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Hypovolaemic Shock: assessment, pathophysiology and nursing care.

Abstract

Hypovolaemia leads to a number of key physiological responses which require systematic assessment and interpretation. This article focuses on a case study involving a patient with Parkinson's disease who became hypovolaemic following a fall at home, to illustrate the impact of hypovolaemia and current recommendations regarding fluid resuscitation. ABCDE assessment is considered as well as arterial blood gas analysis and interpretation is explained in light of the patient scenario.

Key Words

Key Points

- Hypovolaemia can lead to a number of systemic responses
- Careful and systematic interpretation of arterial blood gases is an important element to assessing the impact of hypovolaemia
- Early, balanced fluid resuscitation is an essential aspect of patient management and can prevent patient deterioration

Hypovolaemic Shock: assessment, pathophysiology and nursing care

Hypovolaemic shock refers to the serious clinical condition of acute circulatory failure. It is characterised by loss of 15% or more of intravascular circulating volume, leading to inadequate tissue perfusion and potential tissue necrosis (Zelman et al, 2011; Porth, 2015). There are a number of possible causes of hypovolaemia including severe external haemorrhaging or internal fluid shifts as a result from dehydration (Garretson and Malberti, 2007). This article focuses on a case study, Clive (pseudonym as per NMC requirements (NMC, 2015), who was admitted to critical care from the emergency department (ED). He has Parkinson's disease and fell at home. His case study will illustrate the effects of hypovoalemia and to offer a rationale for the assessment findings, underlying pathophysiology and associated patient care. It will also consider why he may have fallen and how his Parkinson's disease can be effectively managed in a critical care situation.

Case study

Clive was an 80 year old gentleman who was admitted to critical care following a fall at home three days previously. As he was unable to get off the floor following his fall, he was dehydrated and confused, with the risk of developing acute kidney injury. He has Parkinson's disease (PD) and he lives on his own although his family live nearby and he is normally fully independent. In the emergency department, as he had not had his anti-Parkinsons medication for 3 days, he was showing signs of associated motor impairment (stiffness and rigidity) which was noted on his secondary survey. He therefore needed dopamine agonist medication to improve his motor function. As his swallow was not impaired, his usual Parkinson's medication was administered which comprised of Co-careldopa 100/25 mg x 2 orally.

Assessment

On admission, Clive was assessed using the ABCDE (airway, breathing, circulation, disability, and environment) framework developed by the Resuscitation Council (2005). This allowed for a systematic approach to Clive's assessment with life threatening changes identified in a prioritised manner (Jevon, 2010). This allowed for proactive interventions and treatments to avoid further deterioration. THE ABCDE approach is a widely accepted process to assessing

patients who are either already critically ill, or those who face the potential risk of deteriorating. Studies have shown that this framework is particularly useful in an emergency setting and not only serves to provide life-saving care and treatment, but that it also improves the performance of the multi-disciplinary team involved in patients' care (Thim et al, 2012).

Airway:

On assessment Clive's airway did not appear to be compromised as he was able to respond verbally to questions. However as he appeared exhausted and was hypotensive, ongoing airway assessment was required in case his level of consciousness deteriorated further.

Breathing

Assessment of Clive's respiratory function involved counting his respiratory rate, assessing the depth of inspiration and observing for symmetrical chest expansion (Jevon, 2010). On assessment, the following was found:

- Respiration rate: 26 breaths/minute.

- He was dyspnoeic.

- Shallow breaths and use of accessory muscles.

- No signs of central cyanosis

- Symmetrical chest movement with normal rhythm.

- SpO2: 95% on 60% oxygen via a venturi mask

Clive was tachypnoeic with a respiratory rate of 26 breaths per minute. His shallow breathing and use of accessory muscles suggested an increased work of breathing. Clive was self-ventilating on 60% oxygen with a peripheral oxygen saturation (SpO_2) of 95%. Although Clive's recorded SpO_2 was within national guideline targets (SpO_2 94-98%) and no signs of central cyanosis were present, clinical judgement proposes a SpO_2 of 95% is low for someone receiving supplemental 60% oxygen therapy (British Thoracic Society, 2008).

