Review Report

Neurologic dysfunction in newborn calves: understanding neonatal encephalopathy in clinical practice

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Abstract

Neonatal encephalopathy (NE), a clinically important syndrome causing neurologic dysfunction in newborn calves, contributes to early-life morbidity and mortality. Affected calves exhibit impaired adaptation to extrauterine life, including delayed or absent respiration, difficulty standing, and ineffective nursing, compromising colostrum intake, passive immunity transfer, and overall viability. NE reflects a clinical presentation rather than a specific etiologic diagnosis and may result from hypoxic-ischemic injury, metabolic disturbances, trauma, infection, or intoxication. Historical terms such as 'dummy calf' and weak calf syndrome lack diagnostic precision and should be interpreted cautiously. Neonatal maladjustment syndrome, derived largely from equine and human literature, represents a proposed mechanistic subset of NE, potentially involving persistent neurosteroid activity but remains speculative in calves. Emerging hypotheses, including neurosteroid persistence and maternal gut microbiome influences, may contribute to neonatal maladaptation but require further validation. Management is primarily supportive whereas prevention should focus on dystocia reduction, timely obstetric intervention, and optimized periparturient and early neonatal care. Clarifying terminology and understanding underlying mechanisms is essential to improve clinical decision-making and research interpretation.

Keywords: Cattle, calf, neonatal encephalopathy, maladjustment syndrome, perinatal hypoxia, progesterone, gut microbiome

Introduction

Newborn calves must achieve several critical physiologic and behavioral milestones within hours after birth to ensure survival and long-term health. One of the most immediate is initiation of respiration that ideally occurs within 30 seconds after umbilical cord separation.[1-3] This is followed by the ability to assume a sternal position within minutes and to attempt standing within 15-30 minutes postpartum.[2] Effective nursing is equally essential, with the first successful intake of colostrum ideally occurring within the 2 hours after birth.[1-5] This period represents the window of maximal absorption of immunoglobulin (Ig) G and IgA across the small intestine and is critical for establishing passive and mucosal immunity.[6,7] Failure to achieve these early milestones is associated with increased neonatal morbidity and mortality and may reflect underlying systemic or neurologic dysfunction. Such failures are frequently linked to events occurring during late pregnancy, parturition, or the immediate postpartum period, underscoring the importance of periparturient management in determining neonatal neurologic outcomes.

One important cause of impaired neonatal adaptation is neonatal encephalopathy (NE), a clinical syndrome describing neurologic dysfunction occurring within the first days of life.[8-10] NE is characterized by altered mentation, abnormal reflexes, impaired motor function, seizures, abnormal respiratory patterns, or failure to perform expected neonatal behaviors such as standing and nursing. Importantly, NE is a clinical descriptor rather than an etiologic diagnosis. When a specific cause, such as trauma, infection, intoxication, metabolic disease, or hypoxic-ischemic injury (HII) is identified, the diagnosis should reflect that underlying etiology rather than NE itself. The clinical manifestations and severity of NE vary widely depending on the underlying cause and extent of neurologic injury.

The term neonatal maladjustment syndrome (NMS) has been proposed as a mechanistic subset of NE, based largely on work in foals and extrapolated to calves in limited experimental studies.[8-10] In this context, NMS refers to impaired neurologic transition to extrauterine life, hypothesized to involve persistent neurosteroid activity rather than overt structural

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Citation: Clinical Theriogenology 2026, 18, 13247, http://dx.doi.org/10.58292/CT.v18.13247

brain injury.[10-12] However, peer-reviewed evidence supporting NMS as a distinct and clinically defined syndrome in calves remains limited, and the term should therefore be applied cautiously in bovine practice and research.

In clinical settings, the colloquial term 'dummy calf' is often used to describe neonates with abnormal mentation or behavior. However, this label lacks diagnostic specificity and should not be considered synonymous with NMS or NE.[8-12] Similarly, calves with hypoxic-ischemic encephalopathy (HIE) represent a distinct and clinically significant subset of NE with differing pathophysiology, prognosis, and therapeutic implications. Conflating HIE with maladjustment terminology risks obscuring meaningful distinctions relevant to clinical management and outcome prediction.

Calves affected by NE commonly exhibit central nervous system (CNS) dysfunction that interferes with early postnatal behaviors essential for survival. Clinical features may include disorientation or lethargy, impaired maternal bonding, absence or weakness of the suckle reflex, abnormal responses to sensory stimuli, incoordination, seizures, and failure to thrive. These abnormalities may be evident at birth or may develop within the first hours to days of life, depending on the underlying cause and timing of injury.

The historical term weak calf syndrome (WCS) has also been used to describe neonatal calves that are unable to stand, nurse, or thrive. However, WCS represents a broad and nonspecific umbrella encompassing infectious, nutritional, congenital, environmental, and neurologic causes of neonatal weakness.[8,10] Continued use of this term risks obscuring clinically relevant distinctions among disease processes and complicating diagnostic, therapeutic, and preventive decision-making.

Accordingly, this review focuses on NE in calves, emphasizing clinical recognition, differential diagnosis, and established etiologies such as HII, metabolic disturbances, and traumatic birth-related insults. Proposed maladaptive mechanisms are discussed where relevant. In addition, the review considers management and prevention strategies with particular emphasis on periparturient factors, including dystocia, obstetric intervention, and early neonatal care. Clarifying terminology and disease mechanisms is essential to improving clinical decision-making, accurately interpreting the existing literature, and optimizing outcomes for neonatal calves.

Clinical signs

As outlined above, NE encompasses a spectrum of clinical signs reflecting CNS dysfunction occurring within the first days of life and is therefore considered a clinical syndrome rather than a single disease entity.[8-10] Affected calves fail to achieve normal postnatal milestones, including timely initiation of respiration, standing, and nursing, behaviors essential for survival and early neonatal health.

Clinically, calves with NE may appear alert but exhibit abnormal behavior and impaired neurologic function.[11-13]

Common clinical signs include:

- Failure to nurse due to an absent or weak suckle reflex
- Lethargy or obtundation
- Disorientation and impaired maternal bonding
- Poor coordination (ataxia) and generalized weakness
- Wandering behavior or lack of environmental awareness in ambulatory calves

Additional clinical signs may include:

- Abnormal vocalization
- Tongue protrusion or dysphagia
- Irregular or labored respiratory patterns
- Hypothermia
- Blindness or diminished menace response
- Seizures may occur in more severe cases

These neurologic deficits are consistent with NE across species and may be observed in calves with a variety of underlying etiologies, including HII, metabolic derangements, trauma, or other perinatal insults. The presence, severity, or combination of clinical signs does not reliably distinguish among etiologies and must be interpreted in the context of periparturient history, calving events, and concurrent systemic disease. There are 2 general temporal patterns:

1. Immediate onset with neurologic abnormalities evident at birth
2. Delayed onset with clinical signs developing within the first several hours postpartum

In severe cases, NE may result in perinatal mortality, including stillbirth or death within the first 48 hours of life.[14]

Diagnosis

Diagnosis of NE is clinical and presumptive, based on the identification of neurologic dysfunction in the early neonatal period following initial evaluation for infectious, metabolic, or traumatic causes.[10] Definitive etiologic diagnosis is often not possible antemortem, particularly in field settings.

When necropsy is performed, gross and histologic findings may support identification of a specific underlying etiology rather than NE itself. Lesions such as cerebral edema, neuronal necrosis, and malacia are consistent with HIE and indicate hypoxic brain injury as the cause of neurologic dysfunction.[15-23] In some cases, however, no gross or histologic abnormalities are identified, particularly when neurologic dysfunction reflects functional rather than structural disturbances of the CNS.

Prevalence and economic impact

Estimates of the prevalence of NE in calves are limited, in part because of inconsistent terminology and variable case definitions within the literature. Reports describing weak or nonviable calves have suggested overrepresentation in certain beef breeds; however, dairy calves may also be affected, potentially reflecting differences in calving management and periparturient care.[14] These associations are often weak or statistically insignificant, and clear breed predisposition has not been established.[12,13]

At the herd level, NE is typically sporadic, with prevalence influenced by herd size, calving supervision, nutritional status of the dam, and environmental conditions.[12,13] Periodic

Citation: Clinical Theriogenology 2026, 18, 13247, http://dx.doi.org/10.58292/CT.v18.13247

increases in the number of weak or neurologically abnormal calves attributable to NE are often initially attributed to infectious causes; however, noninfectious contributors such as dystocia, prolonged parturition, nutritional deficiencies, and weather-related stressors should also be considered.[13-17]

Management of calves affected by NE frequently requires intensive supportive care, including assisted feeding,[20-22] thermoregulation,[18,19] monitoring, and environmental support.[5,10,13,17-19] These interventions are labor-intensive and may be particularly challenging during peak calving periods. As a result, producers must balance the likelihood of survival and long-term productivity against the availability of labor and resources. Decisions regarding continued treatment versus culling are influenced by operation size, staffing, economic considerations, and prior experience with neonatal morbidity.

From a production standpoint, an annual calf crop loss of ~ 2% or less is generally considered acceptable for most operations. When losses exceed this threshold, herd-level investigation and modification of management practices, particularly those related to dystocia prevention, obstetric intervention, periparturient monitoring, and environmental protection[18,19] may be warranted to reduce economic loss and improve animal welfare.

Etiology

Although the pathophysiology of NE in calves is not fully defined, a substantial body of evidence, particularly from human and equine medicine, has informed current understanding of established etiologies, most notably HIE. Comparative studies across species have identified several periparturient risk factors relevant to calves, including perinatal hypoxia, dystocia, placental insufficiency, umbilical cord compromise, and excessive traction during assisted parturition.[24] Although species-specific differences exist, the fundamental mechanisms underlying hypoxic-ischemic brain injury are broadly conserved across mammals, supporting cautious extrapolation of these models to bovine neonates.

