Acute Asthma: A review of the Pathophysiology, Assessment Findings and Treatment strategies using a Case Study Approach

Abstract
Critical care nursing requires an in depth understanding of the underlying physiology, pathophysiology and rationales for treatment strategies in order to minimise complications and increase the chance of a positive outcome for the patient. The purpose of this care study is to critically evaluate the care of a 25-year-old male who was admitted to the intensive care unit (ICU) with acute asthma. The pathophysiology of acute asthma is reviewed and related to the presenting symptoms and assessment findings. A rationale for treatment is provided with reference to current professional and national guidelines and care pathways. Implications for future nursing practice are also considered.

Keywords
Acute asthma; Pathophysiology; Critical Care; Mechanical Ventilation; Treatment

Introduction
Asthma is a highly prevalent chronic respiratory disorder characterised by reversible airway narrowing, airway hyper-responsiveness and inflammation (Dougherty and Fahy, 2003). In the UK, more than 15% of the population suffer from Asthma. Although asthma can be well controlled, standard treatment of bronchodilators and steroids is ineffective in refractory acute asthma or status asthmaticus, therefore posing a life threatening situation and necessitating the need for management in a critical care setting and consideration of mechanical ventilation. This care study illustrates the critical care requirements of a fictional patient (referred to under the pseudonym ‘John’) who presented to the emergency department with an acute asthmatic episode, which was refractory to nebulised bronchodilators (See appendix A for scenario). Assessment findings are linked to the underlying pathophysiology and treatment strategies, including mechanical ventilation, are critically evaluated and rationalised.

Pathophysiology
A variety of pathological processes including an immune response, bronchoconstriction and inflammation, severely impact the structure and function of the tracheobronchial tree that leads to airway narrowing and obstruction (Cruickshank and Lumley, 1999). An understanding of these processes is required in order to interpret assessment findings and rationalise treatment.

Aetiology
Asthma is classified as atopic (allergic) or non-atopic (non-allergic) depending on the cause (Kaufman, 2011). Atopic asthma is the result of a type 1 hypersensitivity reaction triggered by an extrinsic allergen such as cat dander. This reaction is immunoglobulin E (IgE) mediated, meaning there has been previous exposure and sensitisation. Upon primary exposure, dendritic cells present the allergen to T-Helper 2 lymphocytes, which then stimulate B-lymphocytes to synthesis and release IgE antibodies that subsequently bind to mucosal mast cells in the airways.
Future inhalation of the specific allergen results in degranulation of IgE-bound mast cells and a release of chemical mediators causing a hypersensitivity reaction responsible for the bronchoconstriction and airway inflammation observed in atopic asthma (Ishmael, 2011).

In contrast, it is believed that non-atopic asthma is not associated with an immunological response. Inhalation of cold air, respiratory tract infections and anxiety are widely acknowledged stimulants of bronchoconstriction, however there is debate regarding the exact pathological mechanisms. Humbert et al (1999) found similarities in the immunopathology of both variants of asthma. Their study of bronchial biopsies in non-atopic patients found infiltration of TH2 cells and evidence of localised IgE production. A similar study by Jayaratnam et al (2005) discovered up-regulation of high affinity IgE receptors, which also indicates local IgE synthesis. The evidence suggests that despite variation in the trigger, perhaps non-atopic asthma is also a consequence of an inappropriate immunological response, although more localised. Further research in the importance of the IgE mediated response could possibly reveal new therapeutic possibilities for both types of disease.

Pathogenesis

Asthma is separated into an early (acute) and a late phase response (Porth and Matfin, 2009). Initially, bronchoconstriction is triggered by an array of chemical mediators, such as histamine, prostaglandins and cysteiny leukotrienes, which are released from airway mast cells (O'Byrne and Thomson, 2001). Histamine triggers a parasympathetic nervous system response via stimulation of H1R, H2R and H4R receptors causing bronchial smooth muscle contraction (Thurmond, 2011). Furthermore, histamine initiates an inflammatory response causing vasodilation, increased permeability of the airway microvasculature and leakage of filtrate into the interstitial tissue causing airway oedema, resulting in a narrowed airway lumen and increased airway resistance (Porth and Matfin, 2009).

