

International Journal of Advanced Biochemistry Research



ISSN Print: 2617-4693
ISSN Online: 2617-4707
NAAS Rating (2026): 5.29
IJABR 2026; 10(4): 424-428
www.biochemjournal.com
Received: 15-01-2026
Accepted: 18-02-2026

Dr. Rahul Paul
Assistant Professor,
Department of Veterinary
Surgery & Radiology, Institute
of Veterinary Science &
Animal Husbandry, Siksha 'O'
Anusandhan-Deemed to be
University, Bhubaneswar,
Odisha, India

Dr. Analisha Debbarma
Assistant Professor,
Department of Veterinary
Surgery & Radiology, College
of Veterinary Sciences &
Animal Husbandry, Selesih,
Mizoram, under Central
Agricultural University,
Imphal, India

Corresponding Author:
Dr. Rahul Paul
Assistant Professor,
Department of Veterinary
Surgery & Radiology, Institute
of Veterinary Science &
Animal Husbandry, Siksha 'O'
Anusandhan-Deemed to be
University, Bhubaneswar,
Odisha, India

Importance of blood gas analysis on acid-base imbalance diagnosis in correlation to veterinary anaesthesia & emergency care management: A comprehensive review

Rahul Paul and Analisha Debbarma

DOI: <https://www.doi.org/10.33545/26174693.2026.v10.i4f.8172>

Abstract

Arterial blood gas (ABG) analysis is a highly effective & standard diagnostic tool for detection of acid base imbalances in the body which either due to metabolic or respiratory system alterations. ABG analyzes parameters like blood pH, $p\text{CO}_2$, $p\text{O}_2$, HCO_3^- , lactate, BEb, BEecf etc to determine whether the alterations are due to acidosis or alkalosis. Primary indicators of acid-base imbalance are pH, $p\text{CO}_2$, HCO_3^- . Alterations in these three parameters are correlated in acid-base imbalance & compensatory response mechanism of body. Different types of acid-base imbalances are treated with different treatment protocols with a common concept of elimination of primary cause. Anaesthesia becomes critical as well as challenging in acid-base imbalanced patients. Understanding of drug pharmacology, selection of drugs, doses, anaesthetic techniques, continuous physiological & haematological assessment, prompt responses to emergencies during anaesthesia all these factors plays crucial role for a successful completion of anaesthetic protocol in a acid-base imbalanced patients.

Keywords: ABG analysis, acid-base imbalance, compensatory mechanism, anaesthesia, Anaesthetic emergency

Introduction

Arterial blood gas analysis (ABG) helps to measure the important physiological variables such as tissue oxygen perfusion, carbon dioxide and blood pH. ABG used as standard diagnostic test for metabolic imbalance and respiratory dysfunction (Turlock III *et al.*, 1990, Byrne *et al.*, 2013, Waddell., 2016, Balakrishnan and King, 2014, Swathi *et al.*, 2020) [27, 29, 2, 24]. Analysis of blood gases can provide valuable information on the cardiopulmonary and acid-base status of a critically ill veterinary patient which helps in quick diagnosis along with disease prognosis (Buduk *et al.*, 2019, Desuza *et al.* 2025) [3, 8]. ABG determines the sufficiency of gaseous exchange (O_2 & CO_2) to evaluate oxygenation and ventilation status of animals during emergency & critical patient management. (Topal & Gul 2006, Irizzary & Reiss; 2009, Sidari., 2021) [25, 12, 23]. Blood gas analyzing is more accurate than the pulse oximeter for obtaining information about oxygenation, ventilation and the acid-base status of the body during in ECC & anaesthetic patients (Atalan *et al.*, 2008, Jeong *et al.* 2017) [9, 13]. In general, arterial blood is used for blood gas analysis, but few studies have demonstrated a good correlation of indicators for pulmonary function and acid base balance among arterial, capillary and venous samples in humans and animals (Jeong *et al.* 2017) [13]. In a complete arterial blood gas analysis parameters like blood pH, $p\text{CO}_2$, $p\text{O}_2$, electrolytes (Na^+ , K^+ , Cl^-), blood HCL, bicarbonate (HCO_3^-). Oxygenation, ventilation & circulation are three main factors to assure of adequate tissue & organ perfusion with oxygenated blood during anaesthesia maintenance (Ko & Krimins; 2012) [15]. Capnography monitor & assess the ETCO_2 levels at anaesthetic monitoring stage to evaluate the depth of anaesthesia and although ETCO_2 cannot estimate all ABG variables, but it can provide a brief estimate of HCO_3^- and PaCO_2 in critical, emergency situations.

