

Clinical Approach to Neurological Disorders in Cattle: Etiology, Diagnosis and Management

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INTRODUCTION

The nervous system is responsible for integrating and coordinating physiological activities across the body. In livestock species such as cattle, neurological disorders are often difficult to diagnose because of the complex structure of the nervous system and the limited opportunities for direct examination of neural tissues. Additionally, neurological investigations in farm animals are comparatively fewer than those conducted in companion animals or humans.

In many cases, neurological signs observed in cattle arise as secondary consequences of systemic disturbances rather than primary diseases of the nervous system itself. Metabolic disorders such as hypocalcaemia, pregnancy toxemia, hypoglycaemia,

metabolic acidosis and septic conditions frequently produce neurological manifestations including weakness, altered behaviour or impaired coordination.

Accurate diagnosis requires a systematic clinical approach that begins with a detailed physical examination and is supported by laboratory tests such as serum biochemistry, hematology and cerebrospinal fluid analysis when possible. Once neurological involvement is suspected, determining the anatomical location of the lesion within the nervous system becomes a crucial step in clinical assessment.

From a functional perspective, the nervous system can be divided into four major neuroanatomical regions depicted in Table 1.

Table 1. Neuroanatomic Regions and Abnormalities Associated with Neurologic Disease

Neuroanatomic location	Gait	Mentation	Posture	Spinal reflexes
Cerebrum	Normal	Abnormal	Normal	Normal
Cerebellum	Abnormal	Normal	Abnormal	Abnormal or normal
Brainstem and cranial nerves	Abnormal or normal	Abnormal or normal	Abnormal or normal	Abnormal or normal
Spinal cord and peripheral nerves	Abnormal	Normal	Abnormal	Abnormal or normal

MAJOR CATEGORIES OF NEUROLOGICAL DISORDERS IN FARM ANIMALS

Cerebral Disorders

The cerebrum plays a key role in consciousness, behaviour, learning and voluntary movement. Diseases affecting this region primarily alter the animal's **mental state and behavioural responses**. Affected cattle may exhibit depression, somnolence, stupor or coma, depending on the severity

of the condition. In some situations, animals may display abnormal excitement, aggression or compulsive behaviour.

Another important manifestation of cerebral disease is **central blindness**. Animals with this condition retain normal ocular reflexes but are unable to perceive visual stimuli. They often move slowly, collide with obstacles or appear disoriented in unfamiliar surroundings.

Several metabolic disturbances can impair cerebral function. Hypoglycaemia, electrolyte imbalances and acid–base disorders may interfere with neuronal metabolism and neurotransmission. Therefore, biochemical evaluation of blood parameters is an essential component of diagnostic investigation. Important causes of cerebral disease in cattle include:

- Polioencephalomalacia
- Viral encephalitis
- Bacterial meningitis
- Rabies
- Pseudorabies
- Plant and chemical toxicoses

Polioencephalomalacia

Polioencephalomalacia (PEM), also referred to as cerebrocortical necrosis, is a common neurological disorder in ruminants characterized by necrosis of the cerebral cortex.

The condition may arise due to several factors including:

- Thiamine deficiency
- Excess sulfur intake
- Salt poisoning associated with water deprivation
- Lead toxicity

Under normal conditions, ruminal microorganisms synthesize thiamine. However, disturbances of the rumen environment such as ruminal acidosis can suppress microbial populations and reduce thiamine production. Certain plants and bacteria also produce thiaminase enzymes that degrade thiamine within the rumen. In small ruminants, prolonged administration of anticoccidial drugs such as amprolium may interfere with thiamine metabolism and contribute to the development of PEM. The fundamental pathological mechanism involves disruption of cerebral energy metabolism, leading to accumulation of intracellular sodium, osmotic swelling of neurons and cerebral edema.

Clinical signs may include:

- Depression and incoordination
- Aimless wandering
- Dorsomedial strabismus
- Opisthotonus
- Cortical blindness
- Seizures or convulsions

Early administration of thiamine can significantly improve prognosis if treatment is initiated promptly. Osmotic agents such as mannitol are sometimes used to reduce intracranial pressure.

