

Arterial blood gas analysis in cardiac emergencies of dogs

H. J. Desouza¹, H. Vijayakumar^{2*}, M. Ranjithkumar³ and S. Kavitha⁴

¹Pawsitive Veterinary Speciality Clinic, Cuchelim, Goa- 403 517, Goa, India; ²Department of Clinics, Madras Veterinary College, TANUVAS, Chennai- 600 007, Tamil Nadu, India; ³Resident Veterinary Services Section, Madras Veterinary College, TANUVAS, Chennai- 600 007, Tamil Nadu, India; ⁴Department of Veterinary Clinical Medicine, Madras Veterinary College, TANUVAS, Chennai- 600 007, Tamil Nadu, India

Abstract

This attempt was made to study the prevalence of cardiac emergencies in canines and to analyze the changes in arterial blood gases, electrolytes and metabolites occurring as a result of these cardiac emergency conditions. Dogs presented to emergency and critical care unit were subjected for triage to identify the life-threatening cardiac emergency conditions. Diagnostic approaches included anamnesis, physical examination, ECG, and echocardiography followed by radiography and arterial blood gas, electrolyte and biochemistry analysis. Canine cardiac emergencies were 16 percent of the total emergency cases presented. Among them Dilated Cardiomyopathy (DCM) was common (46%), followed by arrhythmias (38%) and congestive heart failure and myocardial infarction (8% each). Respiratory alkalosis (75%) was the most common acid-base imbalance noted, followed by metabolic acidosis at (17%) and lastly mixed acidosis-alkalosis at (8%). Significant decrease in pCO₂, bicarbonate and base excess were recorded. The observed electrolyte abnormalities were hypocalcemia (92%), hyponatremia (85%), hypokalemia (54%) and hypochloremia (46%). Metabolic abnormalities such as hyperglycemia, hyperlactatemia and azotemia (elevated BUN and creatinine) were also present in these cases.

Keywords: Arterial blood gas, Cardiac diseases, Critical care, DCM, pCO₂

Highlights

- Dogs with DCM were mostly presented for cardiac emergencies.
- Respiratory alkalosis was the most commonly recorded acid-base imbalance among cardiac emergency patients.
- Significant decrease in pCO₂, bicarbonate and base excess were recorded.
- Hyperglycemia, hyperlactatemia and azotemia were also recorded in dogs with cardiac emergencies.

INTRODUCTION

Cardiac diseases in dogs are almost as frequent as in human patients. Approximately 10-15% of all dogs are affected by cardiovascular disease (MacPete, 2018). Even though extensive studies are done in canine cardiac disease, meager information has been reported in India (Hoque *et al.*, 2019). The identification of canine cardiac diseases has been delayed and neglected by owners due to a lack of awareness and knowledge and inadequate diagnostic facilities (Devi *et al.*, 2009). Delayed recognition and advancement of cardiac disease into advanced stages leading to the development of cardiac emergencies. Analysis of arterial blood gases (ABG) can provide valuable information on the cardiopulmonary and acid-base status of a critically ill veterinary patient. The most frequent complaint of patients with heart failure is development of dyspnoea and cardiac congestion leading to cardiogenic pulmonary edema and impaired gas exchange (Gehlbach and Geppert, 2004). Thus,

ABG enables for quick, bedside, point-of-care testing (POCT), providing critical information on oxygenation, ventilation, acid-base balance, and tissue hypoxia, all of which may be abnormal in heart failure (Cook and Rhodes, 2008).

Electrolyte and metabolic disorders are common and potentially life-threatening complications associated with heart failure. This manifests as a result of either pathophysiological compensatory mechanisms leading to neurohumoral activation or adverse events of therapy involved with heart failure. The knowledge of these pathophysiological mechanisms and early identification of these alterations are essential for the management of these patients (Urso *et al.*, 2015). Although the pathophysiological mechanisms involved in heart failure have been extensively studied (Erling and Mazzaferro, 2008), the literature on the role of ABG for early identification of these acid-base, electrolyte and metabolic abnormalities has not been sufficiently documented.

*Corresponding Author, E-mail: vijayvet1985@gmail.com

MATERIALS AND METHODS

Selection of patients and triage

During 3 months study period, seventy nine dogs brought to the Critical Care Unit, Madras Veterinary College Teaching Hospital in unstable conditions presented with signs of cardiac disease were selected for the study. Out of these, thirteen dogs with cardiac diseases were utilized for this study.