Sample collection & analysis

For arterial blood gas (ABG) analysis restraining of animal followed by collection of blood

& immediate analysis of blood sample is required. Arterial blood samples can be collected from femoral artery (very common site), dorsal metatarsal or caudal artery in a heparinised syringe as per standard protocol. For venous blood gas analysis blood can be drawn from jugular vein for best accuracy. Blood sample should be analyzed by using blood gas analyzer machine or stored into a mixture of ice and water until analysis. Ideally blood gas analysis should be done as soon as possible after collection of sample or within 30-60 min because continuous metabolic activity and diffusion of gases in the sample syringe can provide false report & interpretations (Turlock III *et al.*, 1990, Kules *et al.* 2015, Waddell., 2016, Jeong *et al.* 2017, Buduk *et al.*, 2019, Sidari., 2021, Palanivelrajan *et al.* 2025) [7, 16, 29, 13, 3, 23, 18].

Table 1: Normal reference ranges of different ABG parameters (Kules *et al.* 2015) [16]

Parameters	Reference range with unit
pH	7.37-7.44
Partial pressure of carbon dioxide (pCO ₂)	22.62-34.52 mmHg
Partial pressure of oxygen (pO ₂)	91.35-106.05 mmHg
Bicarbonate level (HCO ₃ ⁻)	14.06-22.10 mmol/L
Standard bicarbonate concentration (SBC)	16.59-24.74 mmol/L
Base excess of extracellular fluid (BE _{ecf})	-12.05-1.61 mmol/L
Base excess of the blood (BE _b)	-9.7-0.46 mmol/L
Oxygen saturation (SO ₂)	94.31-100%
Arterial oxygen content (CaO ₂)	13.69-29.77 ml O ₂ /dL
Capillary oxygen content (CcO ₂)	13.59-29.56 ml O ₂ /dL
Respiratory index (Ri)	0.10-0.20
Partial pressure of O ₂ & fraction of inspired O ₂ ratio (pO ₂ /FiO ₂)	437.00-507.45 mmHg
Anion gap (AG)	5.49-19.67 mmol/L
Lactate (Lac)	0.06-3.54 mmol/L
Alveolar oxygen (A)	106.33-120.12 mmHg
Total carbon dioxide (TCO ₂)	14.87-23.06 mmol/L

Discussion on pathophysiology of acid base imbalance, analysis methodology & treatment protocol: The pH value of normal blood is maintained in between 7.35-7.45 by its buffer systems. When pH decreases below 7.35 it is considered as acidosis whereas alkalosis happens when pH increases above 7.45. This change in acid-base balance towards acidosis or alkalosis is due to respiratory or metabolic abnormalities also acid base balance or imbalance of ECF depends on relative quantity of carbonic acid & bicarbonate base which is normally 1:20 (carbonic acid: bicarbonate). Acidosis is a clinical condition which is developed due to development of acidemia where H⁺ ion concentration level in blood increases leads to decrease in blood pH where as alkalosis develops due to alkalemia where H⁺ ion concentration level in blood decreases leads to increase in blood pH. Acidosis is mainly classified into two types: metabolic acidosis & respiratory acidosis; similarly alkalosis is also classified into metabolic alkalosis & respiratory alkalosis. Metabolic acidosis developed due to increase in production of acids in the body which leads to sudden decrease in pH and bicarbonate levels (HCO₃⁻) as decrease in HCO₃⁻ in extra cellular fluid leads to decrease in pH. Most common conditions/diseases related to metabolic acidosis are dehydration, shock, diabetes mellitus, enteritis acute & chronic kidney diseases etc. Respiratory acidosis is mostly related with clinical conditions like respiratory diseases (pneumonia, copd, asthma etc.), anaesthesia, CNS diseases (meningitis, traumatic injury to brain etc.). This

