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Originally published June 2019

An out-of-hospital perspective on hyperventilation syndrome

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Abstract

Hyperventilation and tachypnoea both involve breathing at an increased rate. There are various causes of hyperventilation and conditions associated with it, including acute and chronic hyperventilation syndrome (HVS). The characteristics of HVS are not well defined. It results from a reduction in carbon dioxide (CO₂) and altered pH in the body from over breathing. Symptoms vary between individuals but usually include altered sensations in the peripherals, nausea and headache. Diagnosing patients with this condition can be difficult; however, modern diagnostic tools include the hyperventilation provocation test, voluntary over-breathing and the Nijmegen questionnaire. There are various patient presentations pre-hospitally and differentiating between potential underlying causes is vital to patient treatment and safety. Treatments vary in nature, depending on the desired effect and scope of practice of the clinician. Some aim to reduce the frequency and intensity of attacks whereas others combat the attack when it strikes. This review briefly narrates some of the treatments available at the basic skill level of a pre-hospital clinician.

A lack of research in this topic has been identified with the recommendation for significant research in the future with a focus on the out-of-hospital environment. Due to controversy within the existing research regarding the definition of HVS, an overview of all sources was conducted to produce a definition. It suggests that HVS is a collection of physical and biochemical reactions from an unnecessarily increased respiratory rate that occurs due to an unknown or benign aetiology which can be triggered by anxiety in the absence of other external factors.

Background

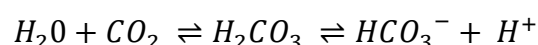
The rate of respiration can vary from normal in a multitude of fashions as a result of different underlying pathologies. Examples include tachypnoea and hyperventilation. Tachypnoea is defined as the respiratory rate of an individual exceeding the expected value for their age group whereas hyperventilation is over-breathing above the required rate of CO₂ elimination resulting in respiratory alkalosis (Whited and Graham, 2019).

Hyperventilation Syndrome (HVS) is one of many conditions that presents with an increased respiratory rate amongst other symptoms. The symptoms of HVS were first recognised during the civil war when DaCosta described the union soldiers as having an “irritable heart” (Evans, 2005). Kerr, Gilebe and Dalton (1938) were the first to use the term HVS and related it to the presentation of hyperventilation associated with anxiety or panic. Due to the ill-defined characteristics of HVS, it has become wildly misused often interchangeably with panic or anxiety disorders.

Defining HVS is somewhat controversial due to the lack of research on the subject. Schildkrout (2005) defines hyperventilation as a type of panic disorder whereas Lum (1975) claims it to be the cause of hyperventilation where no physiological cause can be found. Folgering (1999) defined HVS as the minute ventilation of an individual exceeding their metabolic demands. However, current trends suggest that an alternative term such as behavioural breathlessness or psychogenic dyspnoea may be more appropriate (Kern, 2016). Derbyshire Community Health Services define HVS as a collection of physical and emotional symptoms that arise from breathing too quickly (Derbyshire Community Health Services NHS, 2012). HVS is thought to have both a chronic and an acute form. Waites (1978) believed that the acute form accounted for 1% of patients with this syndrome. Saisch, Wessely and Gardner (1997) believed the label of HVS in a patient with hypocapnia is not beneficial to an individual’s recovery and therefore he aims to abandon the term. It was thought that hyperventilation occurs because of a complex interaction between multiple organ systems and that the label of HVS prevents any further investigation in to the original cause.

Pathophysiology

Blood acidity, measured in pH, is determined by the concentration of hydrogen ions (H^+) present. Blood is comprised of 55% blood plasma, of which, at least 90% is water (IQWIG, 2015). When dissolved in water, CO_2 forms an equilibrium in which the forward reaction produces carbonic acid (H_2CO_3). Once formed, (H_2CO_3) forms a further equilibrium through dissociation - producing bicarbonate (HCO_3^-) and hydrogen (H^+) ions. This equilibrium is the basis for homeostatic balance of blood pH.