One well-characterized pathogenic mechanism underlying NE involves disruption of cerebral blood flow and oxygen delivery to the neonatal CNS. Causes of hypoxia-ischemia include dystocia, excessive force during assisted parturition, umbilical cord compression, in utero infection, premature placental separation, and placental insufficiency.[5,16,25-30] Oxygen and glucose deprivation within the brain results in adenosine triphosphate depletion, lactate accumulation, cellular swelling, mitochondrial dysfunction, and injury to neuronal and glial cells.[31-35] Secondary injury cascades include cytokine production, protease activation, generation of reactive oxygen species and nitric oxide, dysregulation of sodium and calcium fluxes, and excessive excitatory neurotransmitter release.[34,35] Collectively, these processes predispose affected calves to reperfusion injury, cerebral edema, neuronal necrosis, excitotoxic damage, and blood-brain barrier disruption.[36,37] These changes are characteristic of HIE and may result in permanent neurologic dysfunction.

A second proposed mechanism involves persistent neurosteroid activity and impaired neonatal neurologic transition, a hypothesis derived primarily from equine and human literature and extrapolated to calves.[37,38] Neurosteroids are synthesized within the CNS from peripheral steroid precursors and include several compounds, most notably progestogens.[38-42] These substances modulate γ-aminobutyric acid (GABA) and other receptor systems, promoting neuronal hyperpolarization and sedation.[39-42] Allopregnanolone and pregnanolone are among the most potent sedative progestogens.[41-44] Although such effects are physiologically beneficial in utero and during parturition, persistence of elevated progestogen concentrations after birth has been hypothesized to interfere with normal neonatal behavioral adaptation.[44]

Calves delivered by cesarian section may be predisposed to prolonged neurosteroid exposure due to altered parturition signaling and incomplete activation of fetal-to-neonatal transition pathways.[44-52] However, elevated progesterone concentrations have also been documented following vaginal delivery, particularly in cases of dystocia.[49,50] Importantly, neurosteroids may exert context-dependent neuroprotective effects, potentially mitigating ischemic injury or supporting recovery following traumatic brain injury.[5,44-52] At present, persistent neurosteroid activity in calves should be regarded as a proposed contributory mechanism affecting a subset of NE cases, rather than an established or primary cause of hypoxic-ischemic brain injury.

Respiratory acidosis may further contribute to neurologic depression in affected neonates.[53] Initiation of effective respiration represents the calf's first critical postnatal physiologic challenge, and failure to establish ventilation is incompatible with life.[54-57] All calves experience some degree of acidosis during parturition, even following uncomplicated deliveries, due to transient hypoxia and carbon dioxide accumulation.[53,58,59] Most neonates compensate through increased respiratory effort, facilitating carbon dioxide elimination and correction of acid-base imbalance.[53-61] However, prolonged or severe hypercapnia may impair this compensatory response, resulting in CNS depression and delayed initiation of respiration.[54-61]

In such cases, reduced neonatal vigor and survival may occur secondary to hypoxemia and metabolic disturbances rather than primary structural neurologic injury. This mechanism represents a functional rather than structural contributor to NE. Further research is needed to clarify the contribution of respiratory and metabolic derangements to the development and severity of NE in calves.

Risk factors

Many factors described in the literature relate broadly to neonatal morbidity, calf vigor, and survival rather than specifically to neonatal encephalopathy (Figure 1). Where applicable, these factors are discussed here in the context of their potential to contribute to perinatal hypoxia, impaired physiologic transition, or increased susceptibility to neurologic injury, rather than as direct causes of encephalopathy.

Several maternal, fetal, and periparturient factors have been associated with abnormal neonatal behavior or weakness and may influence the development or severity of NE, particularly when they predispose calves to HII. Although NMS has been proposed to describe a subset of calves with impaired postnatal neurologic transition, evidence supporting this entity as a distinct diagnosis in calves remains limited. Accordingly, the factors discussed below should be interpreted primarily as contributors to neonatal morbidity with potential relevance

to NE in specific clinical contexts. The historical designation WCS is used in the literature to describe heterogeneous causes of neonatal weakness and is avoided here as a diagnostic category.

Maternal nutrition

Maternal nutrition, particularly during late pregnancy, has a critical role in fetal growth and neonatal viability. Dams with inadequate protein intake are more likely to produce weak or poorly viable calves.[62-64] Approximately ⅔ of fetal growth occurs during the final 90 days of pregnancy, during which the fetus gains approximately 1 pound per day.[62-64] Diets containing less than 10% crude protein during this period are associated with reduced offspring performance and increased neonatal morbidity.[62-64] Although these studies do not specifically evaluate NE, inadequate maternal nutrition may indirectly increase susceptibility to neurologic dysfunction by contributing to low birth weight, reduced vigor, impaired thermoregulation, or increased dystocia risk. Protein intake is also essential for immunoglobulin synthesis, and undernourished dams may produce calves at greater risk for failed passive transfer.[65]

Thermoregulation and hypothermia

Impaired thermoregulation increases the risk of hypothermia and neonatal morbidity. Severe hypothermia is defined as a body temperature below 95°F (35°C).[66,67] Inadequate thermoregulation is often related to insufficient brown adipose tissue, limiting neonatal heat production.[60] Hypothermic calves may exhibit weakness, delayed standing, reduced suckle reflexes, impaired colostrum absorption, and increased mortality.[66-71] Although hypothyroidism is not considered a common primary cause of neonatal weakness, thyroid hormones have a critical role in thermoregulation, and altered neonatal thyroid hormone concentrations may reflect impaired physiologic adaptation.[69] These factors may exacerbate weakness or delay neonatal transition but should not be interpreted as direct causes of NE.

Damage and parity

Dam age and parity influence neonatal outcomes primarily through their association with dystocia and maternal nutritional demands. Heifers are at increased risk for dystocia and cesarian delivery, and calves born under these conditions may demonstrate reduced vigor or abnormal neonatal behavior.[72-74] These associations likely reflect increased risk of perinatal hypoxia rather than a specific maladaptive neurologic syndrome. Older cows may also produce weaker calves if declining dentition or body condition limits nutrient intake despite acceptable forage quality.

Dystocia

Dystocia is among the most consistently documented and clinically relevant risk factors associated with neonatal mortality and neurologic dysfunction.[5,10,71] Calves born following dystocia are more likely to experience perinatal hypoxia and metabolic acidosis, central mechanisms underlying hypoxic-ischemic encephalopathy, a well-established cause of NE.[5,10,53] Factors contributing to dystocia include malpresentation, fetopelvic disproportion, prolonged labor, parity, fetal sex, pregnancy length, and twinning. Persistent acidosis may impair suckle reflexes and reduce immunoglobulin absorption even in calves that initially appear viable.[5,53] When maternal nutrition is adequate, dystocia remains the most important modifiable risk factor associated with neonatal neurologic injury.

Environmental conditions

Environmental stressors such as cold, moisture, and wind increase heat loss and exacerbate cold stress in neonatal calves, particularly in the absence of adequate shelter.[75,76] Severe weather conditions are associated with reduced dam productivity and increased neonatal mortality.[75,76] Cold exposure increases metabolic demand and glucose utilization, which can be especially problematic for calves unable to nurse effectively. Although these factors contribute to neonatal morbidity and mortality, their role in NE is likely indirect and mediated through hypothermia,[18,19] hypoglycemia,[20-22] or delayed neonatal transition rather than primary neurologic injury.

Micronutrient deficiencies

Micronutrient deficiencies have been associated with neonatal weakness and mortality and may indirectly increase susceptibility to hypoxia or impaired neonatal adaptation. Selenium and vitamin E deficiencies are associated with white muscle disease, placental dysfunction, and increased risk of perinatal hypoxia.[77,78] Severe selenium deficiency may result in fatal cardiac lesions. Iodine deficiency may lead to goiter and hypothyroidism, impairing thermoregulation and neonatal viability.[68] Vitamin A deficiency has been associated with increased calf mortality and a range of clinical signs, including neurologic abnormalities, although these findings reflect systemic developmental effects rather than primary encephalopathy.[77,78] These deficiencies should be considered contributors to generalized neonatal morbidity rather than direct causes of NE.

Infectious causes

During periods of increased neonatal morbidity, infectious diseases must be considered as important differential diagnoses rather than causes of neonatal encephalopathy.[29,79,80] Bovine viral diarrhea virus infection may result in abortion, congenital abnormalities, or persistently infected calves, depending on the timing of fetal exposure.[79] Leptospirosis, particularly infection with *Leptospira hardjo*, may cause abortion, stillbirth, or placental insufficiency.[29] These conditions may produce weak or nonviable calves but represent distinct disease processes with different diagnostic, therapeutic, and preventive implications and should be differentiated from NE.

Major maternal, pregnancy, calving, environmental, early neonatal management risk factors contributing the syndrome. The condition is multifactorial and arises from interactions among prenatal nutrition, gut microbiome, placental and fetal development, parturition stress and environmental exposure at birth and impaired colostrum acquisition. These risk factors predispose to NE or general neonatal weakness, but only some directly contribute to neurologic injury (e.g. dystocia-related hypoxia).

Emerging pathophysiological theories

The following sections describe emerging hypotheses that may contribute to impaired neonatal neurologic adaptation or generalized neonatal morbidity in calves. These concepts are

derived largely from human, rodent, and equine literature and should be interpreted as biologically plausible mechanisms requiring further validation in bovine populations. Where relevant, potential links to NE, neonatal maladjustment, or weak calf syndrome are discussed, with emphasis on distinguishing contributory factors from established etiologies.