Viral Encephalitis

Viral infections affecting the central nervous system represent an important group of neurological diseases in cattle.

Herpesvirus Encephalitis

Bovine herpesvirus types 1 and 5 belong to the alphaherpesvirus group. Although both viruses can infect cattle, BHV-5 is more strongly associated with neurological disease. Infection typically begins in the respiratory tract and the virus subsequently travels

through sensory nerves to reach the central nervous system.

Affected animals may develop fever, behavioural changes, ataxia and progressive neurological deterioration.

Rabies

Rabies is a fatal viral disease affecting all mammals, including livestock. The virus is transmitted primarily through the bite of an infected animal. Following entry into the body, the virus replicates locally in muscle tissue before migrating along peripheral nerves toward the brain.

Once the central nervous system is involved, the disease progresses rapidly. Typical clinical signs include:

- Behavioural abnormalities
- Excessive salivation
- Ataxia
- Paralysis of the throat and jaw
- Increased sensitivity to external stimuli

The disease usually leads to death within a short period after the onset of neurological symptoms.

Pseudorabies

Pseudorabies, caused by Suid herpesvirus-1, primarily affects pigs but may infect cattle and other livestock species as incidental hosts. In cattle, the disease is characterized by intense itching, neurological disturbances and rapid progression to death.

Cerebellar Disorders

The cerebellum plays a critical role in regulating balance and coordinating voluntary movements. Unlike cerebral diseases, cerebellar disorders generally do not affect consciousness or mental status.

Animals with cerebellar dysfunction may exhibit:

- Ataxia
- Dysmetria
- Truncal sway
- Intention tremors

Although muscle strength is typically preserved, movement becomes poorly coordinated.

Pestivirus Infection

Bovine viral diarrhoea virus infection during pregnancy can disrupt fetal brain development. When infection occurs during certain stages of gestation, it may lead to cerebellar hypoplasia in newborn calves. Affected calves appear bright and alert but show severe incoordination, head tremors and difficulty standing or nursing.

Hypomagnesaemia (Grass Tetany)

Grass tetany is a metabolic disorder resulting from low magnesium concentration in blood. It commonly occurs in lactating cattle grazing rapidly growing pastures that contain insufficient magnesium.

Clinical manifestations include:

- Neuromuscular excitability
- Muscle tremors
- Ataxia
- Convulsions
- Tetanic muscle contractions

Older lactating cows are particularly susceptible to this condition.

Brainstem and Cranial Nerve Disorders

The brainstem is a critical structure that connects the brain with the spinal cord and contains the nuclei of most cranial nerves. Lesions affecting this region often result in abnormalities related to cranial nerve function.

Typical clinical signs include:

- Head tilt
- Circling behaviour
- Facial asymmetry
- Nystagmus
- Difficulty swallowing

Such signs are often associated with diseases such as listeriosis, ear infections or brain abscesses.

Listeriosis

Listeriosis is a significant bacterial disease of ruminants caused by *Listeria monocytogenes*. The organism is widely distributed in the environment and

is frequently associated with the consumption of poorly fermented or spoiled silage.

Affected animals often develop unilateral cranial nerve deficits. Clinical features may include facial paralysis, drooping ears, head tilt and difficulty swallowing. Without treatment, affected animals may become recumbent and eventually die.

Early administration of antibiotics such as penicillin or cephalosporins combined with supportive therapy improves the likelihood of recovery.

METABOLIC DISORDERS WITH NEUROLOGICAL MANIFESTATIONS

Several metabolic disorders in cattle can lead to neurological signs even though they do not cause direct structural damage to the nervous system. These conditions are mainly functional disturbances resulting from imbalances in electrolytes, energy metabolism or essential micronutrients. Common examples are below in Table no.2.

