The concepts of venous admixture and shunt

This chapter is most relevant to Section F6(vi) from the 2017 CICM Primary Syllabus, which expects the exam candidates to be able to *"explain the concept of shunt"*, and to Section V(ii), which asks them to *"explain venous admixture, its relationship to shunt and ventilation-perfusion (V/Q) mismatch"*. This specific matter has appeared in Question 6 from the second paper of 2009. The fact that it has only appeared once should not discourage exam candidates from becoming familiar with the topic, as it is fairly fundamental. If one's eyes glaze over from the discussion of the difference between venous admixture and shunt, then at the very least the Berggren equation should be firmly understood and committed to memory, because it is fair game for future questions and viva stations.

In summary:

- **Shunt** is the blood which enters the systemic arterial circulation without participating in gas exchange
- **Venous admixture** is that amount of mixed venous blood which would have to be added to ideal pulmonary end-capillary blood to explain the observed difference between pulmonary end-capillary PO_2 and arterial PO_2
- **Shunt fraction** is the calculated ratio of venous admixture to total cardiac output
- **The shunt equation**, otherwise known as the Berggren equation, is used to calculate the shunt fraction:
  $Qs/Qt = (Cc_{O2} - Ca_{O2}) / (Cc_{O2} + Cv_{O2})$
  where
  Qs/Qt = shunt fraction (shunt flow divided by total cardiac output)
  Cc_{O2} = pulmonary end-capillary O_2 content, same as alveolar O_2 content
  Ca_{O2} = arterial O_2 content
  Cv_{O2} = mixed venous O_2 content
- Sources of venous admixture include:
  - **"True" intrapulmonary shunt**, blood which passes through lung regions where V/Q = 0
  - **V/Q scatter**, blood which passes through lung regions where V/Q < 1.0
  - **Thebesian veins**, which contribute myocardial venous blood with low oxygen content
  - **Bronchial veins**, which drain the bronchial walls
  - **Intracardiac right-to-left shunts**
- Normal shunt fraction in healthy adults is 4-10%

The most detailed explanation of these concepts can be found in "Understanding the meaning of the shunt fraction calculation" by Cruz & Metting (1987), but this article is not freely available, and by virtue of being comprehensive may be unreasonable for last-minute revision. A better reference is probably Bigeleisen (2001), which is not only a free article, but also one which was written with the express intention of explaining these concepts to people who are then expected to teach others.

The relationship of venous admixture to shunt

What is "shunt"? An authoritative-sounding document from the 1970s ("Glossary on respiration and gas exchange", Hughes et al, 1973) defines it as :

> *"Vascular connection between circulatory pathways so that venous blood is diverted into vessels containing arterialized blood"*

So, that's clearly not the sort of shunt we are talking about here. For respiratory physiology, *Wests'* (p.68

of the 10th edition) defines shunt as:

> *"blood that enters the arterial system without going through ventilated areas of the lung"*

West does not try to make a distinction between venous admixture and shunt, but in other textbooks (*Nunn's* and *Levitzky* included) the two terms are made distinct. In the 8th edition of *Nunn's* (p. 123), venous admixture is defined as:

> *"the degree of admixture of mixed venous blood with pulmonary endcapillary blood that would be required to produce the observed difference between the arterial and the pulmonary end-capillary PO_2 (usually taken to equal ideal alveolar PO_2)"*

Thus, "venous admixture" is the calculated estimate of how much hypoxic blood would be required to produce the measured arterial oxygen results, for a given cardiac output. It is a volume of deoxygenated blood from the venous circulation which appears to have bypassed the lungs, not participating in any gas exchange.

So... How is this different to shunt? Well. The two terms are often used interchangeably. For the editors of *Nunn's,* the confusion in students must have been viewed as having such magnitude as to warrant a brief subsection on the nomenclature, at the end of which the authors admitted that, even though the two concepts are distinct, *"venous admixture is ...often loosely termed shunt".*

However, venous admixture is not shunt. It is a calculated volume which *appears* to have bypassed the pulmonary gas exchange surface. It is the product of the shunt equation, which assumes that there are only two kinds of alveoli (perfectly ventilated and perfectly collapsed). "True" intrapulmonary shunt, in contrast, is the volume of venous blood which *actually* bypassed the aerated alveoli, and returned deoxygenated blood to the left heart via the pulmonary circulation. "True" shunt does not integrate the contribution of Thebesian veins and alveolar regions with V/Q ratios between 0 and 1.0, or any other added sources of extra venous blood contributing to the systemic circulation (like intracardiac right-to-left shunts) and therefore the calculated venous admixture volume will usually be larger.

Thus, venous admixture does not accurately estimate the volume of true intrapulmonary shunt, nor does it help to determine exactly where that extra venous blood is coming from. The very term "venous admixture" implies that there is some known amount of hypoxic venous blood which gets mixed with the arterial circulation, but in actual fact, there is no such thing; you never quite know how much shunt blood volume there is, or how hypoxic that blood is. Instead, one *calculates* a certain fraction of the cardiac output which consists of that blood. This is a completely reasonable shortcut, because it is actually impossible to measure "true" shunt, as practically one can never separate the fraction of blood coming from truly unventilated lung units (V/Q =0) from blood which comes from merely *incompletely* ventilated units (V/Q < 1.0). For this reason, we resort to using venous admixture as a surrogate for shunt, and report it as "shunt fraction", or F_{shunt}.

Types of shunt and venous admixture

A classification system seems to exist for shunt, which tends to vary across textbooks (whereas some, eg. *West's*, abandon the whole idea of classifying things). Not only are the categories different, but the same nominal category may have different meanings to different authors. For example, here is a comparison of *Nunn's* and *Levitzky:*

Different Classification Systems for Venous Admixture and Shunt

From *Nunn's*, 8th edition	From Levitzky, 7th edition

• Anatomical shunt:
  ○ Physiological shunt
  ■ Bronchial veins
  ■ Thebesian veins
  ○ "True" shunt
  ○ Intracardiac shunt
• "Virtual" shunt
• Pathological anatomical shunt
• V/Q scatter
• Physiological shunt
	Physiological shunts
	• Physiological anatomical shunt
	  ○ Bronchial veins
	  ○ Thebesian veins
	• Pathological anatomical shunt
	  ○ Intracardiac shunt
	• Intrapulmonary shunt
	  ○ Absolute intrapulmonary shunt ("true" shunt)
	  ○ "Shunt-like states": V/Q scatter, i.e. V/Q < 0

From *Basic Physiology for Anaesthetists* by Chambers et al (2015)

- Physiological shunt:
  - Anatomical shunt
    - Bronchial veins
    - Thebesian veins
  - Functional shunt
    - V/Q scatter
- Pathological shunt:
  - Intracardiac shunt
  - Pulmonary AVM
  - Intrapulmonary shunt (true shunt)

These are only a few of the possible taxonomies. Judging by the lack of literature references, these were not composed by the work of some sort of scientific body, but rather concocted by each textbook author independently. As such, it is impossible to say which of them is "better". The exam candidates are invited to choose a system and stick with it.

Without trying to justify any of the existing classification systems or trying to invent a new one, the following list of shunts and shuntish admixtures is offered in an unordered state.

Different sources of venous admixture

- **"True" shunt through useless lung:** blood passing through a diseased pneumonic lung (or one which has collapsed), with V/Q ratio of 0 (i.e. no V, all Q). This blood will exchange no gas.
- **"V/Q scatter":** Lung regions which have a V/Q ratio less than 1 will have inefficient gas exchange, and will return pulmonary venous blood which is incompletely oxygenated. As the name suggests, the oxygen content of such blood will be available in a wide range, from blood which closely resembles mixed venous, to blood which is only slightly deoxygenated. The category of anatomical shunt for some reason excludes this form of venous admixture.
- **Thebesian veins**, otherwise known as *venae cordis minimae,* are tiny valveless veins in the walls of the four cardiac chambers. Their contribution to blood flow is piddling - examination of anaesthetised subjects has suggested that thebesian vein flow contributes 0.12% to 0.43% of the total aortic flow. However, the oxygen content within these veins is probably very low, and the impact on the A-a difference is not trivial.
- **Bronchial veins** contribute probably no more than 1% of total cardiac output. This is blood which leaves the aorta, nourishes the bronchial wall, and then rejoins the central circulation by draining into the pulmonary veins. According to *Nunn's* , in patients with bronchiectasis or COPD this contribution could be considerable - as much as 10% of the cardiac output.
- **Congenital heart disease** with right-to-left shunting is a possibility that should be mentioned, as

it allows the right heart to eject into the left circulation, bypassing the lungs.

- **Intrapulmonary arteriovenous connection**, such as an AVM or fistula, would do exactly the same thing as the intracardiac shunt
- **Intrapulmonary sources of poorly oxygenated blood**, such as blood draining from the anoxic depths of a lung tumour, or portopulmonary shunts in liver disease, can deliver very oxygen-poor blood to the pulmonary venous circulation
- **Virtual shunt** is virtual in the sense that it may not actually exist. When one's measurement of shunt is performed without a mixed venous blood sample, the resulting shunt is referred to as virtual. As far as one can tell, this terminology is unique to works published by, or about, Nunn (see Lawler & Nunn, 1984). According to the original authors, this is *"the shunt which would explain the relationship between arterial PO2 and inspired oxygen concentration if the arterial-to-mixed venous oxygen concentration difference was 5 vol %".*

Previous chapter: Physiological consequences of increased dead space

Next chapter: Measurement and estimation of shunt

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Physiological consequences of increased shunt

This chapter is most relevant to Section F6(vi) from the 2017 CICM Primary Syllabus, which expects the exam candidates to be able to *"explain the concept of shunt, its physiological effects and its measurement"*. This chapter will focus on *"its physiological effects"*, which could be easily summarised as "all gas-related things get worse". It is remarkable that this has never appeared in the past papers, and by this very fact one may be assured that one day it will.

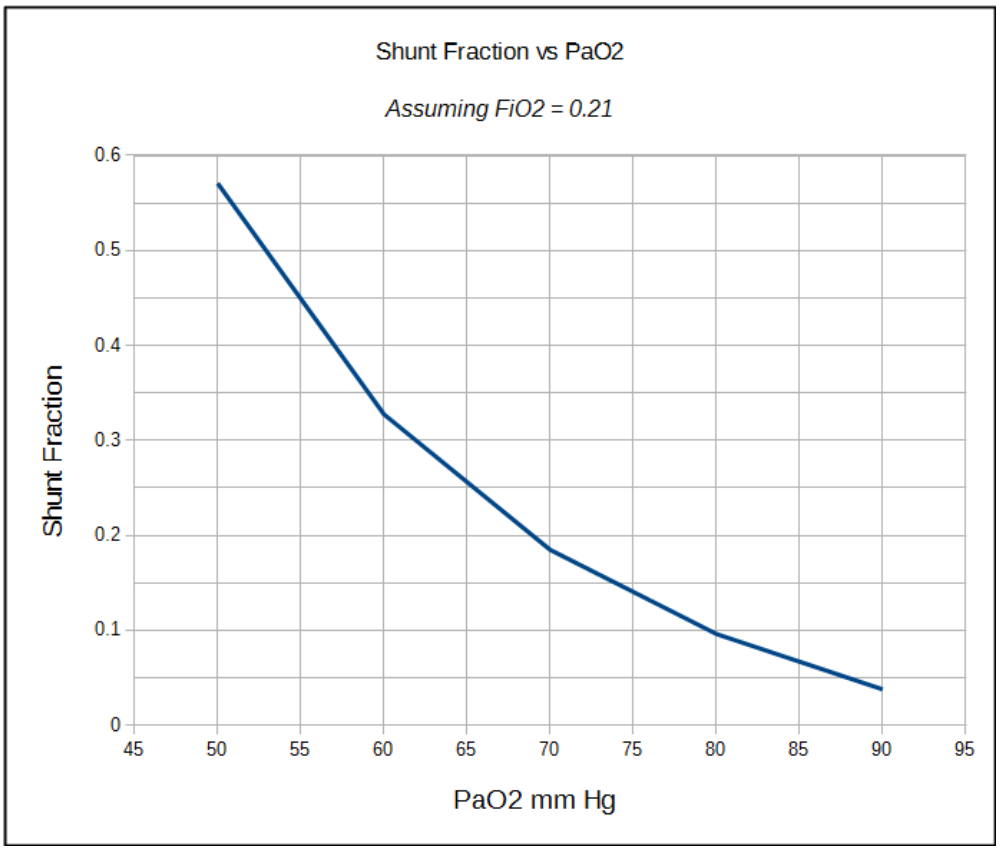
In summary:

- **The effects of shunt on oxygenation:**
  - With worsening shunt, the oxygenation of arterial blood will decrease
  - PaO_2 of arterial blood decreases roughly in proportion to increasing shunt
- **The effects of increasing FiO_2 on shunt:**
  - The greater the shunt, the less effect increasing FiO_2 has on improving oxygenation
  - With a shunt fraction of 50% or more, increasing the FiO_2 will have minimal effect on the PaO_2
- **The effects of shunt on CO_2 clearance:**
  - Shunt has little effect on $PaCO_2$
  - The main reason is the increase in alveolar ventilation associated with hypercapnia
  - In patients who are unable to increase their alveolar ventilation, $PaCO_2$ may increase sllightly (eg. by up to 15-30% with a shunt fraction of 50%)
  - Low cardiac output and metabolic acidosis increase the effect of shunt on $PaCO_2$

The best free peer-reviewed reference for this is Bigeleisen (2001), which is an excellent resource to *"help students master these equations as well as their practical limitations"*. Original papers by J.F Nunn (1966) and later works by Petersson & Glenny (2014) are also useful. From the non-peer-reviewed FOAM world, the shunt post from the PFTforum blog is an excellent overview, though it may earn undeserved scowls of disapproval from the FOAM-weary CICM Illuminati.