History and signalment

Accurate information about signalment, anamnesis and presenting complaint were recorded which included breed, age, sex, body weight, previous symptoms shown, nutritional history, medical history, urination and defecation history, changes in activity levels and mentation.

General inspection and physical examination

A general inspection was performed upon presentation to assess the general appearance, body condition score, presence of edema or ascites, mentation and respiratory effort. Physical examination involved heart auscultation for rate, rhythm, intensity of beats, and presence of any abnormal sounds or murmurs, followed by palpation of the femoral artery for detecting the pulse rate, rhythm, pressure and pulse deficits. Lung auscultation was performed alongside of percussion to assess the presence of any abnormal sounds like crackles, moist rales and wheezes. Assessment of other vital parameters such as measuring rectal temperature, blood pressure, pulse oximetry, conjunctival and buccal mucus membranes, capillary refill time and lymph node palpation were performed. The abdomen was palpated to detect presence of any organomegaly, fluid thrill of ascites or excessive distension of bladder. The limbs were palpated to check for presence of peripheral edema.

Diagnostic approach

ECG and echocardiography: Upon detection of any abnormal findings in cardiac auscultation and pulse

palpation, the patient was subjected to an ECG examination. The ECG was recorded by placing the patient in either sternal recumbency or right lateral recumbency, on a non-conductive surface. The hair at the point of electrode placement was clipped, and electrode gel or alcohol was applied to increase the contact between the skin and electrodes. The placement of the electrodes was based on the colour coding [Yellow (positive) = left forelimb, Red (negative) = right forelimb, Green = left hindlimb, Black = right hindlimb] and were attached to the respective limbs using crocodile clips. ECG was monitored for presence of arrhythmias, conduction abnormalities or metabolic abnormalities. Bedside echocardiographic examination was performed as per standard protocol to further assess abnormalities in cardiovascular structure and function in emergency conditions. Four views were visualized as follows: 1. Four-chamber right-sided parasternal long-axis view, 2. Five-chamber right-sided parasternal long-axis view, 3. Right-sided short-axis view of the left ventricle at the level of the papillary muscles, 4. Right-sided short-axis view at the level of the left atrium and aorta.

Radiography: Radiography of the thorax and abdomen was performed for dogs in relatively stable condition, if found unstable, patients were pre-oxygenated and stabilized before proceeding. Patients were restrained in lateral or sternal recumbency for thoracic radiograph and in lateral recumbency for abdominal radiograph.

Arterial blood gas analysis: The arterial blood sample was collected from femoral artery using heparin coated 1 mL tuberculin syringe and the sample was analysed using Blood gas analyser (epoc® Blood Analysis System) (Fig. 1 and 2). Following parameters were obtained from the epoc® Blood Analysis System. 1. Gases: i. pH – Acidity/ alkalinity of blood, ii. pCO_2 – Partial pressure of carbon dioxide (mmHg), iii. pO_2 – Partial pressure of oxygen (mmHg), iv. $cHCO_3^-$ –



Fig. 1. Arterial blood collection from femoral artery



Fig. 2. Processing of arterial blood in Arterial blood gas analyzer

Bicarbonate concentration (mmol/L), v. BE (ecf) – Base Excess (mmol/L), vi. cSO_2 – Oxygen saturation of blood (%). 2. Chemistry: i. (Na^+) – Sodium (mmol/L), ii. (K^+) – Potassium (mmol/L), iii. (Ca^{++}) – Calcium (mmol/L), iv. (Cl^-) – Chloride (mmol/L), v. TCO_2 – Total carbon dioxide (mmol/L), vi. AGap – Anion gap (mmol/L), vii. Hct – Hematocrit/Packed cell volume (%), viii. cHgb – Haemoglobin concentration (g/dL). 3. Metabolite: i. Glu – Glucose (mg/dL), ii. Lac – Lactate (mmol/L), iii. BUN – Blood Urea Nitrogen (mg/dL), iv. Urea (mmol/L), v. Creatinine (mg/dL), vi. BUN/Creatinine ratio (mmol/mmol), vii. Urea/Creatinine ratio (mmol/mmol)

RESULTS

During the study period of 3 months, 79 canine emergency cases were presented to the Critical Care Unit (CCU), out of which 13 were cardiac emergencies. Cardiac emergencies in dogs contribute about 16% (13/79) of total emergency cases, indicating the significance of cardiac diseases. In this study, the most frequently presented cardiac emergency condition was Dilated Cardiomyopathy (DCM) at 46% (6/13), followed by Arrhythmias at 38% (5/13) and lastly, Congestive Heart Failure and Myocardial Infarction at 8% (1/13) each.

