

Print ISSN : 0972-8813
e-ISSN : 2582-2780

[Vol. 24(2) May-August 2026]

Pantnagar Journal of Research

(Formerly International Journal of Basic and
Applied Agricultural Research ISSN : 2349-8765)



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Arterial blood gas analysis in bovine pneumonia: Comparative study of healthy and diseased cattle

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<https://doi.org/10.67063/pjr.vol.24.issue.02.11>

ABSTRACT: The present study was undertaken to assess blood gas variations in samples from the auricular artery in healthy and cattle affected with pneumonia. Acid–base and blood gas parameters of healthy control animals (n=10) were within normal physiological limits. Diseased animals (n=25) showed significantly increased blood pH along with decreased arterial pCO₂, pO₂, and oxygen saturation, indicating respiratory compromise and compensatory hyperventilation. Bicarbonate concentration, carbonic acid, bicarbonate-to-carbonic acid ratio, and standard base excess remained largely comparable between healthy and affected animals. Different pneumonic conditions exhibited variable acid–base disturbances, with respiratory alkalosis and metabolic alkalosis being the predominant abnormalities. Chronic and chronic active pneumonia cases commonly demonstrated uncompensated metabolic alkalosis, whereas fibrinopurulent and suppurative pneumonia showed fewer apparent acid–base changes despite the presence of hypoxemia (pO₂ <50 mm Hg) in several animals. Reduced arterial oxygen tension and oxygen saturation suggested impaired pulmonary gas exchange and tissue oxygenation. The findings indicate that arterial blood gas analysis is a useful diagnostic tool for evaluating pulmonary dysfunction, oxygenation status, and compensatory acid–base mechanisms in bovine pneumonia.

Keywords: Acid-base, Arterial blood gas, Cattle, Pneumonia

Respiratory diseases significantly impair physiological functions, particularly gas exchange, leading to disturbances in acid–base balance, tissue oxygenation, and carbon dioxide elimination. Blood gas analysis is a valuable tool for assessing acid–base balance and cardiopulmonary function, supporting both diagnosis and therapeutic intervention. Arterial blood gas analysis plays an important role in assessing oxygenation and ventilation status of the animal. Even minor changes in blood pH (± 0.05) can influence ventilation. Although it is widely used in emergency and critical care for companion animals (Sumner and Rozanski, 2013; Humm and Kellett-Gregory, 2016), its application in food-producing animals remains limited. In cattle, it has been primarily employed in neonatology (Bleul, 2009), with more recent use in adult cows for conditions such as mastitis, subacute ruminal acidosis, and the management of downer cow syndrome (Feng *et al.*, 2022; Giancesella *et al.*, 2010; Güzelbekte_ *et al.*, 2006). In calves with respiratory syndrome, varying degrees of venous

blood gas alterations and acid–base imbalances have been reported (‘oltésová *et al.*, 2015; Aslan *et al.*, 2002). While rapid compensatory mechanisms are well documented in dogs and cats, similar disturbances in cattle often manifest as hypoxaemia, hypercapnia, and respiratory acidosis due to impaired respiratory function (Wall, 2001). In addition to disrupting gas exchange, respiratory diseases may also alter other blood constituents, including lactate levels and lactate dehydrogenase activity (Nagy *et al.*, 2013).

The present study was designed to evaluate acid–base balance and arterial blood gas changes in healthy and pneumonic cattle, with the objective of improving understanding of physiological responses to respiratory disease and the extent of metabolic compensation in acid–base homeostasis.

MATERIALS AND METHODS

Twenty-five cows with clinical and radiographic evidence of pneumonia were selected. Ten

apparently healthy cows were included as healthy control. For arterial blood sampling, hair on the dorsal surface of the ear pinna were removed with a disposable razor. After proper restraining, 1 ml blood was collected from medial intermediate auricular branch of the caudal auricular artery in heparinized syringe maintaining the anaerobic conditions (Bleul *et al.*, 2007). The blood samples were placed on ice and immediately taken to the laboratory for acid base analysis. The pH of blood, pCO_2 (mm Hg), bicarbonate concentration (mmol/L), Anion gap (mmol/L), BE (base excess, mmol/L), pO_2 (mm Hg), saturation of oxygen (SAT, %) and electrolytes *viz*, K (mmol/L), Na (mmol/L) and Cl^- (mmol/L) were estimated at 37°C on IDEXX VetSTAT Electrolyte and blood gas analyzer, USA within 1 hour of sample collection to avoid alteration in blood gas tension. Blood gas cassettes were stored at 4°C prior to use and were used immediately once opened.

