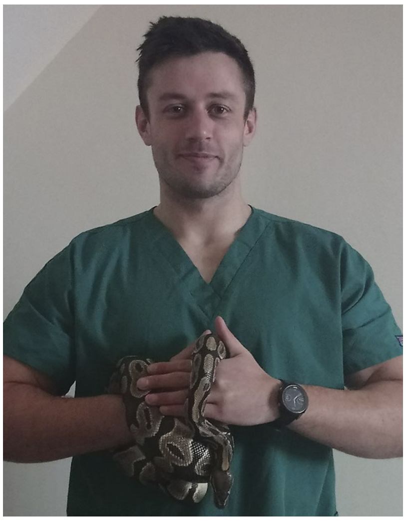

Neil Kemp BSc(Hons) PgDip(Anaesthesia/Analgesia) RVN

Neil graduated with a first-class honours degree from Edinburgh Napier University, before working in first-opinion, referral and emergency out of hours practice with a particular focus in anaesthesia. Then wanting to pursue a path in academia, he became a full-time locum while studying and recently achieved a post graduate diploma in veterinary anaesthesia and analgesia.
Email: Neil004@hotmail.com

DOI: 10.1080/17415349.2021.1882919

Cardiovascular changes in physiology seen in canines with gastric dilation volvulus (GDV)

Neil Kemp BSc(Hons) PgDip(Anaesthesia/Analgesia) RVN

Portknockie, Buckie, UK

ABSTRACT: This article analyses the cardiovascular changes typically present in a patient with a gastric dilation volvulus. This pathogenesis results in the reduction of oxygen delivery to tissues through hypoperfusion, the translocation of GI bacteria and the release of proinflammatory cytokines. By highlighting these physiological changes, in an emergency case that can be seen at most practices, this article will aid more complete monitoring. This monitoring facilitates appropriate interventions and gives a more comprehensive outline of circulatory consequences.

Keywords: gastric dilation volvulus; monitoring; cardiovascular effects

Introduction

Gastric dilation volvulus (GDV) involves rapid distension and rotation of the stomach; with large and giant breeds showing a predisposition. The physiological effects include multiple organ systems consisting of cardiovascular, respiratory, gastrointestinal, coagulation and renal. A process of systemic inflammatory response syndrome progresses to multiple organ dysfunction syndrome (Osterbur et al., 2014). The pathogenesis involves the reduction of oxygen delivery to tissues through hypoperfusion, the translocation of GI bacteria and the release of proinflammatory cytokines (Sharp & Rozanski, 2014). This paper will analyse the cardiovascular effects and monitoring involved in treating this condition.

Cardiovascular effects

The cardiovascular effects of GDV include shock, cardiac arrythmias and dysfunction of the myocardium. The shock presented can be diverse with a combination of obstructive, distributive, hypovolaemic and cardiogenic (Sharp & Rozanski, 2014). These cardiovascular effects can result in the release of troponins, catecholamines and proinflammatory cytokines (Schober et al., 2002).

Obstructive shock

Obstructive shock occurs due to abdominal distension compressing low pressure vessels including the portal and splenic veins and caudal vena cava. Reduction in venous return (VR) impacts preload and subsequently stroke volume (SV). This results in increasing heart rate (HR) to maintain the cardiac output (CO) which equals SV multiplied by HR. This compensatory mechanism is inadequate and causes increased myocardial oxygen demand (Bruchim & Kelmer, 2014).

Distributive shock

Distributive shock involves splanchnic pooling of blood due to the decreased VR and increased venous pressure and is exacerbated by the vasodilatory effect of nitric oxide (Howe & Boothe, 2001) released.

Hypovolaemic shock

Hypovolaemia occurs due to circulating fluid losses including: from vomiting, distension preventing ingestion, splanchnic pooling of blood and portal hypertension causing third spacing of fluid.

Cardiogenic shock

Cardiogenic shock results from myocardial dysfunction and mainly ventricular arrythmias, predominantly because of myocardial ischaemia exacerbated by reduced coronary blood flow and compensatory mechanisms increasing workload (Brockman et al., 1995; Horne et al., 1985).