condition is developed due to decrease in pH level due to increase in plasma carbon dioxide (pCO₂) as a result of decreased ventilation. Metabolic alkalosis occurs due to increase in pH & HCO₃⁻ levels, mostly associated with conditions like vomiting, pyloric stenosis, medications like diuretics etc. Respiratory alkalosis develops most in cases like toxemia, anaemia, hypoxaemia which ultimately leads to hyperventilation where excessive CO₂ is blown off leading to respiratory alkalosis. Compensatory mechanism of acid-base balance is a biological & physiological protective response of the body to correct the significant alteration of pH value (decrease in acidosis or increased in alkalosis) than normal standard level. Compensatory mechanism mainly regulated by 3 physiological responses-buffer systems (bicarbonate buffer, phosphate buffers & protein buffers), renal system compensatory mechanism & respiratory system compensatory mechanism. Buffer systems & respiratory system acts as a front line defence whereas renal mechanism acts as second line of defence to compensate acid-base imbalance. Compensatory mechanism acts by regulating secondary acid-base parameters to counter the disturbances of primary acid base parameter; When acid base imbalance happens due to metabolic disturbance compensatory mechanism acts on respiratory compensatory system to correct acid base balance, similarly it acts on metabolic compensatory systems to counter acid-base disturbances originated due to deviation of respiratory function. To treat different metabolic imbalances, different treatment and managemental protocols are implemented. In all type of acid-base imbalance there is a primary cause so the elimination the route is most important for complete recovery. In case of metabolic acidosis treatment line includes IV fluid therapy with M/6 sodium lactate, ringer lactate along with oral, IV sodium bicarbonate supplementation to reduce acid level as well as compensate excess bicarbonate loss. 100% oxygen supply will improve the ventilation status of patient. Patient of respiratory acidosis can be treated with bronchodilators like deriphylline inj. and broncho-stimulators like doxapram inj., they improves O₂ & CO₂ exchange by bronchodilation & also improves diaphragmatic contractility. Endotracheal intubation with tracheostomy if needed followed by oxygen supply or in severe cases mechanical ventilation to promote ventilation status. Appropriate fluid therapy. Respiratory alkalosis can be treated mainly with route cause or primary cause elimination which triggers hyperventilation and PCO₂ decrease. Anti-anxiety drugs (anxiolytics-example: benzodiazepines) can provide relief from anxiety related hyperventilation which leads to development of respiratory alkalosis. Patient with metabolic alkalosis can be treated with IV fluid therapy of 0.9% NaCl and RL solution. Inj. H₂ blocker like ranitidine or proton pump inhibitors like pantoprazole and antiemetic drugs like ondansetron are very useful if vomiting is the route cause of metabolic alkalosis. Oxygenation can improve the patient condition with concurrent medication. (Irizzary & Reiss; 2009, Chatterjee and Shinde., 2012, Waddell., 2013, Grimm *et al.*, 2015, Waddell., 2016, Thorp & LIss., 2017, King & Boag., 2018, Sidari., 2021, Chakrabarty., 2021, Sharma *et al.*, 2021) [12, 6, 28, 10, 29, 26, 14, 23, 5, 22]. Along with central nervous system, cardiopulmonary & renal systems are also got compromised during general anaesthesia and patients with nonclinical acid-base imbalance can be present with clinical symptoms of acid-base imbalance during or after anaesthesia, so

selection of drugs, doses and techniques should be planned by correlating with high risky patient status is necessary (Hall *et al.*, 2001, Grimm *et al.*, 2015) [11, 10]. Capnography performs multiple tasks like assessment respiratory rate, estimation of PaCO₂, EtCO₂, fraction of inspired CO₂ (FiCO₂) during anaesthesia. Although directly PaCO₂ is not measured by capnograph but usually it is 3-5 mm/Hg higher than EtCO₂. It also detects anaesthetic emergencies like apnoea, hyperventilation (hypocapnia), hypoventilation (hypercapnia) which gives a primary information to diagnose acid base imbalances like (metabolic acidosis, respiratory acidosis, respiratory alkalosis) (Topal & Gul., 2006, Waddell., 2013, King & Boag., 2018, Sidari., 2021, Clancy., 2023) [25, 28, 14, 23, 7].

