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Neurological disorders in dairy animals

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Abstract

Neurological disorders in farm animals are clinically important due to their varied causes and significant economic impact. In most cases, neurological dysfunction arises secondary to systemic or metabolic disturbances rather than primary structural lesions of the central nervous system, making differentiation between metabolic encephalopathies and primary infectious, inflammatory, degenerative, or traumatic conditions essential for proper diagnosis and treatment. Metabolic imbalances such as hypocalcemia, hypomagnesemia, ketosis, and electrolyte or acid–base disturbances can impair neuronal activity and neuromuscular transmission, leading to signs like altered mentation, ataxia, tremors, recumbency, and seizures without obvious gross lesions. Infectious agents (viral, bacterial, or prion-related), toxins, and trauma also contribute to neurological disease, particularly in neonatal and periparturient animals. Accurate neuroanatomical localization supported by clinical examination, hematology, serum biochemistry, electrolyte analysis, and cerebrospinal fluid evaluation remains fundamental for distinguishing systemic disorders from primary neurological conditions and for implementing timely therapeutic interventions to reduce mortality, production losses, and welfare compromise.

Keywords: Cerebral disorders, grass tetany, metabolic disorders, neurological disorders, pestiviral infections, rabies

1. Introduction

The nervous system is critical for maintaining homeostasis by integrating sensory input and coordinating motor, autonomic, endocrine, and behavioural responses necessary for survival and productivity in food-producing animals (Cunningham & Klein, 2020) ^[4]. However, in large animal practice, it remains relatively under investigated due to structural complexity, limited access to advanced diagnostic imaging, and economic constraints (Fecteau, 2015) ^[7]. Neurological symptoms are frequently due to systemic disorders rather than underlying brain disease in cattle and other ruminants. Altered mentation, ataxia, tremors, recumbency, or seizures are common symptoms of metabolic and toxic illnesses include hypocalcemia, hypomagnesemia, pregnancy toxemia, hypoglycemia, hepatic encephalopathy, sepsis, and acid-base imbalances (Constable *et al.*, 2017, Radostits *et al.*, 2007) ^[3, 18]. Therefore, distinguishing primary neurological disease from metabolic encephalopathy is essential for accurate diagnosis and treatment planning.

Altered mentation, ataxia, tremors, recumbency, or seizures are common symptoms of metabolic and toxic illnesses such as hypocalcemia, hypomagnesemia, pregnancy toxemia, hypoglycemia, hepatic encephalopathy, sepsis, and acid-base imbalances (Fecteau, 2015) ^[7]. Once a neurological disorder is suspected, precise neuroanatomical localization of the lesion is fundamental for narrowing differential diagnoses (Mayhew, 2009) ^[16].

Functionally, the nervous system is divided into four major regions: cerebrum, brainstem, cerebellum, and spinal cord (Lorenz *et al.*, 2011) ^[14]. Each region produces characteristic clinical signs. Forebrain lesions typically cause behavioural changes and contralateral proprioceptive deficits, brainstem lesions result in cranial nerve abnormalities and altered consciousness, cerebellar dysfunction leads to hypermetria and intention tremors without weakness, and spinal cord lesions produce paresis or paralysis with characteristic upper or lower motor neuron signs (Constable *et al.*, 2017) ^[3].

2. Specific neurological disease-

2.1. Cerebral Diseases

Cerebral disorders mostly affect an animal's awareness and behaviour. Drowsiness, low reactivity, extreme melancholy, unconsciousness, or extraordinary enthusiasm are all possible symptoms of brain malfunction in animals. Loss of central vision is commonly seen, which may cause slow, purposeless wandering and bumping into objects, particularly in new surroundings. Such clinical signs suggest involvement of the cerebral cortex, especially areas responsible for vision and behaviour (Dutton, 2003) ^[6].

Metabolic abnormalities, including disturbances in blood glucose levels, electrolyte imbalance, and acid base irregularities, can negatively affect brain activity. Therefore, serum biochemical analysis is important to detect these underlying problems. In ruminants, frequent causes of cerebral disorders include conditions such as polio encephalomalacia, bacterial meningitis, lentiviral diseases like caprine encephalitis and Maedi-Visna, herpesvirus-related encephalitis, rabies, pseudorabies, and toxicity caused by certain plants (Filippatos *et al.*, 2017) ^[8].