The late phase response is associated with infiltration of inflammatory cells (eosinophils and lymphocytes) and cytokine release that can cause remodelling of the airways that can exacerbate acute attacks if poorly managed (Fahy et al, 2000). Prolonged inflammation and subsequent shedding of the airway epithelium increases penetration of allergens and exposes sensory nerves resulting in bronchial hyper-responsiveness and increased occurrence and severity of bronchoconstriction (Kaufman, 2011). Additionally, hypertrophy of mucous glands and damage to the ciliated airway epithelium inhibits mucous clearance leading to the formation of intraluminal mucous plugs, which increase airway obstruction (Porth and Matfin, 2009).

Analysis of Assessment Findings

John’s assessment findings were consistent with the manifestations of acute asthma. The key observations are interpreted and related to the underlying pathophysiology.
Breathing

John's breathing assessment (Table 1, Appendix A) noted an abnormally high respiration rate (RR). Hyperventilation is a recognised sign of acute asthma, however the trigger remains somewhat unclear. Totora and Derrickson (2009) suggest a pO2 below 9kPa increases respiratory drive through stimulation of chemoreceptors whilst Osborne et al (2000) attribute hyperventilation to stimulation of stretch receptors due to lung hyperinflation. Both theories could cause the increased RR in view of John's altered lung physiology. It is also likely John was anxious and panicked, which can also exacerbate respiratory distress (O'Bryne, 2001).

The wheezing breath sounds heard on assessment are a cardinal sign of airway narrowing, produced by the increased resistance met by exhaled air (Cruickshank and Lumley, 1999). John likely presented with a 'polyphonic' wheeze indicating disseminated airway obstruction (McGloin and McLeod, 2010). The reduced air entry heard on auscultation is the result of progressive airway narrowing and closure (Porth and Matfin, 2009).

The recruitment of accessory muscles (sternocleidomastoid and scalene muscles) indicates John's increased work of breathing (WB). WB is the sum of the forces required to expand the lungs by overcoming resistance from the airways and the lung/chest wall elastic forces and structures (McGloin and McLeod, 2010). Airway resistance increases due to severe airway narrowing and gas trapping as a result of incomplete exhalation. This leads to an increased alveolar pressure at the end of expiration, known as intrinsic peak expiratory end pressure (iPEEP). Ensuing dynamic hyperinflation of the lungs close to the total lung capacity causes flattening of the diaphragm and imposes a significantly increased workload on the inspiratory muscles despite less effective ventilation (Soni et al, 2003).

John's SpO2 is markedly low and reflects poor ventilation and hypoxia despite the current treatment of 60% oxygen and nebulised salbutamol.

Two arterial blood gases (ABGs) were taken an hour apart to assess respiratory and metabolic function and guide further treatment (Table 2, Appendix A). The alterations in the ABGs show that John is becoming hypoxic, hypercapnic and has developed a mixed acidosis. The altered physiology and function of the lungs is the primary cause of John's deterioration.

John has signs of refractory hypoxaemia and therefore type 1 (hypoxic) respiratory failure (Casey, 2013). The hypoxaemia is a result of V/Q (ventilation/perfusion) mismatching, which is a complication of acute asthma (Soni et al, 2003). Severe airway narrowing and obstruction causes a marked decrease in alveolar ventilation whilst alveolar perfusion remains unaffected. In response to decreased ventilation, the pulmonary vasculature attempts to maximise oxygenation of blood via hypoxic vasoconstriction (HPV) to ensure blood is re-directed to well-ventilated areas of the lung, however this becomes inefficient in severe hypoxia. Consequently, poorly oxygenated blood leaves the pulmonary circulation causing a physiologic right to left shunt (Porth and Matfin, 2009). Studies suggest that HPV is impeded in acute asthma. Moudgil et al (2005) suggest that HPV is hindered during hyperventilation whilst Rodriguez-Roisin (1997) proposes that the presence of inflammatory mediators causes pulmonary
vasodilation and increased perfusion of poorly ventilated alveoli. Both proposals are plausible in view of John’s high RR and presence of airway inflammation.