Discussion on Clinical case Analysis methodology in acid-base imbalances with correlation to anaesthesia & emergency management:

Hypothetically consider a case of chronic pyometra in a old bitch is presented with most common clinical symptoms of pyometra like, depression, anorexia, small pus discharge from vagina, injected mucous membrane as a result of systemic infection, toxemia & septicaemia, secondary urinary infection due to pyometra etc. In-appetite, systemic infection, toxemia & septicaemia all these potential factors mostly leads to development of metabolic acidosis condition in this case. ABG findings in such cases mostly revealed low pH, low HCO₃⁻ and high lactate value, for compensation hyperventilation can be present. For correction of this condition, radical treatment-ovariohysterectomy should be done, with fluid therapy (RL, NS), Oxygen supplementation, antibiotics & supportive treatment can be advise. ABG changes due to metabolic acidosis is an alteration of normal body homeostasis results in decrease of both blood pH & bicarbonate level with increase in acid production. As a result of these changes cardiovascular system is also shows profound alternations with most common changes like-I. decrease in myocardial contractility II. decrease in cardiac output III. decrease in blood pressure (hypotension). IV. early stage tachycardia followed by bradycardia at later stage. All these changes are interlinked and influenced by multiple factors like excess H⁺ ion, hyperkalemia, decrease of catecholamine's response etc. act as a chain reaction if not treated appropriately on emergency basis. Management of such emergency situations needs proper diagnosis, accurate treatment plan and continuous monitoring of patient. Route cause diagnosis & elimination is most important part of treatment plan. IV fluids to correct electrolyte imbalance, antibiotics for control of toxemia/infection, oxygenation to reduce anaerobic metabolism and adequate tissue perfusion, Inj. of sodium bicarbonate etc are already have been discussed earlier. Along with these for symptomatic correction of hypotension inj. nor-epinephrine can be given. To counteract hyperkalemia calcium gluconate can be given intravenously. All those combined treatment protocol can help to resolve these conditions. In addition to cardiopulmonary changes metabolic acidosis also leads to significant variation on pharmacokinetic & pharmacodynamic profile of anaesthetic drugs. In this condition potency or sensitivity of anaesthetic drugs of different classes (benzodiazepines-diazepam, midazolam; opioids-fentanyl; induction agents-propofol, etomidate, thiopentone etc.) are increased. It mainly because of decrease of plasma protein binding ability of drug molecules due to competitive action of excess H⁺ ion. As a

result a higher number of unbound free molecules of anaesthetic drugs remains in plasma. These pharmacological alterations increase potency of drug effect. Metabolic acidosis also decreases hepatic function as a result hepatic metabolism of drug is also delayed. As a result prolong duration of action of drugs are observed. Considering all these factors are very much important before preparing an anaesthetic protocol for a successful anaesthetic outcome (Hall *et al.* 2001, Grimm *et al.* 2015, Novakovski *et al.* 2017, Zeiler and Pang., 2024) [11, 10, 17, 30]. For anaesthetic protocol in such cases, consider those drugs that have minimal or no cardiopulmonary depression. For premedication glycopyrrolate is a better choice over atropine because of better cardiovascular stability and decreases cardiac arrhythmias which is caused by atropine and also decreases excessive salivation. (Hall *et al.* 2001) [11]. For induction midazolam + etomidate or midazolam & ketamine are good options over depending upon drug availability due to better cardiovascular stability of etomidate compared to propofol or alfaxalone (Pascoe *et al.* 1992, Sams *et al.* 2008, Rodriguez *et al.* 2012) [19, 21, 20] similarly ketamine is a respiratory stimulant drug with excellent visceral analgesic property unlike propofol or barbiturates (Hall *et al.* 2001, Grimm *et al.* 2015) [11, 10]. Maintenance of anaesthesia can be achieved either by TIVA bolus or CRI of etomidate or ketamine (similar to induction agent) or by inhalant anaesthesia (isoflurane or sevoflurane) with fresh oxygen supply throughout the anaesthetic procedure and patient monitoring via pulse oximeter & capnograph to maintain SpO₂ & EtCO₂ levels as per anaesthetic requirement on the basis of patient condition. Overventilation should be avoided as it may lead to development of respiratory alkalosis. Cardio-respiratory depressant drugs like alpha2 agonists, phenothiazines, barbiturates, propofol should be avoided while preparing anesthetic protocol. Now let's discuss briefly about a patient showing symptoms of respiratory alkalosis or respiratory acidosis during anaesthesia. While maintenance of anaesthesia, if a patient shows symptoms of respiratory alkalosis (hyperventilation) the most common cause may be inadequate or light level of anaesthesia (may be less dose of anaesthetic than actually required dose, delay in maintenance dose or low concentration of inhalant to maintain surgical plane of anesthesia etc). This condition can also trigger the pain sensation. Another possible cause is inadequate ventilation setting-excess ventilation (respiration rate) or higher tidal volume setting can ultimately decreases PCO₂ levels leading to respiratory alkalosis. To correct this condition route cause analysis is need to be done. If it's due to the light level of anaesthesia, it can be managed by giving a maintenance shot (bolus) of rapid acting IV anaesthetic like propofol, alfaxalone etc., or if patient is in inhalant setup by increasing the concentration of inhalant drug to produce surgical depth anaesthesia. If it's due to improper ventilation setting than correction of ventilation setting decrease in respiration rate and tidal volume will improve the patient condition. Decrease in fresh gas flow along with re-breathing procedure PCO₂ & EtCO₂ level can be increased to normal level. Constant monitoring of patient via vital parameter monitor (EtCO₂, SpO₂, RR), periodic analysis of ABG are also crucial for confirmatory diagnosis. Respiratory acidosis during anaesthesia is marked with contrary causes & symptoms than respiratory alkalosis, mainly too deep anaesthesia or overdosing of anaesthesia,

