

Review

The Basics of Clinical Nutrition for Compromised Ruminants—A Narrative Review

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Simple Summary

Guidelines for clinical nutrition in compromised ruminants are lacking. Clinical nutrition plays a critical role in the recovery of compromised ruminants. Yet, the lack of species-specific data for various compromised states in ruminants makes informed guidelines difficult. The essential role of the forestomaches in ruminant utilization of nutrients demands prioritizing clinical nutrition interventions that deliver the diet through the mouth or directly into the rumen. Softened, shorter and highly palatable diet may be preferred. Total intravenous feeding should be restricted to very expensive or neonatal livestock only, and then it should be for as short a period as possible.

Abstract

A significant gap in specific nutritional guidelines for ruminants with compromised health exists. Due to their unique anatomy, physiology, and metabolic processes, further research is needed to establish accurate, evidence-based recommendations tailored to these animals. This review highlights the critical role of clinical ruminant nutrition and provides provisional recommendations based on studies in other species (e.g., changes in nutrient requirements in different morbidities available for humans and less for companion animals). These suggestions should be interpreted cautiously until more definitive, species-specific data become available. The review includes the foundational principles of clinical nutrition in ruminants, with particular emphasis on the pathophysiology of nutrient utilization. It explores the roles of energy, protein, vitamins, and minerals during illness or injury and discusses how these nutrients can be strategically applied in clinical interventions. Considerations for designing diets for compromised ruminants are also addressed, considering both physiological needs and the challenges posed by illness and injury states. Practical aspects of diet delivery during treatment are examined, including the indications, benefits, limitations, and potential side effects of route of feeding. Clinical nutrition can be administered orally, enterally (including rumen delivery) or parenteral, depending on the localization of the pathology and the integrity of the alimentary tract. Nutrients should be provided based on livestock requirements and pathophysiology and severity of the primary morbidity. Oral or ruminal provision of diet should be prioritized to maintain rumen functionality. Additionally, a list of pharmaconutrients with potential clinical applications in ruminant medicine is presented to encourage future research and integration into veterinary practice. The success of clinical nutritional interventions can be measured by improvements in appetite, behavior, and health of the compromised ruminant.



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1. Introduction

The important roles of nutrition in the growth, health, and wellbeing of ruminants have been extensively explored [1–3]. Where essential nutrients for maintenance are deficient, the result has been immune system disruption with impaired production of immunoglobulins, coupled with compromised cellular and humoral responses [4]. The range of disorders directly or indirectly associated with imbalanced nutrition is extensive, including hypocalcemia [5] and ketosis in dairy cattle [6], pregnancy toxemia in beef cattle [7] and small ruminants, and rumen acidosis [8] in all ruminants. These disorders can be prevented through nutritional management that involves providing a well-balanced diet in appropriate quantities tailored to the specific production stage. However, even with a balanced diet and favorable living conditions, ruminants may still experience health issues [9,10]. Indeed, nutritional status of both humans and animals significantly impacts the progression of infectious diseases [4]. Malnutrition not only delays the recovery and healing process but also disrupts immune function and overall health [11]. Therefore, in cases of illness, prompt detection and intervention are crucial. In the past, ruminant nutrition was not prioritized as a therapeutic element, but it is now recognized as an important component in disease management [11].

Clinical nutrition, a strategy employed in human medicine, utilizes pharmacologically active compounds including, but not limited to, amino acids, antioxidants, buffers, and omega-3 fatty acids to modulate immune responses, facilitate recovery, and promote tissue repair [4,12–15]. Clinical nutrition, discussed in this paper, focuses on the nutritional management of compromised, including ill and injured, ruminants. Clinical nutrition, also referred to as therapeutic nutrition by some [12,16,17], is an area that has received limited attention in ruminants (e.g., [11,18–20]), resulting in a lack of information regarding its benefits and appropriate utilization. Despite many similarities in anatomy and physiology of various species, some variations do exist in anatomy, metabolic response to illness/injury, physiology, and dietary requirements between the species. The uniqueness in anatomy and physiology of ruminants [21] requires differences in the approaches to clinical nutrition interventions compared to humans and other monogastric animals, although the basic principles are the same.

The main objective of clinical nutrition of ruminants is to provide a balanced and healthy diet to compromised livestock, to minimize the use of their body reserves, and facilitate their recovery, ultimately leading to a return to productivity. Similarly to human clinical nutrition, specific molecules can be utilized to modify metabolism and expedite the recovery process in ruminants [22–24].

When formulating a diet for an ill or injured (compromised hereafter) ruminant, the nutritionist/practitioner should consider the clinical situation, nutrition principles applicable to healthy livestock, patient response, and all available information about the diagnosed disorder. A complete lack of research on the nutritional requirements of compromised ruminants (e.g., indigestion, left displaced abomasum) and minimal information regarding approaches to the delivery of clinical nutrition intervention are evident. Without evidence-based information on ruminant clinical nutrition, the need for involvement of an experienced nutritionist to formulate a clinical diet is highlighted. Much of the available data on clinical nutrition comes from experiments with companion animals, horses, and humans. The purpose of this review is to highlight the importance of

ruminant clinical nutrition, its role in addressing health issues, the technologies involved, and the areas where knowledge is currently lacking. Many of the recommendations in this review are extrapolated from evidence in other species, interjected with the experience of the authors. Therefore, many of the recommendations in this paper should be regarded as subjective until definitive data on ruminants become available. We hope that this review will stimulate more research and publications in this important area.

In this paper, we discuss the background of ruminant clinical nutrition, pathophysiology of nutrient utilization in compromised ruminants and applicability to the clinical nutrition intervention, the options for delivery of the diet during clinical nutrition interventions, and, finally, pharmaconutrition for compromised ruminants.

2. The Background of Ruminant Clinical Nutrition

Compromised ruminants often experience a decrease in appetite and frequently encounter alterations in their alimentary functions. In populations of ruminants, compromised ruminants are typically housed together with their healthy herd mates and receive the same diet. In traditional approaches, they are exposed only to pharmaceutical management of their morbidity. Different disorders can result in various modulations of metabolism and physiology [5,6,25]. This is discussed in the next section dealing with the pathophysiology of the utilization of nutrients in compromised ruminants. Therefore, to promote faster recovery, each compromised ruminant requires a diet that specifically addresses the physical and physiological state of the livestock individual. The field of ruminant clinical nutrition focuses on addressing the dietary needs of compromised ruminants and is the subject covered in this paper.

The successful application of clinical nutrition requires an accurate diagnosis and an understanding of the alterations that have occurred because of it. Inappropriate utilization of clinical nutrition may result in an increasing severity of the morbidity, side effects and, death, if the diagnosis is neglected. The clinical nutrition intervention should be in addition to ensuring the provision of basic nutrient requirements of the compromised ruminant. Depending on animal welfare considerations, diagnosis, prognosis, and the value of the affected ruminant, it is important to evaluate whether the intervention is economically feasible.

Treatment of the primary morbidity should always be the top priority [26,27], often supplemented with supportive treatment and appropriate nursing care (Figure 1). Clinical nutrition can be a component of the primary treatment, such as correcting specific deficiencies or excesses, or more commonly, part of the supportive treatment, such as balancing acid–base status, electrolytes, and fluids. For example, in undifferentiated neonatal diarrhea, the problems needing correction include dehydration, electrolyte imbalance, hypoglycemia, and metabolic acidosis [28].

The goal of clinical nutrition should be to optimize body and cellular functionality, immunocompetence, treatment outcomes and/or wound healing; minimize catabolic processes, co-morbidity, metabolic alterations, mortality, oxidative damage, and weight loss; and optimize the intake of nutrients [26–28]. For this purpose, in the initial stages, acid–base, electrolyte, fluid and metabolic alterations should be corrected, and then life and wellbeing should be supported. Ultimately, the objectives of ruminant clinical nutrition are to reduce the duration and severity of morbidity, and prevent mortality [8]. In short, ruminant clinical nutrition should restore the function of the alimentary system and promote the return of the patient to health and productivity, whilst minimizing impacts on animal welfare.

Step 1	Determine if clinical nutritional intervention is required	Nutritional assessment (Signalment, Health interview, Examination of the environment, Clinical examination from distance / from close)	Length of anorexia; Physiologic status; Type of morbidity
Step 2	Determine the mode of clinical nutritional intervention	Available equipment, nutrients, time; Collected data; Facilities; Functionality and Integrity of the alimentary system; Finances	Functionality and integrity of the alimentary system; Patient's signalment and worth; Finances
Step 3	Correct primary morbidity	Management of the primary morbidity (May be the clinical nutritional intervention or it may be only a supportive strategy)	Morbidity-dependent
Step 4	Establish nutrient requirements	Energy; Protein; $\pm$ Acid-base, Electrolyte, Fluid imbalances, $\pm$ Pharmaconutrients	Body condition score; Length of anorexia; Morbidity; Physiologic status; Signalment
Step 5	Design the diet	Ingredients; Type of clinical nutritional intervention: Complete / total diet (oral / enteral / combined, less likely parenteral); Partial diet (oral / enteral / combined, less likely parenteral); Supplemental diet (enteral / parenteral / combined); Hyperalimentation (enteral, parenteral, combined)	
Step 6	Implement clinical nutritional intervention	Plan: Frequency / Meal size / Mode of delivery of the diet; Monitoring and re-adjustments; $\pm$ Need of transfaunation; Slit responsibilities	Body condition score; Functionality and integrity of the alimentary system; Length of anorexia; Morbidity; Physiologic status; Signalment
Step 7	Monitor clinical nutritional intervention	Imbalances: Acid-base, Electrolyte, Energy, Fluids, Protein; Rumen function and integrity; Signs of refeeding syndrome	SMART goals (Specific, Measurable, Achievable, Relevant, Time-bound) and triggers for timely intervention

Figure 1. Steps that need to be taken for any patient that is a potential candidate for a clinical nutrition intervention.