Theory 1. Neurosteroid activity and the role of progesterone in neonatal brain function

Progesterone is widely recognized as a reproductive hormone essential for estrous cycle regulation and maintenance of pregnancy. Increasing evidence, however, demonstrates that progesterone also has important roles in CNS development, modulation, and protection.[81] As a neurosteroid, progesterone is synthesized locally within the CNS and influences neural excitability, neurodevelopment, and adaptive responses to injury. This section reviews established and emerging roles of progesterone in brain physiology, with emphasis on mechanisms potentially relevant to neonatal neurologic transition rather than as primary causes of neonatal encephalopathy.

Progesterone as a neurosteroid

Although progesterone is primarily produced by the ovaries, adrenal glands, and placenta, it is also synthesized within the CNS by neurons and glial cells. This endogenous production classifies progesterone as a neurosteroid, enabling paracrine and autocrine effects within neural tissue. Neurosteroids exert many of their effects through modulation of neurotransmitter systems, particularly GABA system. Progesterone metabolites, most notably 3α, 5α-tetrahydroprogesterone (allopregnanolone), act as positive allosteric modulators of the γ-aminobutyric acid type A (GABAA) receptor and exhibit barbiturate-like pharmacologic activity.[81,82]

Through enhancement of GABAergic inhibition, progesterone and its metabolites exert sedative, anxiolytic, and anticonvulsant effects and contribute to regulation of arousal states and sleep architecture.[81,82] These properties are particularly relevant during the perinatal period, when rapid neuroendocrine transitions are required to facilitate postnatal behavioral adaptation.

Neuroprotective effects of progesterone

Progesterone has well-documented neuroprotective effects in experimental models, including reduction of neuroinflammation, limitation of excitotoxicity, and inhibition of apoptotic pathways following neural injury. In animal models of traumatic brain injury, progesterone treatment has been associated with improved neurologic outcomes, including reductions in cerebral edema, oxidative stress, and neuronal loss.[83,84] Progesterone also supports oligodendrocyte function and myelin synthesis, contributing to axonal conduction and neural network integrity.[85]

Progesterone further modulates expression of neurotrophic factors such as brain-derived neurotrophic factor that has a critical role in neuronal survival, synaptic plasticity, and adaptive learning processes.[85-87] Collectively, these findings support a biologically plausible neuroprotective role for progesterone but do not establish it as a primary driver of neonatal neurologic disease.

Cognitive, behavioral, and affective effects

Progesterone influences multiple domains of cognitive and behavioral function, including learning, attention, mood regulation, and stress responsiveness.[88] Fluctuations in progesterone concentrations have been associated with variable effects on cognition and behavior across species and life stages.[89,90] Dysregulation of progesterone signaling has been implicated in affective disorders such as premenstrual dysphoric disorder, postpartum depression, and perimenopausal mood instability, largely mediated through neuroactive metabolites such as allopregnanolone acting on GABAA receptors.[91,92]

These observations have prompted investigation of progesterone-based therapies for mood and neurologic disorders, including traumatic brain injury, epilepsy, and neurodegenerative disease.[83,84,93-97] However, results from large clinical trials have been mixed, underscoring the complexity of progesterone signaling and the importance of timing, dosage, and physiologic context.

Progesterone in domestic animals and relevance to neonatal calves

In veterinary medicine, progesterone has been evaluated primarily in the context of seizure disorders and behavior modulation in small animals.[98-102] In dogs, progesterone and its metabolites may exert anticonvulsant effects through enhancement of GABAergic inhibition, paralleling mechanisms described in humans.[99-101] In large animal species, fluctuations in progesterone concentrations have been associated with behavioral changes and altered stress responsiveness, suggesting broader neuroendocrine effects.[103]

In ruminants, progesterone is essential for establishment and maintenance of pregnancy and for supporting fetal development.[103-105] It promotes uterine quiescence, endometrial receptivity, and secretion of histotroph necessary for early embryonic and fetal development prior to placental maturation.[106] Throughout pregnancy, progesterone contributes to regulation of uterine blood flow, immune tolerance, and placental function, thereby supporting fetal growth and organ development.[107]

Disruptions in progesterone signaling during pregnancy have been proposed as contributors to syndromes such as WCS, characterized by calves born alive but weak, unresponsive, or unable to stand and nurse.[5,13,17] Inadequate placental development or function, potentially influenced by suboptimal maternal progesterone concentrations during critical pregnancy windows, may impair fetal muscle and neurologic maturation. Conversely, persistence of elevated neurosteroid concentrations in the neonate has been hypothesized to interfere with postnatal arousal and behavioral adaptation, particularly in calves delivered by cesarean section or following dystocia.

At present, altered progesterone or neurosteroid dynamics should be regarded as proposed contributory mechanisms affecting neonatal vigor or behavioral adaptation, rather than established causes of neonatal encephalopathy.

Theory 2. Maternal gut microbiota and fetal gut-brain axis development as a contributor to weak calf syndrome

The maternal gut microbiota is increasingly recognized as an important regulator of fetal development through its influence on gut-brain axis (GBA). Although this concept has been extensively studied in human and rodent models, its relevance to bovine neonatal health remains largely unexplored. Dysbiosis-associated alterations in fetal neurodevelopment, enteric nervous system maturation, immune programming, and stress-response pathways represent a biologically plausible but unproven mechanism contributing to WCS rather than NE per se.

The WCS is characterized by calves born alive but weak, unable to stand or nurse, and exhibiting poor muscle tone, failure to thrive, and increased susceptibility to disease. The etiology of WCS is multifactorial and includes genetic, nutritional, environmental, metabolic, and infectious contributors. Emerging evidence suggests that maternal influences on fetal development, including maternal gut microbiota composition, may represent an underrecognized risk modifier.

Gut-brain axis development and neonatal function

Although the fetal GBA is not functionally active in utero, its structural and regulatory components develop during pregnancy under the influence of maternal microbial signaling.[108] A balanced maternal microbiome produces metabolites such as short-chain fatty acids,[109] neurotransmitter precursors (e.g. serotonin, GABA), and immune-modulating molecules that may cross the placenta and influence fetal CNS and enteric nervous system development, immune priming, and hypothalamic-pituitary-adrenal axis maturation.[110-112]

Disruption of these developmental processes may plausibly contribute to impaired neuromuscular coordination, delayed suckling behavior, gastrointestinal dysfunction, and increased disease susceptibility observed in weak calves.

- Maternal dysbiosis as a potential risk modifier Maternal dysbiosis during pregnancy, resulting from inadequate nutrition, stress, antimicrobial exposure, or systemic illness, may influence fetal development through several proposed mechanisms:
- Altered neurogenesis and myelination, including vagal nerve development, leading to impaired neuromotor control[113]
- Increased fetal inflammatory signaling via cytokine exposure, predisposing neonates to metabolic dysregulation and stress vulnerability[114,115]
- Disrupted ENS development and intestinal barrier function, impairing nutrient absorption after birth[115-118]
- Impaired immune system maturation, increasing susceptibility to early-life infections[116,117]

These mechanisms are consistent with clinical observations in calves born to dams with suboptimal health, body condition, or dietary management but direct causal relationships have not been established.

Comparative evidence and research gaps

Most mechanistic evidence regarding fetal GBA development originates from human and rodent studies.[46,47,109,119-124] Nevertheless, core biologic principles appear conserved across mammalian species. In precocial species such as cattle, late pregnancy represents a critical period for neural myelination, enteric nervous system maturation, and cortisol-mediated physiologic preparation for birth. Disruption of maternal microbiota signaling during this window could plausibly result in calves with reduced neuromotor tone, impaired thermoregulation, ineffective suckling, and delayed gastrointestinal and immune function.[118]

At present, maternal gut dysbiosis should be regarded as a theoretical and investigational contributor to WCS rather than a demonstrated cause of NE. Further controlled studies in cattle are required before management recommendations can be made.

Therapeutic options

Neonatal encephalopathy

Currently, the management remains primarily supportive, focusing on stabilization of vital functions, prevention of secondary injury, and optimization of neonatal adaptation.[52,120,125,126] Therapeutic interventions are largely symptom-directed and must be tailored to the severity of neurologic dysfunction, systemic compromise, and concurrent metabolic disturbances.

Respiratory support

Respiratory dysfunction is a prominent feature of NE, often resulting from HII, perinatal asphyxia, or delayed postnatal adaptation.[58,124] The HII is the primary driver of respiratory compromise in NE. Initial management involves ensuring airway patency, including manual removal of mucus or debris from the nasal and oral cavities. Historical practices such as suspension by the hind limbs to drain fluid are discouraged because of the potential for diaphragmatic compression and impaired ventilation; if used, suspension should not exceed 90 seconds.[122]

Supportive measures include stimulating the nostrils with a finger or straw, mimicking maternal licking through vigorous rubbing, or brief application of cool water to the head to initiate reflexive respiration.[58,123] Positioning in sternal recumbency is critical for optimal lung expansion, whereas lateral recumbency may compromise ventilation in the dependent lung. Supplemental oxygen therapy may be indicated in calves failing to establish spontaneous respiration, delivered via flow-by, resuscitation bag, or endotracheal intubation with mechanical ventilation.[58,124] Manual ventilation (mouth-to-mouth or mouth-to-nose) is less effective and carries zoonotic risk. Pharmacologic respiratory stimulants, including doxapram, caffeine, and epinephrine, have been utilized in emergency scenarios to enhance ventilation, improve arterial oxygenation, and promote bronchodilation, though these remain extra-label interventions in cattle and require adherence to food animal residue avoidance databank (FARAD) withdrawal guidelines.[58,127,128]

Metabolic and acid-base management

Calves with NE frequently exhibit mixed respiratory and metabolic acidosis due to hypoxia, impaired perfusion, and lactate accumulation.[58] Management focuses on restoring

Citation: Clinical Theriogenology 2026, 18, 13247, http://dx.doi.org/10.58292/CT.v18.13247

adequate oxygenation and ventilation rather than solely correcting acid-base imbalance chemically, as indiscriminate use of sodium bicarbonate may exacerbate hypercapnia in hypoxemic calves. Intravenous fluids and glucose supplementation are indicated when hypoglycemia or poor energy reserves are present, supporting cellular metabolism and promoting early neonatal activity. In NE, lactate-driven metabolic acidosis may persist despite restoration of ventilation and perfusion.

