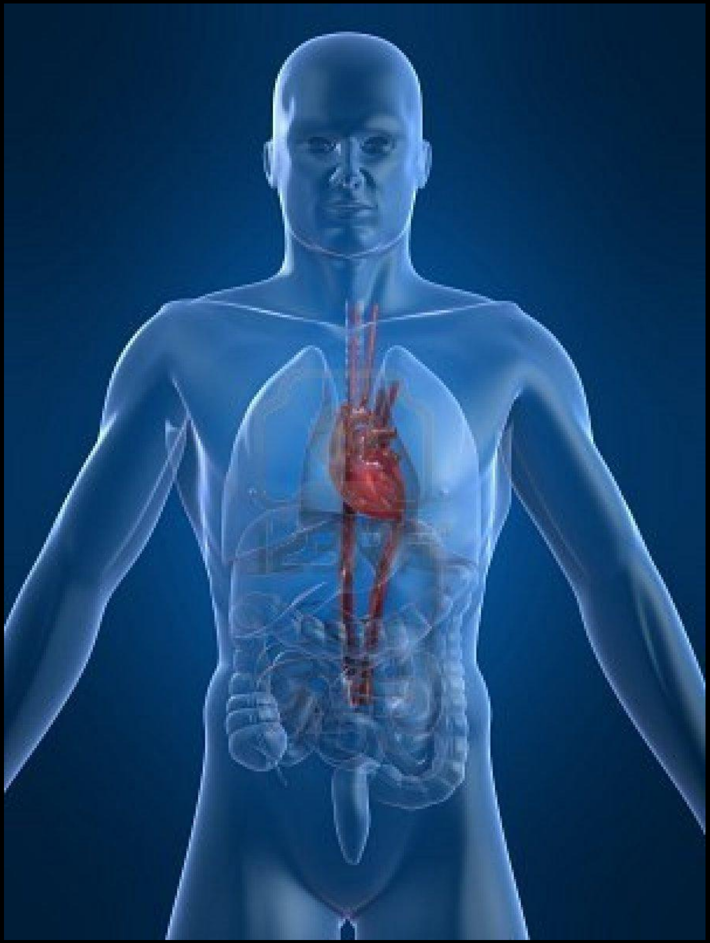
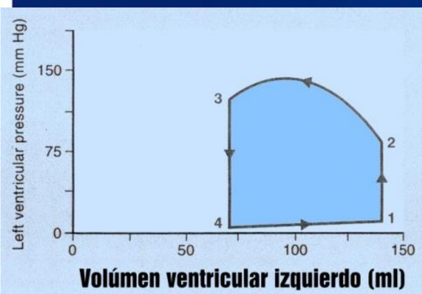
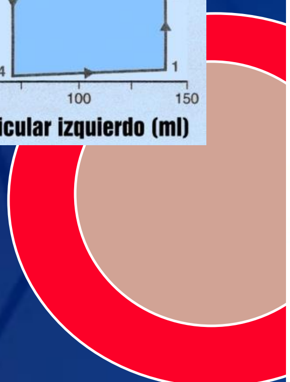
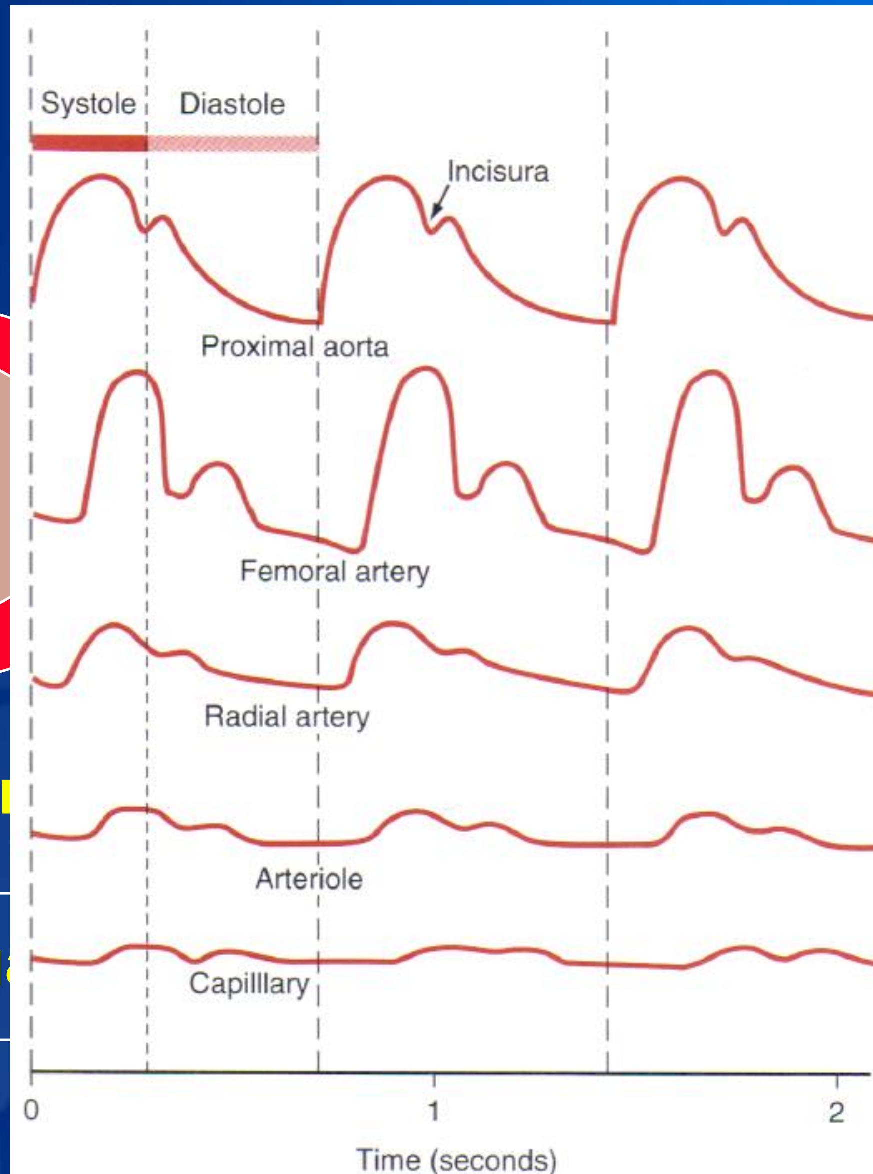
MECANICA CARDIACA:
“EL CORAZON COMO BOMBA”

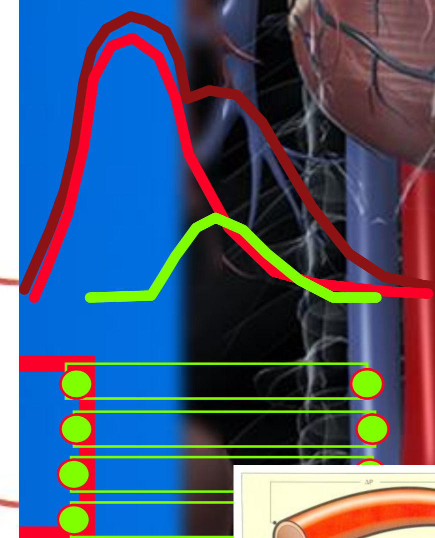


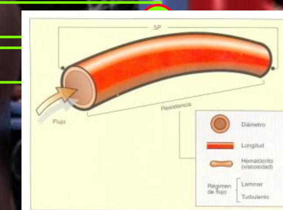
Visión holística de la circulación











corazón

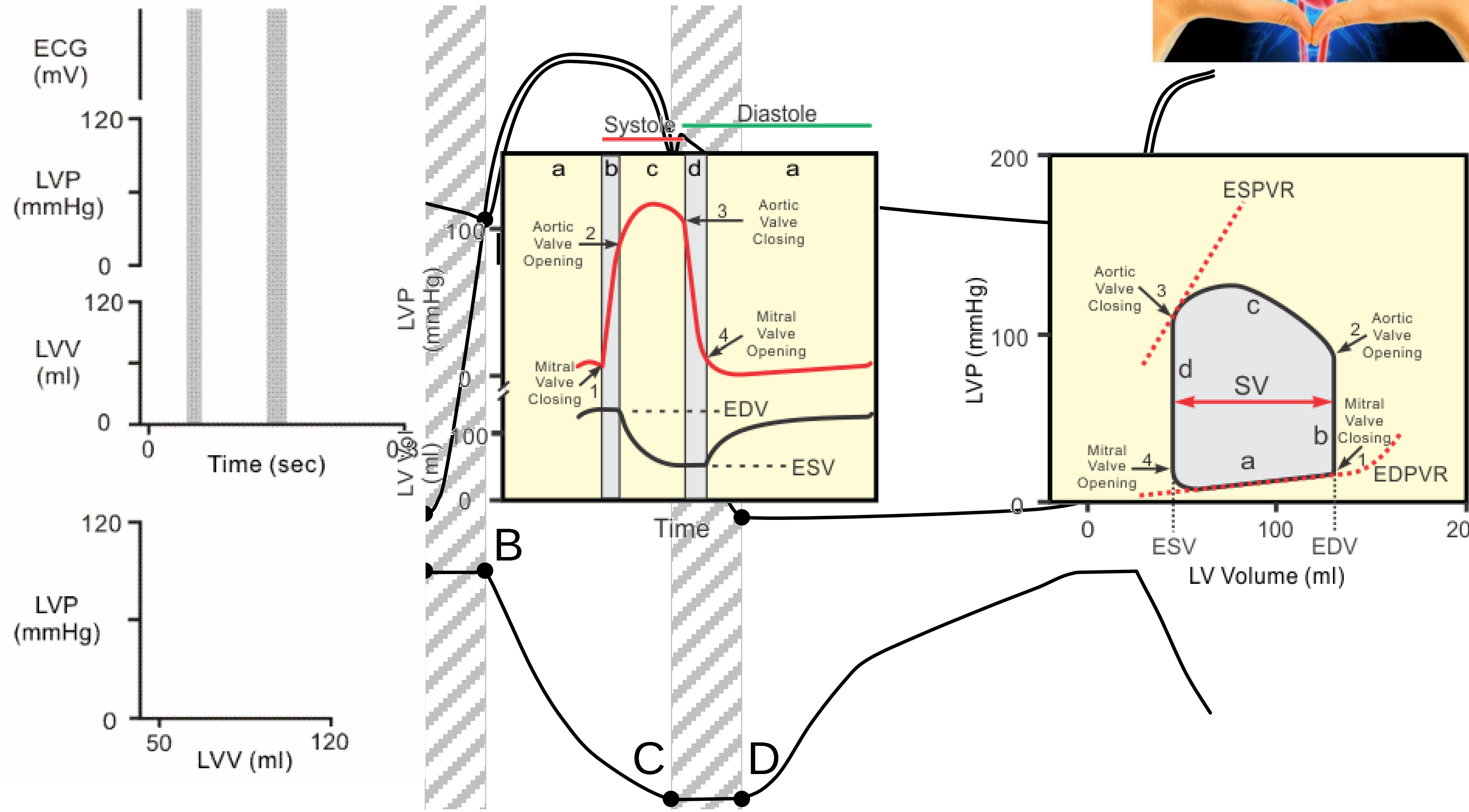
Arteriolas-
capilares

entrega

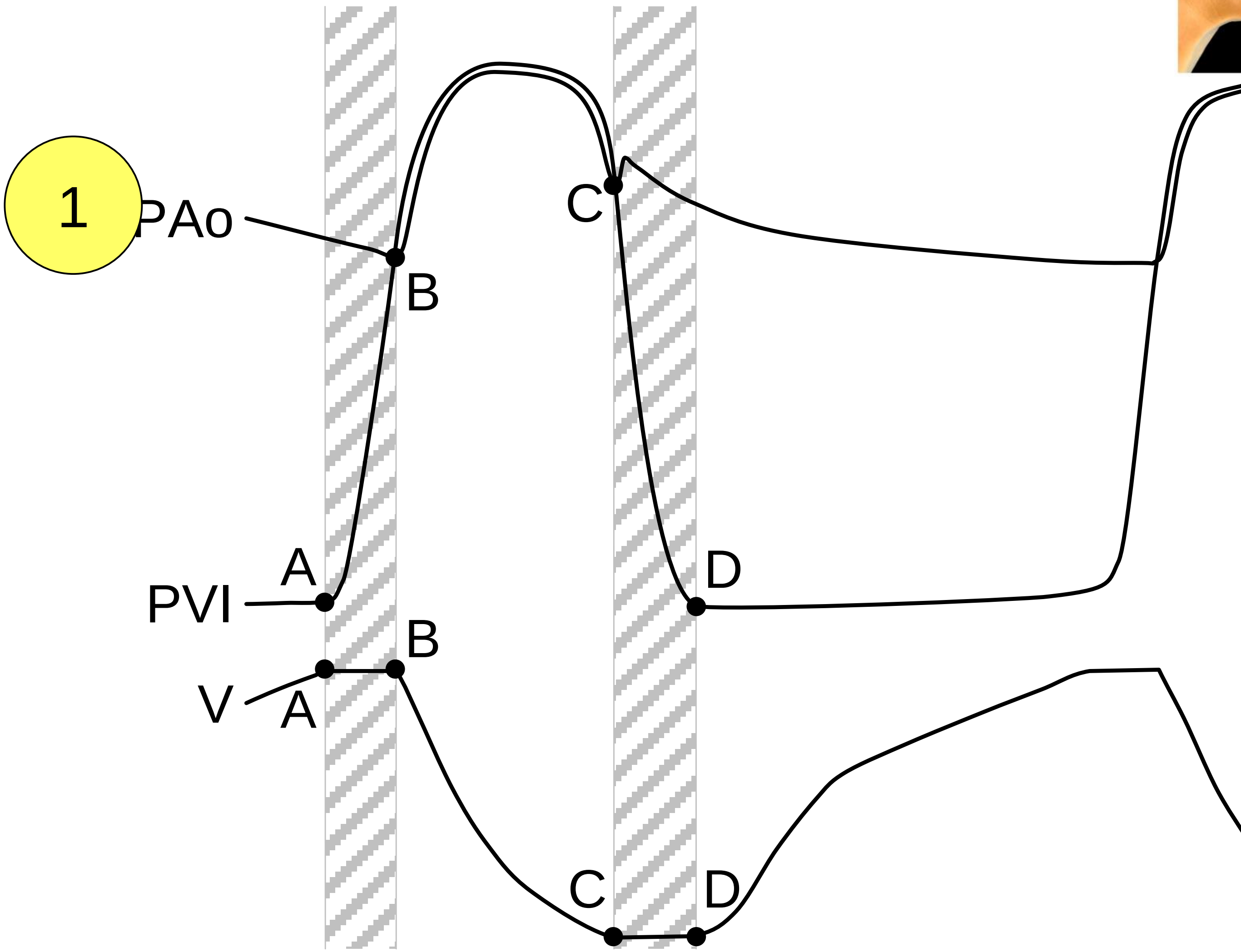
disipación

Cardiac Output:	5 liters/min. (resting)	
	15-25 liters/min. (exercise)	
Heart Rate:	60–80 beats/min. (resting)	
	120–160 beats/min. (exercise)	
Stroke Volume:	70 cc. (resting)	
	160 cc. (exercise)	
Pressures:	Aortic Phasic	120/80 mmHg
	Mean	100 mmHg
	Pulmonary Artery	25/10
	Mean	15
	Venous Mean	5
	Intrathoracic	–5
	1 mmHg = 1330 dynes/cm^2	
Dimensions (diameters):	Aorta	2.5 cm
	Medium Artery	0.5 cm
	Arteriole	30–60 m
	Capillary	8 m
	Vein (medium)	0.5 cm
	Vena Cava	3.0 cm
	Red Blood Cell	7 m
Velocities (approximate):	100 cm/sec. *peak* in aorta	
	0.5–1 mm/sec. in capillaries	
	20 cm/sec. in vena cava	
Viscosities:	Water	1.0 centipoise
	Plasma	1.5 centipoise
	Whole blood	4.0 centipoise
	(1 centipoise = 10^{-2} dyne-sec./cm^2)	
Resistance:	Total Pulmonary	150 dyne-sec. cm^{-5}
	Systemic	1500 dyne-sec. cm^{-5}

