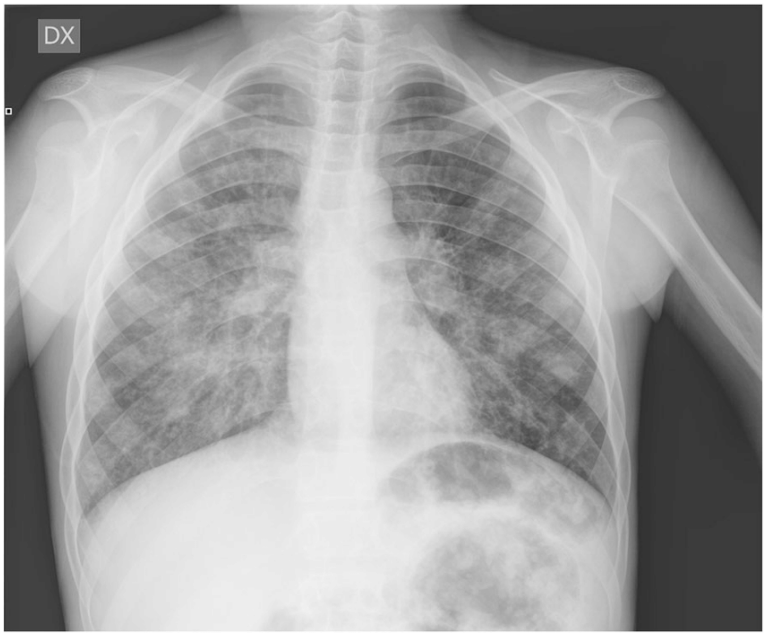
ARDS & Proning

What is ARDS?



So ARDS is non cardiogenic pulmonary oedema., with fluid in the alveolar space.

It has three defining criteria:

- Acute onset of bilateral opacities on the chest x ray
- Low oxygen levels
- Pulmonary oedema is not due to heart failure.

It further defined mild moderate and severe ARDS using the P:F ratio which have increasing levels of mortality.

It can develop after direct lung injury such as in pneumonia or aspiration of gastric contents, when the alveoli are subjected to damage directly.

Or because of indirect lung injury such as in sepsis, pancreatitis, or severe trauma, in which case inflammatory mediators in the circulation will make their way to the lung vasculature.

On the left side of the illustration below, you can see that the normal alveoli have a thin epithelial layer, and the inside is coated with surfactant.

The gas exchange, therefore, can take place very easily across this area by diffusion.

The epithelial layer is normally tight and prevents any fluid from crossing over into the alveoli.

The type II cells are where the surfactant is produced.

On the right side of the illustration, we can see the damaged alveoli where the inside has filled with this protein rich pulmonary oedema fluid.

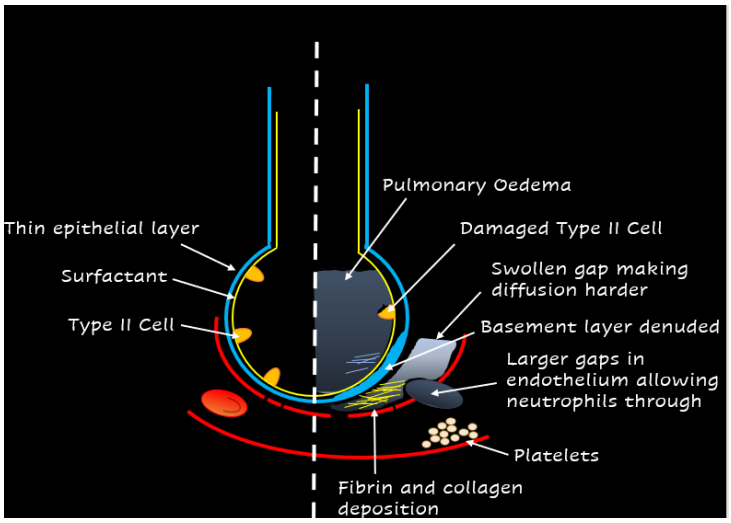
This is filled with inflammatory cells.

These inflammatory cells are releasing chemicals which can make the process worse.

The alveolar macrophages recruit neutrophils and circulating macrophages to the site of injury.

This then goes on to encourage proteases and cytokines amongst others which perpetuate the inflammatory response.

The border between the epithelial and the endothelial layers is swollen, creating a bigger gap for the gases to diffuse across making that process harder.



The basement layer is a thin, pliable sheet-like type of extracellular matrix, that provides cell and tissue support, and protects the alveoli from stress.

This is now denuded or stripped of its covering, and necrotic.

The capillary endothelium is also involved. As the endothelium is acted upon by a range of stimuli it itself becomes dysregulated leading to the endothelial surface becoming abnormal and having larger gaps for fluid and other substances to move through.