2.2 Polioencephelomalacia

A number of underlying conditions, such as thiamine deficiency, high salt consumption combined with dehydration, sulphur toxicity, or lead poisoning, can cause polio encephalomalacia. (Cebra *et al.*, 2004) ^[2]. Ruminal acidosis, which disrupts the normal rumen bacteria responsible for thiamine synthesis, frequently results in thiamine deficiency. (Ma *et al.*, 2021) ^[15]. Additionally, thiaminase-producing bacteria or certain plants such as bracken fern and horsetail can break down thiamine, leading to deficiency. Long-term usage of coccidiostats such as amprolium may also disrupt the metabolism of thiamine in small ruminants. Water deprivation often caused by frozen water sources or restricted access can result in neurologic manifestations when animals suddenly regain access to water (Hogan *et al.*, 2007) ^[10]. High-sulphur feed ingredients (such as distillers' grains), tainted water, or sulphur-rich plants like Brassica species can all cause sulphur poisoning.

Impaired cerebral energy metabolism is the fundamental process, regardless of the etiology. This disturbance causes cerebral edema, osmotic water changes, sodium buildup in brain cells, and ultimately pressure necrosis of brain tissue. Clinically, affected animals may show incoordination, dullness, aimless wandering, dorsomedial strabismus (particularly in thiamine deficiency cases), opisthotonus, central blindness, and seizures.

The goal of treatment is to reduce brain swelling and repair the deficit. Thiamine is administered intravenously at a dose of 10 mg/kg, and mannitol (1–2 g/kg IV given slowly) may be used to decrease cerebral edema.

2.3. Herpesvirus Encephalitis

Bovine herpesvirus type 1 (BHV-1) and bovine herpesvirus type 5 (BHV-5) are members of the genus *Varicellovirus* within the family *Herpesviridae* and are classified as alpha herpesviruses. Although genetically related, these two viruses differ significantly in their neurovirulence and clinical presentation. BHV-5 is more commonly linked to severe meningoencephalitis, especially in young calves, and has a strong neurotropism. The virus replicates in the upper respiratory mucosa after primary infection, most frequently through the respiratory pathway, and then enters peripheral sensory neurons. Through The virus travels centripetally to

the central nervous system (CNS) by retrograde axonal transport, where it causes degenerative and inflammatory lesions. Both BHV-1 and BHV-5 can cause latent infections in brain ganglia, just like other alpha herpesviruses. Stress or immunosuppression can cause viral reactivation, which can lead to increased viral shedding and transmission to vulnerable animals (Kaur & Chandra 2016) ^[13].

Clinically, BHV-1 is primarily associated with infectious bovine rhinotracheitis (IBR), characterized by respiratory signs such as fever, nasal discharge, conjunctivitis, and coughing. Neurological manifestations may occur but are less common and typically accompany respiratory involvement. In contrast, BHV-5 infection predominantly presents as a neurological disorder with comparatively mild or absent respiratory signs. Affected calves may develop depression, ataxia, blindness, circling, seizures, and recumbency, with high case fatality rates. Histopathologically, non-suppurative meningoencephalitis with neuronal necrosis and perivascular cuffing is a common finding in BHV-5 infections (Meyer *et al.*, 2001) ^[17].

2.4. Rabies

One of the most deadly viral encephalitis affecting both domestic and wild animals, including cattle, is rabies, which is caused by a neurotropic RNA virus that belongs to the genus *Lyssavirus* within the family *Rhabdoviridae*. Transmission typically occurs through the bite of an infected animal, whereby virus-laden saliva is introduced into muscle tissue. Initial viral replication occurs locally within myocytes before the virus enters peripheral nerves at neuromuscular junctions. Using retrograde axoplasmic transport, the virus ascends toward the CNS, where it produces a diffuse, non-suppurative encephalitis. Following CNS replication, centrifugal spread through autonomic and parasympathetic nerves allows viral dissemination to salivary glands and other tissues, facilitating further transmission (Scott & Nel, 2021) ^[21].

