Ventilator Associated Lung Injury

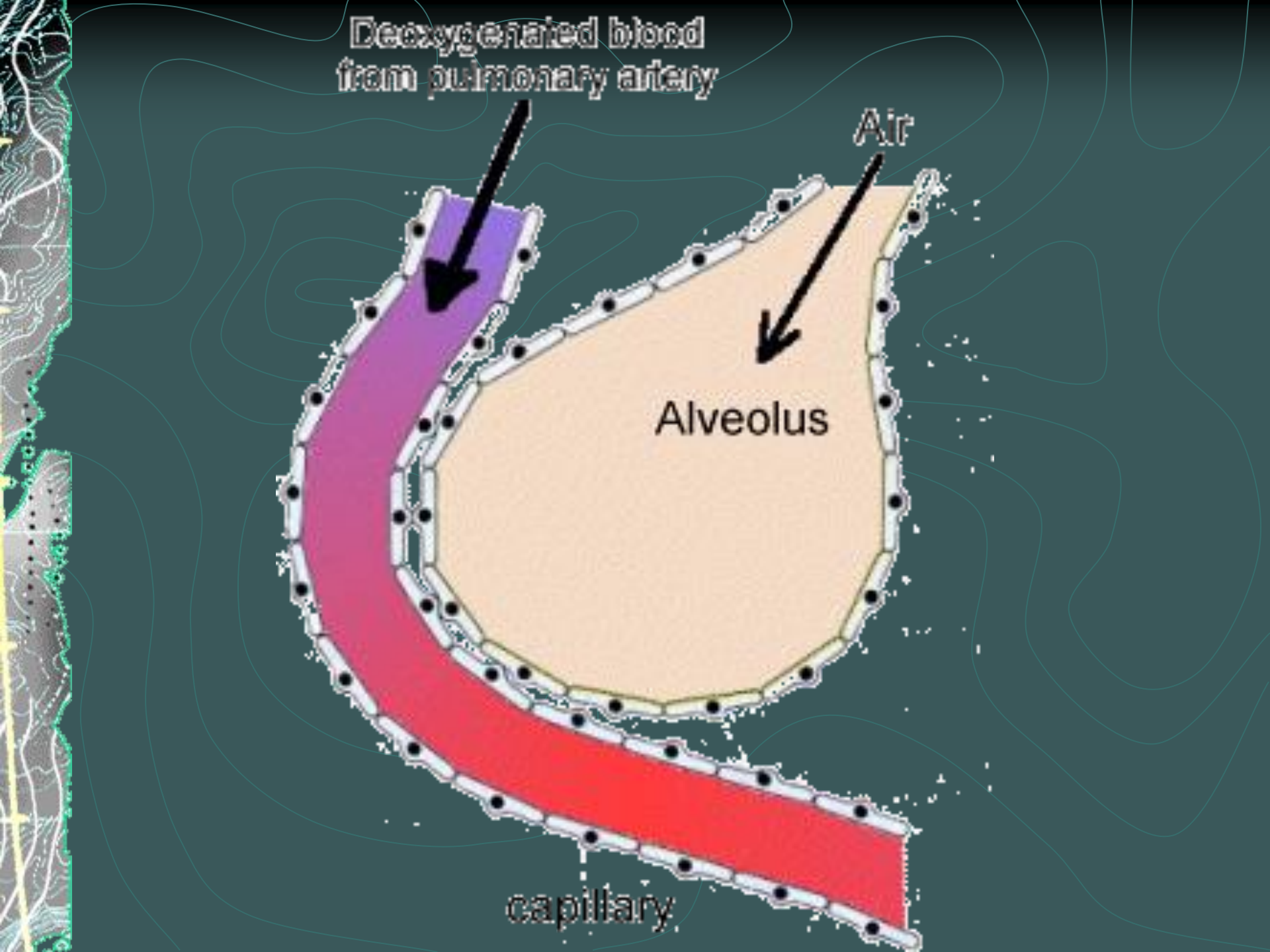
Yaron Bar-Lavie, M.D., FCCP
Director, Dept. of Critical Care Medicine
Rambam Medical Ctr., Haifa

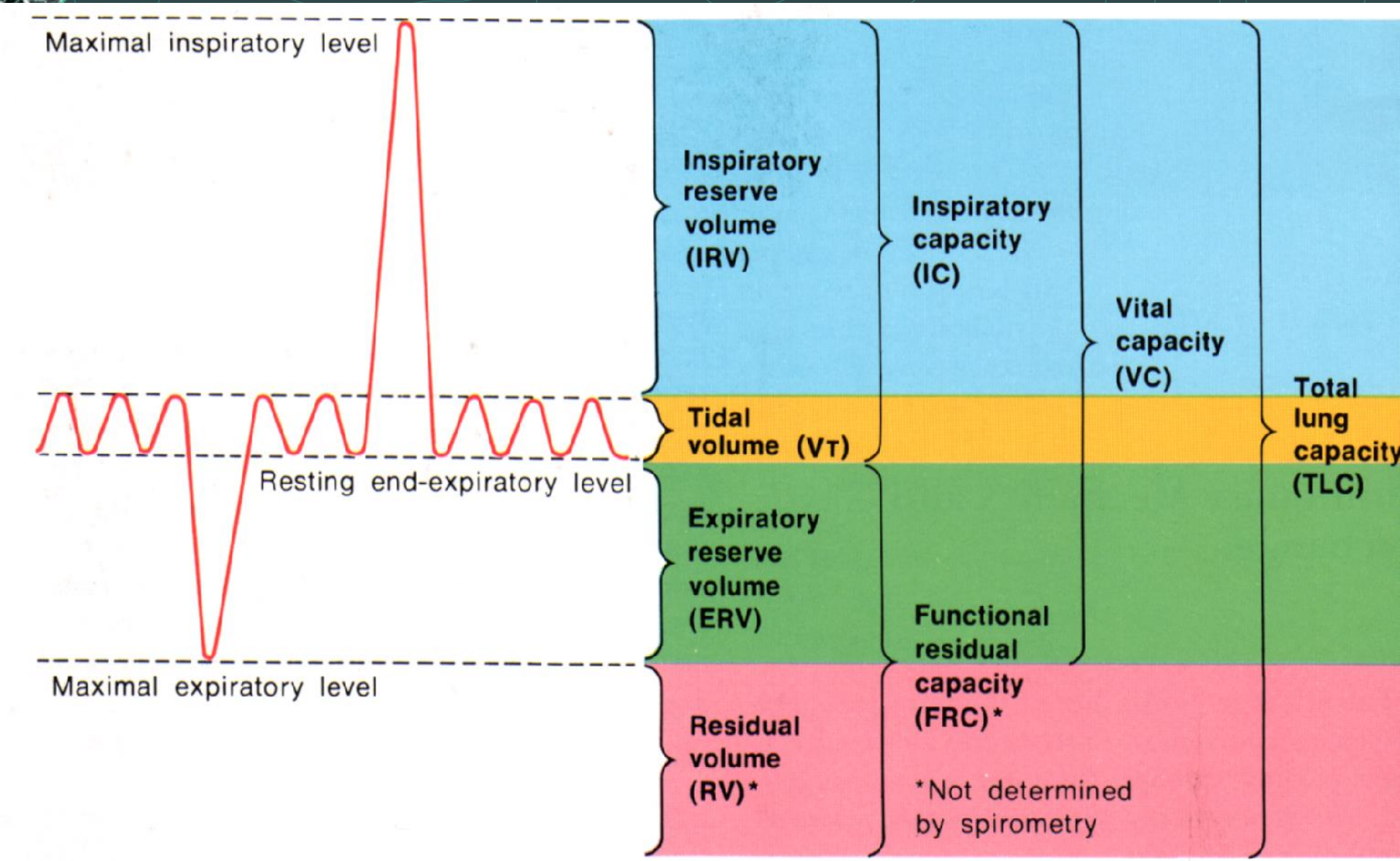
Benefits of Mechanical Ventilation

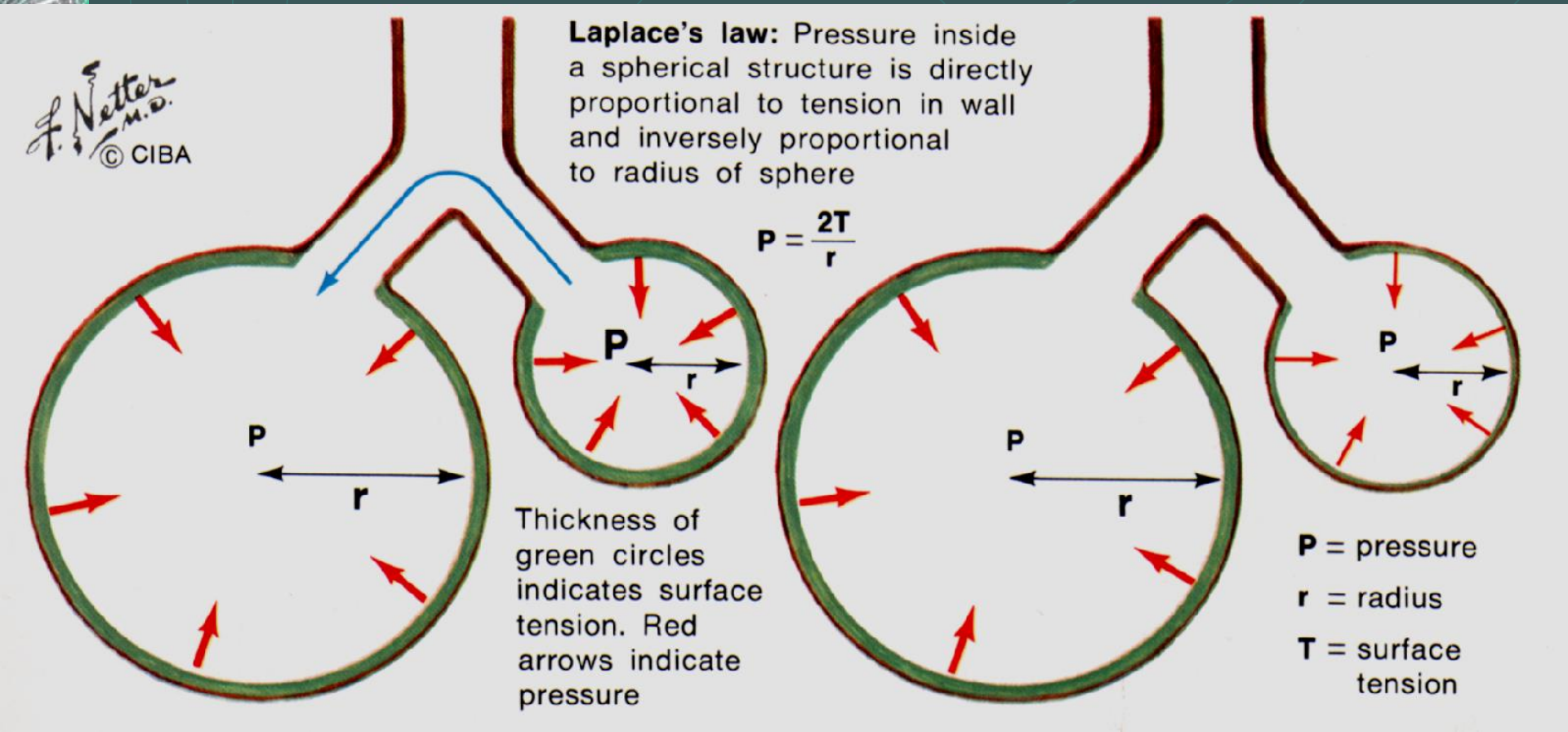
- O2 Delivery
- CO2 Removal
- Reduce WOB
- Recruitment of Atelectatic alveoli
- Time for healing, recovery to occur

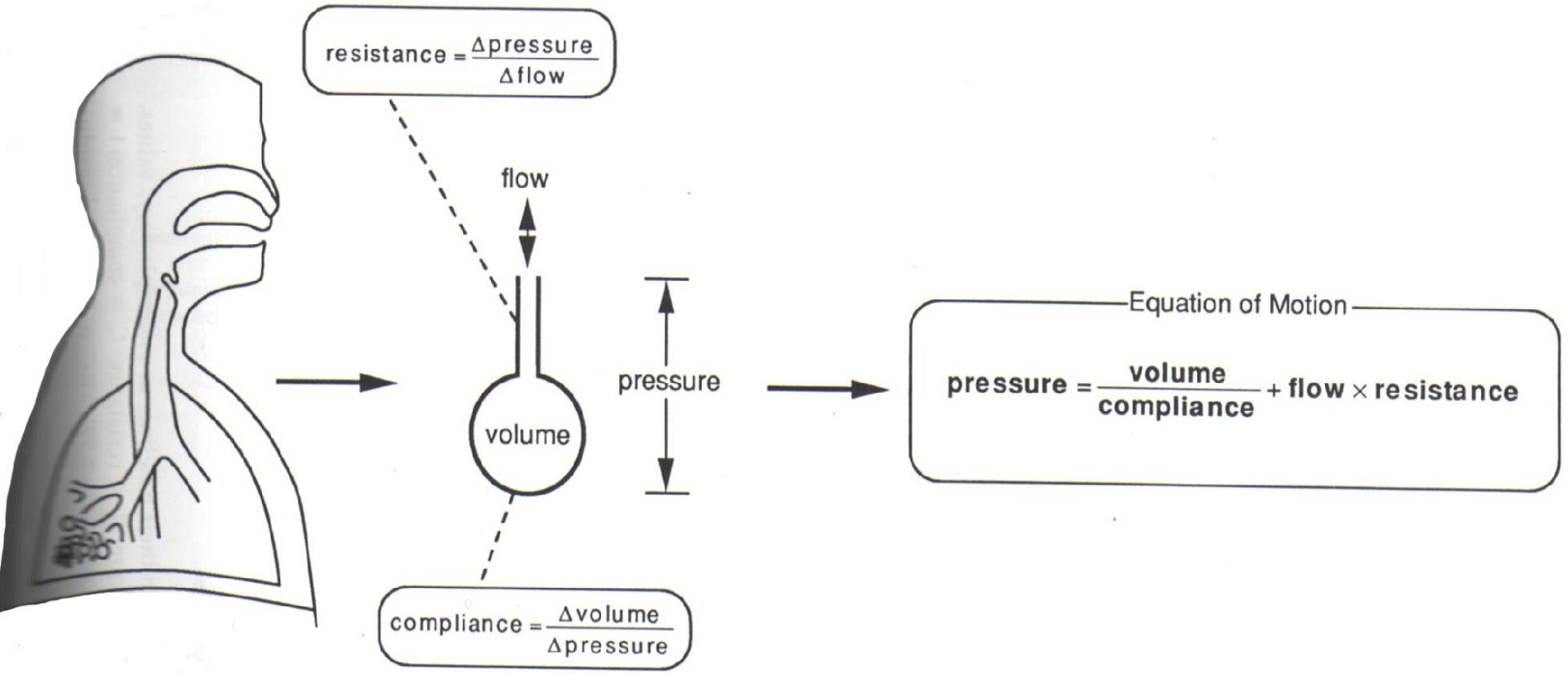
Risks of Mech. Vent.