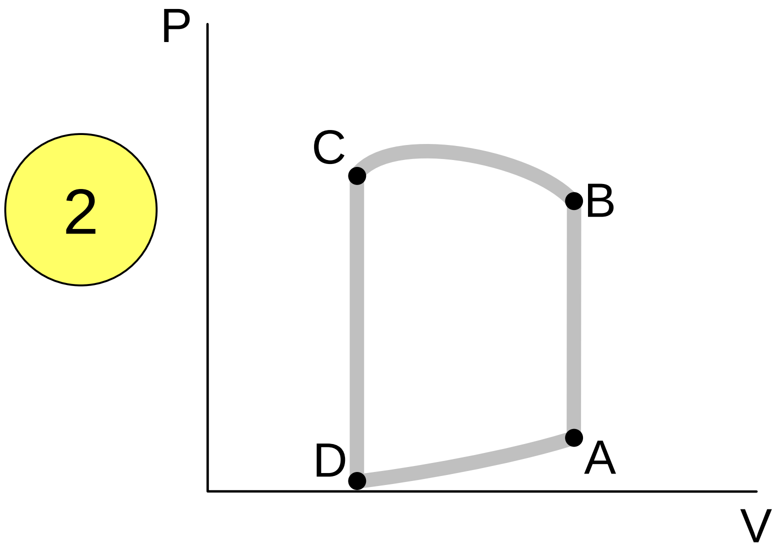
Series temporales

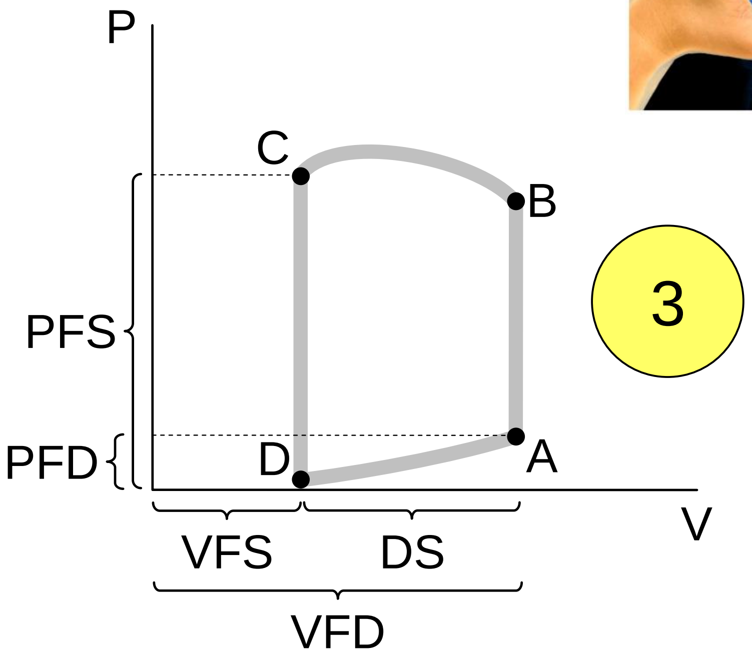


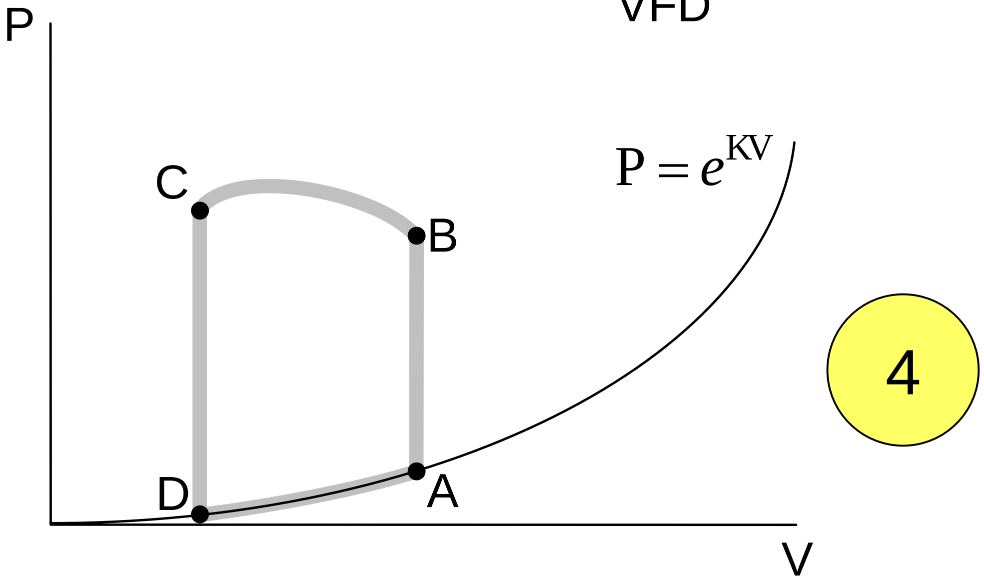
Series temporales



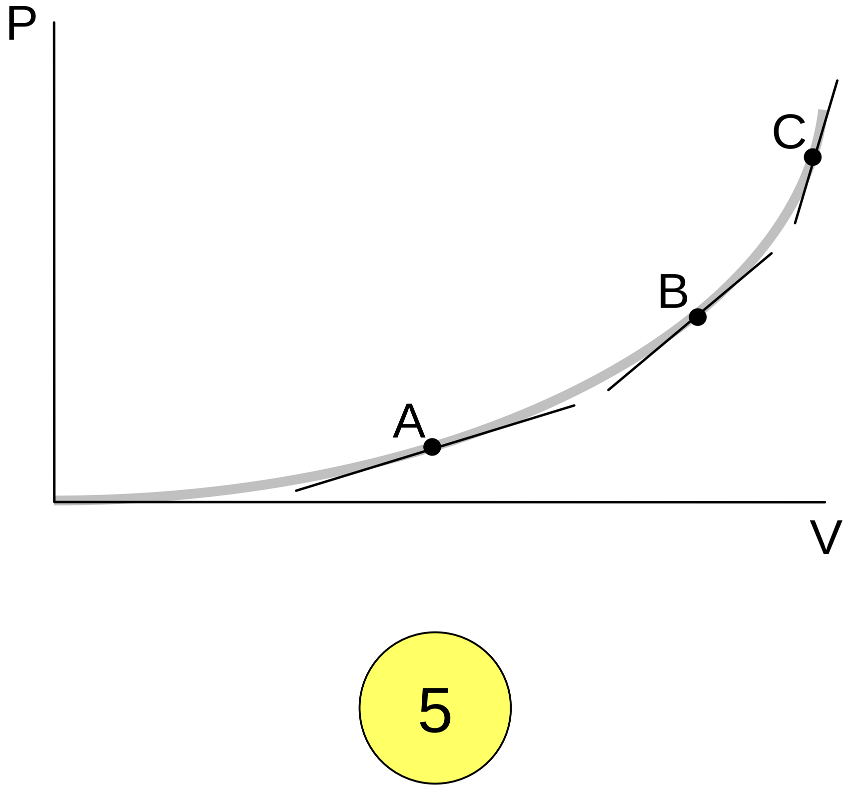
El bucle presión-volumen

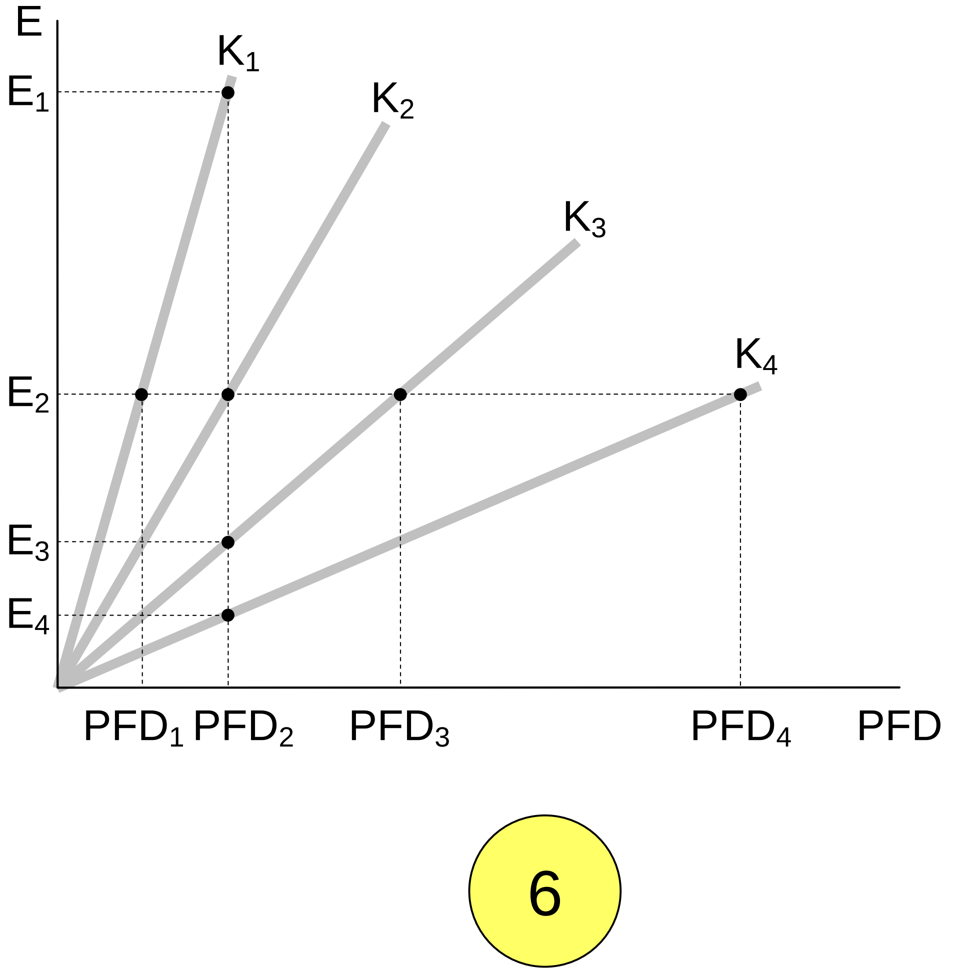






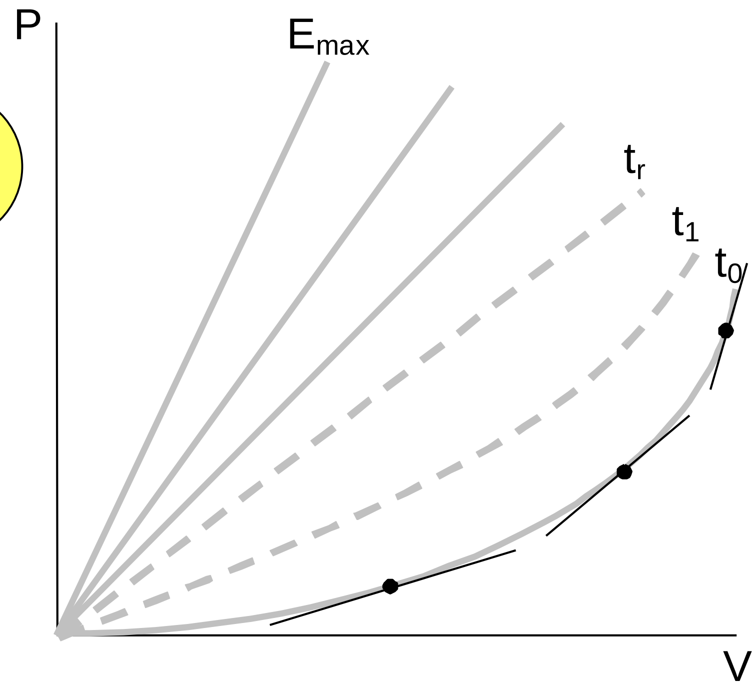
Comportamiento diastólico





Comportamiento sistólico

7



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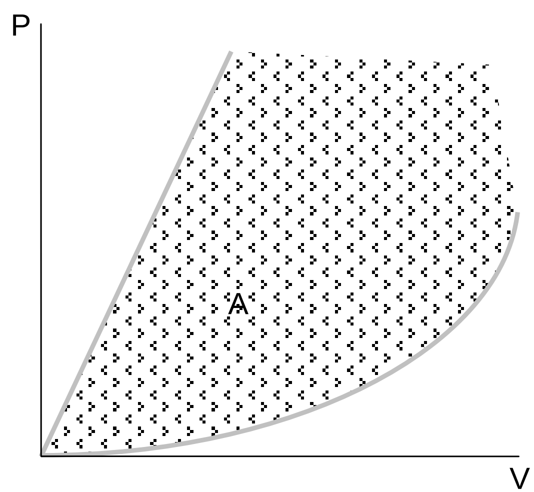

