

MEDICAL PHYSIOLOGY

Respiration Conference - Quiz 5A

November 7, 2000

1. A patient breathing air shows the following values:

arterial PCO₂ = 50 mm Hg

arterial PO₂ = 55 mm Hg

Explain the mechanism responsible for the abnormal PCO₂

The high PaCO₂ is due to hypoventilation. If CO₂ production remains unchanged, there is an inverse relationship between alveolar ventilation and PaCO₂.

2. Is the abnormal PO₂ in the patient of question 1 due to the same mechanism responsible for the abnormal PCO₂ ? What other mechanisms may be involved in lowering the arterial PO₂ ?

The hypoventilation will decrease PO₂ to some extent; however that is relatively minor compared with the effect of other possible mechanisms. Hypoventilation will lower alveolar PO₂ according to the alveolar gas equation.

$$PAO_2 = PIO_2 - (PaCO_2 * f)$$

If the lungs are normal, PAO₂ will be close to PaO₂. In this case, there is a very large difference showing that other factors participate in lowering PO₂. In this case, the major factor is the veno-arterial shunt produced by the alveolar collapse. Impaired diffusion may contribute.

3. Explain why lack of surfactant increases the work of breathing.

Surfactant lowers surface tension of the fluid lining the alveoli. This maintains lung elastic recoil within normal values. If there is insufficient surfactant, elastic recoil increases, compliance decreases, the transpulmonary pressure needed to inflate the lung increases, and this increased transpulmonary pressure is obtained through a more vigorous contraction of the respiratory muscles.

4. Referring to the conference's case, explain why blood flow through the patent ductus - from the aorta to the pulmonary artery- increased as the patient's condition improved.

Flow through the ductus was low when the baby was ill because pulmonary vascular resistance was high. This was due to lung collapse, and, more importantly, to the hypoxia that produces pulmonary vasoconstriction. When the baby's condition improved, the lungs started opening up, PVR decreased because improved oxygenation, pulmonary arterial pressure decreased below systemic arterial pressure, and flow through the ductus increased.

5. Explain why in respiratory distress syndrome positive airway pressure ventilation is more effective in improving blood oxygenation than breathing high O₂ gas mixtures.

The collapsed alveoli do not receive air. On the other hand, blood flowing through the capillaries served by ventilated alveoli is fully saturated. Increasing the inspired PO_2 does not help because the collapsed alveoli do not get the air, and the blood leaving the ventilated alveoli already has as much O_2 as it can get. What is needed is to reverse the collapse of alveoli and prevent further collapse, so the lung is more uniformly ventilated.

MEDICAL PHYSIOLOGY

Respiration Conference - Quiz 5B

November 2, 2000

1. A patient breathing air shows the following values:

arterial $PCO_2 = 25$ mm Hg

arterial $PO_2 = 55$ mm Hg

Explain the mechanism responsible for the abnormal PCO_2

The low PCO_2 is due to alveolar hyperventilation. This due to stimulation of the peripheral chemoreceptors by the low PO_2 .

2. Is the abnormal PO_2 in the patient of question 1 due to the same mechanism responsible for the abnormal PCO_2 ? What other mechanisms may be involved in lowering the arterial PO_2 ?

If anything, the alveolar hypoventilation will tend to increase PaO_2 . However, this effect is small compared to other mechanisms that tend to lower PaO_2 . In this case the major factor is the veno-arterial shunt created by blood flowing through areas of collapsed lung. Impaired diffusion may also contribute. These combined mechanisms tend to lower the arterial PO_2 below the calculated alveolar PO_2 .

3. Explain the effects of low surfactant on lung compliance.

Surfactant lowers surface tension of the fluid lining the alveoli. This maintains the lung's elastic recoil within normal values. If there is insufficient surfactant, elastic recoil increases, the transpulmonary pressure needed to inflate the lung increases and compliance decreases.

4. Referring to the conference's case, explain why blood flow through the patent ductus -from the aorta to the pulmonary artery- was very low initially, when the patient's condition was poor.

Although the ductus was patent the flow through it was low because pulmonary arterial pressure was high. This was due to the areas of lung collapse that increase resistance, and to the hypoxia which produces pulmonary vasoconstriction and increases pulmonary vascular resistance.

5. Explain why in respiratory distress syndrome, breathing high O_2 gas mixtures is less effective in improving blood oxygenation than positive end expiratory pressure ventilation.

The collapse of alveoli produces a veno-arterial shunt whereby insufficiently oxygenated blood mixes with blood draining well ventilated alveoli. Increasing inspired PO_2 does not increase oxygenation of the blood draining the ventilated alveoli, which already is well oxygenated. On the other hand, the inspired air does not reach the collapsed alveoli. On the other hand, maintaining always positive airway pressure helps open collapsed alveoli and prevents further collapse. Accordingly, the previously non-ventilated alveoli now receive ventilation and the blood draining these alveoli is better oxygenated than before.