CHAPTER 19 Cardiovascular System: Blood Vessels: Addendum

III. Capillary Exchange (Flow & Dynamics)

A) Several gradients moving water in & out of vessels into tissues

- Movement in/out of capillaries, over the endothelial surface or between cells.
  - * Diffusion, osmosis, exocytosis, endocytosis.
  - * Concentration gradients must be maintained, which is why blood flow is so important.
  - * Wastes move out of interstitial fluids into blood, nutrients move opposite direction.

- areas where filtration is important have lots of intracellular clefts. Special capillaries are called "Fenestrated capillaries". Kidneys, intestines, etc.
  - ** fenestration = pore.
  - * increase movement of ions, water into tissues = edema. Increase movement of ions/water into capillaries = dehydration.
    - ** Chemicals increase/decrease permeability of capillary walls.
    - ** lymphatics return water to the veins.

B) Hydrostatic and Oncotic (Colloidal) Pressures

- There are two hydrostatic and two oncotic pressures that affect transcapillary fluid exchange:
  1. capillary hydrostatic pressure
  2. tissue (interstitial) hydrostatic pressure
  3. capillary (plasma) colloidal pressure
  4. tissue (interstitial) colloidal pressure.

1) Capillary Hydrostatic Pressure (PC)

- This pressure drives fluid out of the capillary (i.e., filtration), and is highest at the arteriolar end of the capillary and lowest at the venular end.

* Depending upon the organ, the pressure may drop along the length of the capillary (axial pressure gradient) by 15-30 mmHg.

* The average capillary hydrostatic pressure is determined by arterial and venous pressures (PA and PV), and by the ratio of post-to-precapillary resistances (RV/RA). An increase in either arterial or venous pressure will increase capillary pressure; however, a given change in PA is only about one-fifth as effective in changing PC as the same absolute change in PV.

* Therefore, PC is much more influenced by changes in PV than by changes in PA.

* Furthermore, PC is increased by precapillary vasodilation (particularly by arteriolar dilation); precapillary vasoconstriction decreases PC. Venous constriction increases PC, whereas venous dilation decreases PC.

2) Tissue (Interstitial) Hydrostatic Pressure (PT)

-This pressure is determined by the interstitial fluid volume and by the compliance of the tissue, which is related to the ability of the tissue volume to increase.

* Normally, PT is near zero. The rise in PT that occurs with increased interstitial fluid volume decreases the hydrostatic gradient across the capillary thereby limiting filtration.

3) Capillary Plasma Colloidal Pressure (PC)

- Because the capillary barrier is readily permeable to ions, the osmotic pressure within the capillary is principally determined by plasma proteins that are relatively impermeable. Therefore, instead of speaking of "osmotic" pressure, this pressure is referred to as the "oncotic" pressure or "colloid osmotic" pressure because it is generated by colloids.

* Albumin generates about 70% of the oncotic pressure. This pressure is typically 25-30 mmHg.
* The oncotic pressure increases along the length of the capillary. Continuous capillaries have a high amount, whereas discontinuous capillaries which are very "leaky" to proteins have a very amount

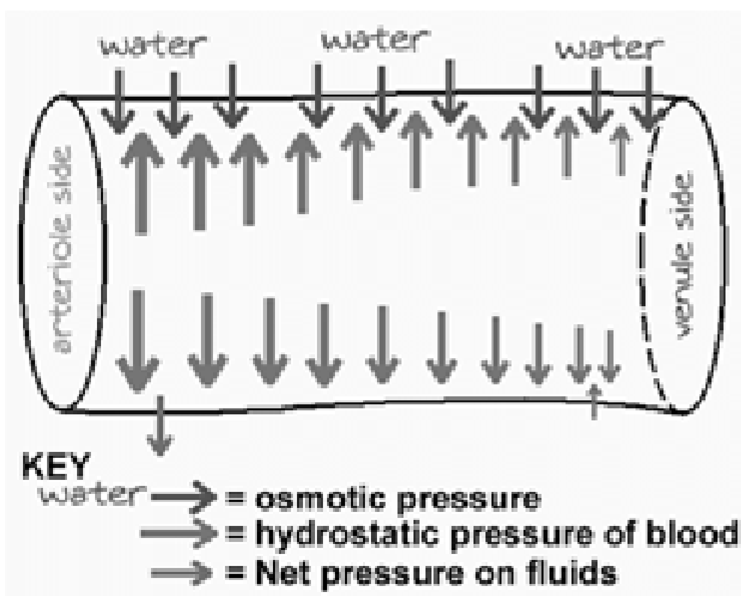
4) Tissue (interstitial) Colloidal Pressure (PT)