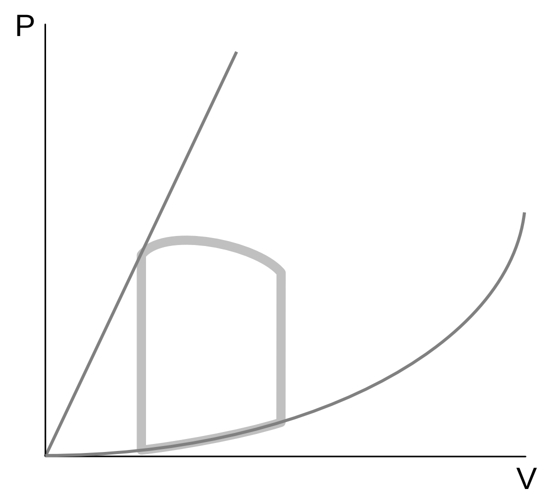

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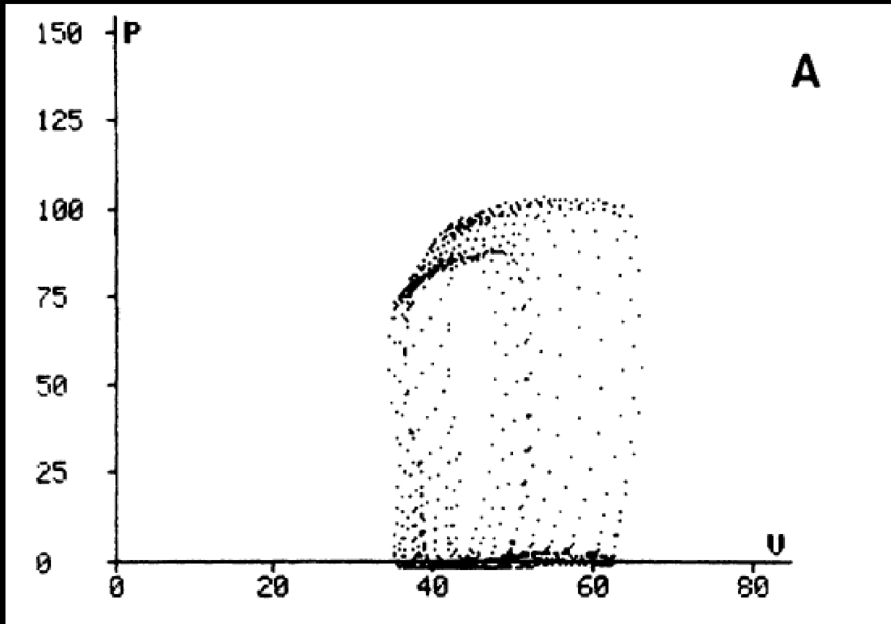
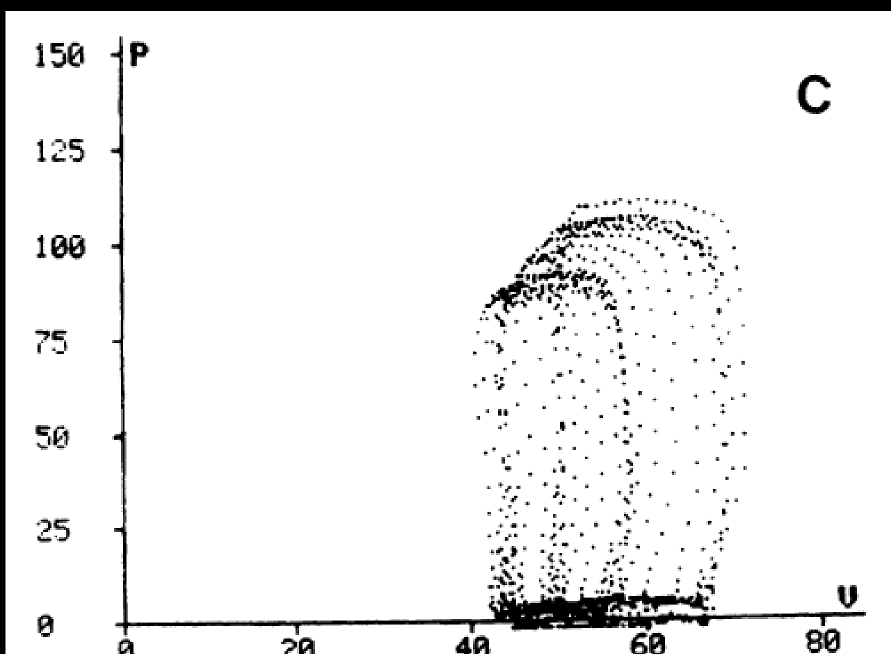


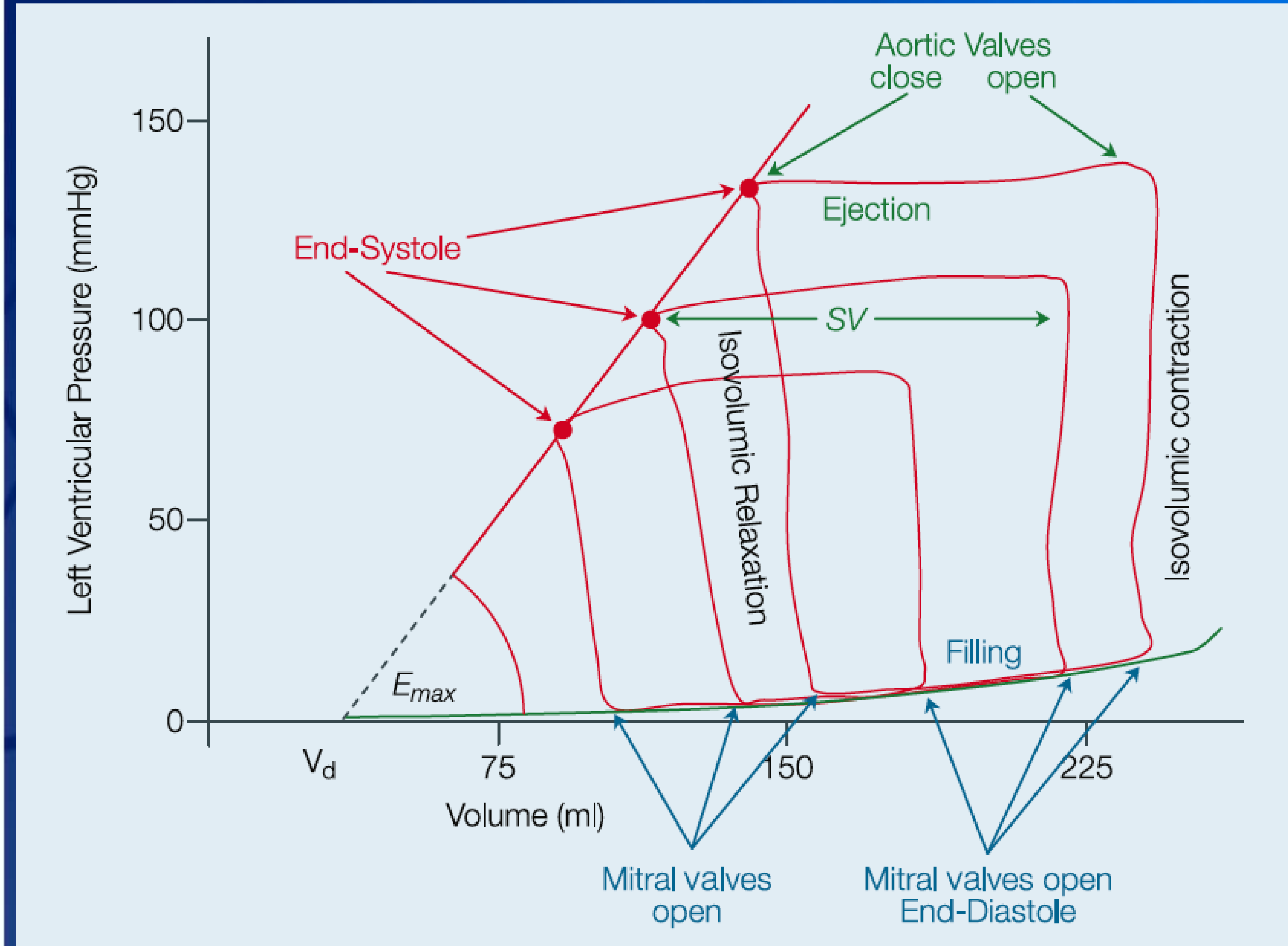
Inconsistency of the slope and the volume intercept of the end-systolic pressure-volume relationship as individual indexes of inotropic state in conscious dogs: presentation of an index combining both variables

AJ Crottogini, P Willshaw, JG Barra, R Armentano, EI Cabrera Fischer

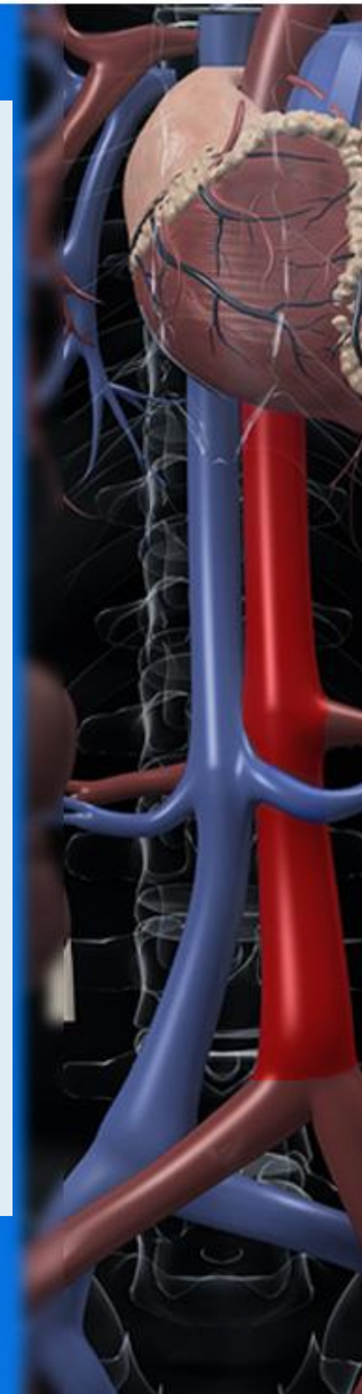
Circulation 1987;76;1115-1126

FIGURE 2. On-line monitoring of pressure-volume loops during vena caval occlusion in dog 3. *A*, Control; *B*, I^+; *C*, I^-. P = pressure (mm Hg); V = volume (ml).