Both oxygen (O_2) and CO_2 can chemically bind to haemoglobin (Hb) for transportation around the body (Marieb, 2016). Hb also acts as a buffer, collecting excess H^+ ions which would otherwise cause acidosis.

Figure 1 shows the oxygen-haemoglobin dissociation curve - a sigmoid curve - and the variables which can cause left and right shift; influencing affinity at different partial pressures (Murray and Nadel, 2016). Through hyperventilation, and the resulting hypocapnia, the Bohr effect describes how the changes in haemoglobins affinity for O_2 are influenced by CO_2/H^+ . Hyperventilation leads to excessive exhalation of CO_2 , and a left shift in the oxygen-haemoglobin dissociation curve resulting in a respiratory alkalosis (Patel and Cooper, 2018). This in turn leads to an impaired release of O_2 within the peripheral tissues, causing the irregular firing of nerves and thus paraesthesia.

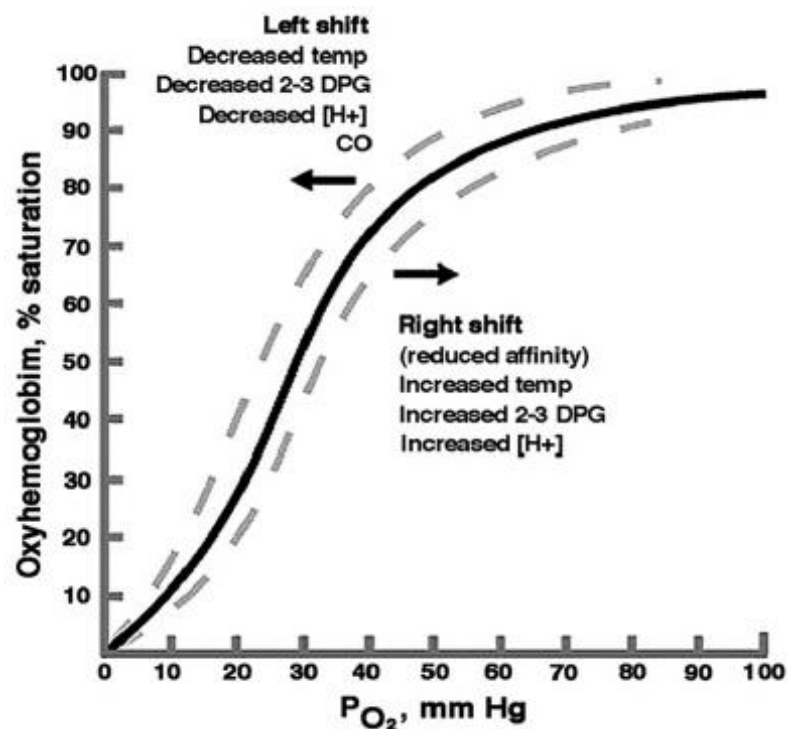


Figure 1. Oxygen-haemoglobin disassociation curve

Nagalakshimi et al (2016)

Due to hypocapnia during hyperventilation, the vasoconstrictive effects of CO_2 are reduced (Cipolla and Rafael, 2009). This leads to a relative hypotension and an estimated reduction in CPP of 30-40% which is more noticeable in patients that suffer from autonomic failure (Pickering, 2000). This contributes to the most frequently

reported symptom of dizziness in patients presenting with hyperventilation (Evans, 2005). To compensate for the reduction in blood pressure; heart rate and stroke volume increase (Marieb, 2016). Tachycardia is a frequently experienced symptom associated with both anxiety disorders and HVS and often presents as palpitations.

Presentation

The signs and symptoms of HVS vary largely between patients, with some being experienced frequently whilst others rarely present. These symptoms are thought to be caused by the metabolic alkalosis and alterations in blood flow as a result of hyperventilation. A wide range of symptoms can be noted across the systems of the body (Table 1).