- The oncotic pressure of the interstitial fluid depends on the interstitial protein concentration and the reflection coefficient of the capillary wall.

* The more permeable the capillary barrier is to proteins, the higher the interstitial oncotic pressure.
* This pressure is also determined by the amount of fluid filtration into the interstitium.

* For example, increased capillary filtration decreases interstitial protein concentration and reduces the oncotic pressure. This effect of filtration on protein concentration also serves as a mechanism to limit capillary filtration. In a "typical" tissue, tissue oncotic (colloidal) pressure is about 5 mmHg (i.e., much lower than capillary plasma oncotic pressure).

C) Why and in what direction does fluid escape from the capillary?



- The capillaries are porous enough that the fluid within them (plasma) is in contact with the fluid outside of them (extracellular fluid, a.k.a., interstitial fluid). Because these fluids can exchange (but must do so through tiny spaces), whether they will do so or not depends on the pressures on the fluids.

- There are two main pressures on the fluids. One is osmotic pressure (shown here using blue arrows for the water). The other is the hydrostatic pressure (blood pressure) on the capillary walls (shown here using red arrows for blood).

  * Osmotic pressure exists whenever two solutions with differing solute concentrations interact. Water always leaves the hypotonic solution, pushing its way into the hypertonic solution. This "pushing" is the osmotic pressure.

  * It turns out that the blood is hypertonic to the extracellular fluid. So water is always trying to push its way into the blood vessel due to osmotic pressure. The pressure due to osmosis remains constant across the entire capillary.

  * The blood pressure, or hydrostatic pressure of the blood (a.k.a., outward hydrostatic pressure) is an outward pressure. That's because the blood is pushing on the walls of the capillary as it runs through the capillary from arteriole to venule ends.

    ** This pressure, however, is not constant throughout the capillary. You see, there is more pressure on the arteriole side because it is closer to the heart (which is applying the pressure in the first place). So it starts off at the arteriole side with a higher pressure than at the venule side.

- Taken together, you will see that the hydrostatic pressure of the blood pushing out is greater at the arteriole end than the osmotic pressure pushing in. That means that the net pressure on all fluids at the arteriole side is outward.

  * Yet, at the venule side, the hydrostatic pressure of the blood pushing out is less than the osmotic pressure pushing in. That means that the net pressure on all fluids at the venule side is inward.

Your book quantifies these pressures in a diagram of capillaries of the systemic circuit. But the overall scheme is that more fluid ends up being filtered out of the capillary than into it.

Fluid in = fluid out = homeostasis
Fluid in > fluid out = dehydration
Fluid in < fluid out = edema

D) Shock

-Circulatory shock
Mechanisms: Generalized - any decrease in blood flow relative to the body's needs; decreased blood pressure(hypotension and shock)

-Antecedents to the classes of circulatory shock:

1. Low output circulatory failure
   * Hypovolemic shock: due to low blood volumeE.g., following hemorrhage or dehydration
   * Obstructive shock: due to decreased movement of blood through heart and great vessels E.g., heart failure (cardiogenic shock) or blockage of pulmonary artery

2. High output circulatory failure
   * Septic/toxic shock (= Vascular shock) : due to loss of vascular resistance. Bacterial endotoxin triggers vasodilation
   * Anaphylactic shock: due to loss of vascular resistance. Histamine triggers vasodilation, increased capillary permeabilityNeurogenic shock: due to loss of vascular resistanceLoss of sympathetic neural stimulation to vasoconstrict.

-Signs and symptoms: see images in textbook