

STARLING'S LAW OF THE CAPILLARIES

by Bill Wojciechowski, MS, RRT



As respiratory therapists, we often encounter patients who exhibit some form of edema. We experience patients who sometimes have pulmonary edema, peripheral edema, pitting edema, interstitial edema, pedal edema, or generalized edema. The list goes on. Part I of this two-part article will explore the mechanism, described by physiologist Ernst Starling in 1896, responsible for maintaining the normal distribution of fluid between the vasculature and the interstitium in the systemic capillary beds. Part II of the article will discuss the same mechanism as it pertains to pulmonary capillaries. In Part II, the consequences of the disruption of this mechanism will also be presented.

Body Fluid Compartments

Generally, 50% to 60% of our total, ideal body weight is water. For newborns, that percentage can be as high as 75%. Obesity causes a decrease in total body water, sometimes to as low as 45%. An adult with an ideal body weight of 70 kg has approximately 40 liters of total body fluid. The 40 liters of fluid are dispersed throughout the body, but this large fluid volume is also "confined" to specific compartments.

The two major fluid compartments of the body are the extracellular fluid (15 liters) compartment and the intracellular fluid (25 liters) compartment. The extracellular fluid space is partitioned into the interstitium, or interstitial space, and the vascular space. The interstitial space is located outside the cells of the body, and encom-

passes all the fluid situated outside the cells of all the body's tissues. The vascular space accommodates the plasma. Specialized fluids such as cerebrospinal fluid, semen, synovial fluid, ocular fluid, etc., are included within the interstitial fluid. The intracellular fluid, on the other hand, is comprised of all the fluid residing inside all the cells of the body, e.g., muscle cells, nerve cells, blood cells, bone cells, etc.

These fluid compartments are cordoned off by semipermeable membranes that normally restrain and limit the movement of the body's fluid volume. Although water can passively diffuse across the majority of the semipermeable membranes, its movement from one fluid space of the body to another is under dynamic control.

The Starling Equation

The Starling equation, shown here, mathematically describes the movement of fluid across the capillary membranes.

$$Q_f = K_f [(P_c - P_i) - \sigma(\pi_c - \pi_i)]$$

where,

- Q_f = net flow of fluid out of the capillaries
- K_f = capillary filtration coefficient
- P_c = capillary hydrostatic pressure
- P_i = interstitial fluid hydrostatic pressure
- σ = reflection coefficient
- π_c = capillary osmotic pressure
- π_i = interstitial fluid osmotic pressure

In the Starling equation, the difference between P_c and P_i represents hydrostatic pressure gradient across the capillary membrane, and influences the filtration of fluid across the capillary membrane, or capillary endothelium. As this gradient widens, more fluid is filtered into the interstitium. The osmotic pressure gradient across the capillary endothelium is symbolized by $\pi_c - \pi_i$. The difference between π_c and π_i determines the amount of fluid reabsorbed across the capillary membrane into the vasculature.

The filtration coefficient, K_f , describes the permeability-surface area characteristics of the capillary endothelium. Its value depends on the nature of the membrane and its surface area. K_f will increase in value if either the membrane's permeability increases, or if the total surface area of the membrane increases. The converse is, likewise, true.

The reflection coefficient, σ , refers to the membrane's ability to prevent the extravasation of protein. The reflection coefficient ranges in value from 0.00 to 1.00. If the membrane is completely soluble to protein, σ equals 0.00. Membranes that are totally impermeable to protein have a σ value of 1.00.

Interacting Pressures

The movement of fluid and solutes dissolved in the blood is regulated by the interplay of four pressures interacting among themselves on both sides of the systemic capillary endothelium. These four pressures are (1) the capillary hydrostatic pressure, (2)