Labrador was the most commonly affected breed at 38% (5/13). Dogs of the geriatric age group 8-12 years had

the highest presentation with 54% (7/13) in comparison to those of the age group less than 1 year and 1-8 years old, both at 23% (3/13) each. Males were more commonly affected sex at 77% (10/13) than females at 23% (3/13).

The most common presenting clinical signs were dyspnea and exercise intolerance at 85% each, followed by sternal or lateral recumbency at 61%, abdominal distention at 38%, limb edema at 23% (Fig. 3) and syncope at 15%. Dull and depressed mentation was a common finding in 85% of cases, while only 15% of cases were active and alert. 54% of cases presented had a normal body condition score, while 38% presented with poor body condition and 8% were obese.

Tachycardia was a common finding in 69% cases along with abnormal rhythm in 61% cases. Respiratory distress was seen in 77% cases, and tachypnea was a common finding among 69% cases, while bradypnea was seen in 15% cases. Pale mucus membranes were seen in 77% with a prolonged capillary refill time of more than 2 seconds, and congested mucus membranes were seen in 23% cases with a shorter capillary refill time of less than 2 seconds.

In ECG, arrhythmias were observed in 38% of cases, among which ventricular tachycardia (Fig. 4) was the most common (50%). In echocardiography, chamber enlargement associated with dilated cardiomyopathy (Fig. 5) was the most common abnormality seen in 46% of cases. Pleural effusion



Fig. 3. Ascites (Pot belly and fluid thrill on tactile percussion) and limb edema in a Labrador

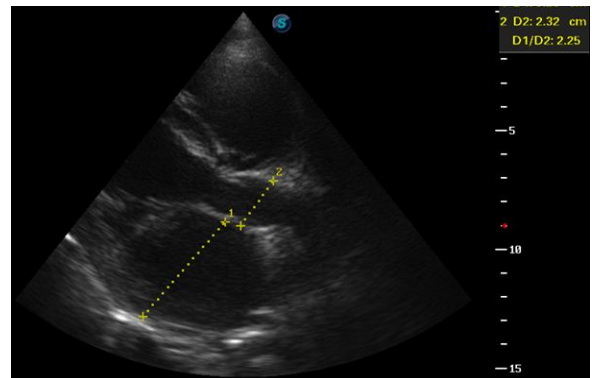


Fig. 5. Right parasternal short axis view of echocardiography with increased LA/Ao ratio (2.25) suggestive of DCM

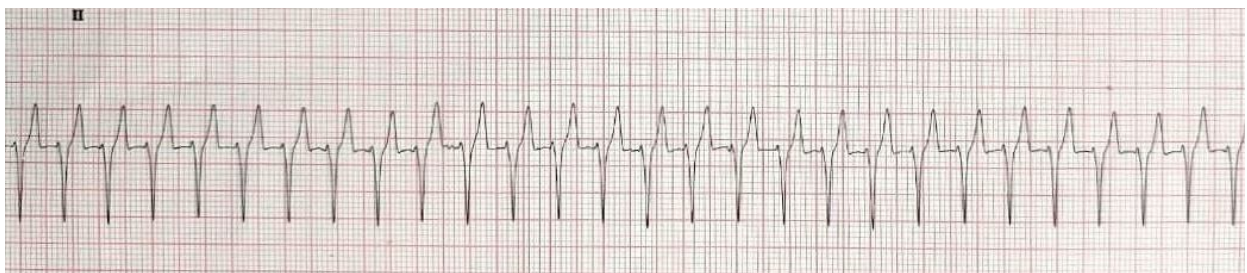


Fig. 4. ECG showing sustained ventricular tachycardia in a dog (absence of P wave, heart rate >150bpm)

(Fig. 6) and ascites were recorded in 46% and 38% cases, respectively on radiographic examination.