The clinical course of rabies in cattle is generally acute and progressive. Early signs may include behavioural changes such as depression or unusual excitability, followed by ataxia, anorexia, hyperesthesia, and ascending paralysis. Pharyngeal paralysis often leads to dysphagia and profuse salivation, giving the characteristic frothy appearance around the mouth. Some animals exhibit aggression or abnormal sexual behaviour. As neuronal dysfunction advances, recumbency, coma, and death typically occur within approximately one week to ten days after onset of clinical signs. Histologically, the disease is characterized by non-suppurative encephalitis with neuronal degeneration and the presence of intracytoplasmic inclusion bodies (Negri bodies) in affected neurons.

Collectively, BHV-associated encephalitis and rabies highlight the critical role of neurotropic viral pathogens in bovine neurological disease. Understanding viral pathogenesis, latency, transmission dynamics, and clinical progression is essential for effective disease surveillance, vaccination strategies, and biosecurity management in livestock populations (Reddy *et al.*, 2026) ^[19].

2.5. Pseudorabies

Cattle, sheep, and goats serve as terminal (dead-end) hosts for pseudorabies, also called Aujeszky's illness, which is caused by suid herpesvirus 1 and mostly affects pigs. Direct contact, such as bites, exposure of mucosal membranes to contaminated fluids, or sporadic aerosol dispersal can all

result in transmission to these species. Affected animals display neurological and behavioural problems after an incubation period of roughly two to seven days. Paresthesia and acute localized pruritus are prominent early symptoms, which frequently result in extensive scratching, self-inflicted damage, and baldness. Other symptoms include circling, sadness, ataxia, fever, and intermittent hostility. Animals experience vocalization, convulsions, recumbency, and severe cerebral dysfunction as the illness worsens, all of which quickly lead to death. The illness is characterized by an abrupt clinical course with significant brain involvement and is usually fatal in non-porcine species (Wilkes & Kattoor, 2022) [22].

2.6. Cerebellar Diseases

Cerebellar dysfunction mainly interferes with coordination, balance, and normal gait, while the animal's level of consciousness and mental status usually remain unaffected. Animals with cerebellar involvement commonly show incoordination (ataxia), exaggerated or misjudged limb movements (dysmetria), swaying of the trunk, and intention tremors that become more pronounced during voluntary movement. Muscle power is generally intact; however, apparent weakness can occur if there is simultaneous damage to the vestibular apparatus, spinal cord, or peripheral nerves.

These clinical manifestations arise because the cerebellum is responsible for fine-tuning and regulating motor activity rather than initiating movement itself. Cerebellar hypoplasia caused by in-utero pestivirus infections, inherited metabolic disorders such as lysosomal storage diseases, or nutritional imbalances like hypomagnesemia that can trigger syndromes like grass tetany are all potential causes of cerebellar disorders (Diab & Krebs 2022) [5].

2.7. Pestiviral Infections (BVDV)

Bovine Viral Diarrhoea Virus (BVDV) is one of the pestiviruses that infect ruminants. BVDV infection can cause fever, respiratory illnesses like pneumonia, reproductive problems, and variable degrees of immunosuppression in animals with healthy immune systems.

The effects may be more serious during pregnancy. Intrauterine infection can cause cerebellar hypoplasia in calves by interfering with normal cerebellar development. Although these calves exhibit incoordination, intention tremors, swaying of the body, and overreaching limb motions (dysmetria), they are often intelligent, attentive, and intellectually normal. When the calf tries to stand or nurse, the incoordination frequently becomes more noticeable.

In other susceptible species including alpacas, deer, goats, pigs, and sheep foetal infection with BVDV can give rise to persistently infected offspring. Such animals continuously shed the virus and commonly exhibit poor growth, reduced weight gain, chronic or intermittent diarrhoea, and repeated secondary bacterial infections due to impaired immune function (Reddy *et al.*, 2026) [19].

2.8. Grass Tetany (Hypomagnesemia)

Grass tetany also referred to as hypomagnesaemic tetany, lactation tetany, grass staggers, winter tetany, or wheat pasture poisoning is a metabolic disorder caused by reduced magnesium levels in the bloodstream. Magnesium deficiency disrupts normal neuromuscular function, leading to characteristic clinical manifestations (Grunes *et al.*, 1970) [9].

In affected cows, signs commonly include incoordination (ataxia), muscle twitching, heightened neuromuscular

excitability, recumbency, convulsions, and eventually rigid tetanic spasms. The condition is termed "grass tetany" because outbreaks frequently occur when animals are first turned out onto lush, rapidly growing spring pasture. A decreased serum magnesium concentration is essential for the development of clinical signs, which arise from both hypomagnesaemia and associated dysfunction of the central nervous system.

