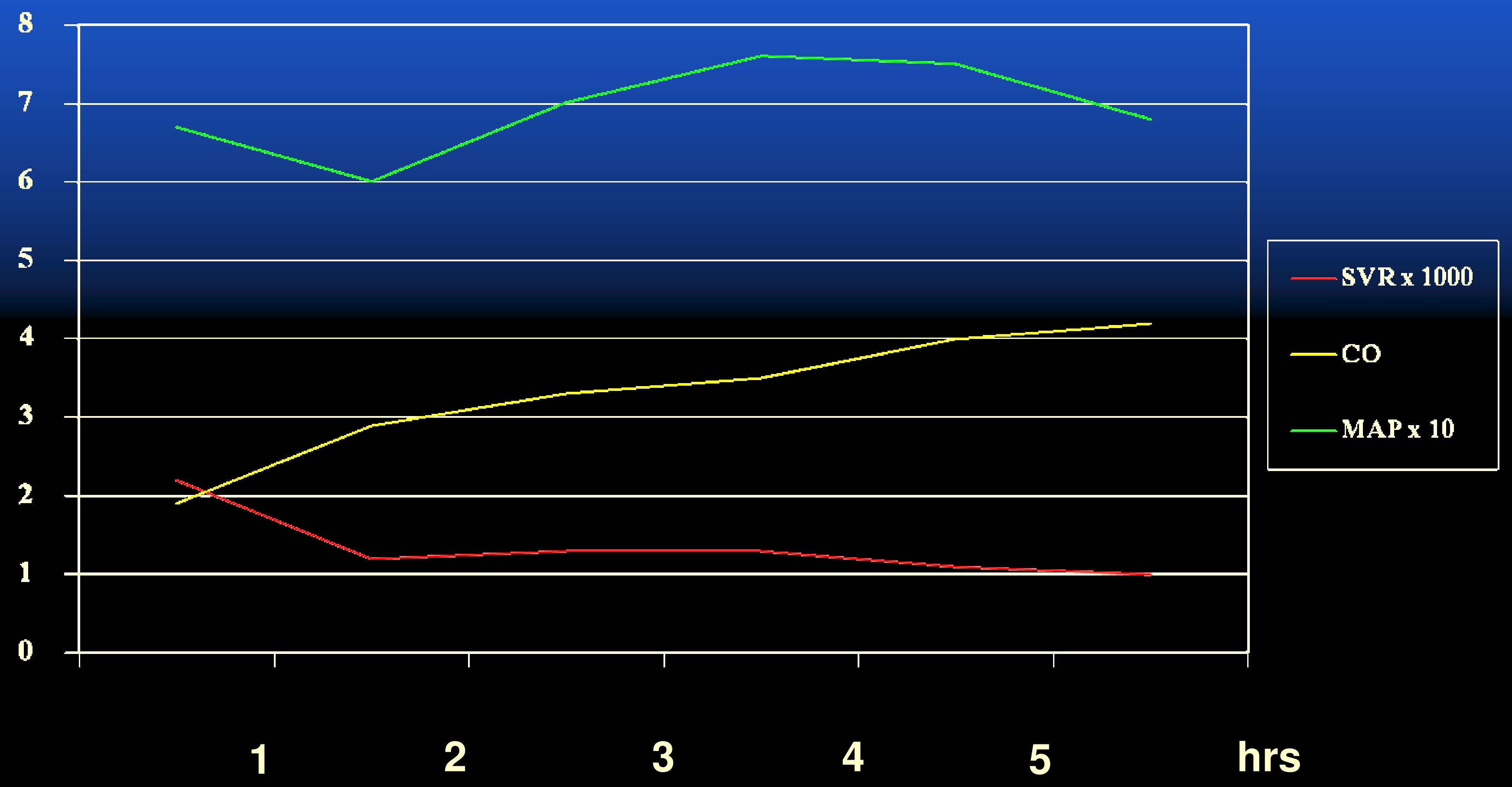
2D Echo

BP	87/58 mmHg
CVP	15 mmHg
PCWP	19 mmHg
CO	1.9 l/min
CI	1.12 $l/min/m^2$
SVR	2190 $dynes.sec/cm^{-5}$
SVRI	3715 $dynes.sec/cm^{-5}$

Treatment

- Adequate oxygenation PaO_2 >70 mmHg
- Correct acid-base disturbance
- Hemodynamic support
  - Inotropic agent if BP < 90 mmHg
  - Diuretic if PCWP is high and pulmonary edema occurs.
  - Vasodilator if SVR is high and has adequate pre-load and BP
  - Intra-aortic balloon pump or left ventricular assisted device.

Relation between SVR and CO



If CO measurement is not available

- Mixed venous O_2 saturation (SvO_2) is a crude guideline for cardiac output.
- Normal SvO_2 saturation 75 %
- The lower SvO_2 means the more O_2 extracted from blood and indicate inadequate blood supply thus lower cardiac output.
- Look for Anemia -> decrease O_2 carrying capacity.