Table 2: Metabolic Diseases Associated with Neurological Signs

Condition	Age/Class Affected	Clinical Signs	Pathology
Milk Fever (Hypocalcaemia)	Periparturient dairy cows	Excitement, tetanic spasms, recumbency, drowsiness, coma, death	None
Transit Tetany	All ages; esp. pregnant cows under heat/travel stress	Excitement, trismus, incoordination, paresis, convulsions, coma, death	None
Hypomagnesaemic Tetany	Lactating cows, housed calves	Hyperaesthesia, fasciculation, tetany, convulsions, sudden death	Renal tubular necrosis in calves
Ketosis	High-yielding dairy cows	Wasting, licking, salivation, incoordination, circling, blindness, delirium	Fatty liver
Water Intoxication / Salt Poisoning	Calves	Restlessness, weakness, tremors, convulsions, coma	Brain edema, hemolysis
Ruminal / Metabolic Acidosis	Fattening cattle, high-yielding cows/calves	Tachycardia, ruminal stasis, diarrhoea, depression, dehydration	Ruminitis, laminitis
Cerebrocortical Necrosis	Young ruminants on low-fibre diets	Nystagmus, opisthotonus, convulsions, recumbency	Polioencephalomalacia
Vitamin A Deficiency	Calves (3–6 months)	Night blindness, xerophthalmia	Not specified

CHEMICAL POISONING

Chemical poisoning in Farm cattle can occur because animals often graze indiscriminately and may ingest harmful substances present in their environment. Exposure may arise from contaminated feed, mouldy grains or materials treated with pesticides such as insecticides, fungicides and herbicides. Toxicity may

also result from accidental over-administration of trace minerals or veterinary medications. The nature and severity of neurological signs depend on the type of toxin involved and the amount consumed. Common chemical toxins and their major neurological effects are summarised in Table 3.

Table 3: Chemical Poisons Associated with Neurological Signs in Cattle

Poison	Principal Effects
Lead	Constipation, ruminal stasis, tremors, hyperaesthesia, blindness, ataxia, convulsions, encephalopathy, laminar necrosis, chronic unthriftiness, aimless wandering, peripheral neuropathy

Arsenic	Tremors, abdominal pain, diarrhoea, blindness, incoordination, convulsions, coma, hyperaemia of mucosa, degeneration of liver, kidney, heart, nerves, cerebral edema, petechiation
Mercuric Compounds	Stomatitis, gastroenteritis, diarrhoea, incoordination, twitching, hyperaesthesia, blindness, convulsions, cerebellar cell depletion, neuronal necrosis, gliosis, nephrosis
Selenium	Respiratory distress, tachycardia, diarrhoea, blindness, head pressing organ necrosis, pulmonary edema, chronic dullness, emaciation, stiffness, cirrhosis, encephalopathy secondary to liver damage
Organophosphates/ Carbamates	Salivation, diarrhoea, bradycardia, tremors, tetany, sweating, ataxia, convulsions, coma, degeneration of peripheral nerves and spinal tracts, signs of cholinesterase inhibition

Intoxications of Biological Origin

Neurological disorders in cattle may also arise from biological toxins, especially bacterial exotoxins (Table 4). These toxins can produce acute or subacute neurological signs depending on the causative organism

and route of exposure. Common clinical manifestations include muscle tremors, convulsions, paralysis and behavioural changes. Diagnosis is generally based on clinical signs, exposure history and supportive pathological findings.

Table 4: Bacterial Exotoxins Causing Neurological Signs

Toxin	Description / Principal Effects
Enterotoxaemia	Salivation, abdominal pain, diarrhoea, excitement, followed by depression, head pressing, recumbency, paddling, opisthotonus, coma. Renal cortical and symmetrical hemorrhages.
Tetanus	Muscle stiffness, tremors, trismus, prolapse of nictitating membrane, hyperaesthesia, spasms, wide-based stance, bloat, tetanic convulsions, opisthotonus, respiratory paralysis. No specific pathology.
Botulism	Progressive muscular paralysis, dysphagia and recumbency. Terminally, respiration becomes abdominal.

TRAUMATIC DISORDERS

Traumatic neurological disorders are most commonly observed in newborns and periparturient animals (Table 5).

