4.4: Pulmonary Microcirculation

Gas exchange is the prime function of the lung. The pulmonary circulation moves the pulmonary blood into close association with the alveoli (at the blood-gas barrier) so that gas exchange is facilitated. The flow involved is large as the pulmonary blood flow is equal to the cardiac output. Efficient gas exchange is facilitated because the blood-gas membrane is thin with a large surface area. At any moment, the pulmonary capillary blood volume is about 80 mls.

The key features of the pulmonary microcirculation are:

- The pulmonary capillaries (and the alveoli) have very thin walls which minimises the barrier to diffusion.
- In the alveolar walls, the capillaries form a dense network which has been considered to be almost a continuous thin film of blood. This provides a large capillary surface area.
- The pressures in the pulmonary circuit are much lower than in the systemic circulation and the pulmonary vascular resistance is very low. The pressure is just sufficient to perfuse the apical areas of the lungs in the erect healthy adult.

The Starling equation can be applied to the pulmonary microcirculation in the same way as any other capillary bed.

**Typical values for the Starling's Forces in Pulmonary Capillaries**

<table>
<thead>
<tr>
<th>Force Description</th>
<th>Value</th>
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<tbody>
<tr>
<td>Capillary hydrostatic pressure (Pc)</td>
<td>13 mmHg (arteriolar end) to 6 mmHg (venous end)</td>
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<tr>
<td>Interstitial hydrostatic pressure (Pi)</td>
<td>Variable but ranges from zero to slightly negative.</td>
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</table>
Oncotic pressure gradient

The interstitial oncotic pressure is high indicating significant leak of protein (mostly albumin) across the thin capillary walls under normal circumstances. The reflection coefficient has been estimated at about 0.5

Considering the typical values and allowing for the reflection coefficient, it can be estimated that the net oncotic gradient is small but favours reabsorption.

Hydrostatic pressure gradient

The capillaries are called intra-alveolar vessels and the pressure they are exposed to is close to alveolar pressure (which has an average value of zero). However, actual measurements of pressure in the alveolar interstitium have found slightly negative pressures (e.g., -2 mmHg). Closer to the hilum, the interstitial pressures become more negative and this favours flow of fluid from the alveolar intersitium into the pulmonary lymphatics.

The capillary hydrostatic pressure is variable because of the effects of gravity. Consider: The erect lung is basically suspended in a gravitational field so the pressure in the vessels at the base of the lung is higher than the pressure at the apex. The pressure difference is equivalent to the height of a static water column from the base to the apex. The distance involved is about 30 cms so the pressure difference is 30 cms H₂O which is about 23 mmHg. If the typical pulmonary artery pressure is 25/8 then it is apparent that the pressure is just adequate for perfusion of the apex of the erect lung.

The pulmonary circuit has a low resistance and about half of this resistance is due to the pulmonary capillaries which have no muscle in their walls. The capillary hydrostatic pressure is quickly affected by changes in pulmonary artery pressure and left atrial pressure without much protective buffering.

Overall Effect

The balance of Starling forces in the lung is generally stated as favouring reabsorption because of the clinical fact that the lungs are generally dry and clearly need to be to facilitate gas exchange. Under normal conditions, there is a small net outward movement of fluid. This is estimated as equal to the pulmonary lymph flow rate. The flow is usually small (e.g., 10 to 20 mls/min) which is only about 2% of the pulmonary blood flow. So despite the net outward hydrostatic pressure gradient and the high reflection coefficient which limits the effectiveness of the oncotic pressure in opposing outward fluid movement, the measured low lymph flow means that the balance of forces is clearly to minimise loss of fluid into the interstitium.
The large surface area and thin capillary walls which assist efficient gas exchange also facilitate filtration from the capillaries to the interstitium. The interstitial fluid move towards the hilum along the spaces beside the vessels and the airways. The interstitial hydrostatic pressure probably becomes more negative as the hilum is approached. The excess filtrate is removed by the pulmonary lymphatics. Lymphatic flow is promoted by the rhythmic external compression that occurs during the ventilatory cycle and by the presence of one way valves.

The Starling equation is not very useful clinically because it is not possible to measure all six of the unknown values. In particular, bedside determination of the interstitial hydrostatic & oncotic pressures and the reflection coefficient is not possible. The clinician is limited to assessments based on plasma protein concentration (as index of capillary oncotic pressure) and values obtainable from use of a pulmonary artery catheter (eg wedge pressure as estimate of left atrial pressure & mean pulmonary venous pressure). A clinical examination and a chest xray are much more useful in assessing & monitoring pulmonary oedema.

Safety Factors Preventing Pulmonary Oedema

For pulmonary oedema to occur, excess fluid must first accumulate in the interstitium (interstitial oedema), then must move into the alveoli (alveolar flooding). The lung is relatively resistant to the onset of pulmonary oedema and this is usually ascribed to several safety factors:

- Increased lymph flow: Increased fluid filtration causes increased lymph flow which tends to remove the fluid.
- Decrease in interstitial oncotic pressure (oncotic buffering mechanism): When filtration increases, the albumin loss in the filtrate decreases. This combined with the increased lymph flow washes the albumin out of the interstitium and interstitial oncotic pressure decreases. This protection does not work if the capillary membrane is damaged eg by septic mediators.
- High interstitial compliance: A large volume of fluid can accumulate in the gel of the interstitium without much pressure rise. Finally, the interstitial tissues become full of fluid, the pressure rises and alveolar flooding occurs. This has been called the bathtub effect: the analogy is that the tub can take a lot of fluid but there comes a point when it is full and suddenly overflows.

These safety mechanisms are quite effective especially in preventing pulmonary oedema associated with rises in capillary hydrostatic pressure. It has been estimated that the capillary hydrostatic pressure can rise to three times normal before alveolar flooding occurs. Surfactant assists in the prevention of alveolar flooding also.