The affected cows also underwent tracheo-bronchial wash (TBW) sampling via the percutaneous technique (Narang *et al.*, 2023), and pneumonia was diagnosed and classified as aspiration pneumonia (n=3), suppurative pneumonia (n=5), fibrinopurulent pneumonia (n=5), chronic pneumonia (n=8), and chronic active pneumonia (n=4) based on tracheo-bronchial aspirate cytology supported by history and comprehensive clinical examination.

Statistical analysis

Arithmetic means and standard errors for each variable evaluated across the animal groups were calculated using descriptive statistical methods. The analysis was performed using the SAS software. One-way ANOVA was employed to determine the significance of differences in mean values among the groups.

RESULTS AND DISCUSSION

The arterial blood gas analysis was done using arterial blood sample from auricular artery. This site of sampling is peculiar to cattle and widely preferred in the dairy practice, as it makes blood collection easier and quicker to perform and requires minimal animal restraint (Santarosa *et al.*, 2023). The mean

acid base parameters of ten healthy animals were within the normal reference interval as reported by Kaneko *et al.* (2008). Mean blood pH in healthy control was 7.39 ± 0.01 and it varied from 7.32 to 7.49 (Table 1).

The blood pH was more ($p < 0.05$) in diseased animals as compared to healthy control. The arterial pCO_2 was significantly lesser than healthy controls. The bicarbonate, carbonic acid and ratio of bicarbonate and carbonic acid did not differ significantly in healthy and affected animals (Table 1). The base excess (BE) in affected animals was 3.2 ± 1.1 mmol/l was also comparable to healthy control. Precisely, only three animals had BE more than the upper range of healthy animals. The partial pressure of oxygen (pO_2) and oxygen saturation (SAT) in diseased animals was significantly lower than healthy controls. The authors could not find studies on arterial blood gas analysis in pneumonic cattle. However, in a study on pneumonic calves, Aslan *et al.* (2002) analysed venous blood gas status and reported alterations in pH along with increased bicarbonate and base excess concentrations concurrent with hypercapnia in the infected calves, indicating the activation of compensatory mechanisms. In contrast to the present study, they found a significant ($p < 0.05$) rise in concentrations of bicarbonate, partial pressure of carbon dioxide, and base excess, and decrease ($p < 0.01$) in partial pressure of oxygen and oxygen saturation concentrations. With primary acid base imbalances, bicarbonate and partial pressure of carbon dioxide have been reported to deviate in same direction, and in animals with imbalanced acid base concentrations, normal pH indicated a mixed acid base disturbance. In the present study, the increased blood pH and decreased arterial pCO_2 observed in pneumonic cattle are indicative of respiratory alkalosis, which commonly occurs during the early stages of pneumonia due to hyperventilation and tachypnea (Johnson, 2017). However, 'oltésová *et al.* (2015) reported compensated respiratory acidosis as the acid- base changes in calves with respiratory diseases.

Pneumonia in cattle is associated with pulmonary

inflammation and impaired ventilation-perfusion matching, which further stimulates tachypnea and contributes to hypocapnia (Caswell, 2014). Pulmonary inflammation and impaired gas exchange in pneumonic animals causes hypoxemia, which in turn increases the respiratory rate. Increased elimination of carbon dioxide is due to excessive alveolar ventilation, which subsequently increases blood pH.