airway obstruction, improper ventilation setting, inadequate supply of oxygen, decrease in fresh gas flow-all these factors can potentially leads to respiratory acidosis. Hypoventilation and build up of CO_2 level in the body. Finding the route cause is essential for appropriate treatment and management. If respiratory acidosis symptoms appears at the end of surgical protocol due to overdosing of IV anaesthetic drug then specific antagonist drug shots can be given for speedy recovery or if it's due to high concentration of inhalant anaesthetic supply then reduce or cut the supply of inhalant and provide fresh gas flow improve ventilation status which ultimately improve the condition. Broncho-stimulant like inj. doxapram can improve the ventilation status. If airway obstruction, mechanical obstruction (faulty ET intubation) or bronchospasm is the route cause than correction of these faults will improve the condition like proper intubation of ET, if upper airway obstruction is there

than emergency tracheotomy followed by ET intubation can be useful, if after proper intubation ET got blocked by mucous, blood, phlegm, suctioning can be done to clear the blockage and repositioning of ET can be done. Bronchodilators like deriphylline inj. reduces bronchospasm and improves ventilation status during anaesthesia. Before connecting the patient into the inhalant anaesthetic machine proper checking of soda lime canister (CO_2 absorber), oxygen cylinder status can reduce the chances of manual error related respiratory acidosis during anaesthesia. Changes in ventilation settings like switching to mechanical ventilation with increased respiration rate and increased tidal volume can counter ongoing respiratory acidosis condition during anaesthesia. Constant patient monitoring via ABG & vitals check are important for assessment of correction of the condition with ongoing treatment line.

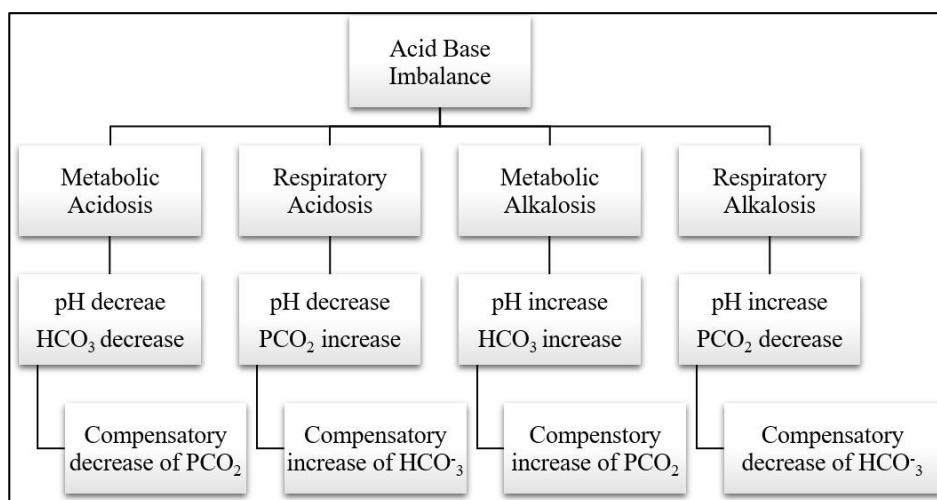


Fig 1: Different types of acid base imbalance & compensatory response

Table 2: Values in different acid-base imbalance condition (Sharma *et al.*, 2021) ^[22]