Table 1. Common signs and symptoms of hyperventilation syndrome

General	Fatigue, nausea, diaphoresis
Neurological	Headache, visual disturbances, dizziness, paraesthesia, syncope
Cardiovascular	Palpitations, tachycardia, chest pain
Gastrointestinal	Dysphagia, epigastric discomfort, belching, dry mouth, flatulence
Psychological	Altered concentration, anxiety
Respiratory	Tachypnoea, tightness of chest
Musculoskeletal	Tremors, weakness, tetany, muscle pains

Evans (2005), Caroline (2016), Clarke and Townsend (2016), Wilson (2018)

Despite there being very little consistency regarding the diagnosis and definition of HVS, the associated symptoms are often similar. Most commonly experienced is the feeling of dizziness or light-headedness. Others experience paraesthesia in their extremities and muscle cramps, with seizures being reported in the most extreme cases. Both peripheral and central tetany are also recorded in a small minority of patients however, this was rarely experienced. Schildkrout (2005) reported derealisation and depersonalisation; however, no other sources reported this as a symptom. The dizziness that is often reported is likely to be due to a combination of the reduction in blood pressure and CPP. When this occurs, the body's blood supply

centralises to ensure O₂ delivery to essential organs (Marieb, 2016). Assuming there is no underlying pathological cause, these symptoms resolve upon resuming eupnoeic breathing once the excess in exhalation of CO₂ has been reversed (Evans, 2005). The sensation of a dry mouth occurs as a result of the rapid breathing through the mouth (Morton, 1990).

Diagnosis

HVS has both chronic and acute presentations. The diagnosis of the chronic form is primarily conducted by the patient's GP after extensive testing. The out-of-hospital setting encounters the acute form more significantly.

There is a lack of prognostic criteria for HVS as there is much controversy amongst the medical community as to which procedures allows for an adequate diagnosis. One diagnostic method proposed for chronic HVS is the hyperventilation provocation test. This involves the patient voluntarily over-breathing for a period significant enough to produce hypocapnia. At this point, if the previously experienced symptoms return then the test is determined a positive result. Hornsveld and Garssen (1996) found that the hyperventilation provocation test gained a positive result in 66% of patients and 60% in a control group. Due to the low specificity of this test, it has largely been discounted, with the scientific background surrounding the hyperventilation provocation test highlighting its flaws. The theory that symptoms will be reproduced upon voluntary hyperventilation is supported by the physiology of over-breathing regardless of diagnosis. This test was also discounted as a diagnostic tool when Howell (1997) found that the reproduction of symptoms was not reliant on the fall in CO₂ thus rendering the test invalid.

An alternative method of diagnosing chronic HVS is the Nijmegen questionnaire. It is a list of symptoms relating to respiratory distress that are scored from zero to four regarding the frequency at which they are experienced. A score above 23 out of a maximum 64 points indicates HVS. This questionnaire has been found to differentiate between HVS sufferers, with non-HVS sufferers with much higher accuracy. Dixhoorn and Duivernvoorden (1985) found a sensitivity of 91% and a specificity of 95% in relation to the clinical diagnosis of chronic HVS. D'Alba et al (2014) conducted similar

research comparing paediatrics with and without asthma using the Nijmegen questionnaire to assess for HVS, and found it to be an accurate tool.

One of the more rudimentary diagnostic methods for chronic HVS revolved around the exclusion of any organic (physiologic) cause (Lum, 1975). This is prevalent for the diagnosis of both the chronic and acute forms and can be conducted in the out-of-hospital setting. This involves assessing all organ systems in-depth for any pathologies that may result in hyperventilation. Only when all other differentials have been discounted can HVS be diagnosed. Often when no cause was found, these symptoms were misdiagnosed as an anxiety related issue. As a result, individuals were often provided with no relevant treatment or guidance on how to manage their condition. This procedure has generally been discounted as a cause of dysfunctional breathing was not discovered. However, this technique is still used in the acute situation. When presented with a patient in the pre-hospital field who is experiencing some of the aforementioned symptoms, it is essential to establish the cause by excluding other differentials. Other differentials that present themselves with hyperventilation or tachypnoea include infection, pulmonary embolus (PE) and diabetic ketoacidosis (DKA). If the increased respiratory rate is caused by an underlying pathology in which the body is attempting to compensate, then reversing the abnormal breathing pattern can cause further harm. Hence the importance of discounting other causes prior to treatment.