3. Pathophysiology of Nutrient Utilization in Compromised Ruminants and Applicability to the Clinical Nutrition Intervention

While inadequate nutrient intake can predispose to disease, short-term nutritional imbalances alone are not sufficient to cause it. However, any nutritional imbalance can create a favorable environment for secondary infections or other disorders to develop [4,29–32]. Nutritional deficiency can also increase the severity of infectious disorders [11] and delay wound healing [27]. Generalized malnourishment and selective nutrient imbalances can lead to immunological impairment, resulting in an increased incidence and severity of illness and injury. This, in turn, results in increased morbidity and mortality rates, often coupled with more severe morbidities. Additionally, there would be reduced feed conversion efficiency and growth rates [9,26], increased treatment costs, and risk of carcass condemnation at slaughter. Furthermore, the depressed immunological response may result in vaccine failures, further increasing the costs of imbalanced nutrition [11,33]. Decreased intake by compromised ruminants results in a higher environmental impact by contamination of the environment with antimicrobials and other medication, and a higher impact on greenhouse gas emissions [34]. Finally, consumer expectations of buying a product derived from healthy ruminants should not be ignored [9]. At the population level, subclinical health conditions are probably costlier but are often unrealized by the client. However, further discussion of subclinical illness and injury is beyond the scope of this review.

3.1. Pathophysiology of Perturbed Utilization of Protein and Energy

To spare available nutrient reserves, in healthy ruminants, any starvation in otherwise healthy livestock results in a reduction in metabolic rate [26,27]. Unwell ruminants with chronic or severe illness or injury often experience a depressed appetite [26,27,33], but lack an adaptive response to starvation [33,35]. The depressed intake may be a direct

result of illness or injury (e.g., inappetence due to depressed activity of the hypothalamic appetite center) or may result from an inability to access (e.g., lameness, paresis/paralysis, or recumbency) or ingest the offered diet (e.g., inability to prehend, masticate and/or swallow) [36,37]. Therefore, as part of the clinical nutrition information, the diet may need to be manipulated to increase access (e.g., portable feeding bunks), energy-density (e.g., fats, grains) [26,32] or intake (e.g., palatability) [26,28,32]. Alternatively, to ensure sufficient intake of nutrients, hyperalimentation and medication may be required.

In compromised ruminants, there is an increase in the mobilization in muscle protein and adipose tissues, and increased metabolic rate $\pm$ body temperature, in addition to the oxidative damage to the cells and repair processes [26,27,38]. These changes are associated with a state of protein hypercatabolism. Additionally, lipids [26] and/or rapidly developing insulin resistance [26,27] and/or insulin release [26] are common in severe illness or injury. These changes occur due to the individual or combined activity of circulating hormones, inflammatory cytokines, and neurotransmitters [26,27,39]. In compromised ruminants with a partially maintained appetite, the provision of diet will minimize catabolic processes associated with starvation. Yet, catabolic processes of chronic disease/inflammation will persist, particularly protein catabolism [26]. Therefore, clinical nutrition intervention should ensure energy and protein requirements of the compromised ruminant are met. Due to the depressed feed intake, meals may need to be smaller and more concentrated [32].

Additionally, during illness or injury, available amino acids are partitioned towards the buildup of acute phase proteins (e.g., ceruloplasmin, fibrinogen) and support of the immune response rather than growth or production [26,33]. During the process of protein hypercatabolism, some of the non-essential amino acids become conditionally essential (e.g., arginine, glutamine). Muscle protein is a major source of glutamine during severe illness, but the liver is the major consumer [30,40]. Therefore, as glutamine is essential for fully functional cellular immunity, a suboptimal response may result due to glutamine deprivation of the immune cells [30,40,41]. Additionally, glutamine has an essential role in brain, liver and kidney function, nitrogen metabolism, and the feeding and resorptive function of the intestines [40]. Arginine should be supplemented to compromised ruminants as it facilitates wound healing, improves nitrogen balance, sometimes with production of dangerous amounts of nitrous oxide, and, finally, it stimulates immunocompetence [38]. Therefore, clinical nutrition intervention should minimize protein catabolism by the provision of essential and conditionally essential amino acids [7,38,41].

To meet the body's maintenance demands under conditions of inappetence, muscle and fat catabolism will occur. This is further complicated by increased metabolic requirements because of fever, general malaise, and/or tissue repair [26]. Catabolism is partially increased due to the release of insulin-like compounds by inflammatory cells. Additionally, under the influence of some inflammatory cytokines, insulin may also be released, which will further enhance the catabolism of carbohydrates [26]. Catabolic processes may contribute to the accumulation of triglycerides in the liver and the production of reactive oxygen species [38]. Prolonged catabolism may lead to conditions such as hepatic lipidosis, ketosis and weight loss. Furthermore, behavior is affected, partly due to an insufficient glucose supply to the brain. Therefore, clinical nutritional intervention should provide a source of easily accessible energy (e.g., glucose). Due to alterations in glucose utilization and a reduced hormonal influence on lipid metabolism, increased fat levels in the diet (up to a maximum of around 5% of dry matter intake) may be beneficial. However, higher fat content between 6 and 8% may result in depressed rumen fermentation and fiber degradation, and should be avoided [42]. Furthermore, high levels of unsaturated fats can react with available calcium to form insoluble calcium soaps in the rumen, reducing calcium availability. These compounds are stable at rumen pH ranging from 5.5 to 6.5, but can

dissociate as the rumen pH decreases, and the free fatty acids may impact the microbial function in the rumen with a detrimental effect on ruminant health [43,44]. It is worth mentioning that ruminants have a low threshold of blood glucose concentrations before the excess is eliminated in the urine, potentially resulting in a glucose-based energy supply limitation, even if provided by the parenteral mode.

3.2. Pathophysiology of Perturbed Utilization of Minerals, Vitamins and Water

Some minerals change their blood concentrations or available forms (e.g., decreases in serum concentrations of iron and zinc, and an increase in copper) [26]. These changes are mediated mainly through various inflammatory cytokines. However, routine inclusion of various minerals in the clinical nutrition intervention cannot be recommended, and supplementation should be based on laboratory confirmation of actual needs. Additionally, to improve immunocompetence, clinical nutrition intervention should provide immunomodulators (e.g., amino acids, minerals, plant extracts, and vitamins) [41].

A major portion of the water-soluble vitamins (particularly the B-complex) are produced by the rumen microbes. Vitamins of the B-complex are particularly important due to their essential role as coenzymes in the Krebs tricarboxylic acid cycle, critical for energy metabolism and cellular function. Vitamins of the B-complex have also roles in the function and protection of the neurologic tissues (B₁, B₃, B₆, and B₉ [45–48]), growth of rumen microbes (B₁ and B₃ [48]), hematopoiesis (B₁₂ [48]), hoof health (B₇ [46–49]), immunity (B₂ [46,50]), integrity and inflammation of the alimentary epithelium (B₁ [45–47]), liver health (B₉ and B₁₂ [48,51]), and reproductive performance (B₉ and B₁₂ [48,52]). Due to an increased metabolic rate in compromised ruminants, B-complex vitamins are utilized in greater amounts. As rumen microbes are likely compromised in ruminant patients at the initiation of the clinical intervention, the need for supplementation with water-soluble vitamins should be considered [26,32,41,48,53]. Although it is probably more important in non-ruminants, Vitamin C can become important during periods of stress, such as illness, transportation, or changes in the environment. This vitamin can also act as an antioxidant and is involved in collagen synthesis for tissue repair and wound healing of injuries [54].

Lipid-soluble vitamins are stored in adipose tissues, and deficiencies can be observed when ruminants experience anorexia or otherwise do not consume enough nutrients, leading to low BCS and potential deficiencies over time. In ruminants with low BCS or those with high metabolic demands, supplementation with lipid-soluble vitamins may also need to be considered [32]. Vitamin A is essential for the repair of epithelial tissues in ruminants [55–58]. Vitamin D is crucial in cases of fractures or bone-related injuries, as it facilitates the absorption of calcium and phosphorus required for bone healing [56,58]. Vitamin E, known for its antioxidant properties, can assist in muscle recovery and help reduce the oxidative stress in damaged tissues, thereby promoting faster healing and reducing inflammation [55,56,58]. It can reduce the incidence of mastitis, metritis and retained placenta [56,58,59]. In cases of wounds, vitamin K plays a vital role in blood clotting for the proper healing of wounds of injuries or post-surgical procedures [55,56,60]. While rumen bacteria generally produce enough vitamin K, ruminants can become vitamin K deficient when consuming feeds containing dicumarol, a vitamin K antagonist, found in moldy sweet clover hay or silage [56,58].

The interactions of minerals and vitamins are crucial to be considered for compromised ruminants, as cobalt is essential for the biosynthesis of vitamin B₁₂ by rumen microbes, and vitamin D is crucial for the absorption of dietary calcium, particularly important during pregnancy and early lactation [61,62].

Chronically or severely compromised ruminants suffer not only from a depressed appetite but also often from depressed water intake [26]. The depressed water intake,

often coupled with a greater water loss, leads to dehydration and various electrolyte imbalances [26,63]. Dehydration in mature ruminants may occur due to drying off the rumen contents or excessive fluid loss (e.g., diarrhea, hemorrhage, polyuria, sialorrhea, and third space loss) [18,36,63]. Hence, clinical nutrition intervention should consider fluid and electrolyte replacement and optimization therapy. Indeed, electrolyte and fluid therapy may also be indicated when there is a need for increased diuresis (e.g., acute renal injury or various toxicoses) [63–65].

3.3. Pathophysiology of Impaired Alimentary Function and Integrity

In ruminants, a large proportion of alimentary system functionality depends on rumen fermentation, followed by abomasal and intestinal digestion, and the smallest fraction is based on hindgut fermentation. Functionality and integrity of the alimentary system, and particularly the intestinal mucosa, are highly dependent on the delivery of nutrients and oxygen to the mucosa [7,41,66]. These are both dependent on the continuous presence of fresh ingesta [7]. Illness can impair the function of the alimentary system (e.g., malabsorption and maldigestion syndromes) [7] and so increase the alimentary system nutrient requirements. Malabsorption and maldigestion syndromes result in decreased feed utilization [7,9,32]. Unfortunately, mucosal damage in compromised ruminants may require prolonged periods to recover, leading to increased costs associated with raising such ruminants [7,66].