Effect of shunt on oxygenation, and effect of oxygen on shunt

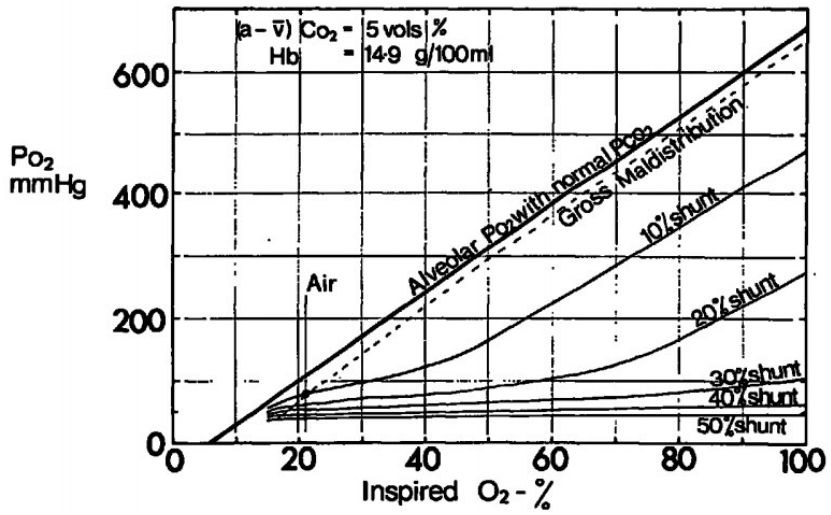
In short, venous admixture gives rise to systemic hypoxemia. The degree of hypoxemia is generally said to be roughly proportional to the shunt fraction. This relationship was discussed at great detail here at the PFTforum, and their graph is reproduced here with zero permission but in the spirit of Creative Commons camaraderie:



This is a demonstration of the range of arterial PO_2 values one may be expected to generate as one's shunt increases. At a shunt of 50%, the arterial oxygen tension at 21% FiO_2 is somewhere in the order of 53 mmHg. A shunt fraction of 25% is enough to drop one's oxygen saturation into the low 90s, prompting worried nursing staff to increase the oxygen flow rates. The latter manoeuvre, however, may have little effect.

Effect of oxygenation on shunt

As is mentioned in the end of the chapter on the measurement of shunt, lung regions with true shunt (V/Q = 0) are not expected to increase their endcapillary oxygen content in response to an increase in alveolar oxygen. From this, one might expect that, as the shunt fraction increases, so the increase of FiO_2 should yield smaller and smaller benefits, ultimately becoming pointless. This is indeed the scenario described by Nunn's iso-shunt lines, which one can find in virtually any physiology textbook (most of all in *Nunn's*).



In short, this is a graphical representation of the effect of increasing the FiO_2 on the arterial oxygenation, at different degrees of shunt. This thing appears to have its origin in a 1966 paper by J.F Nunn, where no experimental data or calculations are offered in support of it (so presumably it comes from another, earlier publication by the same author). The original 1966 image is used here, out of immense respect for the author. Later editions of his textbook justify the existence of this graph by saying that *"it has been found useful to prepare a graph of the relationship at different levels of venous admixture",* because one finds oneself constantly obsessing over the FiO_2 and PaO_2 in the course of clinical practice. It would indeed have been very useful in the distant past where oxygen saturation monitoring was unknown and one needed to adjust the FiO2 on the basis of infrequent PaO_2 measurements. In practice, *"despite their simplicity, the isoshunt lines did not achieve popularity because pulmonary critical care involves treating more than a single parameter and because it was impractical to carry the nomogram around"* (Bigeleisen, 2001). The main use of this graph in the modern era is to demonstrate for students of physiology that, with a true shunt of 50%, one's fiddling with the FiO_2 knob on the ventilator will yield little improvement in oxygenation.

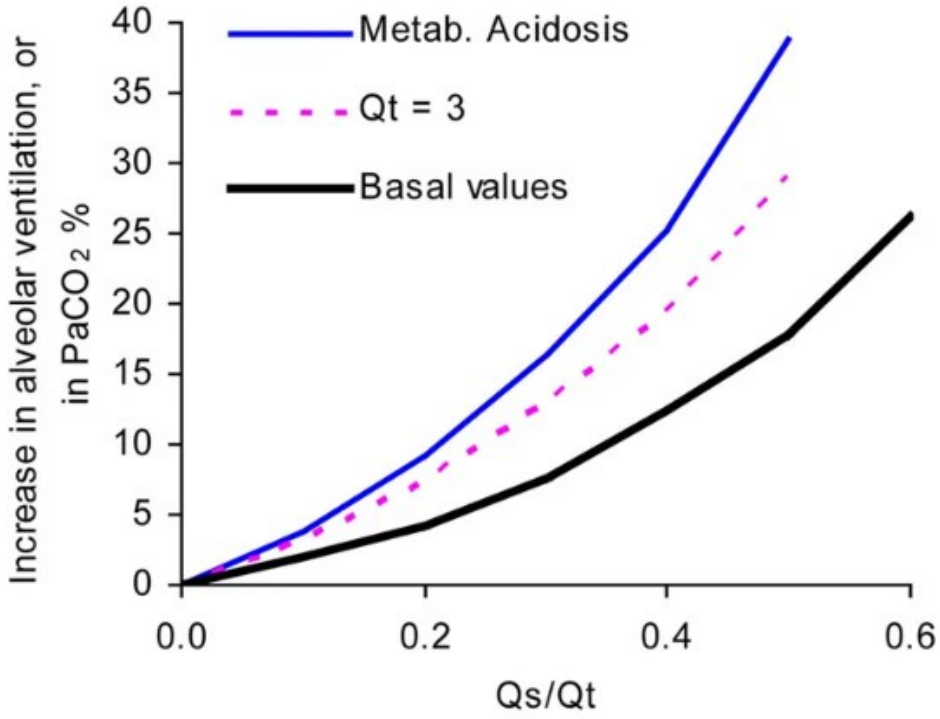
Effects of shunt on CO_2 clearance

In short, shunt does not make much of a difference to CO_2 clearance, and in fact CO_2 clearance may increase in the presence of a massive shunt.

True, the shunted blood has a high CO_2 content. Bypassing the gas exchange surfaces of the lungs, that CO_2 content ends up in the arterial circulation, and the arterial CO_2 should be expected to rise proportionally to the shunt fraction. However, in practice it does not. The main reason for this is that central control of ventilation maintains a very tight grip on the arterial CO_2 concentration, and any rise in arterial CO_2 is matched by an increase in alveolar ventilation.

Now, the blood which travels through non-shunt regions of the lung may already be maximally oxygenated by the available oxygen, and an increase in FiO_2 will not change the arterial oxygenation. However, that non-shunt blood is not maximally *decarboxylated,* and increasing the minute ventilation will still yield a reasonable improvement in CO_2 clearance even in the presence of a large shunt. This is enhanced by the hypoxic ventilatory drive which will increase the respiratory rate irrespective of the arterial CO_2 tension. In short, any patient with the capacity to increase their minute ventilation will be able to compensate for the hypercapnic effects of even a very large shunt.

But let's say the patient was unable to increase their minute volume, and the shunt fraction was substantial. how much would the $PaCO_2$ increase? Turns out, not very much. Niklason et al (2008) determined that a shunt fraction of 50% would only increase the $PaCO_2$ by 15-30%, and that's in a patient whose minute volume was fixed. Their original images are reproduced below:



As one can see, the increase in $PaCO_2$ was greatest where cardiac output was low ("Qt = 3" on their graph means a cardiac output of 3L/min, or a cardiac index around 1.7). The main reason for this was thought to be an increase in mixed venous CO_2, and therefore more CO2 delivery through the shunt. Similarly, where there was a metabolic acidosis, the effect of the shunt on $PaCO_2$ was higher because metabolic acidosis suppresses the Haldane effect (Cavaliere et al, 2002), decreasing the affinity of deoxygenated haemoglobin for CO_2 and therefore increasing the concentration of free CO_2 in the venous blood.

Previous chapter: Measurement and estimation of shunt

Next chapter: The oxygen cascade

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Measurement and estimation of shunt

This chapter is most relevant to Section F6(vi) from the 2017 CICM Primary Syllabus, which expects the exam candidates to be able to *"explain the concept of shunt, its physiological effects and its measurement"*. The concept of shunt and venous admixture being explained elsewhere, this chapter will focus on the *"its measurement"* part of the syllabus. This discussion is made more difficult as in actual fact shunt cannot be measured directly, and so all such "measurements" are actually calculating venous admixture.

In summary:

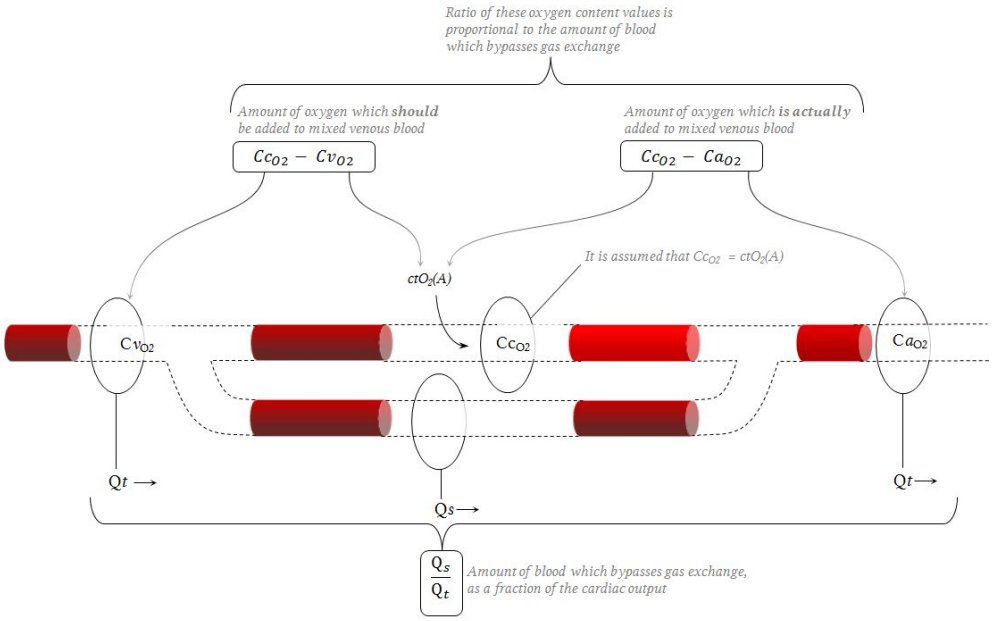
- **The shunt equation**, otherwise known as the Berggren equation, is used to calculate the shunt fraction:
  $Qs/Qt = (Cc_{O2} - Ca_{O2}) / (Cc_{O2} + Cv_{O2})$
  where
  Qs/Qt = shunt fraction (shunt flow divided by total cardiac output)
  Cc_{O2} = pulmonary end-capillary O_2 content, same as alveolar O_2 content
  Ca_{O2} = arterial O_2 content
  Cv_{O2} = mixed venous O_2 content
- **The estimated shunt fraction** ($\dot{Q}shunt_e$) can be calculated if an assumed value is substituted for Cv_{O2}) in the Berggren equation
- "True" shunt can be identified if the subject is made to breathe 100% FiO2
  - This decreases the contribution from V/Q scatter
  - With 100% FiO2, the measured shunt fraction is the "true" intrapulmonary shunt
  - This technique does not separate "true" shunt from anatomical shunt (contribution from thebesian veins and bronchial veins) or cardiac defects

The best free reference is Bigeleisen (2001), which is an excellent resource to "help students master these equations as well as their practical limitations".

The shunt equation

Confusingly, the shunt equation is used to estimate the venous admixture, relying on a model of the lung which divides it conceptually into regions with V/Q of 1 and regions with V/Q of 0.

As always, these things are easier to represent as a big confusing diagram.



Let us dissect these variables.

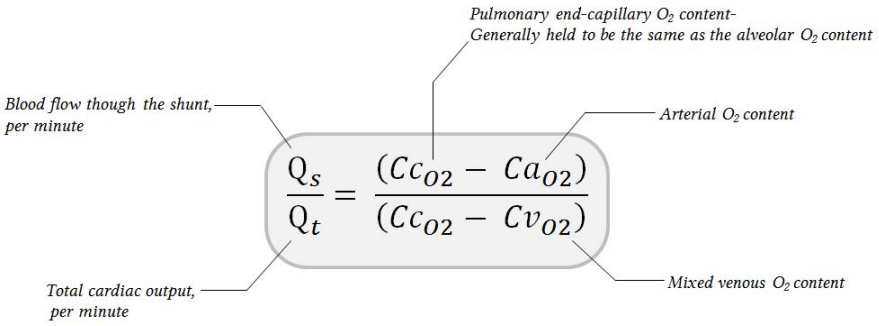
- Cv_{O2}, the oxygen content of mixed venous blood, is a known variable (as you can measure it), and it is returning to the lungs at a flow rate equal to the cardiac output, Qt.
- Cc_{O2} is the oxygen content of pulmonary endcapillary blood, and it is assumed that it is equal to $Ct_{O2}(A)$, the alveolar oxygen content. This assumption is based on a two-compartment model, where this compartment is perfectly oxygenated (i.e. the arterial and alveolar oxygen content is the same).
- $(Cc_{O2} - Cv_{O2})$ is the difference in oxygen content between mixed venous and "perfect" endcapillary blood
- Qs is the flow through the shunt fraction, which is returning perfectly unchanged mixed venous blood back to the systemic arterial circulation
- Ca_{O2} is the oxygen content of systemic arterial blood, which will be lower than the content of "perfect" endcapillary blood because it is mixed with the relatively hypoxic Qs.
- $(Cc_{O2} - Ca_{O2})$ is the difference in oxygen content between "perfect" endcapillary blood and systemic arterial blood; this drop in oxygen content is due to the venous admixture.

