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CHAPTER 25

Lactation and Neonatal Care

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An epidemiologic study in the United Kingdom reported that 4% to 11% of the deaths among llamas and 17% to 33% of deaths in alpacas occur during the first 6 months of life. A high proportion of these deaths occur within the first week of life.[1] In South America, cria mortality rates before weaning may reach 50%, mostly caused by *Clostridium perfringens* (types A and C).[2] As in other domestic animal species, most neonatal deaths are associated with events occurring during parturition and the immediate postpartum period such as dystocia, poor mothering ability, or exposure. Poor mothering ability includes not only behavior problems such as rejection but mostly poor milk production. Neonatal care and early recognition and treatment of neonatal ailments are of the utmost importance to reduce these losses.

Prenatal Care

Care of the neonate begins during the long gestation as strength and viability of the newborn is greatly influenced by health of the dam.[3] More than 80% of fetal growth occurs in the last trimester of pregnancy, and the normal rate and growth pattern of the fetus may be altered by numerous factors, including abnormal hormonal environment, nutrition, genetics, and infectious conditions.[3,4] These factors may cause chronic or acute disruption of development of the fetus, resulting in a stillborn or dysmature neonate without any obvious clinical problems in the dam. Taking these factors into consideration, prenatal care of the dam with proper nutrition, vaccinations, and knowledge of any concurrent herd health problems is important to enhance survival of the newborn. All these aspects have been discussed in detail in other chapters in this text.

Preparation for parturition is crucial. Frequent observation of the dam in the last week of pregnancy is the only way to determine the approaching time of parturition and take measures to ensure a healthy birthing environment and immediate intervention, if needed. Close-up females should ideally be placed in a clean grassy paddock or moved into a clean barn with the facility for easy observation. A clear emergency protocol and communication channel should be established between the breeder and the attending veterinarian. It is preferable not to change the social organization of the periparturient female or move it during the first stage of labor.

Postnatal Care

Even with an optimal uterine environment, the newborn is subject to severe stresses and some degree of oxygen deprivation during parturition. During parturition, it is possible that the neonate may be affected by a damaging degree of anoxia. Several mechanisms help the neonate to adapt to extrauterine life. The first is an increase in fetal cortisol concentration, which triggers parturition and allows adequate levels of surfactant to be produced by type II alveolar pneumocytes. In addition to elevated cortisol concentration, a catecholamine surge occurs. A negative side effect of this physiologic feature is that it may allow potential problems to be masked and the newborn to appear normal immediately after

parturition, even though it may have substantial physiologic impairment.[5]

Early diagnosis and aggressive treatment of neonatal disease, particularly infections, results in greater positive outcome.[6] The clinical signs are often nonspecific and vague, which results in a neonate that is slow to adapt to extrauterine life or that dies suddenly in the first few days of life. If an intrauterine infection exists, the fetus may be born alive or may die in utero. Intrauterine infections of bacterial origin in the newborn camelid are recognized more commonly compared with viral infections. Infections acquired in utero rather than in the postpartum period should be suspected if the newborn has elevated plasma fibrinogen in the first 12 to 24 hours of life, the placenta appears abnormal, or the dam exhibits vaginal discharge during parturition.[6,7]

Normal Behavior of the Newborn Cria

Human intervention on the newborn should be as limited as possible to avoid disturbing the establishment of the dam–cria bond. However, the cria should be monitored from a distance during the period of adjustment to its new environment.

The newborn cria should be evaluated within the first few hours of life for any abnormalities of development or maladjustment to extrauterine life. Physical and behavioral parameters of the normal newborn camelid are presented in Table 25-1. Assessment of the newborn cria includes evaluation of the epidermal membrane, respiration, heart function, and presence of obvious congenital abnormalities. The remaining epidermal membrane should be removed with clean towels. It is normally translucent and may become yellow or brownish because of meconium staining in cases of fetal stress (fecal passage caused by dystocia). Ideally, the placenta should be weighed and examined thoroughly for completeness and signs of inflammation or infection, as described in the chapter on postpartum disorders in this text (Chapter 26, Postpartum Disorders). Clients should be instructed to keep the placenta refrigerated for any subsequent diagnostic examination, as needed.

The cria should be weighed daily to monitor weight gain accurately. Regular weighing of the cria (daily for the first 2 weeks and once every other week thereafter) is warranted to determine the adequacy of milk production and intake. Birth weights vary significantly from one farm to another and reflect most likely the feeding management on that farm as well as genetic influence (Table 25-2). A tendency toward higher birth weight than originally described has been observed in many alpaca farms in North America. In alpacas in South American conditions, birth weights have tended to increase with increasing age of the dam, and low birth weights have been associated with an increased risk of neonatal death.[8] Birth weights tend to decrease after 11 years.[8] No studies have been performed on the effect of the sire on birth weight in these species. It is not uncommon for crias to lose some weight in the first 24 hours (120 to 250 grams [g] in alpacas and 250 to 500 g in llamas). In alpacas, the average daily weight gain in crias is 195 g under grazing conditions in South America.[3]

Disinfection of the umbilical cord is a significant line of defense against infection. If the birth is observed to have occurred in a clean birthing area, the umbilicus should be trimmed and dipped in 2% to 3% tincture of iodine. If the birth did not occur in a clean area, it is advisable to clean the umbilicus with diluted (0.5%) chlorhexidine solution or soapy water and to dip or spray the umbilicus with regular-strength betadine solution. Umbilical cord dipping should be performed two to three times in the first 24 hours. It is important to protect the newborn cria from low or high temperatures.