The ABGs also indicate an increase from an initially low pCO2 (hypocapnia) to a high pCO2 (hypercapnia). The hypocapnia evident in the first ABG is the result of hyperventilation whereby increased alveolar ventilation causes excessive exhalation of CO₂ and subsequently low levels in the blood (Porth and Matfin, 2009). The hypercapnia depicts a shift to type 2 (hypercapnic) respiratory failure as a result of respiratory muscle exhaustion and decreased alveolar ventilation. As CO₂ is a weak acid, the increased levels in the blood contribute to the fall in pH (Casey, 2013).

The fall in pH reflects an increased concentration of hydrogen ions in the body and can have devastating effects on cellular activity (Casey, 2013). Hypoxaemia and ensuing cellular hypoxia causes a shift in cellular respiration from aerobic to anaerobic causing lactate production, which alters the pH of the blood when hydrolysed (Porth and Matfin, 2009). Disturbances in pH are modulated by the buffering action of bases/alkalis (BE) such as bicarbonate (HCO₃⁻). Therefore, the decreased HCO₃⁻, BE and increasing lactate levels indicate cellular hypoxia and the onset of a metabolic acidosis (Casey, 2013).

Circulation

John’s cardiovascular assessment (Table 3, Appendix A) notes he is tachycardic and pyrexial, however his BP, MAP and CVP are reasonable. Tachycardia may be caused by increased Wₘ, which requires an increased blood supply resulting in an increased heart rate to compensate (Martini et al, 2008). This would also cause an increase in core temperature due to an increased metabolic rate and heat production (Martini et al, 2008). The anxiety and panic associated with respiratory distress prompts the release of adrenaline, which is an alpha and beta receptor agonist, therefore causing vasoconstriction and tachycardia respectively (Levick, 2010). Salbutamol also causes tachycardia through stimulation of beta-receptors (Hazeldine, 2013). Arguably, John’s blood pressure is low in view of the stress response, however given John’s young age this would not necessarily be cause for concern.

John has an adequate urine output (UOP) exceeding the recommended minimum of 0.5ml/kg/hour (Resuscitation Council, 2005) however, as there is no indication of UOP prior to admission, it is an unreliable indication of organ perfusion and would require hourly monitoring (Jevon, 2010).

Disability

John was rousable to speech (V on AVPU) indicating a decreased level of consciousness (LOC) (Table 4, Appendix A). This could be the result of cerebral hypoxia or hypocapnia. Hypocapnia and hyperventilation are associated with impaired LOC due to constriction of cerebral blood vessels that impairs cerebral perfusion (Porth and Matfin, 2009).

John’s blood sugar is reasonable, however monitoring would be wise in view of the stress response and inhibition of insulin production (Martini et al, 2008).
Rationale for Treatment

John’s assessment demonstrated exhaustion, altered LOC and refractory hypoxaemia therefore indicating the need for mechanical ventilation (Stanley and Tunnicliffe, 2008). Although life saving, mechanical ventilation can exacerbate gas trapping and increase intrathoracic pressures causing barotrauma and reduced preload resulting in hypotension (Leatherman, 2007). Therefore, an understanding of altered lung physiology is required to minimise the risks of mechanical ventilation.