Analgesia and pain management

Parturition-associated trauma may result in musculoskeletal injury, spinal cord compromise, or visceral trauma, necessitating analgesic intervention. Nonsteroidal antiinflammatory drugs (NSAIDs), including meloxicam, flunixin meglumine, or ketoprofen, have been employed to reduce pain, shorten recumbency periods, and facilitate earlier initiation of colostrum intake.[58,129] Pain management may indirectly support neurologic recovery by improving mobility and systemic oxygen delivery.

Thermoregulation

Hypothermia is a common secondary complication in calves with NE and may exacerbate neurologic and systemic dysfunction. Supportive strategies include drying the neonate, removal from environmental stressors, and the use of external heat sources such as heated pads, lamps, or warm towels. Warm water baths (~ 37.5 °C) may be considered if followed by immediate drying.[53] Rapid rewarming, however, may not confer benefit in calves with primary hypoxic–ischemic brain injury, highlighting the importance of differentiating environmental hypothermia from neurologically mediated thermoregulatory impairment. Therapeutic hypothermia (33.5 °C for 72 hours) has demonstrated neuroprotection in human neonates with HIE,[130] but its efficacy and safety in calves remain untested. It is important to distinguish environmental hypothermia from neurologically mediated thermoregulatory dysfunction.

Nutritional support

Giving colostrum early in life is essential to support passive immunity, energy metabolism, and neonatal adaptation. Calves with NE often demonstrate weak suckle reflexes, necessitating assisted feeding via bottle, orogastric tube, or direct dam nursing.[131] In severe cases, intravenous dextrose may be provided to meet immediate energy requirements. Extended absorption windows and impaired immunoglobulin uptake are common, and failure of passive transfer is indicated by serum total protein < 5.0 mg/dl.[132] Plasma transfusion serves as an alternative when colostrum intake is insufficient, or absorption is inadequate.

Considerations for neurosteroid-associated syndromes

Therapies such as the Madigan squeeze technique (MST), described below, is designed to address persistent neurosteroid activity in maladaptive neonates, are not appropriate for NE resulting from HII, metabolic derangements, or infectious etiologies.[48] In these contexts, interventions must target stabilization of organ function, correction of metabolic disturbances, and mitigation of secondary injury rather than manipulation of neurobehavioral states. Mechanistic rationale is extrapolated from equine studies; controlled trials in calves are lacking.

Neonatal maladjustment syndrome

At present, there is no definitive or disease-specific treatment for NMS in calves, particularly when the underlying etiology is unclear. Management is therefore primarily supportive and directed toward stabilization of vital functions, facilitation of neonatal physiologic transition, and prevention of secondary complications.[52,120,125,126] Therapeutic interventions should be individualized based on clinical presentation, severity of neurologic depression, and the presence of concurrent metabolic, respiratory, or traumatic conditions (Table 1).

Respiratory support

Establishment and maintenance of effective ventilation is the initial priority in calves with suspected NMS. Immediate interventions include clearing the nasal passages and oral cavity of mucus or fluids, which may be performed manually or using a bulb syringe.[58] Historical practices such as suspending calves by the hind limbs to facilitate fluid drainage are discouraged, as gravitational displacement of abdominal viscera may restrict diaphragmatic excursion and impair ventilation.[122] If attempted, suspension should be brief and not exceed 90 seconds.

Additional stimulation techniques aimed at initiating respiration include tactile stimulation of the nostrils with a finger or straw, vigorous rubbing with towels to mimic maternal licking, or pouring a small amount of cold water over the head.[58,123] Proper positioning is critical; sternal recumbency promotes bilateral lung expansion, whereas lateral recumbency compromises ventilation of the dependent lung.

Supplemental oxygen may be indicated for calves that fail to establish spontaneous breathing or remain hypoxemic. Delivery methods include flow-by oxygen, resuscitation bags, or endotracheal intubation with mechanical ventilation.[58,124] Manual ventilation using mouth-to-mouth or mouth-to-nose techniques is less effective and poses zoonotic risk.

Acupuncture at the Governing Vessel 26 (GV-26) point has been described as an adjunctive method to stimulate endogenous catecholamine release and support respiratory drive, particularly in cases of apnea.[58] Although widely used in equine neonatology, evidence supporting its efficacy in calves remains limited.

Pharmacologic respiratory stimulants such as doxapram, caffeine, and epinephrine may be considered in emergency settings.[58,127,128] Doxapram acts as a central respiratory stimulant and may transiently improve ventilation and arterial blood gases. Caffeine and epinephrine promote bronchodilation and cardiovascular support. These agents are used extra-label in cattle, and appropriate withdrawal intervals should be determined using FARAD guidance.

Pain management

Calves experiencing dystocia or traumatic delivery may suffer musculoskeletal injury, soft tissue trauma, or less commonly fractures or spinal cord injury. Appropriate analgesia is therefore an important component of supportive care; NSAIDs, including flunixin meglumine, ketoprofen, or meloxicam (extra-label in the United States), may reduce pain, inflammation, and recumbency time, potentially improving mobility

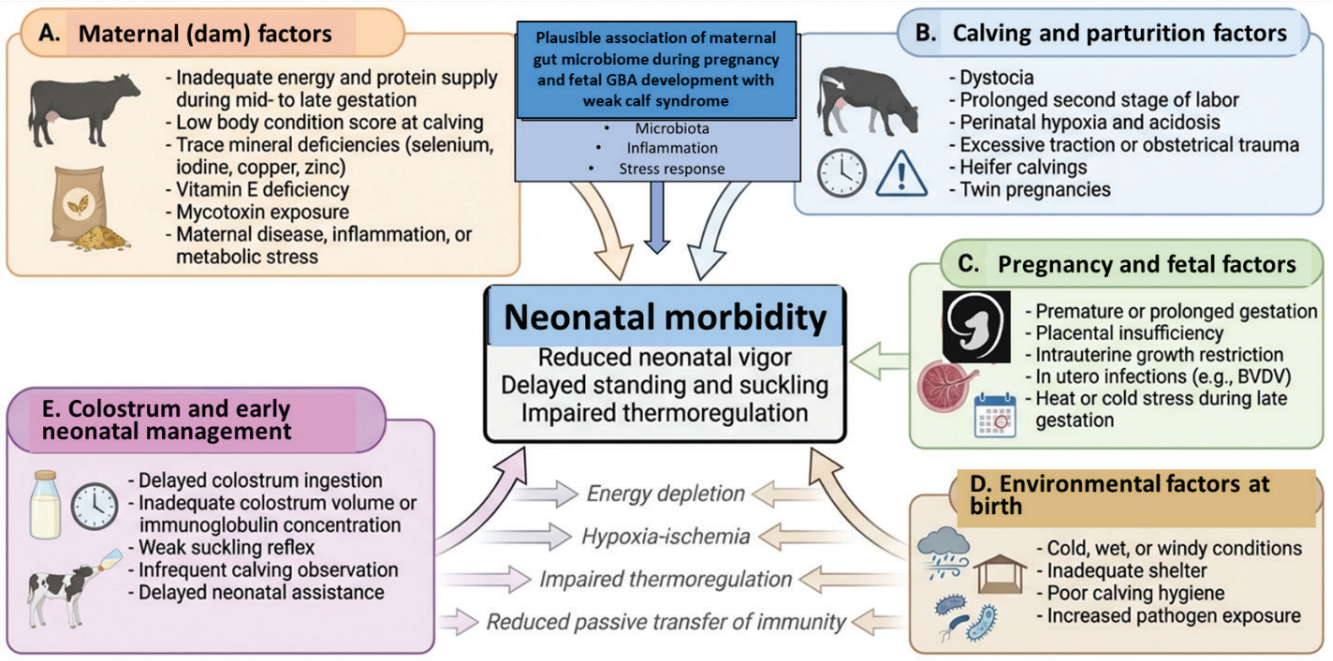

Figure 1. Risk factors for neonatal calf morbidity

and the likelihood of successful colostrum intake.[58,129] Analgesic use should be balanced against hydration status and renal function.

Management of respiratory and metabolic acidosis

Calves with NMS frequently exhibit respiratory acidosis secondary to hypoventilation.[58] Management should focus on improving ventilation and oxygen delivery rather than chemical correction. Sodium bicarbonate treatment is generally discouraged in hypoventilating calves, as it may worsen hypercapnia and intracellular acidosis if carbon dioxide elimination remains impaired.

In cases of perinatal asphyxia, a mixed respiratory-metabolic acidosis may be present. Although respiratory components often improve rapidly with effective ventilation, lactate-driven metabolic acidosis may persist longer and typically resolves with restoration of tissue perfusion and oxygenation rather than direct buffering.

Thermoregulation

Hypothermia is common in compromised neonates and contributes to weakness, hypoglycemia, and impaired immune function. Initial management includes drying the calf, removing environmental stressors, and providing external heat using warm towels, heat lamps, or warming pads. Warm water immersion (~ 37.5 °C) may be considered if followed by thorough drying to prevent evaporative heat loss.

It is important to distinguish environmental hypothermia from neurologic depression due to hypoxic–ischemic injury, as calves with true brain injury may demonstrate limited response to aggressive rewarming.[53] Therapeutic hypothermia has demonstrated neuroprotective benefits in human neonates with hypoxic-ischemic encephalopathy,[130] but this approach has not been validated in calves. Current veterinary management prioritizes controlled rewarming, particularly when hypothermia is associated with hypoglycemia, inadequate colostrum intake, or environmental exposure.