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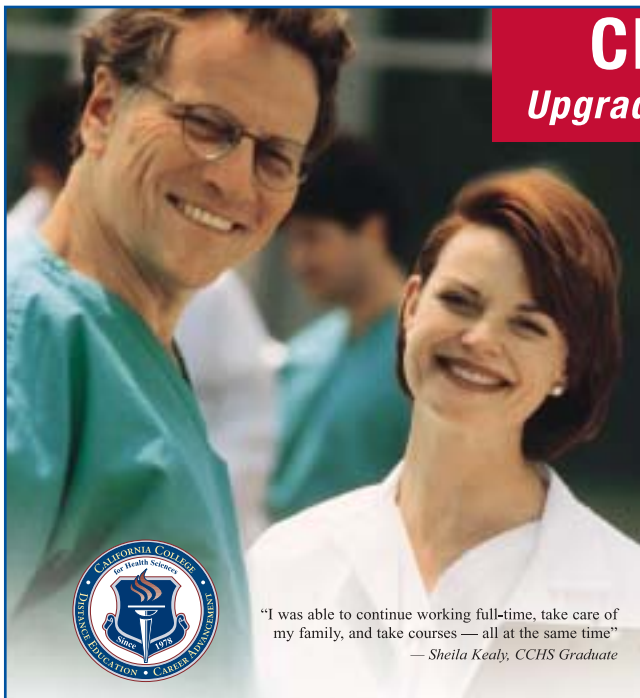
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the interstitial fluid hydrostatic pressure, (3) the capillary osmotic pressure, and (4) the interstitial fluid osmotic pressure.

The capillary hydrostatic pressure (CHP) is the pressure exerted by the blood against the walls of the capillary endothelium. The capillary hydrostatic pressure is essentially equivalent to the blood pressure. The CHP favors fluid movement out of the vasculature into the interstitium, and normally is about 30 torr. Interstitial fluid hydrostatic pressure (IHP) is the pressure applied by the interstitial fluid outside the cells of the body. The IHP tends to pull fluid out of the vasculature because this pressure is subatmospheric, and averages around - 3 torr. The capillary osmotic pressure (COP) results from large protein molecules (albumin, globulin, and fibrinogen) unable to cross the capillary endothelium and remain in the vasculature. The COP pulls water into the vasculature. The usual value for the COP is 25 torr. The interstitial osmotic pressure (IOP) is caused by protein molecules that reside in the interstitium. The value of the IOP is approximately 4 torr.

The $CHP - IHP$, or the $P_c - P_i$, difference constitutes the hydrostatic pressure gradient, and causes filtration out of the vasculature into the interstitium. The $COP - IOP$ gradient is synonymous with the $\pi_c - \pi_i$ gradient, and is responsible for net osmosis of fluid into the vasculature from the interstitium.

Under normal circumstances, three of these pressures – CHP, IHP, and IOP – affect fluid movement out of the vasculature. Only the COP influences fluid to enter the vasculature.

Systemic Capillary Model

Consider blood flowing through a systemic capillary from an arteriole to a venule. All along the capillary endothelium fluid exchange occurs. However, fluid is filtered toward the arteriole end of the capillary, and is reabsorbed toward the venule end.

To determine the net direction and to quantify the magnitude of fluid movement along the systemic capillary, numerical values will be used for each of the four pressures interacting along the systemic capillary. The $CHP = 30$ torr; $IHP = - 3$ torr; $COP = 25$ torr; $IOP = 4$ torr.

Filtration along Arterial End of Systemic Capillary

The magnitude of the filtration that occurs along the arteriole end of the capillary can be calculated. The hydrostatic pressure gradient along the arteriole end is obtained by subtracting the IHP from the CHP.

$$CHP - IHP = \text{hydrostatic pressure gradient at arteriole end} \\ 30 \text{ torr} - (-3 \text{ torr}) = 33 \text{ torr}$$

Because the IHP is subatmospheric, it augments the CHP, and both favor filtration.

Next, the osmotic pressure gradient is calculated by subtracting the IOP from the COP.

$$COP - IOP = \text{osmotic pressure gradient at the arteriole end} \\ 25 \text{ torr} - 4 \text{ torr} = 21 \text{ torr}$$

The osmotic pressure gradient supports the movement of fluid from the interstitium into the vasculature.

Subtracting the osmotic pressure gradient from the hydrostatic pressure gradient provides the filtration pressure at the arteriole end of the systemic capillary.

$$\text{hydrostatic pressure gradient} - \text{osmotic pressure gradient} = \text{filtration pressure} \\ 33 \text{ torr} - 21 \text{ torr} = 12 \text{ torr filtration pressure}$$

Because the difference between the hydrostatic pressure and the osmotic pressure is a positive value, i.e. 12 torr, filtration predominates at the arteriole end of the systemic capillary.