After the initial evaluation, the newborn should be monitored from a distance. The dam's udder could be cleaned with a warm wet towel and the teats examined for any abnormalities and stripped to unplug them. The mother–cria bond is established through humming, nose-to-nose touching, and nuzzling of the cria. If the newborn does not get up and nurse by 3 hours after birth, a problem should be suspected, and intervention is needed.

TABLE 25-1 Biologic Parameters of Normal Healthy Newborn Camelids

Parameter	Reference Range?
Birth weight (kilograms)	5 to 11 alpacas (*Vicugna pacos*) 9 to 18 llamas (*Lama glama*)
Temperature (°C)	37.7 to 38.9
Pulse (beats per minute [beats/min])	60 to 100
Respiration (beats/min)	10 to 30
Time to standing (minutes)	30 (10 to 120)
Time to nursing (minutes)	45 (20 to 180)
Nursing frequency	Several sessions per hour lasting 1 to 3 minutes
Meconium passage (hours)*	<18 hours
Urination (hours)	<18 hours
Daily gain Kg (first 3 months of life)**	0.2 to 0.4 (alpaca), 0.4 to 0.8 (llama)

*Meconium passage should occur about 8 hours after a normal feeding.
**Most neonates will register a slight drop in weight in the first 24 hours.

TABLE 25-2 Variation of Birth Weight in Normal Alpaca Crias from a Herd in the United States

	N	Mean (kg)	SD	Minimum (kg)	Maximum (kg)
Females	49	7.76	1.27	3.76	11.25
Males	64	8.12	1.04	5.26	10.61
Total	113	7.98	1.18	3.76	11.25

kg, Kilogram; *N*, number; *SD*, standard deviation.
From Tibary A, et al: Neonatal care and neonatal emergencies in camelids. In *Proceeding of the Annual Convenetion of the American Association of Bovine Practitioners*,Charlotte NC ?, 177-184, 2008.

Evaluation of Passive Transfer of Immunity

The epitheliochorial microcotyledonary placenta of camelids does not allow passage of immunoglobulins from the dam to the fetus. Therefore, newborn camelids are born hypogammaglobulinemic and rely on passive transfer of immunity through colostrum intake.[9] Increased incidence of illness and death from infections in neonates is associated with inadequate passive transfer of immunoglobulin as measured by low serum immunoglobulin concentrations in sick or dead neonates.[10] Conversely, successful immunoglobulin transfer is associated with low infection rates and the high likelihood of survival. Even though this is true, great differences exist among farms with regard to immunoglobulin concentrations in healthy neonates. The effect of between-farm variations, which could involve factors such as management practices, including biosecurity measures, nutrition, vaccination programs, current pathogen history, herd bloodlines, geographic location, and climate, would enable a neonate with failure of passive transfer to remain healthy.

A relative paucity of research is seen in the area of colostrogenesis in camelids. Concentration of immunoglobulin G (IgG) in mammary secretion is 8 to 10 times higher than that in serum about 3 weeks before parturition.[11] However, data indicate that camelids do not selectively transfer immunoglobulin (specifically IgG_1) from serum to the mammary gland prior to parturition.[11] Although Bravo et al. have suggested that periparturient camelids may produce IgG in the mammary gland, this has not been fully investigated. The concentration of IgG drops quickly in mammary secretion after parturition.[11]

Colostrum production and quality depend on the normal preparation of the mammary gland to concentrate antibodies during pregnancy and the ability of the dam to lactate after birthing. The quality of colostrum depends on several factors, including the immune status of the dam and udder health. In many situations, either colostrum production or quality may be compromised, putting the newborn camelid at risk of contracting infections in the first weeks of life.

Intake of colostrum may be hindered by factors associated with the dam or the cria. Dam factors include primary causes such as poor colostrogenesis or secondary causes such as painful conditions or stress preventing intake by the cria. Agalactia or poor mammary gland development is frequently seen in young females or following hormonal treatment during pregnancy. Fescue toxicosis has been incriminated in agalactia but has not been scientifically verified. Secondary causes of agalactia or poor mothering ability include severe mammary gland edema, mastitis, and pain following severe dystocia or cesarean section and unfamiliar environment. Colostrum intake may also be reduced by the cria's inability to suckle because of weakness (difficulty standing), congenital defects (cleft palate, swollen tongue caused by dystocia), or abnormal teat conformation.