Of these quantities, all are known except the *Qs*, which is the shunt fraction. Well, the pulmonary end-capillary oxygen content is not known, but it is assumed. Obviously nobody is ever going to get into those capillaries and measure it directly. Instead an assumption is made that the ventilated areas of the lung are perfused with "ideal" capillaries, and the gas exchange in them is so perfect that their end-capillary oxygen tension is equal to alveolar oxygen tension, and their saturation is 100%.

Determining *Qs* is therefore a matter of subtracting (Cc_{O2} + Cv_{O2}) from Ca_{O2}. However, we don't know what the *flow* is. We can only say that if *all* blood were ideally oxygenated the Ca_{O2} should be the same as Cc_{O2}, and if all blood were *completely shunted* the Ca_{O2} should be the same as Cv_{O2}.

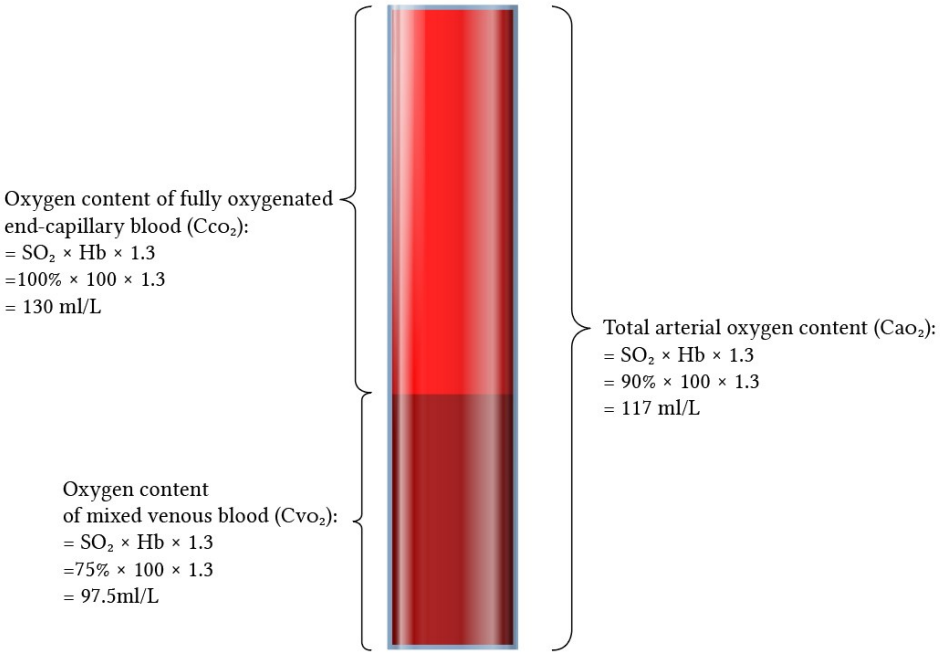
The relationship between the different fractions must therefore remain a ratio, rather than a real oxygen difference in ml. Hence the shunt fraction is usually represented as *Qs/Qt*.

The famous relationship below which determines this ratio is the so-called Berggren equation, which is usually referred to as the "shunt equation" (Sven M. Berggren, 1942). The equation compares arterial oxygen content to pulmonary end-capillary oxygen content (Cc_{O2}):



Thus, we can plug in some values, for a typical hypoxic ICU patient with an arterial saturation of 90%, and a decently anaemic Hb of 100g/L. The oxygen content of blood is calculated as $(SaO_2 \times ctHb \times 1.39) + (PaO_2 \times 0.03)$; though usually people will use 1.3 or 1.34 as these are more "realistic" BO_2 values, and *ct*Hb if the *ce*Hb is not available (because the normal fractions of dyshaemoglobins are in the 1-2% range, and can be safely ignored). The contribution of dissolved oxygen can also be safely ignored, as it is trivial. This solubility coefficient is variably expressed as 0.03ml/L/mmHg (if you use a haemoglobin concentration measured in g/L) or 0.003ml/100ml/mmHg (if you're using haemoglobin measured in g/dL as in this equation from Cornell).

Conceptually, we can represent the output of these calculations as a volume of systemic arterial blood which is separated into two compartments, one of which is fully oxygenated and one which is fully composed of unchanged venous blood:



Thus, the mixed venous blood content is 97.5ml/L, and the fully oxygenated end-capilary blood content is 130ml/L. The shunt equation can now be used to determine how much mixed venous blood one would need to add to the capillary blood to create the systemic arterial oxygen content, which is 117ml/L:

Qs/Qt = (130-117) / (130 - 97.5) = 0.4

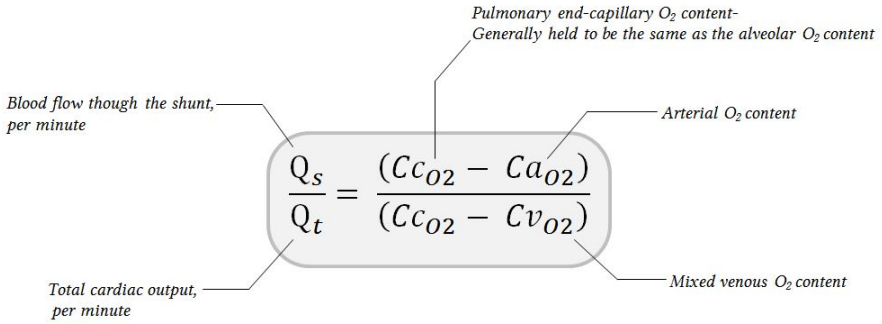
Thus, in this example, the shunt fraction is 40%.

The estimated shunt fraction, $\hat{A}Shunt_e$

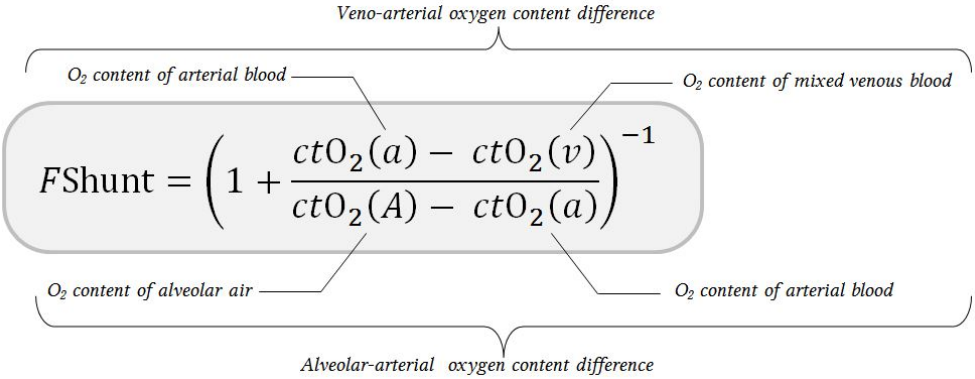
The local ABG machine may be capable of producing some estimate of shunt without any direct

measurement of mixed venous oxygen content. It is possible to make certain assumptions about the mixed venous blood, and substitute those assumptions into the shunt equation. The ABG machine can do this calculation on our behalf; the result is usually reported as FShunt_e where the little "e" indicates "estimate".

Recall the Berggren equation:



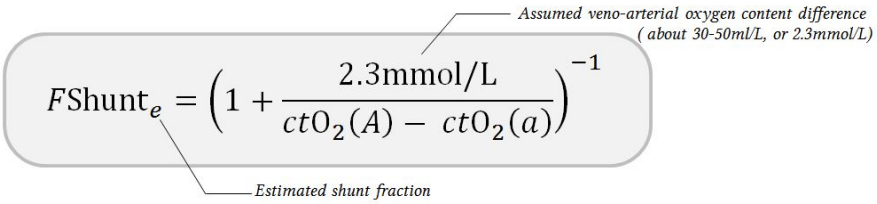
The local ABG machine rearranges the Berggren equation, and uses a slightly different nomenclature.



All of the variables used in this equation are measured in the same ABG, with the exception of the O_2 content of alveolar air, and you can get that from the alveolar gas equation.

Assumptions used in calculating the FShunt_e

In the absence of mixed venous data, the blood gas analyser uses an assumed veno-arterial oxygen content difference. The assumed difference is 2.3mmol/L, according to the reference manual for the local ABG machine.



Let us look at that assumption.

2.3 mmol/L of oxygen seems like a very small difference, but actually it is not.

An easily available online ideal gas law calculator reveals that under standard conditions 2.3mmol of oxygen equates to about 51ml/L, using whatever equation Radiometer happened to use. Now, if the arterial blood is assumed to contain 150g/L of maximally oxygenated haemoglobin, then its oxygen content is actually about 208.8ml/L. The content of venous blood therefore must be about 157.8ml/L.

Running this backwards through the DO_2 equation, one discovers that an oxygen saturation of around 77% is required, which just so happens to be the high end of average mixed venous oxygen saturation.

From this stems the greatest critique of the estimated $\dot{Q}_s/\dot{Q}_t$. The ICU patients can vary greatly in their mixed venous oxygen saturation, from the cyanide-poisoned patient with an SvO_2 of 90%, to the severe sepsis patient with an SvO_2 of 30%. In these two examples, the venoarterial oxygen differences would be 10ml/L and 145.9ml/L, respectively. Obviously, making assumptions about the mixed venous saturation of critically ill patients will lead to errors, as most of them will not have an SvO_2 of 77%

Additionally, a criticism of all shunt calculations is the fact that they use a two-compartment model. If the $\dot{Q}_s/\dot{Q}_t$ is 25%, the equation leads one to believe that in a two-compartment lung, 25% of the blood is travelling through the non-ventilated compartment. In reality, the lung is a mixture of heterogeneous units, each with a different V/Q ratio. The shunt equation is therefore a very gross estimate of oxygenation defects.

Elimination of V/Q scatter: measuring shunt with 100% FiO_2

Let us say one has encountered a sizeable difference between the arterial and alveolar oxygen content. If one were for some reason in need of estimating how much of that difference comes from "true" shunt and how much comes from V/Q scatter, one could use the MIGET technique, which is the gold standard but quite technically complex, or one could see what happens when one increases the FiO_2. V/Q scatter and "true" shunt react quite differently to increases in allveolar oxygen concentration.

As the FiO_2 increases, areas of low V/Q ratio would suddenly find themselves ventilated with a higher oxygen concentration. Even small increases in oxygen concentration here will yield substantial increases if endcapillary oxygenation, because of the steepness of the oxygen-haemoglobin dissociation curve. Thus, the difference between endcapillary blood and arterial blood would diminish, such that at 100% FiO_2 the contribution of V/Q scatter would be minimal. Sure, there would still be lung units with an extremely low V/Q ratio (less than 0.10), but these are usually few, and the amount of blood passing through them would be minimal (ergo their contribution to arterial blood would also be small). However, for lung units with "true" shunt, V/Q = 0, and no amount of extra inspired oxygen is going to improve their oxygenation. As a consequence of this, breathing a high FiO_2 essentially eliminates the contribution of V/Q scatter to the total shunt fraction, and what is left over is the "true" shunt.

Previous chapter: The concepts of venous admixture and shunt

Next chapter: Physiological consequences of increased shunt

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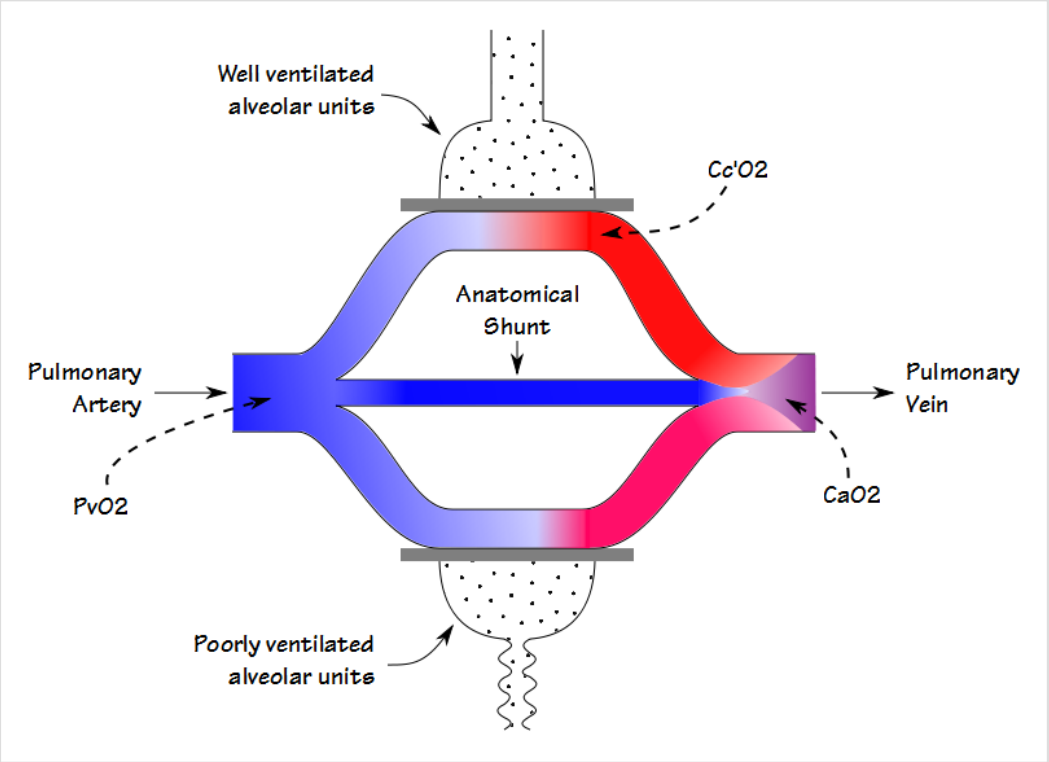
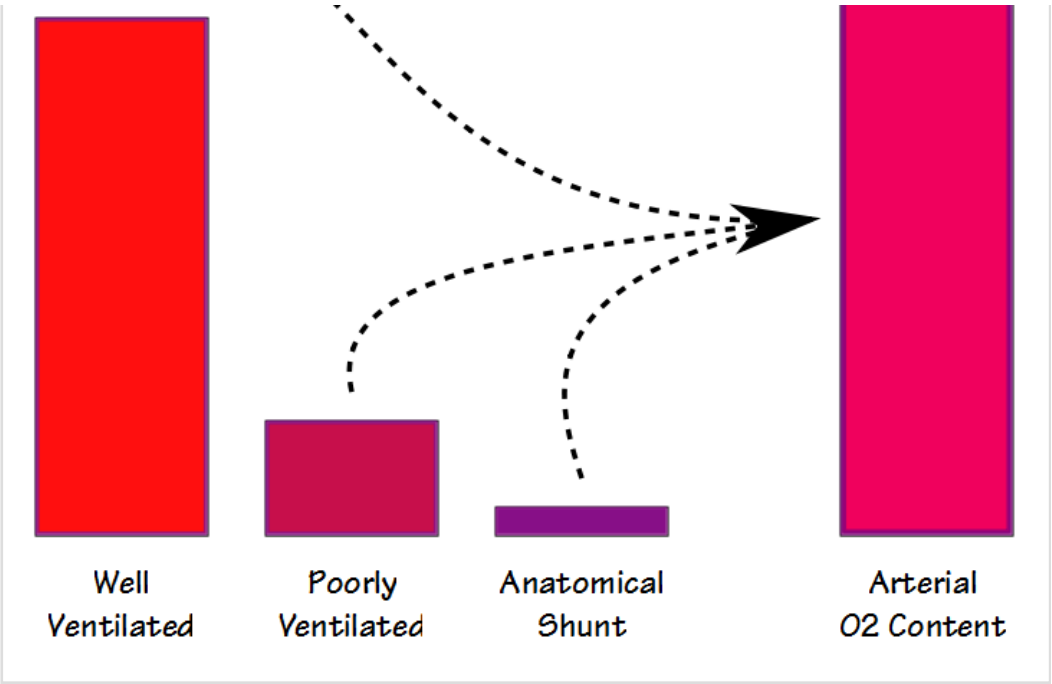
Shunt fraction

