Indian Journal of Animal Sciences **96** (3): 175–180, March 2026/Article
https://doi.org/10.56093/ijans.v96i3.176687

Analysis of cerebrospinal fluid in recumbent cows with different duration of recumbency

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Received:01 March 2026; Accepted:08 May 2026

ABSTRACT

Chronic recumbency results in progressive ischemic muscle damage, systemic inflammation and disruption of blood brain barrier, leading to neuroinflammation and irreversible recumbency in dairy cows. The present study evaluated the CSF changes into recumbent cows with different duration of recumbency from January to November 2025. The recumbent cows were unresponsive to routine therapy for more than 24 hours and were grouped as 2-5 days, 6-10 days and more than 10 days of recumbency. CSF samples were analyzed for biochemical, electrolyte, lactate, bicarbonate and neuro biomarkers. CSF analysis revealed significant reductions in glucose and increased protein, CK, LDH and neuron-specific enolase, as expected in prolonged recumbency. A significant change in CSF indicated the disruption of blood brain barrier and neuroinflammation in more than 10 days of recumbency cases.

Key words: Cow, Chronic recumbency, CSF analysis, Neuromuscular damage

Chronic recumbent cattle unresponsive to routine treatment and management remains a major clinical concern in bovine practice, yet the mechanism is poorly understood. Chronic recumbency leads to pressure associated muscle damage and ischemic necrosis, particularly of thigh and gluteal muscles. Persistent compression of dependent musculature compromises capillary perfusion and oxygen delivery, resulting in an ischemic myonecrosis and muscle atrophy (Puerto-Parada *et al.* 2021). Necrotic muscles subsequently release intracellular constituents and other toxic metabolites, which initiate dissemination of localized inflammation. The accumulation of inflammatory mediators and metabolites further amplify tissue injury and precipitate a systemic inflammatory response analogous to crush syndrome (Constable *et al.* 2017). Moreover, bacterial translocation from pressure sores and necrotic tissue predispose affected animals to bacteremia and sepsis (Bilodeau *et al.* 2018). Once systemic inflammation is established, the circulating pro-inflammatory cytokines (TNF-α, IL-1β and IL-6) activates leukocytes which exert profound effects on the vascular endothelium and disturb the integrity of blood brain barrier (BBB) during sepsis (Millan Solano *et al.* 2023). The choroid plexus epithelium forming the blood cerebrospinal fluid (BCSF) barrier is highly susceptible to inflammatory and oxidative injury, leading to alteration in cerebrospinal fluid (CSF) biodynamics (Reiber 1994, Hillmer *et al.* 2023). This barrier disruption facilitates infiltration of inflammatory mediators and immune cells in to CNS, resulting neuroinflammation, which further impairs neuronal signaling pathways and disrupt CSF homeostasis, ultimately affecting supraspinal control of posture, muscle tone and coordinated motor function(Na Pombejra *et al.* 2017). Thus, CNS injury secondary to systemic inflammation and BBB–BCSF barrier failure likely represents a critical and yet poorly recognized non-responsive recumbent animals.

Despite the biological plausibility of this mechanistic link, direct veterinary evidence characterizing BBB–BCSF barrier integrity, neuroinflammatory changes and their relationship with neuromuscular dysfunction in chronic recumbent cattle remains scarce. Current clinical decision making is, therefore, largely confined to peripheral musculoskeletal and metabolic parameters, with minimal consideration of central neuroinflammatory processes. This substantial knowledge gap critically limits accurate prognostication and targeted therapeutic intervention in advanced recumbency. Hence, the present study was

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designed to evaluate CSF changes in chronic recumbent cows with different duration of recumbency to underscore the pathogenesis of BBB-BCSF barrier dysfunction and neuroinflammation due to prolonged recumbency.