The characteristic, low levels of CO₂ in a hyperventilating individual can be measured using capnography. Capnography is a non-invasive method used to measure CO₂ and can provide information on ventilation and perfusion (Long, et al., 2017). It typically produces a quantitative value and a graphic representation of CO₂ levels (Brandt, 2010). This quantitative value is referred to as end-tidal CO₂ (ETCO₂) and is the level of CO₂ exhaled.

During hyperventilation the capnography waveform will be at an increased frequency in comparison to an individual breathing within the expected range. The amplitude of the waveform which represents the ETCO₂ is also reduced in comparison to eupnoeic breathing. When presented with hyperventilation, capnography can be used alongside patient assessment however it is not a stand-alone test.

Treatment

Once the cause of hyperventilation has been determined as HVS, there are various treatments which have been formalised with the intention of reducing the frequency and severity of hyperventilation attacks (Laurino et al, 2012). These treatments vary from preventative measures to techniques to use when an attack occurs. Respiratory rehabilitation has been used to educate an individual on methods to reduce their breathing rate. This involves techniques such as diaphragmatic breathing or hypoventilation to combat over-breathing (Derbyshire Community Health Services NHS, 2012). Diaphragmatic or deep breathing helps reverse any deviations from eupnoeic breathing. Often when an individual hyperventilates, they utilise accessory muscles in their upper chest. This is an ineffective method of breathing and results in muscle tension which can increase the overall feeling of anxiety, a potential cause of the attack. Ensuring the diaphragm is engaged in the most efficient manner can distract a patient from any potential emotional triggers. It also actively ensures deeper and slower breaths are taken, combatting the decline in CO₂.

Swift, Campbell and McKown (1988) found that inhalation via the nose and exhalation via the mouth can also help reduce the effects of hyperventilation. The nose acts as a filtration system and both moistens and humidifies air prior to contact with the lungs. When inhaling through the nose, the lower lobes of the lungs are stimulated to a greater degree than in comparison with the mouth, this increases the volume of the lungs and overall amount of O₂ distributed to the body. The lower lobes also contain a large quantity of parasympathetic receptors which when stimulated will help reduce excitability thus lowering the desire to inhale. In contrast, the upper lobes of the lungs are stimulated by mouth breathing which stimulates a sympathetic response. This encourages hyperventilation and can induce the fight or flight response.

An additional acute treatment for HVS includes the well-known practice of breathing into a paper bag. This results in any exhaled CO₂ being 'stored', thus increasing the amount readily available to inhale. Brouhard (2017) thought that this rebreathing of the air we exhale increases CO₂ intake thus correcting the imbalance created by hyperventilation. This should reduce the metabolic alkalinity and therefore eradicate the symptoms. This technique must not be performed for any prolonged period or in

an individual who is not suffering from hyperventilation as a result of anxiety as it could cause the opposite of the desired effect and increase levels of CO₂ above the average limits. This technique for correcting ineffective breathing techniques must only occur after the exclusion of any pathological causes. If a patient is hyperventilating to compensate for metabolic acidosis from an underlying cause, then correcting this can have serious repercussions.

The Association of Ambulance Chief Executives (AACE) produced an algorithm for the assessment and management of HVS that begins with assessing the patient's baseline observations and correcting any of these as the issues are identified (AACE, 2016). A rapid transfer must be made when a patient is experiencing an attack with any time critical features such as cyanosis or reduced level of consciousness. This transfer to hospital must be performed when it is their first hyperventilation episode or if their "symptoms have not settled or re-occur within 10 minutes" (AACE, 2016). When the patient has a personalised care plan and can be left in the care of a responsible adult, the option for non-conveyance can be considered. The recommended treatment for an acute episode involves coaching the patient's breathing and providing reassurance. When possible, it is advised to remove the patient from the source of any potential anxiety, with supplementary O₂ only being recommended with accompanied hypoxaemia.