Assessment of the patient

To assess cardiac function and treatment plan efficacy, clinical examination, and assessment of blood pressure (BP), CO and tissue perfusion are utilised.

Clinical examination

Clinical examination presents cardiovascular signs including depressed mentation, pale or injected mucous membranes and tachycardia with weak irregular pulses (Haak et al., 2012). Clinical examination is the primary form of monitoring on admission and should not be overlooked as a rapid form of assessment used to guide more invasive measures.

Electrocardiogram (ECG)

ECG monitoring is essential due to the common occurrence of ventricular arrythmias and possible presentation of irregular pulses from examination. Thought to be caused by myocardial ischaemia, ventricular arrythmias were present in 40% of dogs, showing a failing ability to deal with circulatory deficit (Brockman et al., 1995). The production of ectopic foci of electrical activity throughout the myocardium has been identified in patients with GDV and the monitoring of this condition and treatment efficacy is paramount (Sharp & Rozanski, 2014). Time is an important consideration, ECG is quick, non-invasive and provides a wealth of information.

Other techniques that assess circulation take longer and require more skill, this can be problematic if unable to decompress the stomach and rapid surgery is the only treatment option to stabilise the patient.

Capnography

Capnography provides a continuous reading of respiratory gases, allowing assessment of gas exchange via pulmonary circulation. During spontaneous ventilation capnography can indicate CO and severity of shock (Swenson et al., 2008; Weil et al., 1999).

Pulse oximetry

Pulse oximetry is a limited form of cardiac monitoring that provides an estimation of arterial oxygenation (Thawley & Waddell, 2013). In addition to saturation, the production of a plethysmograph can provide more detailed beat-to-beat analysis of arterial waveform. This method should be used with caution as it shows delayed graph changes and altered amplitude, the basic graph shape can give indications of myocardial contractility, hypovolaemic status and resistance to outflow (Shamir et al., 1999).

Both capnography and pulse oximetry are not prioritised highly in terms of the quality of information provided regarding cardiovascular function, compared to more invasive measures. However, in terms of emergency monitoring, they are useful in providing rapid and easily obtained data, guiding requirement for invasive monitoring techniques to confirm cardiovascular decompensation. Using multiple forms of non-invasive techniques can help to reduce error due to their individual limitations.

Blood analysis

Information regarding oxygenation, carbon dioxide concentration, pH and lactate can be obtained through blood analysis. Sample collection can be facilitated by an arterial catheter for intermittent testing. The presence of both hyperlactataemia and acidosis indicate decreased oxygen delivery to tissues and increased activity of anaerobic pathways (Beer et al., 2013; De Papp et al., 1999). This testing is more accurate than non-invasive monitoring and can be used to confirm data produced by continuous output monitors (capnography/pulse oximetry). Electrolyte analysis can be beneficial at identifying causes of arrythmia. Persistent vomiting of gastric juices rich in potassium, sodium, hydrogen, and chloride ions leads to imbalance (Papazoglou et al., 2003); prolonged vomiting before assessment is shown to increase mortality (Evans et al., 1994).

Non-invasive BP monitoring (NiBP)

Non-invasive methods of BP monitoring include doppler and oscillometry. They provide rapid data, are easy to use and should be a starting point in BP monitoring. BP is a major component of haemodynamic status assessment; however, should not be confused with CO as it is also influenced by systemic vascular resistance (Cooper & Cooper, 2012). Various forms of shock will negatively impact BP, regardless of compensatory mechanisms such as tachycardia. NiBP monitoring can be limited, particularly in compromised patients; however, the use of both forms simultaneously can reduce inaccuracy. Providing mean arterial pressure (MAP) which is considered the most accurate representation of circulating pressure with a minimum of 60 mmHg considered viable for organ perfusion and beat-to-beat analysis provided by doppler ultrasonography (Ribezzo et al., 2014; Wagner & Brodbelt, 1997).

