The oxygen cascade describes the process of declining oxygen tension from atmosphere to mitochondria.

The purpose of the cardio-respiratory system is to extract oxygen from the atmosphere and deliver it to the mitochondria of cells. Oxygen, being a gas, exerts a partial pressure, which is determined by the prevailing environmental pressure. At sea level, the atmospheric pressure is 760mmHg, and oxygen makes up 21% (20.094% to be exact) of inspired air: so oxygen exerts a partial pressure of 760 x 0.21 = 159mmHg. This is the starting point of the oxygen cascade, as one moves down through the body to the cell, oxygen is diluted down, extracted or otherwise lost, so that at cellular level the PO_2 may only be 3 or 4mmHg.

The first obstacle that oxygen encounters is water vapor, which humifies inspired air, and dilutes the amount of oxygen, by reducing the partial pressure by the saturated vapor pressure (47mmHg). This will, obviously, affect the PIO2 (the partial pressure of inspired oxygen), which is recalculated as: (760 - 47) x 0.2094 = 149mmHg.

Air consists of oxygen and nitrogen, but as gas moves into the alveoli, a third gas, carbon dioxide, is present. The alveolar carbon dioxide level, the PACO2, is usually the same as the PaCO2, which can be measured by a blood gas analyzer. The alveolar partial pressure of oxygen PAO2 can be calculated from the following equation: PAO2 = PIO2 – PaCO2/R. R is the respiratory quotient, which represents the amount of carbon dioxide excreted for the amount of oxygen utilized, and this in turn depends on the carbon content of food (carbohydrates high, fat low). For now let us assume that the respiratory quotient is 0.8, the PAO2 will then be 149 – (40/0.8) = 100mmHg (approx).

The next step is the movement of oxygen from alveolus to artery, and as you would expect, there is a significant gradient, usually 5 –10 mmHg, explained by small ventilation perfusion abnormalities, the diffusion gradient and physiologic shunt (from the bronchial arteries).

Oxygen is progressively extracted thru the capillary network, such that the partial pressure of oxygen in mixed venous blood, PVO2, is approx 47mmHg.

What is essential to understand about the oxygen cascade is that if there is any interference to the delivery of oxygen at any point in the cascade, significant injury can occur downstream. The most graphic example of this is ascension to altitude. At 19,000 feet (just above base camp at Mount Everest, the barometric pressure is half that at sea level, and thus, even though the FiO2 is 21%, the PIO2 is only 70mmHg, half that at sea level. Conversely, if the barometric pressure is increased, such as in hyperbaric chambers, the PIO2 will actually be higher.

Four factors influence transmission of oxygen from the alveoli to the capillaries 1. Ventilation perfusion mismatch, 2. Right to left shunt, 3. Diffusion defects, 4. Cardiac output.

The amount of oxygen in the bloodstream is determined by the oxygen carrying capacity, the serum hemoglobin level, the percentage of this hemoglobin saturated with oxygen, the cardiac output and the amount of oxygen dissolved (see below).

The PVO2 is determined by whole body oxygen demand, and the capacity of the tissues to extract oxygen. In sepsis there appears to be a fundamental abnormality of tissue oxygen extraction.