Colostrum management

Early assessment and support of colostrum intake are essential components of care. Calves with NMS often exhibit weak or absent suckle reflexes, necessitating assisted feeding via bottle, orogastric tube, or supervised nursing from the dam.[131] Supplemental intravenous dextrose may be indicated to address hypoglycemia and support energy requirements.

Impaired intestinal motility and delayed gut closure may alter immunoglobulin absorption in affected calves. Serum total protein concentrations below 5.0 g/dl are indicative of failure of passive transfer.[132] When enteral absorption is inadequate or delayed beyond the effective window, plasma transfusion provides an alternative means of passive immunity.

Madigan squeeze technique

A technique that has been proposed as a therapeutic intervention for calves with suspected persistent neurosteroid exposure but is unlikely to benefit calves with hypoxic-ischemic brain injury, infectious disease, nutritional deficiencies, or structural trauma.[48] Originally developed for equine neonates, MST involves application of sustained thoracic pressure intended to simulate birth canal compression and transiently induce a controlled, sleep-like state (Figure 2).

Procedure overview:

- A rope loop is placed around the neck and one forelimb, extending along the thorax

Citation: Clinical Theriogenology 2026, 18, 13247, http://dx.doi.org/10.58292/CT.v18.13247

Figure 2. Simulating birth canal pressure: Madigan squeeze technique in calves

- Gradually increasing pressure (~ 10-20 lb) is applied until the calf becomes recumbent
- Pressure is maintained for ~ 20 minutes
- Upon release, some calves demonstrate improved alertness, suckle reflex, and responsiveness

Calves most likely to benefit include those delivered by cesarian, rapidly delivered calves, or calves born following prolonged but nonasphyxiating parturition. In calves with cardiopulmonary compromise (e.g. rib fractures or suspected thoracic injury) MST is contraindicated. Although the technique is low-cost and minimally invasive, controlled studies in calves are limited, and MST should be considered an adjunct rather than a replacement for standard supportive medical care.

Therapeutic options: Comparison of neonatal encephalopathy and neonatal maladjustment syndrome

Neonatal encephalopathy and neonatal maladjustment syndrome in calves share overlapping clinical features, including neurologic depression, impaired respiration, weak suckle reflex, and metabolic disturbances.[52,120,125,126] Both conditions require prompt stabilization and supportive care, but the underlying pathophysiology dictates differences in therapeutic priorities and expected responses.

Keypoints

- Both NE and NMS require individualized, symptom-directed supportive care, with emphasis on respiratory stabilization, thermoregulation, and nutrition.
- NE management prioritizes correction of hypoxia, metabolic derangements, and secondary injury whereas NMS management may include behavioral stimulation interventions like MST.
- Recognition of the underlying pathophysiology is critical to avoid inappropriate interventions (e.g. MST in hypoxic NE) and to optimize neonatal outcomes.
- Interventions effective for NMS may not be beneficial in NE caused by hypoxia, metabolic derangements, or infection.
- Failure of passive transfer is common in both NE and NMS and may require plasma supplementation

Prevention

Given the absence of definitive curative therapies for NE, prevention remains the most effective strategy. Management of maternal nutrition, minimization of perinatal stressors, and environmental optimization during late pregnancy are central to reducing the risk of NE and supporting fetal growth, neurological maturation, and neonatal vitality.[52,85] Last 60 days of pregnancy are particularly critical, as maternal nutrient requirements escalate and fetal growth accelerates.

Nutritional management

Optimal maternal nutrition is essential for supporting fetal development and reducing the incidence of NE. Feed quality should be assessed through forage analysis, and intake must be carefully monitored, as physical constraints (e.g. reduced rumen volume in late pregnancy) may limit consumption. Provision of low-protein forage alone is insufficient; forages with ≥ 12% crude protein (CP) are recommended. When this target cannot be achieved, supplemental protein should be provided, though adequate intake monitoring is necessary to ensure efficacy. For example, providing hay with > 10% CP in combination with at least 2 lb of supplemental protein per cow per day has been associated with improved neonatal outcomes.[52,85]

Energy requirements vary according to stage of pregnancy, dam parity, environmental conditions, and whether the dam is a growing heifer that may require additional protein and energy to support concurrent maternal growth and fetal development.[52,85]

Micronutrient and vitamin supplementation is equally critical. Herd mineral status may be assessed through liver biopsy or serum sampling, ensuring representative sampling for accuracy. Selenium deficiency, commonly observed in many regions in USA, predisposes neonates to conditions such as white muscle disease (WMD), often accompanied by vitamin E deficiency.[85] Oral supplementation of selenium and vitamin E supports both maternal and fetal health, and whole-blood testing (e.g. 6 weeks later) after supplementation can assess efficacy. Iodine and manganese are also essential during late pregnancy whereas vitamin A supplementation may be required during periods of poor-quality forage, such as drought years.[17]

Important nutrient and micronutrient recommendations for late pregnancy are summarized (Table 2).

Minimizing dystocia

Dystocia is a well-established risk factor for NE due to perinatal hypoxia and trauma.[52,85,131] Preventive measures include culling dams with a history of calving difficulties, selecting sires with demonstrated calving ease, and performing pelvimetry in replacement heifers. Farm personnel should be trained in timely recognition and intervention during dystocia, and established protocols for veterinary consultation are recommended. Proactive management reduces neonatal hypoxia, stress, and trauma, all of which are critical in preventing NE.

Environmental management

Environmental stressors can exacerbate NE risk, particularly in preterm or weak neonates. Providing additional feed energy during periods of severe weather, along with shelter, windbreaks, and straw bedding, supports thermoregulation. A warm, dry environment reduces the energy expenditure required for maintaining body temperature, allowing neonates to prioritize standing, nursing, and establishing early postnatal adaptation.[52,131]

Herd-level infectious disease management

Maternal vaccination and herd biosecurity are essential components of NE prevention. Bovine viral diarrhea virus (BVDV) is managed through vaccination, maintenance of closed herds, screening and isolation of new additions, identification of persistently infected animals, and culling carriers.[18] Preventing viral introduction is generally more effective than attempting eradication after establishment. Leptospirosis control is similarly achieved through vaccination, though vaccines do not cover all serovars, and immunity typically persists for approximately 6 months. Unlike BVDV, complete eradication of Leptospira from a herd is often impractical.[18]

Conclusion

Neonatal encephalopathy in calves is a multifactorial condition arising from a complex interaction of maternal, perinatal, and neonatal factors. Recognized risk factors include inadequate maternal nutrition, micronutrient deficiencies, dystocia, periparturient stress, hypoxic-ischemic events, environmental challenges, and exposure to infectious agents such as BVDV or Leptospira. These contributors may work alone or in combination, producing a spectrum of clinical presentations ranging from mild neurobehavioral depression to severe neurologic

Table 1. Therapeutic considerations for neonatal encephalopathy (NE) versus neonatal maladjustment (NMS)

Therapeutic domain	NE	NMS	Similarities
Respiratory support	Focus on hypoxia and perfusion; oxygenation, mechanical ventilation	Focus on maladaptive neurobehavior; oxygenation plus adjuncts like GV-26 acupuncture	Airway clearance, positioning, supplemental O^2, emergency stimulants
Metabolic/acid-base	Lactate-driven metabolic acidosis; intravenous glucose, restore perfusion	Respiratory acidosis from hypoventilation; resolves with improved ventilation	Prioritize ventilation over chemical correction
Thermoregulation	May reflect brain injury; aggressive rewarming less effective; experimental hypothermia untested	Often secondary to weakness; controlled rewarming effective	Drying, environmental control, external heat sources
Nutritional support	Weak suckle reflex due to neurologic depression; colostrum, intravenous glucose, plasma as needed	Weak suckle due to maladaptive behavior; colostrum, intravenous glucose, MST may assist feeding	Assisted feeding, plasma transfusion if passive transfer fails
Pain management	Supports mobility and systemic recovery	Reduces trauma-related discomfort; facilitates behavioral normalization	NSAIDs as adjunct, shorten recumbency
Condition-specific therapy	MST not indicated; focus on stabilization and metabolic correction	MST may improve neurobehavioral adaptation	Supportive care remains cornerstone

Table 2. Important nutrient and micronutrient recommendations for late pregnancy

Nutrient/ micronutrient	Typical requirement	Notes/recommendations
Protein	10% CP	Increase to ≥ 12% CP
Iodine	0.5 mg/kg	Recommended supplementation
Manganese	50 mg/kg	Supports neonatal development; may not fully meet dam requirements
Selenium	0.3 mg/kg	Maximum safe dosage; oral supplementation preferred

Citation: Clinical Theriogenology 2026, 18, 13247, http://dx.doi.org/10.58292/CT.v18.13247

dysfunction. Persistent neurosteroid activity has been proposed as an emerging mechanistic theory, particularly in calves with NMS, highlighting the heterogeneity of NE and underscoring the need for precise terminology to guide diagnosis, management, and research.

Therapeutic interventions for NE are primarily supportive and symptom-directed. Supportive care remains the cornerstone; disease-specific interventions should be applied only when etiology is clearly identified. Respiratory support, thermoregulation, correction of metabolic disturbances, giving colostrum early, and analgesia form the cornerstone of care. Adjunctive therapies (e.g. pharmacologic respiratory stimulants or the Madigan squeeze technique) may benefit select calves, although evidence in cattle remains limited. Individualized assessment is essential, as therapies effective for NMS associated with neurosteroid persistence may not benefit calves with hypoxic-ischemic or metabolic etiologies.

Preventive strategies remain the most reliable means of improving neonatal survival and long-term productivity. These include optimizing maternal nutrition and micronutrient status during late pregnancy, minimizing dystocia through sire selection and heifer management, ensuring supportive environmental conditions, and implementing herd-level infectious disease control. Vigilant neonatal monitoring allows early recognition and timely intervention, reducing morbidity and improving outcomes.