Invasive BP

Direct arterial BP is considered the 'gold standard', allowing continuous monitoring of arterial pressure and waveform, providing real-time data, and allowing more accurate assessment of treatment efficacy (Waddell & Brown, 2015). However, this requires time spent by skilled staff to set up an arterial cannula with pressure bag and transducer. Canula placement increases risk of infection, haemorrhage, thrombosis, and tissue necrosis (Mazzaferro & Wagner, 2001). Therefore, placement of an arterial catheter should only be undertaken in critical patients such as this patient with GDV and haemodynamic instability (Waddell & Brown, 2015).

Limitations of this technique that alter the signal produced can include both resonance and damping. Resonance frequencies <40Hz from movement of the transducer diaphragm or movement of blood in the catheter can severely distort the waveform produced (Figure 1). Damping is caused by restrictions in transmission (clots and air bubbles). Some damping is desirable for accurate interpretation; however, overdamping causes underreading of SAP and DAP (Figure 1), and vice-versa when underdamping.

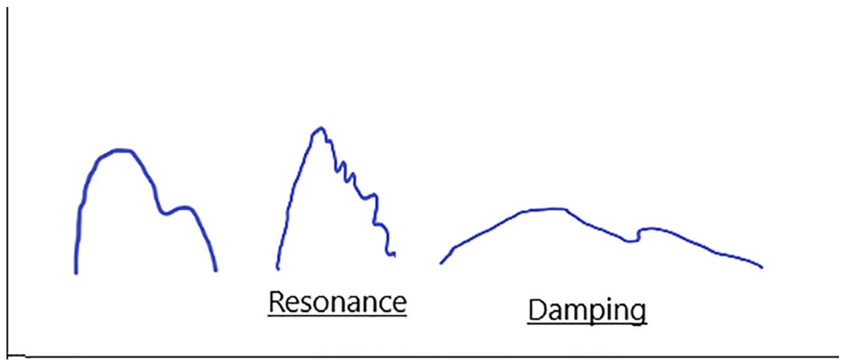

Figure 1. Graph showing normal arterial waveform, compared to resonance and damping interference.

Arterial waveform analysis

When used effectively this method can provide information beyond BP readings. The arterial waveform can assist assessment of myocardial contractility when considering the slope of the upstroke (Figure 2). This could be pertinent for myocardial ischaemia from prolonged reduction in oxygen delivery as this will exhibit reduced contractility. The GDV patient would likely show reduced SV (Figure 3); depicted in the waveform with reduced area under the curve from the upstroke to the dicrotic notch.

Cardiac output (CO)

When considering CO, all techniques are compared to thermodilution as the 'gold standard'; when analysis of arterial waveform was compared to thermodilution in human anaesthesia it produced comparably reliable results (Jansen et al., 2001).

CO represents the cardiac contribution to global oxygen delivery to tissues and is determined by HR, rhythm, contractility, preload, and afterload (Hall, 2016). However, this value should not be looked at in isolation and its priority is dependent on having other cardiac monitoring in place. CO monitoring was not shown to improve outcome in critically ill patients (Ospina-Tascon et al., 2008) and if viewed in isolation can negatively influence treatment. If this patient became septic, CO could appear normal while suffering circulatory failure (Kehlet & Bundgaard-Nielsen, 2009).

Persistently low CO has a significant effect on oxygen delivery and in this patient with reduced VR could be advantageous. CO monitoring also facilitates goal directed fluid therapy which can be beneficial in this patient as actual fluid loses are relatively small. Large volumes of isotonic fluid therapy may not be as advantageous as use of hypertonic and isotonic solutions; to both replace fluid loss and draw fluid from third spacing.

Methods of monitoring CO

Many methods of CO monitoring utilise Fick's principle; detailing the quantity of a marker (oxygen, carbon dioxide, temperature, lithium) entering or leaving an organ as equal to the blood flow divided by the arteriovenous difference. Practically this is typically achieved through a Swan-Ganz pulmonary catheter, that is guided through the right atrium, ventricle, and pulmonary artery before wedging into position. This process is guided through changes in pressure and pressure waveform to isolate the position of the catheter tip (Vigani, 2015). In the research, setting output can be measured directly through electromagnetic flowmetry; however, this requires probe placement and is impractical for the clinical setting (Tabrizchi & Iida, 2003).