Circulation

Assessment of Clive's circulatory condition provided further detail regarding the cause of his condition. Assessment of circulation involves assessing pulses, blood pressure and tissue/ organ perfusion. Clive's assessment showed:

- Heart rate   130 BPM
- Blood pressure   100/40 mmHg
- Mean arterial pressure   60 mmHg
- Central venous pressure   2 mmHg
- Core temp   36.5 ºC
- His limbs felt cool and his capillary refill was 3 seconds
- He had a catheter inserted in the emergency department and there was 20 ml in the bag

Results from the assessment revealed that Clive was tachycardic, hypotensive with a decreased mean arterial pressure (MAP) and a decreased central venous pressure (CVP), suggesting he was in shock. Clive's cool peripheries, prolonged capillary refill time and oliguria suggested that he had decreased tissue perfusion (Clarke and Ketchell, 2011). With a temperature of 36.5 °C, Clive was apyrexial.

As he was showing signs of shock, cardiac output monitoring was commenced on admission to the critical care unit and the following readings were obtained:

- Systemic vascular resistance index   2400 dynes · sec/cm^5/m^2
- Cardiac index   1.3 l/min/ m^2
- Stroke volume   30 ml

The results from Clive's invasive cardiac output monitoring revealed an increased systemic vascular resistance index (SVRI) (normal values: 1,360- 2,200 dynes · sec/cm^5/m^2) and a decreased cardiac index (CI) (normal value: 2.6–4.2 L/min/m^2) and stroke volume (SV) (normal value 60- 80 ml). A reduced SVRI is often presented as a classic sign of sepsis, in response to widespread vasodilation which would contribute to warm, flushed peripheries. However, Clive's raised SVRI suggested he was vasoconstricted and a reduced CI and SV signified a low blood volume as indicated also by the reduced CVP. Therefore, he did not

appear to have developed sepsis or systemic inflammatory response syndrome (SIRS) (Aitken et al, 2015). In view of the background to his admission and that there are no clinical signs of haemorrhage or reported history of a cardiac event, the findings indicated that Clive was experiencing hypovolaemic shock as opposed to haemorrhagic or cardiogenic shock (Clarke and Ketchell, 2011).

The oliguria suggested he had developed acute kidney injury. This could be due to fluid responsive renal failure which can occur when there is reduced renal perfusion. However, in consideration of the prolonged period of time Clive spent lying stationary, intra-renal failure may have developed due to rhabdomyolysis.

Disability

The disability stage of ABCDE focuses on assessment of any neurological alteration and possible causes. Assessment of Clive's neurological state identified that he was:

- Rousable to speech on the AVPU scale.

- He was confused to time, place and person.

- He had regained his motor function following administration of his dopamine agonist.

- Blood sugar levels: 3 mmol/l.

These results demonstrated signs of neurological dysfunction which was of concern as Clive fell at home and could possibly have sustained a head injury. Therefore, he was assessed using the Glasgow coma score (GCS) which showed: eyes: open to speech (3), verbal response: confused (4); motor response: obeying commands (6). His pupils were equal in size (3 mm) and reacting briskly. He had a mild weakness in each limb on assessment of limb power. To exclude any cerebral pathology, such as a subdural haemotoma, a computerised tomography (CT) scan was performed which showed no sign of cerebral haemorrhage or raised intracranial pressure.

Exposure

The last part of ABCDE assessment is focused on assessing for any other signs of injury that have not be noted. Clive's assessment identified:

- An area of redness on his right hip where he had been lying on the floor.

As part of Clive's on-going assessment, an arterial blood gas (ABG) was taken so that vital information regarding his internal respiratory and metabolic status was obtained. This will demonstrate Clive's ability to maintain homeostasis and help explain his respiratory assessment findings (Larkin and Zimmanck, 2015).

Arterial blood gases (ABGs)

For the critically ill patient timely interpretation of appropriate investigations will lead to essential treatment. Arterial blood gases (ABGs) offer useful information about gas exchange, the metabolic status of the patient and the body's ability to maintain a normal acid: base balance. Normal ranges for each parameter are:

pH	7.35- 7.45
pO_2	10- 13.3 kPa
pCO_2	4.6- 6.0 kPa
HCO_3^-	22-26 mmol/l
Base Excess	-2- +2