Posted on **January 27, 2016**

I was reading an article recently that made an off-hand reference to the 100% oxygen shunt fraction test. Results from the test were included in the data analysis but the equations the researchers used were not presented nor were they referenced, nor was the procedure described. This is probably because the shunt fraction test and its equations are very much old-school pulmonary physiology but even if the subject is probably covered at one time or another in physiology classes I suspect that some of the issues involved in the calculation are not as well understood as they should be.



There are some similarities between the deadspace-tidal volume ratio (Vd/Vt) and the shunt fraction but even though they are both are involved in gas exchange (and to some extent they also correlate with each other) they are measuring different things. When blood flows through the lung some blood passes through well ventilated alveoli and becomes fully saturated; some blood passes through poorly ventilated alveoli and is only partially saturated; and some bypasses the alveoli entirely. The resulting arterial oxygen content is the summed average of all of these compartments.



There are two different ways that shunt fraction can be measured and calculated; physiological and anatomical. The physiological shunt equation can be performed at any FiO2 (but usually around the FiO2 of room air) and requires that arterial and mixed venous blood samples be taken more or less simultaneously and then analyzed for PO2 and SaO2. The basic formula is:

$$\frac{\dot{Q}s}{\dot{Q}t} = \frac{(Cc'O2 - CaO2)}{(Cc'O2 - C\bar{v}O2)}$$

Where:

Qs = blood flow through shunt

Qt = total blood flow

Cc'O2 = pulmonary capillary O2 content

CaO2 = arterial O2 content

CvO2 = mixed-venous O2 content

Oxygen content is the milliliters of oxygen per liter of blood and is calculated from:

$$O2\,Content = (Hb \times 1.34 \times (\frac{SO2}{100})) + (PO2 \times 0.0031)$$

Where:

Hb = hemoglobin (grams/decaliter)

SO2 = oxygen saturation (%)

PO2 = oxygen partial pressure

The pulmonary capillary O2 content cannot be measured directly (and strictly speaking it is more of a conceptual value than a real one) and is usually estimated from the alveolar air equation (although "ideal" pulmonary capillary blood has a PO2 gradient of about 1 mm Hg from alveolar air this is insignificant enough that it is usually ignored).

$$PAO2 = ((Pb - 47) \times FiO2) - \frac{PaCO2}{RER}$$

Where:

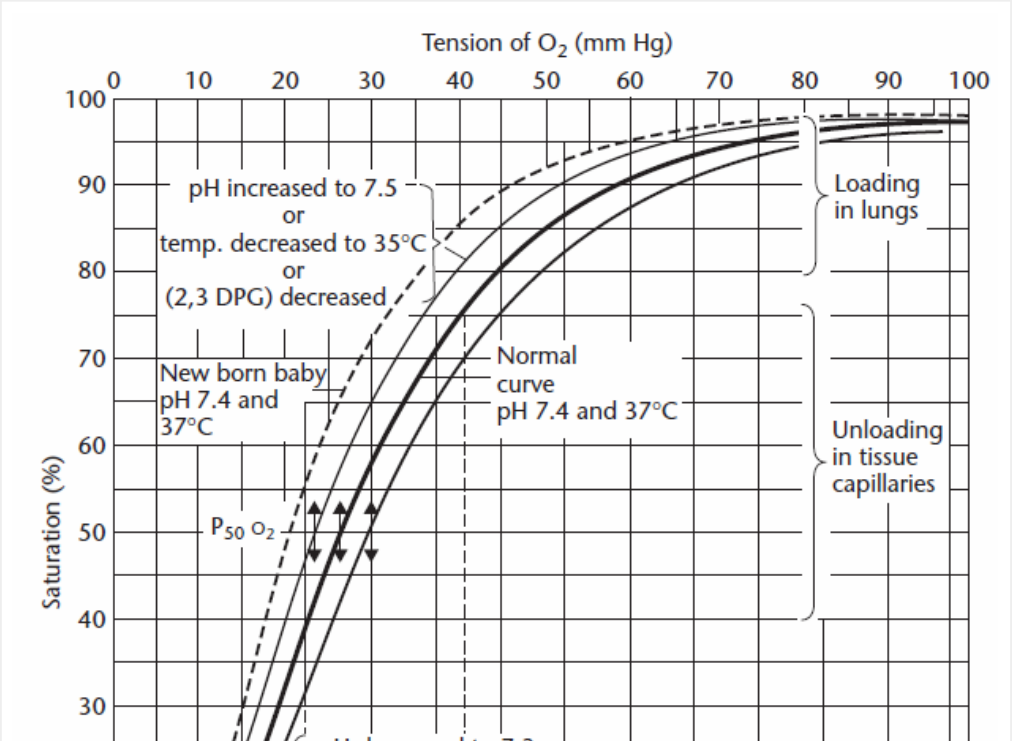
Pb = barometric pressure in mm Hg

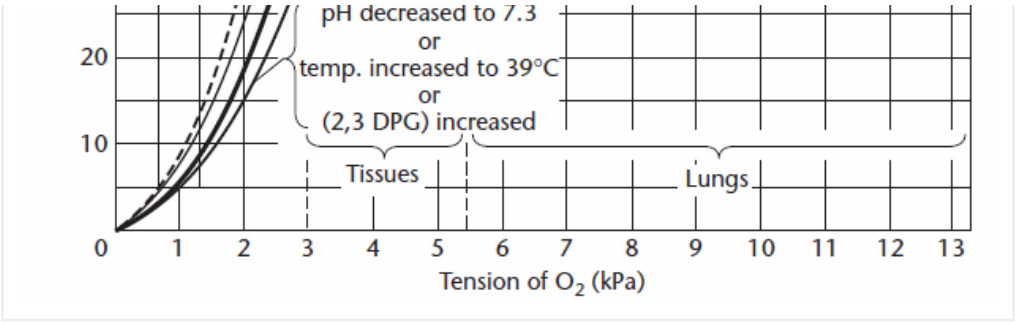
FiO2 = fractional concentration of inspired oxygen

PaCO2 = arterial partial pressure of CO2

RER = respiratory exchange ratio

The oxygen content of the pulmonary capillaries is determined by first estimating the oxygen saturation from the PAO2 and this can be done either visually from the oxygen dissociation curve:





— From Cotes et al, pg. 260.

or from Severinghaus's formula:

$$SO2=((((PO2^3+(150xPO2))^{-1}x23400)+1)^{-1})*100$$

and then calculating Cc'O2 accordingly.

> Note: Interestingly neither the oxygen dissociation curve nor Severinghaus's formula take carboxyhemoglobin (or methemoglobin) into account. For that matter, this issue has not been included in any of the textbook discussions of shunt fraction I've read. COHb skews the relationship between PO2 and SO2 (downwards if you're working from PO2 to SO2, upwards if you're working from SO2 to PO2). Normal COHb levels in non-smokers are 1-2 and this amount of COHb is unlikely to make a significant difference in the shunt fraction calculations. In the absence of any firm guidelines however, when higher levels of COHb are present they should probably be used to adjust Cc'O2 accordingly.

Taking normal values and working backwards, PAO2 is:

$$PAO2=((760-47)x0.21)-(\frac{40}{0.8})=99.73$$

The pulmonary capillary oxygen saturation is therefore:

$$Sc'O2=(((((99.73^3+(150x99.73))^{-1}x23400)+1)^{-1})x100)=97.7$$

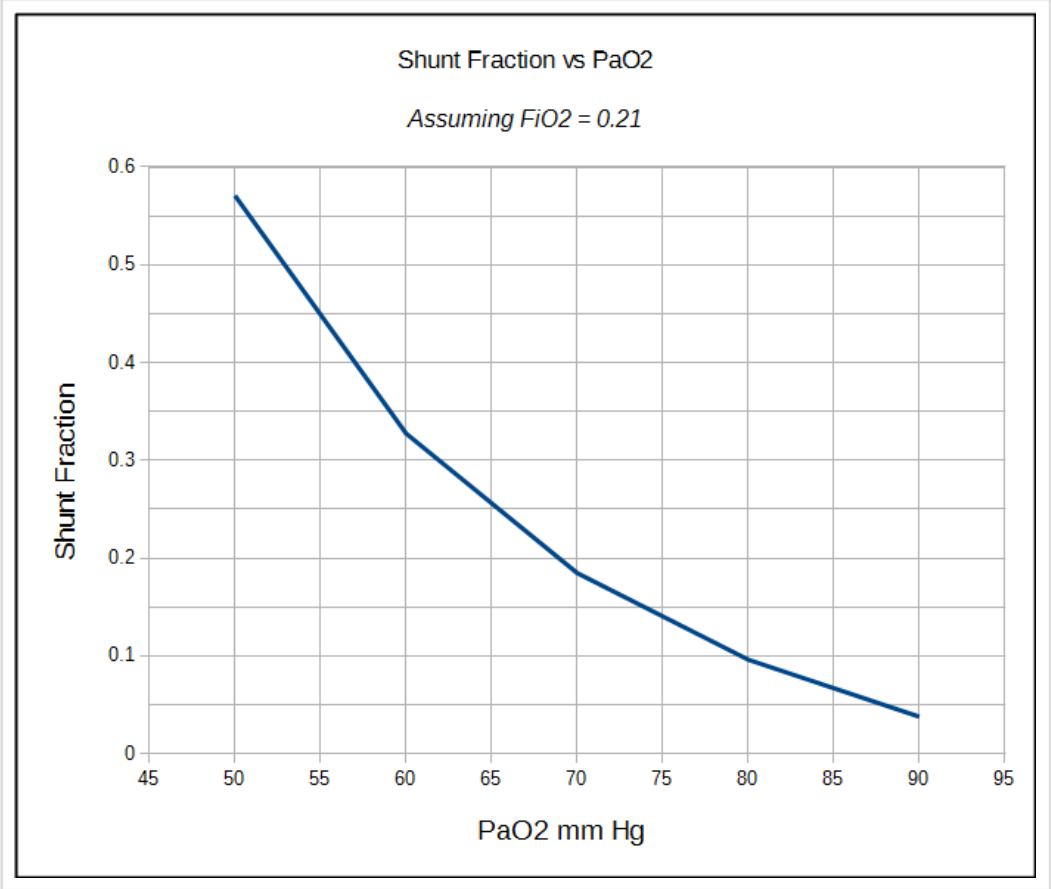
And the pulmonary capillary oxygen content is:

$$Cc'O2=(14.6x1.34x(\frac{97.7}{100}))+(99.73x0.0031)=19.42$$

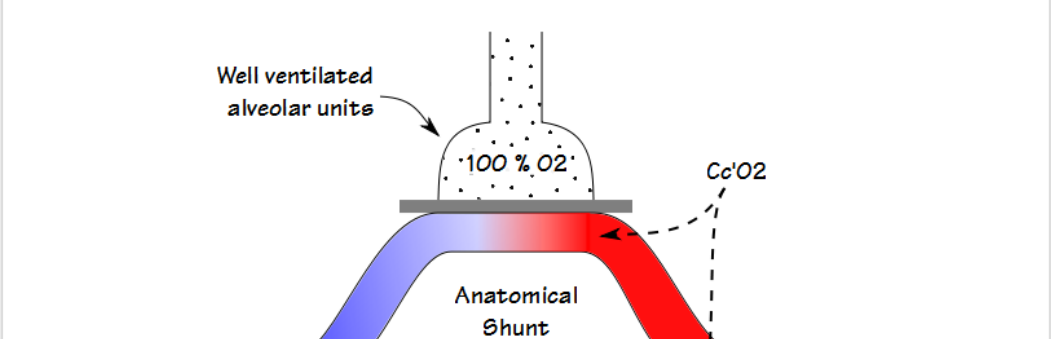
Mixed venous blood nominally has a PO2 of 40 and an oxygen saturation of 75%, so:

