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Approaches to Management and Care of the Neonatal Nondomestic Ruminant

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KEYWORDS

• Neonate • Pediatric • Antelope • Deer • Ungulate

Veterinary care of the newborn nondomestic ruminant can be both rewarding and very challenging, with some practitioners reporting high mortality rates before 6 months of age.¹ Case management during the critical neonatal period is typically modeled after management of domestic ruminants; however, some unique differences exist between nondomestic ungulates and their domestic counterparts that affect neonatal management and medical care. These differences become apparent quickly when the nondomestic neonate requires treatment, and an understanding of the special needs and risks involved can prevent unnecessary problems and losses. The aim of this article is to discuss the unique challenges presented by nondomestic ruminants and approaches to management of neonatal and pediatric cases.

MANAGING THE DAM

Preparation for neonatal and pediatric care should begin when the dam is known to be pregnant. In nondomestic ruminants, early diagnosis of pregnancy is uncommon due to the increased manipulation and stress associated with handling for rectal palpation or ultrasound. For many species, fecal progesterin assays have been developed, providing a means of noninvasive pregnancy diagnosis.^{2–5} However, this procedure often requires time-intensive repeated fecal collection from the dam (generally 2–5 samples per week) for the duration of 1 estrous cycle plus 2 or more weeks to verify maintenance of elevated progesterone. Several zoological institutions have endocrinology laboratories that may provide endocrine assessments as a service. Rectal palpation and ultrasound examination should be conducted opportunistically, such as for annual examination, hoof

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work, or other medical care, for females suspected to be pregnant. However, the risk of anesthesia may outweigh the benefit of early pregnancy diagnosis in many fractious species. Stillbirths are not uncommon in nondomestic ruminants anesthetized repeatedly during pregnancy.

Preparation for successful birth and mother-rearing should emphasize health and reduced disturbance to the dam. If possible, fractious nondomestic ruminants should give birth in an environment to which they are accustomed. Inexperienced dams, particularly those that have not observed maternal behavior in a herd setting, should be monitored closely near parturition. Species with strong herd instincts may be most successful when housed with conspecifics during calving season, and many will demonstrate birth synchrony.^{6,7} However, wild ruminant species vary widely in social grouping and behavior, and captive grouping can affect reproductive success.⁸ Research into the best captive social environment for the species in question before birth can maximize the chance of successful birth and maternal care. Timing exposure of females to males in nonseasonal or seasonally polyestrous species to maximize the chance of late spring or early summer births in temperate zones can also improve neonatal survival in pasture-managed animals. Housing should provide the opportunity for seclusion, adequate ventilation and drainage, and exposure to sunlight to reduce pathogen levels.

Vaccination of dams for diseases of regional risk approximately 2 months before expected parturition can optimize levels of specific immunoglobulins (Igs) in colostrum and improve neonatal health. Vaccination at this time for diseases of particular concern to neonates such as *Clostridium* sp, *Escherichia coli*, rotavirus, coronavirus, and bovine viral diarrhea is common practice in domestic ruminants.⁹⁻¹¹ However, live vaccines should be used with caution in nondomestic ruminants. One month before expected birth, the energy ration should be increased and special attention paid to the provision of adequate calcium and minerals, particularly if the regional soil is deficient in specific minerals, such as selenium.

General signs of impending parturition include teat and udder enlargement, which can occur days to weeks before birth depending on the species, and vulvar swelling/relaxation. Vulvar changes generally occur closer to parturition than udder enlargement. Dams will often seek isolation just before birth and will often give birth to live offspring at night, while stillbirths and abortions can occur at any time.