Thermodilution involves the injection of cold saline and the monitoring of temperature change over time via a thermistor at the end of the pulmonary catheter. A modification of the Swan-Ganz catheter can include a heating coil that heats blood in the caudal vena cava and is then detected by the thermistor, allowing a continuous measurement of CO. Significant risk with placement of a pulmonary arterial catheter include arrythmias, heart block, thromboembolism, pulmonary infarction, valvular damage, and endocarditis (Harvey et al., 2005; Polanczyk et al., 2001; Shah et al., 2005). An additional limitation for this specific case of GDV would be the provision of fluid resuscitation, modifying circulating temperature and giving inaccurate results.

Alternatives that do not require pulmonary catheterisation are available. The pulse contour cardiac output (PiCCO) system uses a central venous and arterial catheter and applies the Stewart-Hamilton equation to form an estimation (Von Spiegel et al., 1996). Cold intravenous injections and an arterial thermistor are required; therefore, the accuracy of the results could be negatively affected by large volumes of intravenous fluid therapy. CO status systems utilise ultrasonography to detect blood velocity following injection of saline. The real benefit with this system is that it is easier to use, does not require pulmonary catheterisation and has been shown to produce accurate results, even in smaller patients (de Boode et al., 2010). CardioQ ultrasound displays a waveform allowing optimum positioning of the probe, producing informing including CO, SV, HR and peak velocity. Analysis can assist with identification of hypovolaemia through reduced flow time, waveform analysis then facilitates goal directed fluid therapy during fluid resuscitation in this patient. Thoracic electrical impedance is another non-invasive technique, however, is unsuitable for this patient as it is shown to provide unreliable results in unstable patients (Yamashita et al., 2007).

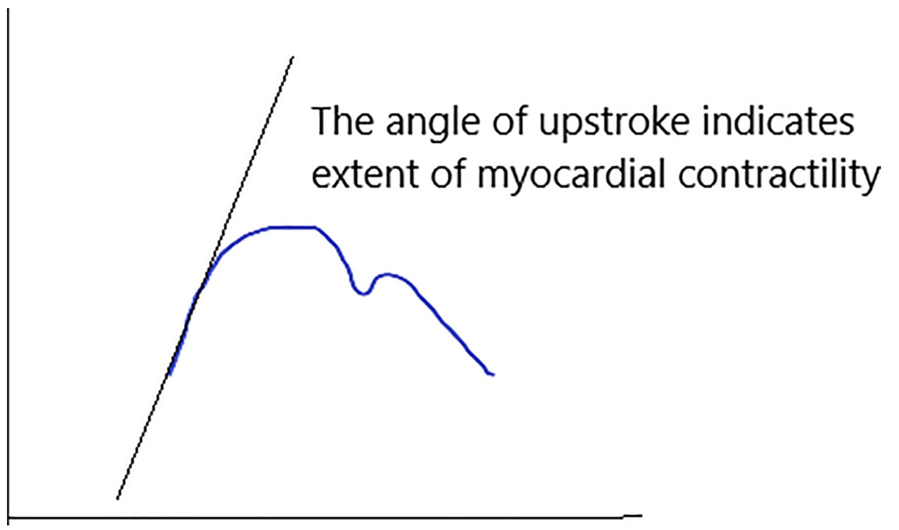

Figure 2. Graph to show the upstroke of arterial waveform and connection to contractility.