- Ventilator Induced Lung Injury – VILI
- Ventilator Associated Pneumonia – VAP
- Disuse Atrophy – of respiratory muscles
- Reduced Cardiac Output
- Gut paresis, ischemia
- Sepsis (Aspiration, colonization, translocation)
- MODS

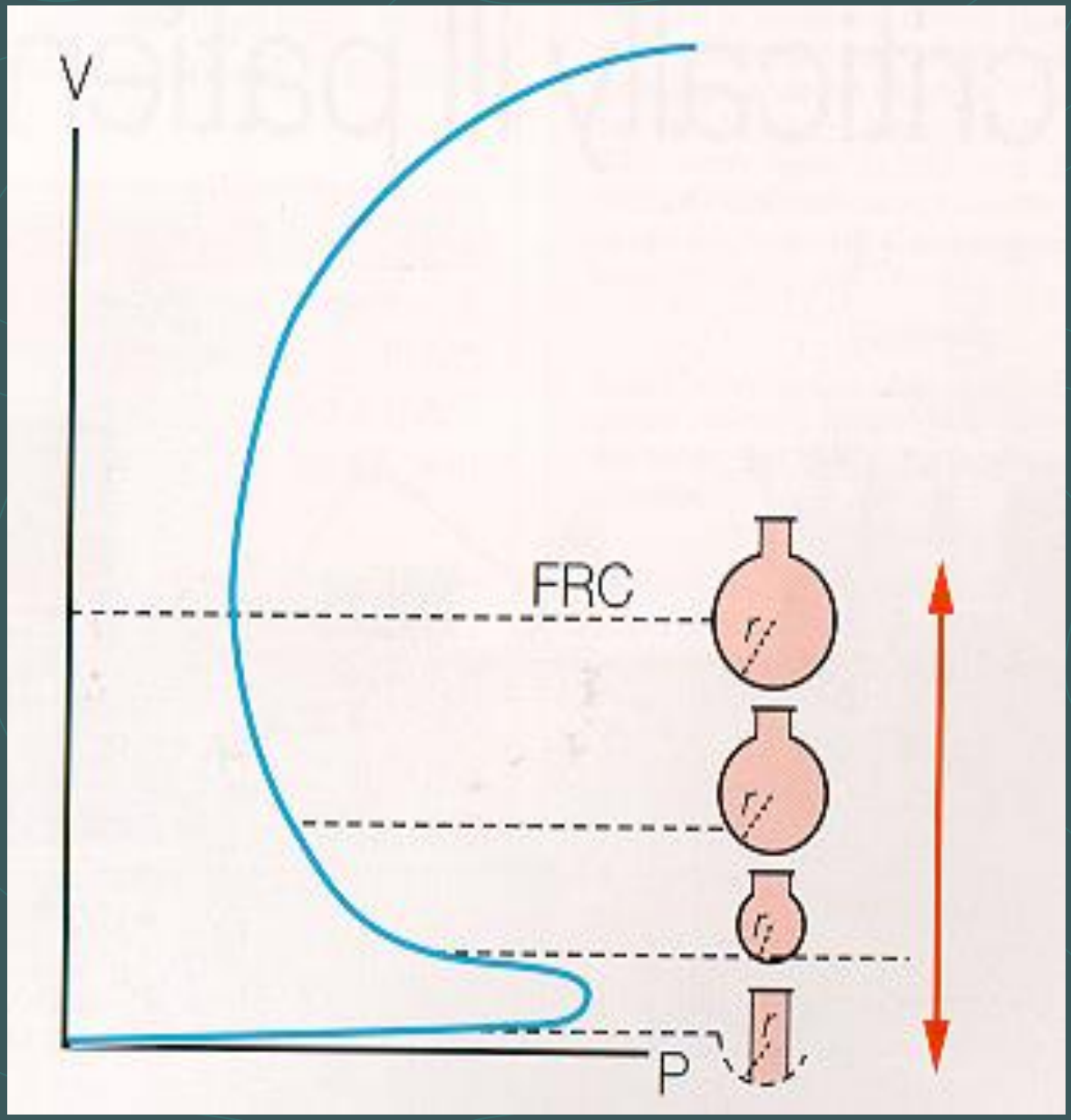

Deoxygenated blood
from pulmonary artery
Air
Alveolus
capillary


Maximal inspiratory level
Resting end-expiratory level
Maximal expiratory level
Inspiratory reserve volume (IRV)
Tidal volume (VT)
Expiratory reserve volume (ERV)
Residual volume (RV)*
Inspiratory capacity (IC)
Functional residual capacity (FRC)*
Vital capacity (VC)
Total lung capacity (TLC)
*Not determined by spirometry

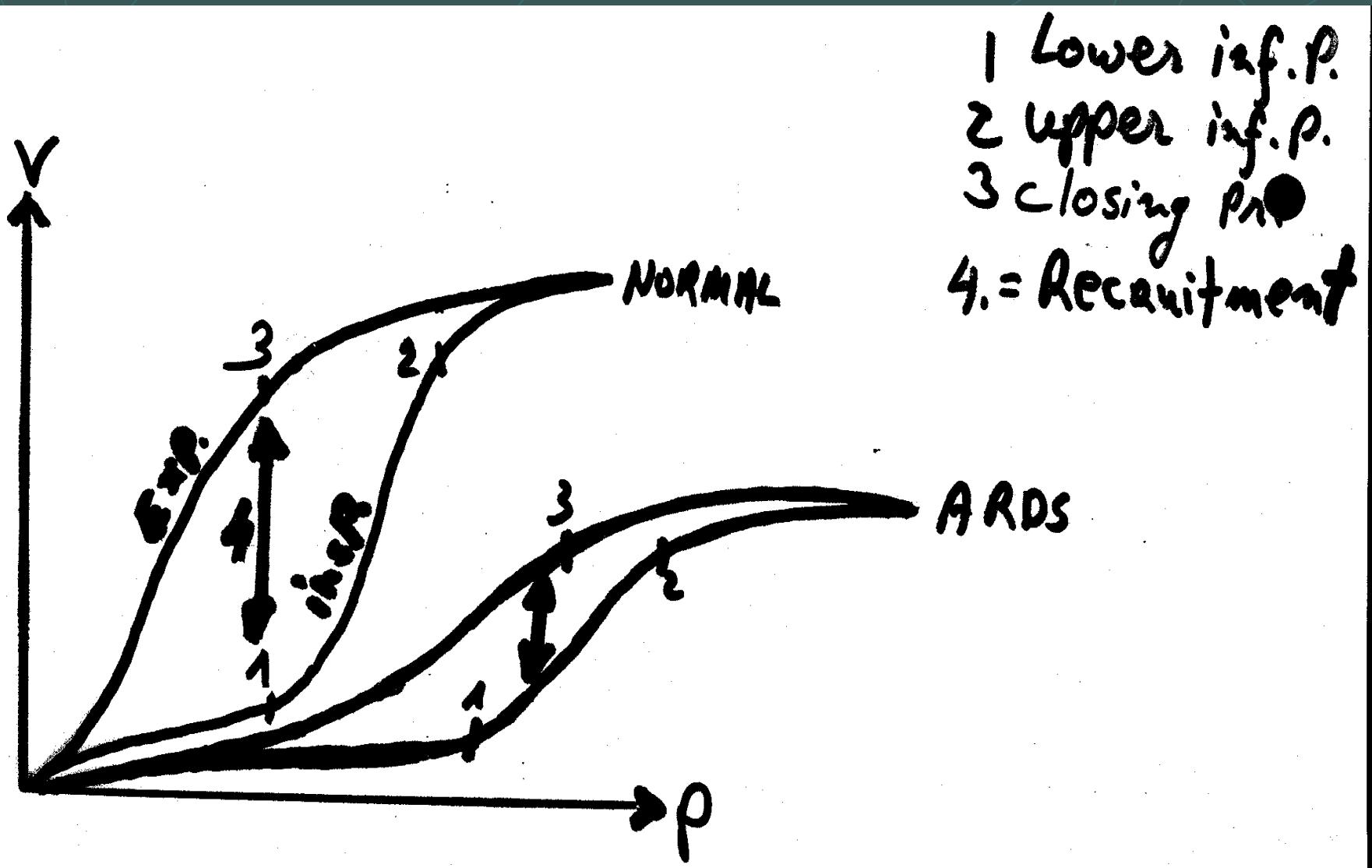

F. Netter M.D.
© CIBA
Laplace's law: Pressure inside a spherical structure is directly proportional to tension in wall and inversely proportional to radius of sphere
$P = \frac{2T}{r}$
P
r
P
r
Thickness of green circles indicates surface tension. Red arrows indicate pressure
P
r
P
r
P = pressure
r = radius
T = surface tension

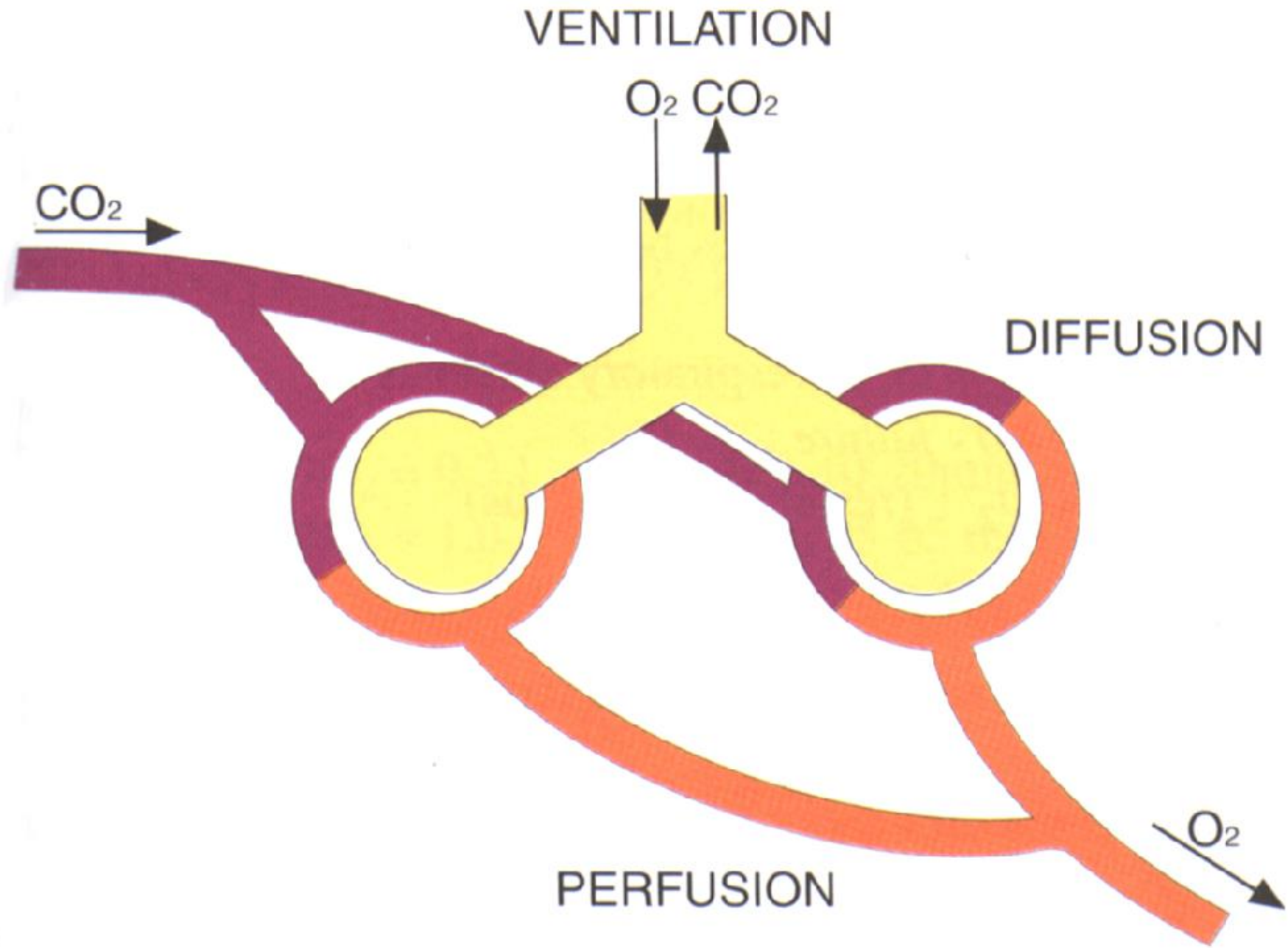

resistance = Δpressure / Δflow
flow
volume
pressure
compliance = Δvolume / Δpressure
Equation of Motion
pressure = volume / compliance + flow × resistance