Future research priorities include validation of emerging therapies, detailed characterization of NE subtypes, and elucidation of maternal and fetal determinants of neonatal susceptibility. A holistic approach integrating both maternal and neonatal care is essential for reducing NE incidence, enhancing welfare, and supporting sustainable herd productivity.

Conflict of interest

None to report.

References

1. Probo M, Veronesi MC: Clinical scoring systems in the newborn calf: an overview. Animals (Basel) 2022;12:3013. doi: 10.3390/ani12213013
2. Mee JF: Newborn dairy calf management. Vet Clin North Am Food Anim Pract 2008;24:1-17. doi: 10.1016/j.cvfa.2007.10.002
3. Mee JF: Neonatal calf vitality. In: Simões J: editor. Encyclopedia of Livestock Medicine for Large Animal and Poultry Production. Springer; Cham: 2025. p. 1-44.
4. Bienboire-Frosini C, Muns R, Marcet-Rius M, et al: Vitality in newborn farm animals: adverse factors, physiological responses, pharmacological therapies, and physical methods to increase neonate vigor. Animals (Basel) 2023;13:1542. doi: 10.3390/ani13091542
5. Murray CF, Leslie KE: Newborn calf vitality: risk factors, characteristics, assessment, resulting outcomes and strategies for improvement. Vet J 2013;198:322-328. doi: 10.1016/j.tvjl.2013.06.007
6. Gamsjäger L, Haines DM, Pajor EA, et al: Impact of volume, immunoglobulin G concentration, and feeding method of colostrum product on neonatal nursing behavior and transfer of passive immunity in beef calves. Animal 2021;15:100345. doi: 10.1016/j.animal.2021.100345
7. Homerosky ER, Caulkett NA, Timsit, et al: Clinical indicators of blood gas disturbances, elevated L-lactate concentration and other abnormal blood parameters in newborn beef calves. Vet J 2017;219:49-57. doi: 10.1016/j.tvjl.2016.12.001
8. Stauber EH: Weak calf syndrome: a continuing enigma. J Am Vet MedAssoc1976;168:223-225.doi:10.2460/javma.1976.168.03.223
9. Constable PD, Hinchcliff KW, Done SH, et al: Perinatal diseases. In: Veterinary Medicine: A Textbook of the Diseases of Cattle, Horses, Sheep, Pigs and Goats. 11th edition, St. Louis, MO; Elsevier: 2017. p. 1830-1903.
10. Bianco AW, Moore GE, Taylor SD: Neonatal encephalopathy in calves presented to a university hospital. J Vet Intern Med 2017;31:1892-1899. doi: 10.1111/jvim.14821
11. Barrier AC, Haskell MJ, Birch S, et al: The impact of dystocia on dairy calf health, welfare, performance and survival. Vet J 2013;195:86-90. doi: 10.1016/j.tvjl.2012.07.031
12. Ward J: Weak calf syndrome. Proc Am Assoc Bovine Pract 1973; p. 97-105.
13. Mee JF: Why do so many calves die on modern dairy farms and what can we do about calf welfare in the future? Animals (Basel) 2013;3:1036-1057. doi: 10.3390/ani3041036
14. Norquay R, Orr J, Norquay B, et al: Perinatal mortality in 23 beef herds in Orkney: incidence, risk factors and aetiology. Vet Rec 2020;187:28. doi: 10.1136/vr.105536
15. Abutarbush SM, Radostits OM: Congenital nutritional muscular dystrophy in a beef calf. Can Vet J 2003;44:738-739.
16. Baker JC: The clinical manifestations of bovine viral diarrhea infection. Vet Clin North Am Food Anim Pract 1995;11:425-445. doi: 10.1016/s0749-0720(15)30460-6
17. Jung Y, Kim B, Ku JY, et al: Physiological alterations and predictors of death in neonatal calves with weak calf syndrome. Vet Rec 2025;197(3):e5327. doi: 10.1002/vetr.5327
18. Rowan TG: Thermoregulation in neonatal ruminants. In: Varley MA, Williams PEV, Lawrence TLJ: editors. Neonatal Survival and Growth. Edinburgh, UK; British Society of Animal Production: 1992. p. 13-24.
19. Mota-Rojas D, Wang D, Titto CG, et al: Neonatal infrared thermography images in the hypothermic ruminant model: anatomical-morphological-physiological aspects and mechanisms for thermoregulation. Front Vet Sci 2022;9:963205. doi: 10.3389/fvets.2022.963205
20. Mohseni, S: Neurologic damage in hypoglycemia. Handb Clin Neurol 2014;126:513-532. doi: 10.1016/B978-0-444-53480-4.00036-9
21. Tennant B, Harrold D, Reina-Guerra M: Hypoglycemia in neonatal calves associated with acute diarrhea. Cornell Vet 1968;58:136-146.
22. Ider M, Naseri A, Ok M, et al: Surveilling brain damage using brain biomarkers in hypoglycemic neonatal calves with diarrhea. Front Vet Sci 2023;10:1240846. doi: 10.3389/fvets.2023.1240846
23. Dore V, Smith G: Cerebral disorders of calves. Vet Clin North Am Food Anim Pract 2017;33:27-41. doi: 10.1016/j.cvfa.2016.09.004
24. Mee JF, Sánchez-Miguel C, Doherty M: Influence of modifiable risk factors on the incidence of stillbirth/perinatal mortality in dairy cattle. Vet J 2014;199:19-23. doi: 10.1016/j.tvjl.2013.08.004
25. Smyth JA, Fitzpatrick DA, Ellis WA: Stillbirth/perinatal weak calf syndrome: a study of calves infected with Leptospira. Vet Rec 1999;145:539-42. doi: 10.1136/vr.145.19.539

26. Gardiner RM: Cerebral blood flow and oxidative metabolism during hypoxia and asphyxia in the new-born calf and lamb. J Physiol 1980;305:357-76. doi: 10.1113/jphysiol.1980.sp013369
27. Hammer CJ, Tyler HD: Effects of early rupture of the umbilical vessels in Jersey calves. J Dairy Sci 1999;82 Suppl:49.
28. Szenci O, Taverne MA, Bakonyi S, et al: Comparison between pre- and postnatal acid-base status of calves and their perinatal mortality. Vet Q 1988;10:140-144. doi: 10.1080/01652176.1988.9694161
29. Mee JF: Premature expulsion of the placenta and bovine perinatal mortality. Vet Rec 1991;128:521-523. doi: 10.1136/vr.128.22.521
30. Smail NL, Adnane M, Wagener K, et al: Roadmap to dystocia management-guiding obstetric interventions in cattle. Life (Basel) 2025;15:457. doi: 10.3390/life15030457
31. Wood CE, Keller-Wood M: Current paradigms and new perspectives on fetal hypoxia: implications for fetal brain development in late gestation. Am J Physiol Regul Integr Comp Physiol 2019;317:R1-R13. doi: 10.1152/ajpregu.00008.2019
32. Mota-Rojas D, Villanueva-García D, Solimano A, et al: Pathophysiology of perinatal asphyxia in humans and animal models. Biomedicines 2022;10:347. doi: 10.3390/biomedicines10020347
33. Garfinkle J, Steven P, Miller SP: The placenta and neurodevelopment in preterm newborns. Neoreviews 2018;19:e456-e466. doi: 10.1542/neo.19-8-e456
34. Almeida A, Delgado-Esteban M, Bolaños JP, et al: Oxygen and glucose deprivation induces mitochondrial dysfunction and oxidative stress in neurones but not in astrocytes in primary culture. J Neurochem 2002;81:207-217. doi: 10.1046/j.1471-4159.2002.00827.x
35. Jagečić D, Petrović DJ, Šimunić I, et al: The oxygen and glucose deprivation of immature cells of the nervous system exerts distinct effects on mitochondria, mitophagy, and autophagy, depending on the cells' differentiation stage. Brain Sci 2023;13:910. doi: 10.3390/brainsci13060910
36. Woods JR Jr: Birth asphyxia: pathophysiologic events and fetal adaptive changes. Clin Perinatol 1983;10:473-486. doi: 10.1016/S0095-5108(18)30980-1
37. Fellman V, Raivio KO: Reperfusion injury as the mechanism of brain damage after perinatal asphyxia. Pediatr Res 1997;41:599-606. doi: 10.1203/00006450-199705000-00001
38. Hirst JJ, Palliser HK, Yates DM, et al: Neurosteroids in the fetus and neonate: potential protective role in compromised pregnancies. Neurochem Int 2008;52:602-610. doi: 10.1016/j.neuint.2007.07.018
39. Slotkin TA, Lagercrantz H: The stress of being born. Sci Am 1986;254:100. doi: 10.1038/scientificamerican0486-100
40. Baulieu EE: Neurosteroids: of the nervous system, by the nervous system, for the nervous system. Recent Prog Horm Res 1997;52:1-32.
41. Zhu W, Huang L, Cheng H, et al: GABA and its receptors' mechanisms in the treatment of insomnia. Heliyon 2024;10:e40665. doi: 10.1016/j.heliyon.2024.e40665
42. Chen RJ, Sharma S: GABA receptor. In: StatPearls [Internet]. Treasure Island, FL; StatPearls Publishing: 2025. [updated 18 February 2025]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK526124/ [cited 25 June 2025].
43. Pinna G, Uzunova V, Matsumoto K, et al: Brain allopregnanolone regulates the potency of the GABAA receptor agonist muscimol. Neuropharmacology 2000:39:440-448. doi: 10.1016/S0028-3908(99)00149-5
44. Timby E, Balgård M, Nyberg S, et al: Pharmacokinetic and behavioral effects of allopregnanolone in healthy women. Psychopharmacology (Berl) 2006;186:414-424. doi: 10.1007/s00213-005-0148-7
45. van Broekhoven F, Bäckström T, van Luijtelaar G, et al: Effects of allopregnanolone on sedation in men, and in women on oral contraceptives. Psychoneuroendocrinology 2007;32:555-564. doi: 10.1016/j.psyneuen.2007.03.009
46. Hu Y, Jin P, Peng J, et al: Different immunological responses to early-life antibiotic exposure affecting autoimmune diabetes development in NOD mice. J Autoimmun 2016;72:47-56. doi: 10.1016/j.jaut.2016.05.001
47. Braniste V, Al-Asmakh M, Kowal C, et al: The gut microbiota influences blood-brain barrier permeability in mice. Sci Transl Med.2014;6:263ra158. doi: 10.1126/scitranslmed.3009759
48. Stilwell G, Mellor DJ, Holdsworth SE: Potential benefit of a thoracic squeeze technique in two newborn calves delivered by caesarean section. N Z Vet J 2020;68:65-68. doi: 10.1080/00480169.2019.1670115
49. Jöchle W, Esparza H, Giménez T, et al: Inhibition of corticoid-induced parturition by progesterone in cattle: effect on delivery and calf viability. J Reprod Fertil 1972;28:407-412. doi: 10.1530/jrf.0.0280407
50. Kindahl H, Kornmatitsuk B, Königsson K, et al: Endocrine changes in late bovine pregnancy with special emphasis on fetal well-being. Domest Anim Endocrinol 2002;23:321-328. doi: 10.1016/s0739-7240(02)00167-4
51. Hirst JJ, Yawno T, Nguyen P, et al: Stress in pregnancy activates neurosteroid production in the fetal brain. Neuroendocrinology 2006;84:264-274. doi: 10.1159/000097990
52. Brunton PJ, Russell JA, Hirst JJ: Allopregnanolone in the brain: protecting pregnancy and birth outcomes. Prog Neurobiol 2014;113:106-136. doi: 10.1016/j.pneurobio.2013.08.005
53. Bleul U: Respiratory distress syndrome in calves. Vet Clin North Am Food Anim Pract 2009;25:179-193. doi: 10.1016/j.cvfa.2008.10.002
54. Bellows RA, Lammoglia MA: Effects of severity of dystocia on cold tolerance and serum concentrations of glucose and cortisol in neonatal beef calves. Theriogenology 2000;53:803-813. doi: 10.1016/S0093-691X(99)00275-7
55. Frosali S, Di Simplicio P, Perrone S, et al: Glutathione recycling and antioxidant enzyme activities in erythrocytes of term and preterm newborns at birth. Biol Neonate 2004;85:188-194. doi: 10.1159/000075814
56. Rice LE: Dystocia-related risk factors. Vet Clin North Am Food Anim Pract 1994;10:53-68. doi: 10.1016/s0749-0720(15)30589-2
57. Kirovski D: Endocrine and metabolic adaptations of calves to extra-uterine life. Acta Vet Beograd 2015;65:297-318. doi: 10.1515/acve-2015-0025
58. Smith GW: Resuscitation of the newborn calf. Proc Am Assoc Bovine Pract 2022;55:108-110. doi: 10.21423/aabppro20228617
59. Bleul U, Wey C, Meira C, et al: Assessment of postnatal pulmonary adaption in bovine neonates using electric impedance tomography (EIT). Animals (Basel) 2021;11:3216. doi: 10.3390/ani11113216