Table 5: Traumatic Causes of Neurological Dysfunction

Cause	Affected Group	Principal Effects
Cervical meningeal haemorrhage	Newborn calves	Laboured respiration, slow pulse, trapezius paralysis, forelimb gait disturbance, coma
Femoral nerve avulsion	Newborn (posterior presentation)	Quadriceps paralysis, muscle atrophy and inability to extend stifle
Obturator/sciatic nerve injury	Periparturient cows	Hindlimb weakness, inability to rise, dragging limbs
Ischemic nerve damage	Downer cows	Paralysis due to pressure on tibial, peroneal or radial nerves
Spinal cord compression	All ages (rare)	Paralysis, sensory loss below lesion, hyperesthesia cranial to lesion
Spinal abscesses	Calves	Progressive paresis, spinal pain, fever
Spondylosis and ligament fracture	Older bulls (AI centers)	Sudden recumbency due to vertebral instability

CLINICAL MANIFESTATIONS OF NERVOUS SYSTEM DISORDERS

Neurological disorders in cattle present with various behavioural, motor and sensory abnormalities resulting from dysfunction of the central or peripheral nervous system. These signs can generally be grouped

into changes in mental status, involuntary movements, gait disturbances and sensory deficits. Recognising these clinical features is important for accurate neuroanatomical localisation and differential diagnosis. A summary of the major clinical manifestations is presented in Table 6.

Table 6: Clinical Manifestations of Neurological Dysfunction

Category	Manifestation	Description / Features	Common Causes
Mental State	Mania	Bizarre behaviour, licking objects, bellowing, wandering, drunken gait	Rabies, pseudorabies, polioencephalomalacia, lead toxicosis

	Frenzy	Violent, uncontrolled activity, dangerous behaviour	Pseudorabies, encephalitis, colic, photosensitization
	Aggression	Hostility toward animals or handlers	Early rabies, pseudorabies
	Syncope	Sudden loss of consciousness	Heart failure, cerebral haemorrhage
	Narcolepsy / Catalepsy	Excessive sleep or sudden rigidity	Rare, idiopathic
	Stupor	Partial unconsciousness, response only to strong stimuli	Severe CNS depression
	Coma	Complete unconsciousness	Terminal CNS depression
	Head Pressing	Persistent pushing of head against objects	Cerebral dysfunction
	Compulsive Walking	Continuous walking with lowered head, apparent blindness	Encephalitis, metabolic disorders
	Aimless Wandering	Purposeless movement, depression, blindness, tongue protrusion	Severe cerebral disease
Involuntary Movements	Tremors	Continuous twitching of skeletal muscles	Toxic/metabolic causes
	Tics	Spasmodic twitching, longer intervals	Spinal nerve trauma
	Tetany	Generalised symmetrical muscle contraction	Tetanus, grass tetany
	Spasms	Involuntary contraction of muscle groups	Various CNS disorders
	Convulsions (Tonic)	Sustained muscle rigidity	Severe CNS insult
	Convulsions (Clonic)	Rhythmic jerking with relaxation phases	Epileptic or toxic causes
	Chorea	Fast, irregular movements of face and limbs	Rare, degenerative conditions
Gait Abnormalities	Weakness / Paresis	Reduced muscle strength, dragging limbs, knuckling	Parturient paresis, peripheral neuropathy
	Ataxia	Incoordination without spasticity	Cerebellar or spinal lesions
	Hypermetria	Exaggerated limb movement	Cerebellar dysfunction
	Hypometria	Stiff, reduced limb movement	Upper motor neuron lesions
	Dysmetria	Combination of hypermetria and hypometria	Cerebellar or proprioceptive deficits
Sensory Disturbances	Anaesthesia	Complete loss of sensation	Peripheral nerve damage
	Hypoaesthesia	Reduced sensitivity	Mild nerve injury
	Hyperaesthesia	Increased sensitivity to stimuli	Inflammatory or toxic conditions

TREATMENT AND MANAGEMENT

Effective treatment of neurological disorders requires a comprehensive approach that includes neuroprotective agents, appropriate antibiotics, symptomatic management, anti-inflammatory and osmotic therapy, along with adequate supportive care. Early and timely intervention is crucial to control disease progression and improve recovery outcomes.