Emergency pre-hospital care

The lack of published research into HVS, particularly within the pre-hospital setting, may be due to the belief that it will have very little impact. Clinicians currently utilise the required skills in assessing a patient and coaching breathing that would be required in the management of acute HVS. Being aware of the alternative differentials that present in a similar way that have a more dangerous pathology may be more important. For example, pulmonary embolism, severe infection or an aspirin overdose all present with an increased respiratory rate thus producing the biochemical symptoms including paraesthesia and dizziness. There are current guidelines produced by AACE that suggests the most appropriate method to use in assessing this group of patients within the out-of-hospital setting (AACE, 2016).

Medical attention is sought by 32% of panic attack sufferers which results in extensive and expensive examinations. (Coley, 2007). Due to the variety of symptoms experienced including chest pain and paraesthesia HVS patients often seek help for fear their symptoms may represent a more sinister pathology. Wilson et al (2017) found a pre-hospital diagnosis ratio that was near perfect with paramedics and emergency medical technicians. With this high specificity and sensitivity surrounding the acute diagnosis of HVS in the pre-hospital setting, the option for non-conveyance is pertinent. This would help reduce the economic burden of hospitals as well as the anxiety of patients.

Is HVS dangerous?

Despite many individuals that suffer an HVS episode fearing a sinister pathology and seeking medical attention, general sources concur that an attack of HVS is unlikely to cause any significant or chronic damage. Although understandably distressing and overwhelming in the moment, the associated symptoms are in response to hyperventilation. Once eupnoeic breathing recommences, and the CO₂ debt has been reversed, all symptoms resolve. Although wildly different, there is a consistency within the nature of the experienced symptoms that can be explained by the biochemical alterations that result from over breathing.

Discussion

There is no overall conclusion that can be gained from the current research to suggest the ideal diagnostic tool and treatment methods as the exact cause of HVS is still unknown. After reviewing the current research, it may be that there is no exact cause for HVS. Evidence of HVS often originates from anxiety related disorders. It delves into the physical reasons as to why an individual would experience these symptoms, including hyperventilation. However, no research considers the possibility that these ideas are interlinked. Anxiety and hyperventilation are shown to be related and it is often thought that the anxiety causes the hyperventilation. However, it may be that rather than a cause-effect relationship, this is more correlational. Hyperventilation occurs, whether it be of a known pathology or of no apparent cause, but this triggers anxiety. This cycle then continues until the individual is in an intense state where they

are no longer able to control either the hyperventilation or the anxiety which would be manageable separately. When occurring simultaneously, the overall result is so overwhelming for the body that it enters a symptomatic loop.

There are significant gaps within the research surrounding HVS with controversy between opinions. Additional research is required to fill these gaps, with an emphasis on the out-of-hospital relevance. As the scope of pre-hospital medicine expands, the degree of understanding behind pathologies needs to increase and mirror these changes which is not possible without additional research.

Conclusion

Different sources have varying definitions for HVS which has resulted in poor understanding of the condition and the term is often misused. The collation of information to provide a conclusive definition may improve understanding within the medical and non-medical communities. Hyperventilation syndrome is a collection of physical and biochemical reactions to an unnecessarily increased respiratory rate that occurs due to an unknown or benign aetiology, often triggered by anxiety in the absence of other external factors. Symptoms include dizziness, headache and anxiety.

A significant lack of research surrounding the cause of hyperventilation syndrome has resulted in controversy regarding the ideal diagnostic tool and treatments. It is still unknown as to whether HVS is as a result of an underlying pathology or is anxiety related. More research is essential to allow the creation of these tools to improve treatment of patients suffering from both acute and chronic HVS.

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