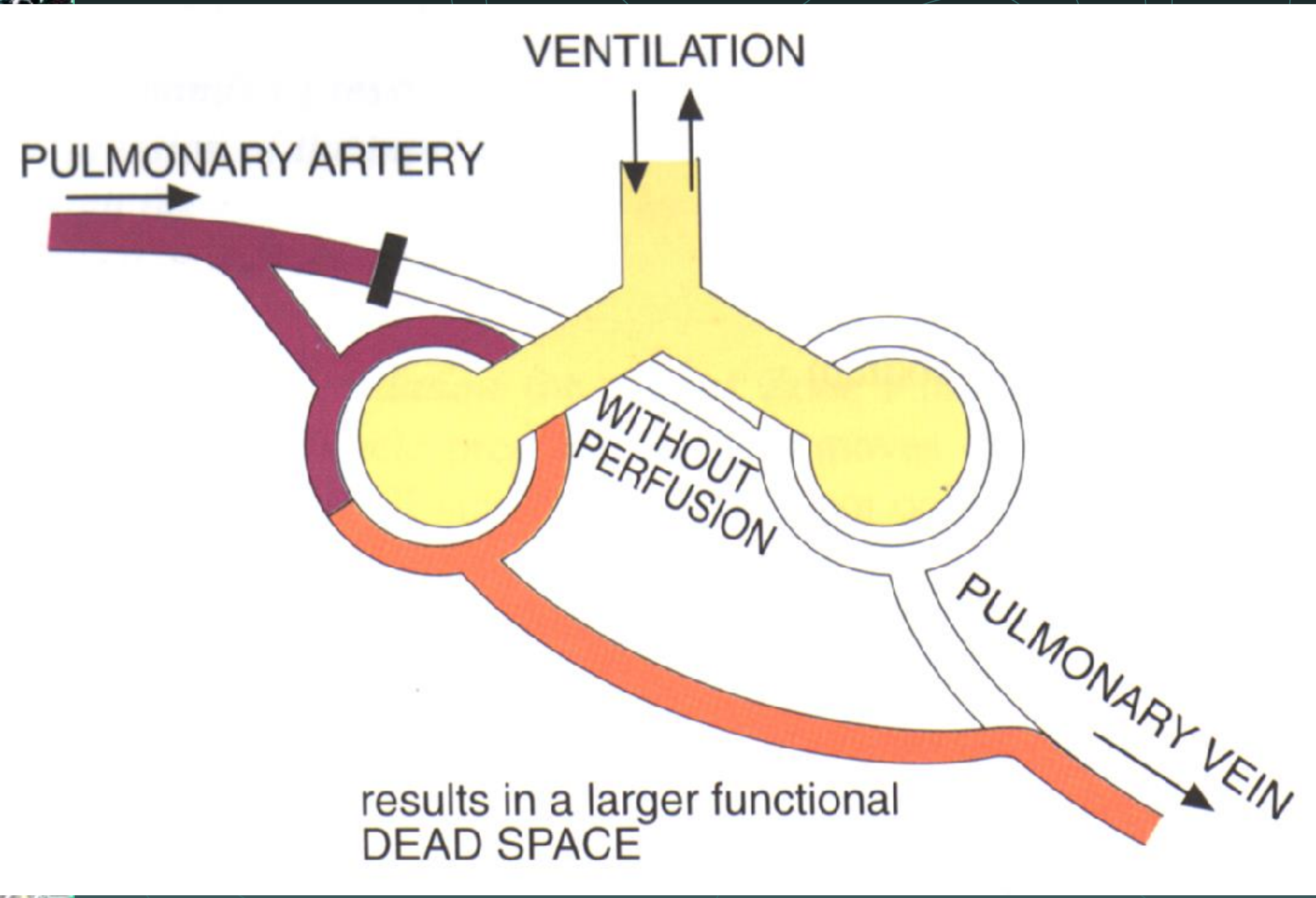
Lung Inflation

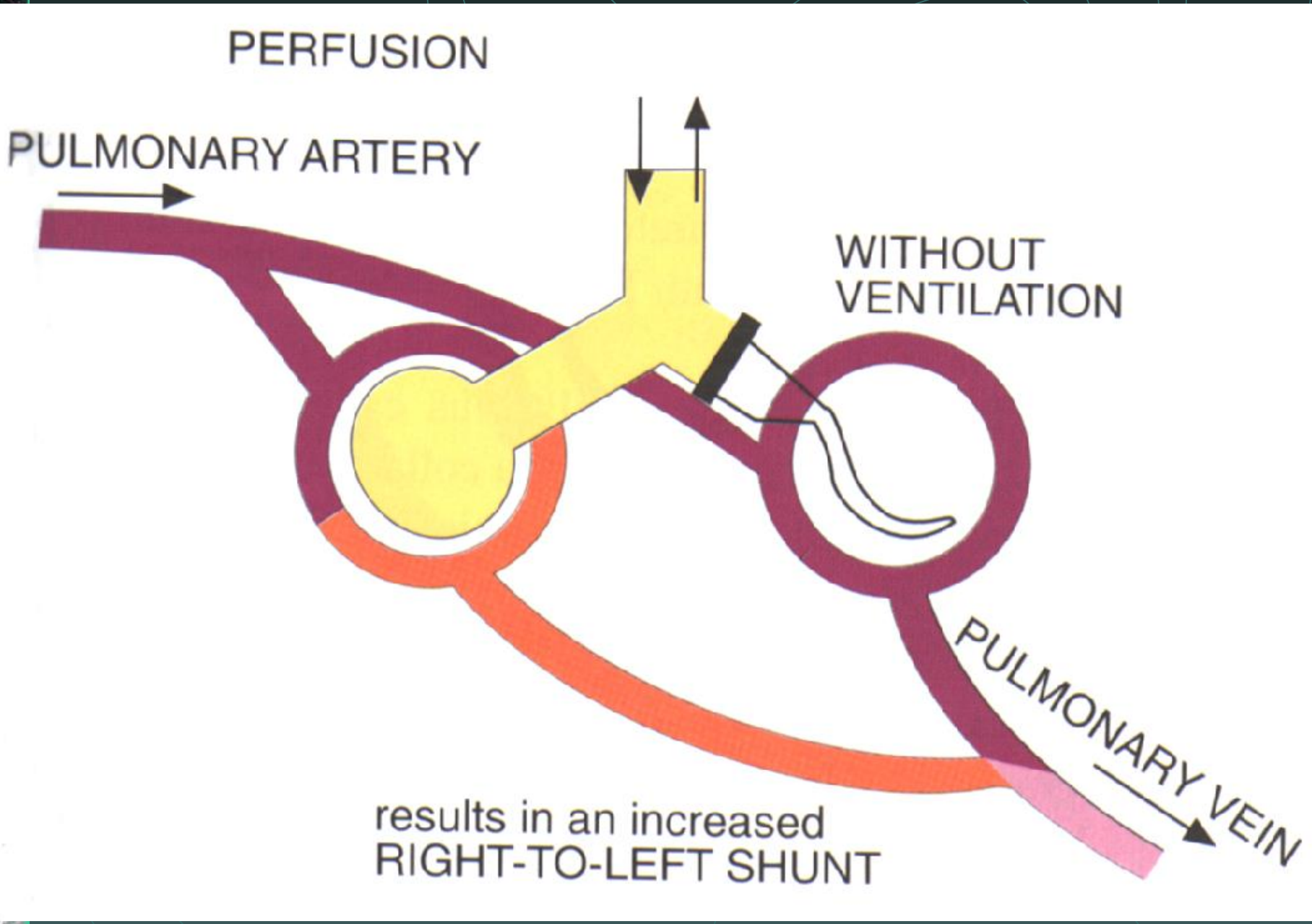


Lung Inflation - deflation

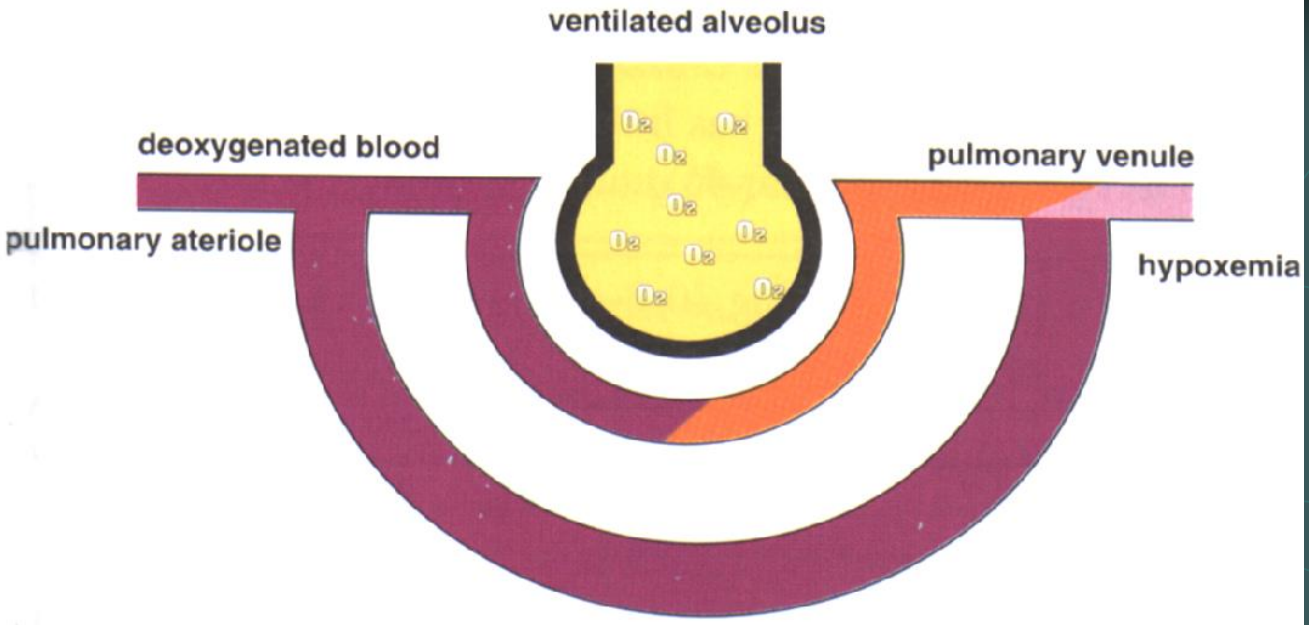



VENTILATION
O_2 CO_2
CO_2
DIFFUSION
O_2
PERFUSION

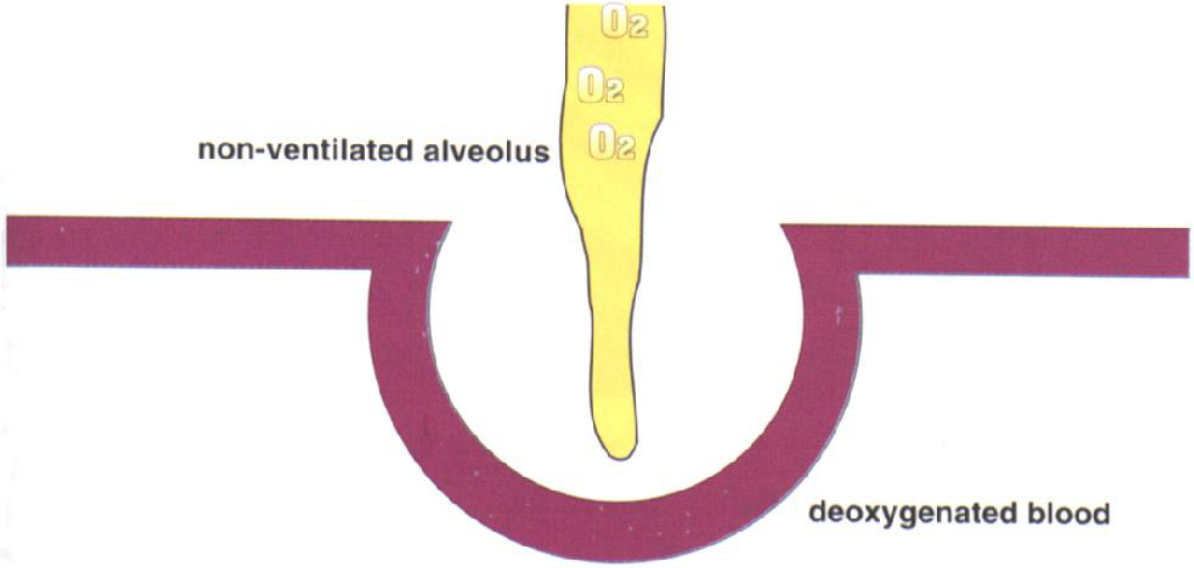

VENTILATION
PULMONARY ARTERY
WITHOUT PERFUSION
PULMONARY VEIN
results in a larger functional DEAD SPACE


PERFUSION
PULMONARY ARTERY
WITHOUT
VENTILATION
PULMONARY VEIN
results in an increased
RIGHT-TO-LEFT SHUNT

Anatomical Shunt



Functional Shunt



Compartment model of the lung

A group of identical breathing mechanical sub-units is referred to as a Compartment

Compartment model of the lung

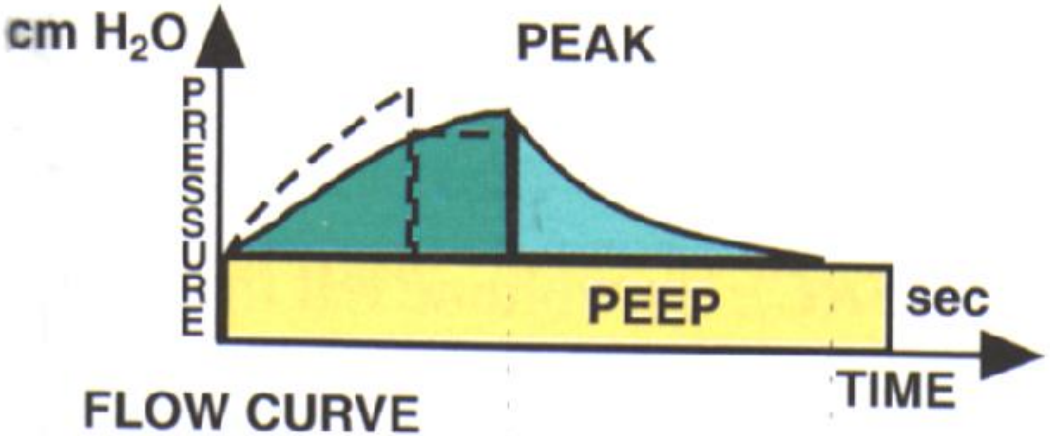
- Time constant τ=RxC
- τ= insp. or exp. time in seconds
- One τ= 63% exhalation
- 3τ = 95% exhalation
- High Resistance increases τ
- Low Compliance reduces τ

Different time constants

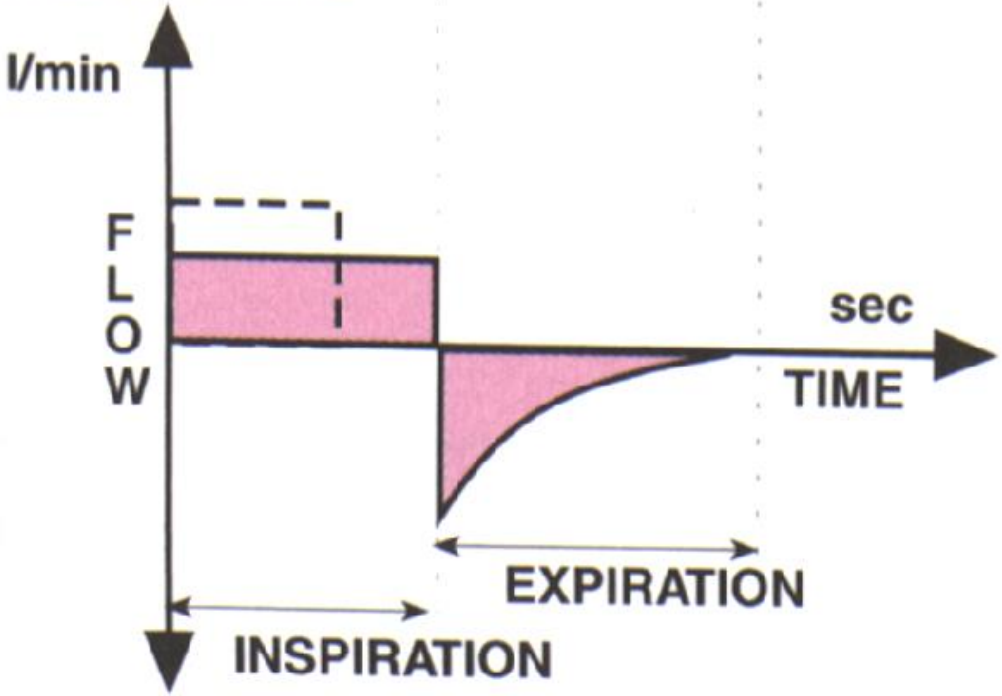
Status	R	C	τ
Normal	2	0.1	0.2 sec
Post surgery	5	0.06	0.3
COPD	15	0.06	0.9
ARDS	8	0.03	0.24