PREPARATION FOR BIRTH

While most ruminants are capable of successfully delivering and raising offspring, nondomestic ungulates in zoos and related institutions are often housed in unnatural social or environmental situations, increasing the chances of dystocia, conspecific trauma, and maternal neglect. If a neonate requires medical care, it may be impractical to house the animal with its dam due to the need for frequent separation, stress on the dam, and the likelihood of maternal rejection. Therefore, preparations for neonatal care should include a hand-rearing protocol, which should detail record keeping, equipment/supplies, formula and supplements needed, housing, and socialization. The Association of Zoos and Aquariums' Nutrition Advisory Group (<http://www.nagonline.net/>) and the book, *Hand-Rearing Wild and Domestic Animals*,¹² are practical hand-rearing resources. Veterinary preparations include plans for treatment of failure of passive transfer (FPT, discussed later). Plans to acquire plasma, colostrum, and milk replacer formulas should be in place before birth.

BIRTH AND POSTPARTUM CARE

Dystocia

Identification of dystocia requiring intervention can be complicated in nondomestic ruminants. While anterior dorsosacral presentation of the fetus with forelimbs extended is by far the most common, posterior dorsosacral presentation with the hindlimbs extended is very common in some species, such as Pere David's deer (*Elaphurus davidianus*) and red deer (*Cervus elaphus*) and may or may not cause difficulty in parturition. The most common causes of dystocia in cattle are malpresentation of the fetus and maternofetal disproportion. Dystocia is not uncommon in nondomestic ruminants, particularly in species of limited genetic diversity, and represents an important risk factor for morbidity and mortality in domestic¹³ and nondomestic calves. In fact, beef calves born following dystocia are 2 to 6 times more likely than normal-birth calves to develop disease within 45 days of birth.^{14,15} Nondomestic ruminant neonates are at even greater risk than domestic calves following dystocia due to the necessity of general anesthesia for birth intervention. Furthermore, many dams will not accept an offspring following general anesthesia, resulting in FPT and the need for hand-rearing. Hypoxemia and acidosis in the calf (discussed later) are possible sequelae to dystocia that may also lead to FPT, although it is unclear whether this is due to a direct effect on Ig absorption or to resulting weakness and inability of the neonate to stand and nurse.¹⁶

NEONATAL TRIAGE AND TREATMENT

The Decision to Treat

In beef cattle, 69% of calf losses before weaning occur within 96 hours of birth,¹⁷ underscoring the need for early intervention. While early intervention is also critical in nondomestic ruminants, the decision to treat can be more involved than in domestic operations.

Maternal neglect is a common occurrence resulting in the need for neonatal care in nondomestic ruminants. Dams that are inexperienced, ill, housed in unnatural social or environmental situations, or stressed by human presence are the most likely to reject a neonate. However, the practitioner must keep in mind during the decision-making process that when an experienced dam abandons her offspring, it may be due to a congenital, infectious, or metabolic problem compromising its viability. In such cases, a significant amount of money and time can be put into a neonate whose survival is unlikely.

If treatment requires that the dam and offspring be brought into confinement, the dam may become inappetent, reject the neonate, or both. In some species, such as sable antelope (*Hippotragus niger*), reintroduction of the dam back into the herd following isolation with her calf can be problematic, leading to conspecific aggression and injury.

In many species, the sex of the neonate is a significant factor in the decision to treat. Hand-raised animals will usually demonstrate altered social behaviors as adults, including an inappropriate response to humans. Male ruminants that are hand-reared may thereby pose an intolerable risk as adults in captivity by demonstrating a lack of fear and increased aggression toward humans. Alternatively, hand-reared females may provide a benefit to the herd by showing less aversion to human presence than their herdmates. Such females are useful in "calming" the herd during intensive management, as companions to conspecifics requiring hospitalization, or as surrogate dams to hand-reared neonates, easing the transition of the neonate into the herd. Strategies for socialization of hand-reared neonates are discussed later. The monetary and labor costs of committing to treatment and hand-rearing, the risk to the dam, the likelihood of

success, and the future of the neonate must all be taken into account in the decision to treat a compromised newborn nondomestic ruminant.