As evident from Table 1, there was no significant difference in bicarbonate concentration, carbonic acid, their ratio, and base excess between healthy and affected cattle, thus the disorder appeared to be primarily of respiratory origin rather than metabolic. The BE values suggested that there was little or no metabolic compensation at the time of examination. In acute respiratory alkalosis, renal compensation is usually minimal because compensatory bicarbonate loss develops slowly over hours to days.

The changes in partial pressure of oxygen and oxygen saturation concentrations observed suggested the coping of lungs with concentration of blood carbon dioxide by hyperventilation. Eight cows were hypoxemic ($pO_2 < 50$ mm Hg), out of which five showed normal blood gas parameters. Abnormal arterial gas results have been attributed to poor perfusion which can be because of dehydration, cardiac output dysfunction and/or lung pathology.

The parameters were also compared within the

different forms of pneumonia. Mean blood pH, arterial pCO_2 and BE did not differ significantly in aspiration pneumonia and suppurative pneumonia as compared to healthy controls (Table 2) suggesting no apparent acid base tensions. However, two cows with aspiration pneumonia had uncompensated respiratory alkalosis ($pH > 7.45$; $pCO_2 < 35$ mm Hg) and one had uncompensated metabolic alkalosis ($pH > 7.45$; $BD > +4$). All the five cows with suppurative pneumonia showed no blood gas tensions. Mean blood pH was significantly higher in chronic pneumonia (7.50 ± 0.04) with non-significantly increased SBE (6.00 ± 2.50 mmol/L) and decreased arterial pCO_2 (36.5 ± 3.10 mm Hg), so indicating uncompensated metabolic alkalosis. There was uncompensated respiratory alkalosis in two cows, uncompensated metabolic alkalosis in two, metabolic alkalosis with compensatory respiratory acidosis ($pH > 7.45$; $BD > +2.4$; $pCO_2 > 45$ mm Hg) in two and metabolic acidosis with compensatory respiratory alkalosis ($pH 7.31$; $BD: -1.7$; $pCO_2 -26$ mm Hg) in one cow in chronic pneumonia. Blood gas tensions were apparently normal in one cow. Respiratory acidosis is characterized by reduced pH levels accompanied by elevated pCO_2 concentrations. In a study by Nagy *et al.* (2016), calves with chronic bronchopneumonia showed severe hypoxaemia and varying degrees of hypercapnia due to impaired lung function. The increased carbon dioxide caused respiratory acidosis, which was partially compensated metabolically, keeping blood pH near normal in most cases. Despite clinical improvement during treatment, blood gas

Table 1: Acid-base balance and blood gas tensions in cattle affected by respiratory disease

	Healthy (n=10)	Diseased (n=25)
pH	7.39 ± 0.01^a (7.32-7.49)	7.48 ± 0.08^b (7.31-7.61)
pCO_2 (mm Hg)	42.5 ± 2.10^a (31-49)	35.9 ± 1.50^b (24-51)
HCO_3^- (mmol/L)	24.6 ± 1.20^a (19-29)	24.9 ± 1.20^a (19-40.9)
H_2CO_3 (mmol/L)	1.22 ± 0.05^a (0.93-1.47)	1.08 ± 0.05^a (0.72-1.53)
HCO_3^-/H_2CO_3	20.5 ± 1.25^a (15.4-26.8)	23.6 ± 0.82^a (15.1-31.7)
Anion Gap (mmol/L)	14.4 ± 0.8^a (11.8-16.4)	7.4 ± 1.3^b (-3.9-23.5)
SBE (mmol/L)	3.20 ± 0.30^a (1.7-4.1)	3.20 ± 1.10^a (-2.3-16.5)
pO_2 (mm Hg)	101.6 ± 3.90^a (87-123)	66.2 ± 5.40^b (31-141)
SAT (%)	92.3 ± 2.00^a (80-99)	84.3 ± 2.70^b (60-98)
Na^+ (mmol/L)	139.9 ± 2.40^a (129-147)	125.5 ± 2.50^b (93.0-150.0)
K^+ (mmol/L)	3.90 ± 0.20^a (3.3-4.8)	3.40 ± 0.10^b (2.0-4.2)
Cl^- (mmol/L)	99.9 ± 1.40^a (95-108)	98.4 ± 0.80^a (91-106.2)

