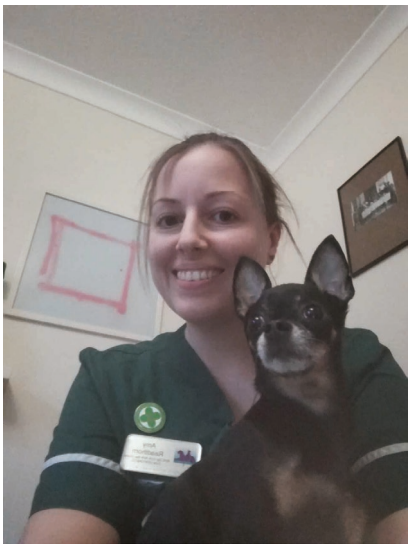

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What is lactate and why are we measuring it?

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ABSTRACT: Lactate measuring machines are becoming more commonly utilised in veterinary practice to assess perfusion in our patients, but why is it produced and how can we reduce the levels to avoid blood pH disturbances?

Keywords: lactate; pH; metabolic acidosis; hypovolaemia

Why is lactate produced?

Hypovolaemia is caused by a reduction in intravascular volume leading to a decrease in venous return and ventricular filling, resulting in a reduction in stroke volume and cardiac output (Battaglia & Steel, 2016). Hypotension then develops causing an inadequate oxygen delivery and decreased tissue perfusion (Battaglia & Steel, 2016). This creates an energy production shift from mitochondrial oxidative metabolism (aerobic metabolism) to ineffective oxygen dependent glycolysis (anaerobic metabolism) as a protective mechanism, due to this decrease in oxygen supply (Jasani, 2012; King & Boag, 2007). The production of lactate is due to the reduction of pryuvate, which is a by-product of glycolysis, using the enzyme lactate dehydrogenase, which can be found in all tissues (Battaglia & Steel, 2016; Burkiti-Creed & Davis, 2012).

Lactic acidosis

It is vital that blood pH is maintained within a strict range to allow for normal cellular function which is achieved by use of buffers, mainly bicarbonate, in the body system (see Appendix 1 in Supplemental Materials) (King & Boag, 2007). Carbon dioxide (CO_2) is a by-product of carbohydrates and fat metabolism excreted by the lungs, whereas hydrogen ions (H+) are a by-product of protein and phospholipid metabolism excreted by the kidneys (King & Boag, 2007). Acid: base disturbances occur due to alterations in non-volatile acids (non-carbon dioxide) in the blood, with metabolic acidosis implying that either an acid has been added to the blood or the bicarbonate has been lost (King & Boag, 2007).

Increased levels of lactate in the bloodstream (hyperlactataemia) result in hydrogen ions being buffered from bicarbonate, leading to metabolic acidosis. Lactic acidosis derived from an inadequate oxygen supply or an increase in oxygen demand, such as hypovolaemic shock, is classified as type A (Battaglia & Steel, 2016). Type B occurs when there is inadequate utilisation of oxygen, for example, with systemic immune response to sepsis (Battaglia & Steel, 2016). Handheld lactometers can be use to determine the level of lactate in the bloodstream as well as blood gas machines which also show the current pH of the sample alongside other gas analysis (see Figure 1).

Fluid resuscitation

Resuscitation fluids are essential for treatment of hypovolaemic shock and should be used to correct any pathological conditions, such as hyperlactatemia, immediately (Kushimoto et al., 2016; Silverstein, 2011). The aim is to restore adequate tissue perfusion by maintaining an effective intravascular circulation, which when combined with an increased cardiac output and oxygen delivery will improve organ oxygen supply (Jasani, 2012; King & Boag, 2007). As lactate accumulation develops due to anaerobic metabolism once oxygen delivery is improved, aerobic metabolism should return causing a reduction in lactate production.

Isotonic fluids such as Lactated Ringer's solution (LRS) and saline equilibrate in the interstitial fluid rapidly, therefore, high doses are required in the form of boluses to expand the intravascular system (Jasani, 2012). The resuscitation rate is the total amount to be administered

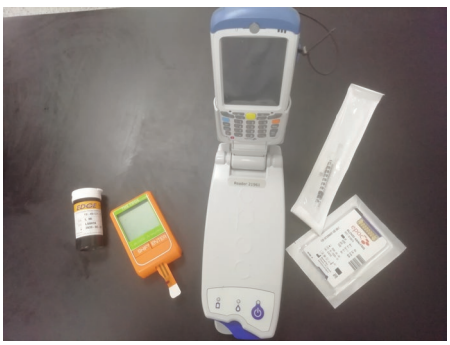

Figure 1.

advisably over an hour and is equivalent to the patient's blood volume, for example in a dog this would be 90 ml/kg/hr (Jasani, 2012; Silverstein, 2011). The aim is to give boluses of fluid at 10–40 ml/kg over 15–20 minutes when using isotonic fluids to allow for regular assessments to establish when endpoints suggestive of adequate perfusion are achieved (Jasani, 2012). These endpoint assessments should include pulse quality, heart rate and capillary refill time alongside lactate readings.