Typically, classical tetanic signs develop within a few days after animals begin grazing fresh pasture. Older cattle particularly those in their third or subsequent lactations are at greater risk. Unlike milk fever, this disorder is not directly linked to calving. It is most often observed in ruminants grazing young spring grasses, whereas "winter tetany" tends to occur in animals consuming cereal forages such as wheat (*Triticum aestivum*), barley (*Hordeum vulgare*), oats (*Avena sativa*), and rye (*Secale cereale*). Lactating dairy cows are especially susceptible, particularly during the transition from winter silage feeding to nutrient-rich, fast-growing spring pastures. (Grunes *et al.*, 1970) [9].

2.9. Brainstem and Cranial Nerve Diseases

Lesions involving the brainstem commonly lead to alterations in consciousness, abnormalities in posture, and deficits affecting multiple cranial nerves. The brainstem houses the nuclei of most cranial nerves and is vital for maintaining awareness, coordinating motor activity, and regulating essential autonomic functions. Consequently, animals with brainstem damage may exhibit depression, stupor, or even coma, along with signs such as head tilt, circling, and various cranial nerve abnormalities (Hurley *et al.*, 2010) [11].

Cranial nerves are evaluated as individual functional units during neurological examination. Damage to the optic nerve (CN II) can result in vision loss despite intact ocular structures. Dysfunction of the oculomotor (CN III), trochlear (CN IV), or abducens (CN VI) nerves may cause abnormal eye position (strabismus) and impaired pupillary light responses. Asymmetry of the face, along with diminished palpebral or corneal reflexes, indicates possible involvement of the trigeminal (CN V) and facial (CN VII) nerves (Wilson *et al.*, 2010) [23].

Brainstem and cranial nerve dysfunction may arise from several conditions, including middle or inner ear infections (otitis media or interna), pituitary abscess syndrome, and aberrant migration of parasites. In young calves, otitis media or interna can extend from the ear structures into the brainstem, leading to prominent vestibular disturbances (Wilson *et al.*, 2010) [23].

2.10. Listeriosis

Listeriosis is a common cause of brainstem and cranial nerve disorders in ruminants and is produced by the bacterium *Listeria monocytogenes*. This organism is widely distributed in the farm environment and may be present in the faeces of clinically normal animals. In cattle, the primary predisposing factor is the consumption of poorly fermented or spoiled silage. In sheep and goats, additional sources such as decaying forage and the lower portions of large round hay bales can serve as important reservoirs. Goats, in particular, appear to be highly susceptible to infection.

Clinical presentation can vary, but severe depression and unilateral cranial nerve deficits are typical features. Affected animals may develop facial paralysis characterized by drooping of the ear and eyelid on one side, along with head tilt. Reduced blinking can lead to exposure keratitis.

Involvement of the glossopharyngeal and vagus nerves often results in difficulty swallowing and excessive salivation, which subsequently reduces feed intake and predisposes the animal to dehydration and weight loss.

Prompt intervention is critical for a favourable outcome. Early administration of appropriate antibiotics, such as penicillin or cephalosporins including ceftriaxone, is recommended and therapy should generally continue for at least 5–7 days. Supportive management is equally important, particularly in animals that are unable to swallow or remain standing, and may include fluid therapy and assisted nutritional support (Boileau & Gilliam, 2017) [1].

3. Metabolic Disorders Associate with Neurological Sign

Metabolic disorders causing neurological signs are of major concern in cattle, especially in high-yielding dairy cows and intensively reared calves. These disorders typically arise from imbalances in minerals, energy metabolism, vitamins, or electrolytes, but their most obvious clinical effects often involve the nervous system. Disruptions in metabolic homeostasis impair neuromuscular transmission, neuronal function, and brain activity, producing neurological signs ranging from mild behavioural changes to severe seizures and coma. A classic example is periparturient hypocalcemia (milk fever), frequently observed in high-producing dairy cows around calving. During early lactation, the rapid calcium demand for colostrum and milk may surpass the cow's capacity to mobilize calcium from bone or absorb it from the gut, leading to a drop in blood calcium. This impairs synaptic transmission and muscle contraction, resulting in progressive weakness, tremors, recumbency, reduced alertness, and, in severe cases, flaccid paralysis that can be fatal if untreated. Gross pathological lesions are typically absent, emphasizing the functional and potentially reversible nature of this condition when promptly addressed.