Acid-base: In this study, 92% of patients showed acid-base imbalances (Table 1). Among these, most common acid-base abnormalities were associated with respiratory alkalosis (75%), followed by metabolic acidosis (17%) and mixed acidosis-alkalosis (8%), and the rest 8% cases showed a normal acid-base balance (Table 1). The mean change in pH (7.44 ± 0.02) was shown to be statistically non-significant, but on individual case basis, the acidotic patients had a pH from as low as 7.263 to as high as 7.559 in alkalotic dogs.

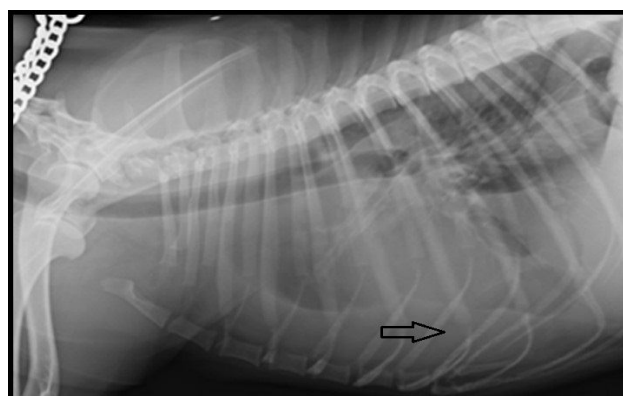


Fig. 6. Left lateral thoracic radiograph of a dog showing pleural effusion (arrow)

Table 1. Arterial blood gas analysis in cardiac emergencies

Parameter	Groups		PV	F
	Healthy Control (Mean $\pm$ S.E.)	Cardiac Disease (Mean $\pm$ S.E.)	alue	Value
pH	7.40 ± 0.02	7.44 ± 0.02^{ns}	0.290	0.427
pCO ₂ (mmHg)	37.26 ± 1.33	$20.77 \pm 1.72^{**}$	0.000	3.968
pO ₂ (mmHg)	99.06 ± 2.35	83.58 ± 9.91^{ns}	0.313	13.621
cHCO ₃ ⁻ (mmol/L)	20.34 ± 0.86	$13.82 \pm 1.08^{**}$	0.001	1.657
BE (ecf) (mmol/L)	0.11 ± 0.97	$-10.35 \pm 1.17^{**}$	0.000	1.140
cSO ₂ (%)	97.60 ± 0.90	93.56 ± 1.62^{ns}	0.122	5.259

ns – Non significant ($P \geq 0.05$), ** – Highly significant ($P \leq 0.01$)

Table 2. Blood biochemistry analysis in cardiac emergencies

Parameter	Groups		P	F
	Healthy Contro (Mean $\pm$ SE)	Cardiac Disease (Mean $\pm$ SE)	Value	Value
Na ⁺ (mmol/L)	141.83 ± 1.25	$126.92 \pm 2.88^{**}$	0.003	6.190
K ⁺ (mmol/L)	4.81 ± 0.15	$4.08 \pm 0.16^{**}$	0.012	1.622
Ca ⁺⁺ (mmol/L)	1.25 ± 0.03	$0.88 \pm 0.06^{**}$	0.001	4.834
Cl ⁻ (mmol/L)	110.83 ± 1.42	105.31 ± 2.81^{ns}	0.216	2.090
TCO ₂ (mmol/L)	24.77 ± 0.95	$14.82 \pm 0.99^{**}$	0.000	1.529
A Gap (mmol/L)	10.90 ± 2.29	8.85 ± 1.51^{ns}	0.458	0.406
Hct (%)	30.83 ± 2.29	24.62 ± 2.94^{ns}	0.198	1.057
cHgb (g/dL)	11.85 ± 0.98	9.39 ± 1.02^{ns}	0.132	0.120
BE (b) (mmol/L)	0.50 ± 0.99	$-8.73 \pm 1.10^{**}$	0.000	0.557

ns – Non significant ($P \geq 0.05$), ** – Highly significant ($P \leq 0.01$)

Partial pressure of carbon dioxide and oxygen: A highly significant drop in mean pCO₂ (20.77 ± 1.72 mmHg) along with TCO₂ (14.82 ± 0.99 mmol/L) was recorded. Non-significant change was found in mean pO₂ levels (83.58 ± 9.91 mmHg). This might be partly due to flow by oxygen supply to the patient as part of the initial stabilisation. Although in cases of DCM drastic reduction in the levels of pO₂ has been recorded even with prior oxygenation.

