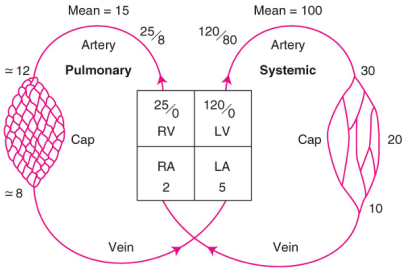
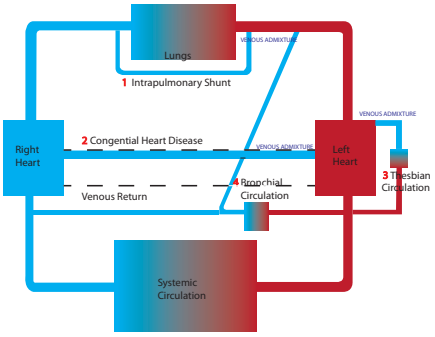
# PULMONARY CIRCULATION




**Physiological features of the pulmonary versus systemic circulation** Blood flow to the pulmonary circulation is roughly equal to that of the systemic circulation (6 litres at rest, up to 25 litres at full exercise). The pressure however is greatly reduced and as a consequence resistance. (R=△P/Blood Flow). As shown in the diagram adjacent the mean pressure for the pulmonary circulation is 15 mmHg compared to 100mmHg for the systemic circulation. The pressure decreases by roughly the same amount in the arterioles, capillaries and venules as opposed to the systemic circulation where the majority of decrease is in the arterioles. A consequence of the reduced pressures is also a right ventricle which has only half the muscle mass of the left ventricle. Whilst the high pressures and resistance of the systemic circulation enable changes to perfusion of specific organs and regions, the control in the pulmonary system is significantly reduced and subject to much greater variation with respect to gravity (discussed in detail below) resulting in the under and over perfusion of parts of the lung. The benefit of this system however is that it is well designed to perform it's primary function of gas exchange, providing pressures which are just enough to perfuse the top of the lungs (and therefore in disease states this may be the first impaired). The reduced pressures also reduce the likelyhood of transudation into the aveoli (pulmonary oedema) which causes significant impairment of gas exchange. The pulmonary arterioles contain little smooth muscle (although vasoconstricition is still possible) and the venules and veins are almost devoid of smooth muscle and are therefore very distensible. These features ensure that the pulmonary system is also able to act as a blood reserve and increase its volume up to five times when systemic return is increased from 0.5-1.0 litres to 2.5-3.0 litres.



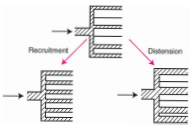

**Pulmonary blood volume** is influenced by both posture (changing from supine to erect decreases volume by a third due to pooling in dependent regions of the sytemic circ) and systemic vascular tone (endogenous or exogenous pressors, g-suits or diving).

**Venous Admixture** refers to the degree of mixed venous blood with pulmonary end capillary Po2 that would be required to produce the observed difference in the endcap Po2 and the arterial Po2. Not all blood that passes through the pulmonary circualtion is oxygenated. This consitutes the intrapulmonary shunt. There is also the bronchial circulation which usually arises from the aorta or intercostal arteries. This supplies the pulmonary system down to the respiratory bronchioles therefore a majority does not tke part in gas exchange. It returns via the pulmonary veins (unoxygenated to the left heart) or the normal venous return (via the right heart therefore not shunted). This represents two of the four types of venous admixture (the others are the Thebsian circulation which supplies the left heart and congenital heart defects). This will be discussed in greater detail later in the respiratory section VQ mismatch.

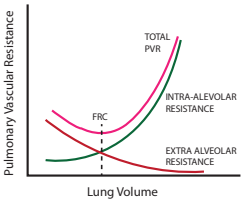
**Pulmonary vascular pressures** As stated previously the pulmonary artery pressure is only about a sixth of the systemic system. There is only a small pressure drop in the pulmonary arterioles and therefore unlike the systemic system the control is reduced. In order to correctly interpret the physiological parameters there are three different pressures that are measured and used in clinical practice. The most common in the intravascular pressure. This is calculated by measuring the pressure in the pulmonary system and comparing to atmospheric pressure. The same method is used in the systemic circulation. The second method is the transmural pressure, which is the difference in the vessel compared to that in the tissue surrounding the vessel. It is measured by an oesophageal balloon (which equates to pleural pressures) and is helpful when you need to exclude the effect of raised intrathoracic pressures (which may suggest a much higher intravascular pressure). The final pressure is the driving pressure. This is the difference between one point in the system and another and is usually used for the calculation of system vascular resistance by comparing the pressures in pulmonary artery and left atrium (△P).