Critical Care of the Postpartum Neonate

Neonates born following dystocia or under extreme environmental conditions are more likely to require resuscitation and medical care than those experiencing normal birth conditions. Once the decision is made to treat these cases, early intervention is essential. Dystocia is likely to cause both respiratory and metabolic acidosis. Following normal birth, mild respiratory acidosis may occur for a few hours and metabolic acidosis for up to 48 hours.¹⁸ However, severe acidosis can result in reduced vigor, decreased suckling response and an increased chance of FPT.¹⁹ A good indicator of acidosis is time to sternal recumbency: in cattle, more than 15 minutes to sternal recumbency is associated with low survival.²⁰ Poor muscle tone and decreased pedal reflexes are also suggestive of acidosis. Scleral and conjunctival hemorrhage indicate both hypoxia and acidosis and can also signal a poor prognosis.²¹

Establishment of a patent airway, initiation of a normal breathing pattern, and establishing adequate circulation are the first priorities in the treatment of a critical neonate. In general, cardiac resuscitation is not indicated, as neonates born without a heart beat are not likely to survive.¹⁵ Attention should be paid to physical stimulation of the neonate, as severe acidosis may cause depression of the central reflexes that initiate respiration.¹⁵ To clear the airway, place the neonate in sternal recumbency and clear fluids and physical obstructions by hand or suction. Rub the neonate vigorously with towels or bedding to simulate the phrenic nerve, which innervates the diaphragm. If respiration is not immediately initiated, stimulation of the nasal passage or pharynx with a finger or piece of straw should induce an inspiratory reflex. Acupuncture at the philtrum may also stimulate respiration.²²

Neonates born to anesthetized dams may have respiratory depression resulting from pharmacologic sedation, and specific antagonists should be administered to the neonate as well as to the dam. Doxapram hydrochloride stimulates central chemoreceptors and may be beneficial in calves born with mild respiratory suppression, but it is unlikely to be effective in severely depressed neonates. Use of doxapram as a respiratory stimulant has demonstrated varying results,^{23–25} but it may improve acid-base balance.²² If these methods are unsuccessful, endotracheal intubation and positive pressure ventilation are preferable to mouth-to-nose ventilation for providing respiratory support. Oxygen therapy, if available, has been shown to improve neonatal survival in at-risk domestic calves and is best administered via endotracheal tube.¹⁵

Placement of an intravenous catheter and fluid therapy as necessary can improve the chance of survival in compromised neonates, and provides a means of treating metabolic abnormalities rapidly. Metabolic acidosis can be treated with a bolus of sodium bicarbonate (1–2 mEq/kg)¹⁹ following the establishment of a normal breathing pattern. Treatment before establishing normal breathing, however, is likely to exacerbate respiratory acidosis.

Maintenance of appropriate body temperature is easily overlooked during resuscitation and care of the neonate but essential to its survival, particularly in critical cases that fail to demonstrate normal thermoregulatory mechanisms such as shivering. In domestic calves, provision of an external heat source for 24 hours postpartum has been shown to improve not only body temperature but also oxygen saturation, tidal volume, and respiratory rate.²⁶

IDENTIFYING ILLNESS IN YOUNG RUMINANTS

Despite apparently normal birth, behavior and maternal care, illness is not uncommon in nondomestic ruminants under 30 days of age. Most mortality in domestic calves following a normal birth is due to prematurity, congenital defects, or infection.¹⁵ Close observation of neonates soon after birth is important to document normal behavior and suckling, but an understanding of species-specific behavior is necessary. While domestic calves are precocial “followers” and stand and follow the dam soon after birth, 75% of nondomestic ruminants are characterized as “hiders.”⁶ These neonates are left by their mothers to lie motionless, reunited only for infrequent nursing periods for the first days to months, depending on the species. In these species, failure to observe the neonate nursing, or to find it, may lead the inexperienced observer to assume the neonate is ill. Hippotragine antelopes and deer are frequently “hider” neonates, whereas wild cattle, goat-like species, and gazelles are usually “followers.” Neonates should be observed nursing within a few hours of birth. Observation of a calf that is weak or unresponsive to stimulation by the dam, demonstrates prolonged unsuccessful or no attempts to nurse, or is too weak to stand soon after birth indicates a need for evaluation.