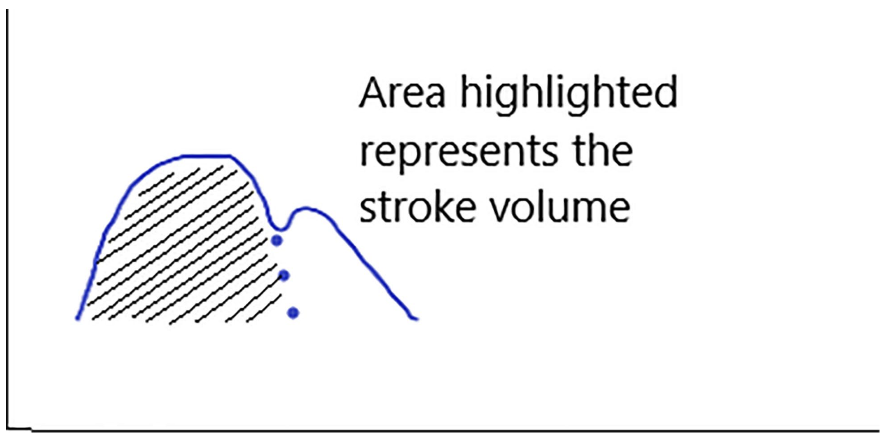

Figure 3. Graph highlighting the area of an arterial waveform that represents stroke volume.

Lithium dilution technique involves a bolus injection of lithium and CO can be estimated through time taken for circulation of the marker. The marker can be administered through peripheral catheterisation and the lithium concentration is determined through a lithium selective electrode connected to an arterial catheter (Linton et al., 1993). The major advantage being simple to set up and execute, reducing treatment time compared to techniques reliant on central catheterisation. Therefore, this technique is suitable for the clinical environment, producing reliable results in dogs (Mason et al., 2001); however, for this case it would be limited by the presence of tachyarrhythmias and fluid resuscitation (Linton et al., 1993). This could be a more viable option if the patient is able to be stabilised with fluid deficits replaced, dilation reduced and arrythmias addressed prior to surgery.

Conclusion

When analysing the efficacy of available cardiac monitoring equipment, it is clear that no system reliably provides all data required. Therefore, for these patients, I would prioritise the monitoring equipment as explained; drawing maximum information from quickly applied techniques prior to establishing more accurate, advanced methods. It is the combination of various techniques that produce the most reliable indication of haemodynamic stability and efficacy of treatment. The reliance on advanced monitoring techniques can be misplaced. For example, CO in the septic patient appearing normal during the early (hyperdynamic) stages despite tachyarrhythmias and failing haemodynamic status.

References

Beer, K. A. S., Syring, R. S., & Drobatz, K. J. (2013). Evaluation of plasma lactate concentration and base excess at the time of hospital admission as predictors of gastric necrosis and outcome and correlation between those variables in dogs with gastric dilatation-volvulus: 78 cases (2004-2009). *Journal of the American Veterinary Medical Association*, *242*(1), 54–58. https://doi.org/10.2460/javma.242.1.54

Brockman, D. J., Washabau, R. J., & Drobatz, K. J. (1995). Canine gastric dilatation/volvulus syndrome in a veterinary critical care unit: 295 cases (1986-1992). *Journal of the American Veterinary Medical Association*, *207*(4), 460–464.

Bruchim, Y., & Kelmer, E. (2014). Postoperative management of dogs with gastric dilatation and volvulus. *Topics in Companion Animal Medicine*, *29*(3), 81–85. https://doi.org/10.1053/j.tcam.2014.09.003

Cooper, E., & Cooper, S. (2012). Direct systemic arterial blood pressure monitoring. In J. M. B. Creed & H. Davis (Eds.), *Advanced monitoring and procedures for small animal emergency and critical care* (pp. 122–133). John Wiley & Sons.

de Boode, W. P., van Heijst, A. F., Hopman, J. C., Tanke, R. B., van der Hoeven, H. G., & Liem, K. D. (2010). Cardiac output measurement using an ultrasound dilution method: A validation study in ventilated piglets. *Pediatric Critical Care Medicine*, *11*(1), 103–108. https://doi.org/10.1097/PCC.0b013e3181b064ea

De Papp, E., Drobatz, K. J., & Hughes, D. (1999). Plasma lactate concentration as a predictor of gastric necrosis and survival among dogs with gastric dilatation-volvulus: 102 cases (1995-1998). *Journal of the American Veterinary Medical Association*, *215*(1), 49–52.

Evans, K. L., Smeak, D. D., & Biller, D. S. (1994). Gastrointestinal linear foreign bodies in 32 dogs: A retrospective evaluation and feline comparison. *Journal of the American Animal Hospital Association*, *30*(5), 445–450.