ABGs reveal the pH (normal range 7.35-7.45) which indicates the concentration of hydrogen ions (H+). Changes in pH can be due to respiratory or metabolic causes. If the alteration in pH is due to respiratory causes, then this will be reflected within the partial pressure of carbon dioxide (pCO2) (normal range 4.6-6.0 kPa), with an increase in pCO2 leading to an acidosis (and a decrease leading to an alkalosis). The partial pressure of oxygen (pO2) (normal range 10-13.3 kPa) is also obtained and therefore any hypoxaemia can be identified but the pO2 does not alter the pH. The measurements of bicarbonate ions (HCO_3^-) (normal range 22-26 mmol/L) and base excess (normal range -2 to +2) identify whether any change in pH is due to metabolic causes; a decrease in HCO_3^- and base excess are associated with a metabolic acidosis whereas an increase in these values is suggestive of a metabolic alkalosis. Careful and systematic interpretation of ABGs therefore assists in differentiating between metabolic and respiratory causes of changes in pH and also allow for identification of

whether any compensation is happening. Through assessment of these measurements, suitable management can be identified (McLeod, 2016).

pH control

As a by-product of many metabolic processes, H+ ions are continuously generated. A high concentration of H+ constitutes for an acidic environment equalling to a low pH, which can have detrimental effects on cellular activity. In health, the normal range of pH is maintained by the balancing of acid and base via three mechanisms: buffering, the respiratory response and the renal response (McLeod, 2016):

1) Buffers are able to rapidly and reversibly combine with H+, neutralising its effect thus maintaining a relatively constant pH. Bicarbonate, protein, phosphate and haemoglobin form the key buffers and the availability of buffers to combine with hydrogen is reflected within the base excess reading on the ABG. A negative reading implies that buffers are depleted whereas a positive reading suggests that there is available buffer. (Porth, 2015)

2) The respiratory response involves HCO_3^- and H^+ combining to form carbonic acid (H_2CO_3). This molecule can easily dissociate into water (H_2O) and carbon dioxide (CO_2), both of which can be expelled via the lungs:

$$HCO_3^- + H \longrightarrow H_2CO_3 \longrightarrow H_2O + CO_2$$

Increased levels of H^+ will additionally be detected by chemoreceptors in the brain stem and carotid and aortic arteries. Chemoreceptors signal the respiratory centre in the medulla oblongata which initiates an increase in pulmonary ventilation, aiming to breathe out hydrogen in the form of carbon dioxide (McLeod, 2016).

3) The renal response involves the renal tubular cells forming HCO_3^- from the combination of CO_2 and H_2O within the tubular cells. The HCO_3^- which is formed when H_2CO_3 dissociates into HCO_3^- and H^+, passes into the blood whereas the H^+ is secreted into the filtrate within the renal tubules. The movement of the HCO_3^- into the blood stream helps to correct the pH.

Interpretation of ABGs

Clive's arterial blood gases were:

• pH	7.33
• pO_2	9 kPa
• pCO_2	3.8 kPa
• HCO^-_3	18 mmol/l
• BE	-5
• Lactate	2.4 mmol/l

In comparison to the normal values of ABGs, Clive's pH, pO_2, pCO_2, HCO^-_3 and base excess are all reduced. This indicates that he had a primary metabolic acidosis (low pH, low HCO^-_3 and reduced base excess) with partial respiratory compensation (low pCO_2).

Clive's low pH of 7.33 identified that he was acidotic. In order to establish the primary cause of Clive's acidosis, the pCO_2, HCO^-_3 and base excess levels were assessed. As both the HCO^-_3 and base excess were low, there was a metabolic acidosis present as opposed to a respiratory acidosis which would have been caused by an increased pCO_2.

Conversely, Clive's pCO_2 was low. The Henderson-Hasselbalch equation describes the normal relationship between HCO^-_3 and pCO_2 as inverse, however Clive's readings showed that both variables are low (McLeod, 2016). In a compensatory response to the high levels of H^+, Clive's respiratory centre was stimulated by the increased hydrogen within his blood. This led to an increase in respiratory rate and therefore the tachypnoea which was observed, was caused by the respiratory response as previously outlined. As the pH of Clive's arterial blood had not fully returned to a normal range of 7.35- 7.45, he was in a state of metabolic acidosis with partial respiratory compensation (McLeod, 2016).

Clive's pO_2 was low and therefore he had a hypoxaemia which was suggested also by the low SpO_2. Additional tests revealed an elevated level of lactate of 2.4 mmol/L, normal levels are below 2mmol/L. Lactate is a by-product of anaerobic cellular respiration, which occurs when there is inadequate oxygen delivery or perfusion to the cells (Bench and Brown, 2011). This suggested that Clive's acidotic state was due to possible shock or sepsis (McLeod,

2016). To successfully treat a metabolic acidosis the underlying cause must be identified and treated.