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Reabsorption along Venule End of Systemic Capillary

The extent of the reabsorption that occurs along the venule end of the capillary can also be calculated. What needs to be taken in consideration at the venule end is that fluid has filtered out of the vasculature at the arteriole end, thereby altering the CHP and COP. The CHP decreases to about 10 torr because fluid left at the arteriole end. The loss of fluid at the arteriole end makes the proteins in the plasma more concentrated at the venule end. The COP increases slightly to around 28 torr. The IHP and IOP remain the same because this fluid compartment is so vast that the filtered fluid essentially has no impact on these two pressures.

The hydrostatic pressure gradient along the venule end is calculated by subtracting the IHP from the CHP.

$$\text{CHP} - \text{IHP} = \text{hydrostatic pressure gradient at venule end} \\ 10 \text{ torr} - (-3 \text{ torr}) = 13 \text{ torr}$$

The osmotic pressure gradient along the venule end is determined again by subtracting the IOP from the COP.

$$\text{COP} - \text{IOP} = \text{osmotic pressure gradient at the venule end} \\ 28 \text{ torr} - 4 \text{ torr} = 24 \text{ torr}$$

Subtracting the osmotic pressure gradient from the hydrostatic pressure gradient provides the reabsorption pressure at the venule end of the systemic capillary.

$$\text{hydrostatic pressure gradient} - \text{osmotic pressure gradient} = \text{reabsorption pressure} \\ 13 \text{ torr} - 24 \text{ torr} = -11 \text{ torr}$$

The negative sign does not indicate subatmospheric pressure. Instead, it represents the direction of the fluid movement at the

venule end of the capillary. This direction is opposite that at the arteriole end, and indicates that reabsorption predominates here.

Filtration-Reabsorption Pressure Difference

The net magnitude of the fluid movement along the systemic capillary can be obtained by subtracting the reabsorption pressure of 11 torr from the filtration pressure of 12 torr

$$12 \text{ torr} - 11 \text{ torr} = 1 \text{ torr}$$

A net movement of 1 torr has left the vasculature during the blood's passage through the systemic capillary. Fluid does not accumulate within the interstitium. If so, edema would develop.

What happens to this filtered fluid? Does it merely continue to accumulate in the interstitium? No, lymph vessels, beginning as blind microscopic end-pouches in the outreaches of the interstitium, prevent the accumulation of this transudated fluid. The lymphatic system continuously siphons fluid away from the interstitium and deposits it ultimately into general circulation.

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The Language of Lung Injury... Continued from page 24

counterbalance end inspiratory forces and is related to the static end inspiratory elastic recoil pressure of the total respiratory system. Airway pressure measured during an end inspiratory occlusion replicates the elastic threshold stress to the pulmonary system sans the inevitable resistive forces present during active inspiration. PPLAT faithfully approximates alveolar pressure and as such is a very useful clinical assessment tool.

Elevated PPLAT will alert the clinician to increased alveolar pressure. Incremental changes in PPLAT are inversely related to lung compliance. An increase in the plateau pressure signals a fall in the global lung compliance. Indeed, a PPLAT of 35 cmH₂O represents the normal peak alveolar pressure necessary to reach TLC. It has been suggested that PPLAT equal to, or in excess of that needed to reach TLC would facilitate lung injury or impede efforts to ventilate the already hyper-inflated lung. Plateau pressure is needed to calculate total lung compliance as the relationship between PPLAT and delivered volume. This lung and chest wall compliance is derived in the following manner.

$$CI = Vt / (\text{PPLAT} - \text{PEEP}_{\text{tot}})$$

Common causes of decreased compliance in the ventilated patient include; main-stem intubation, pneumothorax, CHF, ARDS, pleural effusion, and chest wall deformity.

The difference between Peak Airway Pressure and PPLAT is a function of resistive forces in the patient ventilator system. Raw is calculated by looking at the pressure gradient between the peak airway pressure and the plateau divided by the flow.

$$\text{Raw} = \text{PAP} - \text{PPLAT} / \text{Flow (L/sec.)}$$

Causes of increased Raw include; bronchospasm, bronchoconstriction, secretions, airway obstruction, narrow endotracheal tube and mucosal edema. Bear in mind that both inspiratory and expiratory Raw may vary widely in different pathologies.

Repetitive Alveolar Collapse & Expansion – RACE; This form of VILI is described as a product of alveolar instability that is non-homogenous. The inflation deflation pattern in the acutely injured lung causes this form of VILI. Healthy alveoli with stable

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