Haak, C. E., Rudloff, E., & Kirby, R. (2012). Comparison of Hb-200 and 6% hetastarch 450/0.7 during initial fluid resuscitation of 20 dogs with gastric dilatation-volvulus. *Journal of Veterinary Emergency and Critical Care*, *22*(2), 201–210. https://doi.org/10.1111/j.1476-4431.2012.00726.x

Hall, J. E. (2016). *Guyton and hall textbook of medical physiology* (13th ed., pp. 245–258). Elsevier.

Harvey, S., Harrison, D. A., Singer, M., Ashcroft, J., Jones, C. M., Elbourne, D., Brampton, W., Williams, D., Young, D., & Rowan, K. (2005). Assessment of the clinical effectiveness of pulmonary artery catheters in management of patients in intensive care (PAC-Man): A randomised controlled trial. *The Lancet*, *366*(9484), 472–477. https://doi.org/10.1016/S0140-6736(05)67061-4

Horne, W. A., Gilmore, D. R., Dietze, A. E., Freden, G. O., & Short, C. E. (1985). Effects of gastric distention-volvulus on coronary blood flow and myocardial oxygen consumption in the dog. *American Journal of Veterinary Research*, *46*(1), 98–104.

Howe, L. M., & Boothe, H. W. Jr, (2001). Nitric oxide: A review for veterinary surgeons. *Veterinary Surgery: VS*, *30*(1), 44–57. https://doi.org/10.1053/jvet.2001.20341

Jansen, J. R. C., Schreuder, J. J., Mulier, J. P., Smith, N. T., Settels, J. J., & Wesseling, K. H. (2001). A comparison of cardiac output derived from the arterial pressure wave against thermodilution in cardiac surgery patients. *British Journal of Anaesthesia*, *87*(2), 212–222. https://doi.org/10.1093/bja/87.2.212

Kehlet, H., & Bundgaard-Nielsen, M. (2009). Goal-directed perioperative fluid management: Why, when, and how? *Anesthesiology*, *110*(3), 453–455. https://doi.org/10.1097/ALN.0b013e3181984217

Linton, R. A. F., Band, D. M., & Haire, K. M. (1993). A new method of measuring cardiac output in man using lithium dilution. *British Journal of Anaesthesia*, *71*(2), 262–266. https://doi.org/10.1093/bja/71.2.262

Mason, D. J., O'Grady, M., Woods, J. P., & McDonell, W. (2001). Assessment of lithium dilution cardiac output as a technique for measurement of cardiac output in dogs. *American Journal of Veterinary Research*, *62*(8), 1255–1261. https://doi.org/10.2460/ajvr.2001.62.1255

Mazzaferro, E., & Wagner, A. E. (2001). Hypotension during anesthesia in dogs and cats: Recognition, causes and treatment. *Compendium*, *23*(8), 728–737.

Ospina-Tascon, G. A., Cordioli, R. L., & Vincent, J. L. (2008). What type of monitoring has been shown to improve outcomes in acutely ill patients? *Intensive Care Medicine*, *34*(5), 800–820. https://doi.org/10.1007/s00134-007-0967-6

Osterbur, K., Mann, F. A., Kuroki, K., & DeClue, A. (2014). Multiple organ dysfunction syndrome in humans and animals. *Journal of Veterinary Internal Medicine*, *28*(4), 1141–1151. https://doi.org/10.1111/jvim.12364

Papazoglou, L. G., Patsikas, M. N., & Rallis, T. (2003). Intestinal foreign bodies in dogs and cats. *Compendium on Continuing Education for the Practising Veterinarian-North American Edition*, *25*(11), 830–845.