Underlying physiology/ pathophysiology

On assessment, Clive was showing signs of a compensatory response to the hypovolaemia shock.

Shock Mechanisms

Starling's law of the heart states that there is a direct relationship between the preload (the volume of blood within the ventricle stretching the myocardium fibres) and contractility (the forcefulness of the myocardial contraction). This will affect the stroke volume which is the volume of blood ejected by the ventricle in one contraction and ultimately the cardiac output which is HR x SV and equates to the amount of blood ejected in one minute (Aitken et al, 2015). A depleted circulating volume reduced Clive's preload, resulting in a decreased SV and therefore a low CI, which is his cardiac output in relation to his body mass index. This was reflected within his low systolic blood pressure (BP) (Jevon and Ewens, 2012). Although deemed sufficient for some patients, a systolic BP of 100 mmHg is not expected for an 80 year old man. However, in comparison to his low CI, Clive's BP appeared to be sufficient. This suggested that Clive was in a compensated stage of hypovolaemic shock (Aitken et al, 2015).

Sympathetic Nervous System response

In response to a drop in BP, the sympathetic nervous system initiates a compensatory response, aiming to maintain a sufficient CI. Baroreceptors in the aortic arch and carotid sinus detect a decreased circulating volume and instigate a message to the cardiac vasomotor centre, located in the medulla oblongata (Clarke and Ketchell, 2011). Here, stimulation of sympathetic nerves initiates the release of catecholamines (epinephrine and norepinephrine) from the adrenal glands, resulting in vasoconstriction and increased myocardial contractility. This response attempts to maintain organ perfusion by improving cardiac output (Aitken et al, 2015). Therefore, BP= CO x SVR. Clive's tachycardia and signs of

poor peripheral perfusion, which contributed to the development of metabolic acidosis, reiterate the physiological process of hypovolaemic shock.

Renin angiotensin aldosterone system (RAAS)

As an additional compensatory measure, the kidneys stimulate the homeostatic mechanism which is the renin- angiotensin- aldosterone system (RAAS). A decreased renal arterial pressure, caused by low blood volume, leads to a reduced filtrate flow within the renal nephron which is detected by the juxtaglomerular apparatus and renin is released. Renin modifies circulating angiotensinogen into angiotensin I. Once in the lungs and in the presence of angiotensin converting enzyme, angiotensin I is converted into the active angiotensin II, which causes arterial vasoconstriction. This increases systemic blood pressure, and therefore glomerular filtration pressure (Porth, 2015).

Simultaneously, circulating angiotensin II stimulates the secretion of aldosterone, from the adrenal cortex. Aldosterone promotes the reabsorption of sodium from the renal tubules, thus increasing the intracapillary oncotic pressure causing retention of water. Additionally, the increased osmolality of the blood stimulates the secretion of anti-diuretic hormone (ADH) from the posterior pituitary gland. ADH also promotes water reabsorption by increasing the permeability of the renal tubules (Jevon and Ewens, 2012). Both mechanisms aim to improve circulatory function by increasing circulating volume however, over a period of time these mechanisms reduced Clive's glomerular filtration rate resulting in oliguria. Based on Clive's weight his normal urine output should be 30mls per hour, but only 20mls was reported. This indicated that Clive was developing acute kidney injury. (Creed and Spiers, 2010).

Cerebral perfusion

Clive was showing signs of confusion on admission, although he was responsive to verbal stimulation. This suggested that he had a reduced level of consciousness which was likely to be due to a reduction in cerebral perfusion. The brain requires a cerebral blood flow of 50ml/100g/min to meet the metabolic requirements of neurones, and utilises around 3-5 ml O_2 /min/100g and 5 mg glucose/min/100g. Clive had a low blood pressure and was hypoglycaemic- neurones cannot store glucose and have limited ability to undergo

anaerobic respiration as there no local supply of glycogen to convert into glucose. Therefore neurones rely on a continuous supply of both oxygen and glucose; should the supply be inadequate, then alterations in level of consciousness is observed as in Clive's situation.

Rhabdomyolysis

Clive's profile along with the noted erythema on his right hip indicated that he may have been immobile for an extensive period of time: this suggested the possibility of the development of rhabdomyolysis. Alongside Clive's underlying pathophysiology, rhabdomyolysis can place his metabolic state and renal function at further risk.