- The lung has many compartments with different τ

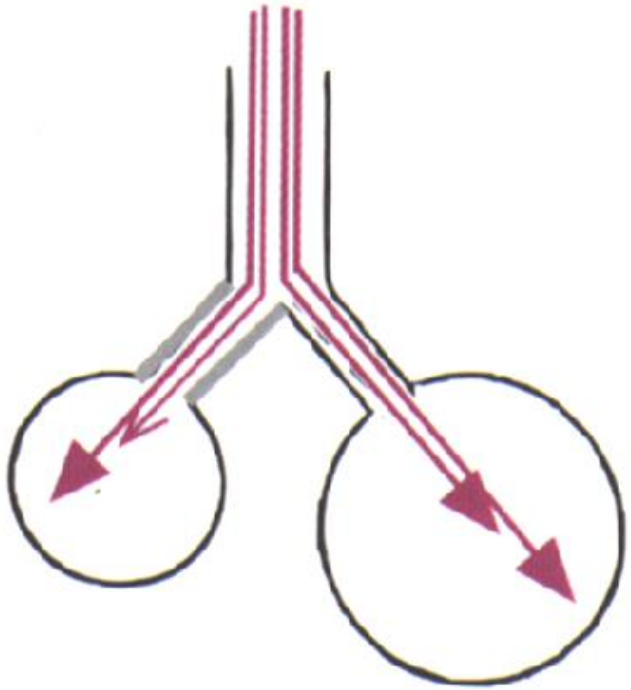
PRESSURE CURVE



FLOW CURVE

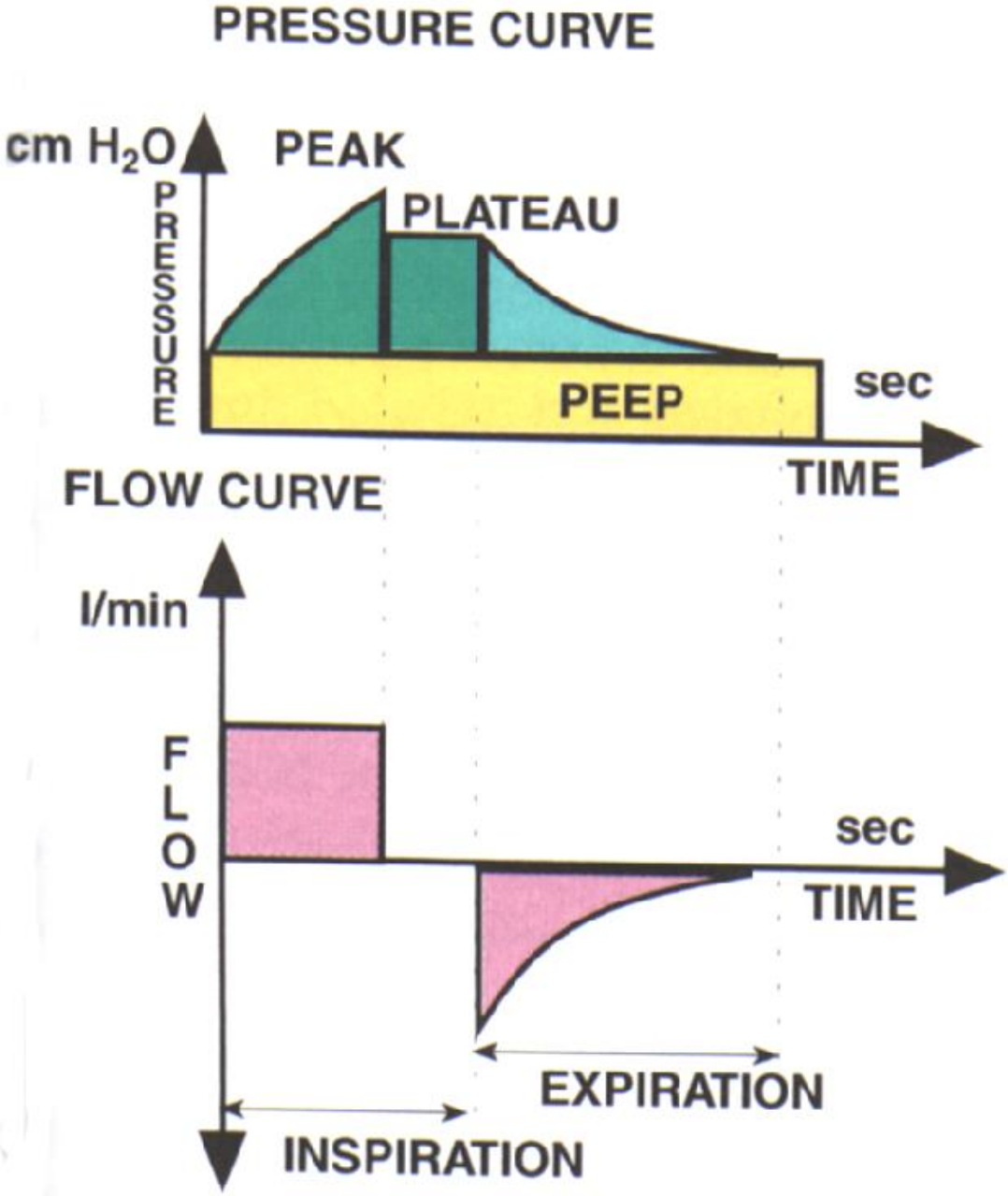


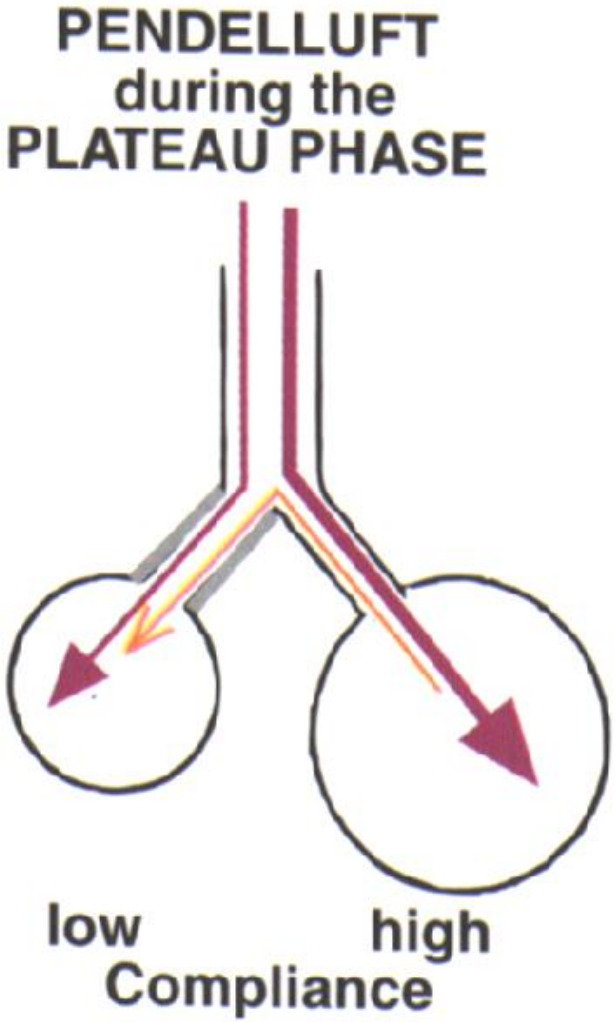
No PENDELLUFT because of constant low flow



low high

Compliance


PRESSURE CURVE
cm H_2O
PEAK
PRESSURE
PLATEAU
PEEP
sec
TIME
FLOW CURVE
l/min
FLOW
sec
TIME
EXPIRATION
INSPIRATION


PENDELLUFT
during the
PLATEAU PHASE
low
high
Compliance

In MV Flow and Pressure are applied to the Airway in order to effect Lung Volume

- Reduced Surfactant production and it's loss
- Time constants are changed
- Dependent atelectasis
- Non dependent over-distension
- Alveolar edema, bleeding, hyaline membranes
- FRC is reduced
- Dead Space is enlarged
- Shunt fraction is enlarged - v/q changes

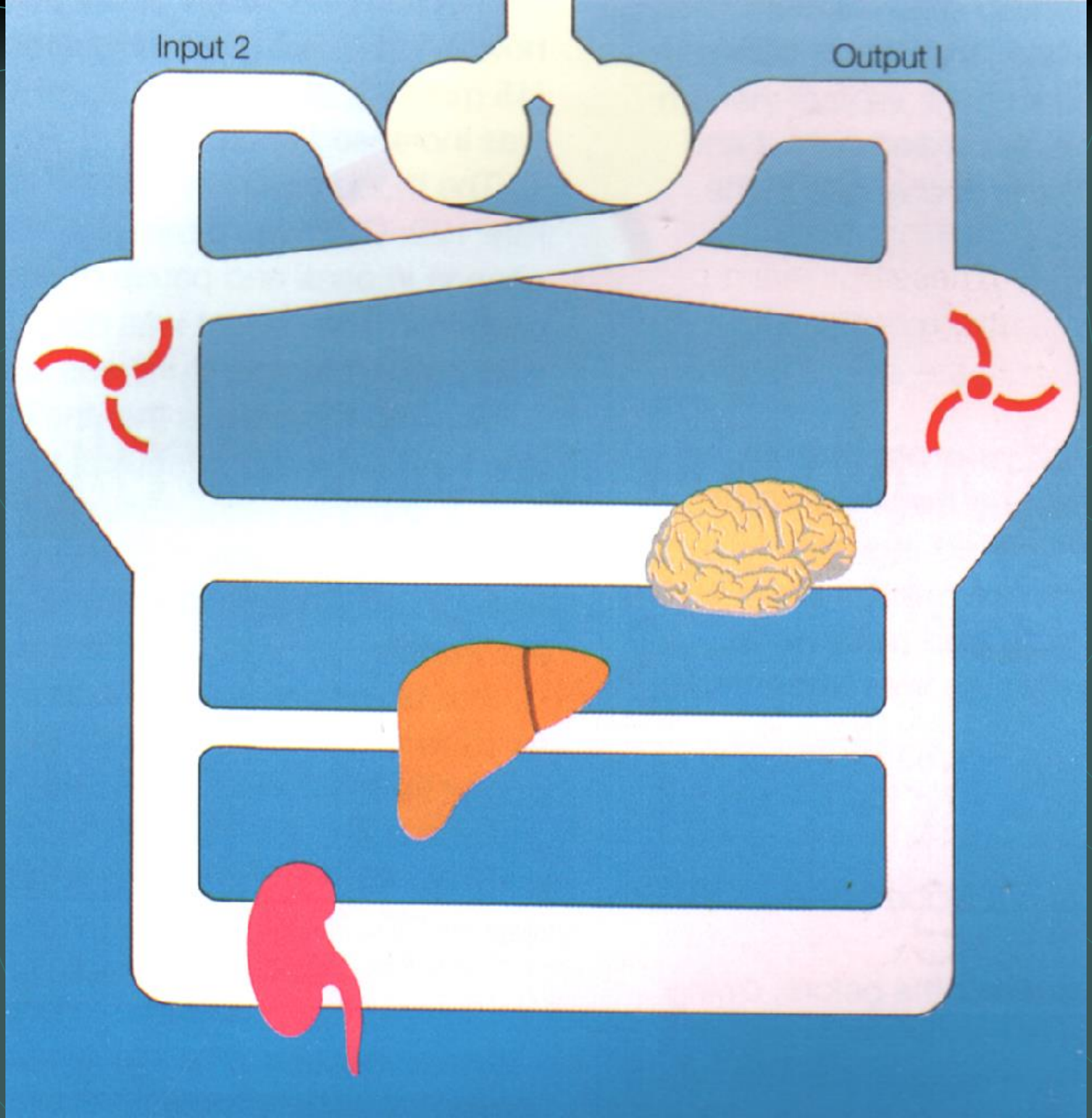
Complications – Barotrauma, Volutrauma

- Interstitial emphysema, pneumo-mediastinum, pneumo-peritoneum, sq emphysema, bulla formation, pneumo-thorax, tension pneumo-thorax, Broncho-Pleural Fistula

Conclusion

- Prolonged hyper-expansion of normal lung results in Parenchimal injury due to the mechanical ventilation itself !!!