60. Rollin F, Danlois F, Alianoui H, et al: Respiratory distress syndrome in full-term newborn calves. Academia.edu: 2017. Available from: https://calfsessions.com/wp-content/uploads/2017/10/Calf-Sessions-template_Respiratory-Distress-Syndrome.pdf [cited 4 August 2025].
61. Donnelly CG, Quinn CT, Nielsen SG, et al: Respiratory support for pharmacologically induced hypoxia in neonatal calves. Vet Med Int 2016;2016:2129362. doi: 10.1155/2016/2129362
62. Wichman LG, Redifer CA, Meyer AM: Maternal nutrient restriction during late gestation reduces vigor and alters blood chemistry and hematology in neonatal beef calves. J Anim Sci 2023;101:skad342. doi: 10.1093/jas/skad342
63. Caton JS, Hess BW: Maternal plane of nutrition: impacts on fetal outcomes and postnatal offspring responses. In: Proc Graz Lives Nutr Conf, Casper, WY, 2010, pp. 104-119.
64. Funston RN, Larson DM, Vonnahme KA: Effects of maternal nutrition on conceptus growth and offspring performance: implications for beef cattle production. J Anim Sci 2010;88(13 Suppl):E205-E215. doi: 10.2527/jas.2009-2351
65. Quigley JD 3rd, Drewry JJ: Nutrient and immunity transfer from cow to calf pre- and postcalving. J Dairy Sci 1998;81:2779-2790. doi: 10.3168/jds.S0022-0302(98)75836-9
66. Carstens GE: Cold thermoregulation in the newborn calf. Vet Clin North Am Food Anim Pract 1994;10:69-106. doi: 10.1016/S0749-0720(15)30590-9
67. Okamoto M, Robinson JB, Christopherson RJ, et al: Summit metabolism of newborn calves with and without colostrum feeding. Can J Anim Sci 1986;66:937-944. doi: 10.4141/cjas86-103
68. Azzam SM, Kinder JE, Nielsen MK, et al: Environmental effects on neonatal mortality of beef calves. J Anim Sci 1993;71:282-290. doi: 10.2527/1993.712282x
69. Takahashi K, Takahashi E, Ducusin RJ, et al: Changes in serum thyroid hormone levels in newborn calves as a diagnostic index of endemic goiter. J Vet Med Sci 2001;63:175-178. doi: 10.1292/jvms.63.175
70. USDA. Death loss in U.S. cattle and calves due to predator and nonpredator causes, 2015. Fort Collins (CO); USDA–APHIS–VS–CEAH: 2017. Available from: https://www.aphis.usda.gov/sites/default/files/cattle_calves_deathloss_2015.pdf [cited 25 June 2025]
71. Lombard JE, Garry FB, Tomlinson SM, et al: Impacts of dystocia on health and survival of dairy calves. J Dairy Sci 2007;90:1751-1760. doi: 10.3168/jds.2006-295
72. Aleman M, Chigerwe M, Varga A, et al: Steroid precursors, steroids, neuroactive steroids, and neurosteroids concentrations in serum and saliva of healthy neonatal heifer Holstein calves. J Vet Intern Med 2020;34:2767-2775. doi: 10.1111/jvim.15957
73. Bernard WV, Reimer JM, Cudd T: Historical factors, clinicopathologic findings, clinical features, and outcome of equine neonates presenting with or developing signs of central nervous system disease. Proc Am Assoc Equine Pract 1995;41:222-224.
74. Mee JF: Impacts of dairy cow nutrition precalving on calf health. JDS Commun 2023;4:245-249. doi: 10.3168/jdsc.2022-0281
75. Nonnecke BJ, Foote MR, Miller BL, et al: Effects of chronic environmental cold on growth, health, and select metabolic and immunologic responses of preruminant calves. J Dairy Sci 2009;92:6134-6143. doi: 10.3168/jds.2009-2517
76. Young BA: Ruminant cold stress: effect on production. J Anim Sci 1983;57(6):1601-1607. doi: 10.2527/jas1983.5761601x
77. Majors C, Myers A, Kasimanickam R: Selenium in cattle diseases and reproductive health. Clinical Theriogenology 2025;17:1-18. doi: 10.58292/CT.v17.12015
78. Waldner CL, Uehlinger, FU: Factors associated with serum vitamin A and vitamin E concentrations in beef calves from Alberta and Saskatchewan and the relationship between vitamin concentrations and calf health outcomes. Can J Anim Sci 2017;97:65-82. doi: 10.1139/cjas-2016-0055
79. Grooms DL: Reproductive consequences of infection with bovine viral diarrhea virus. Vet Clin North Am Food Anim Pract 2004;20:5-19. doi: 10.1016/j.cvfa.2003.11.006
80. Anderson ML: Disorders of cattle. In: Njaan BL: editor. Kirkbride's Diagnosis of Abortion and Neonatal Loss in Animals. Chichester, West Sussex, UK; Wiley-Blackwell: 2012. p. 30-32.
81. Kolatorova L, Vitku J, Suchopar J, et al: Progesterone: a steroid with wide range of effects in physiology as well as human medicine. Int J Mol Sci 2022;23:7989. doi: 10.3390/ijms23147989
82. Hauser CA, Chesnoy-Marchais D, Robel P, et al: Modulation of recombinant alpha 6 beta 2 gamma 2 GABAA receptors by neuroactive steroids. Eur J Pharmacol 1995;289:249-257. doi: 10.1016/0922-4106(95)90101-9
83. Espinoza TR, Wright DW: The role of progesterone in traumatic brain injury. J Head Trauma Rehabil 2011;26:497-499. doi: 10.1097/HTR.0b013e31823088fa
84. Zheng C, Gong J, Zang L, et al: Mechanism of progesterone in treatment of traumatic brain injury based on network pharmacology and molecular docking technology. Med Sci Monit 2022;28:e937564. doi: 10.12659/MSM.937564
85. Schumacher M, Sitruk-Ware R, De Nicola AF: Progesterone and progestins: neuroprotection and myelin repair. Curr Opin Pharmacol 2008;8:740-746. doi: 10.1016/j.coph.2008.10.002
86. Singh M, Su C: Progesterone, brain-derived neurotrophic factor and neuroprotection. Neuroscience 2013;239:84-91. doi: 10.1016/j.neuroscience.2012.09.056
87. Singh M, Krishnamoorthy VR, Kim S, et al: Brain-derived neuerotrophic factor and related mechanisms that mediate and influence progesterone-induced neuroprotection. Front Endocrinol 2024;15:1286066. doi: 10.3389/fendo.2024.1286066
88. Henderson VW: Progesterone and human cognition. Climacteric 2018;21:333-340. doi: 10.1080/13697137.2018.1476484
89. Sundström Poromaa I, Gingnell M: Menstrual cycle influence on cognitive function and emotion processing-from a reproductive perspective. Front Neurosci 2014;8:380. doi: 10.3389/fnins.2014.00380
90. Wang J, Chen A: High progesterone levels facilitate women's social information processing by optimizing attention allocation. Psychoneuroendocrinology 2020;122:104882. doi: 10.1016/j.psyneuen.2020.104882
91. Conde DM, Verdade RC, Valadares ALR, et al: Menopause and cognitive impairment: a narrative review of current knowledge. World J Psychiatry 2021;11:412-428. doi: 10.5498/wjp.v11.i8.412
92. Borozan S, Kamrul-Hasan ABM, Pappachan JM: Hormone replacement therapy for menopausal mood swings and sleep

