Discussion Notes for Gas Exchange Computer Lab

Effects of Ventilation

1. Changes in respiratory rate cause proportionate changes in minute ventilation and also in alveolar ventilation.

2. Changes in alveolar ventilation cause reciprocal changes in alveolar \( PCO_2 \).

3. Alveolar \( PO_2 \) can be calculated using the alveolar air equation (see Gas Exchange lecture notes, page 5-3).

4. Changing inspired \( O_2 \) has no direct effect on \( PCO_2 \). Alveolar (and hence arterial) \( PCO_2 \) is determined only by \( CO_2 \) production and alveolar ventilation. We could have followed Experiment 1 by keeping the reduced alveolar ventilation but then lowering \( CO_2 \) production by half to show that the \( PCO_2 \) then falls to 40. This can be done by changing \( O_2 \) uptake to 150 ml/ min. and keeping \( R \) at 0.8.

5. Changes in \( PCO_2 \) are not inversely proportional to changes in \( VT \), but rather to changes in \( (VT - VD) \).

Diffusion

1. \( DL \) is the ratio of \( VO_2 \) to the mean \( PO_2 \) difference between alveolar and capillary blood as the RBC traverses the pulmonary capillary. Normally there is plenty of A-c difference, and equilibrium occurs before the RBC leaves the capillary.

2. If \( DL \) becomes abnormally low, the reserve is used up, initially with hardly any effect on \( O_2 \) uptake. As \( DL \) falls lower, the capillary \( PO_2 \) will then fail to equilibrate, and arterial \( PO_2 \) will drop.

3. Increasing \( O_2 \) uptake, as with exercise, requires a larger gradient and thus exaggerates the problem resulting from a low \( DL \).

4. A n increase in \( FIO_2 \) raises \( PAO_2 \) and therefore the \( O_2 \) gradient so that equilibration is restored.

5. \( CO_2 \) is so diffusible that no significant A-a \( CO_2 \) gradient can exist, even when \( O_2 \) transport is severely impaired.
Shunt

1. When poorly oxygenated mixed venous blood mixes with well-oxygenated pulmonary capillary blood, the result is always less than fully oxygenated arterial blood. The larger the fraction of cardiac output that is shunted, the lower will be the arterial \( P_{O_2} \).

2. \( P_{CO_2} \) in arterial blood is very little affected because \( P_{CO_2} \) of mixed venous blood is only about 6 mm Hg higher than arterial. The model shows a slight rise in \( P_{CO_2} \) of arterial blood, but in real life the \( P_{CO_2} \) would be adjusted by a slight reflex increase in alveolar ventilation. The rule is that alveolar \( P_{CO_2} \) is determined by \( V_{CO_2} \) and \( VA \); \( PACO_2 \) and \( PaCO_2 \) are usually not significantly different.

3. Unlike the situation with hypoxemia due to all other causes, increasing \( F_{IO_2} \) in the presence of a shunt results in only a slight rise in arterial \( P_{O_2} \). This is because a high \( F_{IO_2} \) affects only the non-shunted blood, which is already almost fully saturated and cannot take up enough extra \( O_2 \) to make up for the low saturation in shunted blood.

4. It is possible to calculate \( O_2 \) values resulting from shunt, but it requires use of the Hb dissociation curve and is best left to the computer.

V/Q Imbalance

1. If a portion of the lungs has a low V/Q ratio compared with the rest of the lung, that portion contributes poorly oxygenated blood to the mixture. The effect is similar to having a portion with normal V/Q ratio plus a shunt.

2. If there is a portion with high V/Q ratio, the effect is similar to a portion with normal V/Q ratio plus a dead space. If there is no compensatory increase in total minute ventilation, the dead space ventilation results in a lower alveolar ventilation, and this causes a rise in \( P_{CO_2} \).

3. Calculation of physiological dead space and physiological shunt per the Riley Model permits a good assessment of the severity of V/Q imbalance.

4. Increasing inspired \( O_2 \) raises alveolar \( O_2 \) so that even an underventilated portion may then oxygenate the blood fully.

5. The V/Q ratio for both normal lungs is close to 1.0, but remember that it is not the total that counts. It is only when the ratio is not the same in all parts of the lung that an abnormality of gas exchange results.
Cardiac Output

1. In a normal resting subject, blood flow to the tissues delivers enough O\textsubscript{2} so that blood leaving the systemic capillaries is still about 75% saturated, which corresponds to a P\textsubscript{O\textsubscript{2}} of about 40 mm Hg. This P\textsubscript{O\textsubscript{2}} is sufficient to allow diffusion of O\textsubscript{2} into the tissues. During exercise, as much as half of the O\textsubscript{2} delivered is taken up by the tissues, so the mixed venous P\textsubscript{O\textsubscript{2}} may fall to as low as 25 mm Hg.

2. If cardiac output falls below normal, less oxygen is delivered to the tissues, and venous P\textsubscript{O\textsubscript{2}} (and hence tissue capillary P\textsubscript{O\textsubscript{2}}) falls. The lungs will continue to oxygenate the blood normally, but tissues may suffer hypoxia.

Hemoglobin

1. Even when cardiac output is normal, anemia (low hemoglobin in the blood) reduces delivery of oxygen to the tissues. While the arterial blood P\textsubscript{O\textsubscript{2}} and saturation are normal, the oxygen content is low, and mixed venous P\textsubscript{O\textsubscript{2}} falls.

2. Under some circumstances, raising the hemoglobin level may compensate for a low cardiac output. Increasing cardiac output is a very effective physiological compensation for anemia.

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