Para2001: integrated clinical case case studies example

Health & Medicine, Stress

Written Case Study – Pathophysiology II

(Location of the University)

1. Describe the underlying pathology of COPD. What impacts do these pathological changes have on normal physiology? In particular, describe alveolar ventilation in a normal individual and discuss how this might be different in Mr Wenham. (20 marks)

Chronic Obstructive Pulmonary Disease or COPD is touted to be the third leading cause of death in the United States of America. In 2010, it has claimed the lives of 134, 676 individuals (CDC 2013). By definition, COPD presents persistent blockage of airflow in the lungs — thus interfering normal breathing. Those afflicted with such condition generally present symptoms of the classic triad: chronic bronchitis, emphysema and asthma.

The primary cause of Chronic Obstructive Pulmonary Disease is tobacco use (WHO 2014). The lungs become vulnerable to elastolytic destruction which would eventually lead to chronic bronchitis and emphysema (Spurzem & Rennard 2011).

The pathophysiology of COPD starts at the cellular level. Epithelial cells — the cells lining the airways (i. e. bronchi, bronchioles) — undergo cellular changes as a response to long-term exposure to cigarette smoke, an airway irritant. The effects are devastating to these cells, giving rise to muco-ciliary impairment and parenchymal destruction.

Marked thick mucus secretions remain static because of the absence of a ciliated epithelium, thus further impeding airflow and causing repeated infection (bronchitis). Chronic inflammation associated with the release of proteolytic enzymes such as elastases causes more damage to the

bronchiolar and alveolar wall by breaking down elastic fibers.

In a healthy individual, thoracic volume and lung volume decrease during quiet expiration because of lung recoil. Lung recoil is possible because of two factors: presence of elastic fibers in the connective tissues of the lungs and surface tension of the film of fluid in the alveoli. Because the elastic fibers have been destroyed in the lungs of a patient with COPD, the chest wall enlarges (hence, the symptom “ barrel chest”), the total lung capacity goes up and the lateral chest films show a lot of air below the sternum.

In this case, part of the inhaled air becomes retained in the air passages. Coughing, as a means to remove mucus, increases the pressure in the alveoli — thus, resulting in alveolar wall rupture and destruction (Seeley, Stephens & Tate 2005).

On another note, reduction of the alveolar surface area decreases gas exchange. This would ultimately result to the decrease of oxygen and increase in carbon dioxide in the bloodstream, signalling respiratory acidosis (Byrd 2014). By virtue of homeostasis, the body will try to compensate for depleting oxygen stores by increasing the respiratory rate — which is evident in Mr. Wenham’s case.

2. Discuss why you would administer salbutamol and describe how it works at the cellular level. (10 marks)

The smooth muscles of the bronchial passages have two types of receptors: the β2-adrenergic receptors and muscarinic receptors. The M3 muscarinic receptors are activated by acetylcholine released from parasympathetic fibers, causing bronchoconstriction. On the contrary, activation of the β2-adrenergic receptors results to bronchodilation (Proskocil and Fryer 2005).

Salbutamol is a medication that is classified as a short-acting beta 2-adrenergic receptor agonist. It is usually given through aerosol administration for patients who exhibit bronchoconstriction and bronchospasm (i. e. asthma, COPD). This drug stimulates the beta 2-adrenergic receptors of bronchial smooth muscles, causing them to relax and dilate. In addition, salbutamol inhibits release of bronchoconstricting agents from mast cells — a granulocyte that produces products responsible for bronchial hyperresponsiveness and mucous gland secretion (Bradding, Walls & Holgate 2006).

3. Discuss why they would take an arterial blood gas and explain how the results relate to the pathophysiology you described. (10 marks)

An arterial blood gas measurement is necessary in assessing critically ill patients. This provides valuable information about the patient’s blood pH and partial pressures of arterial oxygen and carbon dioxide. Generally, it also reflects serum bicarbonate.

In COPD, ABG analysis is imperative, as it provides the best clues as to the acuteness and severity of the disease exacerbation (Kleinschmidt 2012). Ineffective breathing pattern and alveolar hypoventilation are present in COPD, thus leading to hypoxia and hypercapnia. An increase of carbon dioxide in the bloodstream results in a decreasing blood pH (Seeley, Stephens & Tate 2005).

Normally, hypoxia and hypercapnia stimulate the respiratory centers of the brain to increase ventilation. In Mr. Wenham’s case, there is evidence of tachypnea — a mechanism characterized by rapid breathing that aims at decreasing carbon dioxide levels of the blood. His ABG results show a

decrease in blood pH and a compensating serum bicarbonate level — indicative of an acute respiratory acidosis secondary to exacerbation of COPD (Byrd 2014).

4. Discuss the issues surrounding the use of supplemental oxygen therapy in patients with severe exacerbations of COPD. What problems can it cause and why? (20 marks)

In managing COPD patients, it is important to be aware of the concepts of hypercapnic and hypoxic drives. Respiration is controlled by these two drives. Hypercapnic means that if the CO2 level is high, the body is going to adjust by breathing faster to blow off excess carbon dioxide. Hypoxic, on the other hand, means that if the oxygen level is low, the person tends to breathe faster because the body demands more oxygen (Campbell 1967). Individuals afflicted with COPD, however, have adjusted their tolerance of a higher than normal CO2 levels in the body brought about by the alterations in the respiratory tract. For example, one patient may have become accustomed to a PaCO2 of 50 mmHg than the normal range of 35 to 45 mmHg (Campbell 1967).

