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Acute Bronchospasm Under General Anesthesia

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Background

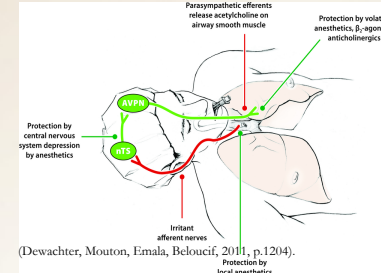
The topic presented in this poster is acute bronchospasm during general anesthesia. Bronchospasm is of interest to me because I am studying to become a certified registered nurse anesthetist (CRNA) and this is an acute pathophysiological process that I will encounter as an anesthesia provider. It's essential for all anesthesia providers to have an understanding of the pathophysiology of bronchospasm and how to appropriately respond when it occurs.

Implications for CRNA'S

Bronchospasm has potential to become a life-threatening emergency for a patient while they are under anesthesia care. If severe enough, a bronchospasm can make it impossible for the anesthesia provider to ventilate the patient. If not treated immediately, the inability to ventilate a patient will cause hypoxemia, hypercarbia, and metabolic acidosis, all of which places the patient at risk for sudden cardiovascular collapse, anoxic brain injury, and or death (Gaba, 2015, pg. 187). It is because of the serious nature and acuity of bronchospasm that a CRNA is obligated to have a knowledge of its pathophysiology, signs and symptoms, differential diagnosis, and how to effectively respond in treating it (Nagelhout, 2014).

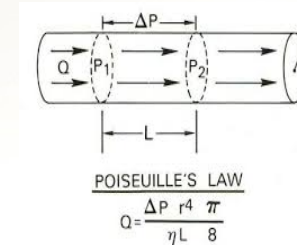
Pathophysiology

Essentially, a bronchospasm is the constriction of bronchial smooth muscle due to a stimulus. The types of stimulus that can cause a bronchospasm are either mechanical or chemical in nature (Dewachter, Mouton, Emala, Beloucif, 2011). Mechanical stimulation is that it is the cause of a reflex-induced bronchoconstriction. The reflex pathway begins with a physical object (such as an endotracheal tube) that stimulates the nerves in the airway which can potentially send an afferent signal to the brainstem which in response sends an efferent signal back to the airway through the vagus nerve. Once the efferent signal arrives at the airway, it causes a release of acetylcholine into the neuromuscular junction which binds to the muscarinic receptors of the smooth muscle. The release and binding of acetylcholine within the bronchial smooth muscle cause a neuromuscular action potential to be propagated causing smooth muscle constriction and bronchospasm (Dewachter, Mouton, Emala, Beloucif, 2011, p.1204). Chemical stimulation for bronchospasm is that it is either of an asthmatic or anaphylactic origin. Bronchospasm that precipitates from an asthmatic origin is essentially an acute exacerbation of asthma while a patient is under anesthesia (Nagelhout, 2014).



Significance of Pathology

Based on Poiseuille's Law, the flow of air into the lungs is primarily determined by the pressure of air and the radius of the conducting passageways (the bronchi). When bronchial smooth muscle constriction occurs, the radius of the of the conducting passageways is reduced which causes an increased resistance to the fourth power. This increased resistance causes a decrease in the flow of air into the lungs. To compensate, the pressure of air going into the lungs must be increased in order to achieve the same minute volume. In Bronchospasm, it is possible that the constriction is severe enough that increased pressure won't be sufficient enough to ventilate the patient and hypoventilation and hypoxemia results (Trips, 2014).



Identifying Bronchospasm

- Difficulty ventilating the patient
- Prolonged expiratory time
- Wheezing breath sounds
- Diminished breath sounds (Looseley, 2011, pg. 17)
- Increased PIP
- Decreased tidal volume
- Diminished ETCO2
- Decreased SaO2 or O2 saturation (Gaba, 2015, pg. 185)

Treatment

- Deepen anesthesia with sevoflurane, propofol, or another anesthetic agent
- Give 100% FIO2
- Auscultate chest for bilateral breath sounds
- Verify ETT position & suction ETT tube
- Optimize ventilation (Gaba, 2015, pg 186)
- Give a short-acting B2-agonist (e.g. Albuterol).
- Severe bronchospasm: Intravenous epinephrine and intravenous corticosteroids (Nagelhout, 2014, pg. 625)

Conclusion

Bronchospasm can become a life threatening condition for a patient under general anesthesia. It is an acute bronchial smooth muscle constriction that can cause hypoventilation, hypoxemia, and metabolic acidosis. It's imperative for any anesthesia provider to understand what a bronchospasm is, its pathophysiology, differential diagnosis, and algorithm of treatments.

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