Lactated Ringer's solution vs saline

The isotonic effects of LRS and saline results in an electrolyte concentration similar to that of plasma and they are indicated as ideal choices for fluid resuscitation with hypovolaemic cases (Jasani, 2012; Kirby & Linklater, 2017). Saline has a pH level of 5 which is more acidic than plasma pH and can have a mild acidotic effect, whereas LRS has a pH of 6.7 which is more alkaline (Kirby & Linklater, 2017). LRS also contains a buffer, in the form of sodium lactate. This lactate is converted by the liver and kidneys to bicarbonate and is unlikely to cause a clinically sustained alteration in plasma lactate concentration, instead the resulting bicarbonate acts as a buffer to reduce the amount of H+ (Kirby & Linklater, 2017). Saline contains a higher chloride concentration than LRS which can result in a hyperchloraemic metabolic acidosis and this combined with the lack of a buffer may suggest use with metabolic alkalosis rather than acidosis, therefore its use could be contra-indicated in cases of hyperlactataemia (Jasani, 2012; Kirby & Linklater, 2017). Its use will also dilute bicarbonate already present in the plasma (Burkiti-Creedon & Davis, 2012). It has however been suggested that in requirement of resuscitation fluids these factors are not of clinical significance and that both LRS and saline should be considered in hypovolaemic emergencies as correction of perfusion alone will reduce the hyperlactataemia, therefore resolving the metabolic acidosis (Jasani, 2012). LRS does contain a small concentration of calcium and potassium and it has been discussed that if a patient presents with hypercalcaemia or hyperkalcaemia saline, should be indicated, however, the concentration of potassium is so small that this is unlikely to cause any significant alterations to blood concentration (Jasani, 2012). Therefore, despite all the above factors LRS is often preferred in these cases due to the advantage of the buffer in correcting the acidosis.

Monitoring intravenous fluid therapy

When monitoring a patient undergoing fluid therapy, three presumptions should be considered; volume overload is bad, inadequate volume is bad and what is concluded about the patient's fluid balance should be correct (Kirby & Linklater, 2017). Using lactate as a marker can help to evaluate the effectiveness of resuscitation fluid therapy, however a stress response hyperlactataemia should also be considered to avoid over infusion (Battaglia & Steel, 2016; Kushimoto et al., 2016). Therefore, other perfusion parameters should be monitored alongside this. Studies have, however, shown that if more than 50% of lactate clearance is shown 16 hours post diagnosis then survival rates increase (Burkiti-Creedon & Davis, 2012).

Monitoring and assessment of adequate perfusion should include assessment of perfusion parameters by use of physiological monitoring and ongoing laboratory testing to evaluate cellular function (Battaglia & Steel, 2016). Resuscitation endpoints should be assessed after every bolus which should include assessment of mucus membrane colour and capillary refill time, heart rate, pulse pattern and intensity, blood pressure and central venous pressure (Kirby & Linklater, 2017). Prior to fluid resuscitation cardiac performance should be evaluated to avoid the risk of fluid overload in cases of cardiogenic shock (Kirby & Linklater, 2017). Oxygen supplementation should be considered and saturation levels should be monitored to ensure this maintains above 95%. Measuring arterial blood pressure will provide information on stroke volume, heart rate and systemic vascular resistance. An electrocardiogram (ECG) should be utilised if any bradycardia or tachycardia develops as this may highlight an abnormal rhythm (Battaglia & Steel, 2016) (see Figure 2).

There is no expiration date for catheter placements and given the limited number of insertion sites, catheters should be regularly assessed and only removed if a complication arises (Kirby & Linklater, 2017). It is suggested that the catheter should be checked twice daily for signs of heat, erythema, swelling, fluid leakage and swelling above or below the bandage (King & Boag, 2007). This should involve removing the bandage at each assessment and if any phlebitis or pyrexia of unexplained cause develops that the catheter should be removed and the tip sent for microbiological culture. RCVS (2016) suggested utilising a checklist to ensure a high standard of peripheral catheter care (see Appendix 2 in Supplemental Materials). This advises assessment of 14 indicators involving patient interference, bandage appearance, assessment of catheter area alongside hygiene guidance and flushing of the port every 24 hours. This can be easily implemented in practice to allow guidance for nurses and trainees.

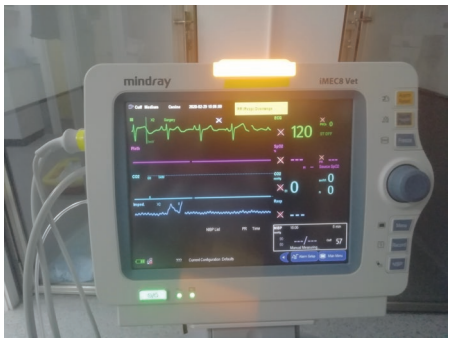

Figure 2.

Conclusion

Although lactate can be utilised as a marker for diagnosis and improvements in perfusion during hypovolaemic shock, it is important to consider and rule out other cases of hyperlactataemia such as anaemia or cardiogenic shock. These conditions also result in a lack of oxygen delivery resulting in lactate accumulation but would require a different treatment plan than hypovolaemic shock. Lactate readings are proving more useful within veterinary practice and a good understanding of accumulation in the blood is essential to enable us to utilise them. Patients commonly present with hypovolaemic shock and as nurses it is important we understand what clinical signs to monitor to allow us to highlight improvements seen during a treatment plan and lactate readings can prove beneficial to assess alongside physiological measurements.

Supplementary material

Supplemental data for this article is available online at https://doi.org/10.1080/17415349.2020.1790447

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