quality: the current evidence. World J Psychiatry 2024;14:1605-1610. doi: 10.5498/wjp.v14.i10.1605
93. Stefaniak M, Dmoch-Gajzlerska E, Jankowska K, et al: Progesterone and its metabolites play a beneficial role in affect regulation in the female brain. Pharmaceuticals (Basel) 2023;16:520. doi: 10.3390/ph16040520
94. Paul SM, Pinna G, Guidotti A: Allopregnanolone: from molecular pathophysiology to therapeutics. A historical perspective. Neurobiol Stress 2020;12:100215. doi: 10.1016/j.ynstr.2020.100215
95. Voskuhl R: It is time to conduct phase 3 clinical trials of sex hormones in MS - yes. Mult Scler 2018;24:1413-1415. doi: 10.1177/1352458518768764
96. Harden CL, Pennell PB: Neuroendocrine considerations in the treatment of men and women with epilepsy. Lancet Neurol 2013;12:72-83. doi: 10.1016/s1474-4422(12)70239-9
97. Honjo H, Iwasa K, Kawata M, et al: Progestins and estrogens and Alzheimer's disease. J Steroid Biochem Mol Biol 2005;93:305-308. doi: 10.1016/j.jsbmb.2004.12.001
98. Partridge B, Rossmeisl JH Jr: Companion animal models of neurological disease. J Neurosci Methods 2020;331:108484. doi: 10.1016/j.jneumeth.2019.108484
99. De Nicola AF, Coronel F, Garay LI, et al: Therapeutic effects of progesterone in animal models of neurological disorders. CNS Neurol Disord Drug Targets 2013;12:1205-1218. doi: 10.2174/18715273113120012O
100. Grone BP, Baraban SC: Animal models in epilepsy research: legacies and new directions. Nat Neurosci 2015;18:339-343. doi: 10.1038/nn.3934
101. Van Meervenne SA, Volk HA, Matiasek K, et al: The influence of sex hormones on seizures in dogs and humans. Vet J 2014;201:15-20. doi: 10.1016/j.tvjl.2014.05.008
102. Van Meervenne SA, Volk HA, Van Ham LM: Association between estrus and onset of seizures in dogs with idiopathic epilepsy. J Vet Intern Med 2015;29(1):251-253. doi: 10.1111/jvim.12505
103. Freitas-de-Melo A, Banchero G, Hötzel MJ, et al: Progesterone administration reduces the behavioural and physiological responses of ewes to abrupt weaning of lambs. Animal 2013;7:1367-1373. doi: 10.1017/S1751731113000621
104. Lonergan P, Sánchez JM: Symposium review: progesterone effects on early embryo development in cattle. J Dairy Sci 2020;103:8698-8707. doi: 10.3168/jds.2020-18583
105. Spencer TE, Burghardt RC, Johnson GA, et al: Conceptus signals for establishment and maintenance of pregnancy. Anim Reprod Sci 2004;82-83:537-550. doi: 10.1016/j.anireprosci.2004.04.014
106. Pokharel K, Peippo J, Weldenegodguad M, et al: Gene expression profiling of corpus luteum reveals important insights about early pregnancy in domestic sheep. Genes 2020;11:415. doi: 10.3390/genes11040415
107. Hansen PJ: Regulation of immune cells in the uterus during pregnancy in ruminants. J Anim Sci 2007;85(13 Suppl):E30-E31. doi: 10.2527/jas.2006-487
108. Dicks LMT: Gut bacteria and neurotransmitters. Microorganisms 2022;10:1838. doi: 10.3390/microorganisms10091838
109. Ziętek M, Celewicz Z, Szczuko M: Short-chain fatty acids, maternal microbiota and metabolism in pregnancy. Nutrients 2021;13:1244. doi: 10.3390/nu13041244
110. Vuong HE, Pronovost GN, Williams DW, et al: The maternal microbiome modulates fetal neurodevelopment in mice. Nature 2020;586:281-286. doi: 10.1038/s41586-020-2745-3
111. Clarke G, Grenham S, Scully P, et al: The microbiome-gut-brain axis during early life regulates the hippocampal serotonergic system in a sex-dependent manner. Mol Psychiatry 2013;18:666-673. doi: 10.1038/mp.2012.77
112. Bonnin A, Levitt P: Fetal, maternal, and placental sources of serotonin and new implications for developmental programming of the brain. Neuroscience 2011;197:1-7. doi: 10.1016/j.neuroscience.2011.10.005
113. Sachis PN, Armstrong DL, Becker LE, et al: Myelination of the human vagus nerve from 24 weeks postconceptional age to adolescence. J Neuropathol Exp Neurol 1982;41:466-472. doi: 10.1097/00005072-198207000-00009
114. Dash S, Syed YA, Khan MR: Understanding the role of the gut microbiome in brain development and its association with neurodevelopmental psychiatric disorders. Front Cell Dev Biol 2022;10:880544. doi: 10.3389/fcell.2022.880544
115. Garzoni L, Faure C, Frasch MG: Fetal cholinergic anti-inflammatory pathway and necrotizing enterocolitis: the brain-gut connection begins in utero. Front Integr Neurosci 2013;7:57. doi: 10.3389/fnint.2013.00057
116. Nyangahu DD, Jaspan HB: Influence of maternal microbiota during pregnancy on infant immunity. Clin Exp Immunol 2019;198:47-56. doi: 10.1111/cei.13331
117. Nyangahu DD, Lennard KS, Brown BP, et al: Disruption of maternal gut microbiota during gestation alters offspring microbiota and immunity. Microbiome 2018;6:124. doi: 10.1186/s40168-018-0511-7
118. Joly A, Leulier F, De Vadder F: Microbial modulation of the development and physiology of the enteric nervous system. Trends Microbiol 2021;29(8):686-699. doi: 10.1016/j.tim.2020.11.007
119. Yang LL, Millischer V, Rodin S, et al: Enteric short-chain fatty acids promote proliferation of human neural progenitor cells. J Neurochem 2020;154:635-646. doi: 10.1111/jnc.14928
120. Rogers GB, Keating DJ, Young RL, et al: From gut dysbiosis to altered brain function and mental illness: mechanisms and pathways. Mol Psychiatry 2016;21:738-748. doi: 10.1038/mp.2016.50
121. Warner BB: The contribution of the gut microbiome to neurodevelopment and neuropsychiatric disorders. Pediatr Res 2019;85:216-224. doi: 10.1038/s41390-018-0191-9
122. Uystepruyst C, Coghe J, Dorts T, et al: Sternal recumbency or suspension by the hind legs immediately after delivery improves respiratory and metabolic adaptation to extra uterine life in newborn calves delivered by caesarean section. Vet Res 2002;33:709-724. doi: 10.1051/vetres:2002051
123. Uystepruyst C, Coghe J, Dorts T, et al: Effect of three resuscitation procedures on respiratory and metabolic adaptation to extra uterine life in newborn calves. Vet J 2002;163:30-44. doi: 10.1053/tvjl.2001.0633
124. Armstrong L, Caulkett N, Pearson JM, et al: Assessing the efficacy of ventilation of anesthetized neonatal calves using a laryngeal mask airway or mask resuscitator. Front Vet Sci 2018;5:292. doi: 10.3389/fvets.2018.00292

125. Di Gesù CM, Matz LM, Buffington SA: Diet-induced dysbiosis of the maternal gut microbiome in early life programming of neurodevelopmental disorders. Neurosci Res 2021;168:3-19. doi: 10.1016/j.neures.2021.05.003
126. Kim SW, Youk T, Kim J: Maternal and neonatal risk factors affecting the occurrence of neurodevelopmental disorders: a population-based nationwide study. Asia Pac J Public Health 2022; 34:199-205. doi: 10.1177/10105395211066383
127. Bleul U, Bircher B, Jud RS, et al: Respiratory and cardiovascular effects of doxapram and theophylline for the treatment of asphyxia in neonatal calves. Theriogenology 2010;73:612-619. doi: 10.1016/j.theriogenology.2009.10.017
128. Bleul U, Bylang T: Effects of doxapram, prethcamide and lobeline on spirometric, blood gas and acid-base variables in healthy new-born calves. Vet J 2012;194:240-246. doi: 10.1016/j.tvjl.2012.04.007
129. Constable PD, Trefz FM, Sen I, et al: Intravenous and oral fluid therapy in neonatal calves with diarrhea or sepsis and in adult cattle. Front Vet Sci 2021;7:603358. doi: 10.3389/fvets.2020.603358
130. Shankaran S, Laptook AR, Ehrenkranz RA, et al: Whole-body hypothermia for neonates with hypoxic-ischemic encephalopathy. N Engl J Med 2005;353:1574-1584. doi: 10.1056/NEJMcps050929
131. Godden SM, Lombard JE, Woolums AR: Colostrum management for dairy calves. Vet Clin North Am Food Anim Pract 2019;35: 535-556. doi: 10.1016/j.cvfa.2019.07.005
132. Abdelfattah E, Fausak E, Maier G: Failure of passive immune transfer in neonatal beef calves: A scoping review. Animals (Basel) 2025;15:2072. doi: 10.3390/ani15142072