MATERIALS AND METHODS

The present study was conducted from January 2025 to November 2025 on 18 cows. Inclusion criteria included animals with recumbency exceeding for more than 24 hours and that had failed to respond to therapeutic management. Animals that were recumbent due to lameness, fractures or recent surgical procedures were excluded from this study. The selected animals were divided into three groups based on the duration of recumbency as Group I: 2-5 days of recumbency (n= 6); Group II: 6-10 days of recumbency (n= 6); Group III: More than 10 days of recumbency (n= 6). All the selected cows were systematically screened and subjected to comprehensive clinical evaluation, which included detailed physical examination with particular emphasis on neurological assessment to identify abnormalities associated with recumbency and such cases were excluded.

CSF was collected aseptically from the lumbosacral (LS) space by using sterile 18 or 20-gauge spinal needles (0.9 x 120 mm or 0.9 x152 mm) according to animal body size (Scott, 2004). The CSF samples were divided in two aliquots, for immediate evaluation of physical and microscopic examination and stored at -80 ° C for further analysis (Smith *et al.* 2020, Ferrini, 2024). The CSF samples were estimated for total nucleated cell count and cytology (after centrifugation at 500 rpm for 3 minutes). The supernatant fluid was removed and sediment fluid was made into smear and stained with Giemsa satin for cytological examination (Puerto-Parada *et al.* 2021). The specific gravity of the CSF was determined using a handheld refractometer (Stokol *et al.* 2009). CSF cultured for bacterial identification (Sturgis *et al.*1997).

Biochemical analysis of CSF was carried out by Selectra PRO XS® automated biochemistry analyser (ELITECH, Netherlands). The following parameters were estimated: urea nitrogen, creatinine, glucose, protein, albumin, alkaline phosphatase (ALP), aspartate aminotransferase (AST), lactate dehydrogenase (LDH), creatine kinase (CK), phosphorus and magnesium. Fully automated ST-200 aQua Electrolyte Analyser (SENSA CORE®) was used to determine the concentrations of sodium (Na), potassium (K), chloride and bicarbonate. Immediately after collection of CSF, lactate was measured by Lacto Spark meter (SENSA CORE®). Neuron-specific enolase (NSE) was estimated in CSF by using Bovine Neuron-Specific Enolase ELISA assay (BT LAB ®bioassay laboratory).

The data obtained in this study was subjected to statistical analysis (Snedecor and Cochran, 1994). Biochemical, electrolytes and NSE of CSF was analysed by descriptive statistics. One-way ANOVA was performed to determine significant differences among groups of recumbent animals at different duration of recumbency (2-5 days, 6-10 days and more than 10 days), with significance at the level of 95% ($p < 0.05$).

RESULTS AND DISCUSSION

A significant increase in total nucleated cell in CSF was observed in animals group III (108.67±32.42 cells/µl) which indicating a definitive pleocytosis consistent with neuro inflammation or CSF barrier disruption in the affected cows (Table 1). The inflammation was graded further as mild (10–50 cells/mL), moderate (51–100 cells/mL) and severe (>100 cells/mL) based on TNCC. Bellino *et al.* (1994) reported that CSF is considered abnormal even if the total cell count exceeded 10 cells/mm³ and based on TNCC level in CSF (Bilodeau *et al.* 2018), it could be used as prognositic indicator. According to Stokol *et al.* (2009), listeriosis, viral encephalitis, trauma and neoplasia in adult cattle were more frequently associated with mononuclear pleocytosis, while neonatal calves bacterial meningitis showed neutrophilic CSF pleocytosis. A mild to moderate mixed-cell pleocytosis arise from necrosis or secondary inflammation due to conditions such as intervertebral disc disease, hemorrhagic myelomalacia, ischemia or infarction (Freeman and Raskin, 2001). Prolonged of recumbency resulted in elevated TNCC with neutrophilic pleocytosis and bacteriological evidence suggestive of active

Table 1. Mean (±SE) values of cerebrospinal fluid parameters in recumbent cows at different durations of recumbency