Electrolyte changes: Changes in the electrolyte levels were recorded in Table 2. In the present study, hyponatremia was a common finding in 85% of cases. Significantly decreased levels in mean sodium (126.92 ± 2.88 mmol/L) and mean potassium (4.08 ± 0.16 mmol/L) were recorded in cardiac emergency cases. Hypokalemia was recorded in 54% of cases. Hypocalcaemia was seen in 92% of cases with a significant decrease in mean calcium (90.88 ± 0.06 mmol/L).

Bicarbonate and base excess:

The mean bicarbonate concentration (13.82 ± 1.08 mmol/L) significantly decreased. All affected dogs showed low bicarbonate values below the reference range. Significant change in mean base excess (-10.35 ± 1.17 mmol/L) was found in 92% of cases.

Anion Gap: Mean Anion Gap (8.85 ± 1.51 mmol/L) change in the diseased group was non-significant as compared to the healthy control (Table 2).

mmol/L) level as compared to healthy control. The extent of hypochloremia with mean chloride (105.31 ± 2.81 mmol/L) was statistically non-significant. However, 46% of cases have independently shown the presence of hypochloremia.

Metabolite changes:

Changes in the blood metabolites are presented in Table 3. The mean glucose

(106.46 ± 5.86 mg/dL) value was statistically non-significant. The change in mean lactate (3.12 ± 0.88 mmol/L) has been shown to be statistically non-significant in the disease group from the healthy group. There is no statistical significance to the change in mean BUN (32.62 ± 4.75 mg/dL) level. A significant increase in the mean creatinine (2.10 ± 0.36 mg/dL) level was recorded in 61% of total cases.

DISCUSSION

MacPete (2018) stated that approximately 10-15% of all dogs globally are affected by cardiovascular disease. Common acid-base abnormality was respiratory alkalosis, which is in agreement with the study by Kuwahara and Kawai (1992). Low $p\text{CO}_2$ levels can be attributed due to development of pleural effusion resulting in hyperventilation (Natanzon and Kronzon, 2009). However, a reduced bicarbonate level is contradictory to studies done in human models of heart failure, which showed elevated levels of bicarbonate (Cooper *et al.*, 2016). The base deficit can be attributed to the presence of alkalotic state due to reduced level of bicarbonate concentration. However, Nakano *et al.* (2020) stated that a low base excess holds no significant prognostic value. Mean anion gap change was non-significant, which is in contrast to studies of Gabow *et al.* (1980), who had stated that a 62% of elevation in anion gap in heart failure. Hyponatremia could be due to volume overload causing dilutional hypervolemic hyponatremia as stated by Rodriguez *et al.* (2019). Hypokalemia in canine cardiac emergency cases was in agreement with Alper *et al.* (2009). Jensen *et al.* (2019) also observed hypocalcemia in cardiac cases, which was observed in this study. In this study, 46% of cases independently showed the presence of hypochloremia, which is in line with the studies done by Magder and Emami (2015). The non-significant increased level of glucose

Table 3. Blood metabolite changes in cardiac emergencies

Parameter	Groups		P Value	F Value
	Healthy Contro (Mean $\pm$ SE)	Cardiac Disease (Mean $\pm$ SE)		
Glucose (mg/dL)	88.83 $\pm$ 3.96	106.46 $\pm$ 5.86 ^{ns}	0.071	4.957
Lactate (mmol/L)	0.74 $\pm$ 0.16	3.12 $\pm$ 0.88 ^{ns}	0.090	3.694
BUN (mg/dL)	19.83 $\pm$ 2.27	32.62 $\pm$ 4.75 ^{ns}	0.096	10.097
Urea (mmol/L)	5.31 $\pm$ 0.92	11.23 $\pm$ 1.78*	0.039	6.625
Creatinine (mg/dL)	0.88 $\pm$ 0.17	2.10 $\pm$ 0.36*	0.040	6.179
BUN/Creatinine	15.83 $\pm$ 1.17	18.19 $\pm$ 2.05 ^{ns}	0.466	2.894
Urea/Creatinine	66.95 $\pm$ 5.08	74.45 $\pm$ 11.95 ^{ns}	0.674	1.398

ns – Non significant ($P \geq 0.05$), * – Significant ($P \leq 0.05$)