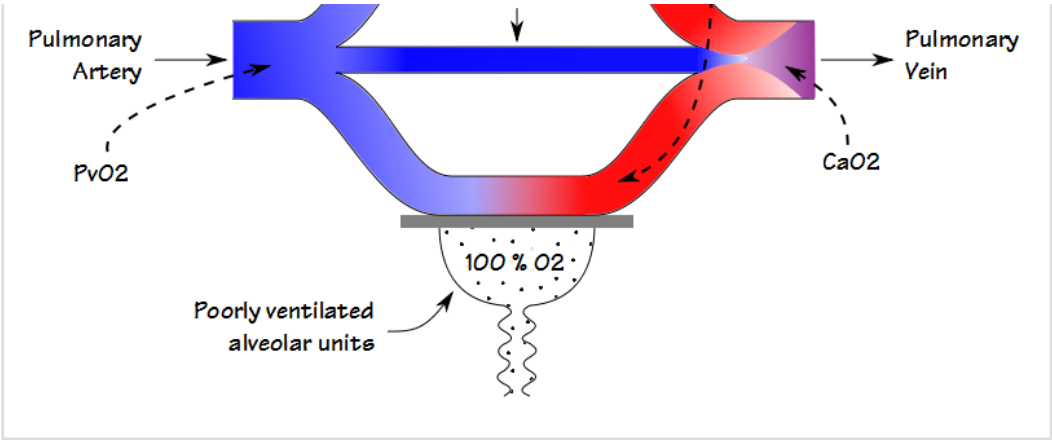
$$C\bar{v}O2=(14.6\,x\,1.34\,x\,(\frac{75}{100}))+(40\,x\,0.0031)=14.8$$

CaO2 will then be calculated from an individual's actual PaO2 and SaO2. Depending on the specific results the shunt fraction will be:



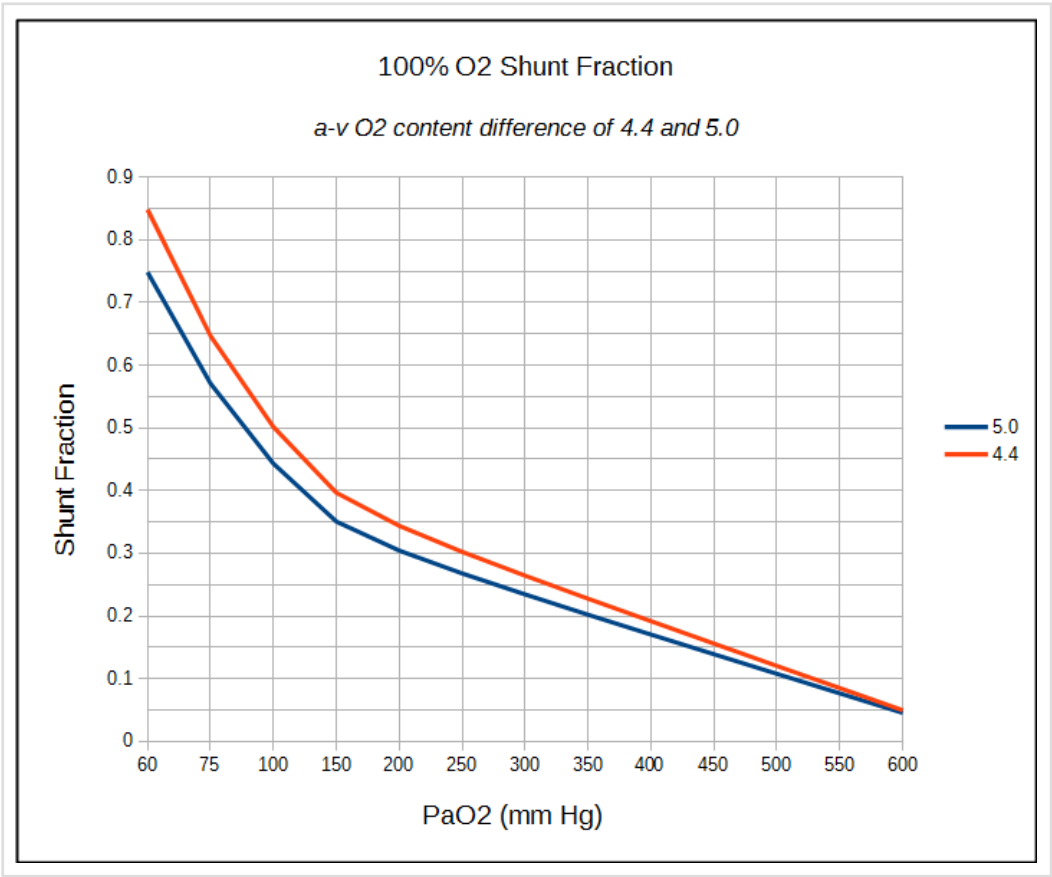
The physiological shunt fraction can only be calculated when both the arterial and the mixed-venous PO2 and SO2 are known. For this reason it is most often performed in a cardiac cath lab, operating room or intensive care unit where indwelling arterial and central venous lines are relatively common. The physiological shunt calculation cannot differentiate between the shunting caused by poorly ventilated alveolar units and that from an anatomical shunt, however. The anatomical shunt fraction can be calculated by a separate procedure however, and this is where the 100% O2 test comes into play.





By having a patient breath 100% O2 until the nitrogen has been washed out of their lung (nominally 20 minutes), the oxygen concentration in even poorly ventilated units will approach 100%. This means that the partial pressure and saturation of blood leaving both the poorly and the well ventilated alveolar units will be the same. For this reason, any decrease in the arterial oxygen content will be due solely to an anatomical shunt.

If a patient has an indwelling central venous catheter, the calculation of anatomical shunt can proceed the same way as already detailed. If only an arterial sample can be obtained (which is usually the case in a PFT Lab) an arterial-venous O2 content difference of between 4.4 and 5.0 can be assumed and the shunt fraction calculated accordingly.



The limitations of the shunt fraction calculations have to do in part with some of the assumptions about normal values and in part with the accuracy of blood gas

measurements. The alveolar air equation, for example, assumes that the respiratory exchange ration (RER) is 0.8 but the only way to be sure is by actually measuring VO2 and VCO2. Strictly speaking, an RER that is different than 0.8 will probably not make a significant difference in calculated PAO2, Sc'O2 and Cc'O2, but it is still an assumption. Using an a-v O2 content difference of 4.4 to 5.0 on the other hand, is a much larger assumption. It is justified to some extent by the fact that the 100% O2 test is usually made at rest and these are reasonable values for an individual at rest but again, it is an assumption.

Far more concerning are the limitations in accurately measuring PaO2 and SaO2, particularly at higher FiO2's. Two different studies have shown that the type of syringe used to obtain an ABG (glass versus plastic) and how it was stored (on ice or at room temperature) made a significant difference in the calculated shunt fraction even when the ABG samples were analyzed quickly. When there was a longer wait before analysis the error in PaO2 could cause the calculated shunt fraction to be twice as large as it really was. The reason this happens is partly due to diffusion through the plastic syringes and partily to continued metabolism within a blood sample when kept at room temperature. The least amount of change was seen when glass syringes kept on ice.

Interestingly, a similar study with ABG samples taken at normal FiO2 (PO2 ≈ 100) showed the opposite effect. The measured PO2 tended to rise, again more in plastic syringes than in glass, and again this likely due to diffusion. Interestingly, PO2 fell in glass syringes kept on ice and the authors, Knowles et al, point out that the solubility of O2 rises as temperature falls and that with more O2 in solution PO2 may decrease.

Finally, blood gas analyzers are usually calibrated using gas concentrations in the normal physiological range. Any arterial blood sample with PO2 above 200 mm Hg is well outside this range and I am concerned about what kind of error bar there is for PO2's that are even higher. Pretto et al used blood tonometered with 95% O2 and 5% CO2 but interestingly they did not report the measured PO2 but only the change in PO2 over time. Smeenk et al obtained blood samples from individuals undergoing the 100% oxygen test as a pre-op assessment for coronary bypass surgery and the average PO2 of their gold standard samples (glass syringe, iced, 5 minute delay) was 590 mm Hg. This is an A-a gradient of around 80 mm Hg and may well be appropriate, but it also means that the average anatomical shunt fraction was 10% and Cotes et al indicates that the normal anatomical shunt for individuals in the same age range is around 4%.

The shunt fraction test is not commonly performed in pulmonary function labs. True anatomic shunts are relatively rare and the most appropriate patient for the 100% O2 shunt fraction test would be one with a reduced SaO2 at rest that does not significantly improve with supplemental O2.

The physiological shunt fraction could be considered the reverse side of Vd/Vt. Perfusion inhomogeneities exist just as much as ventilation inhomogeneities but this may be overlooked because pulmonary function testing is oriented far more around the ventilation

side of respiration than the perfusion side. Ventilation and perfusion inhomogeneities are core features of many pulmonary diseases. For this reason the shunt fraction and the differences between its physiological and anatomical components need to be part of the education of all pulmonary technologists. Like Vd/Vt however, there are also limitations to the accuracy of the shunt fraction calculation both from assumptions that may or may not be reasonable, and from the measurement accuracy of PO2 and SO2.

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8 THOUGHTS ON "SHUNT FRACTION"

MODELS OF VENOUS ADMIXTURE

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Medical students, residents, and allied health professionals often have difficulty quantitating ventilation-perfusion mismatch in ill patients. This manuscript quantitates ventilation-perfusion mismatch using the underlying physiological concepts and equations that describe mismatch. In addition, clinical problems with diagrams and worked-out solutions are supplied to help students master these equations as well as their practical limitations.

ADV PHYSIOL EDUC 25: 159–166, 2001.

Key words: ventilation

When patients have hypoxemia due to ventilation-perfusion mismatch, clinicians need quantitative methods to measure the severity of illness as a function of time. Medical students, residents, and allied health professionals usually have some familiarity with the virtual shunt equation, shunt nomogram, alveolar-arterial gradient, and arterial/alveolar ratio. These techniques are all commonly used to assess respiratory failure. Nonetheless, students often have difficulty quantitating these measures and do not understand when these methods fail. Standard textbooks may provide the theoretical background for the material above, but they rarely provide clinical examples to solidify these concepts (1, 3, 4). This manuscript presents the derivation of equations describing ventilation-perfusion mismatch. In addition, clinical examples with worked solutions are provided to help students learn to manipulate the formulas necessary to assess respiratory failure. The practical limitations of these formulas are also discussed.

MODEL

The formal method of modeling pulmonary dysfunction in a hypoxemic patient is the patient's virtual shunt through his lungs. Figures 1 and 2 and *Equations 1-3* explain how this is done.

Figure 1*A* shows a healthy alveolus and pulmonary capillary. In this part of the lung, ventilation and perfusion are matched, and there is no venous admixture. Figure 1*B* shows a collapsed alveolus and a healthy capillary. Here, all of the mixed venous blood is shunted past the functional part of the lung. This is called venous admixture. Figure 1*C* shows a healthy alveolus and collapsed pulmonary capillary. This is the definition of dead space. It is included here to avoid confusion, although it is not part of the model.

Figure 2 shows how venous admixture to the systemic arterial blood can occur.

Conservation of blood flow dictates that the pulmonary capillary blood flow and shunted blood flow must equal the total cardiac output through the lungs (*Eq. 1*)

$$\dot{Q}_T = \dot{Q}_C + \dot{Q}_S \qquad (1)$$

where $\dot{Q}_T$, $\dot{Q}_C$, and $\dot{Q}_S$ are cardiac output, pulmonary capillary blood flow, and shunted pulmonary capillary blood flow, respectively.

Conservation of mass dictates that the transport of oxygen through the lung is also conserved (*Eq. 2*)

$$\dot{Q}_T Ca_{O_2} = \dot{Q}_c C_c O_2 + \dot{Q}_s C_{\bar{v}} O_2 \qquad (2)$$

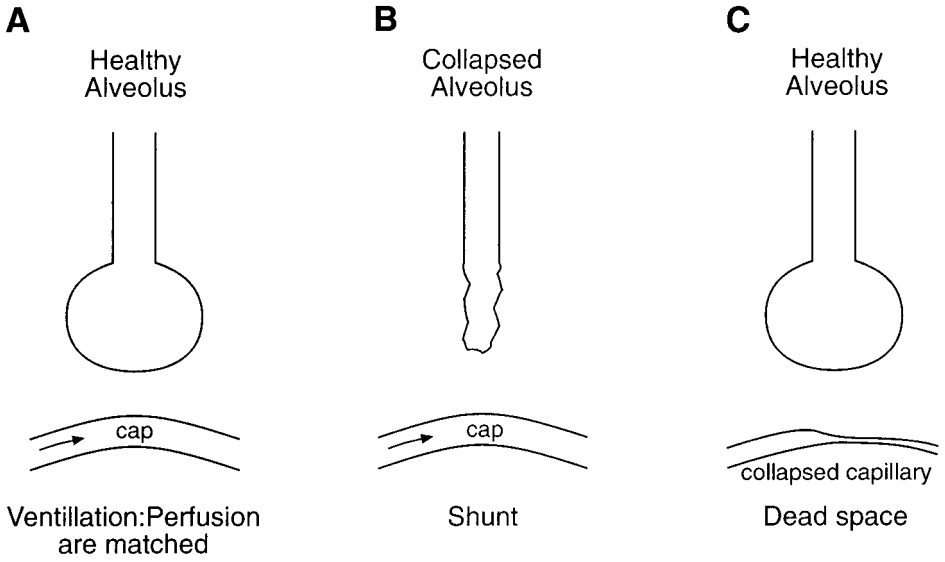

FIG. 1.
Oxygen and blood flow in the lung. Cap, pulmonary capillary.