S. No	Parameters	Group I	Group II	Group III	F value
1	Colour	Clear in 6/6	Clear in 6/6	Clear in 5/6 and turbid 1/6	
2	pH	7.51±0.10	7.28±0.04	7.27±0.05	3.38^{ns}
3	Specific gravity	1.01±0.00	1.01±0.00	1.01±0.00	2.77^{ns}
4	TNCC (Cells/µL)	$50.00±8.56^{a}$	$61.67±9.80^{ab}$	$108.67±32.42^{b}$	4.89^{*}
5	Neutrophils (Cells/µL)	$18.5±5.37^{a}$	$27.33±11.21^{ab}$	$40.00±12.43^{b}$	3.65^{*}
6	Lymphocytes (Cells/µL)	24.67±5.66	27.17±6.81	53.67±18.62	3.03^{ns}
7	Monocytes (Cells/µL)	6.83±2.04	7.33±1.80	15.00±4.81	2.683^{ns}

Where Group I (2-5 days of recumbency; n=6), Group II (6-10 days of recumbency; n=6), Group III (>10 days of recumbency; n=6); TNCC-Total Nucleated Cell Count.

inflammation with progressive breakdown of the blood CSF barrier (Scott, 1995 and Ferrini *et al.* 2024).

CSF of cattle is transparent, colourless, slightly alkaline (pH 7.0–7.6) and specific gravity of 1.007–1.010 (Soliman *et al.* 1965). Similarly, in the present study the CSF colour remained clear in all groups except in one cow of Group III, which was turbid in nature and the turbidity of CSF was revealed mixed bacterial infection (Table 1).

In the present study, the CSF culture from all three groups revealed *Staphylococcal* infection with variation related to severity of recumbency. One out of six samples in Group I (2-5 days of recumbency) cow was positive for *S. aureus*. In Group II (6-10 days of recumbency) out of six, three were positive with *S. aureus and S. epidermidis*. Group III (more than 10 days of recumbency) revealed *Staphylococcal spp* infection and amongst those, two samples were mixed infections with enteric organisms (*E. coli and Proteus spp*). This suggests that polymicrobial involvement with Gram-negative enteric bacteria was more prevalent in advanced or prolonged recumbency cases. According to Coureuil *et al.* (2017), extracellular infections enter into the brain due to BBB dysfunction or transcytosing through endothelial cells. Na Pombejra *et al.* (2017) had also reported that microorganisms could penetrate the BBB either by traversing between damaged brain endothelial cells (BECs). Due to prolonged recumbency leading to systemic inflammation and metabolic stress, the blood brain barrier could have got damaged and created a pathway to opportunistic pathogens resulting in meningitis. During sepsis altered cerebral circulation or BBB damage leads to astrocytic dysfunction with detachment of astrocytes from the vasculature progressing into sepsis associated encephalopathy (Nishiko *et al*. 2009 and Taccone *et al.* 2010).

The osmolarity of CSF is similar to that of serum, typically around 280-290 mOsm/kg. Any minor differences in osmotic pressure between the two compartments were quickly corrected through transport of water and solutes across the BBB and blood CSF barriers. Under normal physiological conditions, large or hydrophilic serum constituents cannot cross the intact BBB. The choroid plexus (blood CSF barrier) and the endothelial BBB both tightly regulate and restrict passive diffusion (Kadry *et al.* 2020). However, small lipophilic molecules could enter CSF via passive diffusion, while others utilize specific carrier mediated transport systems such as GLUT1 for glucose and various ion transporters or receptor mediated transcytosis for certain proteins and peptides. Additionally, the choroid plexus epithelium actively secrete CSF and regulates solute transport into the ventricular system (Solar *et al.* 2020).

Gurre-Romer *et al.* (1992) noted that in bacterial meningitis, a shift to anaerobic cerebral metabolism elevates CSF lactate and produces hypoglycorrhachia. Curti *et al.* (2020) reported that CSF-lactate concentration was two to three times greater than in plasma, can be indicative of lactate production inside the central nervous system. Similarly in hypoxic conditions, central nervous system cells and infiltrating leukocytes such as granulocytes and macrophages could substantially augment lactate levels, depending upon the extent and intensity of CNS inflammation (Haji-Michael *et al.* 1999 and Wellmer *et al.* 2001).

