



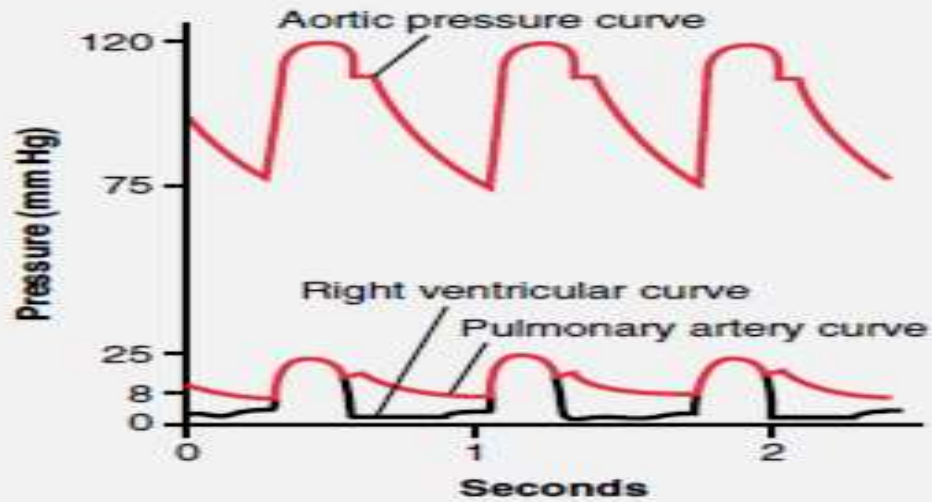
# 5- Pulmonary Capillary Dynamics

By

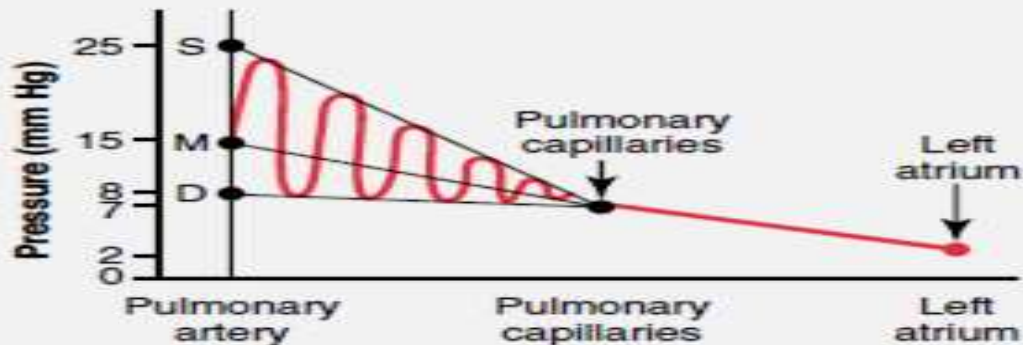
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**Figure** Pressure pulse contours in the right ventricle, pulmonary artery, and aorta.



**Figure** Pressures in the different vessels of the lungs. The red curve denotes arterial pulsations. D, diastolic; M, mean; S, systolic.

# Pressures Causing Fluid Movement

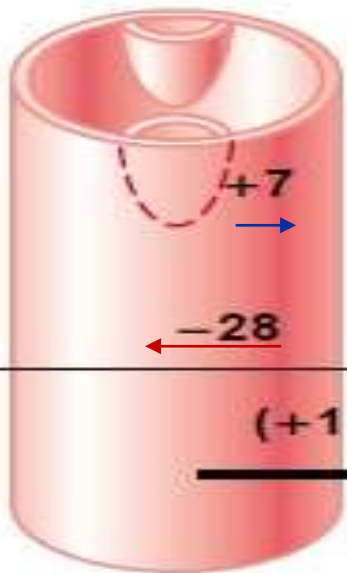
CAPILLARY

ALVEOLUS

Hydrostatic pressure

Osmotic pressure

Net pressure



-8  
-14

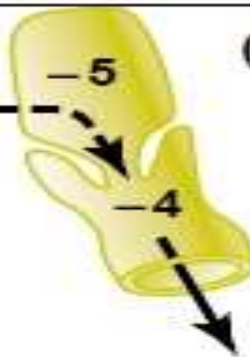
(+1)

-8

-8 (Surface tension at pore)

(0)

(Evaporation)