(hyperglycemia) was seen in 61% of cases which is suspected to be a result of insulin receptor antagonism, defects in insulin signal transduction and impaired insulin-mediated glucose uptake in heart failure (Tidholm and Jonnson, 1997; Friehs *et al.*, 1999; Paolisso *et al.*, 1999). Nevertheless, hyperlactatemia was seen in 38% of cases as previously studied by Stanley and Chandler (2002). Peripheral vasoconstriction in heart failure causes reduced blood flow, causing anaerobic metabolism, and is thus used as an indicator for impaired tissue perfusion (Boag and Hughes, 2005). Elevated BUN in the present study could be a result of renal reabsorption simultaneously with sodium and water as a compensatory mechanism of heart failure, as stated by Aronson and Burger (2010). Increase in creatinine levels could be a result of impaired cardiac output and renal ischemia as stated by Winton (1931). This rise in creatinine in patients with heart failure is known as Cardio-Renal Syndrome and is consistent with findings of Adams *et al.* (2005).

In conclusion, Arterial blood gas analysis is an effective tool to measure acid base, blood gas, electrolyte and metabolic abnormalities in the emergency patient. The rapid and mobile nature of this tool is of major benefit in the emergency and critical care setup in which timely diagnosis and management is of utmost importance. It includes assessing parameters of disease progression as well as prognostic importance. It has shown to be essential in the detection of changes in the blood picture which aids with quick diagnosis. This specific analysis also aids in the selection of an optimum treatment plan tailored to each patient's requirements and, thus providing effective and efficient therapy and recovery.

Conflict of interest: Authors have no conflict of interest in this study.

Author's contribution: HJD: Involved in the investigation, data generation, and preparing original draft; HVK: Engaged in conceptualization, data curation, supervision and final editing; MR: Involved in statistical analyses, methodology and editing; SK: Conceptualization and overall supervision.