Hypoxic drive is also a critical concept to understand, especially when it comes to giving oxygen therapy to clients with COPD. Many of these patients are given supplemental oxygen for life. Progressive hypoxemia (PaO2 of less than 55 mmHg or O2 saturation of less than 90%) can be helped through oxygen supplementation for 15-19 hours a day (Duiverman, Wempe, Bladder, Jansen, Kerstjens & Zijlstra 2008). However, long-term oxygen therapy can cause a decrease in hypoxic drive. This means that the patient may lose his drive to breathe on his own.

Some noteworthy key points presented by Murphy and Driscoll (2001) include: 1) Oxygen can cause carbon dioxide retention in patients with COPD, 2) The degree of carbon dioxide retention that develops in response to oxygen is varied, 3) There is evidence to suggest that high concentration oxygen causes more carbon dioxide retention and more acidosis than low concentration oxygen, 4) Carbon dioxide narcosis can often be managed by reducing the concentration of inspired oxygen, and 5) Carbon dioxide retention below 80-100 mmHg may produce no symptoms in some patients, especially if long standing and not associated with academia and may resolve despite continuing oxygen therapy.

5. Do you think it is a good idea to remove Mr Wenham’s oxygen? Provide an argument supporting why it is OR why it is not. (10 marks)

Campell (1967) hypothesized that an increase of inspired oxygen concentration to hypoxic patients causes them to underventilate because they mainly rely on their hypoxic drive to breathe. That is, the more oxygen given, the more they stop breathing. And when that happens, carbon dioxide levels in the body would increase — thus, resulting to carbon dioxide narcosis.

Symptoms of CO2 retention include tachypnea, tachycardia and low blood pressure. To stabilize Mr. Wenham’s condition, it is best to remove the oxygen supplementation.

6. What is BiPAP? How might BiPAP help to improve Mr Wenham’s clinical condition? (10 marks)

BiPAP machine, otherwise known as a bilevel positive airway pressure machine, is a small device used to assist the patient’s breathing. A face

mask that is connected to a flexible tubing is worn by the patient. BiPAP machine supports the user's breathing by pushing air and oxygen into the lungs. Unlike CPAP, this particular non-invasive ventilation equipment allows the delivered air to be set at a specific pressure for inhalation and another for exhalation. Variable levels of air pressure give the patient ease and comfort because there is no need to exhale against extra pressure (Soo Hoo 2014).

A study conducted by Confaloneri, Aiolfi and others (1994) supports that Bi-level pressure support ventilation via nasal mask (NIPSV) can be used as an initial approach to severe exacerbations of COPD. In this case, however, patients with an Apache II score of less than 29 have high success rates with the use of NIPSV. Multiple controlled trials have shown that BiPAP, along with other noninvasive ventilation strategies, decreases the need for endotracheal intubation and length of stay in patients with COPD (Soo Hoo 2014).

7. What is spirometry? (5 marks)

A common lung test function, spirometry seeks to help diagnose various lung conditions particularly COPD. It is also used to monitor the patient's response to treatment.

Spirometry gauges the amount of air inhaled and exhaled (Seeley, Stephens & Tate 2005). It also measures the flow of air that goes in and out of the lungs. Measurements used include the forced expiratory volume in one second (FEV1), forced vital capacity (FVC), and FEV1 divided by FVC (Seeley, Stephens & Tate 2005).

8. Discuss the significance of the results by examining the differences

between Mr Wenham’s spirometry and that of a normal individual. (10 marks)

Readings usually differ depending on the age, size and sex. Medical practitioners refer to a chart for the spirometry readings.

In general, the spirometry reading of a healthy individual is considered normal when it reflects an FVC and FEV1 above 80% predicted, plus an FEV1/FVC ratio of greater than 70%. Narrowed airways affect the amount of air that a person can blow quickly (Kenny 2013). An FEV1/FVC ratio of 0. 7 or less—such as in Mr. Wenham’s case—can be classified as an obstructive pattern on spirometry (Tidy 2013).

Aside from diagnosing COPD, spirometry can be used to measure the extent of damage to the lungs. As for Mr. Wenham’s case, the spirometry results could help in assessing the effectiveness of the treatment modalities used (i. e. inhalers). The readings would improve if the narrowed airways have become wider as a result of the medication (Tidy 2013).

9. How does the pathology of COPD explain these differences? (5 marks)

Chronic Obstructive Pulmonary Disease is a condition characterized with narrowed airways. An obstructive airflow pattern happens when the airways are narrowed to the point that the amount of air the patient can blow out at a faster rate is reduced. A reduction in the forced vital capacity in patients with COPD is caused by the loss of elasticity of the lung tissue and as well as alveolar damage and coalescence (Tidy 2013).

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LIST OF ILLUSTRATIONS