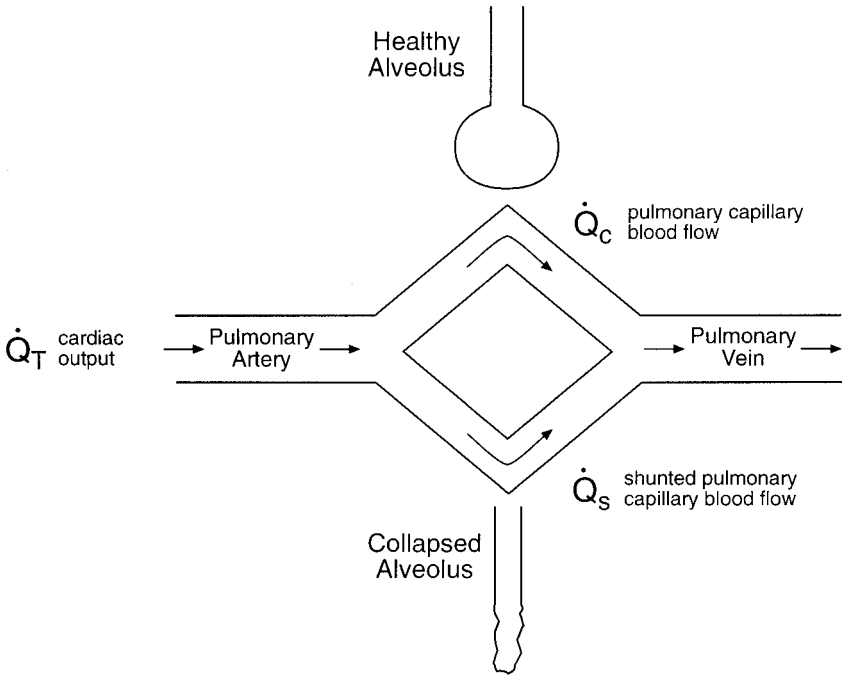

FIG. 2.
Virtual shunt model.

where Ca_{O_2} is the O_2 content in the systemic arterial blood (pulmonary vein), C_cO_2 is the O_2 content in the pulmonary capillary, and $C_{\bar{v}}O_2$ is the O_2 content in the shunted or mixed venous blood. Substituting *Eq. 1* into *Eq. 2* and rearranging yields the familiar virtual shunt equation.

$$\frac{\dot{Q}_S}{\dot{Q}_T} = \frac{C_cO_2 - C_aO_2}{C_cO_2 - C_{\bar{v}}O_2} \quad (3)$$

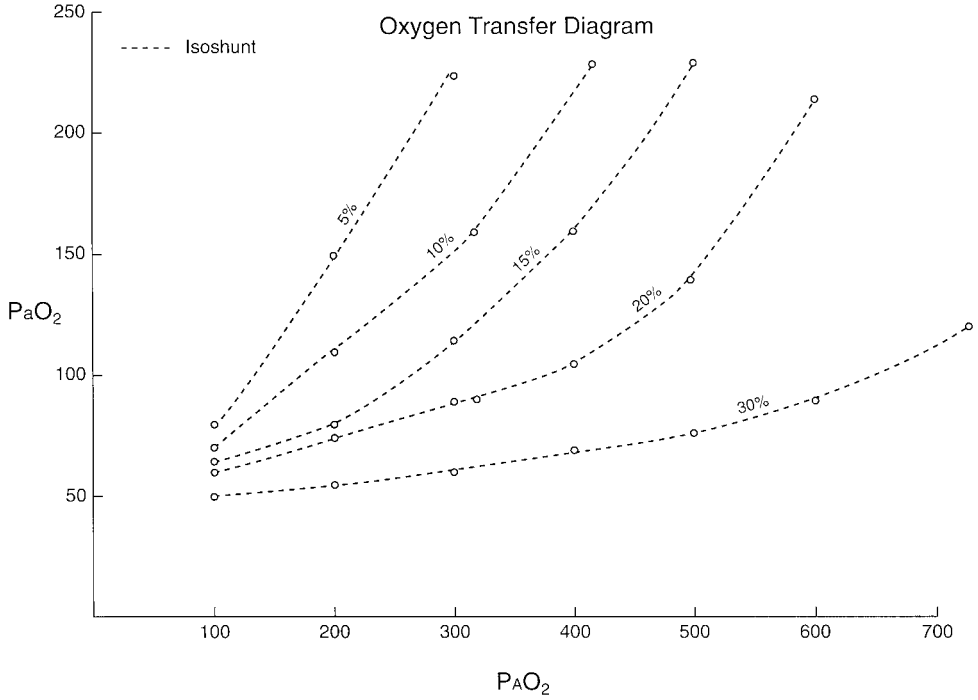

FIG. 3.
Pa_{O_2}, arterial oxygen pressure in Torr; PA_{O_2}, alveolar oxygen pressure in Torr; isoshunt, isoshunt lines in %.

To calculate shunt (*Eq. 3*), one must measure the partial pressure of O_2 in the mixed venous and systemic arterial blood. In addition, one must estimate the partial pressure of O_2 in the pulmonary capillary by calculating the partial pressure of oxygen delivered to the alveolus (PA_{O_2}; *Eq. 3A*)

$$PA_{O_2} = (PB - PH_2O) \cdot FI_{O_2} - \frac{Pa_{CO_2}}{R} \qquad (3A)$$

where PB is atmospheric pressure, and PH_2O is the pressure of water vapor in the lung. Pa_{CO_2} is the partial pressure of CO_2 in the systemic arterial blood, and R is the respiratory quotient, which is assumed to be 1 in this model. The use of *Eq. 3* is impractical in routine clinical care because the mixed venous blood is rarely sampled.

To get around this, *Eq. 3* was recast in nomogram form. Nunn assumed that the C_vO_2 had a constant value and that the hemoglobin and Pa_{CO_2} were within common ranges (2). Using these assumptions, he created the isoshunt lines shown in Fig. 3.

This nomogram allows one to estimate the patient's virtual shunt and follow his pulmonary dysfunction as his FI_{O_2} and ventillatory therapy are adjusted by measuring his Pa_{O_2}. Despite their simplicity, the isoshunt lines did not achieve popularity because pulmonary critical care involves treating more than a single parameter and because it was impractical to carry the nomogram around.

A number of years later, interest was renewed in *Eq. 3* with the advent of pulse oximeters and oxymetric pulmonary artery catheters. Manipulation of *Eq. 3* shows why. The oxygen content of blood is given by *Eq. 4*

$$Co_2 = 1.34 \cdot Hgb \cdot saturation + 0.003\ Po_2 \qquad (4)$$

where Po_2 is the partial pressure of oxygen in the blood. The term ($0.003\ Po_2$) represents dissolved O_2 in the plasma and will be ignored here. If we assume that the amount of dissolved oxygen is negligible, then we can substitute *Eq. 4* into *Eq. 3* to yield.

$$\frac{\dot{Q}_S}{\dot{Q}_T} = \frac{1.34 \cdot \text{Hgb (end-capillary sat} - \text{arterial sat)}}{1.34 \cdot \text{Hgb (end-capillary sat} - \text{mixed venous sat)}} \quad (5)$$

With oximetry, both the arterial saturation and mixed venous saturation could be measured continuously to give a good estimate of shunt. Unfortunately, *Eq. 5* still involved a cumbersome calculation and the cost and morbidity of an oxymetric pulmonary artery catheter. For these reasons, *Eq. 5* never achieved common usage either. *Eq. 5*, however, can be simplified to make it useful for clinicians.

In patients with stable cardiac outputs who are administered a high concentration of O_2 (FI_{O_2}), the difference between end-capillary saturation and mixed venous saturation is ~0.25. Thus *Eq. 5* becomes

$$\frac{\dot{Q}_S}{\dot{Q}_T} = \frac{\text{end-capillary sat} - \text{arterial sat}}{0.25} \quad (6)$$

Equation 6 is made usable by relating O_2 saturation to Pa_{O_2}. Reference to the oxyhemoglobin dissociation curve shows how this is done (Fig. 4).

At a Pa_{O_2} >100 Torr, the graph is nearly a flat line. Thus O_2 saturation and Pa_{O_2} are related by the formula for a straight line (*Eq. 7*)

$$O_2 \text{ saturation} = m \cdot Pa_{O_2} + b \quad (7)$$

where m is the slope of the line, and b is the intercept on the saturation axis. Substituting *Eq. 7* into *Eq. 6* yields

$$\frac{\dot{Q}_S}{\dot{Q}_T} = \frac{m(P_{A_{O_2}} - Pa_{O_2})}{0.25} \quad (8)$$

where we have assumed that the $P_{A_{O_2}}$ is the same as P_{O_2} in the pulmonary capillary. Using the slope (m = 1.4×10^{-4} saturation/Torr) on the flattest part of Fig. 4 allows *Eq. 8* to be recast as

$$\frac{\dot{Q}_S}{\dot{Q}_T} = \frac{1.4 \cdot 10^{-4}(P_{A_{O_2}} - Pa_{O_2})}{0.25} \quad (9)$$

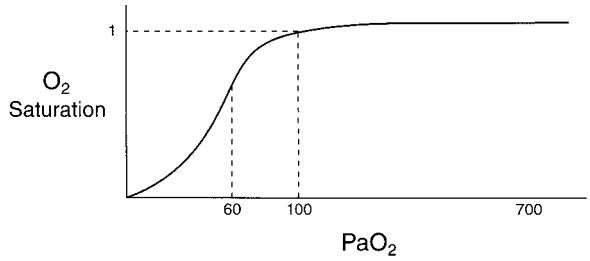

FIG. 4.
Oxyhemoglobin dissociation curve.

If we desire to use shunt fraction rather than shunt, then *Eq. 9* becomes

$$\text{Shunt fraction} = \frac{\dot{Q}_S}{\dot{Q}_T} \cdot 100 = \frac{PA_{O_2} - Pa_{O_2}}{18} \quad (10)$$

Equation 10 shows that the commonly used alveolar-arterial gradient divided by a constant yields an easy estimate of shunt when the patient's cardiac output and hemoglobin are stable and when the $P_{A_{O_2}}$ and Pa_{O_2} lie along the flat part of the oxyhemoglobin dissociation curve. Using a similar approach, the shunt fraction can be estimated when the $P_{A_{O_2}}$ < 100 Torr and the Pa_{O_2} lies between 50 and 100 Torr. In this case, *Eq. 10* becomes

$$\text{Shunt fraction} = \frac{PA_{O_2} - Pa_{O_2}}{2} \quad (10A)$$

Equations 10 and *10A* are only estimates of shunt fraction and are insensitive to larger gradients in the denominator of *Eq. 5*. Larger gradients in the denominator of *Eq. 5* may occur in some patients with decreased cardiac output or significant shunt. Nonetheless, as long as cardiac output is stable, *Eqs. 10* and *10A* will track changes in shunt fraction as the patient's pulmonary function improves or declines. If one wishes to account for larger gradients in *Eq. 5*, then the slope in *Eq. 7* must be adjusted to fit *Eqs. 10* and *10A* to clinical data.

The following clinical cases below are illustrative:

An 18-yr-old male with a history of asthma presents to the emergency department in respiratory distress. A nasal cannula supplies oxygen at 28%. His blood gas shows the following values: 7.32/31/74/23, O_2 saturation 95%. In this case, his alveolar O_2 ($P_{A_{O_2}}$) is given

TABLE 1

	$F_{I_{O_2}}$	$P_{A_{O_2}}$	Pa_{O_2}	A-a Gradient	Virtual Shunt	Estimated Shunt
Patient 1 (asthma)	0.28	168	74	94	15%–20%	6%
Patient 2 (surgery)	0.5	322	115	207	10%–15%	12%

by *Eq. 3A*. Thus $P_{A_{O_2}} = (760 - 47) \cdot 0.28 - 31 = 168$ Torr. The patients' alveolar-arterial gradient is given by $P_{A_{O_2}} - [Pa_{O_2}] = 168 - 74 = 94$ Torr. Reference to Fig. 3 shows the patient has a virtual shunt of 15–20%. His estimated shunt using the $P_{A_{O_2}}$-Pa_{O_2} gradient (*Eq. 10*) is ~6%.

Now consider a very different patient. A 74-yr-old male with a 50-pack/yr history of cigarettes undergoes a Whipple procedure. At the end of the surgery, the patient is intubated and ventilated with an $F_{I_{O_2}}$ of 50%. His blood gas shows the following values: 7.32/34/115/27, O_2 saturation 98%. Using the same analysis as above, we find that this patient has $P_{A_{O_2}}$-Pa_{O_2} gradient of 207 Torr, a virtual shunt of 10–15%, and estimated shunt using the $P_{A_{O_2}}$-Pa_{O_2} gradient (*Eq. 10*) of 12%. This is summarized in Table 1.

Notice that *patient 1* (asthma) is sicker (virtual shunt = 15–20%) even though his $P_{A_{O_2}}$-Pa_{O_2} gradient is smaller than *patient 2*. This shows the value of the virtual shunt method, i.e., virtual shunt remains constant regardless of the $F_{I_{O_2}}$ administered. The $P_{A_{O_2}}$-Pa_{O_2} gradient, however, widens as the $F_{I_{O_2}}$ is increased, even in the absence of increased pulmonary dysfunction. The estimated shunt of *patient 1* (*Eq. 10*) is 6%, which compares poorly with his virtual shunt (*Eq. 3*). This is because his Pa_{O_2} lies along the steep part of the oxyhemoglobin dissociation curve. In this situation, *Eq. 10* is not a good measure of venous admixture. Conversely, the estimated shunt of *patient 2* (12%) is similar to his virtual shunt. This is because both his $P_{A_{O_2}}$ and Pa_{O_2} lie along the flat part of the oxyhemoglobin dissociation curve (Fig. 4).

Figure 5 shows the relationship between virtual shunt and estimated shunt. Notice that when the Pa_{O_2} is greater than 100 Torr, the virtual shunt lines and estimated shunt lines have slopes and absolute values that are similar. Below 100 Torr, the estimated shunt ($P_{A_{O_2}} - Pa_{O_2}$) reproduces the virtual shunt poorly.