Neutrophils can cross over into the interstitium and there is greater platelet activation. These neutrophils will release proteases which can break down the elastic tissue around the alveoli affecting the compliance.

The overwhelming activation of neutrophils contributes to surrounding tissue damage and even lung dysfunction.

In COVID-19 ARDS patients, higher counts of neutrophil are observed and represent a predictor of poor outcome.

The worsening inflammation and injury will damage the type II endothelial cells reducing the production of surfactant causing a decreased compliance.

Type II cells also have a role in managing lung fluid.

Activated fibroblasts secrete several extracellular matrix proteins within the interstitium but also migrate into the alveolar space where they form attachments to damaged basement membranes and contribute to the intra-alveolar fibrosis which can predominate in some cases.

This can lead to established fibrosis and the obliteration of alveolar spaces with a dense irregular matrix.

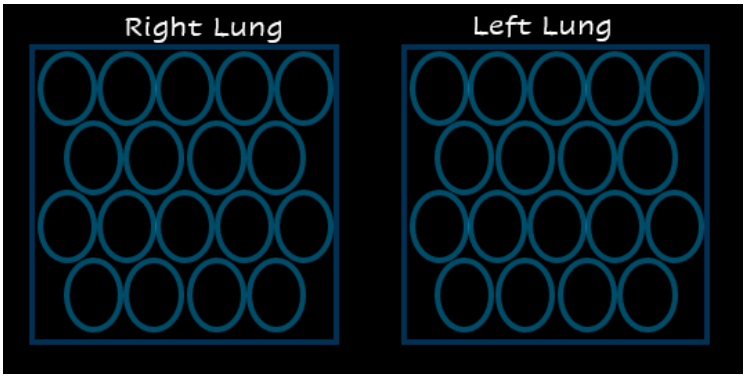
Over time scarring will occur which will go on to reduce diffusion across the membranes.

Why do we prone?

The physiology of why we prone is key to its understanding and, I think, gives a greater appreciation of its efficacy.

There are three terms that play a key part in the understanding of why we prone-

- Ventilation,
- Perfusion
- and Gravity.



The first part to understand is the shape matching of the lung and the effect that has on the patient's ability to ventilate well.

Let us start with a simple diagram of the lung which we will gradually add to, to aid understanding.

Imagine that the patient is now lying on their back and that we have taken a slice through their lungs.

We are now looking at that slice from the position of the feet up.

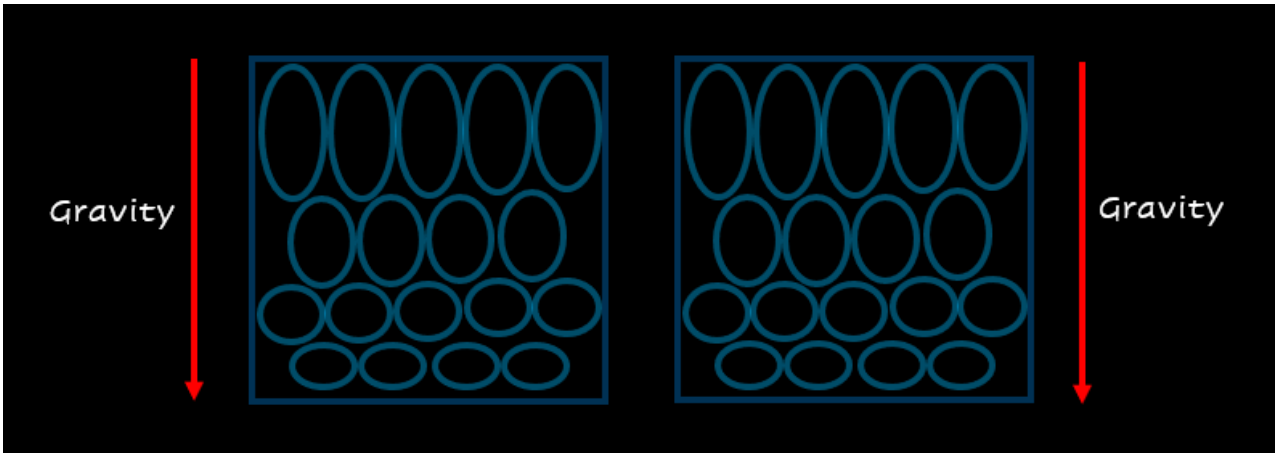
We represent each lung in an enclosed box, which represents the pleura and the chest wall, giving the lung some limitations as to how it can expand.

You can see the individual alveoli represented by the circles within the box. In this diagram they are all an equal shape and size.

However, our lungs are subject to the force of gravity, just like everything else and when we add that, as above, you can see that the picture changes in the illustration below.