Acid-base state	pH	HCO ₃	PCO ₂
Normal	7.4	24	40
Metabolic acidosis (Uncompensated)	7.2	15	40
Metabolic acidosis (Partially compensated)	7.3	15	32
Respiratory acidosis (Uncompensated)	7.2	24	90
Respiratory acidosis (Partially compensated)	7.32	38	90
Metabolic alkalosis (Uncompensated)	7.6	24	20
Metabolic alkalosis (Partially compensated)	7.5	20	20
Respiratory alkalosis (Uncompensated)	7.55	38	40
Respiratory alkalosis (Partially compensated)	7.5	38	50

Table 3: Compensatory mechanism in different acid base imbalance (Chatterjee and Shinde., 2012, Grimm *et al.*, 2015, Thorp and Liss., 2017) ^[6, 10, 26]

Acid-base imbalance	pH	pCO ₂	HCO ₃	Compensatory response
Metabolic Acidosis	Decreased	Compensatory decrease	Decreased	Hyperventilation-due to Respiratory compensation to eliminate CO ₂ (Rapid & deep breathing)
Respiratory Acidosis	Decreased	Increased	Compensatory increase	Buffer system (haemoglobin/protein/bicarbonate) regulates serum HCO ₃ generation. Kidney increases HCO ₃ reabsorption with increase H ⁺ secretion
Metabolic Alkalosis	Increased	Compensatory increase	Increased	Respiratory compensation to retain CO ₂ (Depression-slow & shallow breathing)
Respiratory Alkalosis	Increased	Decreased	Compensatory decrease	Buffer system (haemoglobin/protein/bicarbonate) regulates serum HCO ₃ depletion. Kidney increases H ⁺ reabsorption with increase HCO ₃ secretion

Conclusion

Route cause finding in acid-base imbalance is important step for proper treatment and restoring acid-base haemostasis.

Arterial blood gas analysis provides more accurate diagnosis than venous blood sample to detect acid-base imbalance, blood gas fluctuations, ventilations, oxygenations,

electrolyte and metabolic alterations in the critically emergency and anaesthetic patients. ABG allows to find the underlying route cause of metabolic alterations and helps to select the therapeutic measures. ABG analysis is a useful tool for assessment of cardiac, pulmonary, renal, gastrointestinal, infectious patients treatment responses. Pre-operative ABG, CBC, LFT, KFT and intraoperative pulseoximeter, capnography monitoring, ventilatory support finally postoperative fluid therapy, antibiotic, analgesics protocol all plays crucial role for a successful completion of anaesthetic protocol and surgical procedure.

Declaration

Author hereby declare that use of google scholar, pub-med, research gate etc. platforms to study & analyse different research, review, short communication articles, for collection of conclusive data's & statements related to the article topic. All the sources are properly acknowledged by the authors wherever necessary.