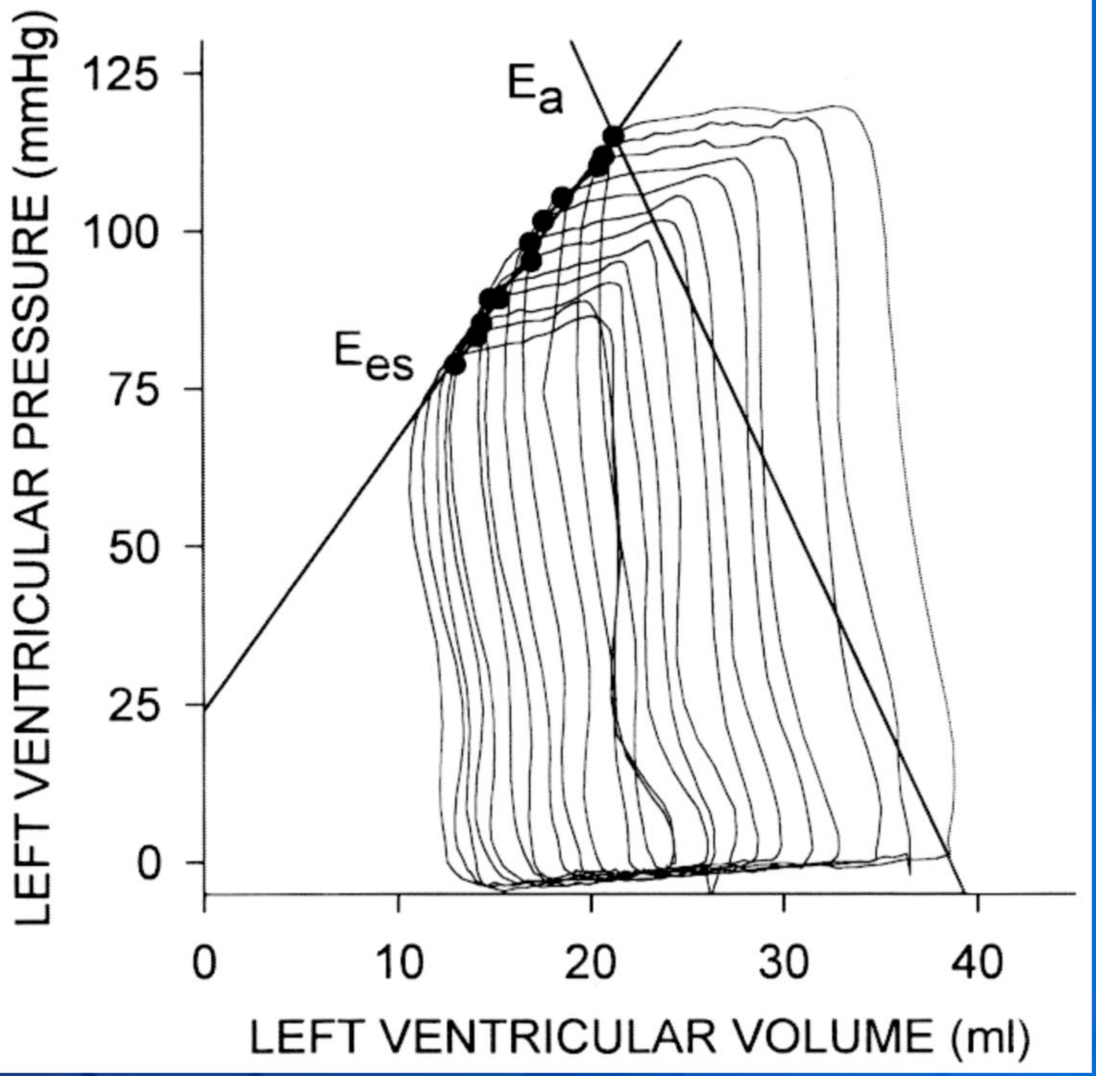


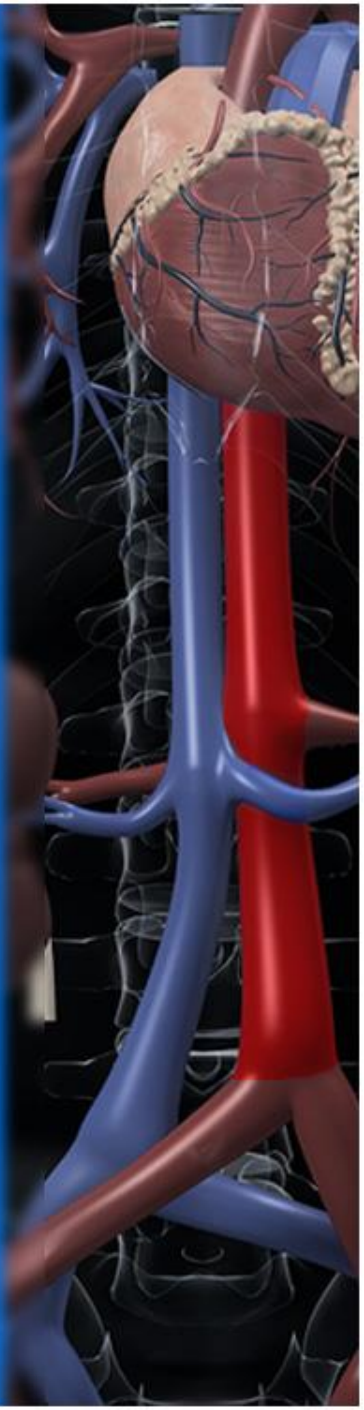


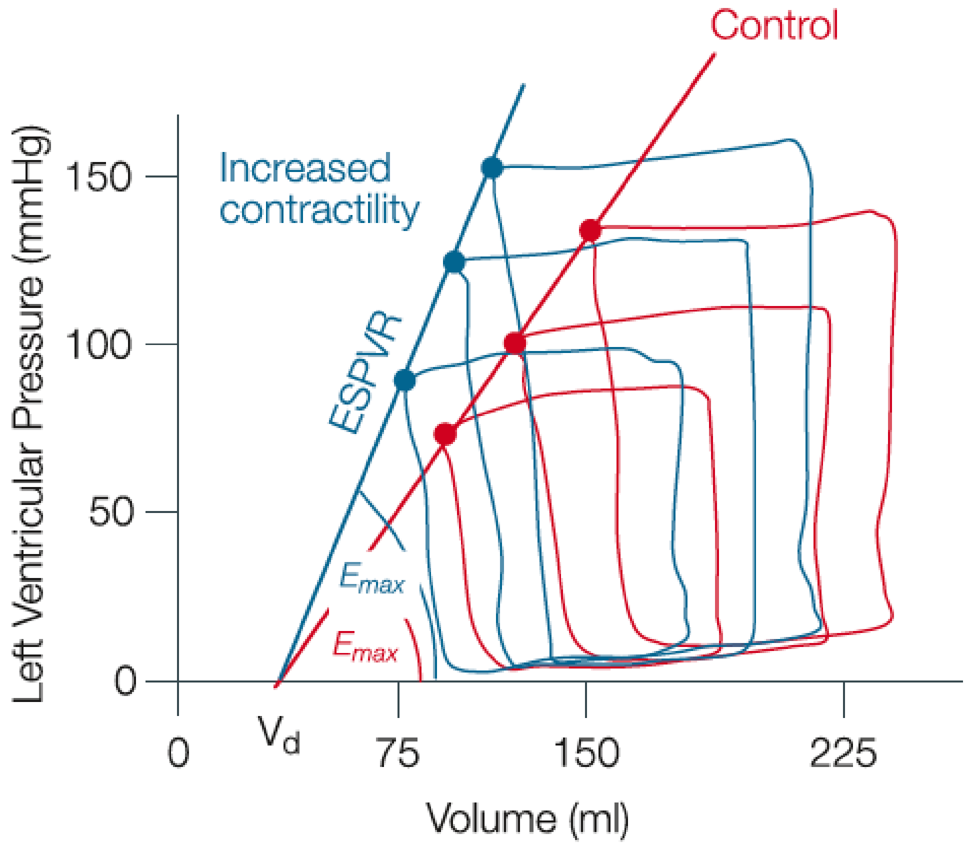
Left Ventricular Pressure (mmHg)
150
100
50
0
Volume (ml)
V$_d$
75
150
225
Aortic Valves
close
open
Ejection
End-Systole
SV
Isovolumic Relaxation
Isovolumic contraction
Filling
E_{max}
Mitral valves
open
Mitral valves open
End-Diastole



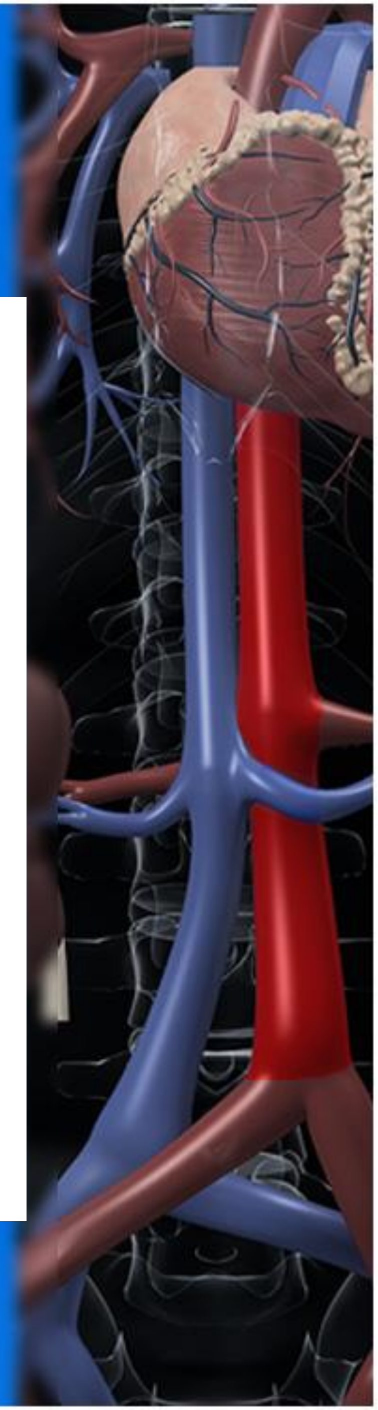
ELASTANCIA de FDS

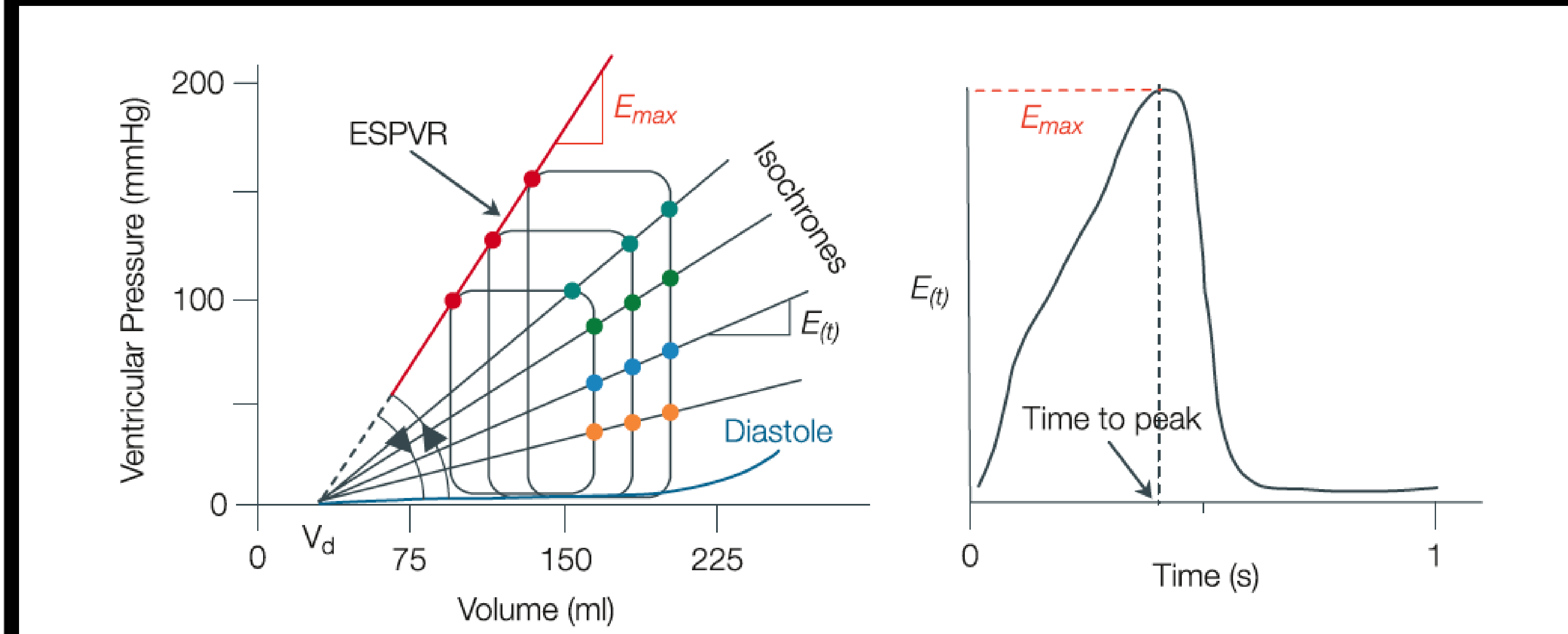




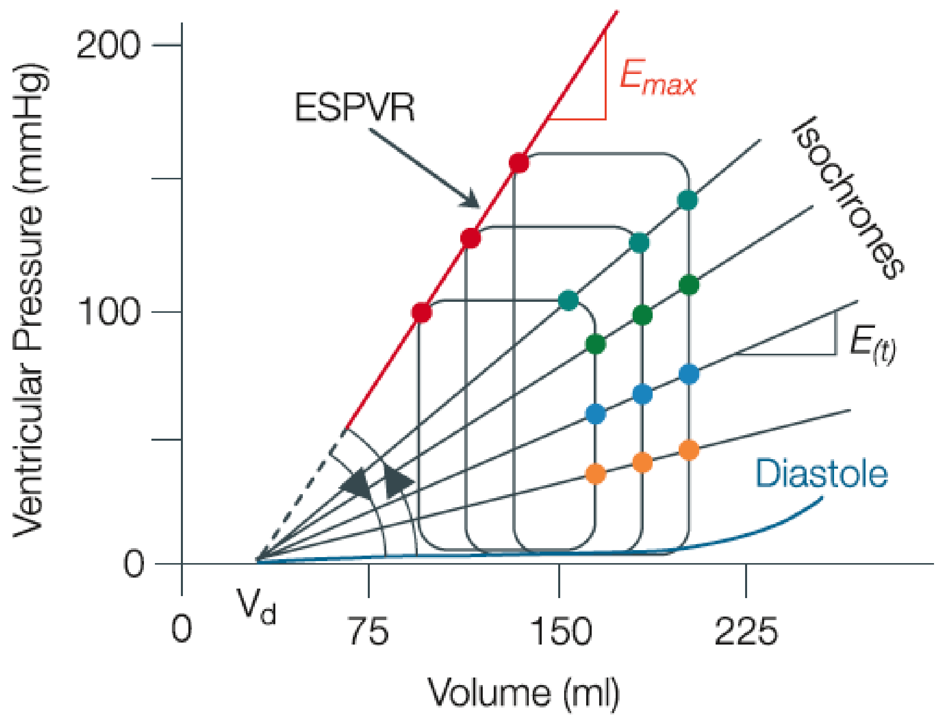


Control
Increased
contractility
ESPVR
E_{max}
E_{max}
Left Ventricular Pressure (mmHg)
150
100
50
0
0
V_d
75
150
225
Volume (ml)

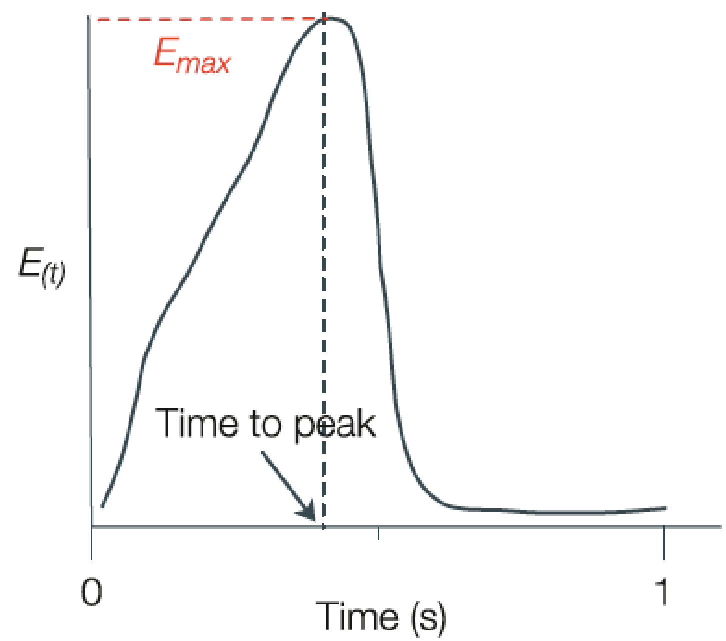




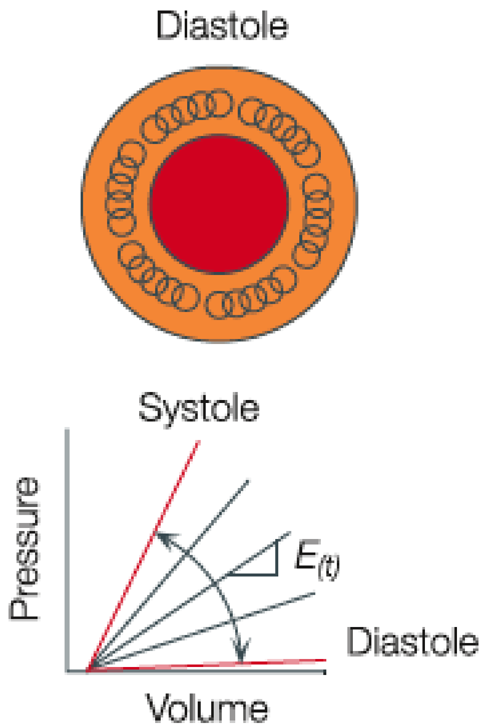
Ventricular Pressure (mmHg)
200
100
0
ESPVR
E_{max}
Isochrones
$E_{(t)}$
Diastole
V_d
0
75
150
225
Volume (ml)
E_{max}
$E_{(t)}$
Time to peak
0
1
Time (s)