Approach to Illness in Neonatal and Young Ruminants

Common causes of illness in neonatal ruminants include acidosis, hypothermia, hypoglycemia, dehydration, pneumonia, and septicemia.^{10,15} Each of these conditions can progress rapidly to death, and often more than one is present in the sick neonate,²⁷ necessitating expeditious diagnosis and treatment of existing conditions. **Table 1** describes the diagnosis and treatment of common conditions in the sick neonatal ruminant.

Bacterial septicemia is common in compromised neonatal ruminants, primarily due to inadequate colostrum intake and establishment of immunocompetence. In the neonate, the primary source of bacterial contamination of blood is the intestine due to nonspecific pinocytosis. In older calves, enteritis may cause septicemia through loss of integrity of the intestinal wall. By the second or third week of age, omphalophlebitis, arthritis, pneumonia, and meningitis may result in septicemia.²⁸ Septicemic calves between 2 and 6 days of age present with vague signs such as altered mentation (ranging from mild signs to coma), depression, or decreased suckle response with or without diarrhea. The condition is most often rapidly fatal. Physical exam findings may include hypothermia or hyperthermia, tachycardia, tachypnea, hyperemia or petechiae of mucous membranes, increased capillary refill time, cold extremities, and diminished peripheral pulse. Point-of-care testing is likely to reveal hypoglycemia, metabolic acidosis, hypoxia, and hypotension. Observation of any combination of these signs should lead the practitioner to suspect septicemia and/or meningitis and act quickly. Blood culture before initiation of antibiotics is prudent. Treatment should include fluid therapy and intravenous antibiotics, the choice of which should include activity against both gram-negative and gram-positive bacteria. Commonly encountered pathogens and resistance patterns at the institution should also be considered when choosing antibiotics. Nonsteroidal anti-inflammatory drugs are helpful in modulating the inflammatory response once hydration is corrected. Correction of hypothermia and hypoglycemia should be addressed immediately (see **Table 1**). When treating a hypoglycemic neonate, dextrose may be delivered intraperitoneally if venous access is a challenge due to profound hypotension or dehydration. Check blood glucose frequently during the initial 24 hours of treatment. Inability to correct hypoglycemia despite treatment is suggestive of septicemia.

Table 1 Approach to diagnosis and treatment of common conditions in the sick neonate		
Condition	Diagnosis	Treatment
Hypothermia	94– 99°F (34.4–37°C) = hypothermia Below 94°F = severe hypothermia	Warm neonate slowly (2°F per hour) using dry circulating air, blankets, heat lamps and administration of warmed IV fluids or warmed oral colostrum.
Hypoglycemia	Blood glucose <60 mg/dL	Deliver 500 mg/kg (10 mL/kg 5% solution) dextrose IV over several minutes. Repeated dosing may be necessary to increase blood glucose to an acceptable level. Once corrected, maintain glucose administration at least 250 mg/kg/day until neonate accepts food, or begin parenteral nutrition.
Metabolic acidosis	Base deficit >10 mmol/L Blood pH <7.25	Intravenous sodium bicarbonate (1.3%): • $\text{mEq HCO}_3 = \text{Base deficit} \times \text{body wt} \times 0.5$. • Empirical treatment with 1–2 mEq/kg
Hypoxemia	$\text{SaO}_2 <90\%$ $\text{PaO}_2 <70 \text{ mmHg}$	Nasal insufflation with O_2 5–10 L/h or ventilation
Septicemia	a. Blood culture positive for bacterial pathogens b. Elevated or reduced white blood cell count c. Fibrinogen >500 mg/dL d. >2% band neutrophils	Initiate broad spectrum intravenous antibiotics; consider plasma transfusion; monitor and correct aberrations in body temperature, hydration, acid/base balance, and blood glucose; provide adequate nutrition.