The Effects of MV on Vital Organ Function


Input 2
Output I

When ***ventilating with PEEP*** considerations must be given to

venous return ↓

|

cardiac output ↓

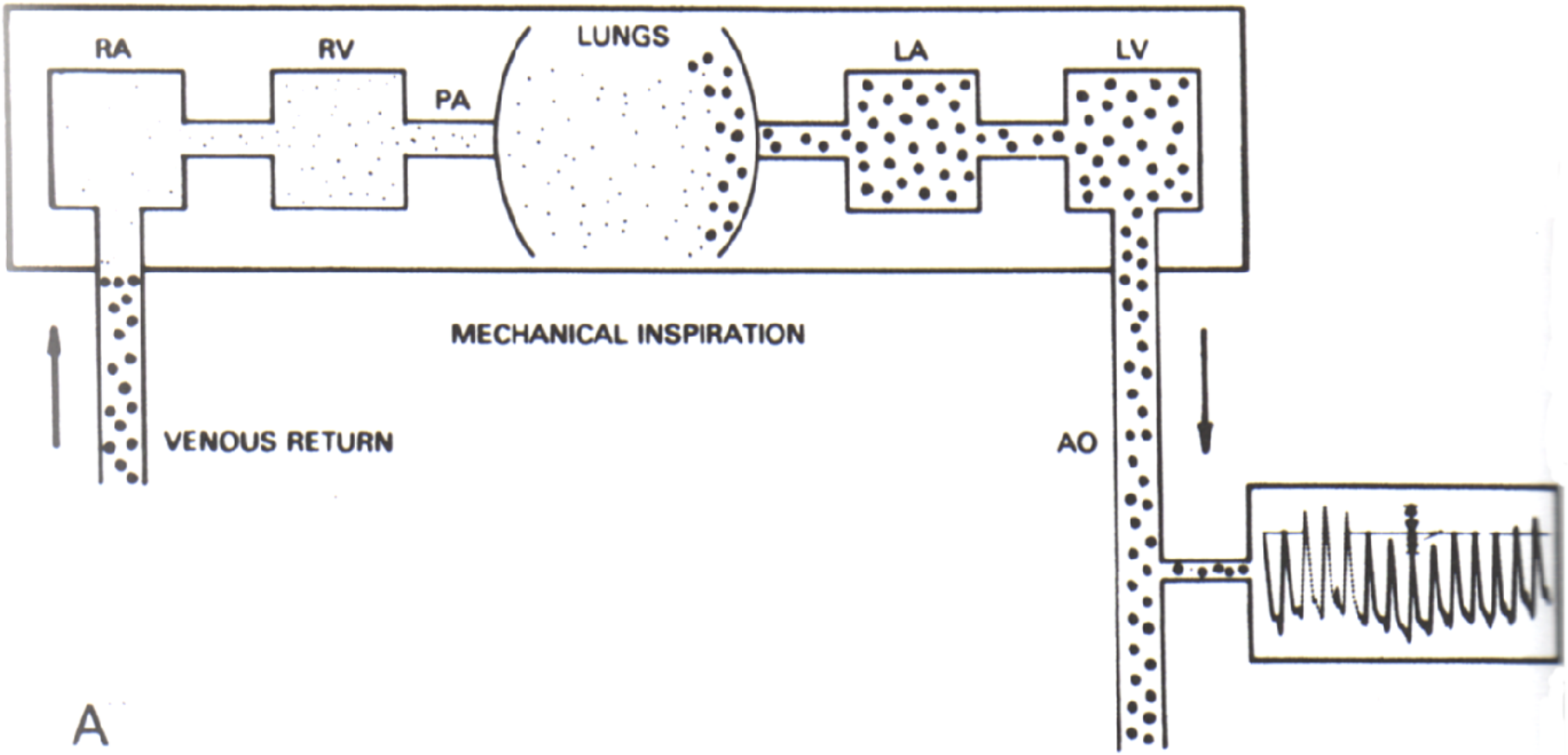
|

blood pressure ↓

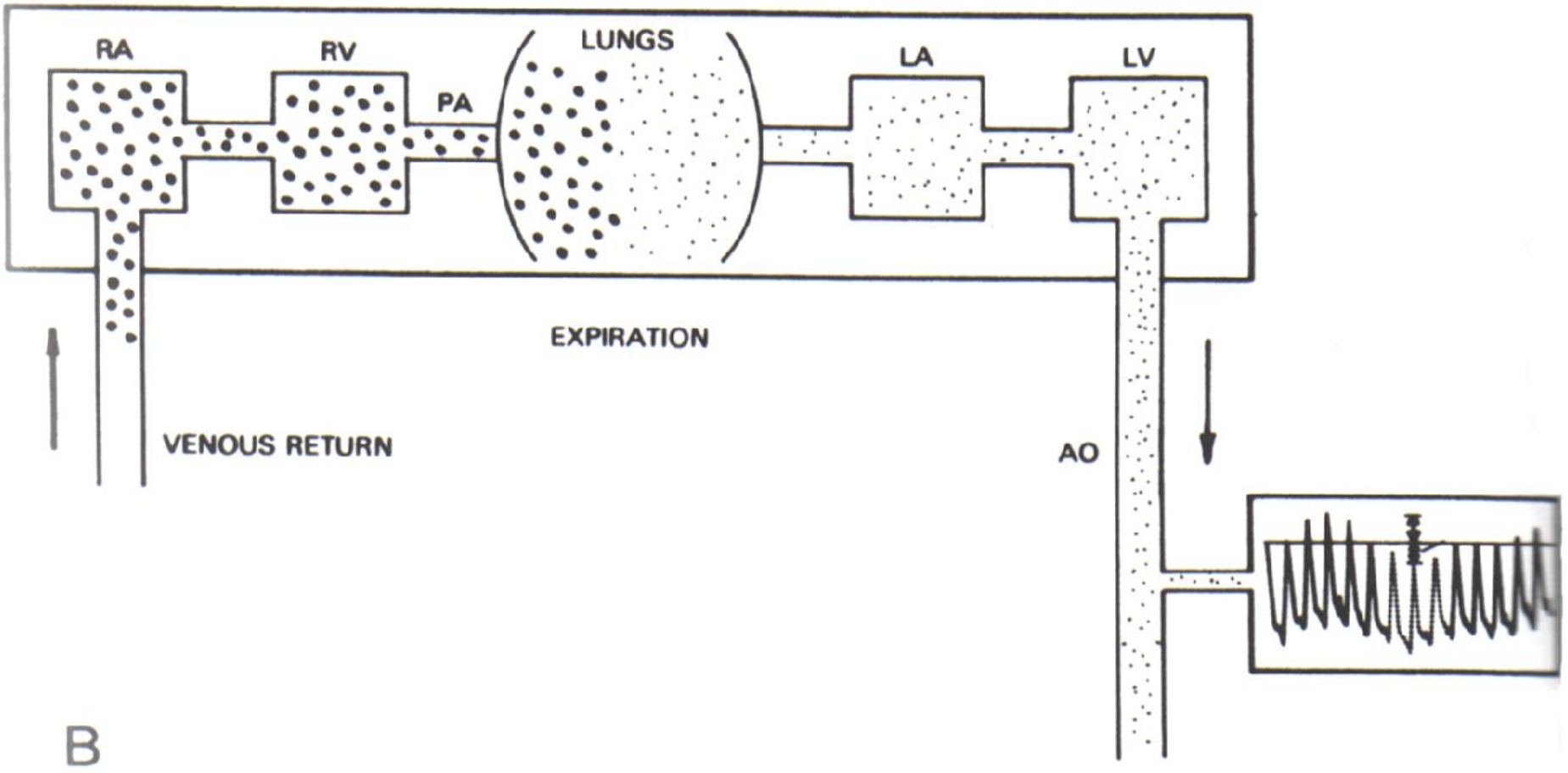
|

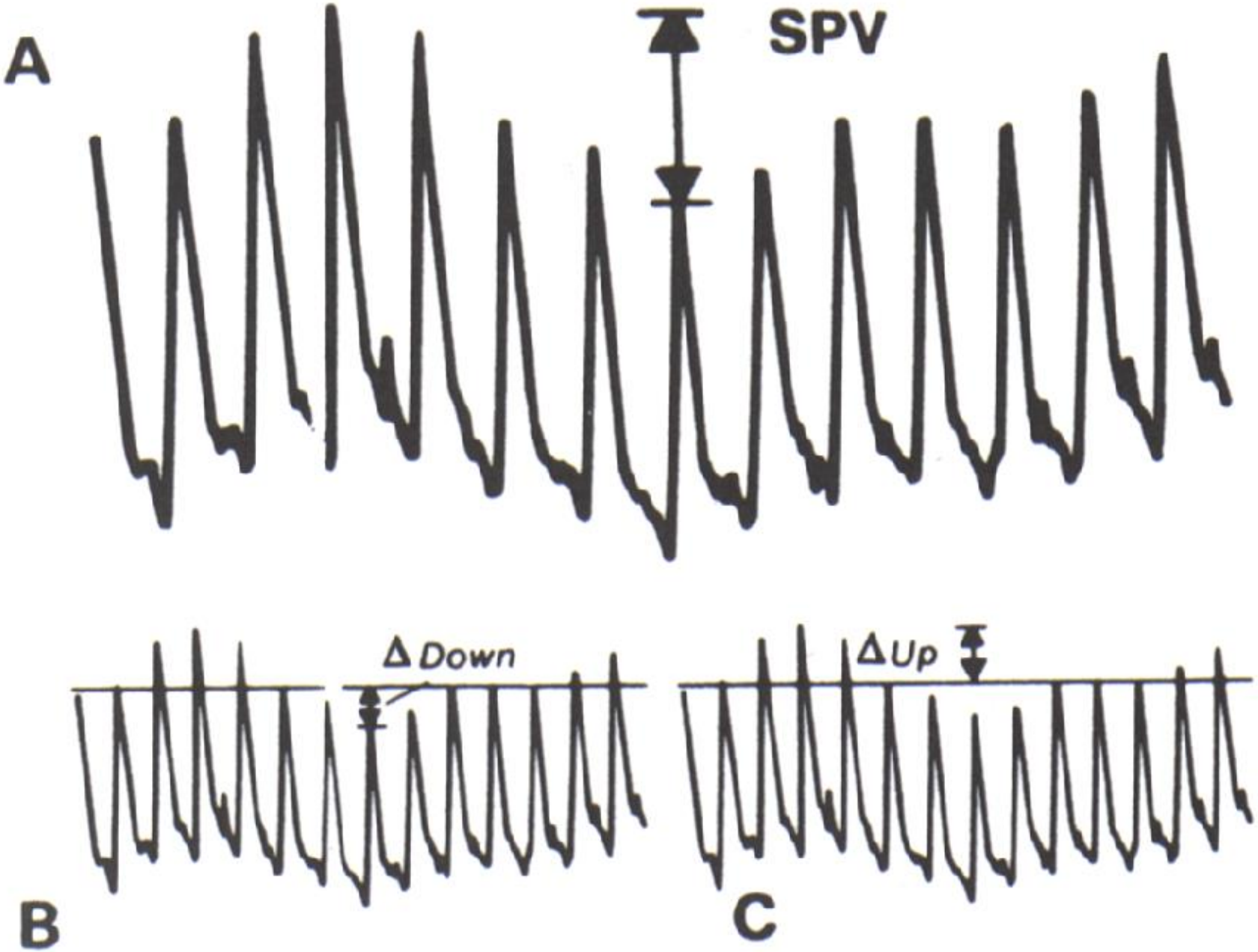
organ perfusion ↓

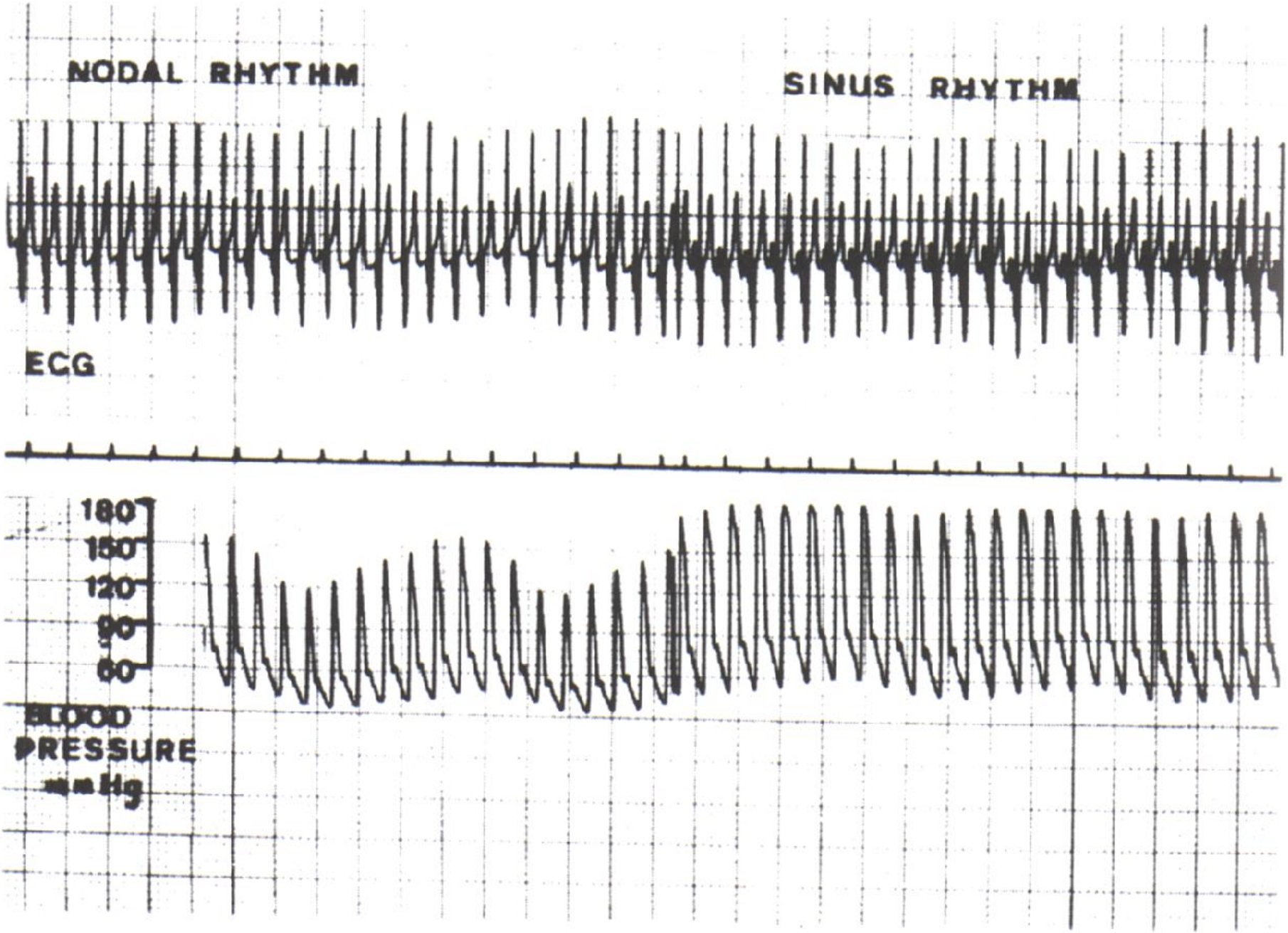
Cardio-Vascular System

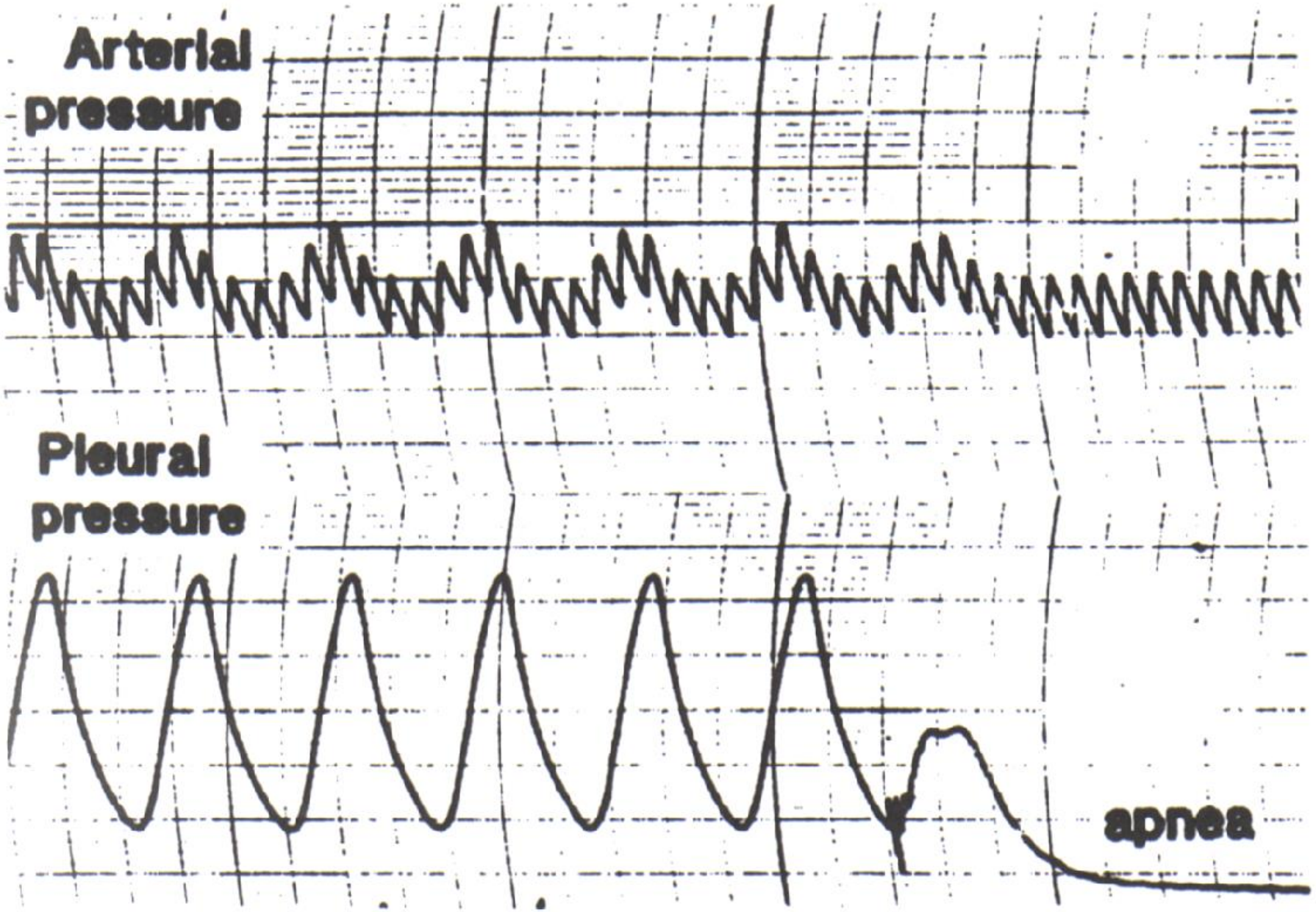


Cardio-Vascular System

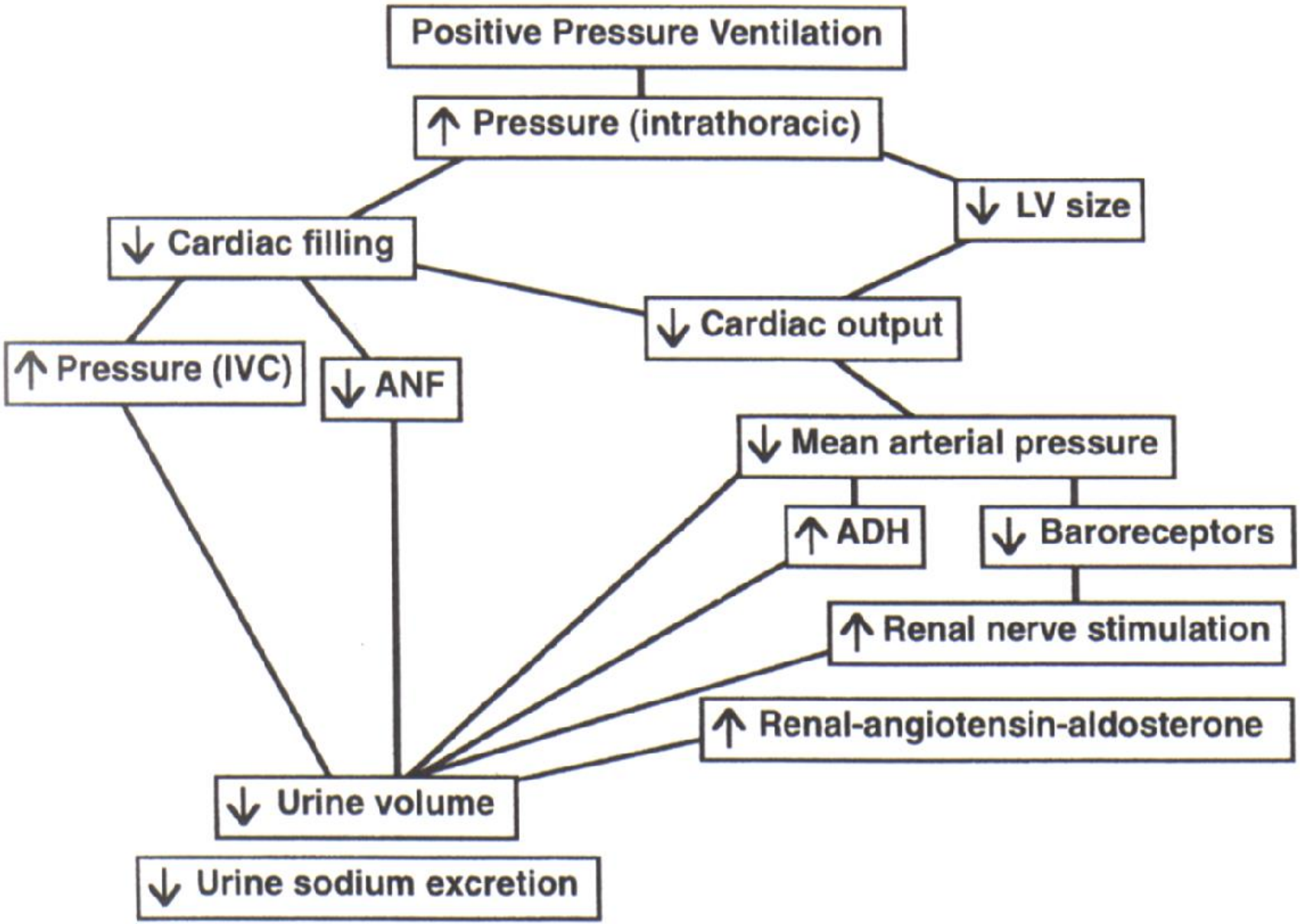



A
SPV
ΔDown
ΔUp
B
C


NODAL RHYTHM
SINUS RHYTHM
ECG
180
150
120
90
60
BLOOD PRESSURE
mm Hg


Arterial pressure
Pleural pressure
apnea