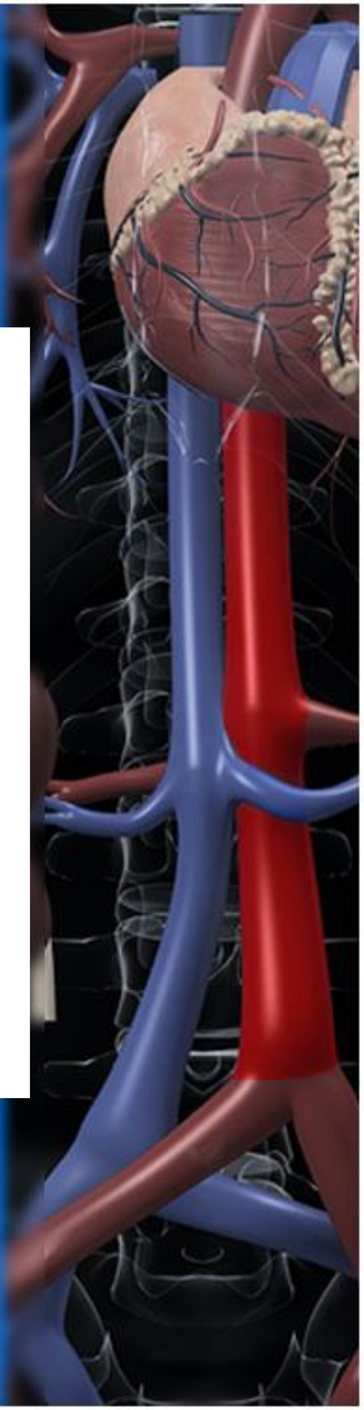


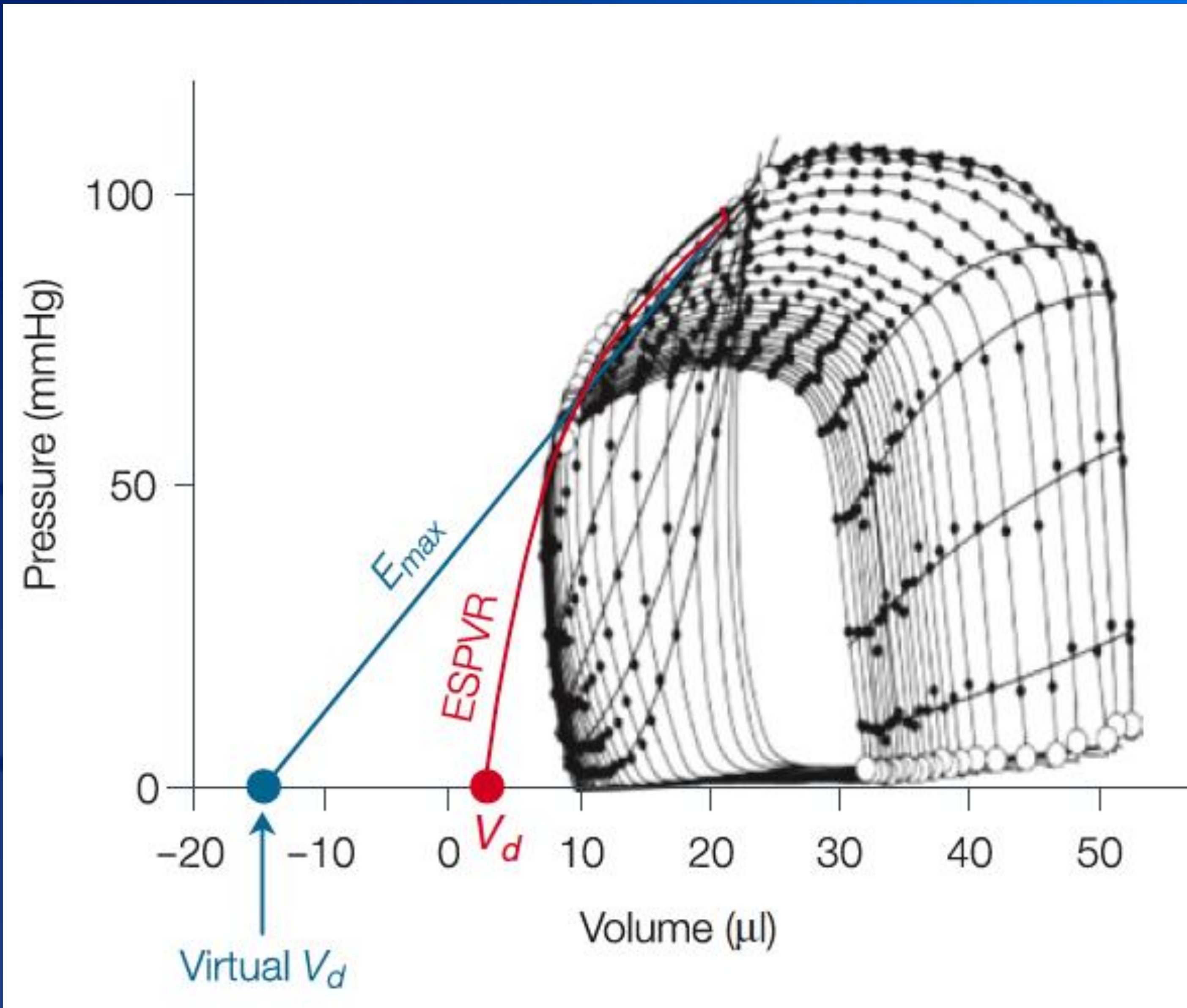
200
100
0
Ventricular Pressure (mmHg)
ESPVR
E_{max}
Isochrones
$E_{(t)}$
Diastole
0
V_d
75
150
225
Volume (ml)

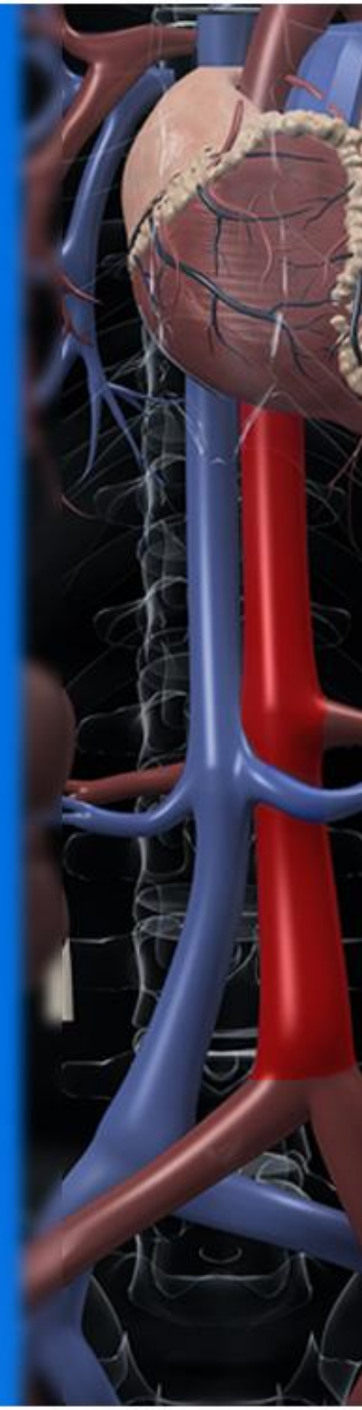


E_{max}
$E_{(t)}$
Time to peak
0
1
Time (s)


Diastole
Systole
Systole
Pressure
$E_{(t)}$
Diastole
Volume
Elastance
Time




Pressure (mmHg)
100
50
0
Volume (µl)
-20
-10
0
10
20
30
40
50
E_{max}
ESPVR
V_d
Virtual V_d



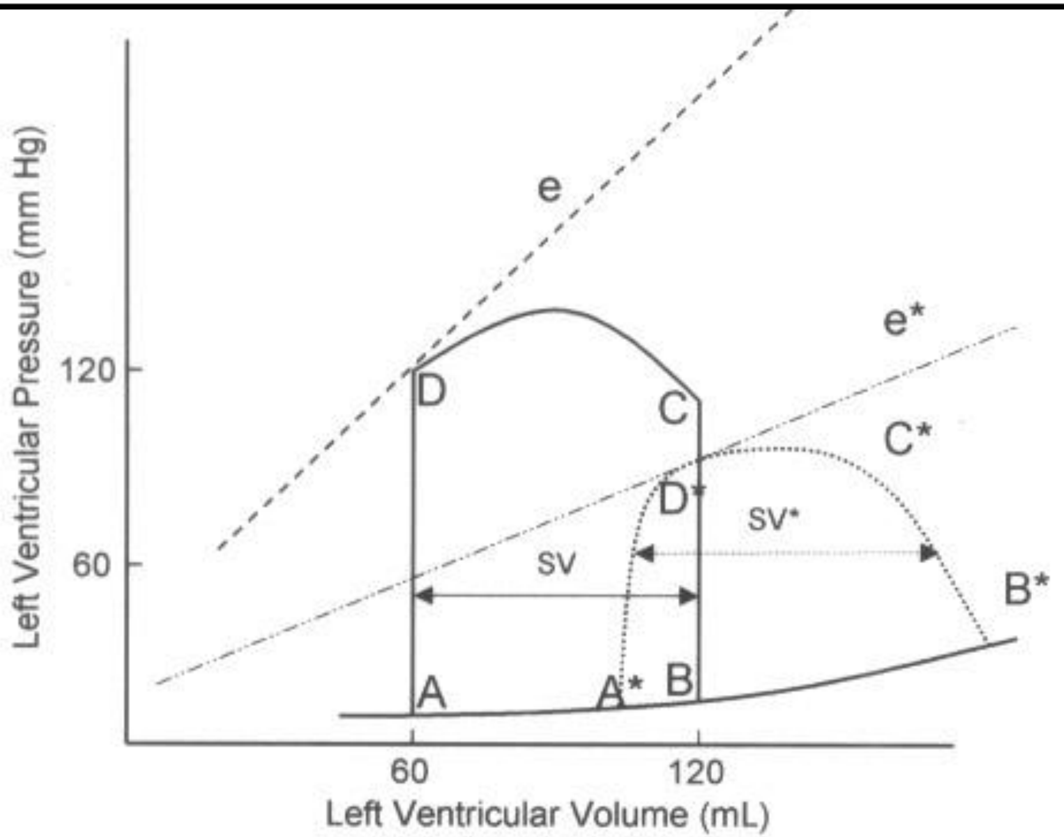
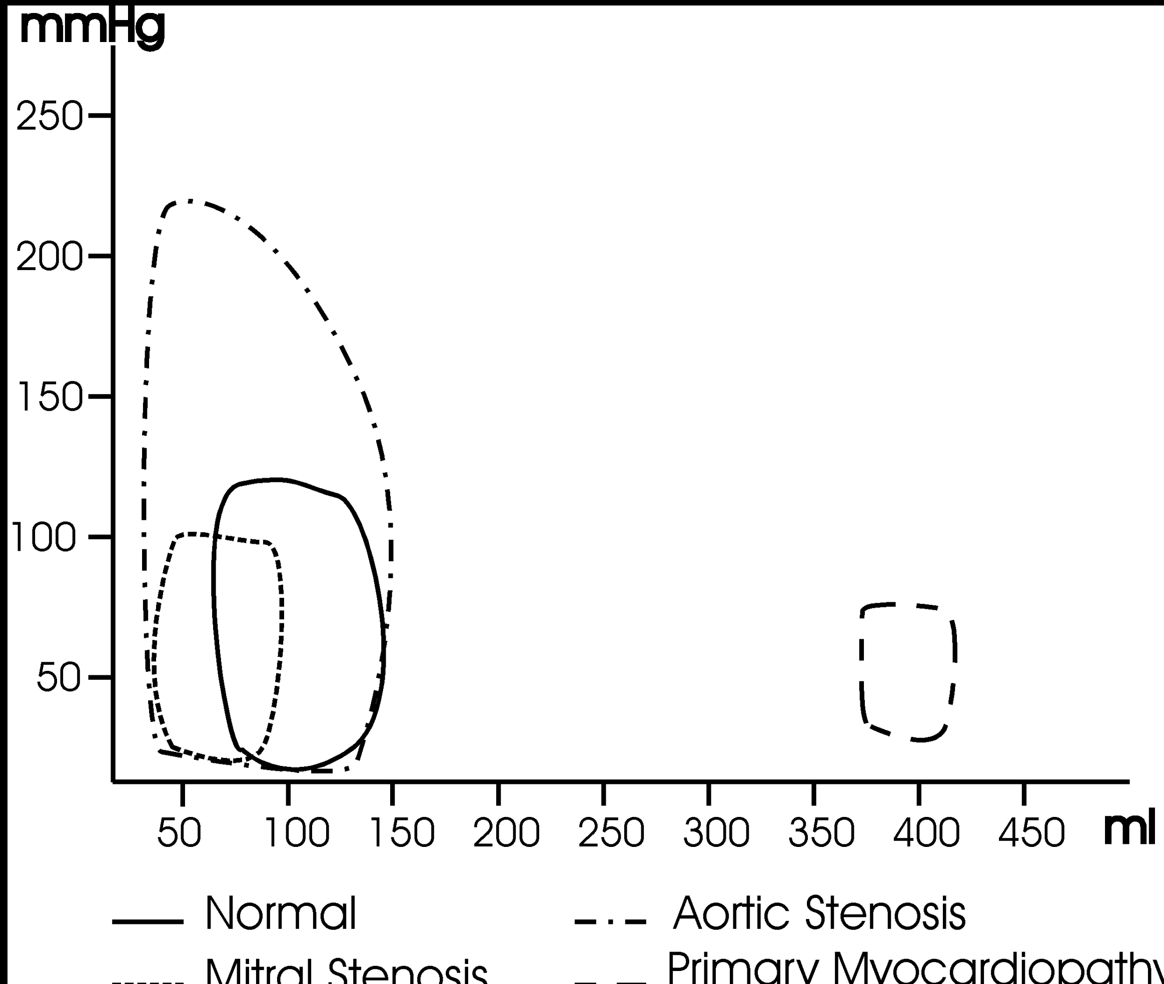
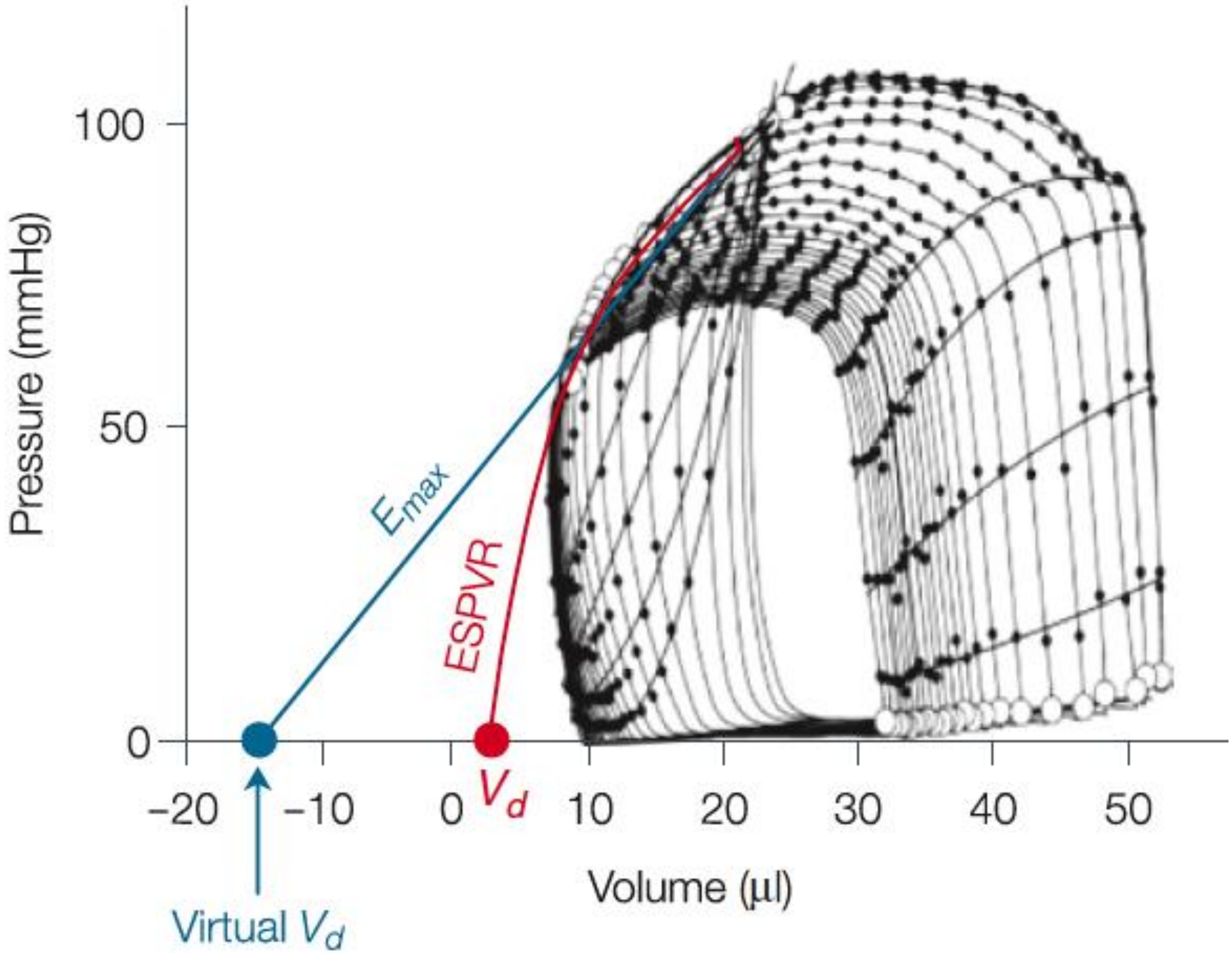