Supportive treatment for neonatal septicemia should include provision of a clean, warm, lowly lit environment; soft, clean bedding; intravenous fluid supplementation; and plasma transfusion, colostrum supplementation, or milk-based nutritional support, depending on the age of the neonate. Bedding should be changed frequently as septicemic neonates are usually too weak to stand and therefore susceptible to urine scald and corneal irritation/ulceration. If the patient is normothermic ($\geq 98^\circ\text{F}$) and responsive, offer a bottle as soon as it appears strong enough to suckle.

Assessing Passive Transfer

Maternal immunity is not transferred in utero in ruminants. Therefore, neonatal ruminants rely on the intestinal absorption of antibodies from colostrum, produced by the dam in the first 24 hours postpartum, for humoral protection from environmental pathogens. Endogenous production of antibodies by the calf does not reach protective levels until at least 1 month of age. While observation of a strong neonate suckling normally from the dam in the first hours after birth is a practical indicator of successful passive transfer, the performance of neonatal physical exams on all calves is a good practice. This exam allows the practitioner to check for medical and congenital conditions, apply an identification tag, supplement vitamin E and selenium if needed, and collect blood for passive transfer testing if maternal care is in question. Detection of IgG in plasma is possible as soon as 2 hours after a colostrum feeding. Therefore, timing of neonatal physical exams may be determined by many factors, such as the mobility

Table 2

Common tests for assessing passive transfer.

Passive Transfer Test	Description	Result Indicating FPT
Sodium Sulfite Turbidity Test	Sodium sulfite causes precipitation of antibodies. Mix 14%, 16% and 18% sodium sulfite. Add 0.1 ml serum to 1.9 mL of each concentration in separate tubes. Mix and observe at 1 hour. Commercially available as a kit; simple and quick; high sensitivity, low specificity ¹⁶	Precipitation or flakes at all 3 concentrations 3+ = >1500 mg/dL. 2+ = 500–1500 mg/dl (partial FPT). 1+ = <500 mg/dL (FPT). 86% accurate in detecting FPT. ²⁹
Zinc Sulfate Turbidity Test	Similar to sodium sulfite test but more susceptible to error due to hemolysis. Add 0.1 ml serum to 6 ml of a 350 mg/mL ZnSO ₄ solution. Cap, mix and observe turbidity at 1 hour.	Turbidity insufficient to obscure newsprint indicates FPT. ²⁹
Serum Total Protein	Simple refractometer test. TP may be affected by dehydration, leading to a false positive result.	<5.2–5.5 g/dL TP indicates FPT. ³⁰
Glutaraldehyde Coagulation Test	Relies on the induction of coagulation of antibodies by glutaraldehyde. Add 1 mL serum to 50 μ L 10% glutaraldehyde and observe clot formation in 1 hour. High specificity, low sensitivity.	Complete clot = >600 mg/dL. Semisolid clot = 400–600 mg/dl. Failure to observe clot formation indicates FPT. ^{29,31}
Serum Gamma-Glutamyltransferase (GGT)	Serum levels of GGT are high following colostrum feeding. Relies on in-house serum testing ability or laboratory time. Best as an adjunct to other tests.	<50 IU/l in domestic calves indicates FPT. ^{16,30} Varies with species, but GGT levels <50X normal adult serum levels are suggestive of FPT.

of the neonate, the observation (or lack thereof) of nursing and the ability to find “hider” species in large pastures. Generally, the behavior of the dam of hider species is a very poor guide to the location of the neonate.

Common tests for passive transfer are listed in **Table 2**. While radial immunodiffusion is considered the gold standard test for passive transfer, it is a species-specific laboratory test and not practical for rapid diagnosis of FPT in nondomestic ruminants. Similarly, an enzyme-linked immunosorbent assay, while useful as a stall-side test for domestic calves, is also a species-specific test and therefore not practical for nondomestic species. Several commercially available tests, as described in **Table 2**, are sufficiently sensitive to rapidly identify FPT to allow for the immediate development of a treatment plan.