One of the important functions of the intestinal mucosa is to be a barrier to the absorption of unwanted intestinal compounds (e.g., D-Lactate and various bacterial toxins, such as lipopolysaccharide; LPS) and/or pathogen incursion [29,41,53,63,67]. This is achieved through the tight junctions between enterocytes [63,68]. Additional contributions to the prevention of infection are achieved through gut-associated lymphoid tissue [41]. The lack of sufficient feed intake deprives the intestinal mucosa of nutrients, followed by alterations in absorptive capacity and vascular supply, damage to intestinal villi $\pm$ crypts, and loss of barrier protective function, with increased paracellular transport [53,63,68]. Any alteration in hemodynamic stability (e.g., shock) may additionally affect oxygenation of the gut mucosa and, due to splanchnic ischemia, may result in reperfusion injury [41]. Absorption of unwanted intestinal compounds and/or bacterial toxins results in changes in behavior, endotoxemia, and/or inflammatory reactions [68] and translocation of intestinal pathogens results in various infections. Therefore, clinical nutrition intervention should provide fresh ingesta as soon as it is safe to do.

Increased metabolic rate with hyper-catabolism, coupled with a decreased diet intake, often results in overloading of the liver. The excess triglycerides in the liver increase the risk of hepatic lipidosis and other hepatopathies [6]. Therefore, clinical nutrition intervention may need to consider the hepatoprotective activity of some nutrients (e.g., branched and sulfur-containing amino acids, water-soluble and E vitamins).

Pathophysiology of Forestomach Impaired Function and Integrity

When discussing the pathophysiology of nutrient utilization in ruminants, an important factor to consider is forestomach function in regard to alterations in microbes and motility. Ruminants are fed by the amazing, commensal, and symbiotic microbes that produce a large number of nutrients for their host, including microbial protein, vitamins, and volatile fatty acids [66,69,70]. In healthy ruminants, forestomach fermentation provides some 80% of fiber digestion and supplies around 60% of the total amino acids [71]. As such, the rumen microbes require a constant supply of fresh nutrients [69,72,73] and meeting the increased specific dietary requirements in compromised ruminants is crucial for rapid recovery [4]. Yet, ruminants recover more slowly from starvation compared to

monogastric animals, partly due to the requirements for forestomach microbes to adjust to the diet [74,75]. Rumen transfaunation may speed up the process of recovery [7,20].

The lack of dietary intake slows down and may even cease forestomach motility. The microbes change (dysbiosis) by loss of active protozoa and alterations in proportions of live microbes, slowly resulting in the death of a large proportion of all microbes [7,18,20]. These changes result in various forms of indigestion and several metabolic disorders (e.g., displaced abomasum) [73]. As rumen pH decreases, the growth of Gram-negative bacteria is facilitated, ultimately resulting in the production of excessive amounts of LPS [71,76]. As LPS is absorbed, it results in endotoxemia $\pm$ inflammation [53]. However, in ruminants fed on high-concentrate diets, LPS may also be produced by hindgut fermentation [76]. The ultimate result of the nutritional imbalance related to the forestomach is indigestion [7,26,76].

Additionally, due to systemic dehydration or the buildup of osmotically active compounds in the rumen, the existing rumen content dries out [18,77]. Therefore, clinical nutrition intervention should initially provide fluids into the reticulorumen $\pm$ transfaunation. Paradoxically, as these compounds pass into the intestines, diarrhea may be the only sign of the build-up of osmotically active compounds in the rumen [77].

The next step should be the provision of feeds to the reticulorumen. The feed provided must contain sufficient effective fiber (e.g., in cattle, structural carbohydrates longer than 2.5 cm), which means a mainly forage-based diet. The roles of the fiber include, but are not limited to, maintenance of the health and functionality of forestomachs (e.g., stimulation of mixing of the rumen contents), production of milk fat in lactating females (by production of its precursor volatile fatty acids, e.g., acetate, butyrate); and stimulation of chewing, rumination, and salivation, indirectly controlling rumen pH [36,72]. However, in ruminants with high metabolic demands (e.g., young growing individuals, lactating and heavily pregnant females), forages may not be able to provide enough energy and protein.

3.4. Pre-Ruminant Stage

Neonates are more prone to illness than are mature ruminants [26] due to the close contact with other neonates and/or mature ruminants, higher exposure to unfamiliar pathogens, lower immunocompetence, and a very inquisitive nature. Body energy and protein reserves in ruminant neonates are significantly smaller compared to mature ruminants. Additionally, their metabolic rate is higher than in mature ruminants. Therefore, energy is often seriously lacking in ill and injured ruminant neonates. Crystalloid-based oral/enteral or parenteral fluids used initially in the clinical nutrition intervention should contain insufficient energy and protein to satisfy their requirements [28,76]. Therefore, clinical nutrition intervention should consider a suitable approach to replenish and maintain energy and protein to meet their requirements.

Any serious illness may lead to loss of the suckle reflex, and oral administration of fluids may inadvertently result in aspiration pneumonia. Additionally, any serious illness or injury may result in a non-functional alimentary system, and force feeding the ruminant with fluids and/or milk may result in ruminal deposition, bloat and rumen putrefaction [26].

The body of the neonate contains a much larger proportion of water than an adult does, and any disorder may quickly result in dehydration and electrolyte imbalances [26,63,78]. Enteric morbidity has the highest incidence in neonatal ruminants. Notably, due to the replacement of intracellular potassium with hydrogen ions, with potassium being expelled extracellularly, neonatal ruminants quickly develop apparent hyperkalemia with real hypokalemia. Neonatal ruminants rapidly develop metabolic acidosis, particularly with any diarrheic disorder [26,78,79]. Therefore, clinical nutrition intervention should consider

restoration and optimization of acid–base, electrolyte, and fluid balances. Fluid therapy should ensure that at least chloride, potassium and sodium losses are replaced.

Oral and enteral feeding are necessary for neonates for the proper development of the alimentary system and the gut microbiota [80,81]. In humans, in addition to suboptimal gut microbiota, the developments of the intestines, pancreas, and stomach have been delayed and incomplete in neonates on an exclusively parenteral diet [82,83]. This has been, in part, explained as due to decreased secretion of gut hormones that stimulate the development of the alimentary system [82].

3.5. Summary of Pathophysiologic Alterations in Ruminants Important to Clinical Nutrition Intervention

In summary, based on pathophysiologic alterations in body functions and diagnosis, the clinical nutrition intervention should consider any or all of water, carbohydrates (both structural and non-structural), proteins, lipids, electrolytes, minerals and vitamins [10,26,32] (Table 1). Additionally, clinical nutrition intervention should enhance/support the ruminant's immune system [3,33,84].

Table 1. The relation between alteration in body function, syndromes indicative of the alteration, and common causes. Italicized text is directly associated with the nutritional management of ruminants.

Alteration in	Common Causes	
	Syndromes	Diagnoses
Appetite	Alimentary syndromes	Actinobacillosis
	Generalized malaise	Actinomycosis
	Heat stress	Any generalized morbidity
	Neurologic syndromes	Cheilitis
	Severe fever/inflammation/pain/sepsis/toxemia	Foreign body
	<i>Insufficient Diet delivery/preparation/Nutrient supply</i>	Glossitis
	<i>Offensive diet smell/taste</i>	Mandibular Fracture
	<i>Poor diet quality/spoilage</i>	Sinusitis
	<i>Sudden diet change/Unfamiliar diet</i>	Stomatitis
Prehension	Congenital abnormalities	Actinobacillosis
	Cranial nerve dysfunction	Cheilitis
	Lameness	Foreign body
	Localized pain	Glossitis
	Oral trauma	Mandibular Fracture
	Stomatitis	Sinusitis
	Tongue disorders	Stomatitis
	<i>Offensive diet smell/taste</i>	
	<i>Poor diet quality</i>	
Mastication	<i>Presence of corrosive compounds</i>	
	Congenital abnormalities	Actinobacillosis
	Cranial nerve dysfunction	Actinomycosis
	Dental problems	Balling gun injury
	Localized pain	Botulism
	Oral trauma	Foreign body
	Pharyngeal abscessation/cellulitis/trauma	Lead/Salt/Sulfur/Water toxicosis
	Quidding	Listeriosis
	Stomatitis	Mandibular fracture
	Tongue disorders	Tetanus
	<i>Offensive diet smell/taste</i>	
	<i>Poor diet quality</i>	
	<i>Presence of corrosive compounds</i>	

Table 1. Cont.