Neuroprotective Therapy

Cobalamin (Vitamin B₁₂) is a water-soluble vitamin essential for cellular growth, nervous system function and hematopoiesis. Cobalamin acts as a cofactor in several enzymatic pathways, including nucleic acid synthesis, fatty acid metabolism, amino acid metabolism, myelin formation and red blood cell production. The three natural forms of Vitamin B₁₂ are

methylcobalamin (MeCbl), adenosylcobalamin (AdCbl) and hydroxycobalamin (OHCbl). Among these, MeCbl is the most bioavailable and neurologically active form. It does not require conversion in body and is better retained in tissues such as the liver and nervous system.

MeCbl supports DNA synthesis and maintains chromosomal stability, particularly in regions like centromeres and subtelomeric DNA. It is also essential for preserving myelin sheath and synthesising neurotransmitters. In Veterinary neurology, MeCbl is administered in combination with Pyridoxine and Nicotinamide at a dose of 5–10 ml IM/IV. This combination supports neuronal repair, metabolic stabilisation and clinical recovery in conditions such as

polioencephalomalacia, peripheral neuropathies, lead toxicity and thiamine-deficiency-related disorders.

Antibiotic Therapy

Broad-spectrum antibiotics are indicated in cases of bacterial encephalitis, meningitis or suspected CNS infections. Preference is given to drugs that can cross the blood-brain barrier (BBB). Lipophilic antibiotics and third-/fourth-generation Cephalosporins are particularly effective due to their enhanced CNS penetration.

Commonly used antibiotics include:

Cefquinome: 1 mg/kg IM/IV

Ceftriaxone: 5–10 mg/kg IM/IV

Symptomatic Treatment

To manage convulsions, agitation and behavioural disturbances, sedatives and tranquillisers are administered. These agents help stabilise neurological activity and improve patient comfort. Commonly used sedatives and tranquillisers include:

Triflupromazine HCl: 0.1 mg/kg IV every 8–12 hours

Diazepam: 0.25–0.5 mg/kg IM every 6 hours

Chlorpromazine HCl: 1 mg/kg IM every 8–12 hours

Anti-inflammatory Therapy

Reducing intracranial inflammation and pressure is critical in many neurological conditions. These agents help mitigate cerebral edema and inflammatory damage. Two classes of drugs are commonly used:

NSAIDs: Meloxicam: 30 ml/300 kg IM/IV/SC

Corticosteroids: Dexamethasone: 0.04–1 mg/kg IM

Isoflupredone @ 5-10 ml IM

Osmotic Therapy

Osmotic agents are used to reduce cerebral edema and intracranial pressure:

Mannitol 10–20%: 0.5–2 g/kg IV

Dextrose 20%: 500–2000 ml IV

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These hypertonic solutions draw excess fluid from brain tissue, relieving pressure and improving neurological function.

Supportive Care

Supportive care is fundamental to recovery and includes:

- Providing soft bedding to prevent pressure sores
- Frequent repositioning of recumbent animals
- Ensuring adequate hydration and nutrition
- Monitoring comfort and vital signs

Proper nursing care reduces complications and enhances the effectiveness of medical treatment.

CONCLUSION

Neurological disorders in cattle can arise from multiple causes, including metabolic disturbances, infectious agents, toxic substances and traumatic injuries. These conditions often present with complex or subtle clinical signs, making early detection and accurate localisation of the affected region of the nervous system essential for proper diagnosis and management. A structured diagnostic approach, beginning with a thorough clinical examination and supported by laboratory investigations, helps veterinarians differentiate primary neurological diseases from systemic conditions that produce secondary neurological signs.

Management should focus on addressing the underlying cause and may involve the use of neuroprotective agents such as methylcobalamin, appropriate antimicrobial therapy, symptomatic treatment and adequate supportive care. Early and appropriate intervention enhances recovery, reduces the risk of persistent neurological deficits and helps minimise economic losses in cattle production.