Di Terlizzi and Platt (2006) reported that CSF glucose concentration was approximately 60–80% of the blood glucose level. The reduction in CSF glucose (hypoglycorrhachia) is likely be due to bacterial consumption or reduced cerebral glucose uptake or altered glucose transport mechanisms secondary to neuronal dysfunction, hypoxia or inflammation within the central nervous system (Guerra-Romera *et al.* 1992). Such a pattern of glucose depletion was also observed in the present study as significantly reduced CSF glucose was noticed in group III animals followed by group II animals. These findings were consistent with energy compromise and neuronal stress associated with prolonged recumbency (Table 2).

Lactate dehydrogenase (LDH) activity in CSF was significantly ($p<0.05$) higher in cows recumbent for more than 10 days (Gr III) followed by 6-10 days (Gr II) of recumbency, which indicated presence of cellular and tissue damage due to prolonged recumbency. Likewise, creatine kinase (CK) was also significantly ($p<0.01$) high with duration of recumbency especially in more than 10 days (Gr III) of recumbency (Table 2). Estimation of lactate dehydrogenase (LDH) and creatine kinase (CK) in cerebrospinal fluid considered as an important indicator of cellular injury in CNS. Elevated CSF-LDH concentrations usually noticed with inflammatory conditions such as meningitis, traumatic brain injury and neoplasia (Supritha *et al.* 2025).In the present investigation, increased LDH and CK activities were observed in prolonged recumbent cows which might be attributed to ischemia induced neuronal and glial injury, leading to neuroinflammation and subsequent BBB disruption, thereby permitting leakage of intracellular enzymes into the CSF.

Neuron-specific enolase (NSE) concentrations in CSF was significantly ($p<0.01$) high among the groups. A progressive and significant increase in NSE was observed with prolonged duration of recumbency, with Group III cows (more than 10 days of recumbency) showed the highest NSE level (6.76±0.49 ng/ml), this trend suggested that association of neuronal involvement or neuroaxonal damage with prolonged recumbency (Table 2). Garcia-Alix and Arnaez (2022) had reported that CSF-NSE is released only after hypoxicischemic injury and encephalopathy. In the present study the progressive elevation in CSF NSE levels was observed across the recumbent groups indicating a rising magnitude of neuronal or glial cell injury, reflecting the extent of central nervous system involvement during the recumbency period. The pattern suggested that the magnitude of NSE elevation correlated with the severity or duration of neurological compromise, supporting its potential role as a sensitive biomarker for neuronal damage in recumbent cows. Czarniak *et al.* (2023) reported that

Table 2. Mean (±SE) values of CSF biochemical, electrolytes and NSE in recumbent cows at different durations of recumbency

S. No	Parameters	Group I	Group II	Group III	F value
1	BUN (mg/dL)	27.00±3.62	32.33±6.10	30.00±7.27	0.28 ^{ns}
2	Creatinine (mg/dL)	1.46±0.03	1.32±0.06	1.49±0.14	1.02^{ns}
3	Glucose (mg/dL)	58.33±7.17^{b}	33.16±3.49^{a}	28.00±1.90^{a}	11.75*
4	Protein (g/dL)	0.83±0.17	0.85±0.18	1.20±0.18	1.36^{ns}
5	Albumin (g/dL)	0.24±0.10	0.37±0.07	0.35±0.03	0.21^{ns}
6	Globulin (g/dL)	0.41±0.14	0.48±0.16	0.85±0.19	2.06^{ns}
7	A/G ratio	1.84±0.59	1.45±0.73	0.65±0.23	1.18^{ns}
8	ALP (U/L)	9.33±3.39	6.33±0.84	5.33±0.84	1.01^{ns}
9	AST (U/L)	104.10±15.99	79.00±7.89	66.00±14.87	2.08^{ns}
10	LDH (U/L)	5.33±1.02^{a}	59.66±12.63^{b}	41.00±4.76^{b}	12.48*
11	CK (U/L)	21.12±0.55^{a}	373.92±56.43^{b}	566.24±92.37^{c}	21.05**
12	Calcium (mg/dL)	5.97±0.26	7.08±1.30	8.02±1.01	1.13^{ns}
13	Phosphorus (mg/dL)	2.02±0.42	1.70±0.12	2.08±0.22	0.51^{ns}
14	Magnesium (mg/dL)	2.23±0.11	2.27±0.06	2.19±0.11	0.18^{ns}
15	Sodium (mmol/L)	144.77±1.11	139.60±1.94	144.63±2.12	2.75^{ns}
16	Potassium (mmol/L)	2.81±0.04	2.76±0.09	2.83±0.05	0.34^{ns}
17	Chloride (mmol/L)	108.15±1.90	107.58±1.93	110.53±1.53	0.76^{ns}
18	Bicarbonate (mmol/L)	21.98±1.10	21.58±1.31	20.67±1.03	0.34^{ns}
19	Lactate (mmol/L)	5.34±0.59	6.37±0.29	6.57±0.50	1.89^{ns}
20	NSE (ng/mL)	2.55±0.13^{a}	4.23±0.68^{b}	6.76±0.49^{c}	18.50**