Prolonged compression on an isolated area can result in localised ischaemia, due to impaired perfusion to skeletal muscles (Baaijens et al. 2015). The ischaemic environment leads to anaerobic cellular respiration, resulting in depleted levels of ATP within myocytes and production of lactate; constituting for further metabolic acidosis. Low levels of ATP hinder the action of calcium ATPase pumps leading to increased levels of calcium within the sarcoplasm, which initiates myofibril contraction. A constant state of contraction exhausts all energy resources resulting in lysis of the myocyte and over time skeletal muscle necrosis (Williams and Thorpe, 2014).

Disintegration of the myocyte results in a release of the intracellular contents, including numerous ions and the protein myoglobin into circulation. Once in circulation myoglobin is rapidly filtered by the glomerulus, leading to myoglobinuria manifesting as dark red/brown coloured urine (Porth, 2015). Within acidic environments, as found in the renal tubules myoglobin is nephrotoxic causing damage and obstruction (Williams and Thorpe, 2014). For Clive this additional insult would further exacerbate the development of acute kidney injury. Alongside clinical manifestations such as localised erythema and muscle weakness further laboratory investigation can provide a definitive diagnosis of rhabdomyolysis for Clive. High elevated levels of creatine kinase (> ten times normal) indicates a breakdown of myocytes and is used as a primary indicator for the condition. Metabolic acidosis and an increased level of serum electrolytes, such as potassium further support the diagnosis (Aitken et al, 2015)

Patient Management

Hypovolaemia

Treatment of hypovolaemic shock focuses on regaining adequate tissue perfusion via restoration of fluid volume and BP. Consequently, fluid resuscitation will improve oxygen delivery to cells, enabling aerobic respiration, which in-turn will help restore the acid-base disparity (Garretson and Malberti, 2007). Guidelines proposed by the National Institute for Health Care and Excellence (NICE) (2013) recommend a fluid challenge of a 500ml crystalloid bolus, over 15 minutes for any patient suspected of hypovolaemia. Based on Clive's physiological profile, his age was suggestive of potential reduced cardiovascular function. Administration of too much fluid, too quickly increases the risk of iatrogenic chronic heart failure, as the heart is unable to cope with the sudden rise in blood volume (Creed and Spires, 2010). Therefore, Clive was prescribed boluses of 250ml crystalloid to assess for fluid responsiveness: boluses should be given until there is no further response and at that point, fluid resuscitation should not continue as preload should be adequate (Aitken et al, 2015) Fluid resuscitation using crystalloids provides a transient intravascular expansion and further replaces accompanying fluid loss in interstitial and intracellular spaces. Hypertonic colloids are normally only indicated to replace large volumes of fluid loss, or to restore low haemoglobin levels, due to their higher cost and associated risks such as alteration in clotting (Garretson and Malberti, 2007). The CRISTAL trial (Annane et al, 2013) did not show any significant different in 28 day mortality rates in hypovolaemic patients in intensive care who received either crystalloids or colloids.

Sequential monitoring of vital signs and cardiac output are imperative when managing a patient receiving a fluid challenge. Fluid boluses should be repeated until a rise of 3mmHg or more is seen in the CVP, 5 to 10 minutes after the bolus has been fully administered (Aitken et al, 2015). The CVP indicates the amount of blood returning to the heart and thus cardiac output, therefore it is a useful guide when observing the effect of a fluid challenge (Porth, 2015). However, CVP readings should not be used independently, as CVP can also be sensitive to vasoconstriction. Vincent and Weil (2006) recommended using the CVP as a

safety guide to prevent hypervolaemia, using quantitative clinical goals such as, blood pressure, heart rate and MAP to help observe an increased peripheral perfusion.