References

1. Ak A, Ogun CO, Bayir A, Kayis SA, Koylu R. Prediction of arterial blood gas values from venous blood gas values in patients with acute exacerbation of chronic obstructive pulmonary disease. *Tohoku J Exp Med.* 2006;210:285-290.
2. Balakrishnan A, King LG. Updates on pulmonary function testing in small animals. *Vet Clin North Am Small Anim Pract.* 2014;44:1-18.
3. Buduk SY, Garud KV, Gaikwad RV, Galdha CN, Ingole SD, Bhoite RS, Swadi K. Comparative analysis of blood gas parameters from arterial, peripheral and central venous sites in dogs with renal dysfunction. *Indian J Vet Med.* 2019;39(2):12-15.
4. Byrne AL, Bennett MH, Pace NL, Thomas P. Peripheral venous blood gas analysis versus arterial blood gas analysis for the diagnosis of respiratory failure and metabolic disturbance in adults. *Cochrane Database Syst Rev.* 2013;(11). doi:10.1002/14651858.CD010841.
5. Chakrabarty A. Textbook of clinical veterinary medicine. India: Kalyani Publishers; 2021. p.113-130.
6. Chatterjea MN, Shinde R. Textbook of medical biochemistry. 8th ed. New Delhi: Jaypee Publishers; 2012. p.708-724.
7. Clancy N. Capnography for veterinary nurse. *Vet Nurse.* 2023;14(5):232-237.
8. Desouza HJ, Vijayakumar H, Ranjithkumar M, Kavitha S. Arterial blood gas analysis in cardiac emergencies of dogs. *Indian J Anim Health.* 2025;64(1):57-62. doi:10.36062/ijah.2024.05224.
9. Atalan G, Gunes V, Uzlu E, Yapar K, Atalan G. Comparison of arterial and venous blood gas values in conscious dogs and dogs under anaesthesia induced by ketamine. *Revue Med Vet.* 2008;159(5):288-292.
10. Grimm KA, Lamont LA, Tranquilli WJ, Greene SA, Robertson SA. Veterinary anaesthesia and analgesia. 5th ed. USA: Wiley Blackwell; 2015. p.357-371.
11. Hall LW, Clarke KW, Trim CM. Veterinary anaesthesia. 10th ed. London: W.B. Saunders, Harcourt Publishers Ltd; 2001. p.123-125, 396-397.
12. Irizarry R, Reiss A. Arterial and venous blood gases: indications, interpretations, and clinical applications. *Compend Contin Educ Vet.* 2009;E1-E7.
13. Jeong DH, Yang JJ, Lee L, Yeon SC. Prediction of arterial blood gas values from venous blood gas values in Asiatic black bears (*Ursus thibetanus*) anesthetized with intramuscular medetomidine and zolazepam-tiletamine. *J Vet Med Sci.* 2017;79(10):1757-1763.
14. King LG, Boag A. BSAVA manual of canine & feline emergency & critical care. 3rd ed. UK: BSAVA; 2018. p.170-207, 17-55, 334-353.
15. Ko J, Krims R. Anesthetic monitoring: devices to use and what the results mean. *Today's Vet Pract.* 2012;23-31.
16. Kules J, Brkljacic M, Crnogaj M, Potocnjak D, Grden D, Torti M, Matijacko V, Mrljak V, Rafaj RB. Arterial blood acid-base and electrolyte values in dogs: conventional and strong ion approach. *Vet Arhiv.* 2015;85(5):533-545.
17. Novakovski D, Vries D, Seymour C. BSAVA manual of canine & feline anaesthesia & analgesia. 3rd ed. UK: BSAVA; 2017. p.170-207.
18. Palanivelrajan M, Sumathi D, Nambi AP, Ramesh S. Arterial blood gas analysis in Labrador retriever puppies with parvoviral enteritis. *Indian J Vet Med.* 2025;45:19-20.
19. Pascoe PJ, Ilkiw JE, Haskins SC. Cardiopulmonary effects of etomidate in hypovolemic dogs. *Am J Vet Res.* 1992;53(11):2178-2182.
20. Rodriguez JM, Rascon PM, Calvo RN, Rafael JGV, Perez JMD, Sarmiento JAF, Carmona SQ, Machuca MMG. Comparison of cardiopulmonary parameters after induction of anaesthesia with alfaxalone or etomidate in dogs. *Vet Anaesth Analg.* 2012;39:357-365.
21. Sams L, Braun C, Allman D, Hofmeister E. Comparison of the effects of propofol and etomidate on induction of anaesthesia and cardiopulmonary parameters in dogs. *Vet Anaesth Analg.* 2008;35:488-494.
22. Sharma MC, Kumar M, Sharma RD. Textbook of clinical veterinary medicine. New Delhi: ICAR; 2021. p.88-92.
23. Sidari HA. Diagnostics: the veterinary nurse's role in reading blood gases. *Today's Vet Nurse.* 2021;(Summer):62-67.
24. Swathi S, Ajithkumar S, Unny NM, Usha NP, Beena V, Sunanda C. Arterial blood gas analysis in Labrador retriever dogs with upper and lower respiratory tract diseases. *J Vet Anim Sci.* 2020;51(1):75-78.
25. Topal A, Gul N. Comparison of arterial blood gas, arterial oxyhaemoglobin saturation and end-tidal carbon dioxide tension during sevoflurane or isoflurane anaesthesia in rabbits. *Ir Vet J.* 2006;59(5):278-281.
26. Thorp AR, Liss D. Acid-base and electrolyte handbook for veterinary technicians. United Kingdom: Wiley Blackwell; 2017. p.1-12.
27. Turlock EP III, Walker HK, Hall WD, Hurst JW. Arterial blood gases. In: Clinical methods: the history, physical, and laboratory examinations. 3rd ed. Boston: Butterworths; 1990. p.254-257.
28. Waddell LS. The practitioner's acid-base primer: differential diagnoses and treatment. *Today's Vet Pract.* 2013;25-30.
29. Waddell LS. Blood gas analysis. *Management Tree Pulmonol Crit Care Med.* 2016:20-22.
30. Zeiler GE, Pang DSJ. Fundamental principles of veterinary anesthesia. 1st ed. United Kingdom: Wiley Blackwell; 2024.