Lymphatic pump

## Starling forces affecting pulmonary cap.

1. **The pulmonary capillary pressure is low, about 7 mm Hg**, in comparison with a considerably higher functional capillary pressure in the peripheral tissues of about 17 mm Hg.
2. **The interstitial fluid pressure in the lung is slightly more negative** than that in the peripheral subcutaneous tissue. (This has been measured by measuring the absorption pressure of fluid from the alveoli, giving a value of **about -8 mm Hg.**)
3. The pulmonary capillaries are relatively leaky to protein molecules, so that the **colloid osmotic pressure of the pulmonary interstitial fluid** is about **14 mm Hg**, in comparison with less than half this value in the peripheral tissues.
4. **Plasma colloid osmotic pressure = 28 mm Hg.**

-The alveolar walls are extremely **thin**, and the alveolar epithelium is so weak that it can be ruptured by any **positive pressure** in the interstitial spaces greater than alveolar air pressure (greater than 0 mm Hg), which allows dumping of fluid from the interstitial spaces into the alveoli.

The dynamics of fluid exchange across the lung capillary membranes are **qualitatively** the same as for peripheral tissues. However, **quantitatively**, there are important differences, as follows:

	mm Hg
<i>Forces tending to cause movement of fluid outward from the capillaries and into the pulmonary interstitium:</i>	
Capillary pressure	7
Interstitial fluid colloid osmotic pressure	14
Negative interstitial fluid pressure	8
<b>TOTAL OUTWARD FORCE</b>	<b>29</b>
<i>Forces tending to cause absorption of fluid into the capillaries:</i>	
Plasma colloid osmotic pressure	28
<b>TOTAL INWARD FORCE</b>	<b>28</b>

Thus, the normal **outward** forces are slightly greater than the **inward** forces, providing a mean filtration pressure at the pulmonary capillary membrane; this can be calculated as follows:

	<b>mm Hg</b>
Total outward force	+29
Total inward force	<u>-28</u>
<b>MEAN FILTRATION PRESSURE</b>	<b>+1</b>

This filtration pressure causes a slight continual flow of fluid from the pulmonary capillaries into the interstitial spaces, and except for a small amount that evaporates in the alveoli, this fluid is pumped back to the circulation through the pulmonary lymphatic system.

## Negative Pulmonary Interstitial Pressure and the Mechanism for Keeping the Alveoli “Dry.”

One of the most important problems in lung function is to understand why the alveoli do not normally fill with fluid. One’s first inclination is to think that the alveolar epithelium is strong enough and continuous enough to keep fluid from leaking out of the interstitial spaces into the alveoli.

This **is not true**, because experiments have shown that there are always openings between the alveolar epithelial cells through which even large protein molecules, as well as water and electrolytes, can pass.

It is clear that whenever **extra fluid** appears in the alveoli, it will simply be sucked mechanically into the lung interstitium through the small openings between the alveolar epithelial cells. Then the excess fluid is either carried away through the **pulmonary lymphatics** or absorbed into the pulmonary capillaries.

Thus, under normal conditions, the alveoli are kept “**dry,**” except for a small amount of fluid that seeps from the epithelium onto the lining surfaces of the alveoli to keep them moist.



# Pulmonary Edema

The most common **causes** of pulmonary edema are as follows:

1. **Left-sided heart failure or mitral valve disease**, with consequent great **increases in pulmonary venous pressure and pulmonary capillary pressure**.
2. **Damage to the pulmonary blood capillary membranes** caused by **infections** such as pneumonia or by breathing **noxious substances** such as chlorine gas or sulfur dioxide gas.

## -Edema safety factors:

- A) **Low pulmonary capillary pressure (7 mmHg)** while the **osmotic (oncotic) pressure of plasma proteins = 28 mmHg**, so alveoli are always dry.
- B) **Surfactant** decreases surface tension, which attract fluid towards alveoli.
- C) **The rich lymphatic circulation** carries any fluid remains out the capillaries.
- D) **Drainage away of proteins by continuous lymph flow.**
- E) **-ve ISF.**

## -Safety Factor in Chronic Conditions:

When the pulmonary capillary pressure remains elevated **chronically** (for at least **2 weeks**), the lungs become even **more resistant** to pulmonary edema because the **lymph vessels expand greatly**, increasing their capability of carrying fluid away from the interstitial spaces perhaps as much as **10-fold**.

## Rapidity of Death in Acute Pulmonary Edema

When the pulmonary capillary pressure rises even slightly above the safety factor level, lethal pulmonary edema can occur within hours, or even within **20 to 30 minutes** if the capillary pressure rises **25 to 30 mm Hg** above the safety factor level.

Thus, in **acute left-sided heart failure**, in which the pulmonary capillary pressure occasionally does rise to **50 mm Hg**, death frequently ensues in less than **30 minutes** from acute pulmonary edema.

**Thank You**