Values in parenthesis depict range: Values having different superscript in same column differ significantly at $P < 0.05$

Table 2: Acid-base balance and blood gas tensions in cattle affected by different types of pneumonia

	Healthy control (n=10)	Aspiration pneumonia (n=3)	Suppurative pneumonia (n=5)	Fibrinopurulent pneumonia (n=8)	Chronic pneumonia (n=5)	Chronic active pneumonia (n=4)
pH	7.39±0.01 ^c (7.32-7.49)	7.46±0.01 ^{abc} (7.46-7.47)	7.43±0.01 ^b ^c (7.4-7.46)	7.46±0.02 ^{abc} (7.39-7.56)	7.50±0.04 ^{ab} (7.31-7.57)	7.54±0.02 ^a (7.49-7.61)
pCO ₂ (mm Hg)	42.5±2.10 ^a (31-49)	35.7±4.3 ^{ab} (30-44)	39.4±2.0 ^{ab} (34-47)	30.6±1.8 ^b (24-35)	36.5±3.1 ^{ab} (28-49)	37.5±5.7 ^{ab} (27-51)
HCO ₃ ⁻ (mmol/L)	24.6±1.20 ^a (19-29)	25.5±2.7 ^a (19-31)	24.4±0.9 ^a (21.8-26.6)	21.4±0.6 ^a (20.2-23.4)	27.5±2.8 ^a (20.1-40.3)	26.0±5.0 ^a (19-40.9)
H ₂ CO ₃ (mmol/L)	1.22±0.05 ^a (0.93-1.47)	1.07±0.13 ^a (0.90-1.32)	1.18±0.07 ^a (1.02-1.41)	0.92±0.05 ^a (0.72-1.05)	1.13±0.10 ^a (0.78-1.47)	1.12±0.17 ^a (0.81-1.53)
HCO ₃ ⁻ /H ₂ CO ₃	20.5±1.25 ^a (15.4-26.8)	22.0±0.16 ^a (21.8-22.3)	20.7±0.57 ^a (18.9-22.3)	23.7±1.80 ^a (21.4-30.8)	24.9±1.70 ^a (15.1-30.9)	25.8±2.65 ^a (18.9-31.7)
Anion Gap (mmol/L)	14.4±0.80 ^a (11.8-16.4)	6.70±2.80 ^{bc} (2.3-11.8)	12.6±3.60 ^{ab} (5.2-23.5)	9.10±1.30 ^{abc} (6-12.7)	3.20±1.90 ^c (-3.9-11.2)	7.50±3.20 ^{abc} (-0.8-14.8)
SBE (mmol/L)	3.20±0.30 ^{ab} (1.7-4.1)	0.70±2.00 ^{ab} (-0.7-4.7)	1.20±0.60 ^{ab} (-1.1- 2.3)	-0.60±0.90 ^b (-2.2-2.3)	6.00±2.50 ^a (-1.9-16.5)	6.40±3.40 ^a (2.2-16.5)
pO ₂ (mm Hg)	101.6±3.90 ^a (87-123)	54.7±17.7 ^b (35-90)	58.2±13.7 ^b (31-92)	77.4±19.0 ^{ab} (50-141)	66.9±7.80 ^{ab} (31-97)	69.8±3.90 ^{ab} (58-75)
SAT (%)	92.3±2.00 ^a (80-99)	78.3±8.40 ^{ab} (68-95)	74.6±6.30 ^b (60-95)	85.2±6.80 ^{ab} (60-98)	87.8±4.80 ^{ab} (60-98)	93.2±2.90 ^a (85-98)
Na ⁺ (mmol/L)	139.9±2.40 ^a (129-147)	111.3±9.20 ^c (93-122)	134.8±3.20 ^{ab} (125-142.4)	122.4±3.70 ^{bc} (108-129)	122.6±3.30 ^{bc} (111-141)	134.3±7.20 ^{ab} (121-150)
K ⁺ (mmol/L)	3.90±0.60 ^a (3.3-4.8)	3.60±0.20 ^{ab} (3.1-3.9)	3.08±0.30 ^b (2.3-3.8)	3.50±0.10 ^{ab} (3.3-3.8)	3.40±0.20 ^{ab} (2-4.2)	3.30±0.10 ^{ab} (3.1-3.4)
Cl ⁻ (mmol/L)	99.9±1.40 ^a (95-108)	97.0±2.10 ^a (94-101)	101.6±1.80 ^a (98-106.2)	96.0±1.50 ^a (92-100)	97.8±1.60 ^a (91-103)	100.0±1.50 ^a (97-104)