For safe and effective administration of IV fluids NICE (2013) recommend using the principles of the five R's: Resuscitation, Routine maintenance, Replacement, Redistribution and Reassessment. Once Clive appears haemodynamically stable, after resuscitation, treatment should focus on replacement of any electrolyte deficits (Sherratt, 2014). Therefore, a maintenance plan including 500ml Hartmanns solution and 500ml 5% dextrose was prescribed for Clive. The British consensus guidelines on intravenous fluid therapy for adult surgical patients (Powell-Tuck et al. 2011) endorse Hartmanns solution over 0.9% saline as this can contribute to hyperchloraemic acidosis, particularly in the elderly. The isotonic nature of crystalloids such as Hartmanns solution, result in distribution within the interstitial space, ensuring adequate replacement of lost electrolytes, such as potassium and sodium (Dougherty and Lamb, 2008). To help distribute fluid evenly amongst the body's compartments, the British National Formulary (British Medical Association and Royal Pharmaceutical Society, 2016), advise for an equal infusion of 500ml 5% dextrose, to aid intracellular hydration. The metabolism of glucose, within dextrose, lowers the osmolarity of extracellular fluid creating a hypotonic environment, causing a net movement of fluid into the cells moving down its pressure gradient (Porth, 2015). Care though needs to be taken if there is any suggestion of altered intracerebral pressure (ICP) as dextrose containing solutions can increase ICP. As Clive's CT scan did not show any signs of raised ICP, it was safe to administer 5% dextrose.

Rhabdomyolysis

 If rhabdomyolysis is confirmed, treatment is aimed at preserving renal function and reversing metabolic abnormalities. Volume replacement using 0.9% sodium chloride is supported, at a rate of 1.5 l/h to maintain a urine output of 200- 300 ml/ hr (Zut et al, 2014). This allows for an increase in glomerular filtration rate and therefore removal, or washing out, of myoglobin. This should continue until the serum creatinine kinase has declined to 1000 IU/l or lower- careful monitoring is required as this volume of fluid could precipitate fluid overload and therefore pulmonary oedema.

In Clive's situation, although he was at risk of developing rhabdomyolysis, his creatinine kinase was not more than 10 times normal, so he was not diagnosed as having developed this.

Orthostatic hypotension

It was important when Clive had been stabilised to establish why he fell at home. Woodford and Walker (2005) report that 14% of patients with PD who are admitted to hospital as an emergency admission is due to them falling. Other reasons for admission are infective diseases (21%), orthostatic hypotension (OH) (4%) and hypovolaemia (2%). Infective disorders, such as urinary tract infections, can lead to falls as can OH, and hypovolaemia can result from falling as in Clive's situation. On discussion with Clive and his family, it became apparent that he had been having signs of OH shortly before falling. OH is one of the main non- motor features of PD. It is thought to occur because of degeneration of the autonomic system as the disease progresses (Velseboer et al, 2011), leading to ineffective vasoconstriction and excessive pooling of blood in the venous system. This then leads to cerebral hypoperfusion and can lead to dizziness and fainting: Clive had been experiencing dizziness on standing up.

Drugs are known to bring about and aggravate symptoms of OH (Perez- Lloret et al, 2012). Levodopa and dopamine agonists can lead to and worsen OH. Other drugs such as antipsychotics, antidepressants, diuretics and antihypertensives can also worsen OH. Perez-Lloret et al (2012) found that age, polypharmacy and use of amantadine and diuretics were the main factors related to OH. It was therefore important that Clive's drug therapy was reviewed to reduce the OH he was experiencing.

Management of his PD in critical care

When Clive was admitted he was able to take his dopamine agonist orally which relieved the PD symptoms he was displaying in the ED. If he had been unable to take these orally, it would have been essential to administer dopamine agonists via an alternative route. Patches can be used to administer the dopamine agonist Rotigotine. It is important to correctly convert the oral dosage into the equivalent patch dose which may require

consultation with the pharmacist or Parkinson's disease nurse specialist (PDNS). Madopar dispersible can be given via a nasogastric tube. Additionally, Apomorphine can be administered subcutaneously, however the cardiovascular effects of this drug would need to be observed for: it can cause hypotension and bradycardia both of which would be problematic in Clive's shock status. If this was given, Domperidone should be administered prior to the Apomorphine as Apomorphine can lead to nausea and vomiting.