Alteration in	Common Causes	
	Syndromes	Diagnoses
Swallowing	Congenital abnormalities Cranial nerve dysfunction Dysphagia Neoplasia Odynophagia Oral trauma Esophageal abscessation/cellulitis/diverticulum/neoplasia/trauma Pharyngeal abscessation/cellulitis/trauma Stomatitis Tongue disorders Presence of corrosive compounds	Balling gun injury Botulism Choke Foreign body Goiter Hypocalcemia Lead/Sulfur toxicosis Megaesophagus Rabies
Rumen fermentation	Alimentary syndromes Poor diet quality/spoilage Sudden diet change	Imbalanced diet/Indigestion Rumen acidosis (less likely alkalosis)
Eructation	Alimentary syndromes Congenital abnormalities Neurologic syndromes	Bloat Choke Foreign body
Rumination	Alimentary syndromes Congenital abnormalities Neurologic syndromes	Rumen acidosis (less likely alkalosis)/impaction Rumenitis Traumatic reticulo-peritonitis Vagus indigestion
Chime passage	Alimentary syndromes Congenital abnormalities Neoplasia Neurologic syndromes	Foreign body Herniation Omasal impaction Obstructive disorders Use of laxatives Vagus indigestion
Digestion	Alimentary syndromes Congenital abnormalities Maldigestion syndrome (rare in ruminants)	Abomasal displacement/impaction/ulceration Abomasitis Cholestasis Enteritis
Absorption	Alimentary syndromes Iatrogenic interventions Diarrhea Malabsorption syndrome	Parasitic gastro-enteritis Use of binders in diet
Utilization	Alimentary/Hepatic/Urinary loss Alimentary/Endocrine syndromes Cachectic state Chronic morbidity Heat stress Neoplasia Parasitism Severe morbidity (fever/inflammation/pain/sepsis/toxemia) Toxicities resulting in the prevention of nutrient transport and metabolism Malnutrition	Amyloidosis Cholestasis Congestive heart failure Endocrinopathy Enteritis/Enteropathy Hepatitis/Hepatopathy Nephritis/Nephropathy

4. Establishing the Need for Clinical Nutrition Intervention in Compromised Ruminants

The need for clinical nutrition intervention should be established by considering several factors. These may include, but are not limited to, ability to assess the nutritional status of the patient and the need of clinical nutrition intervention, capacity to carry out the entire clinical nutrition intervention while maintaining the minimum animal welfare standards, and patient wellbeing not being further compromised by the clinical nutrition intervention, the economic value of the patient, and the diagnosis and prognosis for that particular patient [32,85,86].

The volume of the alimentary system in ruminants is sufficient to provide some cushioning effect regarding the demand of regular diet intake [7]. Mature ruminants that voluntarily consume at least 80% of their dietary requirements do not require any prompt intervention and can go without food for 36 to 98 h [7,26]. However, as indicated below, this will depend on the type of morbidity as in certain conditions, due to severe changes in body physiology and metabolism, clinical nutrition intervention may be required sooner.

- Cachectic or debilitated ruminants (immediately);
- Growing ruminants (particularly during the exponential growth phase) when anorexic > 24–36 h;
- High-producing lactating cows expend significant amounts of energy to produce milk. A failure to consume sufficient food to meet the demands of milk production will result in a loss of substantial amounts of muscle and fat mass;
- Lactating females when anorexic for >12–24 h;
- Mature ruminants in average body condition scores (BCS; 3–4/5) being anorexic for a maximum of 2–3 days;
- Neonates, due to their limited energy reserves, can only go without food for about 12 h [22,59];
- Pregnant females in the last half of pregnancy, and particularly in the last trimester;
- Ruminants from extensively managed enterprises with irregular supervision should always be considered anorexic for a few days before detection;
- Ruminants with a high BCS ($\geq 4/5$), due to the risk of hepatic lipidosis;
- Ruminants with a low body condition score (BCS; $\leq 1.5/5$) and prolonged malnutrition often have an unstable microbiota, underdeveloped rumen papillae, and increased requirements for specific vitamins, minerals, and amino acids;
- Ruminants that have undergone surgery experience high stress levels and may experience pain in the days following the procedure, which can affect their food intake;
- Ruminants with pre-existing metabolic derangements that would worsen with further anorexia;
- Severely immunocompromised ruminants;
- Specific morbidities and metabolic disorders, such as diarrhea.

Assessment of the need for clinical nutrition intervention in compromised ruminants is yet to be prepared.

5. Considerations in Designing a Clinical Diet for Compromised Ruminants

Once the necessity of clinical nutrition intervention is established, other steps to consider include the nutritional requirements of the compromised ruminant. Additional aspects to consider include the age of the patient, body condition score, body weight, degree of inappetence, duration of anorexia, growth rate, level of activity, locomotion score, phase of production, and reproductive status.

5.1. Nutritional Requirements

The first step in preparing a plan for clinical nutrition intervention is to calculate the nutritional requirements of the compromised ruminant, recognizing that these requirements will differ from those of a healthy livestock individual. In cases of illness, production will decrease, or cease altogether, while the immune system responds with heightened activity. The changes in nutrient requirements vary depending on the type and severity of the disorder [4]. When discussing ruminants, rumen microbes play a crucial role in the recovery process and must be given special consideration in ruminant clinical nutrition [20].

5.1.1. Energy Requirements

In horses, compromised individuals have been estimated to need only their resting energy requirement (RER) [87]. The authors of this paper believe that the same would apply to compromised ruminants, and the net energy for maintenance (NEm) should be known. A rule of thumb would be not to exceed 2–3% of BW in dry matter intake per day. This guideline should still consider the physiological, productive and reproductive status of the ruminant as discussed in the section above (e.g., a lactating dairy cow should be provided enough to support her lactation).

Adding fat to the diet, within the limits that maintain rumen function at a maximum of 5–7% of the total diet dry matter, can increase the energy density in the diet and make it easier for the ruminant to meet its energy requirements [88–90]. It is important to note that an ill ruminant reduces its demand for energy and nutrients for growth, production, and activity, but increases the energy requirements for the activated immune system and associated metabolic changes [27]. These requirements will vary depending on the stage and type of disorder. It is possible that the energy demands of a compromised ruminant that was previously in production or growth remain the same due to the increased energy demands of the immune system and metabolic changes offsetting the lower production demands. However, clinical diet specifications may be underestimated when there are reductions in alimentary absorption capability and excessive excretion of nutrients via urine or feces [27]. Changes in the physical characteristics of the diet, sometimes being part of the clinical nutrition intervention, can also alter the rate of passage through the alimentary system, affecting the absorption rate. Additionally, the metabolic rate is elevated by 5–13% per degree Centigrade of fever [27]. Following immune activation, energy requirements of leukocytes increase 2–3-fold per cell [11]. Most of what is known about clinical nutrition comes from studies on humans, horses, and pets, and based on their information, some alterations in ruminant nutritional requirements are suggested, based on NEm as follows [11,27,91–93]:

- 10% increase following elective surgery;
- 10–30% increase during heat stress/rumen failure;
- 10–30% increase in acute liver failure;
- 10–40% increase following parenteral loss of nutrients (e.g., nephropathy);
- 10–50% increase following enteral loss of nutrients (e.g., intestinal resection, severe parasitic gastroenteritis);
- 20–50% increase following bone fracture/major trauma;
- 30–50% increase following clostridial myositis/severe muscle trauma;
- 30–60% increase following major infection/sepsis/toxemia (approximately 10–13% increase for each 1 °C decrease/increase in body temperature);
- 40% increase following major peritonitis;
- 50–110% increase following major burns.

5.1.2. Protein Requirements

In mature ruminants with a normal appetite when not having a high demand for protein, requirements are about 1–3 g/kg BW [32,94]. In neonates, protein requirements have been estimated at 2–4 g/kg BW [26]. These requirements are probably increased in compromised ruminants. Highly digestible proteins (e.g., brewers'/distillers' grains, canola/soybean meal) are preferred [28], and should be included in concentrations that do not cause alimentary disturbances. Some specific amino acids that may need to be considered are arginine, branched amino acids (BAA; isoleucine, leucine, and valine), glutamine, and sulfur-containing amino acids (lysine and methionine).

Stressed calves have similar protein requirements to unstressed calves, but due to reduced intake, the protein concentration in the diet should be higher [95,96]. The same recommendation applies to compromised calves as they exhibit a hypermetabolic response with increased nitrogen excretion.

5.2. Meal Offering Frequency and Quantity

At the beginning of the clinical nutrition intervention, based on the ruminant's condition and severity of the morbidity, the meal size should be adjusted. Unfortunately, there is a lack of research addressing meal size and frequency of meal offering in compromised ruminants. The recommendations below are based on non-evidence-based information. Whatever approach to clinical nutrition intervention is taken, the initiation should not be sudden but gradual [26]. The maximum recommended start should not exceed the NEm and should be gradually increased over time. Acutely morbid ruminants are recommended to start at 50% of their daily requirement and then increase by 20% per day until reaching their total requirement. Ruminants diagnosed with chronic morbidity should start at 25% of their requirement and increase by 25% daily until total requirement is accomplished [18,32,97]. To stimulate the ruminant to eat and avoid overloading the alimentary system, the frequency of diet offering should be 2–6 times daily [18]. Starting at a higher percentage of the requirements may result in a refeeding syndrome [97]. The number of meal offerings per day depends on labor availability, rumen capacity [18], and type of diet delivery.

Since the main volume of the diet in mature ruminants is roughage, there may be a significant restriction in the amount delivered per meal offering [21]. For liquid feeds, each feeding bout should not exceed 1.5 L/100 kg BW.

5.3. Medication–Nutrient Interaction

Appropriate considerations should be given to compromised ruminant receiving medications or supplements. These products may interact with nutrients in the diet and potentially interfere with their function. It is important to consider the possibilities of medication–nutrient interactions that can alter the function and/or metabolism, potentially diminishing the effectiveness or possibly causing toxicity [27,98–100]. Additionally, absorption of medications/nutrients/supplements may be affected (e.g., ionophores may increase the risk of copper toxicosis, particularly in sheep; tetracyclines may be chelated with calcium or high crude protein, decreasing their orally administered absorption) [100,101]. The interaction may be even more evident under conditions of compromised gut integrity (e.g., requirements to adjust nutritional management in ruminants with ulcers associated with the prolonged use of non-steroidal anti-inflammatory drugs; use of any oral antimicrobials on the rumen microbes in any rumen dysbiosis) [102,103].

6. Options for the Delivery of the Diet During Clinical Nutrition Interventions to Compromised Ruminants

Ideally, clinical nutrition should prioritize the voluntary intake of the ruminant’s usual diet while incorporating corrective ingredients that do not significantly alter appearance, smell, and taste (e.g., antioxidants, buffers, electrolytes, enzymes, vitamins, or yeasts). When this is not feasible, changes in the diet composition will necessitate an adjustment of the rumen microbes and the rumen itself [8,104]. Unfortunately, the time of adaptation of the rumen and the rumen itself may delay the desired effect and recovery [105]. Depending on the underlying cause requiring clinical nutrition intervention, modifications to the diet may be considered to make it more appealing to the compromised ruminant. For instance, finely chopping the feedstuffs or wetting the diet could be implemented for a patient with oral/pharyngeal pain.