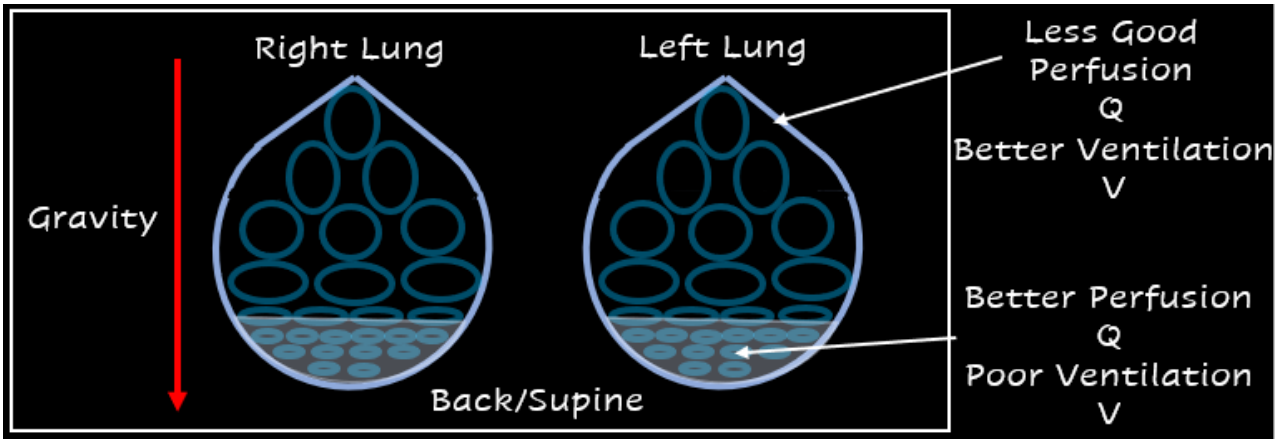
Now, the alveoli at the top of the image, or the ventral part of the lung are pressing down on the alveoli in the lower part of the lung or the dorsal part.

So, due to this compression effect we have larger alveoli higher up and more compressed alveoli lower down.



We now consider the true shape of the lung and I represent this by the tear shaped lung I have illustrated below.

This is an exaggeration of the true shape but helps illustrate the principles we need to understand.



This shape means that, when the patient is on their back, or supine, there is slightly less room at the top then the bottom.

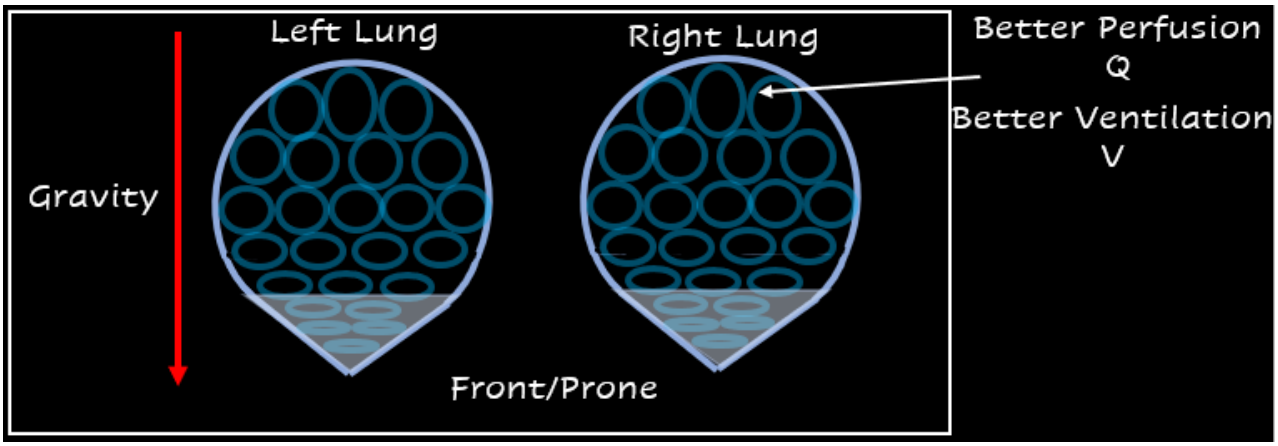
Consequently, with the added gravity much of the lung tends to drop into the lower part where it is compressed by the lung above it.

The key point here is that there is a lot of room for this compressed lung to fall into.

Add to this the fluid that will also be affected by gravity and you can see that now we have compressed alveoli surrounded by the fluid- making diffusion in these alveoli much harder.