Nursing considerations

Amongst the machines, noises and continuous observations it can be easy for nurses, in the critical care environment, to lose sight of the patient in the centre and the impact the experience has on their psychological wellbeing. Ramsay et al. (2014) highlights the importance effective rehabilitation can have on improving the patients quality of life, post critical care admission. A number of physiological and psychological complications are experienced following discharge from critical care, including a reduced appetite, depression and weakness. Experience of critical illness can precipitate feelings akin to post traumatic stress disorder (PTDS). Jackson et al (2007) found that reported PTDS rates in medical intensive care varied from 5%- 63% when they reviewed 16 studies. This is largely attributed to the critical care environment as well as the use of sedation- Clive do not require sedation to facilitate critical care interventions however, it was important to maintain sensory balance and avoid stressors. Nurses within critical care units play an important role in humanising the experience for the patient and their family. Woodrow (2011) advocate nurses to regularly orientate the patient and balance sensory stimulation by actively promoting an ambient, interesting environment to prevent adverse psychological outcomes. Committed to reducing associated long-term consequences of critical care, NICE (2009) advocate the application of a multidisciplinary rehabilitation care pathway, starting from admission. Early comprehensive assessment can also ensure for a smooth supportive discharge. For example in Clive's case, understanding the nature of his fall allowed for prompt referral to the necessary professionals, such as occupational therapists, facilitating an optimised recovery. He also required closer follow up by community based PDNS to ensure his symptoms were controlled and being managed effectively. Therefore, the PDNS team were consulted and involved in his care within the acute setting prior to his discharge back to primary care.

Conclusion

Having an in-depth understanding of Clive's pathophysiological response allowed for clear, effective treatment and management. Although this level of knowledge is essential when providing care in critical care, its speciality should not allow for discrepancies in knowledge in other care settings. Nurses outside of critical care areas need to be aware of the consequences of shock so that early patient management can be initiated to avoid patient deterioration. Early and thorough assessment using an ABCDE process and escalation to critical care outreach is important to prevent the complications of a reduced cardiac output, whatever the cause. It was also important to establish the underlying cause of why he fell at home and therefore further assessment of his Parkinson's disease and drug management was a very important element to avoid a further similar situation and to enable a safe discharge back to the community setting.

References

Aitken, L; Marshall, A; Chaboyer, W (2015) ACCCN's Critical Care Nursing 3rd ed. Chatswood: Elsevier

Annane, D; Siami, S; Jaber, S; Martin, C; Elatrous, S; Declere, AD; Preiser, JC; Outin, H; Troche, G; Charpentier, C; Trouillet, JL; Kimmoun, A; Forceville, X; Darmon, M; Lesur, O; Regnier, J; Abroug, F; Berger, P; Clec'h, C; Cousson, J; Thibault, L; Chevret, S; (2013) Effects of Fluid Resuscitation With Colloids vs Crystalloids on Mortality in Critically Ill Patients Presenting With Hypovolemic Shock The CRISTAL Randomized Trial *JAMA*. 310 (17) pp1809-1817

Baaijens, F., Oomens, C.C., Bader, D.D. and Loerakker, S.S. (2015) 'Pressure induced deep tissue injury explained', *Annals of Biomedical Engineering,* 43 (2), pp.297.

Bench, S. and Brown, K. (2011) *Critical care nursing: learning from practice.* Chichester: Wiley-Blackwell.

British Medical Association and Royal Pharmaceutical Society (2016) *British National Formulary.* London: Pharmaceutical Press. Available at: https://www.medicinescomplete.com/mc/bnf/current/PHP78566-fluids-and-electrolytes.htm?q=intravenous%20sodium&t=advanced&ss=ts&tot=39&p=3#_hit (Accessed 19th April 2016).

British Thoracic Society (2008) *Guideline for emergency oxygen use in adult patients.* Available at: https://www.brit-thoracic.org.uk/document-library/clinical-information/oxygen/emergency-oxygen-use-in-adult-patients-guideline/emergency-oxygen-use-in-adult-patients-guideline/ (Accessed: 12th April 2016).

Clarke, D. and Ketchell, A. (eds.) (2011) *Nursing the acutely ill adult: priorities in assessment and management.* Basingstoke: Palgrave Macmillan.

Creed, F. and Spiers, C. (2010) *Care of the acutely ill adult: an essential guide for nurses.* Oxford: Oxford University Press.

Criddle, L.M. (2003) 'Rhabdomyolysis. Pathophysiology, recognition, and management', *Critical Care Nurse,* 23 (6), pp.14.

Dougherty, L. and Lamb, J. (2008) *Intravenous therapy in nursing practice.* 2nd ed. Oxford: Blackwell.

Garretson, S. and Malberti, S. (2007) 'Understanding hypovolaemic, cardiogenic and septic shock', *Nursing standard,* 21 (50), pp.46.