Fig. 5. Effect of ventricular failure on the pressure–volume loop. In heart failure, the myocardium compensates its inability to contract by increasing preload in an attempt to maintain stroke volume. Excessive preload eventually leads to worsening of heart failure. e and e*, contractility lines; SV, stroke volume.

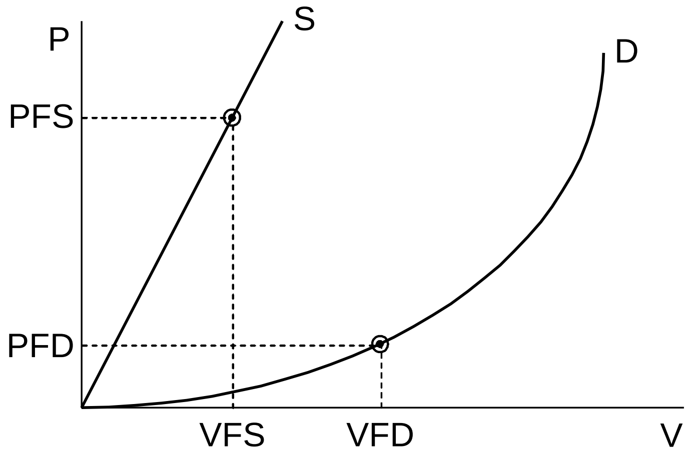


Left ventricular pressure-volume relationship in normal and diseased hearts. Note the large displacement shown by Primary Myocardiopathy characterized by severe ventricular enlargement and low systolic pressures.

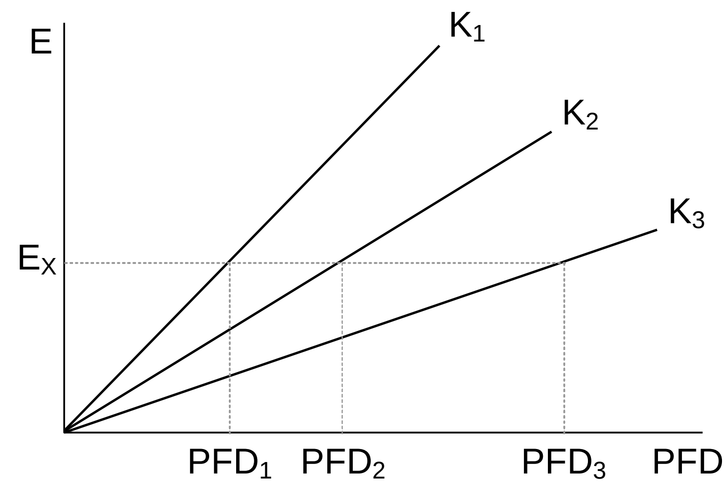


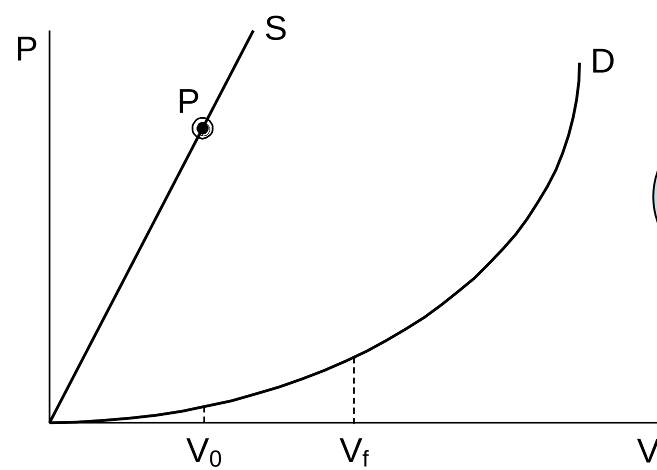
Pressure (mmHg)
100
50
0
E_{max}
ESPVR
V_d
−20
−10
0
10
20
30
40
50
Volume (µl)
Virtual V_d

1



2

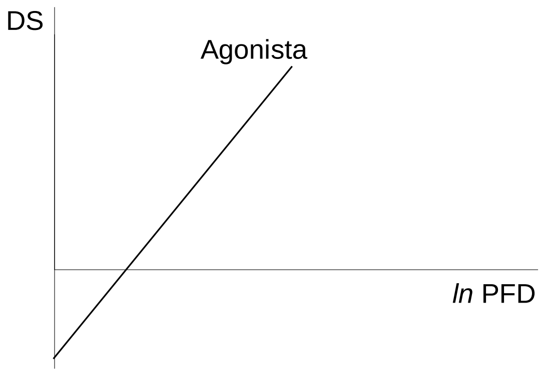




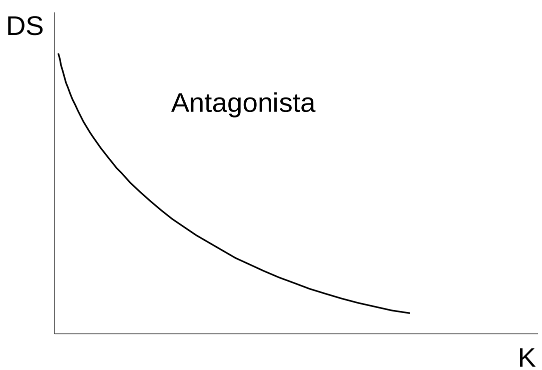
3

Extrínsecas

4

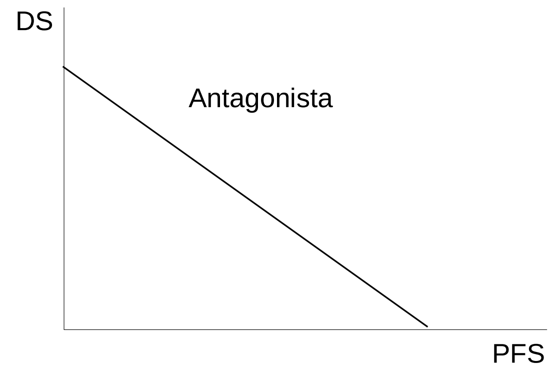


6

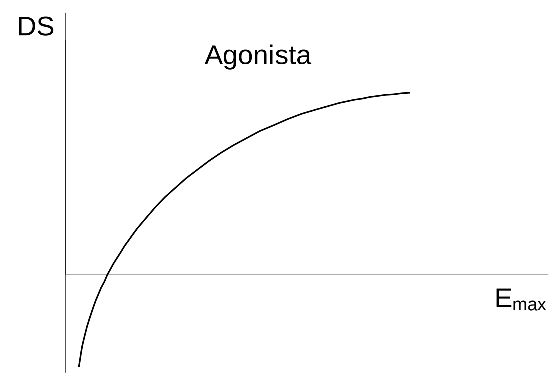


Intrínsecas

5



7



Determinantes de DS

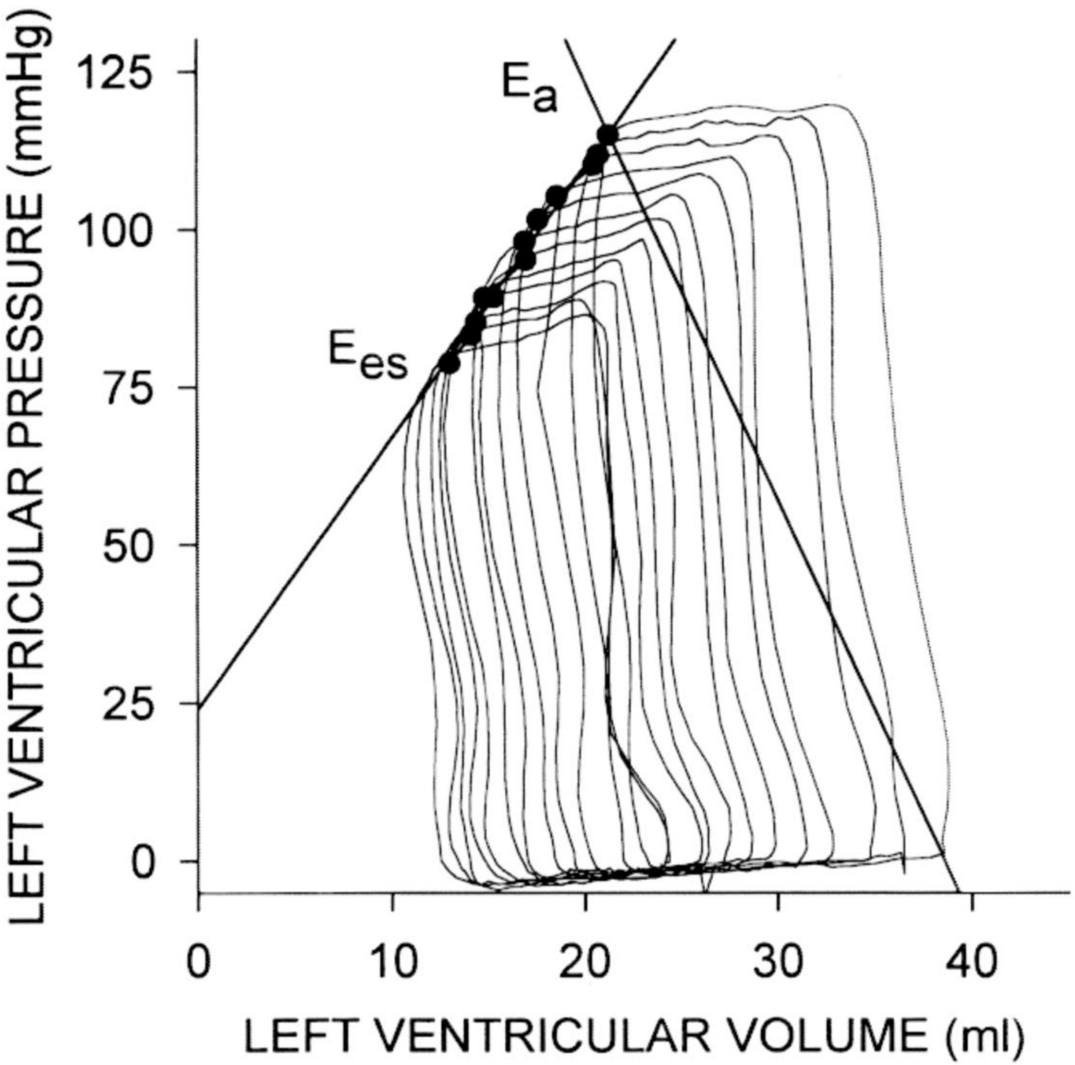
Propiedades que determinan la descarga sistólica (DS)

- Intrínsecas
  - Agonistas: E_{max}
  - Antagonistas: K
- Extrínsecas
  - Agonistas: PFD
  - Antagonistas: PFS