Hypomagnesemic tetany, including grass tetany and transport tetany, predominantly affects lactating or stressed cattle fed magnesium-deficient diets. Magnesium is critical for stabilizing nerve membranes and controlling acetylcholine release at neuromuscular junctions. Low serum magnesium increases neuronal excitability, leading to hyperesthesia, muscle tremors, jaw rigidity (trismus), incoordination, and convulsions. Sudden death may occur in some animals, often with minimal post-mortem lesions, though mild degenerative changes in renal tubules have occasionally been observed.

Energy metabolism disorders, particularly ketosis in early lactation, are also associated with neurological signs. Negative energy balance triggers excessive mobilization of body fat, resulting in accumulation of ketone bodies that can alter brain function. Affected cows may exhibit abnormal licking, hypersalivation, aimless wandering, circling, apparent blindness, or episodes of excitability, commonly referred to as nervous ketosis. Fatty infiltration of the liver is a typical pathological finding, reflecting prolonged metabolic stress.

Electrolyte imbalances, such as water intoxication or salt toxicity, mainly affect calves and arise from osmotic disturbances that induce cerebral edema. Affected animals often exhibit restlessness, tremors, seizures, opisthotonus, and may progress to coma. Histopathology can reveal brain swelling and laminar cortical necrosis. Similarly, ruminal or metabolic acidosis, commonly linked to high-concentrate feeding, leads to systemic acid–base disturbances, dehydration, and endotoxemia. Although primarily a gastrointestinal disorder, severe acidosis can cause depression and secondary neurological signs, with ruminitis and laminitis

being characteristic pathological lesions.

Cerebro cortical necrosis (polio encephalomalacia) is a significant metabolic encephalopathy in young ruminants, often associated with thiamine deficiency or high dietary sulfur intake. It is characterized by cortical neuronal necrosis and cerebral edema, producing signs such as blindness, nystagmus, opisthotonus, seizures, and recumbency. Similarly, vitamin A deficiency in growing cattle affects retinal and optic nerve function, leading to night blindness, ocular dryness (xerophthalmia), and, in chronic cases, optic nerve degeneration due to disrupted cerebrospinal fluid dynamics (Kang *et al.*, 2025) [12].

4. Neurological disorder caused by microorganism

Infectious neurological diseases form an important category of disorders in cattle, impacting animals at various ages and production stages. These diseases may result from viral, bacterial, or prion infections, with clinical signs influenced by the animal's age, immune status, and mode of exposure. Each pathogen induces specific neurological and pathological changes, which are critical for accurate diagnosis and effective management.

Viral infections, such as Bovine Viral Diarrhoea Virus (BVDV), are significant contributors to both congenital and postnatal neurological disorders in cattle. Fetal infection during mid-gestation can severely impair neural development, causing congenital abnormalities such as cerebellar hypoplasia, hydrocephalus, arthrogryposis, and microencephaly. Affected calves may be stillborn or born weak, tremulous, and uncoordinated. In persistently infected animals, viral mutations can later trigger mucosal disease, leading to systemic illness and progressive neurological deterioration due to widespread tissue damage.

Bacterial infections, such as meningitis, are frequently observed in neonatal calves and often arise secondary to septicaemia or localized infections. Clinically, affected calves show fever, neck stiffness, opisthotonus, nystagmus, and depression, which can progress to coma. Gross examination typically reveals thickened, congested meninges coated with fibrin or pus. Common bacterial pathogens include *Escherichia coli*, *Staphylococcus* spp., *Streptococcus* spp., and *Histophilus somni*. Risk factors include inadequate colostrum intake, umbilical infections, and poor hygiene or management practices.

Brain and spinal abscesses commonly affect young cattle and usually develop secondary to bacterial dissemination following trauma (e.g., dehorning), otitis media, sinusitis, or bacteraemia. Clinical manifestations vary with lesion location and may include head tilt, cranial nerve deficits, ataxia, or localized paresis. Frequently isolated pathogens include *Trueperella (Corynebacterium) pyogenes*, *Staphylococcus* spp., and *Streptococcus* spp. Grossly, abscesses appear as well-demarcated purulent lesions within neural tissue.