Drugs commonly used

Dopamine	0.5-3.0 mcg/kg/min	dopaminergic
	3.0-7.5 mcg/kg/min	β adrenergic
	> 7.5 mcg/kg/min	α adrenergic
Dobutamine	β1 agonist with mild β2 agonist	
	5-15 mcg/kg/min usual dose	
Epinephrine	very potent α agonist	
	0.01-0.1 mcg/kg/min	
Nitroglycerine	< 40 mcg/min	venous dilator
	> 200 mcg/min	arterial dilator
Nitroprusside	very potent arterial dilator	
	0.5-2.0 mcg/kg/min in CHF	
	2.0-5.0 mcg/kg/min in hypertension	

Kidney receives enormous blood flow

TABLE 7-1 BLOOD FLOW IN MAJOR ORGANS OF THE BODY

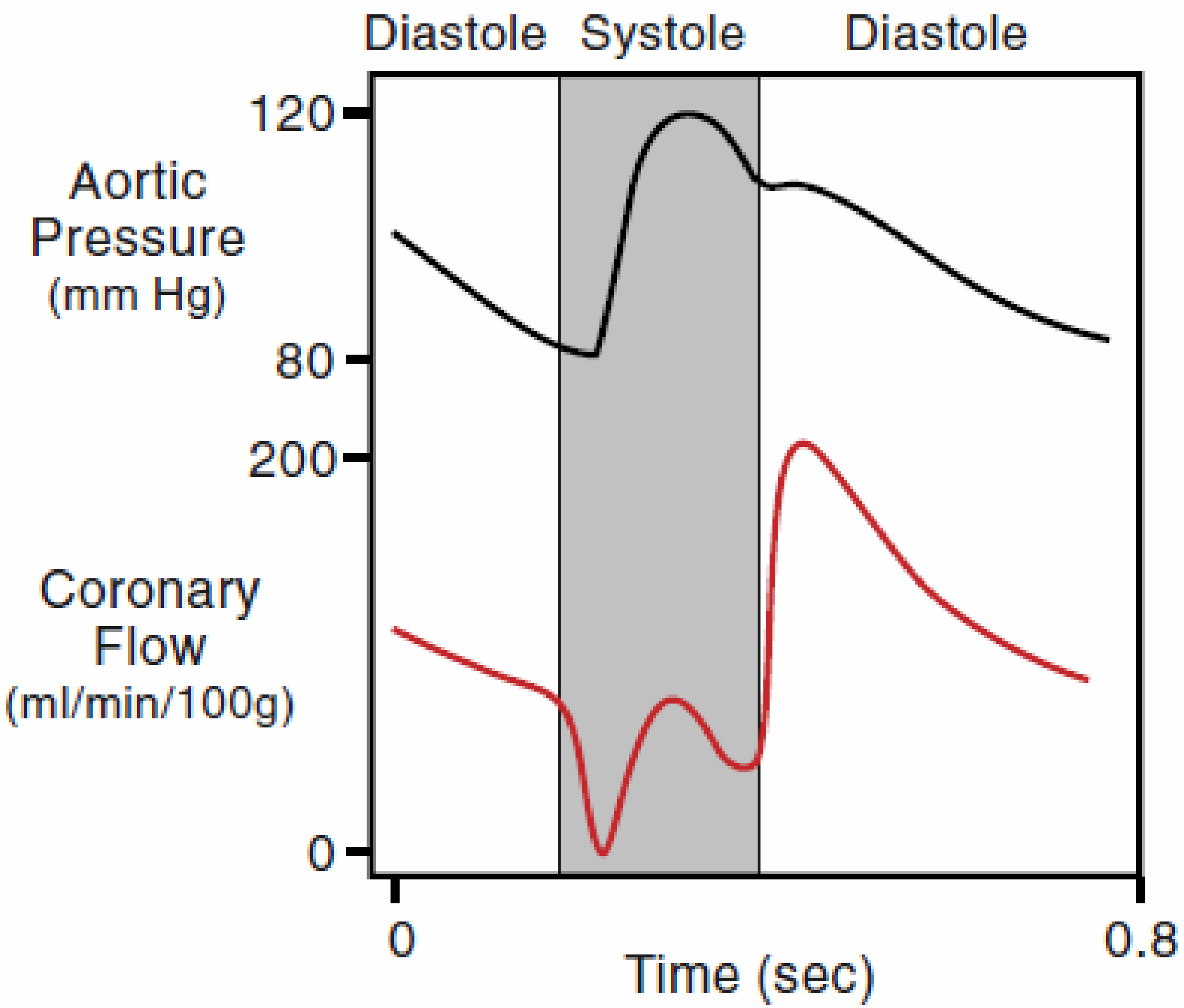
ORGAN	PERCENT BODY WEIGHT	PERCENT CARDIAC OUTPUT AT REST	NORMAL FLOW (ML/MIN PER 100 G)	MAXIMAL FLOW (ML/MIN PER 100 G)
Heart	0.4	5	80	400
Brain	2	14	55	150
Skeletal muscle	40	18	3	60
Skin	3	4	10	150
Stomach, intestine, liver, spleen, pancreas	6	23	30	250
Kidneys	0.4	20	400	600
Other	48	16	-	-

Normal and maximal flows are approximate values for the whole organ. Many organs (e.g., brain, muscle, kidney, and intestine) have considerable heterogeneity of flow within the organ depending on the type of tissue or region of organ being perfused. The liver receives blood flow from the gastrointestinal venous drainage as well as from the hepatic artery (only hepatic artery flow is included in this table). "Other" includes reproductive organs, bone, fat, and connective tissue.

Pre-renal Azothemia in state of hypervolumia

- Renal disease is often associated with coronary artery disease.
- Coronary artery disease is quite common cause of LV dysfunction (diastolic or both systolic and diastolic).
- So patients with renal disease who develop acute heart failure showed markedly reduce RBF and hemodynamically resambles pre-renal azothemia.
- But in this setting, vasodilator and ACEI or ARB are quite useful.

Coronary artery perfuses during diastole



Patients with very low diastolic blood pressure may significantly impair there myocardial perfusion and have ischemia especially during stress.

Beware of patients with AV shunt and significant coronary artery disease

AT Last

- No monitoring device, no matter how simple or complex, invasive or non-invasive, inaccurate or precise will improve outcome unless coupled to a *treatment*, which itself improves outcome.

Pinsky & Payen. Functional Hemodynamic Monitoring, Springer, 2004