Few modes are available for delivery of the diet during clinical nutrition interventions (Table 2) [18,20,21,26,35,36,63,65,87,97,106–108]. These modes are not mutually exclusive, and a few combinations are possible. For example, a ruminant patient may have a partially maintained appetite, and the oral mode may need to be supplemented with an enteral or parenteral mode. The order of preference, particularly in mature ruminants, should be oral, followed by enteral, with the least preferred being a parenteral mode of delivery of the clinical nutrition intervention (Figure 2). Yet, the final decision regarding the choice of the mode of delivery of clinical nutrition intervention differs from case to case, depending on available equipment, facilities and nutrients; feasibility of implementing the proposed clinical nutrition intervention; financial or time constraints; functionality, health and integrity of the alimentary system; duration, severity, and the type of illness (e.g., inappetence to anorexia); and patient signalment (e.g., age) [18,26,39,85,87]. In some cases, particularly in neonatal ruminants, parenteral nutrition must be initiated but supported or gradually replaced by an oral/enteral route as soon as possible [39,109]. Whilst economics will be one of the major factors influencing the decision on the mode used in clinical nutrition information, changes in the socio-economic structure of society and the larger number of ruminants being kept as pets may mean an anthropomorphic approach needs to be considered, as applicable.

Table 2. Main characteristics of modes for the delivery of nutrients to the patient during clinical nutrition interventions.

Parameter	Oral	Enteral ¹	Parenteral
Indication/s	Alimentary function is grossly maintained	Alimentary health is minimally affected	Alimentary health is grossly affected
	Alimentary health is minimally affected	Caudal alimentary function is grossly maintained	Complete lack of alimentary function/patency
	Alimentary patency maintained	Caudal alimentary patency is maintained	Dehydration > 8%
	Some appetite present	Complete absence of dietary intake	Failure to obtain adequate nutrition by other modes
		Inability to obtain adequate nutrition by the oral route	Severely compromised metabolic states (e.g., hepatic lipidosis, pregnancy toxemia)
			Severe hemodynamic compromise
Absolute contraindication	Completely interrupted patency of the alimentary system caudal to the forestomaches	Completely interrupted patency of the alimentary system caudal to the forestomaches	
	Congenital abnormalities	Congenital abnormalities	
	Hemodynamic instability (e.g., shock)	Hemodynamic instability	
	Lack of swallowing reflex	Non-functional alimentary system	
	Non-functional alimentary system		

Table 2. Cont.

Parameter	Oral	Enteral ¹	Parenteral
Advantages	Allows for the provision of roughage ² Cheapest Improved protein utilization through microbial digestion Lowest labor requirements Least stressful for the ruminant individual Maintenance of the functionality of the entire alimentary system Maintenance of the gut-associated lymphoid tissue (GALT) Maintenance of the integrity of the entire alimentary system Maintenance of the rumen microbes Maximized enteral resistance to pathogens Minimal gut permeability Physiologic function	Allows for provision of roughage ² Attenuation of hypermetabolic response to injury ³ Cheap Improved protein utilization through microbial digestion Lower labor requirements than parenteral feeding Rumenostomy after the intervention is less stressful Maintenance of functionality of a major portion of the alimentary system Maintenance of the GALT Maintenance of the integrity of a major portion of the alimentary system Maintenance of the rumen microbes Maximized enteral resistance to pathogens Minimal gut permeability Minimal risk of fluid overload More physiologic compared to parenteral	Providing essential nutrients when other modes are not possible Rapid rehydration and correction of acid–base, electrolyte, fluid, and nutrient imbalances
Disadvantages	Intake must be carefully monitored, as the diet may not be eaten, or selection of feedstuffs may occur	Administration of a liquid diet generally results in a shorter transit rate Higher demand for nutrients for the enterocytes Requirements for specialized equipment Supplementation of effective fiber	Altered function of the alimentary system (e.g., enzymatic dysfunction, rumen inactivity/loss of microbes) Altered integrity of the alimentary system (e.g., increased gut permeability, intestinal atrophy) Does not allow for the provision of roughage Expensive High risk of fluid overload Major labor requirements (e.g., requirement of regular monitoring and checks for side effects) Minimized enteral resistance to pathogens Requirements for specialized equipment
Risk of side effects	Minimal	Medium	The highest
Common side effects	Aspiration pneumonia Refeeding syndrome Worsening of diarrhea	Refeeding syndrome Worsening of diarrhea	Imbalanced nutrient supplementation (e.g., amino acids, electrolytes, fluid) High risk of infection (e.g., catheter/giving set contamination; fluids being excellent media for microbial growth) High risk of liver disorders Refeeding syndrome Thrombophlebitis
What can be fed	Usual diet, usually finally chopped/maximized palatability ⁴ /moistened	Usual diet, usually finally chopped/moistened Blended diet Ground/Pulverized diet	Simple nutrients (e.g., amino acids, minerals, mono- to di-saccharides, peptides, and vitamins)
Commonly used methods	Bunk feeding/Pasture grazing	Oro-ruminal tubing ⁵ Permanent/Temporary rumenostomy ⁶	Intravenous route

¹ Includes ruminal feeding; ² If combined with transfaunation, also for transfer of live microbes, nutrients, and volatile fatty acids; ³ When instituted early; ⁴ Additives (e.g., molasses)/Avoiding additives with bitter taste or low palatability/Avoiding feedstuffs with offensive smell and taste/Avoid feedstuffs with poor nutritional quality/Avoiding feedstuffs with rough appearance or texture/Avoiding diets with presence of decomposition or molds/Avoiding diets that the patient is unfamiliar with/Fresh grass/Freshly prepared ration, Offering fresh grass/hay; ⁵ Allows delivery of pulverized and/or very finely chopped diet; ⁶ Allows delivery of nearly any diet; Information in this table is deducted from the following references: [18,20,21,26,35,36,63,65,87,97,106–108].

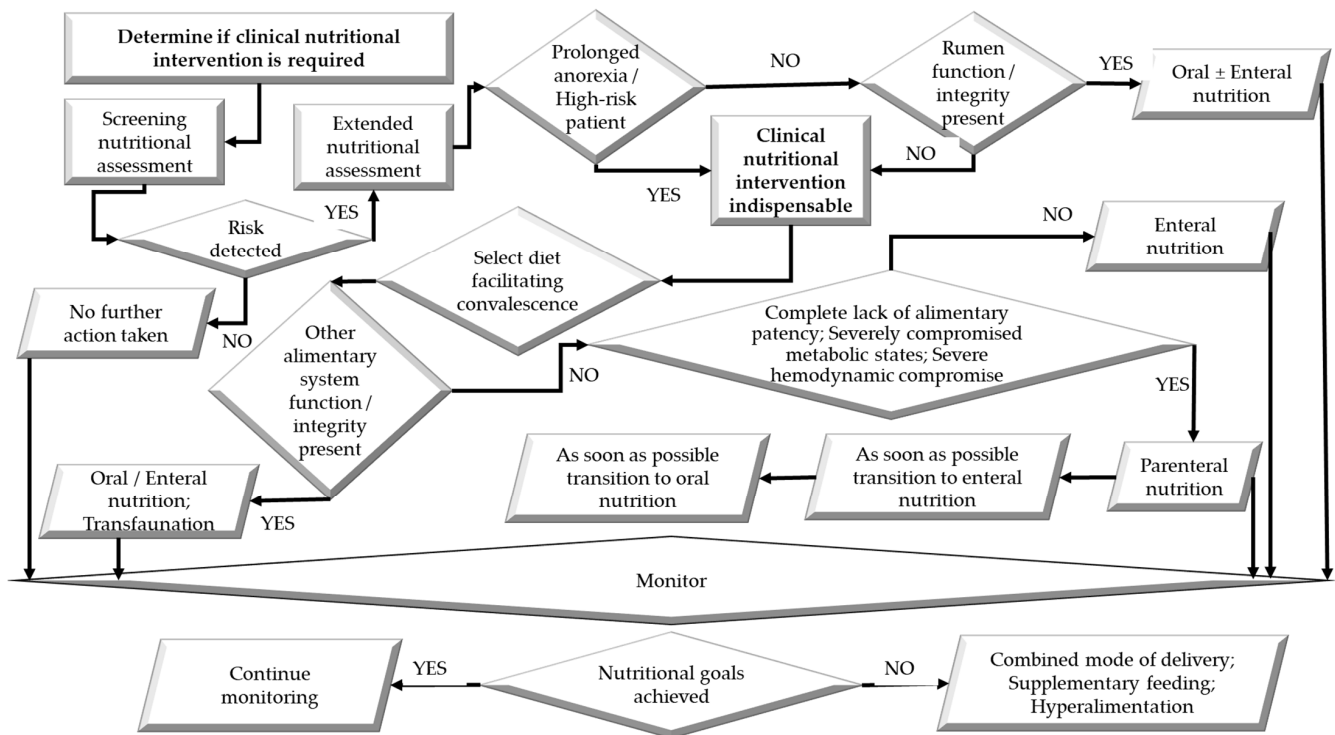


Figure 2. Decision tree for the choice of the mode of delivery of the clinical nutritional intervention.

6.1. Oral Feeding Mode

Ruminants have evolved to consume forage, which is likely the ideal diet for them. Forage is usually attractive to ruminants, even when in an unfamiliar environment [32], and this advantage should be utilized. Fresh grass is usually the last feed that may be rejected by compromised ruminants [26]. Nevertheless, this recommendation is contingent upon the specific system and usual diet, as many ruminants are fed a significant proportion of non-forage components (e.g., feedlot ruminants or high-producing dairy cows). Therefore, a blanket recommendation of providing high-quality forage may not be appropriate, and the ideal diet would be the one to which the ruminant is accustomed. This is because of the essential function of the rumen microbes in the fermentation of feedstuffs otherwise non-digested by mammalian enzymes. For each type of alteration in ruminant physiology, there are specific dietary strategies that may help maintain voluntary intake (Table 3; [26,27,36]).