**Pulmonary Vascular Resistance** is an expression of the relationship between driving pressure (DP) and flow (often simplified to the cardiac output CO), as in the case of resistance to gas flow. As previously stated it may be expressed as; PVR = DP/CO. It is important to note however that this relationship is non linear, which is mainly due to passive changes in the pulmonary circulatory system although active changes may play some part.

**Passive changes in pulmonary blood flow and resistance.** Three main mechanisms contribute to passive changes in PBF/PVR. Recruitment and distension due to increases in cardiac output, changes due to the opposing influences of different lung volumes have on intra and extra alveolar blood vessels and the effect of gravity and the so called west's zones of perfusion. Each are considered seperately below.

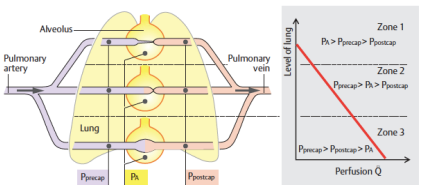



The pulmonary circulation can adapt to large changes in cardiac output with only small increases in the pulmonary pressures. Therefore by using the equation shown above there must be corresponding decrease in vascular resistance with inrcrease in CO (PVR x CO=DP). This is due to two adaptations, recruitment of previously underperfused pulmonary vessels and the other is distension of the entire pulmonary vasculature. Distension is the more important adaptation.




**Lung inflation** is the second passive influence on pulmonary blood flow. At low lung volumes the dependent tissues are compressed which increases the resistance overall due to compression of the extra-alveolar vessels (in the parenchyma). At high lung volumes the vessels that are within alveolar become compressed which also contributes to a significant increase in resistance, and the extra alveolar vessels become slightly distensed due to traction which slightly reduces resistance. Thus overall there is increased resistance at both low and high volumes, with the least resistance around the point of FRC.

**Gravity and West's Zones of the Lung** The interplay between of alveolar pressure, flow rate and vascular resistance is best considered by dividing the lung field into three zones. This was first described by West and colleages using a starling resistor model. It is best explained using a weir model. In zone 1 the alveolar pressure exceeds the precapillary pressure therefore there is no flow. This does not occur normally although may be present if the pulmonary artery pressure is decreased due to shock or iatrogenically due to PPV. Zone 2 is a situation where the alveolar pressure is less than the precap but more then the post cap. In this setting, the flow is determined by the pressure difference between the precap and the alveolar. In the third zone the alveolar pressure is less than both the precap and post cap, therefore it is these two parameters which determine flow. A Zone 4 is sometimes referred to where an increase in extralveolar pressures due to gravity increase resistance and thus flow as described above. Therefore at the very base there is actually a reduction in flow compared to zone 3.




**Active changes in pulmonary blood flow and resistance.** In addition to the three mechanisms described above the pulmonary vasculatrue is able to control resistance by both active vasoconstiction and vasodilation. It is believed that the lung is held in a state of active vasodilation. Cellular mechanisms which include receptors and their associated second messengers do influence vascular tone but many of these are poorly understood. There is good evidence that the basal production of NO occurs in normal lungs and contributes to maintenence of low pulmonary vascular resistance. There are three autonomic systems which influence active control of vascular tone, adrenergic, cholinergic and non-adrenergic non-cholinergic (NANC). Adrenergic control is the most important, alpha 1 receptors mediate vasoconstriction and predominate, beta 2 receptors will cause vasodilation to a lesser extent. Humoral control actioned by catecholamines, Eicosanoids (which may mediate the development of sepsis associated PHTN) amines and peptides play a role. Drugs are also important, especially inhaled NO which causes localised vasodilation, prostacyclin, ACE inhibitors and calcium channel antagonists reduce pulomnary pressures. Phosphodiesterase inhibitors such as sildenafil have significant effects as do endothelin receptor antagonists such as bosanten.

**Hypoxic Pulmonary Vasoconstriction** is the last active controller of vascular resistance considered here and is it is own section. It is influenced by both alveolar and mixed venous oxygen tensions although the alveolar is most important. Regional vasocontriction to poorly ventilated alveoli is beneficial as a means of improving V/Q matching. It is also critical in the neonatal period for establishing normal breathing. Long term however it leads to changes which result in PHTN. Both respiratory and metabolic acidosis augment HPV and alkalosis of any cause results in pulmonary vasodilation and thus attenuates HPV.