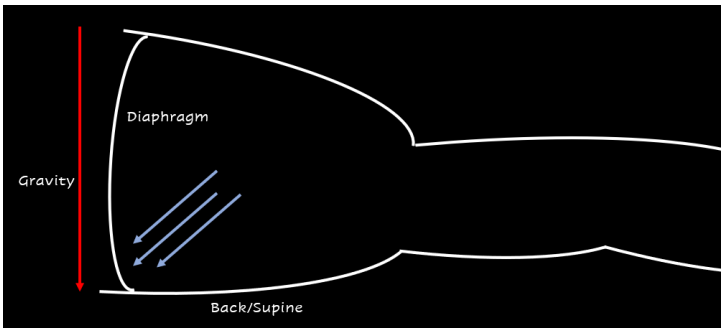
The perfusion to the dorsal part of the lung is a little better than the perfusion to the ventral part- and the key here is that does not change significantly when the patient is proned.

So, you can see in the illustration above, where the patient is supine, we have the better perfusion where there is the poorest ventilation and the better ventilation with the slightly less good perfusion- in other words a V/Q mismatch.



In the illustration above, we have proned our patient so, because of the shape matching, and the effects of gravity on the fluid in the patient's lungs we now have the better perfusion taking place where there is the better ventilation- improving the V/Q balance.

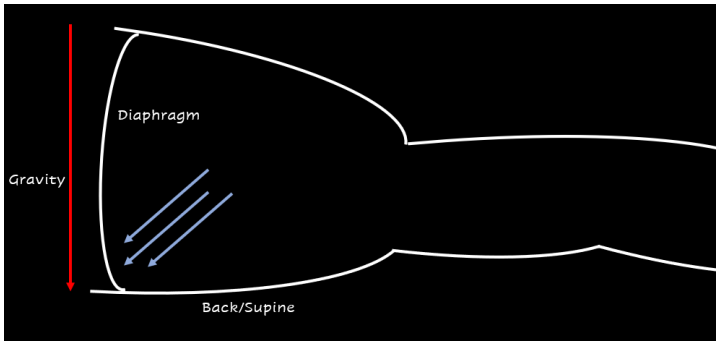
The other effect to be considered is the way the abdominal contents can add pressure to the diaphragm when it is moving up and down.



The arrows indicate the pressure from the abdomen on the diaphragm.

Remember that there is already a V/Q mismatch in this region when the patient is supine, and this added pressure contributes to make it worse.

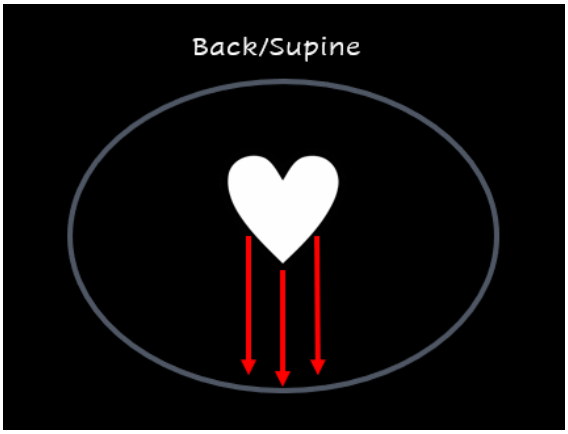
If we then turn our patient onto their front, as in the second illustration the pressure remains the same but is now pressing on the front of the diaphragm and no longer the back.
Again, remember that the back of the lung, when proned, is where we have the improved V/Q matching, so now we have also relieved some of that pressure also.

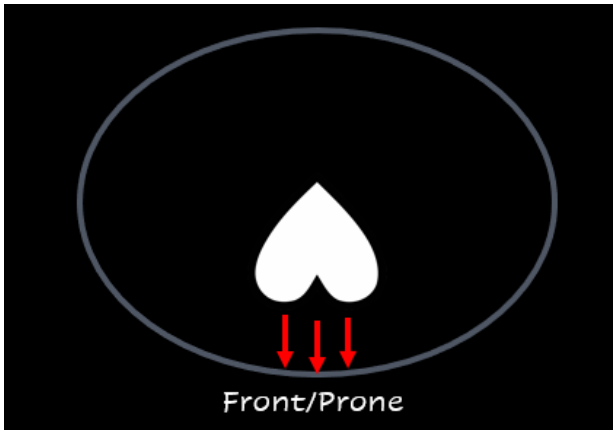


The final point is about the position of the heart within the chest cavity.

The heart is nearer to the front of the chest than the back.

This means that, when the patient is lying on their back the weight of the heart, gravity in action again, is lying on top of much of the lung, thereby adding to the compression.





You can see here that, when we lie the patient on their front there is less lung for the heart to lie on top of and much of it is supported by the sternum.

This again reduces some of the compression on the alveoli and certainly takes some of the weight off that area of the lung with the best V/Q match.