In compromised ruminants, there are commonly reductions in appetite. To assist in the maintenance of feed intake, modifications to the diet's accessibility and preparation may be required [26,27,36,110]. Therefore, any clinical nutrition intervention should ensure diet is easily accessible, easy to digest, and has improved palatability.

Depending on the type of illness/injury, to allow for maximum diet intake, the offered diet may need to be in a portable feed bunk or similar or, when fed in a group, the patient should have sufficient feeding space without significant effects of social interactions. When fed from a feed bunk in the 'hospital mob', the clinical nutrition intervention should ensure at least twice the feeding spaces for the number of ruminants. Additionally, to minimize the risk of spoilage and diet refusal, the feeding area should be cleaned and refusals removed daily [36,72]. Similarly, for pasture-based management of the hospital mob, there needs to be enough access to fresh grass. As the weather has a greater effect on compromised ruminants, the paddock where the hospital mob is located should have some type of shelter provided.

Table 3. The order of preference of clinical nutrition intervention depends on the pathophysiological mode that is affected within the alimentary system in the compromised ruminant.

Alteration in	Method of Delivery of Clinical Nutrition	
	Type in Order of Preference	Notes
Appetite *	Maximize palatability ² Use of pharmaceutical appetite stimulators ¹	Mainly generalized morbidity
Prehension *	Moistened diet ² Finely chopped and moistened diet ² Ruminal feeding Pellets	Mainly localized morbidity (e.g., Dental morbidity, Dysfunction of some cranial nerves, Stomatitis, Trauma) but also some generalized morbidity (e.g., Botulism, Tetanus)
Mastication *	Moistened diet ² Finely chopped and moistened diet ² Ruminal feeding	Mainly localized morbidity (e.g., Actinobacillosis, Dental morbidity, Dysfunction of some cranial nerves, Osteomyelitis, Stomatitis, Trauma) but also some generalized morbidity (e.g., Botulism, Tetanus)
Swallowing *	Moistened diet ² Finely chopped and moistened diet ² Ruminal feeding	For esophageal ± pharyngeal alterations, feeding from an elevated surface (30–50 cm above the ground)
Rumen fermentation	Decrease/Increase particle size (as required) ² Use of ruminal buffers ¹ Transfaunation	Water contents may need modification, particularly after prolonged anorexia
Eructation	Use of condensed tannins ² Use of methane abatement products ¹ Use of plant secondary compounds ^{1,2} Transfaunation	
Rumination	Increase particle size ²	
Chyme passage	Increase the inclusion of highly fibrous components in the diet	
Digestion	Feed manufacturing, involving water and heat High-quality forage Grain processing, involving chemical, physical, or thermal methods	Mainly a syndrome of maldigestion
Absorption	Oral rehydration compounds containing glutamine, glycine, or glucose	Mainly a syndrome of malabsorption Severe internal parasitism
Elimination	Water intake Physical activity Improving rumen function Emesis Use of pharmaceuticals (e.g., laxatives) ¹	Mainly syndromes of constipation and diarrhea, but also the hindgut fermentation
Utilization	Amino acids Enzymes Glucose Minerals Vitamins	Mainly generalized morbidity

¹ Approval for use differs between countries; ² Requires voluntary intake (or ruminal feeding); * Force feeding may be required.

To increase the chances of the offered diet being ingested, minor modifications may be required (e.g., cut and carry the pasture, finely chopped/moistened diet). Additionally, when fed from a feed bunk, the diet should be offered in smaller amounts but more frequently. Yet, if the compromised ruminant refuses to eat fresh grass for a few days, it is a candidate for other modes of delivery of the clinical nutrition intervention (enteral or parenteral). In ruminants unaccustomed to grass, risks of bloat and indigestion should be considered. Additionally, lush grass may be associated with a lower chewing activity and so, an increased risk of rumen acidosis.

To increase diet palatability, any of the manipulations mentioned in the footnote of Table 2 may be utilized, often in combination. Intending to allow rumen adaptation, any alterations in the diet composition or appearance require gradual introduction, usually around 25% of the change every 2–3 days. When energy density in the diet is not sufficient for the requirements of the compromised ruminant, grains/molasses/propylene

glycol/vegetable oil may be added to the diet with the caution to not exceed the recommended levels for rumen health [26,53]. When protein density in the diet is not sufficient to meet the requirements of the compromised ruminant, canola/lucerne/soybean meal or whey protein may be added to the diet [26,32,53]. In pasture-based systems, providing legume-based pastures (e.g., clover, lucerne) is recommended over grass-based pastures [26,37]. Yet, some grass varieties (e.g., rye grass) may contain enough protein.

In cases where the ruminant is not eating or is unable to eat independently, enteral or parenteral feeding may be necessary (Table 3). However, it is important to note that these methods require additional labor and limit the use of fiber, thereby increasing treatment costs. As dehydration is a common problem in ruminants, it is worth noting that a functional rumen is capable of absorbing large volumes of water and electrolytes, particularly when hypotonic [65].

Pre-Ruminant Stage

Bright and alert ruminant neonates with maintained suckle reflex and functionality and, at least, partial integrity of the alimentary system, should be offered milk from the dam, bottle with teat, or bucket, dependent on their usual nutritional management; their dam's milk is the preferred choice. When a dam's milk is limited in supply or not available, providing milk from another lactating female from the same species is recommended throughout the neonatal period [39]. If milk is not available at all, then careful introduction of a suitable milk replacer may need to be considered [28]. The historic belief that, due to being a suitable medium for the growth of pathogens, milk should not be given to calves with diarrhea has been overcome by research findings. The current information indicates that milk should not be withheld from ruminant neonates for >12 (to a maximum of 24) h [76].

When dehydration is present, it should be corrected by oral fluid and electrolyte replacement therapy. Some evidence states that due to the prevention of clotting of the milk within the abomasum, oral electrolytes should not contain bicarbonate [111]. This is now believed to be incorrect [76,112,113]. Electrolytes containing antacids that result in the production of glucose (e.g., acetate, lactate) may be preferred due to the improved absorption of sodium, not due to lack of interaction with clotting of milk within the abomasum [112].

6.2. Enteral Feeding Mode

When a ruminant patient is unable to maintain sufficient feed intake but still has functional digestion and absorption in the alimentary system, enteral feeding becomes necessary [7,21,26,32,53,66,109,114]. The inability to maintain a sufficient intake may result from alterations in appetite, prehension, mastication, swallowing, rumination, eructation, or passage, or neurologic alterations (e.g., obtundancy).

For our purposes, ruminal feeding is included in the section of enteral feeding. In such cases, the diet should ideally (in order of preference) be moistened, finely chopped, or blended into a liquid form [21].

When passage of ingesta is interrupted or there is anorexia or inappetence, a few approaches to ruminant feed delivery may be considered. These include feeding through the mouth when the passage of the bolus is uninterrupted, a surgically placed esophageal tube (rarely used in ruminants as rumenostomy is easily carried out), oro-ruminal tube, or temporary or permanent rumenostomy (common synonyms: rumen cannulation/fistulation). Placement of an esophageal tube and rumenostomy are suitable for prolonged extra-oral feeding.

For blending, the livestock individual's usual diet is blended and suspended in water to the desired consistency. For administration by oro-ruminal tube, each kg of blended (pulverized) diet should be mixed with 3–5 L of water. The aim of adding water is to achieve a final 'slurry-like' or gruel consistency that will flow through the esophageal/oro-ruminal tube [21]. When the usual diet is unavailable or difficult to provide, a blended complete pelleted diet is the simplest method for providing ruminants with nutritious and balanced feed [87]. Additionally, pelleted food is usually cheap compared to other balanced options. The downside of the pelleted food in unaccustomed ruminants is the increased risk of alimentary upset, similar to high-concentrate diets [21].

Commercial diets for enteral feeding are currently too expensive to be considered under field conditions. Additionally, their composition is often not suitable as a ruminant diet. However, in exceptional circumstances, elemental component feeding (e.g., amino acids, particular carbohydrates, or peptides) may be considered. To maintain the ideal pH in the rumen, as the reduction in fiber particle size can diminish rumination and salivary gland stimulation by chewing, maintenance of the rumen pH may require the use of buffers.

Unfortunately, any prolonged fasting in ruminants leads to alterations in rumen microbes and indigestion [115,116]. An important objective of the enteric feeding should be the rapid and multidimensional restoration of the forestomach function.

6.2.1. Transfaunation

One potential solution to altered forestomach function in ruminants is transfaunation [19,20,117,118]. The term transfaunation is probably a misnomer, as the process transfers not only microbes but also various nutrients, including volatile fatty acids [20,118]. Detailed discussion on the selection of donor ruminant, collection and administration of the rumen fluid is beyond the scope of this paper, but they have been well described elsewhere [19,20,106,117,118]. Briefly, collection of rumen fluid for transfaunation can be carried out by abattoir or, less preferred, post-mortem opening of the rumen, or from live ruminants, using oro-ruminal tube or by rumenostomy (temporary or permanent) [19–21,117,118]. Administration of collected rumen fluid from one ruminant to the compromised ruminant individual can be carried out using an oro-ruminal tube or by rumenostomy (temporary or permanent) [19,21,117,118].

A single transfaunation is usually sufficient. Repeated transfaunation may be required for some chronic disorders, either forestomach (e.g., rumen putrefaction) or generalized (e.g., botulism; [21]). Historically, large volumes of rumen fluid were recommended for transfaunation (e.g., in cattle ≥ 10 L; [20]). However, newer research suggests that as little as 1 L of rumen fluid being transferred during transfaunation may be sufficient [118]. Transfaunation has been proven to be an efficacious supportive treatment strategy for various alimentary or metabolic disorders (e.g., anorexia, indigestion, ketosis, left displaced abomasum, and rumen acidosis; [19,117,118]).