Where Group I (2-5 days of recumbency; n=6), Group II (6-10 days of recumbency; n=6), Group III (>10 days of recumbency; n=6); BUN – Blood urea nitrogen; A/G ratio – Albumin to globulin ratio; ALP- Alkaline phosphatase; AST - Aspartate aminotransferase, LDH-Lactate dehydrogenase (LDH), CK-Creatine kinase, NSE: Neuron-Specific Enolase.

the NSE can be used to assess the integrity of BBB and suggested that elevated concentration was an indicative of damage to the neural tissue. When neuronal injury occurred as in cases of ischemia, inflammation or trauma, the intracellular NSE was released into the extracellular space and subsequently, diffused into the CSF and the systemic circulation, and increasing serum NSE concentrations (You *et al.* 2019).

Further, the CSF protein, albumin and lactate levels did not vary significantly among the different groups (Table 2). However, these parameters showed consistently higher values as compared to reference level documented by Welles *et al.* (1992). Albumin synthesis exclusively occurs in the liver and it unable to cross an intact blood CSF barrier in healthy animals, hence it serves as a reliable marker of blood brain barrier permeability (Reiber, 1994, Zetterberg *et al.* 2013, Czarniak *et al.* 2023). The elevated CSF-protein directly reflected structural or functional compromise of the BBB. The elevated CSF albumin and protein levels in the prolonged recumbency resulted in hypoxic and ischemic insults to the central nervous system (CNS), leading to endothelial dysfunction and increased permeability of the choroid plexus epithelium. Similar findings have been reported in both human and veterinary studies, where elevated CSF albumin and protein concentrations were associated with systemic inflammation, ischemia or trauma-induced blood brain barrier disruption (Hillmer *et al.* 2023). Similarly, CSF-serum albumin ratio was greater than 2.4 in foals, which indicated CNS inflammation secondary to prolonged pressure induced ischemic necrosis of muscle, leading to the release of inflammatory mediators that adversely affected CNS microvasculature and aggravated blood CSF barrier leakage (Andrew *et al.* 1994) and Blennow *et al.* 1994). Therefore, consistent elevation of protein and albumin quotients correlated with the chronicity and severity of recumbency, highlighting their potential diagnostic relevance for assessing barrier integrity in recumbent cattle.

The study provided evidence-based blood–brain barrier dysfunction and ongoing neuroinflammation in prolonged recumbency. Significant alterations in CSF glucose, albumin, LDH, CK and bovine NSE confirmed CNS

involvement in chronic recumbency, especially in more than 10 days in recumbent cows. Further, these findings underscored the diagnostic and prognostic value of cerebrospinal fluid analysis in chronic recumbent cows and supported its incorporation into clinical decision making protocols as well as formulating tailor made treatment strategies for the individual cows.

ACKNOWLEDGEMENT

The authors express their sincere gratitude to the Dean and the Professor and Head of the Veterinary Clinical Complex, Veterinary College and Research Institute, Orathanadu for their valuable support and guidance.

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