Mech. Vent. and the Kidney

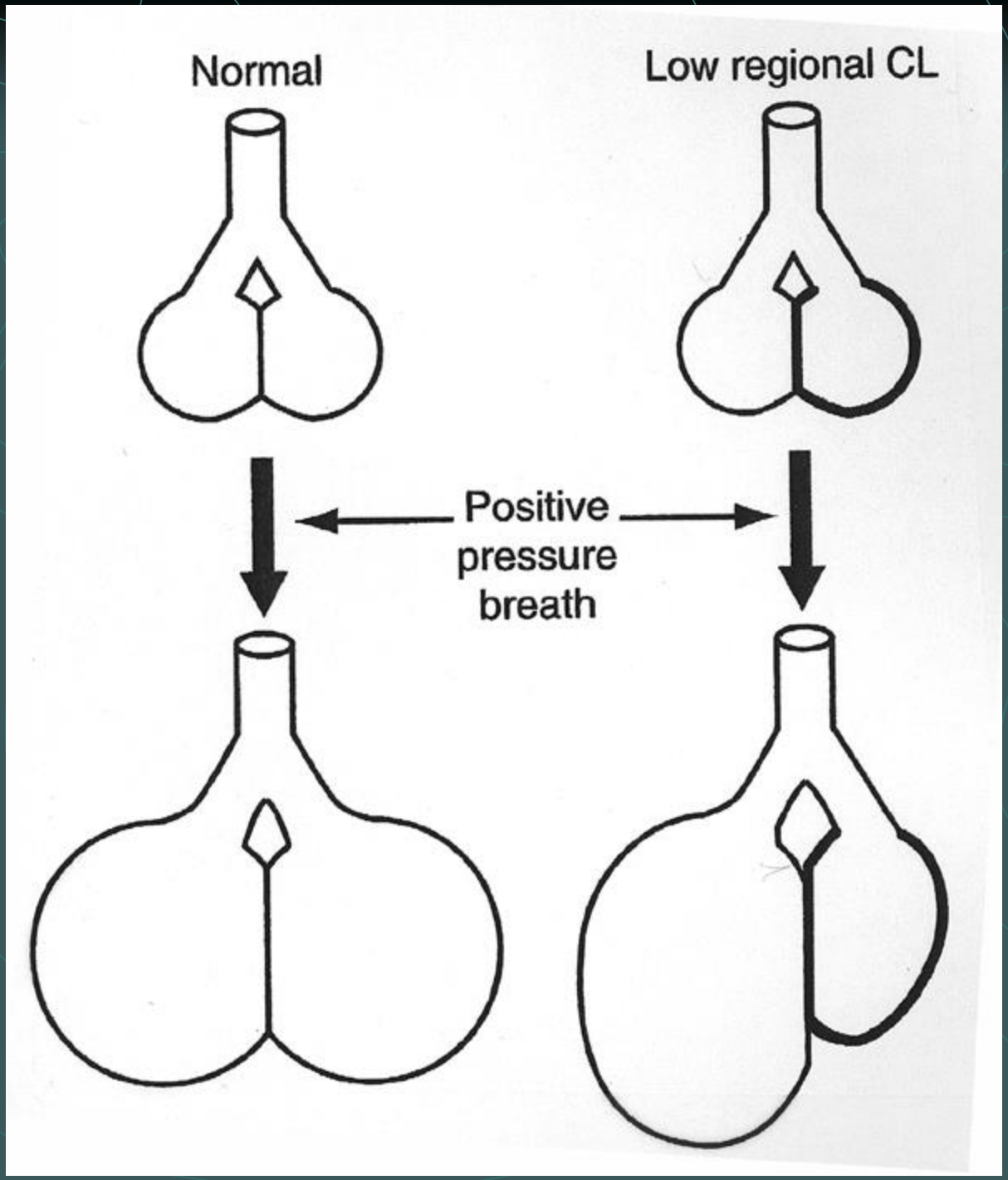


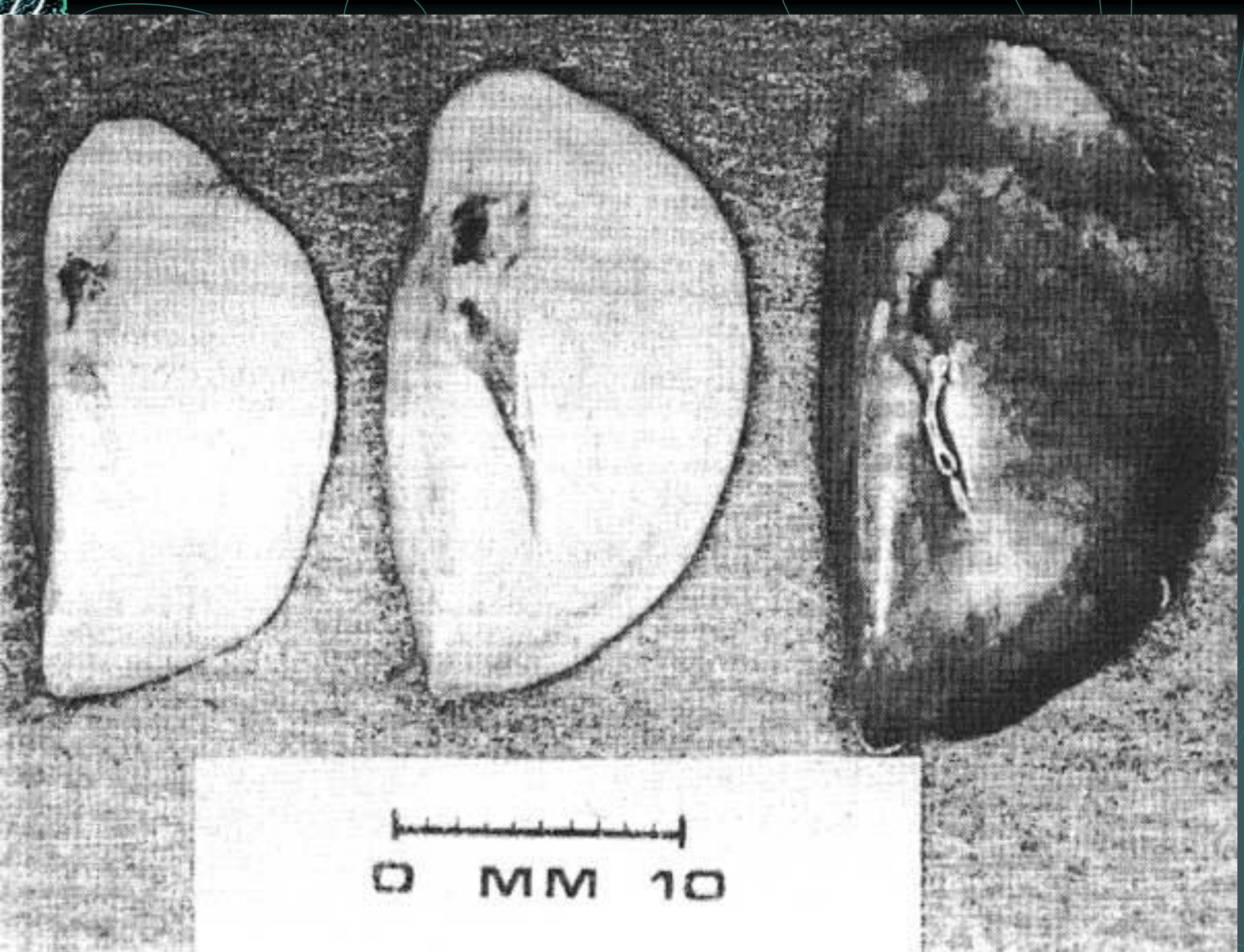
Brain

- CPP=MAP-ICP
- High PEEP reduces cerebral venous return
- Reduced C.O. lowers CBF
- BOTH processes elevate ICP and create more Ischemia and Edema

Mechanisms and pathogenesis of VILI

- Alveolar over-distension and development of lung injury
- Trans-alveolar pressure over 30-35 cm H2O
- Tidal Volume above 10 cc/Kg (safe= 6-8 cc/Kg)
- Rate (speed) of lung distension = stretch, shear
- Frequency (Resp. Rate)
- Pulmonary hypertension, Capillary leak syndrome
- Duration (Insp. Time), Flow Pattern do NOT effect VILI