Treating Failure of Passive Transfer

In domestic calves, colostrum absorption occurs during the first 24 to 36 hours of life. The timing of this nonselective absorption of macromolecules including IgG ends approximately 24 hours after the first meal.³² Peak Ig transfer across the enterocytes occurs during the first 4 hours postpartum and declines rapidly after 12

hours^{33–35}; therefore early colostrum intake should result in higher IgG serum concentration. Peak serum IgG concentration occurs 32 hours postpartum.¹⁵ FPT occurs when inadequate levels of IgG are absorbed, predisposing the neonate to the development of disease. Even calves nursing from their dams can experience FPT by ingesting an inadequate volume of colostrum during the critical time period or by ingesting colostrum of relatively low IgG content. FPT calves experience higher rates of neonatal mortality, and this higher mortality rate can extend to the postweaning period.¹⁶

The decision to treat a calf with FPT and the method of treatment should be based on several factors including the animal's age, species, value, and environment and the availability of colostrum, plasma, and other resources. Calves suffering from FPT are at greater risk for developing disease but can survive if placed in a clean environment with low exposure to infectious pathogens. Immunocompetent calves are able to produce approximately 1 g of IgG per day.^{35,36} Despite this, they cannot respond to certain antigens, such as gram-negative bacteria, until 30 days of age.³⁷ The prophylactic use of broad-spectrum, parenteral antimicrobials in calves with FPT should be considered but must be combined with colostrum replacement and management practices that minimize pathogen exposure.¹⁶

FPT calves less than 24 hours of age can be treated orally with fresh colostrum, commercial colostrum replacer, or plasma. It is very difficult to collect a volume of colostrum sufficient to prevent FPT from a lactating nondomestic ruminant. Bovine colostrum is an alternative, but there is an inherent risk of disease transmission (eg, Johne disease) associated with this practice. Commercial colostrum replacers are therefore recommended for neonatal nondomestic ruminants. Serum contains significantly lower IgG concentrations than does colostrum and is thereby not a good choice for oral administration. Commercial hyperimmune plasma, however, is a viable alternative. Oral administration should be attempted first with a bottle, but weak calves or those that do not take readily to a bottle should receive colostrum via oesophageal intubation (see below) or parenteral plasma transfusion.

During the first 24 hours of life, colostrum should be fed at a volume equal to 10% of the neonate's body weight divided over several feedings. Higher volumes should be fed (up to 15% body weight) if the source is poor quality colostrum or plasma. Ideally, as much colostrum as possible should be fed within the first 4 hours to maximize absorption of IgG. In domestic calves, 100 g colostrum IgG delivered in the first feeding is associated with lower rates of FPT.³⁸ When using a colostrum replacer purchased as a powder, the actual IgG dose fed can be varied by mixing the powder with different volumes of water. However, while making a very concentrated formula may seem logical, this may not be palatable to the calf. A recent study in springbok (*Antidorcas marsupialis*) calves has shown that using a commercial bovine colostrum replacer at a dose of greater than 4.68 g/dL of IgG per kg of body weight divided into 5 feedings over 24 hours resulted in passive transfer rates that were comparable to calves consuming maternal colostrum (Lamberski, personal communication, 2011). Colostrum can still be administered orally beyond 24 hours of age, as intraluminal colostrum provides local protection.

When colostrum is not available, plasma can be administered orally or parenterally in calves less than 24 hours old, or parenterally after 24 hours of age. Plasma is typically administered at a rate of 20–40 mL/kg IV. Whole blood can be administered instead of plasma, but the volume should be increased to account for the presence of red blood cells. If plasma or whole blood is unavailable, the use of commercial plasma is an option. Commercially available equine plasma has been used successfully in nondomestic ruminants to prevent FPT.³⁹ Plasma can also be administered