Jackson, JC; Hart, RP; Gordon, SM; Hopkins, RO; Girard, TD; Ely, E (2007) Post traumatic stress disorder and post- traumatic stress symptoms following critical illness in medical intensive care unit patients: assessing the magnitude of the problem *Critical Care* 11: R27

Jevon, P. and Ewens, B. (2012) *Monitoring the critically ill patient.* 3rd ed. Chichester: Wiley-Blackwell.

Larkin, B.G. and Zimmanck, R.J. (2015) 'Interpreting Arterial Blood Gases Successfully', *AORN Journal,* 102 (4), pp.343.

McLeod, A. (2016). *Arterial Blood Gas Analysis - making it easy.* Keswick: MK Publishing.

National Institute for Health and Care Excellence (2013) *Intravenous fluid therapy in adults in hospital (CG174).* Available at:

https://www.nice.org.uk/guidance/cg174/resources/intravenous-fluid-therapy-in-over-16s-in-hospital-35109752233669 (Accessed: 15 April 2016).

National Institute for Health and Care Excellence (2009) *Rehabilitation after critical illness in Rehabilitation after critical illness in adults (CG83).* Available at:

https://www.nice.org.uk/guidance/cg83/resources/rehabilitation-after-critical-illness-in-adults-975687209413 (Accessed: 17th April 2016).

Nursing and Midwifery Council (2015) *The Code* NMC: London

Perez- Lloret, S; Rey, MV; Fabre, N; Ory, F; Spampinato, U; Senard, JM; Pavy- Le Traon, A; Montastruc, JL; Rascol, O (2012) Factors related to orthostatic hypotension in Parkinson's disease *Parkinsonism and related disorders* 18 (5) pp. 501-505

Powell-Tuck, J., Gosling, P., Lobo, D.N., Allison, S.P., Carlson, G.L., Gore, M., Lewington, A.J., Pearse, R.M. & Mythen, M.G. (2011) *British Consensus Guidelines on Intravenous Fluid Therapy for Adult Surgical Patients.* Available at:

http://www.bapen.org.uk/pdfs/bapen_pubs/giftasup.pdf (Accessed: 16th April 2016).

Ramsay, P., Salisbury, L.G., Merriweather, J.L., Huby, G., Rattray, J.E., Hull, A.M., Brett, S.J., Mackenzie, S.J., Murray, G.D., Forbes, J.F. and Walsh, T.S. (2014) 'A rehabilitation intervention to promote physical recovery following intensive care: a detailed description of construct development, rationale and content together with proposed taxonomy to capture processes in a randomised controlled trial', *Trials,* 15 pp.38-6215-15-38.

Sherratt, R. (2014) 'NICE guidance on giving intravenous fluids', *Nursing times,* 110 (6), pp.12.

Thim, T., Krarup, N.H., Grove, E.L., Rohde, C.V. & Lofgren, B. (2012), Initial assessment and treatment with the Airway, Breathing, Circulation, Disability, Exposure (ABCDE) approach, *International Journal of General Medicine,* 5, pp. 117-121.

Velseboer, DC; de Haan, RJ; Wieling, W; Goldstein, DS; de Bie, R (2011) Prevalence of orthostatic hypotension in Parkinson's disease: A systematic review and meta-analysis *Parkinsonism and Related Disorders* 17 (10) pp. 724- 729

Vincent, J. and Weil, M.H. (2006) 'Fluid challenge revisited', *Critical Care Medicine,* 34 (5), pp.1333-1337.

Williams, J. and Thorpe, C. (2014) 'Rhabdomyolysis', *Continuing Education in Anaesthesia, Critical Care & Pain,* 14 (4), pp.163-166.

Woodford, H and Walker, R (2005) Emergency Hospital Admissions in Idiopathic Parkinson's Disease *Movement Disorders* 20 (9), pp. 1104–1108

Woodrow, P. (2011) *Intensive care nursing: a framework for practice.* 3rd ed. New York: Routledge.

Zelman, M., Tompary, E., Raymond, J., Holdaway, P., Mulvihill, M.L., Steggall, M. and Dingle, M. (2011) *Introductory pathophysiology for nursing and healthcare professionals.* Harlow: Pearson.

Zutt, R; van der Kooi, AJ, Linthorst, GE; Wanderes, RJA; de Visser, M (2014) Rhabdomyolysis: Review of the literature *Neuromuscular Disorders* 24 (8) 651- 659