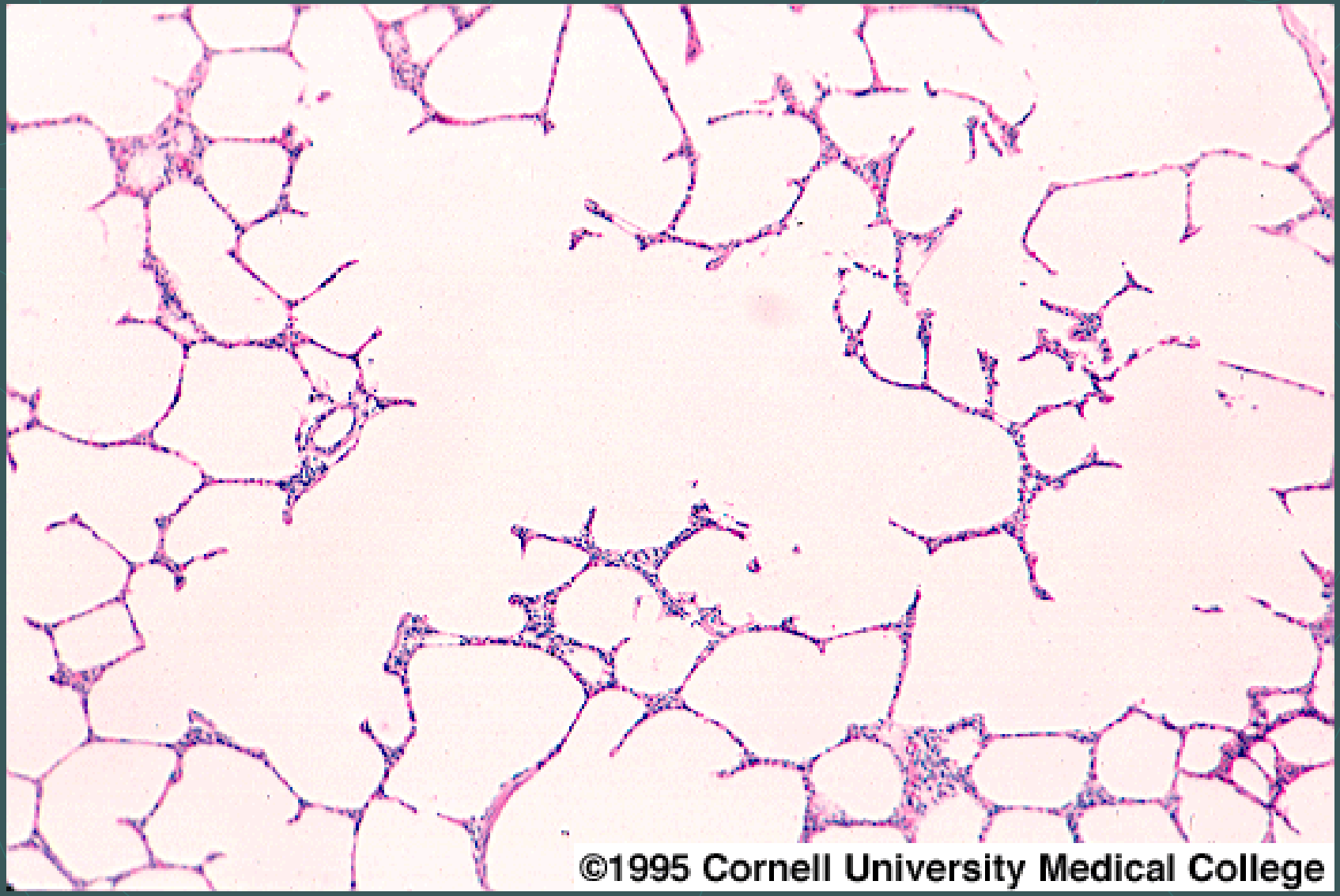

Normal
Low regional CL
Positive
pressure
breath


0 MM 10

Mechanisms of lung injury

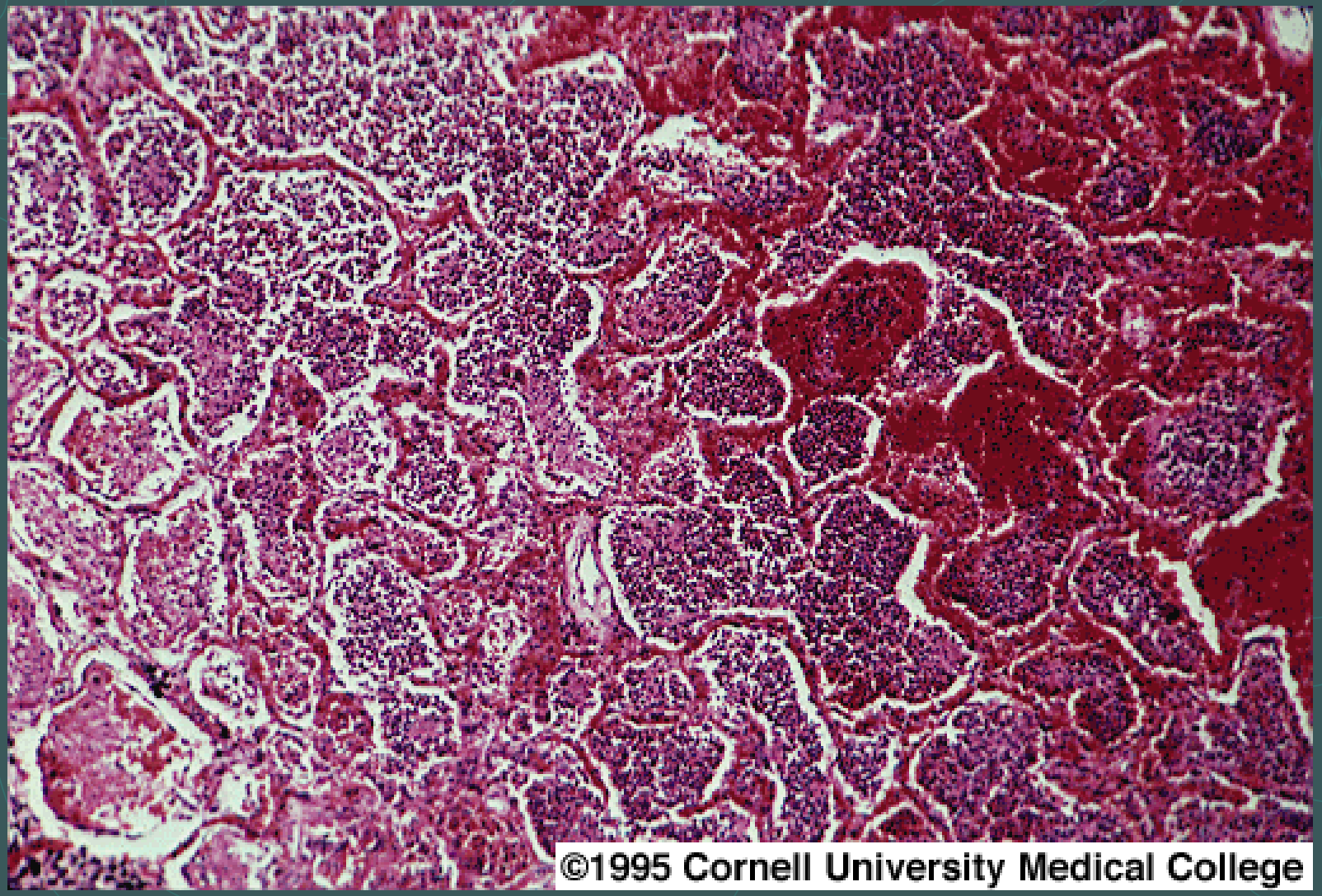
- Cyclical Strain, Stretch, Shearing force
- Cyclical Alveolar Collapse – “atelectrauma”
- Biochemical Mechanisms – neutrophil activation, Cytokines, inflammatory processes
- Stetch--->IL-8, nfKb, TNFa, ….