Values in parenthesis depict range: Values having different superscript in same column differ significantly at P<0.05

values improved little and often worsened later. Blood lactate estimation had limited diagnostic value except in critically ill calves nearing death. Respiratory acidosis develops because of decreased effective alveolar ventilation or breathing an atmosphere with elevated carbon dioxide. Respiratory alkalosis is characterized by elevated pH and decreased pCO₂ levels. It commonly results from hyperventilation, which may be triggered by hypoxemia associated with pulmonary disorders, congestive heart failure, or severe anemia. Respiratory alkalosis might be seen in animals in pain or under psychological stress. Compensating responses for chronic respiratory alkalosis lasting several weeks may actually be sufficient to return pH to normal.

Mean blood pH was significantly higher chronic active pneumonia (7.54±0.02) with non-significantly increased BE and decreased arterial pCO₂ (Table 2), suggesting uncompensated metabolic alkalosis. Two

cows with chronic active pneumonia had uncompensated respiratory alkalosis, one had uncompensated metabolic alkalosis and one cow had metabolic alkalosis with compensatory respiratory acidosis. Mean arterial pCO₂ was significantly lesser in fibrinopurulent pneumonia suggesting respiratory alkalosis. The bicarbonate, carbonic acid and ratio of bicarbonate and carbonic acid were comparable in healthy and all the pneumonias (Table 2). Two cows with fibrinopurulent pneumonia had uncompensated respiratory alkalosis and three cows had normal blood gas tensions.

Arterial partial pressure of oxygen (PO₂) in diseased animals was lower in all types of pneumonia than healthy controls though significantly in aspiration and suppurative pneumonia (Table 2). Likewise, oxygen saturation (SAT) was significantly lower in suppurative pneumonia and non-significantly lower in all other groups except chronic active pneumonia. The alterations in pO₂ and oxygen saturation levels

observed in the infected animals suggest that the lungs were attempting to regulate blood carbon dioxide concentrations through hyperventilation. Aslan *et al* (2002) reported pH changes along with increase in bicarbonate and base excess concentrations with hypercapnia in pneumonic calves as indications of the functioning of compensatory mechanisms.

All the five cows with suppurative pneumonia showed blood gas values within the normal range, however three of them were hypoxemic. Respiratory diseases lead to various degrees of hypoxaemia with possible subsequent tissue hypoxia. Other possible consequences include accompanying hypercapnia and respiratory acidosis. In contrast, hypoxemia does not stimulate increased ventilation until arterial pO₂ levels are markedly reduced.

CONCLUSION

Arterial blood gas analysis using the auricular artery proved to be a practical and reliable method for evaluating respiratory and acid–base alterations in cattle with pneumonia. Diseased animals generally exhibited higher blood pH, lower arterial pCO₂, reduced pO₂, and decreased oxygen saturation, indicating the presence of respiratory compromise and compensatory hyperventilation. Although bicarbonate and base excess values remained largely within normal ranges, different forms of pneumonia showed variable acid–base disturbances, with respiratory alkalosis and metabolic alkalosis being the most common findings. Hypoxemia was observed in several animals even when blood gas parameters appeared normal, emphasizing that impaired oxygenation can occur without overt acid–base imbalance. Overall, the study demonstrates that arterial blood gas analysis is a valuable diagnostic aid for assessing pulmonary dysfunction, oxygenation status, and compensatory mechanisms in bovine pneumonia.

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Received: May 20, 2026

Accepted: June 05, 2026