Intubation and Sedation

The management plan is to sedate John using propofol and fentanyl and intubate. Intubation is necessary to facilitate invasive mechanical ventilation whilst sedation is required for toleration of the endotracheal tube and to depress respiratory function for mechanical ventilation (Phipps and Garrard, 2003). Propofol is commonly used in ICU as it enables rapid titration of sedation level (Phipps and Garrard, 2003). Fentanyl is also administered for pain relief and to increase patient comfort. Morphine is avoided in asthmatic patients due to the association with histamine release (Stanley and Tunnicliffe, 2008). Although sedation is required to facilitate mechanical ventilation and effective oxygenation, sedation itself is associated with undesirable consequences such as hypotension and ongoing psychological difficulties such as hallucinations. This can lead to situations akin to post-traumatic distress syndrome which require careful counselling following the critical care experience during follow up (McGloin and McLeod, 2010).

Mechanical Ventilation

The mechanics of normal breathing rely on three key variables: pressure, volume and flow. Mechanical ventilators can be used to replace or supplement spontaneous breathing by controlling any of these variables; hence the classification of pressure, volume or flow controlled ventilation (McGloin and McLeod, 2010). In pressure-cycled ventilation, airway pressure is the controlled variable whilst the volume and flow depend on lung compliance (elasticity of the lungs) and airway resistance. In volume-cycled ventilation, the volume of air is the controlled variable and determines the airway pressure and flow. Choice of ventilation depends on what variable needs to be controlled in order to gain optimal gas control with minimal disturbance to the lung structure (McGloin and McLeod, 2010).

John was placed on pressure-cycled ventilation on the Bilevel mode (Table 5, Appendix A). Bilevel is commonly used in asthmatic patients due to the requirement for tight regulation of intrathoracic pressures and reduced risk of hyperinflation (Marino, 1998). In view of mechanics, breaths are delivered between alternating pressures. According to John’s ventilator settings, flow starts at 1 (PEEP) and increases to the pressure control level of 18 above the PEEP, creating a flow of air into the airways and an increase in volume dependent on John’s lung compliance.

John’s ventilator settings are consistent with the recommendations from professional guidelines. Stather and Stewart (2005) recommend relatively low rates (RR of 11-14) to allow for an increased expiratory time (e.g. an I:E
ratio of 1:3, where the expiratory time is three times the length of the inspiratory time in each breath cycle) achieving tidal volumes of 6-8ml/kg, and extrinsic PEEP (ePEEP) of 0-5cmH₂O. These settings aim to minimise the development of iPEEP and dynamic hyperinflation as a result of airway obstruction (Phipps and Garrard, 2003). There is debate over the role of ePEEP in asthmatic patients. Soni et al (2003) suggest no ePEEP should be used initially as an increase in total PEEP (iPEEP + ePEEP) will worsen gas trapping. However, Stather and Stewart (2005) propose that a minimal amount of ePEEP keeps the alveoli open and reduces the Wₑ.

John’s actual tidal volume (TV) is below the target TV indicating poor lung compliance as a result of dynamic hyperinflation. Reducing the respiratory rate and increasing the I:E ratio would allow longer and more effective expiration of gas, potentially reduce iPEEP and improve lung compliance (Soni et al, 2003). Stather and Stewart (2005) also suggest increased sedation and muscle relaxants can lower the Wₑ, reduce O₂ consumption and CO₂ production. This may improve John’s outcome in view of the hypercapnia.

**Medical Management**
Drug therapies are utilised in conjunction with mechanical ventilation to reverse bronchostriction and airway inflammation (Stanley and Tunnicliffe, 2008). John’s treatment plan considers commencement of IV salbutamol, hydrocortisone, magnesium and nebulised ipratropium bromide.

The British Thoracic Society (BTS) and Scottish Intercollegiate Guidelines Network (SIGN) guidelines (2012) advise the administration of continuous 5mg salbutamol nebulisers, which is consistent with John’s initial treatment. However, Soni et al (2003) suggests IV salbutamol should be considered if there is no response to nebulization as severe airway obstruction could prevent nebulised drug delivery. Conversely, IV beta-agonists can cause lactic acidosis and worsen V/Q mismatching (Stanley and Tunnicliffe, 2008), therefore the risks and benefits must be considered before administration.