Normal Acini

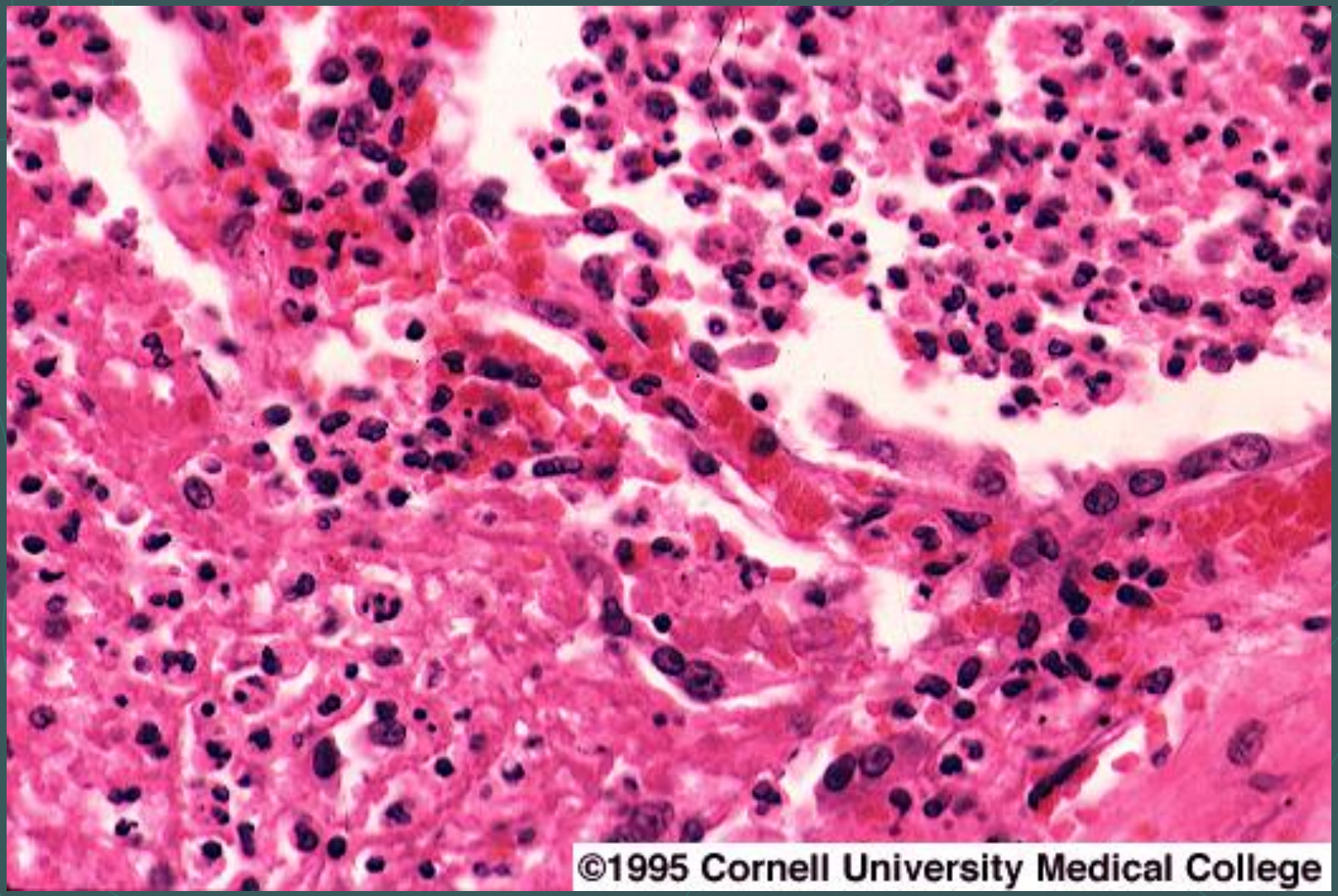


Normal Alveoli

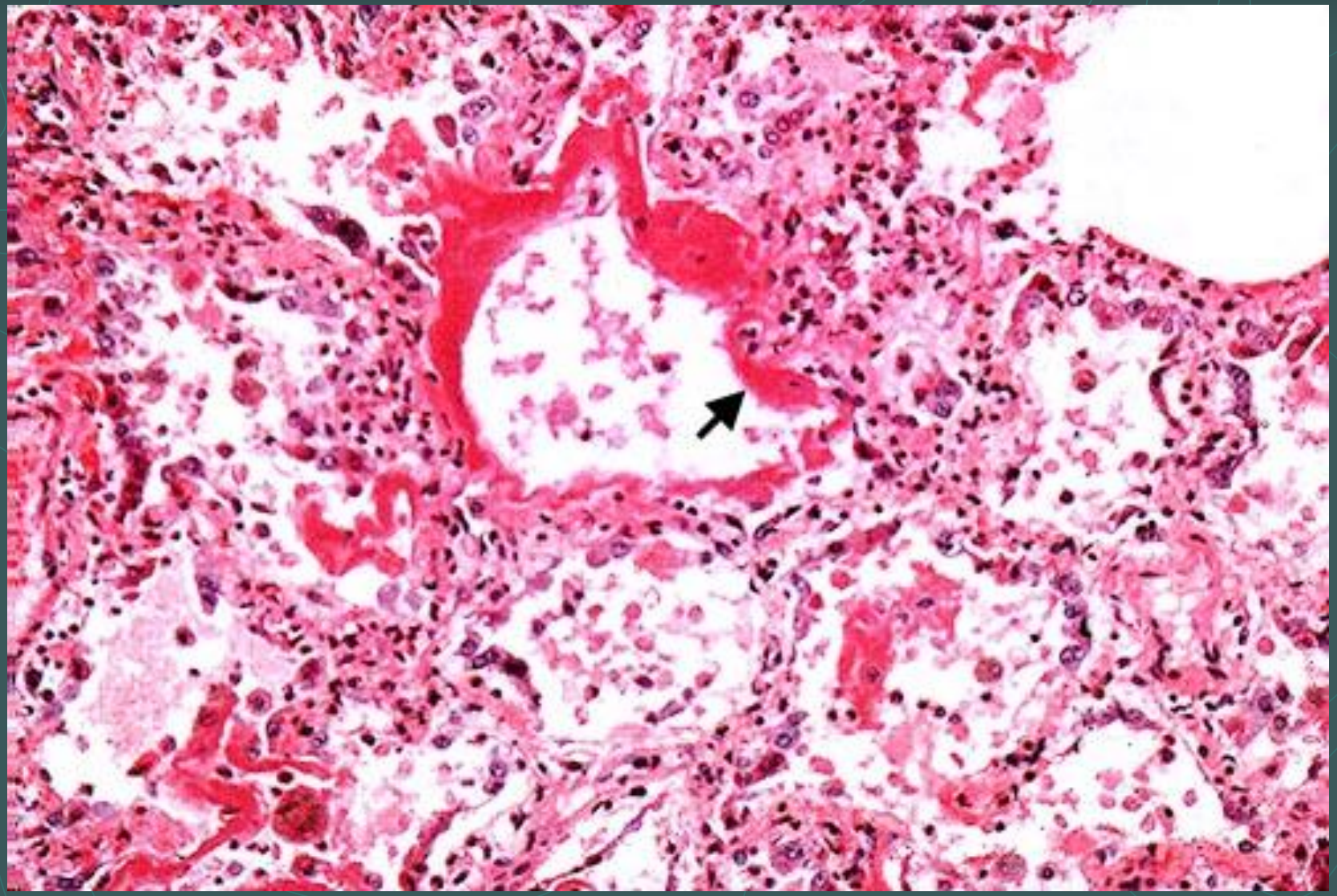
Neutrophil Invasion



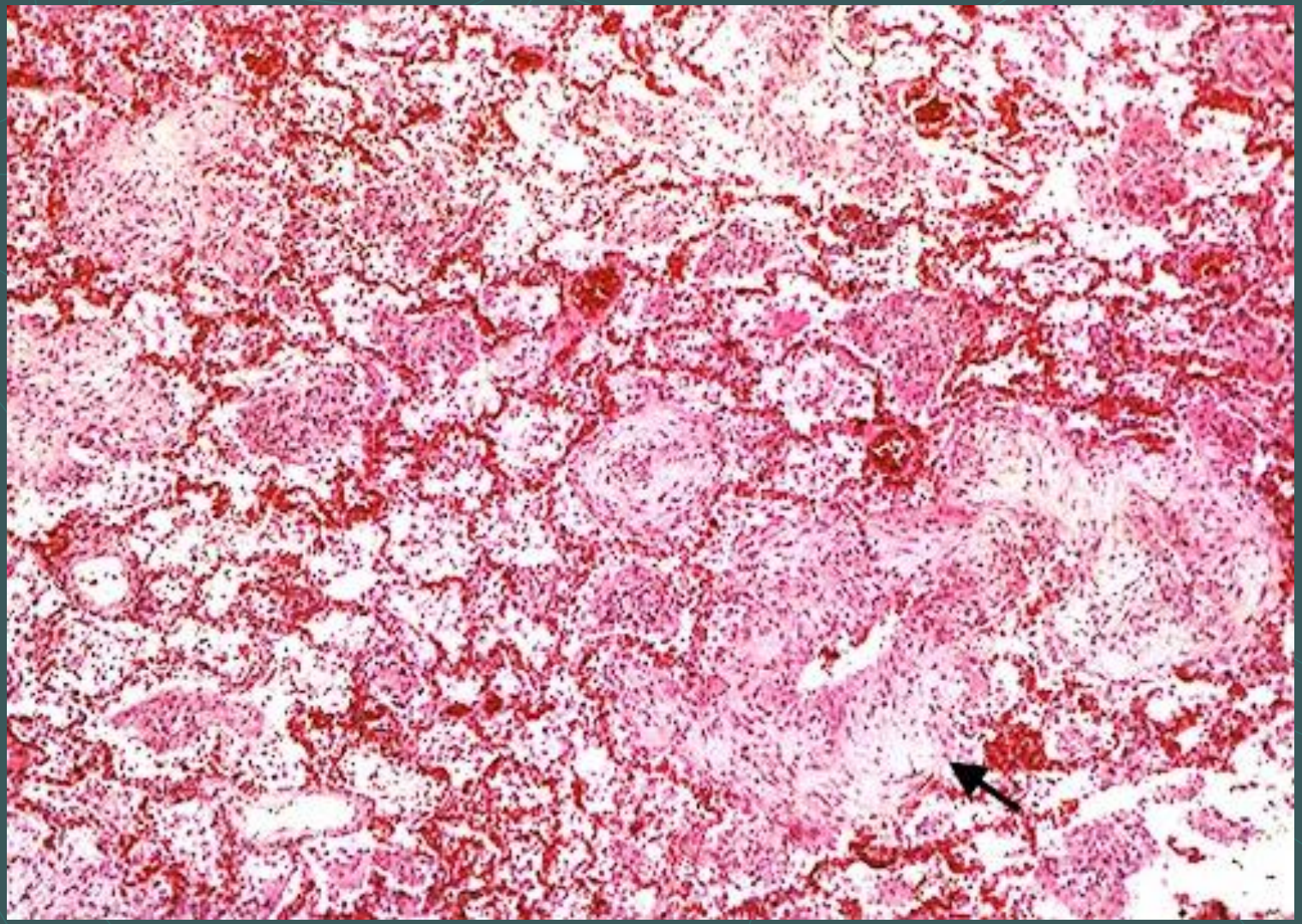
Neutrophil invasion 2



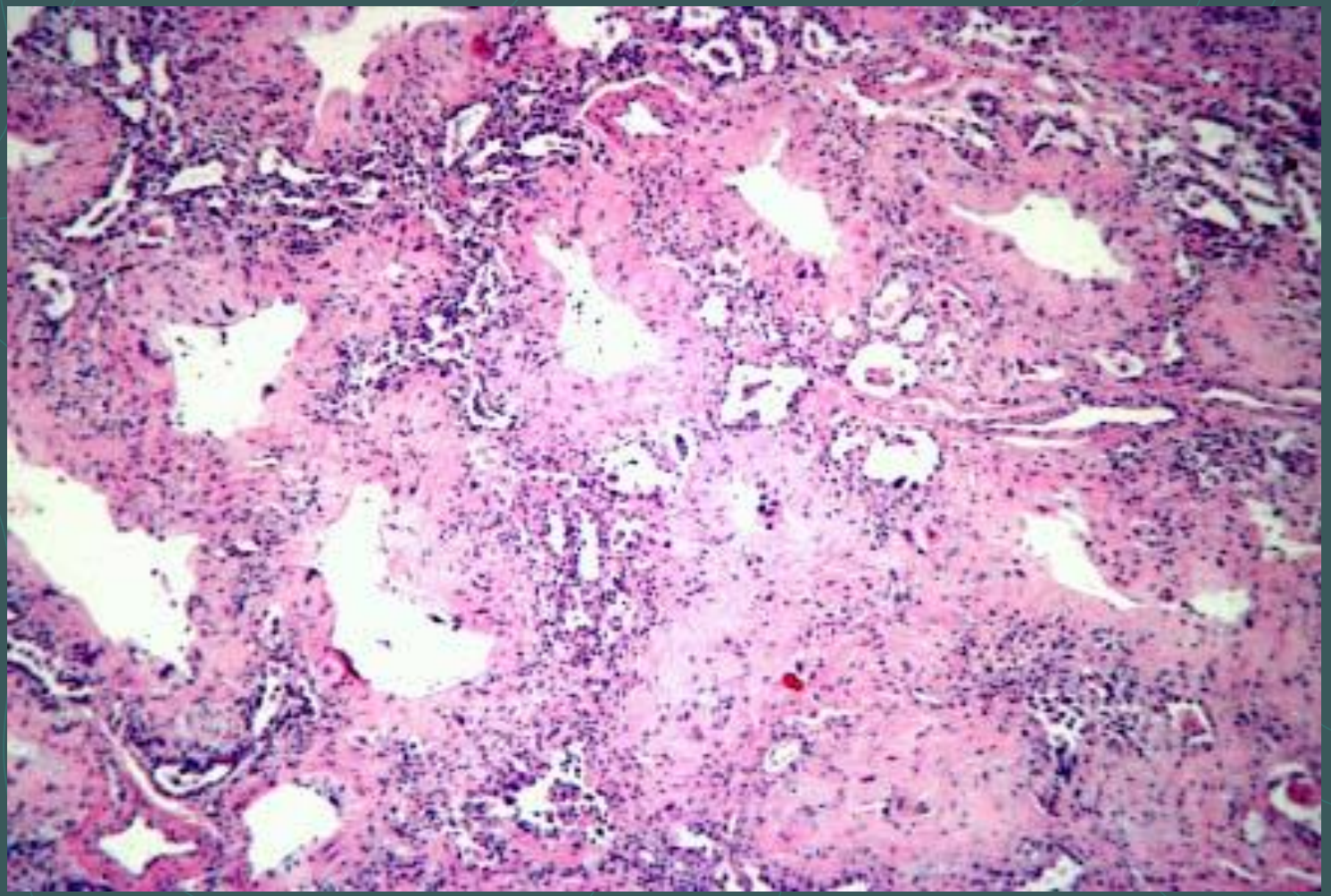
Diffuse Alveolar Damage - Acute



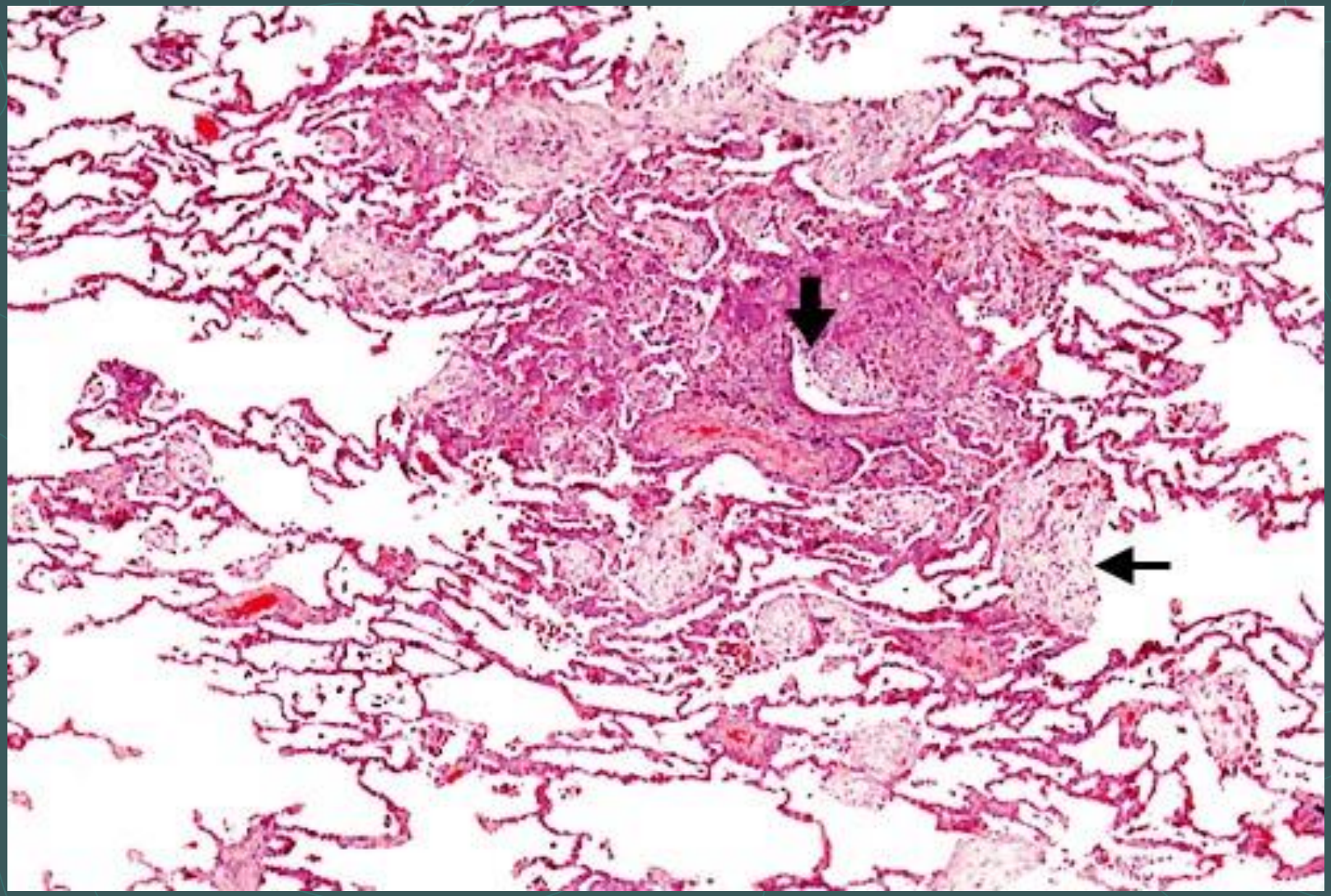
DAD - Organizing



DAD - Fibrotic



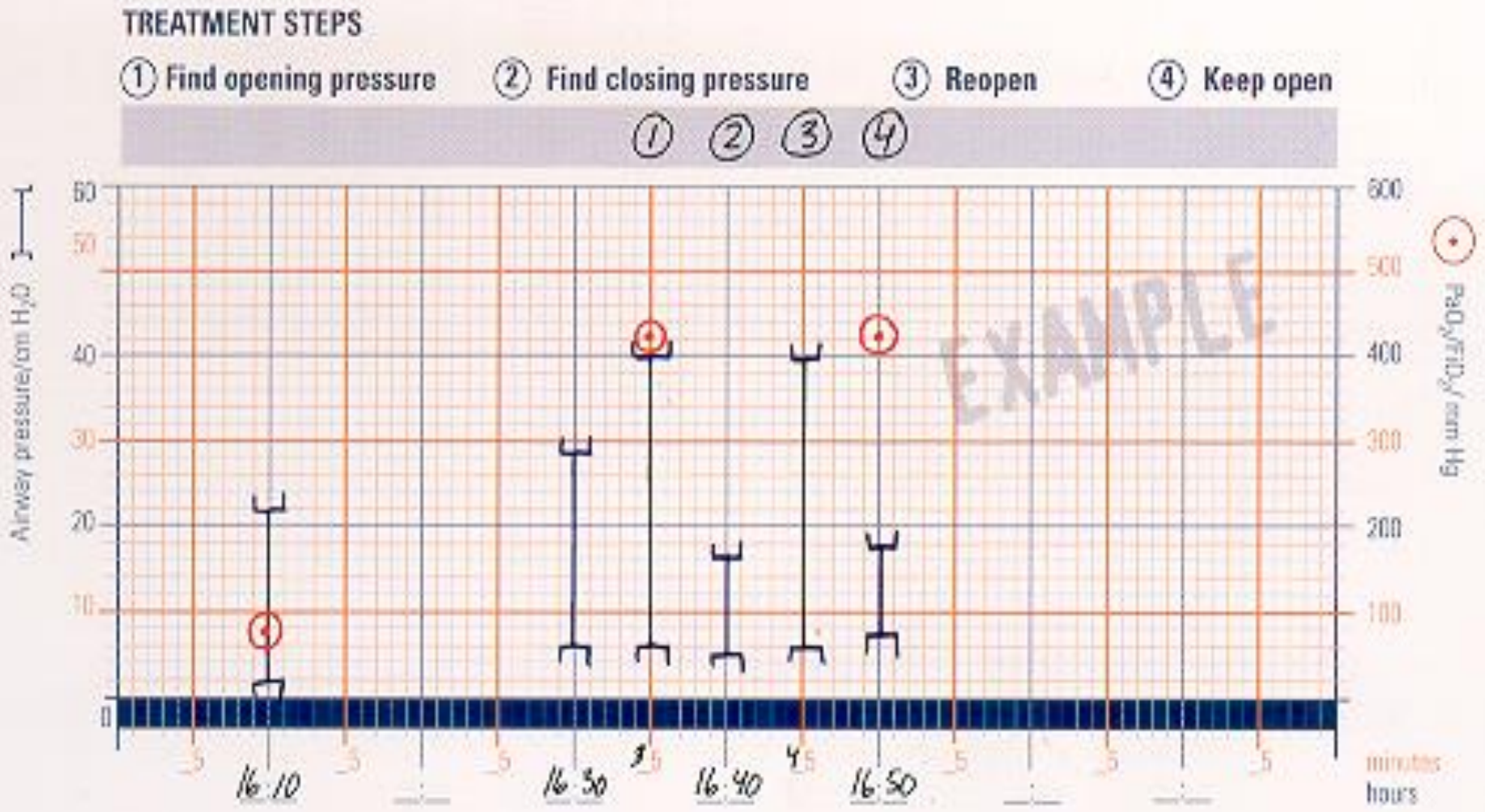
BOOP



Honeycomb Lung

Recruitment

Recruitment Maneuver - Lachmann



VILI Prevention & Management

- Lung protective ventilation strategies – Vol, Press, Rate
- Lung Rest
- Avoid Cyclical Alveolar Collapse (PEEP)
- Recruit and keep open
- Prone Position
- Treat Pulm. Hypertension
- Avoid, treat VAP
- Steroids?

Recommendations - Kolobow

- Limit peak and plateau pressures, Maintain MAWP
- Limit Tidal Volume and Minute Volume
- Adjust PEEP and FiO_2 to maintain a saturation of 90%
- Tolerate an elevated $PaCO_2$, Maintain pH
- Change body position
- Avoid Edema and Malnutrition

Thank You