

STARLING'S LAW OF THE CAPILLARIES - PART II

by Bill Wojciechowski, MS, RRT



In Part I of this two-part article concerning Starling's law of the capillaries, the discussion focused on (1) body fluid compartments, (2) the Starling equation, and (3) the fluid dynamics across a systemic capillary. In Part II normal capillary dynamics will be reviewed for the benefit of those who did not read Part I. Also, in Part II the fluid dynamics across a pulmonary capillary, and the consequences of the disruption of the dynamic equilibrium that normally exists across the capillary endothelium will be presented.

Normal Capillary Dynamics

The physiologic model that will be developed here involves the interaction among the pressures influencing the exchange of intravascular and interstitial fluid across the capillary endothelium. Two hydrostatic and two osmotic pressures affect transcapillary fluid exchange. These interacting pressures are:

- capillary hydrostatic pressure;
- capillary osmotic pressure;
- interstitial hydrostatic pressure;
- interstitial osmotic pressure.

The capillary endothelium functions as a selectively permeable membrane between the intravascular fluid (blood) and the interstitial fluid. This membrane confines blood cells, platelets, and large protein molecules, i.e., albumin, fibrinogen, and globulin, within the intravascular space. At the same time, this membrane enables the exchange of fluid and small molecules, i.e.,

crystalloids, in accordance with the relationship among the four pressures interacting at this site.

The systemic capillary endothelium is "divided" into an arterial end and a venous end. This division is not marked by a definitive anatomic structure that partitions the capillary into these two segments. Despite this indiscreet conversion, distinct physical events occur at the arterial and venous ends of a systemic capillary, as the blood evolves from arterial to venous. At the arterial end of the capillary the process of filtration is favored. Along the venous end reabsorption predominates.

Hydrostatic Pressure Gradient

The capillary hydrostatic pressure drives fluid out of the capillary (i.e., filtration), and is higher at the arterial end of the capillary and lower at the venous end. The interstitial hydrostatic pressure varies throughout the body. However, it tends to be sub-atmospheric in most locations. Therefore, in most locations the interstitial hydrostatic pressure helps filter fluid from the vasculature into the interstitium. The difference between the capillary hydrostatic pressure and the interstitial hydrostatic pressure constitutes the capillary hydrostatic pressure gradient. This gradient normally facilitates fluid filtration.

Osmotic Pressure Gradient

The capillary endothelium is permeable to certain solutes, i.e., ions and low-molecular weight proteins. The osmotic pressure within the capillary is principally determined by large-molecular weight proteins, which are relatively impermeable to the capillary endothelium. Therefore, this pressure is referred to as the colloid osmotic pressure because it is generated by colloids such as albumin, fibrinogen, and globulin. The colloid osmotic pressure typically ranges between 25 and 30 mmHg.

The osmotic pressure of the interstitial fluid depends on the interstitial protein concentration and the reflection coefficient (σ) of the capillary wall. The more permeable the capillary endothelium is to proteins (σ approaches 0), the higher the interstitial osmotic pressure. The osmotic pressure gradient is obtained by subtracting the interstitial osmotic pressure from the capillary osmotic pressure. This gradient favors the reabsorption of fluid.

Fluid Dynamics across Pulmonary Capillary Endothelium

The conditions that exist at the capillary level in the pulmonary vasculature differ from those of the systemic circulation. The major difference is that the magnitude of the hydrostatic pressure in the pulmonary capillary is less. The mean pulmonary capillary hydrostatic pressure is 7 torr, as compared to 30 torr in the systemic capillary. What accounts for the lower hydrostatic pressure in the pulmonary circulation is the lower pulmonary vascular resistance experienced by the blood flowing through the pulmonary vasculature, compared to the higher resistance encountered by blood flowing through the systemic network.

Recruitment

Two physiologic phenomena peculiar to the pulmonary vascular bed, namely recruitment and distension, account for the

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vascular pressures characteristic of the pulmonary system. The term recruitment refers to the fact that under normal resting conditions not all pulmonary capillaries are perfused. This situation enables the accommodation of increased blood flow during periods of increased right ventricular cardiac output. What occurs when the right ventricular cardiac output increases is that non-perfused capillaries are recruited ("opened"), thereby providing additional blood vessels for the increased pulmonary blood flow. In essence, the total cross-sectional area of the pulmonary capillary bed increases. (As an aside, this situation exemplifies a manifestation of the law of continuity, which states that an inverse relationship exists between the cross-sectional area and the velocity of the flowing fluid.) Despite the increased pulmonary blood flow, the pulmonary capillary hydrostatic pressure does not increase. In fact it remains constant.