intraperitoneally (IP). This procedure is less invasive and intensive than IV administration and allows the calf to be returned to the dam in a shorter period of time. While the recommended plasma dose for intravenous administration is 40 ml/kg, IP administration may require a larger volume of plasma. To administer plasma IP, an area in the left paralumbar fossa is clipped and surgically prepared. A needle is inserted through the skin, abdominal musculature, and peritoneum in the center of the paralumbar fossa. Alternatively, local anesthesia can be used before making a stab incision and then inserting an 18 gauge \times 2 inch over-the-needle indwelling catheter. Plasma can be delivered at a relatively rapid drip rate.¹⁶ Calves should be monitored for adverse reactions (lethargy, fever, tachypnea, tachycardia) during parenteral plasma administration regardless of the route, and those receiving plasma from an animal anesthetized using opioid anesthetics may benefit from naltrexone administration during plasma administration.

Despite the method of treatment, tests for FPT should be repeated following treatment and the animal retreated if adequate passive transfer is not confirmed.

LONG-TERM CARE AND SOCIALIZATION

Nutrition

Neonatal ruminants requiring treatment are most often, as mentioned above, raised in the absence of their dam, necessitating hand-rearing. Even if a calf is allowed to remain with the dam, supplemental feeding may be necessary. In some cases, neonates can be returned to the natal group following a period of stabilization and training to ensure reliable consumption of formula from a bottle. This approach has the added benefits of optimal socialization and minimal staff time.

Milk composition is influenced by genetics, nutrition, and environmental factors, and the components vary substantially between species.⁴⁰ Therefore, it is not possible to recommend a hand-raising formula that is suitable for all nondomestic ruminant species. A formula's composition should mimic that of the dam's milk in protein, carbohydrate (lactose), fat, and total solids ratios. Many commercial milk replacers are available but the authors have experience with LAND O LAKES Doe's Match (Land O'Lakes Animal Milk Products, Shoreview, MN, USA)^a and Zoologic Milk Matrix (PetAg, Inc., Hampshire, IL, USA).^b Goat's milk is often used and is a good choice for many species, used alone or in combination with a milk replacer. However, unless fortified, goat's milk does not contain vitamin E and is relatively low in zinc and copper. Most milk is naturally low in iron. For these reasons, a vitamin and mineral supplement is often added to the formula. Adding probiotics to the formula may be beneficial in establishing a healthy rumen flora.

In the first 2 to 3 weeks of a ruminant's life, termed the preruminant phase, digestion of milk occurs in the abomasum and small intestine. During this phase, milk deposited into the nonfunctional rumenoreticulum (eg, via tube feeding) is not digested and often leads to rumenitis and septicemia.⁴¹ Reflex closure of the esophageal groove, stimulated by suckling, normally prevents this deposition. If tube feeding is required during this time, the tube can be passed to the mid-esophagus to stimulate swallowing and closure of the groove. Additionally, milk feeding can be preceded by oral administration of 10% sodium bicarbonate or 2–5% copper sulfate, which facilitates groove closure for several minutes.⁴² Esophageal feeding should be conducted using gravity flow.

^a<http://www.lolmilkreplacer.com/KID/DoesMatchMilkReplacer/default.aspx>.

^b<http://www.petag.com/industry/zoologic-formulation-mixing/>.

During the transitional phase, which begins at 2 to 3 weeks of age, the calf begins to take in small amounts of dry feed and the volatile fatty acids butyric and propionic acid stimulate differentiation of the ruminal papillae.⁴³ By this age, the neonate should be provided with a variety of appropriate hay, pelleted ration, and calf supplements to begin to sample, in addition to the milk ration. From initiation of solid feed through weaning, sand or dirt ingestion is common in some areas. Sand ingestion can be due to formula imbalances, gastrointestinal diseases, stress, or boredom, but determining the exact etiology is often difficult. Impaction can result as can the need for medical and/or surgical intervention. Since ingestion can lead to impaction, it is best recognized early as a symptom of an underlying problem. The calf should be housed away from sand or dirt while the etiologies are explored, and it may be necessary to attempt formula changes in lieu of a diagnosis as the exact imbalance may not be obvious. Early weaning may be considered in some cases.