6.2.2. Pre-Ruminant Stage

In pre-ruminant individuals (neonates), the initial resuscitation approach involves administration of electrolytes and fluids (oral and/or parenteral) [110,119]. This approach should correct dehydration, major electrolyte imbalances and metabolic acidosis.

To facilitate intestinal absorption of sodium, orally/enterally administered fluids should contain any of glutamine, glycine, or glucose, individually or in combination.

To supply enough energy to the neonate, milk is the preferred choice for enteral supplementation. The final aim of clinical nutrition intervention should be to provide the ruminant neonate with at least 10% of its body weight of milk per day in at least 2, preferably more, daily feeding bouts. Initial feeding bouts should be of smaller volume,

and, thereafter, the volume should be increased gradually to the desired daily volume. Milk can be provided by (in order of preference) suckling, bottle/container with a teat, bucket, or enteral feeding tube for ruminant neonates.

Note that suckling, bottle/container with a teat, and bucket feeding require a functional suckle reflex. Syringe dosing is not recommended for compromised ruminant neonates as it is often associated with a high risk of aspiration pneumonia. As neonates are, in essence, monogastric animals, a naso-gastric feeding tube may be used for the delivery of liquids directly into the abomasum. To stimulate the esophageal groove, the tube may not need to extend beyond the mid-esophagus [63].

6.3. Parenteral Feeding Mode

When there is dysfunction of the alimentary system, or the patient is unable to digest and absorb nutrients due to prolonged anorexia (0.5–1 day in neonates, >3–4 days in mature ruminants), intravenous parenteral nutrition is typically required. Intraosseous and intraperitoneal administration are not suitable for parenteral nutrition but are suitable for electrolyte and fluid therapy. Parenteral nutrition can be complete/total, partial or hyperalimentation [26,65]. In complete parenteral nutrition, the entire feeding is satisfied parenterally (e.g., completely non-functional alimentary system). In clinical practice, due to the expense, complete parenteral nutrition is restricted to neonates and valuable mature ruminants [26,27,32,63,65]. In partial parenteral feeding, a portion of the requirements is satisfied orally/enterally (e.g., inappetence). The cost of the partial parenteral nutrition makes it suitable for intervention in any ruminant. In hyperalimentation, more nutrients are supplied than required (e.g., major burns, sepsis, or severe cachexia). During parenteral feeding, a commercially available and properly formulated liquid diet should be used (Table 4) [26,120], and the ruminant must receive appropriate nursing and supportive care. Parenteral nutrition should be via a separate line and use a 1.9 to 2.2 mm catheter in mature ruminants and a 1.4 to 1.8 mm catheter in neonates.

Table 4. Composition of published parenteral fluids for ruminants.

Compound	Type of Ruminant			
	Calves 1	Calves 2	Small Ruminants 1	Small Ruminants 2
Vehicle	Balanced electrolyte solution or Sterile water	Balanced electrolyte solution or Sterile water	Balanced electrolyte solution	Balanced electrolyte solution
Dextrose 50%	20%	16.6%	10%	10%
Amino acids 8.5%	3.4%	2.8%	20%	20%
Lipids	2%	3.3%		10%
B-complex	Separate line	Separate line	4 mL/L	
Potassium chloride (as indicated)	Separate line	Separate line	20–40 mEq/L	
Calcium gluconate 23% (as indicated)	Separate line	Separate line	4–10 mL/L	
Reference	[26,110]	[26,110]	[120]	[65]

The line and the catheter used for administration of the parenteral nutrition should not be used for administration of any other compounds, including medicines [120]. The catheter should switch veins every 3–5 days, provided there is no evidence of an inflammatory reaction before that [26]. At initiation, the parenteral nutrition flow should be at 25–50% of the desired rate for the first 3–4 h, and if there is no hyperglycemia, the rate is gradually increased (over 0.5 h) to the desired rate (usually about 5% of BW per day) [120]. Another recommendation is to start at 25% of the requirements and raise by another 25% every 12–24 h [63]. To prevent lipid embolism and severe metabolic disturbances, the initial

parenteral fluid composition should also contain only 50% of the final lipid composition and should be gradually increased after the first 3–4 h until the desired concentration is reached [26]. The parenteral nutrition fluid and the administration set should be changed daily [26,65,120]. At discontinuation, a gradual decrease in the delivery rate is required over 24–48 h [63,65,120]. Sudden stops in parenteral feeding may result in life-threatening complications such as rebound hypoglycemia [26] or shock. If insulin therapy is used as part of the parenteral nutrition, it should be discontinued at least 24 h before discontinuation of the parenteral feeding [63]. The risk of rebound hypoglycemia is particularly high in neonatal kids [26]. For maintenance of a constant flow rate, parenteral nutrition as part of the clinical nutrition intervention should utilize an infusion pump [26,65,120].

The role of dextrose and lipids is the provision of energy to prevent the utilization of amino acids/proteins as an energy source. Sparing of amino acids/proteins from being used as an energy source allows them to be used in the repair of tissue damage [26]. Glucose is cheaper and better utilized by ruminants, particularly neonates, but is not sufficiently energy dense. Hence, some lipids are essential in parenteral fluids. The lipids can be included at a maximum of around 30–60% of the required energy support in the patient [65]. In patients with suspected hepatopathies, the lower end (around 30%) should be considered [65]. Due to the risk of interactions between compounds, amino acids should be added to the vehicle first, followed by lipids, and then dextrose [13,121].

As the major problem with parenteral nutrition is sepsis, mixing of compounds must be carried out using aseptic principles [13,121]. The site of catheter attachment should be checked for evidence of heat or pain 3–4 times per day [26]. Hematologic assessment, including differential white counts, should be performed daily.

As metabolic derangements with parenteral nutrition are common, daily checks of BW, and monitoring for evidence of creatinine increase, electrolyte imbalance, lipidemia, overhydration (less likely underhydration), and alterations in liver enzymes are required [110,120]. Checks of vital signs and glucose levels, at least initially, should be carried out 3–4 times daily [26,110,120]. Serum should be checked for evidence of lipemia, also at least daily [26]. Urine should be checked for evidence of excess glucose and protein at least twice daily.

Although regular monitoring should prevent serious events, another potential disadvantage of parenteral feeding is the refeeding syndrome [120]. Common alterations, among other metabolic alterations, that should be looked for include hypokalemia, hypomagnesemia and hypophosphatemia [120]. However, clinical and laboratory hypokalemia alone may also occur due to the intracellular shift of potassium, due to the correction of the metabolic acidosis (in neonates) and increases in glucose levels [110]. Some ruminants, particularly goats, are prone to chewing the administration line and regular checks are required to prevent intravenous administration of air and, often fatal, air embolism [121].

Pre-Ruminant Stage

Parenteral nutrition of pre-ruminant neonates is more feasible than for mature ruminants and has been proven to save some neonates that would have died otherwise [108,119]. However, most of the parenteral nutrition interventions reported are related to correction of acid–base, electrolyte, and fluid imbalances. Detailed discussion on fluid therapy in neonatal ruminants is beyond the scope of this review and has been previously addressed [27,110,122]. However, a few notes are worthy of discussion. In neonates with diarrhea, the parenteral approach is best if supported by an oral/enteral approach; the oral approach always includes colostrum/milk [108,119,123]. The combination approach has resulted in the fastest recovery rates, lowest expense, with no adverse long-term effects on growth, health, and wellbeing. Initial treatment may require an exclusive parenteral mode (e.g., rapid correction of acid–base, electrolyte, and fluid imbalances), at least until a suckle

reflex is present. In many ruminant neonates, in the initial stages of the clinical nutrition intervention enteral feeding cannot satisfy the entire nutritional support required. Hence, to ensure complete nutritional support of the ruminant neonate, the combined approach can be used, at least initially. Additionally, parenteral nutrition in the initial stages can minimize complications with potential overloading of a compromised gut [26,80]. Parenteral fluids should be delivered by the intravenous route, with the subcutaneous route being considered only as supportive therapy following corrections of hypovolemia and metabolic acidosis [123].

7. Pharmaconutrition for Compromised Ruminants

Pharmaconutrition is a concept that focuses on the effects of pharmacologic doses of individual nutrients on immune function and subsequent clinical outcomes [11]. These nutrients could enhance drug delivery and pharmacological action, improve gut microbiology and gastrointestinal health, and/or modulate the inflammatory and immune responses [27]. For example, to aid the therapeutic recovery process, arginine, glutamine, omega-3 fatty acids, and selenium are commonly recommended as adjuncts to various medications or as special nutritional supplements [13]. However, few data are available on ruminants, with most data referring to pharmaconutrition in humans (Table 5). Following oral administration, many of the nutrients discussed below undergo fermentation in the rumen. Therefore, the applicability to ruminants is yet to be confirmed. It is worth noting that, as with any medicament, products used in pharmaconutrition may have side effects, and so uncontrolled dosing or overdosing is not recommended. Additionally, the field of pharmaconutrition is still in development, and discoveries occur daily. The inclusion of pharmaconutrients can be successful if the primary energy and protein requirements are met. Pharmaconutrition is not a replacement for, but only a support within, the clinical nutrition intervention.

Some electrolytes (e.g., chloride, potassium, and sodium) are essential for the functionality of various systems and should be closely monitored and supplemented as required [32,120]. Some minerals (e.g., calcium, phosphorus) are important to be supplemented with prolonged anorexia [32] and in bone tissue repair processes. Note that exogenous amino acids are insufficient and ineffective in minimizing protein catabolism in chronic disease/inflammation, but can supply precursors for protein synthesis and result in decreased net protein catabolism [7]. Phenylalanine is used to aid pain relief.

Overall, immunomodulators that can be used in clinical nutrition intervention may include amino acids, fatty acids, minerals, plant-based products, probiotics, and vitamins. Any antioxidant is also an immunomodulator. Indeed, a functional immune system requires sufficient energy and protein supply [9,32].

Table 5. Pharmaconutrients that can be used in ruminant clinical nutrition interventions.