Now let's look at the problem in a different way. Suppose we consider the right heart and lungs to be an engine whose job is to convert mixed venous blood into oxygenated blood. If the engine is ideal (Fig. 6*A*), then the blood will be fully saturated with oxygen when it leaves the engine.

If the engine is less than ideal (Fig. 6*B*), then the blood will be only partially oxygenated when it leaves the heart-lung engine. We can calculate an efficiency for this real engine by comparing it with the ideal engine (*Eq. 11*).

$$\text{Efficiency} = \frac{O_2 \text{ content of partially oxygenated blood}}{O_2 \text{ content of fully oxygenated blood}} \quad (11)$$

Substituting *Eq. 4* into *Eq. 11* yields

$$\text{Efficiency} = \frac{1.34 \cdot \text{Hgb} \cdot \text{arterial sat}}{1.34 \cdot \text{Hgb} \cdot \text{end-capillary sat}} \quad (12)$$

Now, recall that on the flat part of the oxyhemoglobin dissociation curve (Fig. 4), the O_2 saturation = $m \times P_{O_2} + b$ (*Eq. 7*). Substituting *Eq. 7* into *Eq. 11* yields

$$\text{Efficiency} = \frac{Pa_{O_2}}{PA_{O_2}} \quad (13)$$

This is commonly called the a/A ratio. Our derivation shows that the a/A ratio is only a good measure of pulmonary dysfunction (venous admixture) when the Pa_{O_2} and $P_{A_{O_2}}$ both lie along the flat part of the oxyhemoglobin dissociation curve.

Figure 7 shows the graphical relationship of $Pa_{O_2}/P_{A_{O_2}}$ to the isoshunt lines. When the Pa_{O_2}-$P_{A_{O_2}}$ ratio is near 1, isoshunt and $Pa_{O_2}/P_{A_{O_2}}$ correlate well. As the ratio

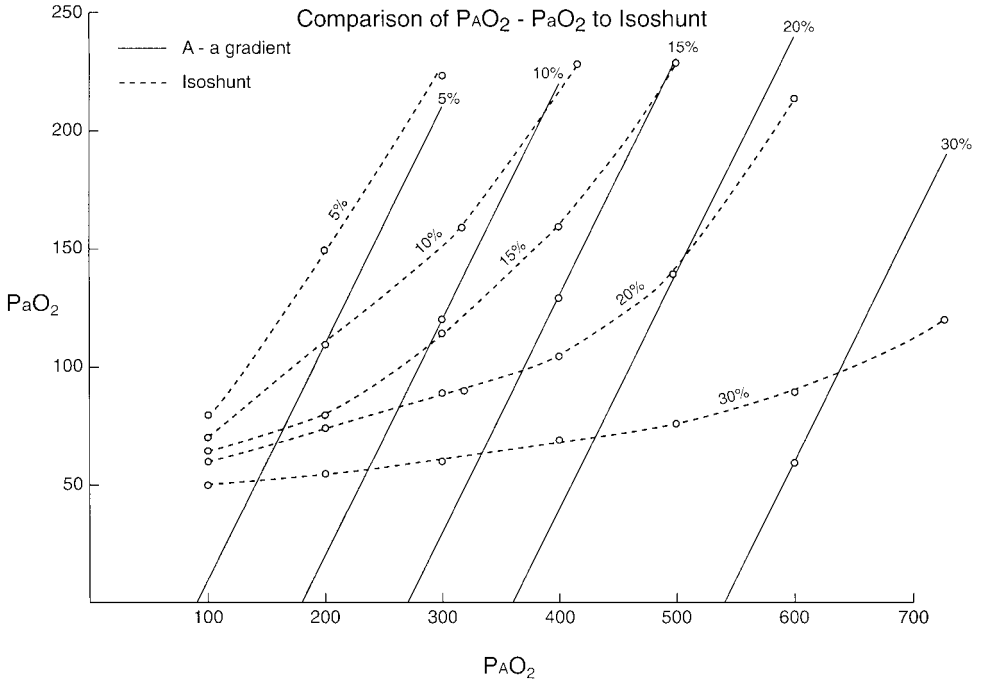

FIG. 5.
A-a gradient, estimated shunt fraction; isoshunt, isoshunt lines in percent.

falls, the correlation becomes poorer because the Pa_{O_2} no longer lies along the flat part of the oxyhemoglobin dissociation curve.

As an example, consider *patients 1* and *2* again. The efficiency of *patient 1* (Pa_{O_2}/PA_{O_2}) is 0.44, whereas that of *patient 2* is 0.36. By comparing a/A ratios, *patient 1* appears healthier than *patient 2*, although his virtual shunt is greater. This is because his Pa_{O_2} lies along the steep part of the oxyhemoglobin dissociation curve, and the a/A ratio is less accurate in this case. This is summarized in Table 2.

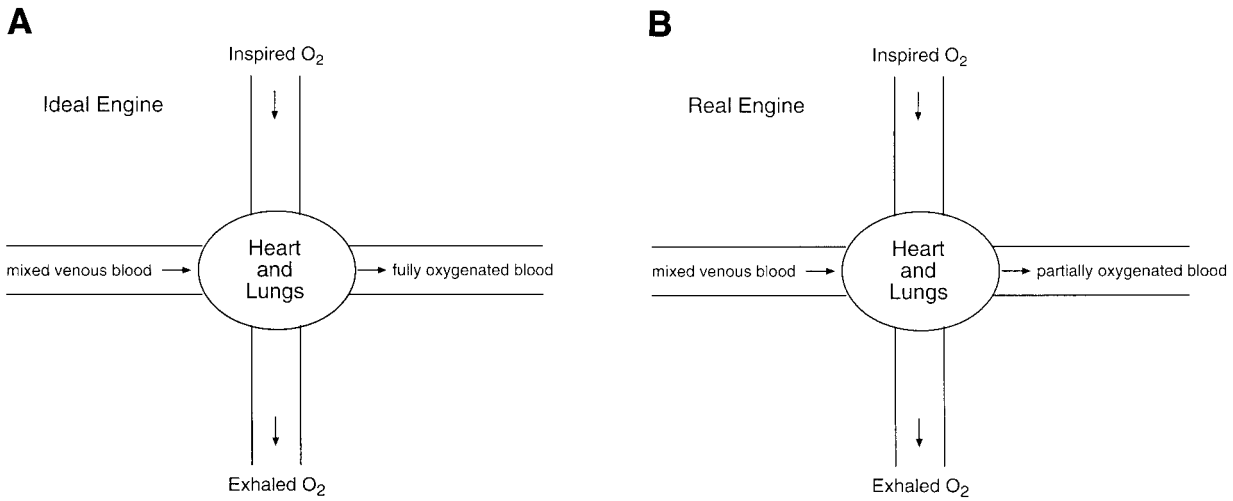

FIG. 6.
Heart-lung engine model.

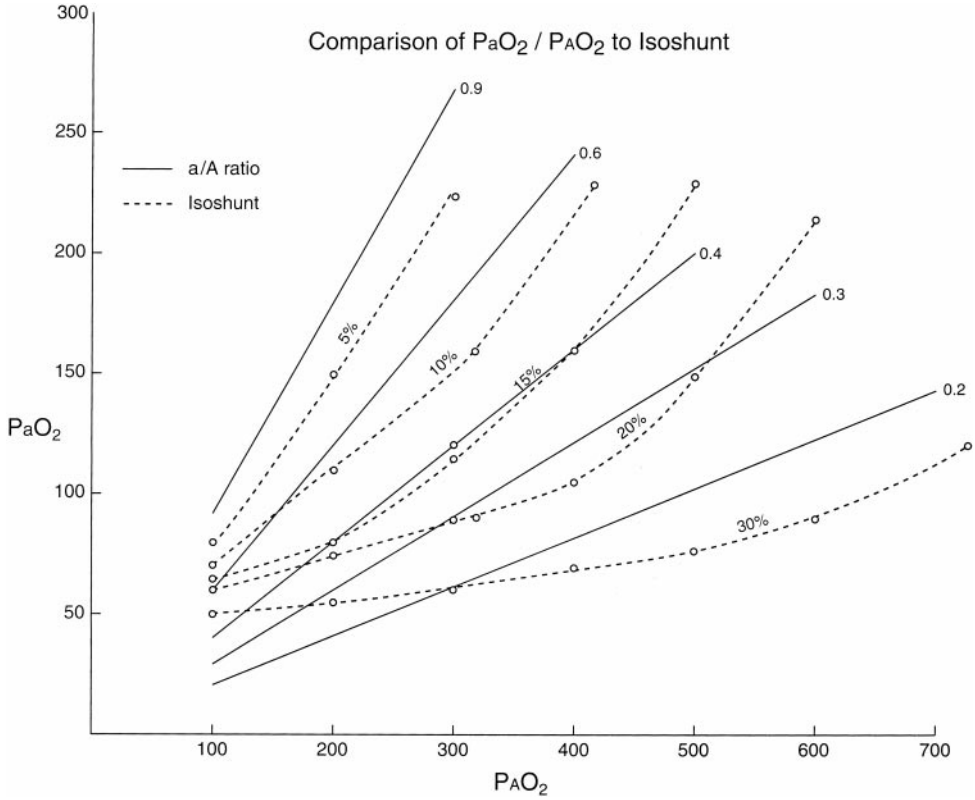

FIG. 7.
a-A ratio, arterial-to-alveolar oxygen pressure ratio.

SUMMARY

Virtual shunt remains the best way of quantitating venous admixture in patients with pulmonary pathology. It is a cumbersome technique that requires pulmonary artery catheterization and data from the oxyhemoglobin dissociation curve. The isoshunt lines approximate virtual shunt when the mixed venous saturation, hemoglobin, and Pa_{CO_2} are within common ranges and are stable. Most clinicians do not carry this nomogram with them. The A-a gradient and a/A ratio both give a good estimate of venous admixture when the conditions for the isoshunt lines are met and when both Pa_{O_2} and $P_{A_{O_2}}$ lie along the flat part of the oxyhemoglobin dissociation curve. Pa_{O_2} is easily measured, and $P_{A_{O_2}}$ is easily calculated. For these reasons, they have become the standards in clinical care.

PRACTICE PROBLEM

An 18-mo-old child presents to the emergency department with respiratory distress and a chest X-ray consistent with pneumonia. A facemask supplies oxygen at 35%. His arterial blood gas shows the following values: 7.31/29/67/23, O_2 saturation 93%.

TABLE 2

	FI_{O_2}	$P_{A_{O_2}}$	Pa_{O_2}	A-a Gradient	Virtual Shunt	Estimated Shunt	a/A
Patient 1 (asthma)	0.28	168	74	94	15%–20%	6%	0.44
Patient 2 (surgery)	0.5	322	115	207	10%–15%	12%	0.36

1) Use *Eq. 3A* to calculate the patient's alveolar O_2 (PAO_2). Answer, 220 Torr.

2) Calculate the patient's alveolar arterial gradient, i.e., $P_{AO_2} - Pa_{O_2}$. Answer, 153 Torr.

3) Use Fig. 3 to estimate the patient's virtual shunt. Answer, 25%.

4) Now use *Eq. 10* to estimate the patient's shunt. Answer, 9%. Can you explain why *Eq. 10* gives such a poor estimate of virtual shunt? Hint, see the discussion of *patient 1*.

5) Now use *Eq. 19* to calculate the patient's arterial-alveolar ratio. Answer, 0.3. Does *Eq. 13* overestimate or underestimate the patient's illness and why? Hint, see the discussion of *patient 1*.