Polanczyk, C. A., Rohde, L. E., Goldman, L., Cook, E. F., Thomas, E. J., Marcantonio, E. R., Mangione, C. M., & Lee, T. H. (2001). Right heart catheterization and cardiac complications in patients undergoing noncardiac surgery: An observational study. *JAMA*, *286*(3), 309–314. https://doi.org/10.1001/jama.286.3.309

Ribezzo, S., Spina, E., Di Bartolomeo, S., & Sanson, G. (2014). Noninvasive techniques for blood pressure measurement are not a reliable alternative to direct measurement: A randomized crossover trial in ICU. *The Scientific World Journal*, *2014*, 1–8. https://doi.org/10.1155/2014/353628

Schober, K. E., Cornand, C., Kirbach, B., Aupperle, H., & Oechtering, G. (2002). Serum cardiac troponin I and cardiac troponin T concentrations in dogs with gastric dilatation-volvulus. *Journal of the American Veterinary Medical Association*, *221*(3), 381–388. https://doi.org/10.2460/javma.2002.221.381

Shah, M. R., Hasselblad, V., Stevenson, L. W., Binanay, C., O'Connor, C. M., Sopko, G., & Califf, R. M. (2005). Impact of the pulmonary artery catheter in critically ill patients: Meta-analysis of randomized clinical trials. *JAMA*, *294*(13), 1664–1670. https://doi.org/10.1001/jama.294.13.1664

Shamir, M., Eidelman, L. A., Floman, Y., Kaplan, L., & Pizov, R. (1999). Pulse oximetry plethysmographic waveform during changes in blood volume. *British Journal of Anaesthesia*, *82*(2), 178–181. https://doi.org/10.1093/bja/82.2.178

Sharp, C. R., & Rozanski, E. A. (2014). Cardiovascular and systemic effects of gastric dilatation and volvulus in dogs. *Topics in Companion Animal Medicine*, *29*(3), 67–70. https://doi.org/10.1053/j.tcam.2014.09.007

Swenson, J., Henao-Guerrero, P. N., & Carpenter, J. W. (2008). Clinical technique: Use of capnography in small mammal anesthesia. *Journal of Exotic Pet Medicine*, *17*(3), 175–180. https://doi.org/10.1053/j.jepm.2008.05.004

Tabrizchi, R., & Iida, N. (2003). Electromagnetic blood flow measurements. In J. Moore & G. Zouridakis (Eds.), *Biomedical technology and devices handbook* (pp. 50–69). CRC Press LLC.

Thawley, V., & Waddell, L. S. (2013). Pulse oximetry and capnometry. *Topics in Companion Animal Medicine*, *28*(3), 124–128. https://doi.org/10.1053/j.tcam.2013.06.006

Vigani, A. (2015). Cardiac output measurement. In K. A. Grimm, L. A. Lamont, W. J. Tranquilli, S. A. Greene, & S. A. Robertson (Eds.), *Veterinary anesthesia and analgesia: The fifth edition of lumb and jones* (pp. 473–482). John Wiley & Sons.

Von Spiegel, T., Wietasch, G., Bürsch, J., & Hoeft, A. (1996). [Cardiac output determination with transpulmonary thermodilution. An alternative to pulmonary catheterization?] *Der Anaesthesist*, *45*(11), 1045–1050. https://doi.org/10.1007/s001010050338

Waddell, L. S., & Brown, A. J. (2015). Hemodynamic monitoring. In D. C. Silverstein & K. Hopper (Eds.), *Small animal critical care medicine* (2nd ed., pp. 957–962). Elsevier.

Wagner, A. E., & Brodbelt, D. C. (1997). Arterial blood pressure monitoring in anesthetized animals. *Journal of the American Veterinary Medical Association*, *210*(9), 1279–1285.

Weil, M. H., Nakagawa, Y., Tang, W., Sato, Y., Ercoli, F., Finegan, R., Grayman, G., & Bisera, J. (1999). Sublingual capnometry: A new noninvasive measurement for diagnosis and quantitation of severity of circulatory shock. *Critical Care Medicine*, *27*(7), 1225–1229. https://doi.org/10.1097/00003246-199907000-00001

Yamashita, K., Ueyama, Y., Miyoshi, K., Igarashi, R., Kushiro, T., Umar, M. A., & Muir, W. W. (2007). Minimally invasive determination of cardiac output by transthoracic bioimpedance, partial carbon dioxide rebreathing, and transesophageal Doppler echocardiography in beagle dogs. *The Journal of Veterinary Medical Science*, *69*(1), 43–47. https://doi.org/10.1292/jvms.69.43