Antioxidants ¹	Appetite Stimulators ²	Gut Integrity Protectors ³	Immunostimulants	Liver Protectants	Rumen Modifiers	Wound Healing Stimulants
Amino acids Arginine, Glutamine [38,87,109,124] Branched chain amino acids (e.g., isoleucine, leucine, and valine) [99] Sulfur-containing amino acids (e.g., lysine, methionine) Minerals Copper, Selenium, Zinc [10,41,125] Plants or Plant-based products Condensed tannins, Garlic (<i>Allium sativum</i>), Oregano (<i>Origanum vulgare</i>) [126–128] Vitamins Beta-carotene, Vitamins C and E [10,41,125]	Acetyl-Coenzyme-A- binding protein [129] Benzodiazepines (although more known in monogastric animals) with a note that all have more or less sedative activity as well Beta-agonists [130–132] Vitamins of the B-complex [32] Various hormones, medications, plant products, proteins, and vitamins [130–132]	Orally administered glutamine [7,29,30,39,41,53,63,87,124,133–135] Plant-based products [136] Prebiotics [67] Probiotics [67,80,137] Vitamin A [125] Zinc [125] Yeasts [137]	Amino acids, including arginine, glutamine and leucine [30,38,41,53,63,87,107,124,133] Fatty acids, including omega-3 fatty acids [11,41,138] Minerals, including calcium, chromium, copper, iodine, iron, selenium, and zinc [31,32,125,139–141] Plants or plant-based products including ashwagandha/ winter cherry (<i>Withania somnifera</i>), black-berry nightshade (<i>Solanum nigrum</i>), cinnamon (<i>Cinnamomum verum</i>), echinacea (<i>Echinacea purpurea</i>), fenugreek (<i>Trigonella foenum-graecum</i>), fire-flame bush (<i>Woodfordia fruticosa</i>), garlic (<i>Allium sativum</i>), guduchi/heart-leaved moonseed (<i>Tinospora cordifolia</i>), oregano (<i>Origanum vulgare</i>), shatawari (<i>Asparagus racemosus</i>), tulsi/holy basil (<i>Ocimum sanctum</i>), turmeric (<i>Curcuma longa</i>), and thyme (<i>Thymus vulgaris</i>) [125–127,136,142] Some probiotics [24,32] Vitamins including beta-carotenes, Vitamin A, B-complex, C, and E [26,31,32,125]	Branched chain amino acids [39,99,143–146] Fatt acids [147,148] Glutamine [30] Sulfur-containing amino acids [68,144,146]	A large group of products have been tested to improve rumen health (e.g., antimicrobials, mannan-oligosaccharides, rumen buffers, and yeasts). Many of these compounds improve rumen health by maintaining optimal rumen pH [2,3,67]. Others are used to decrease the inflammatory response of the rumen [67] and/or improve nutrient utilization [2,3,67] and/or prevent bacterial or toxin translocation from the rumen into the general circulation [67].	Arginine [53] Glutamine [134,135] Vitamin A [125] Vitamin K [60] Zinc [125]

¹ Supplementation of antioxidants to the diet used in the clinical nutrition intervention is important in all ill and injured ruminants, but particularly those with signs of endotoxemia and sepsis [7,41]. Whenever possible, these should be provided by oral/enteral route; ² The approvals for use differ significantly between countries.; ³ This group includes nutrients specifically improving absorption of some nutrients (e.g., sodium), manipulating the gut microbes, being nutritious/protective of the intestinal mucosa, and/or preventing absorption of bacterial products.

8. Future Directions

Ruminant management and nutrition will become progressively more affected by transformative approaches, including big data, real-time monitoring, precision technologies, and tailored, individualized feeding. Precision nutrition aims to tailor diets to meet the specific nutritional requirements of livestock, based on their production and reproductive status, as well as the stage of the productive cycle, all while considering environmental conditions and health. Precision nutrition, facilitated by intelligent feeding systems, has a two-fold potential, satisfying tailored nutritional requirements by livestock and, by reducing nutritional waste, decreasing environmental pollution. Additionally, it reduces cost, labor requirements, and risk of human-related mistakes [149].

As the understanding of pathophysiology and epidemiology of nutritional disorders increases (e.g., incorporation of metabolomics and nutrigenomics in diagnostics [150,151]), the role of precision nutrition is likely to become more important in clinical nutrition, both in prevention and treatment. Yet, due to the current cost and time delays in obtaining results, the widespread use of precision nutrition, or heavily tailored clinical nutrition in ruminants, is not yet practical.

9. Conclusions

Clinical nutritional interventions should be based on knowledge of normal and abnormal physiology and potential morphological changes. Utilization of nutrients in mature ruminants is dependent on rumen microbes. Rumen health must be considered and maintained as much as possible during clinical interventions. Whenever possible, diet should be delivered orally. Second, one of the possibilities of diet delivery is ruminal feeding, and the least favored options are direct enteral and parenteral feeding. The success of interventions can be measured by improvements in appetite, behavior, and health. However, further research is needed to better understand the specific dietary requirements of ill and injured ruminants.

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Glossary

Acidosis	A condition characterized by excess acids in the body fluids
Anorexia	A complete absence of appetite (Synonym: aphagia)
Appetite	The desire to eat the offered diet
Big data	Aggregation and analysis of large/big data sets from multiple sources in management decisions in livestock enterprises (e.g., diet analysis, genomics, health records, production and product quality)
Bloat	Bloat is distention of a portion of the forestomachs (i.e., rumen) resulting from the accumulation of free gas or froth (Synonym: Tympany)
Clinical nutrition	The study and overall analysis of the interaction of nutrition and overall growth, health, and wellbeing of the (ruminant) body/individual
Colostrum	The first secretion from the mammary gland after calving (giving birth), rich in antibodies, growth-stimulating factors, other immune factors, and nutrients

Dysphagia	Difficulty swallowing; In broader term, it means difficulty in taking feed and/or liquids through the mouth, pharynx, and/or esophagus, so preventing entry into the stomach
Dysbiosis	An imbalance between the types of organism present in an animal's microbiota, especially that of the alimentary system, thought to contribute to a range of ill health effects
Effective fiber (eNDF)	The fraction of fiber (NDF) that stimulates chewing activity, primarily related to the particle size (Synonym: physically effective fiber; peNDF)
Forage	The most important feed resource for ruminants globally. Representatives are grasses, forage crops, and legumes. May be fed as pastures or preserved forages (e.g., baleage, hay, or silage)
Hepatic lipidosis	A major metabolic disorder, most frequently in the very late pregnancy or the early lactation in female livestock as a result of overproduction of fatty acids and accumulation of lipids within the liver (synonym fatty liver disease)
Hyperalimentation	Administration of excess nutrients by enteral/parenteral route, particularly in patients unable to ingest enough diet orally
Immunocompetence	The ability of an individual's immune system to work properly, allowing its body to mount an appropriate immune response as required
Immunostimulant	A substance of natural or pharmaceutical origin that stimulates the body immune system, usually in a non-specific manner, by activating or enhancing any of its components
Inappetence	A decreased appetite (Synonym: hypophagia)
Indigestion	A disruption of the 'normal' function of the reticulorumen (main portion of the forestomaches in ruminants) that may affect forestomach motility or microbial fermentation or both
Intelligent feeding system	A feeding system that incorporates an algorithm developed to monitor livestock ration intake, dependent on the data collected by precision technologies and/or human input
Morbidity	Any ill state in an individual; Proportion of the population affected by a particular condition/disorder/problem; State of being affected
Neonate	Newborn individual; In ruminants, typically first 3–4 weeks of life before any forestomach activity is present (pre-ruminant stage)
Nutrigenomics	The study of the complex interaction of genes and nutrients in livestock, to understand how various genes are expressed in response to specific nutrients
Obtundancy	A dulled or reduced levels of alertness or consciousness of an individual (common misnomer in veterinary medicine is depression, which is a symptom, not a sign)
Parenteral	Given/Occurring/Situated outside the intestines
Patency	The quality and state of a tubular organ/system being open and passage being uninterrupted
Prebiotic	A non-digestible food ingredient that promotes the growth of beneficial gut microbiota
Probiotic	Directly fed microbe which stimulates the growth of particular microbiota, especially those with beneficial properties (such as those of the gut microbiota)

Real-time monitoring	Monitoring involving automated systems, sensors, or wearable devices to continuously collect data on behavior, health, physiological parameters, and production
Refeeding syndrome	A potentially fatal shift in electrolytes and fluids that may occur in severely malnourished patients receiving artificial refeeding, whether oral, enteral, or parenteral
Reperfusion	The restoration of the blood flow to an organ or tissue after being significantly to completely blocked
Resting energy requirement (RER)	The energy requirement of a livestock individual at rest in a thermoneutral environment
Rumen	The first forestomach and the largest in mature ruminant. It is a muscular sac that contains large number of microbes involved in fermentation of the ingested diet. Fermented ingesta is passed into the reticulum. The fermentation of diet components unable to be digested by mammalian enzymes makes ruminants valuable in the eco system
Rumen acidosis	A metabolic disease that affects all ruminants. In cattle, both feedlot as well as dairy cattle. Rumen acidosis is usually associated with the ingestion of large amounts of highly fermentable, carbohydrate-rich feeds (e.g., cereal grains), which result in the excessive production and accumulation of acids in the rumen (pH of the rumen contents changes from mildly alkaline [around 7] to acidic [<5.6 down to <4.5])
Rumenostomy	Surgical creation of temporary or permanent (insertion of rumen cannula) opening between the rumen and environment, including incising the skin, subcutaneous tissues, abdominal muscles, peritoneum, and the rumen wall
Siallorrhoea	Excessive flow of saliva ('drooling')
Splanchnic	Related to organ/s within the abdominal cavity
Stress	A Non-specific response of the body to any demand, usually from the environment where the livestock individual resides, management or nutrition
Transfaunation	Procedure consisting of removal of rumen fluid with healthy microbiota and good quality from one ruminant, and transfer of the removed fluid into the rumen of another ruminant individual

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