Causas de descenso de DS

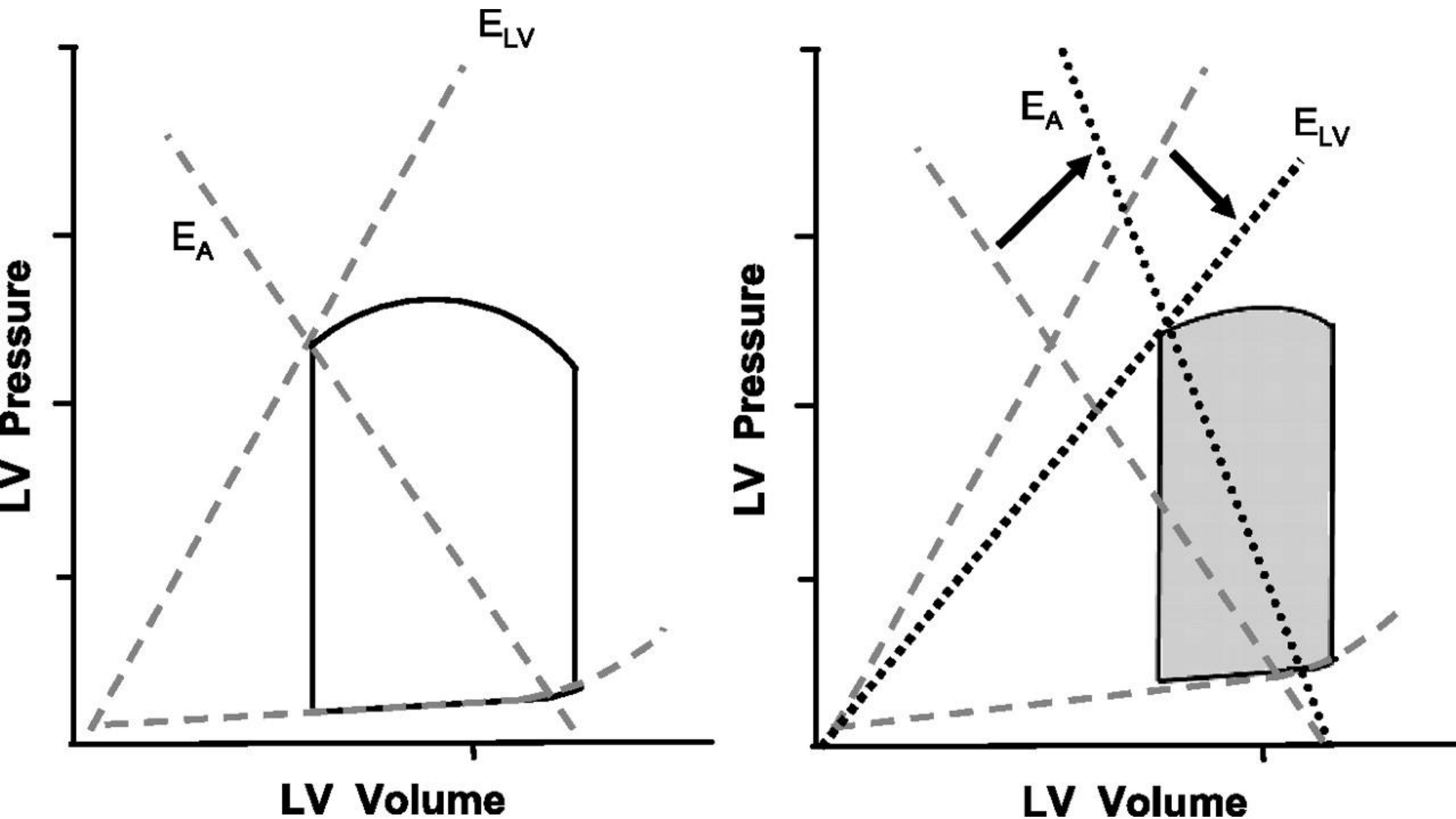
Causa de descenso de la descarga sistólica (DS)

- A. Patologías de causa intrínseca
  - A1. Descenso del agonista intrínseco E_{max} (Insuficiencia cardíaca)
  - A2. Aumento del antagonista intrínseco K (Miocardiopatías hipertróficas)
- B. Patologías de causa extrínseca
  - B1. Descenso del agonista extrínseco PFD (Shock)
  - B2. Aumento del antagonista extrínseco PFS (Hipertensión)

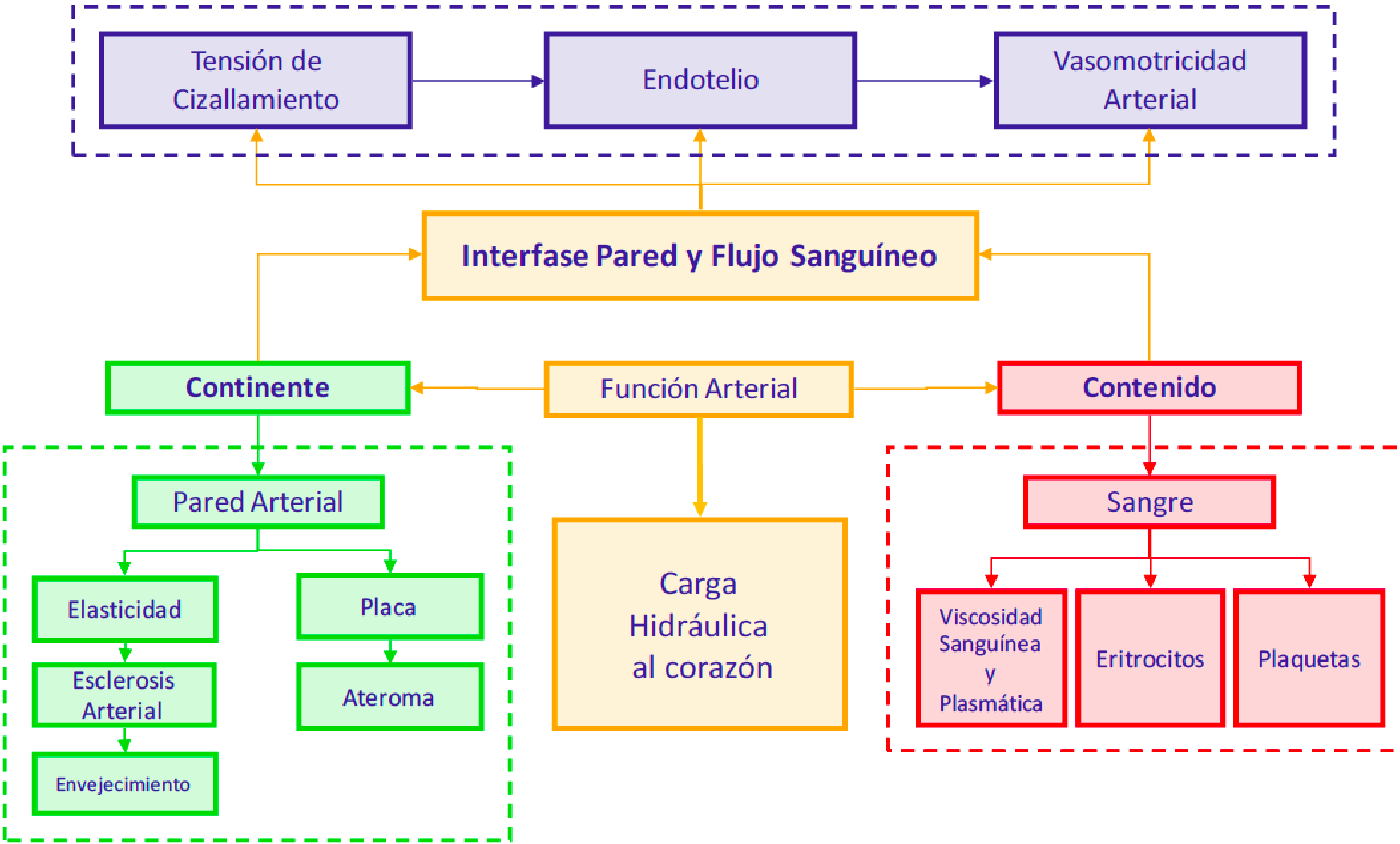


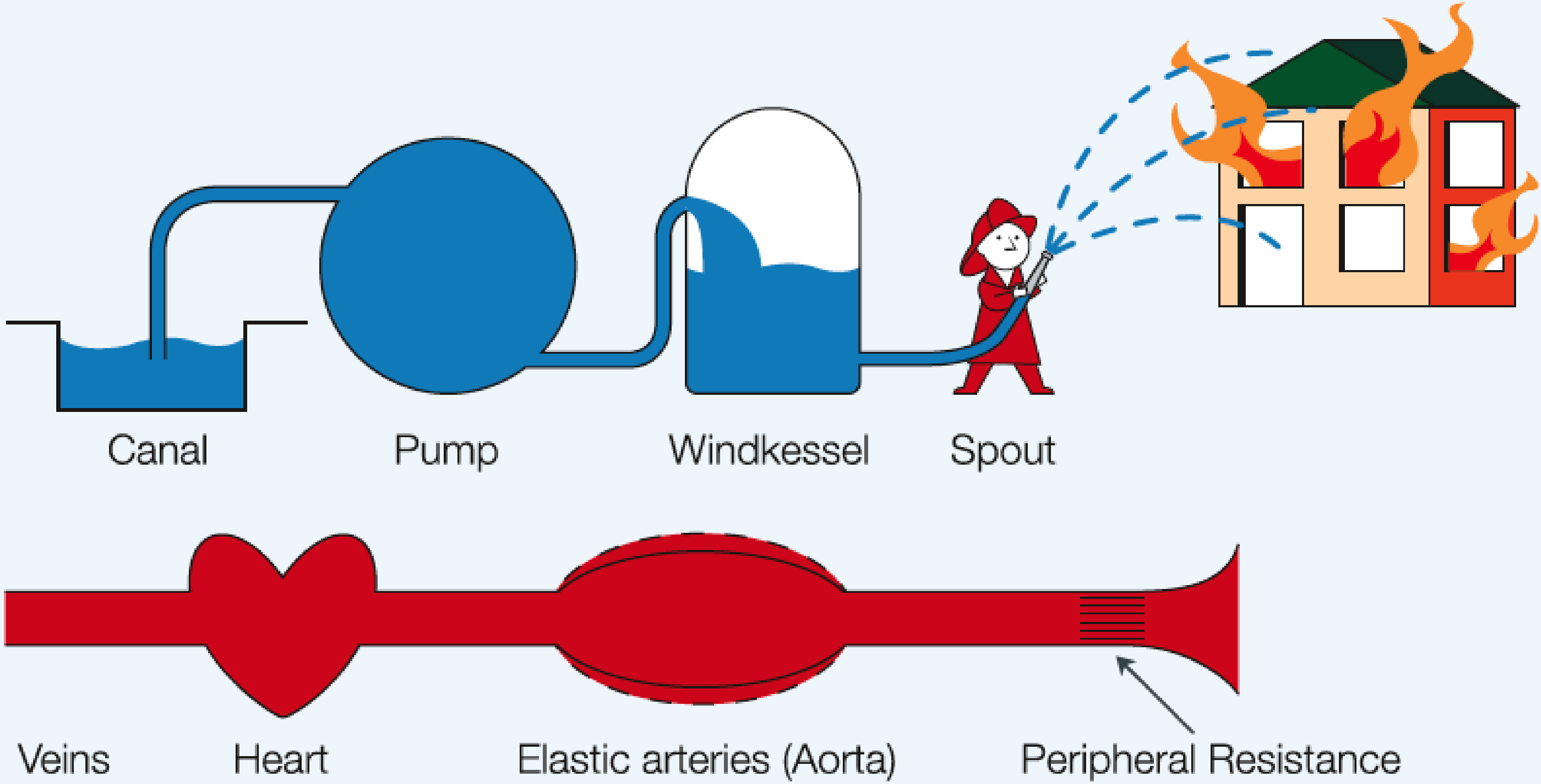
LEFT VENTRICULAR PRESSURE (mmHg)
125
100
75
50
25
0
E_a
E_{es}
0
10
20
30
40
LEFT VENTRICULAR VOLUME (ml)

ACOPLAMIENTO



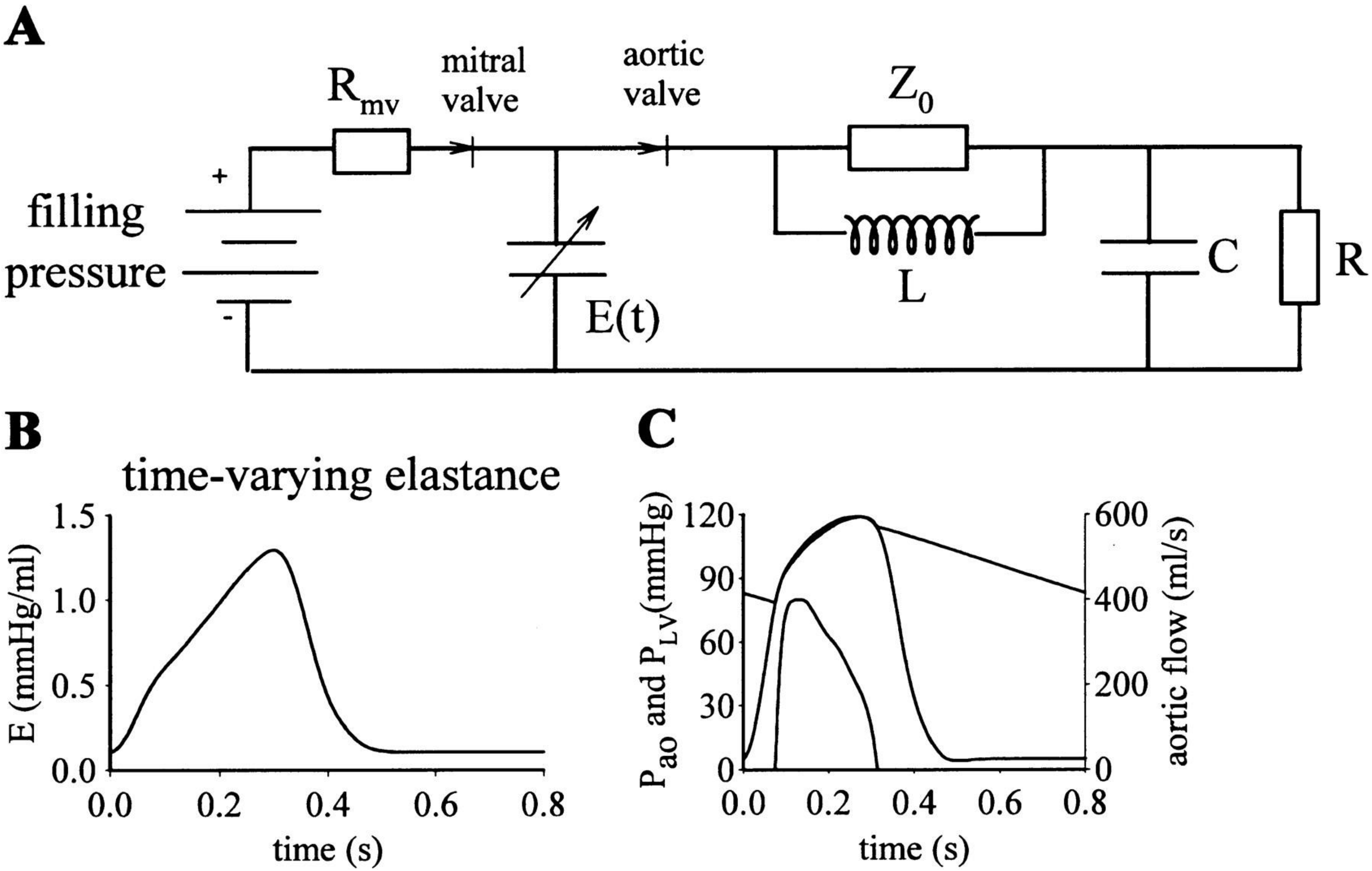
_ASTANCIA ARTERIAL?