Abnormal fecal consistency (liquid, loose, log-shaped, or clumped stool) is not uncommon during the first few months, particularly if the animal is being hand-reared. There are many etiologies for changes in fecal consistency, ranging from formula intolerance to enteritis, necessitating close monitoring of fecal output and consistency until weaning. The transition from preruminant to ruminant and the weaning process both result in changes in gastrointestinal flora and, often, fecal consistency. High-carbohydrate feeds or an overconsumption of carbohydrates can result in clostridial overgrowth and diarrhea. As in domestic ruminants, nondomestic ruminants may benefit from vaccination against *Clostridium perfringens* type C and D at 4 and 8 weeks of age.

The weaning process is often initiated at 2 months of age but is not complete until 3 to 4 months of age. This varies with species and health status and can be accelerated in the interest of returning the animal to its herd, or if formula intolerance is a concern. Conversely, the weaning process can be slowed considerably if an animal has difficulty accepting solid food.

Socialization

Ruminants that are hand-raised need to be socialized before introduction to their herd. If animals will remain in the collection, plans to reintroduce the animal to the herd should be in place by the end of the weaning period. It is ideal to hand-raise nondomestic ruminants with conspecifics or other nondomestic ruminants of similar age. Herd-adapted species need the psychological benefits that result from companionship. They also need a playmate to ensure adequate bouts of activity and exercise. If another hand-reared animal is not available, an older, calm "aunt" is a suitable substitute. Using an older animal has the added benefit of exposing the hand-reared animal to normal fecal flora. Before reintroduction to the herd, it is beneficial to first introduce it to a companion animal to reduce the chance of rejection by the herd. Once bonded, the pair can be introduced together. This is particularly valuable when the animal was raised with a species other than its own.

In some cases it is of benefit to raise young ruminants without imprinting them significantly on humans. For instance, hand-reared male elk and other species imprinted on humans can be very dangerous as adults. A plan to reduce imprinting should be in place within the first few days of life and continue until weaning and complete introduction to the herd. **Fig. 1** depicts a device for hand-rearing that minimizes exposure to human presence and scent. A framed box large enough for a person to stand in is covered with tarp or heavy cloth. The front of the box consists of a 2-way mirror and 2 hand-sized ports for feeding the calf. The handler



Fig. 1. An approach to hand-rearing that minimizes social imprinting on humans. The handler enters the box from the rear and views the neonate through a two-way mirror, feeding through ports in the front of the box.

enters the box from behind, avoiding being seen by the calf, and carries the box toward the calf. Alternatively, the calf can be trained with a clicker to approach the box. The handler uses leather gloves that may be covered with conspecific female urine to encourage appropriate scent recognition and feeds the neonate with a bottle using the ports in the front of the box, avoiding exposure of the arms. The 2-way mirror allows the handler to see the neonate, but the neonate sees itself. This type of device has been used in combination with early weaning and reintroduction to the herd to raise orphaned male ungulates to be socially normal herd members as adults.

SUMMARY

Management and care of the nondomestic ruminant neonate are similar in principle to domestic animal practice. Housing of the dam, conditions for birth, preparation for intervention, and plans for treatment and hand-rearing of sick neonates must all be considered carefully before undertaking nondomestic ruminant breeding. Unfortunately, neonatal losses tend to be much higher in nondomestic calves before weaning than in domestic cattle, sheep, and goat herds.¹ With continued habitat and population declines in wild species, successful captive breeding of nondomestic herds becomes more important to species sustainability and potential reintroduction programs. The primary challenges contributing to neonatal losses in nondomestic ruminants are often animal temperament and adaptation to captivity. Only through experience can some of these challenges be overcome. However, by understanding some species-specific behavioral tendencies and the fractious nature of nondomestic ruminants in

general, we can improve our success in managing and maintaining healthy populations of nondomestic ruminants in captivity.

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