Nebulised Ipratropium bromide acts to decrease cholinergic mediated parasympathetic responses i.e. bronchoconstriction. It is supported in current guidelines (BTS and SIGN, 2012) and known to improve pulmonary function when used with beta-agonists (Stoodley et al, 1999)

IV hydrocortisone is administered in view of the inflammatory response. It is known to increase beta-responsiveness of airway smooth muscle, suppress inflammation and decrease mucous secretion (Soni et al, 2003). Corticosteroids do not take effect until 6 to 12 hours after administration (Hazeldine, 2013,) suggesting treatment should have been considered earlier in John’s case given the severity of his attack.

Finally, IV magnesium stimulates bronchodilation through blockage of calcium channels (Hazeldine, 2013). The BTS and SIGN guidelines (2012) advocate its use when inhaled bronchodilator therapy is ineffective.
Implications for Future Nursing Practice

Nurses play a crucial role in the patient’s journey from admission to discharge. The Department of Health (2005) emphasises the need for a transitional approach to ensure patients are restored back to their former physiological and psychological health status. There are a number of implications for future nursing practice that can minimise the physiological and psychological effects imposed on patients whilst in ICU and after discharge.

The role of sedation in ICU has been considered with regards to facilitating mechanical ventilation, however the physiological and psychological impacts have not been addressed. Sedation deprives the brain of stimulation and results in the creation of false memories, which can be extremely traumatic for the patient (McGloin and McLeod, 2010). This can be an on-going problem for patients post discharge as false memories can contribute to other psychological conditions such as post traumatic stress disorder (PTSD). There are also physiological side effects such as bradycardia, inappropriate respiratory depression and deep vein thrombosis, which emphasises the importance of conducting continuous nursing assessments in view of preventing deterioration and patient safety (Shelley, 1998).

Sedation scoring is an invaluable assessment, which should be continuously applied in nursing practice to minimalize the adverse effects of over sedation. Rowe and Fletcher (2008) recommend the Ramsey sedation scale, which provides a quantitative score depending on clinical findings. Use of such scoring systems can guide titration of sedation depending on the individual’s needs. Additionally, It would provide a medium for nurses to accurately record sedation levels, which would improve patient safety and continuity of care in future practice.

Rowe and Fletcher (2008) also suggest that sedation ‘holidays’ should be been considered in order to increase patient comfort and minimise complications. Kress et al (2000) found the duration of mechanical ventilation to be reduced from an average 7.3 days to 4.9 days therefore reducing the risk of ventilation-acquired pneumonia. Kress et al (2000) also noted increased risks of under sedation including self-extubation and increased pain and anxiety. Given the evidence, sedations holidays may improve the patient outcomes in future practice, however this must be conducted with increased monitoring and vigilance from the nurse to ensure patient safety is maintained.

Despite the requirement for the above more ‘technical’ interventions, it is also crucial that essential nursing care is not lost in the ICU. Ensuring hygiene care is upheld, maintaining dignity and adopting a compassionate approach proves to be just as influential on patient outcomes as technical interventions are (Coyer et al, 2007). A balance should therefore be maintained between skills relating to technical equipment/interventions and the caring role of the nurse of observing and relating to their patient and their family’s needs.

Conclusion

This care study has reviewed the pathophysiology of acute asthma and has provided a rationale for the manifestations and assessment findings. Treatment in acute asthma has been discussed and future implications for nursing practice have been considered.
References


Appendix A: Patient scenario

A 25-year-old man (70kg) has been admitted to the intensive care unit, from the emergency department, with acute asthma. On assessment you find:

Airway:
He is rousable to speech and appears exhausted.