The analogy between the fire engine with the Windkessel and the arterial system. The peripheral resistance is the summed resistance of all small arteries, arterioles and capillaries. Total arterial compliance is the sum of the compliances of all arteries, mainly the conduit arteries. The Windkessel model can help us to understand how the arterial system functions, can be used as a realistic load in isolated heart studies, can be used in modeling, and can form the basis for estimating arterial system parameters. Adapted from [1], used by permission.

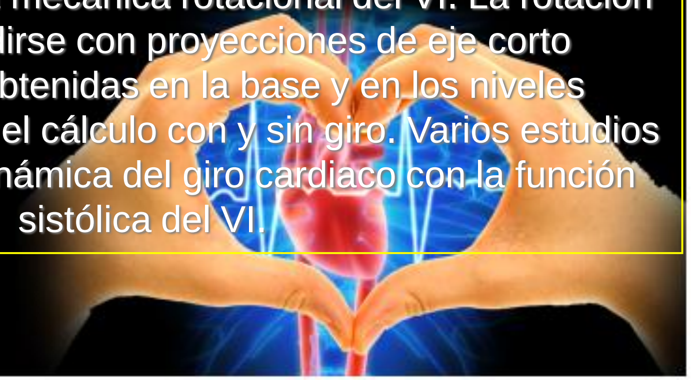
ELASTANCIA ARTERIAL



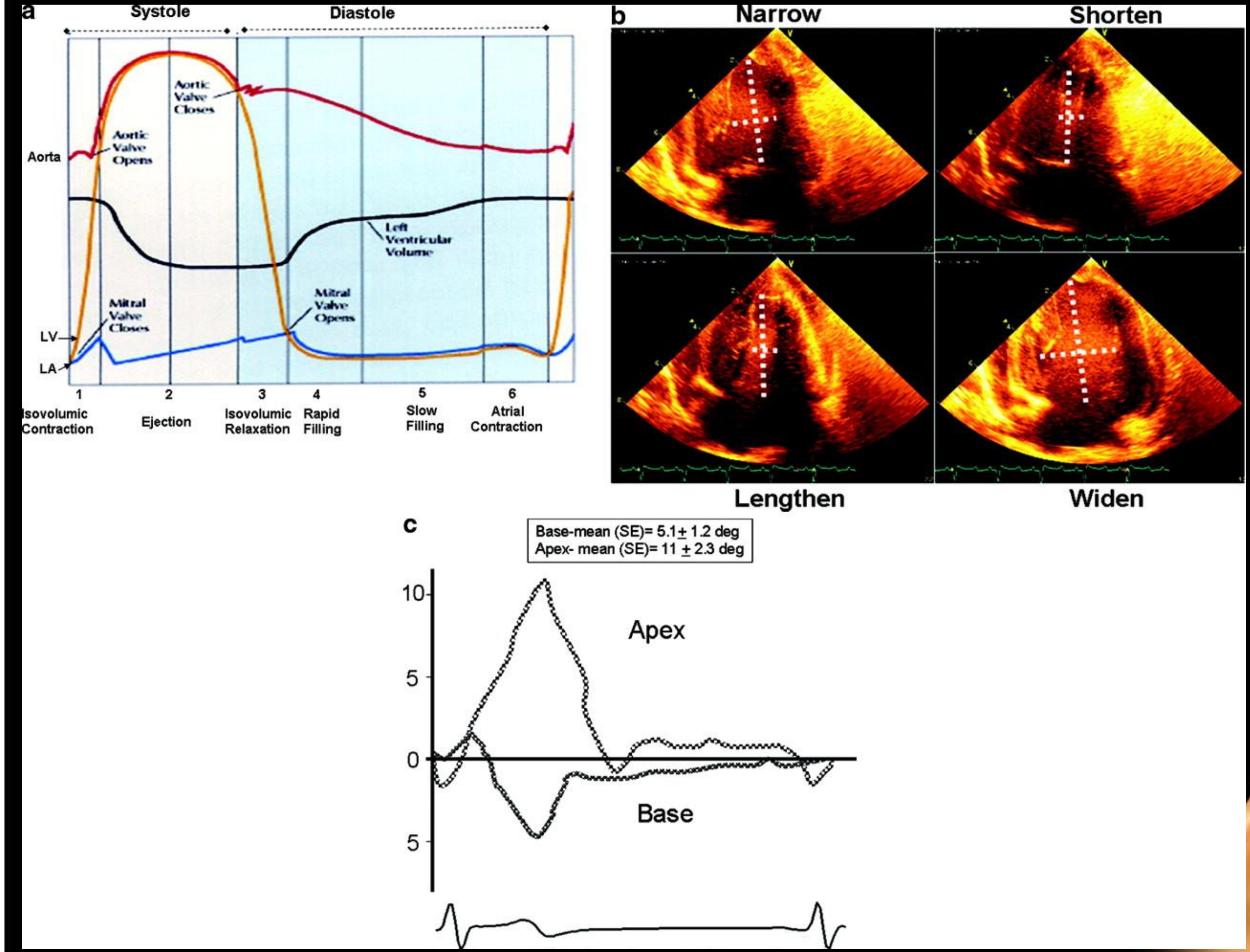
TORSION VENTRICULAR

Mecánica ventricular izquierda mediante ecocardiografía speckle tracking bidimensional

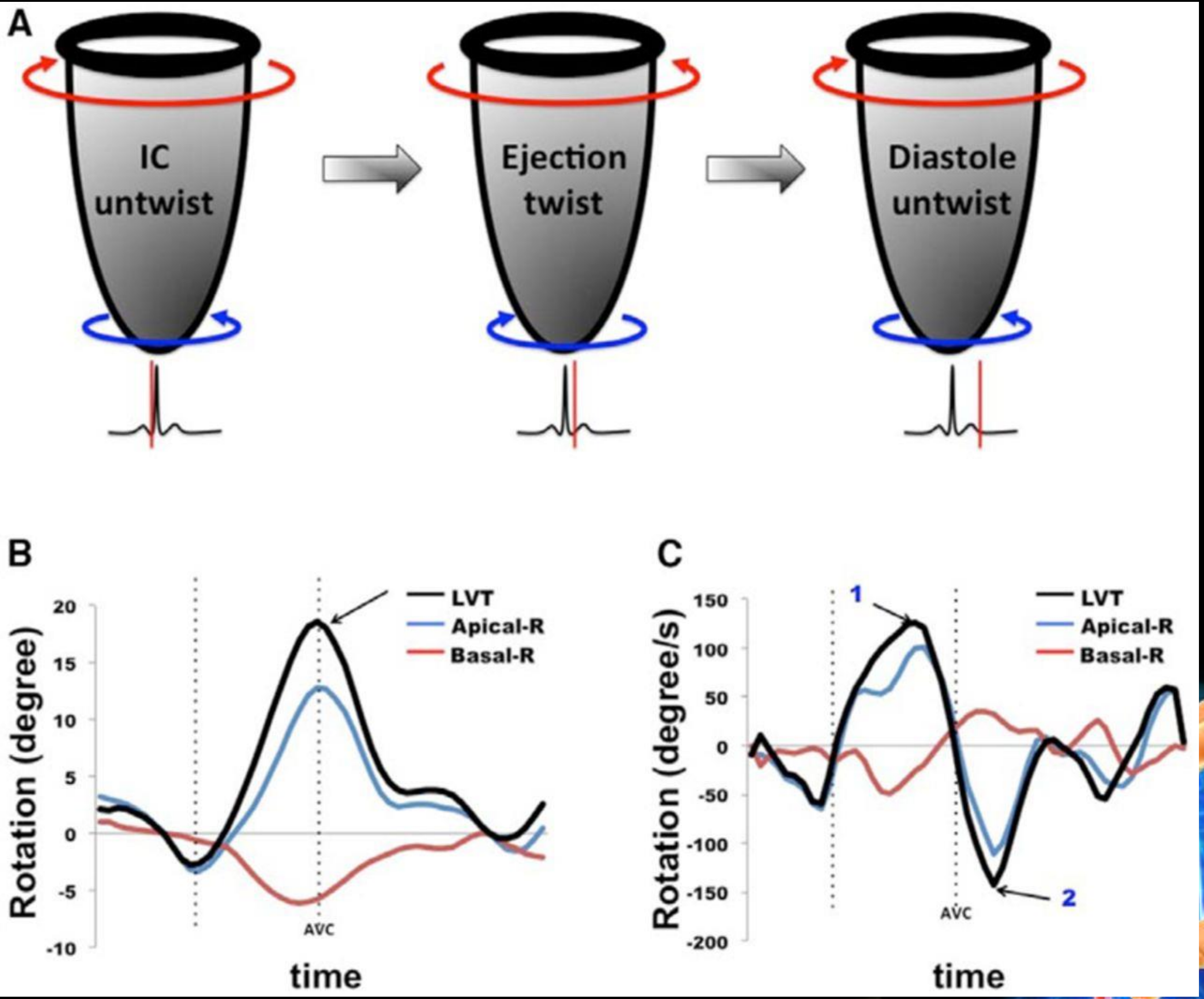
La ecocardiografía con speckle tracking bidimensional puede usarse para evaluar la mecánica rotacional del VI. La rotación del VI puede medirse con proyecciones de eje corto bidimensionales obtenidas en la base y en los niveles apicales, para permitir el cálculo con y sin giro. Varios estudios han relacionado la dinámica del giro cardiaco con la función sistólica del VI.



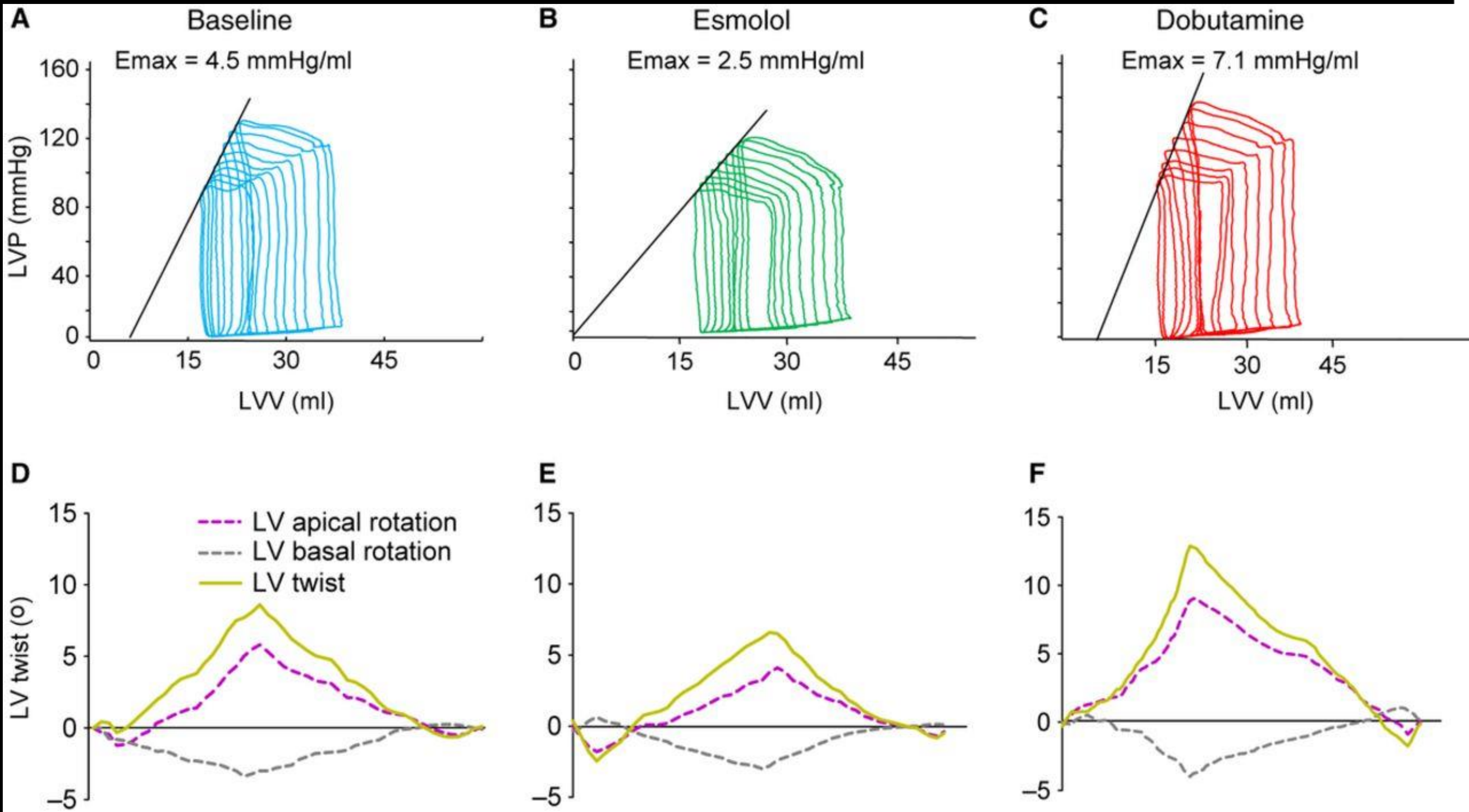
Mecánica ventricular izquierda mediante ecocardiografía speckle tracking bidimensional



TWIST


A
IC
untwist
Ejection
twist
Diastole
untwist
B
Rotation (degree)
20
15
10
5
0
-5
-10
AVC
time
LVT
Apical-R
Basal-R
C
Rotation (degree/s)
150
100
50
0
-50
-100
-150
-200
1
2
AVC
time
LVT
Apical-R
Basal-R

TWIST y Emax



A
Baseline
Emax = 4.5 mmHg/ml
B
Esmolol
Emax = 2.5 mmHg/ml
C
Dobutamine
Emax = 7.1 mmHg/ml
LVP (mmHg)
160
120
80
40
0
0
15
30
45
LVV (ml)
D
E
F
LV apical rotation
LV basal rotation
LV twist
LV twist (º)
15
10
5
0
−5

ANALISIS DE DATOS

Ver archivo Datos Ventriculares

Muchas Gracias