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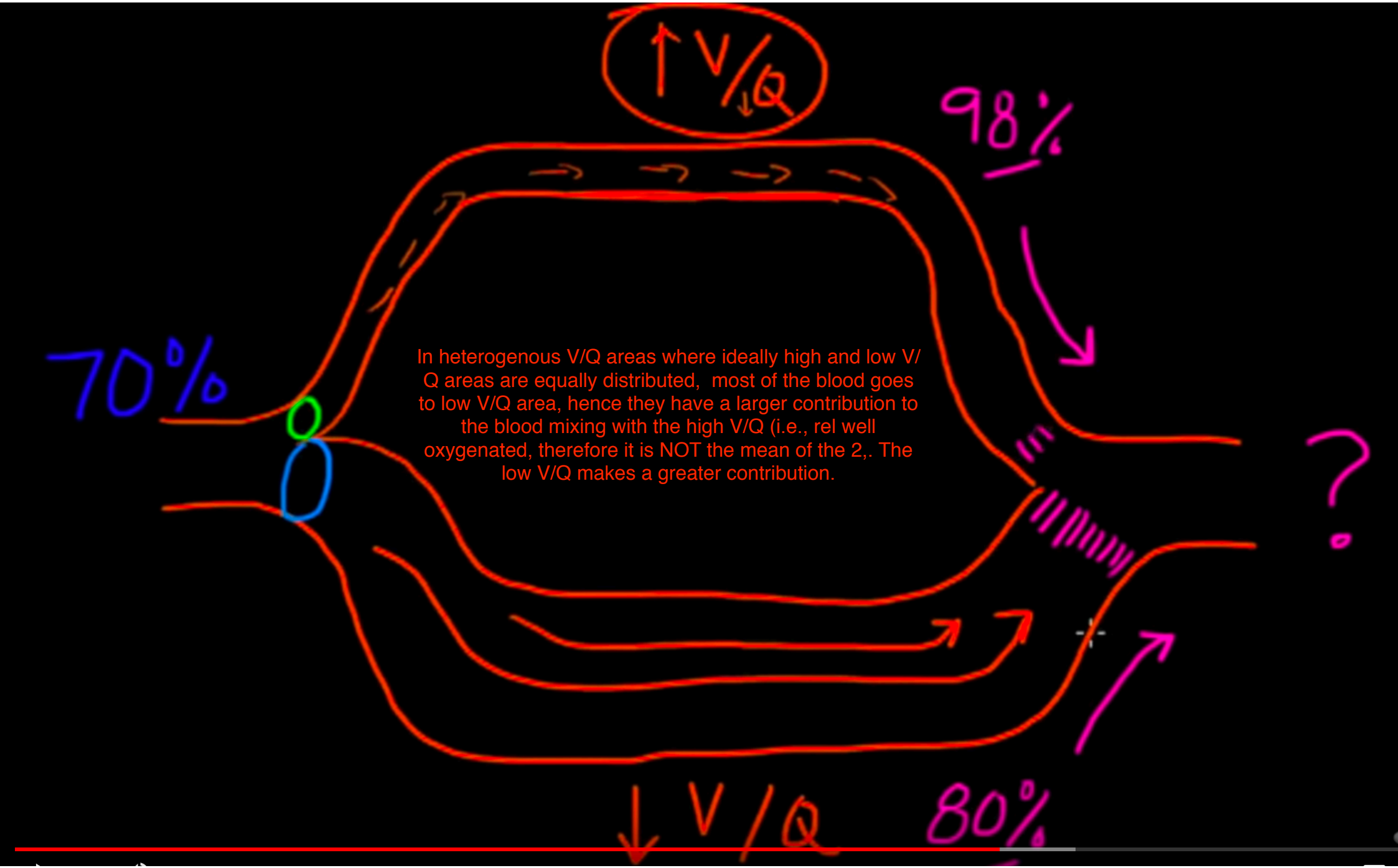
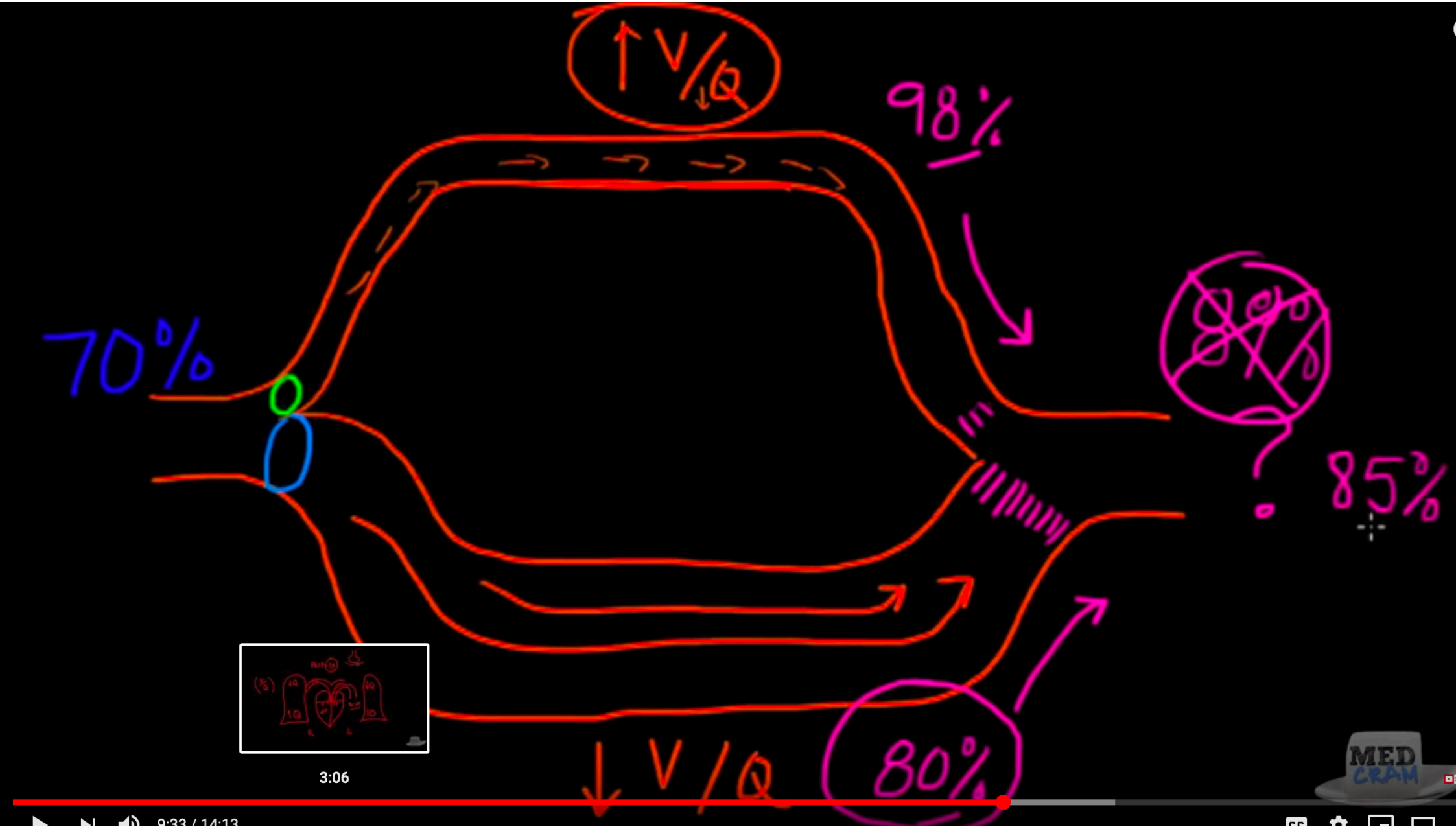
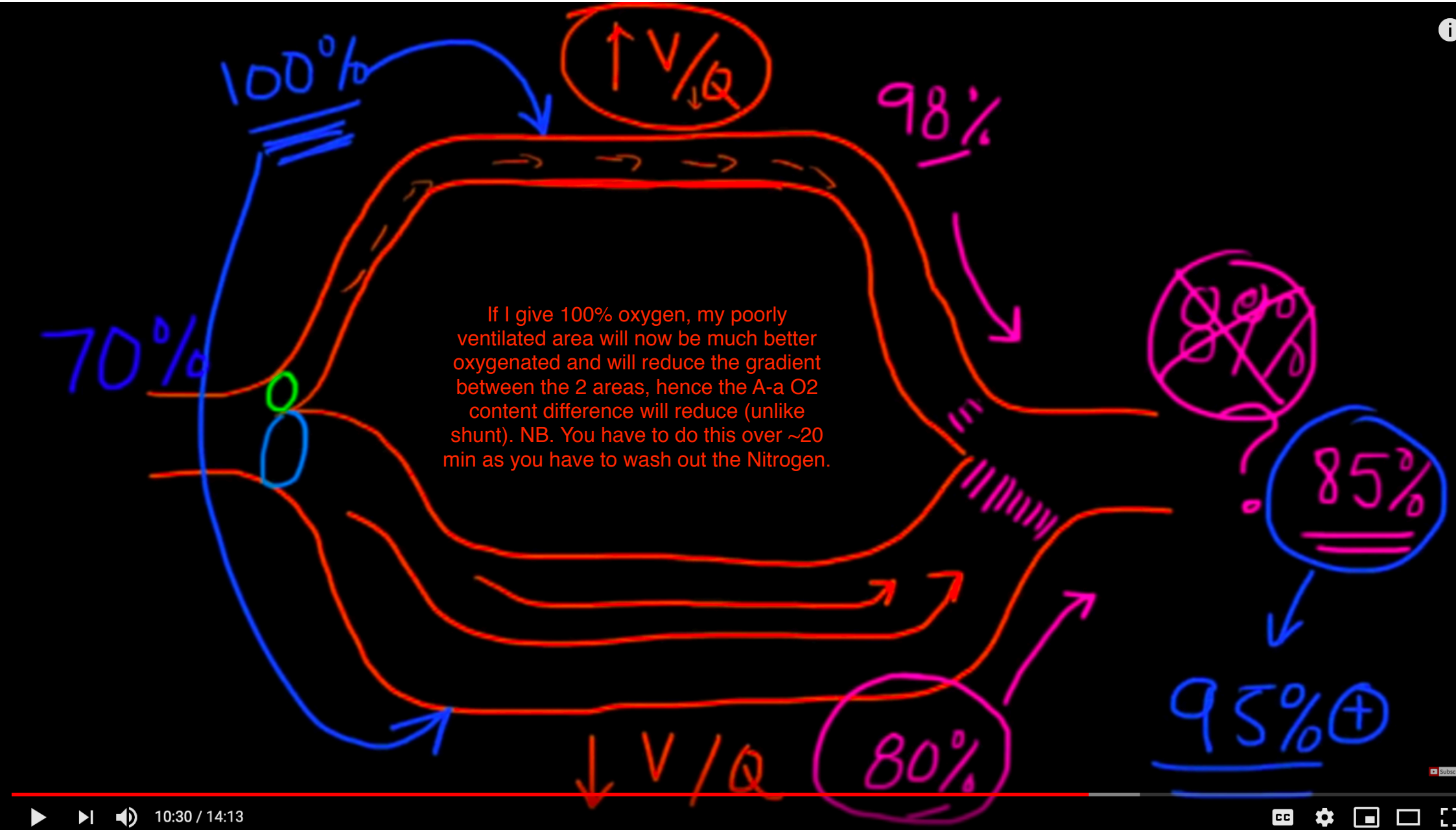
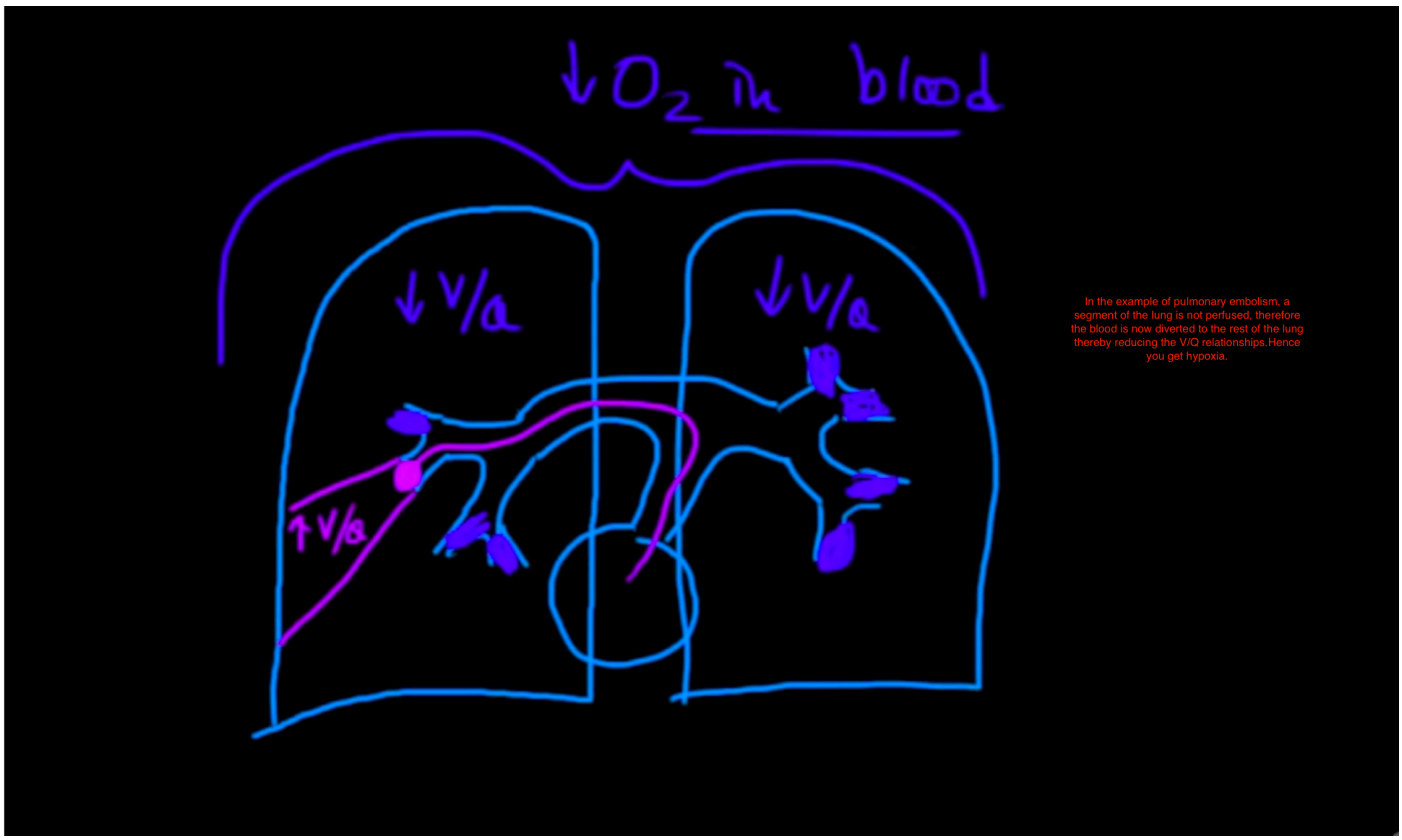

↑V/Q
98%
70%
In heterogenous V/Q areas where ideally high and low V/Q areas are equally distributed, most of the blood goes to low V/Q area, hence they have a larger contribution to the blood mixing with the high V/Q (i.e., rel well oxygenated, therefore it is NOT the mean of the 2,. The low V/Q makes a greater contribution.
?
↓V/Q
80%


↑V/Q
98%
70%
85%
↓V/Q
80%
MED CRAM
3:06
9:33 / 14:13


100%
↑V/Q
98%
70%
If I give 100% oxygen, my poorly ventilated area will now be much better oxygenated and will reduce the gradient between the 2 areas, hence the A-a O2 content difference will reduce (unlike shunt). NB. You have to do this over ~20 min as you have to wash out the Nitrogen.
85%
95%+
↓V/Q
80%
10:30 / 14:13



In the example of pulmonary embolism, a segment of the lung is not perfused, therefore the blood is now diverted to the rest of the lung thereby reducing the V/Q relationships.Hence you get hypoxia.