Breathing:

<table>
<thead>
<tr>
<th>TABLE 1: BREATHING</th>
<th>Observations</th>
<th>Normal Ranges (Baille, 2005)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiration Rate:</td>
<td>35 breaths/minute</td>
<td>12-20 breaths/minute</td>
</tr>
<tr>
<td>Shallow, wheezing breaths with equal lung expansion</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Use of accessory muscles</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Chest auscultation: Audible wheeze and reduced air entry throughout</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>SpO2: 90%</td>
<td>95-100%</td>
<td></td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>TABLE 2: ARTERIAL BLOOD GAS RESULTS</th>
<th>First ABG</th>
<th>Second ABG (After 1 Hour)</th>
<th>Normal Ranges (Casey, 2013)</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.38</td>
<td>7.33</td>
<td>7.35-7.45</td>
</tr>
<tr>
<td>pO2</td>
<td>8kPa</td>
<td>7kPa</td>
<td>10.7-13.3 kPa</td>
</tr>
<tr>
<td>pCO2</td>
<td>3.5kPa</td>
<td>6.5kPa</td>
<td>4.7-6 kPa</td>
</tr>
<tr>
<td>HCO3</td>
<td>22 mmol/L</td>
<td>18 mmol/L</td>
<td>22-32 mmol/L</td>
</tr>
<tr>
<td>Base Excess</td>
<td>-5</td>
<td>-5</td>
<td>+2 - 2 mmol/L</td>
</tr>
<tr>
<td>Lactate</td>
<td>2.0</td>
<td>2.3</td>
<td>0.5-2.0 mmol/L</td>
</tr>
</tbody>
</table>

Circulation:

<table>
<thead>
<tr>
<th>TABLE 3: CIRCULATION</th>
<th>Observations</th>
<th>Normal Ranges (Levick, 2010)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart Rate:</td>
<td>130 beats/minute</td>
<td>60-80 beats/minute</td>
</tr>
<tr>
<td>Blood Pressure:</td>
<td>110/65 mmHg</td>
<td>90/60 – 140/90 mmHg</td>
</tr>
<tr>
<td>MAP:</td>
<td>80 mmHg</td>
<td>&gt; 60 mmHg</td>
</tr>
<tr>
<td>CVP:</td>
<td>6 mmHg</td>
<td>2-6 mmHg</td>
</tr>
<tr>
<td>Core Temp:</td>
<td>37.8 Degrees Celsius</td>
<td>36-37.2°C</td>
</tr>
<tr>
<td>Capillary Refill:</td>
<td>2 Seconds, cool limbs</td>
<td>&lt; 2 seconds</td>
</tr>
<tr>
<td>Urine Output:</td>
<td>Catheter inserted in A&amp;E and 60mls passed</td>
<td>Minimum 0.5ml/kg/hour</td>
</tr>
</tbody>
</table>
Disability:

<table>
<thead>
<tr>
<th>Observations</th>
<th>Normal Ranges (Baille, 2005)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rousable to speech (V on AVPU)</td>
<td>Alert (A on AVPU)</td>
</tr>
<tr>
<td>Blood sugar: 5 mmol/L</td>
<td>4-7 mmol/L</td>
</tr>
</tbody>
</table>

Exposure:

Nothing unusual noted.

The management plan is to intubate and commence mechanical ventilation on Bilevel settings as follows:

<table>
<thead>
<tr>
<th>TABLE 5: VENTILATOR SETTINGS</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiration Rate (RR): 14</td>
<td>Actual Tidal Volume (TV): 220mls</td>
</tr>
<tr>
<td>Pressure Control (PC): 18</td>
<td>Minute Volume (MV): 3.0 L</td>
</tr>
<tr>
<td>Pressure Support (PS): 18</td>
<td>Airway Pressure (AP): 19cmH₂O</td>
</tr>
<tr>
<td>PEEP: 1</td>
<td>Fraction of Inspired Oxygen (FiO₂): 0.6</td>
</tr>
<tr>
<td>Target Tidal Volume (TV): 350ml (5mls/kg)</td>
<td></td>
</tr>
</tbody>
</table>

Consider commencing IV salbutamol, IV hydrocortisone, single dose of IV magnesium 2mg and commence nebulised ipratropium bromide 0.5mg 4-6 hourly.