Data availability statement: All data underlying the

REFERENCES

- Adams KF Jr, Fonarow GC, Emerman CL, LeJemtel TH and Costanzo MR, 2005. Characteristics and outcomes of patients hospitalized for heart failure in the United States: rationale, design, and preliminary observations from the first 100,000 cases in the Acute Decompensated Heart Failure National Registry (ADHERE). *Am Heart J*, 149(2): 209-216, doi: 10.1016/j.ahj.2004.08.005
- Alper AB, Campbell RC, Anker SD, Bakris G, Wahle C *et al.*, 2009. A propensity-matched study of low serum potassium and mortality in older adults with chronic heart failure. *Int J Cardiol*, 137(1): 1-8, doi: 10.1016/j.ijcard.2008.05.047
- Aronson D and Burger AJ, 2010. The relationship between transient and persistent worsening renal function and mortality in patients with acute decompensated heart failure. *J Card Fail*, 16(7): 541-547, doi: 10.1016/j.cardfail.2010.02.001
- Boag AK and Hughes D, 2005. Assessment and treatment of perfusion abnormalities in the emergency patient. *Vet Clin North Am Small Anim Pract*, 35(2): 319-342, doi: 10.1016/j.cvsm.2004.10.01
- Cook MJ and Rhodes A, 2008. Arterial blood gas analysis in acute heart failure syndrome. In: *Acute Heart Failure* (Mebazaa A, Gheorgiade M, Zannad FM, Parrillo JE, eds). Springer, London, doi: 10.1007/978-1-84628-782-4_42
- Cooper LB, Mentz RJ, Gallup D, Lala A, DeVore AD *et al.*, 2016. Serum bicarbonate in acute heart failure: relationship to treatment strategies and clinical outcomes. *J Card Fail*, 22(9): 738-742, doi: 10.1016/j.cardfail.2016.01.00
- Devi S, Jani RG, Anne FK and Singh RD, 2009. Study on clinical symptoms in canine cardiac diseases. *Vet World*, 2(8): 307-309
- Erling P and Mazzaferro EM, 2008. Left-sided congestive heart failure in dogs: pathophysiology and diagnosis. *Compendium (Yardley, PA)*, 30(2): 79-91
- Friebs I, Moran A, Stamm C, Colan S, Takeuchi K *et al.*, 1999. Impaired glucose transporter activity in pressure-overload hypertrophy is an early indicator of progression to failure. *Circulation*, 100(19 Suppl): II 187-93, doi: 10.1161/01.cir.100.suppl_2.ii-187
- Gabow PA, Kaehny WD, Fennessey PV, Goodman SI, Gross PA *et al.*, 1980. Diagnostic importance of an increased serum anion gap. *N Engl J Med*, 303(15): 854-858, doi: 10.1056/NEJM198010093031505
- Gehlback BK and Geppert E, 2004. The pulmonary manifestations of left heart failure. *Chest*, 125(2): 669-682, doi: 10.1378/chest.125.2.669
- Hoque M, Saxena AC, Bashir M and Bodh D, 2019. Cardiac diseases in dogs. *Indian J Anim Health*, 58(1): 01-20, doi: 10.36062/ijah.58.1.2019.01-20
- Jensen AC, Polcwiartek C, Sogaard P, Mortensen RN, Davidsen L *et al.*, 2019. The association between serum calcium levels and short-term mortality in patients with chronic heart failure. *Am J Med*, 132(2): 200-208, doi: 10.1016/j.amjmed.2018.10.006
- Kuwahara T and Kawai C, 1992. Acid-base disturbances in heart failure. *Nihon Rinsho*, 50(9): 2173-2177
- MacPete R, 2018. *Dogs and Heart Disease: An Overview*. IDEXX Laboratories Inc
- Magder S and Emami A, 2015. Practical approach to physical-chemical acid-base management. *Stewart at the bedside. Ann Am Thorac Soc*, 12(1): 111-117, doi: 10.1513/AnnalsATS.201409-426OI
- Nakano H, Nagai T, Honda Y, Honda S, Iwakami N *et al.*, 2020. Prognostic value of base excess as indicator of acid-base balance in acute heart failure. *Euro Heart J Acute Cardiovasc Care*, 9(5): 399-405, doi: 10.1177/2048872619898781
- Natanzon A and Kronzon I, 2009. Pericardial and pleural effusions in congestive heart failure-anatomical, pathophysiologic, and clinical considerations. *Am J Med Sci*, 338(3): 211-216, doi: 10.1097/MAJ.0b013e3181a3936f
- Paolisso G, Tagliamonte MR, Rizzo MR, Gambardella A, Gualdiero P *et al.*, 1999. Prognostic importance of insulin-mediated glucose uptake in aged patients with congestive heart failure secondary to mitral and/or aortic valve disease. *Am J Cardiol*, 83(9): 1338-1344, doi: 10.1016/s0002-9149(99)00097-1
- Rodriguez M, Hernandez M, Cheungpasitporn W, Kashani KB, Riaz I *et al.*, 2019. Hyponatremia in heart failure: pathogenesis and management. *Curr Cardiol Rev*, 15(4): 252-261, doi: <https://doi.org/10.2174/1573403X15666190306111812>
- Stanley WC and Chandler MP, 2002. Energy metabolism in the normal and failing heart: potential for therapeutic interventions. *Heart Fail Rev*, 7(2): 115-130, doi: 10.1023/a:1015320423577
- Tidholm A and Jönsson L, 1997. A retrospective study of canine dilated cardiomyopathy (189 cases). *J Am Anim Hosp Assoc*, 33(6): 544-550, doi: 10.5326/15473317-33-6-544
- Urso C, Brucculeri S and Caimi G, 2015. Acid-base and electrolyte abnormalities in heart failure: pathophysiology and implications. *Heart Fail Rev*, 20(4): 493-503, doi: 10.1007/s10741-015-9482-y
- Winton FR, 1931. The influence of venous pressure on the isolated mammalian kidney. *J Physiol*, 72(1): 49-61, doi: 10.1113/jphysiol.1931.sp002761

Received- 05.04.2024, Accepted- 05.09.2024, Published- 03.10.2024 (Online), 01.06.2025 (Print)

Section Editor: Dr. S. Biswas, Section Editor