Aujeszky's disease (pseudorabies), caused by Suid herpesvirus 1, affects cattle of all ages, although they are terminal hosts and do not transmit the virus further. The disease is characterized by severe localized pruritus, particularly around the head and neck, often resulting in self-inflicted injuries. Additional clinical signs include dysphagia, hypersalivation, respiratory distress, and sudden death. Histopathology typically reveals non-suppurative encephalomyelitis, indicative of viral inflammation.

Malignant catarrhal fever (MCF), caused by gamma herpesviruses, predominantly affects adult cattle and is characterized by high fever, nasal and ocular discharge,

corneal opacity, facial swelling, depression, and neurological signs such as head pressing and convulsions. Pathologically, the disease involves widespread vasculitis and lymphoid proliferation, leading to cerebral edema and vascular damage that contribute to neurological dysfunction.

Bovine Spongiform Encephalopathy (BSE) is a fatal prion disease primarily affecting cattle aged three to six years. It manifests as a slowly progressive neurodegenerative disorder, with signs including behavioural changes, nervousness, hypersensitivity to stimuli, tremors, ataxia, and eventual recumbency. Histopathologically, neurons and neuropil exhibit vacuolation, producing a characteristic spongiform appearance without inflammation, which is a hallmark of prion-induced neurodegeneration.

Traumatic disorders

Traumatic conditions represent an important category of neurological disorders in cattle, particularly in newborn calves and periparturient animals. These injuries often arise from difficult births (dystocia), excessive or improper obstetrical intervention, prolonged recumbency, mechanical compression, or vertebral instability. Depending on the site and severity of trauma, peripheral nerves, the spinal cord, or associated vascular structures may be affected, resulting in variable neurological deficits.

In neonatal calves, trauma is most commonly associated with complicated parturition. Excessive traction or abnormal fetal positioning can cause cervical meningeal haemorrhage, leading to clinical signs such as respiratory distress, weak or slow pulse, trapezius muscle paralysis, poor forelimb coordination, and in severe cases, progression to coma. Haemorrhagic lesions disrupt cervical spinal cord function and adjacent neural pathways, producing early-onset neurological deficits.

Peripheral nerve injuries are frequently observed in calves born in posterior presentation. Severe stretching or avulsion of the femoral nerve can cause quadriceps paralysis, noticeable muscle atrophy, and inability to extend the stifle joint, leaving the calf recumbent with the affected hind limb flexed. Compression or trauma to the obturator or sciatic nerves during dystocia may result in hindlimb weakness, difficulty rising, and limb dragging. Similar neuropathies can also occur in periparturient cows following prolonged labour or calving-related trauma.

In adult cattle, especially downer cows, prolonged recumbency increases the risk of ischemic nerve injury. Sustained pressure on major peripheral nerves such as the tibial, peroneal, or radial nerves can lead to compression neuropathies, causing partial or complete paralysis. Prognosis depends on the duration and severity of ischemia, with severe cases potentially resulting in muscle necrosis and permanent neurological deficits.

Spinal cord dysfunction in cattle can result from vertebral fractures, luxations, or space-occupying lesions such as spinal abscesses. Clinical signs typically include paralysis, loss of sensation below the lesion, and sometimes hyperesthesia. In calves, spinal abscesses often develop secondary to septicemia or localized infections, presenting as progressive weakness, spinal pain, and fever. In older breeding bulls or intensively managed cattle, degenerative vertebral changes or ligamentous injuries may cause spinal instability, leading to sudden recumbency due to acute spinal cord compression (Scott *et al.*, 2004) ^[20].

5. Conclusion

Neurological disorders in livestock stem from a combination of metabolic imbalances and central nervous system lesions. Factors such as mineral deficiencies, energy deficits, and electrolyte disturbances can lead to clinical symptoms like depression, ataxia, and seizures, often without visible lesions, emphasizing the need for laboratory diagnostics. Infectious agents including viruses, bacteria, and prions also contribute to neurological diseases, presenting various clinical signs. Toxins and traumatic events further impair neurological function. Accurate neuroanatomical localization and thorough examinations are crucial for diagnosis. Prompt management of these disorders is vital for animal welfare, focusing on metabolic support, infection treatment, and preventive strategies through careful handling, ultimately enhancing livestock production.

Conflict of Interest

Not available

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