Distention

A further increase in the right ventricular cardiac output can be accommodated, even after all pulmonary vessels have been recruited. Once pulmonary vessels have been recruited, by virtue of the pliability of their walls, they have the capability to distend (widen). This physical event is called distention. The thin architecture of the walls of the pulmonary vessels enables them to increase their caliber; these vessels are extremely distensible. This vascular distension is accomplished via passive vessel wall expansion, and not through any neural or humoral vasodilatory mechanism.

Pulmonary Capillary Model

Consider mixed venous blood flowing through a pulmonary capillary after having been ejected from the right ventricle. Fluid exchange occurs across the pulmonary capillary endothelium along the entire length of the pulmonary capillary. Fluid exchange entails both filtration and reabsorption.

To determine the direction and to quantify the magnitude of fluid exchange along the pulmonary capillary, numerical values will be provided for each of the four pressures interacting along the pulmonary capillary. The values presented here are all within the normal ranges for these pressures. The capillary hydrostatic pressure (CHP) is 7 torr; the interstitial hydrostatic pressure (IHP) is -8 torr. The capillary osmotic pressure (COP) and the interstitial osmotic pressure (IOP) are 28 torr and 14 torr, respectively.

Hydrostatic Pressure Gradient (Filtration)

The pulmonary capillary hydrostatic pressure (CHP) favors the movement of fluid out of the pulmonary vasculature and into the pulmonary interstitium. By virtue of the fact that the pulmonary interstitial hydrostatic pressure (IHP) is subatmospheric (negative), it also causes the transudation of fluid from pulmonary circulation into the pulmonary interstitium. Therefore, both the CHP and IHP bring about filtration. The hydrostatic pressure gradient across the pulmonary endothelium is calculated as follows:

$$\text{CHP} - \text{IHP} = \text{pulmonary capillary hydrostatic pressure gradient}$$

$$7 \text{ torr} - (-8 \text{ torr}) = 15 \text{ torr}$$

Because the hydrostatic pressure gradient in this example has a positive value, filtration is favored. The magnitude of pressure of the fluid leaving the vasculature caused by the hydrostatic pressure gradient is 15 torr.

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Osmotic Pressure Gradient (Reabsorption)

The colloid osmotic pressure influences the reabsorption of fluid from the pulmonary interstitium into the pulmonary capillary. Two osmotic pressures oppose each other at this site. They are the capillary osmotic pressure, known as the COP and the interstitial osmotic pressure, known as the IOP. Because these two pressures are in opposition to each other, they are subtracted from one another. Therefore, the colloid osmotic pressure gradient is calculated as follows:

$$\begin{aligned} \text{COP} - \text{IOP} &= \text{colloid osmotic pressure gradient} \\ 28 \text{ torr} - 14 \text{ torr} &= 14 \text{ torr} \end{aligned}$$

Because the colloid osmotic pressure gradient in this example has a positive value, reabsorption is favored. The magnitude of pressure of the fluid re-entering the pulmonary vasculature because of the osmotic pressure gradient is 14 torr.

Filtration Versus Reabsorption

Subtracting the colloid osmotic pressure gradient from the hydrostatic pressure gradient will indicate the net flow of fluid either into (reabsorption) or out of (filtration) the vasculature. The calculation of the net flow of fluid and the direction of the flow (positive = filtration; negative = reabsorption) is as follows:

$$\begin{aligned} \text{hydrostatic pressure gradient} - \text{colloid osmotic pressure} \\ \text{gradient} &= \text{direction of fluid flow} \\ 15 \text{ torr} - 14 \text{ torr} &= 1 \text{ torr} \end{aligned}$$

If the difference between these two gradients were a negative number, reabsorption would predominate. However, since the difference is a positive value, filtration prevails. In other words more fluid is being filtered across the pulmonary endothelium than is being reabsorbed.

Finally, the role of the pulmonary lymphatics is to remove filtered fluid, and to re-deposit it into general circulation. If the amount of fluid filtered overwhelms the ability of the pulmonary lymphatics to remove fluid, fluid will accumulate in the pulmonary interstitium. Furthermore, if the volume of fluid in the pulmonary interstitium exceeds the capacity of that compartment, fluid will transude across the pulmonary epithelium and enter the alveoli, resulting in pulmonary edema.

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