Absorption of colostral antibodies by the neonatal gastrointestinal tract is possible for only a short period and decreases significantly after 18 hours of life. In normal conditions, serum IgG levels of crias are usually above 2000 milligrams per deciliter (mg/dL) by 24 hours of life. Recommendations for colostrum intake in camelids are extrapolated from other species; accordingly, neonates should receive good-quality colostrum at a rate of about 10% of their body weight in the first 12 hours with preferably half consumed in the first 6 hours. However, many factors such as cold weather or heat stress, prematurity or dysmaturity, and metabolic disturbances may negatively affect absorption of immunoglobulin.

A strategy for colostrum supplementation should be immediately instituted upon discovery of any problem with regard to colostrum production or intake. The best approach is to provide colostrum from the same species. If this is not possible, the second best choice is goat colostrum.[12] It is important that the source of noncamelid colostrum be free from major infectious diseases (i.e., bovine viral diarrhea virus [BVDV], Johne disease, brucellosis, etc.). Cattle colostrum is the second best choice for supplementation in crias. Powdered colostrum supplements are available for some species but have not been critically evaluated in camelids.

Establishment of a colostrum bank is a possibility for camelids. However, it is important to realize that the IgG concentration drops abruptly after the first two milkings. It is highly recommended that alpaca and llama breeding operations be prepared for a possible need for colostrum supplementation by having a stock of frozen caprine or bovine colostrum. Veterinary clinics can play a role in colostrum banking for their clients. Colostrum should be collected from healthy dams and stored properly. Our recommendation is to store colostrum in individual feeding units (60 to 90 milliliters [mL]) using regular freezer bags or freezer bags for human breast milk. All bags should be labeled with identification of the dam and date of collection. Ideally, colostrum should be frozen at –20°C or in a scientific freezer. Colostrum should not be frozen in a frost-free freezer. Adequately prepared and frozen colostrum keeps its properties for at least 1 year. Frozen colostrum should be thawed out slowly and brought up to body temperature before administration. Rapid thawing at high temperature or in a high-power microwave is not good because it may denature the antibodies in colostrum. Thawing is best accomplished by placing small quantities of colostrum-containing bags in a water bath at body temperature.

As a general rule, colostrum administration should be started immediately in cases of agalactia or mastitis or if the newborn has not been seen suckling by 2 hours after birth. Crias should receive 10% of their body weight in the first 12 hours of life divided into meals every 2 hours. This usually represents about 60 to 90 mL every 2 hours. Some practitioners prefer to give 5% of body weight in a feeding followed by another feeding of equal quantity 6 to 8 hours later because small intake volumes may accelerate gut closure for colostrum. The best way to administer colostrum is by bottle-feeding. In case of poor suckling reflex, colostrum may be administered via orogastric intubation (24-French). Care should be taken not to place the tube in the respiratory tract (trachea). The tube should be felt by palpation as it goes down the left side of the neck. It should be kept within the esophagus to provide closure of the esophageal groove and avoid depositing milk within the first stomach compartment, which would increase the risks of fermentation. Repeated tube feeding may cause esophagitis.[13]

Poor IgG status of the newborn is the most important risk factor for neonatal infectious diseases. Most insurance companies require determination of the passive immunity status for

crias. The concentration of IgG increases rapidly after colostrum ingestion and reach a peak between 24 and 48 hours of age. Therefore, determination of IgG levels in serum is ideally performed 24 to 36 hours after birth. Methods used for evaluation of passive transfer of IgG include serum radial immunodiffusion (SRID), serum total solids measurement using a refractometer, serum total protein, globulin concentration, and sodium sulfite turbidity test.

SRID is the gold standard for evaluation of passive immunity status in the newborn alpaca and llama. This technique is quantitative, specific, and highly accurate.[14–16] Since the SRID is species specific, it will not be accurate, if colostrum other than that of camelids was used. Using Triple J Plates (Triple J Farms, Bellingham WA) levels greater than 800 mg/dL are considered adequate, and levels less than 400 mg/dL are considered an indication of failure of passive transfer. However, most studies consider 1000 mg/mL to be the minimum cutoff value for adequate passive transfer.[14,15]

Failure of passive transfer should be suspected if total solids determined by refractometer are less than 4.5 g/dL, whereas values greater than 5.5 g/dL are consistent with adequate passive transfer.[14] However, others have found that this test is not very accurate.[17] If samples are taken at birth and compared with samples at 24 hours, the packed cell volume (PCV) will be observed to be reduced and the total solids increased, indicating that the cria is more hydrated and that the rise of total protein is caused by significant passive transfer. Serum total protein and globulin should be at least 5.5 g/dL and 2 g/dL, respectively.[17] However, it is important to remember that these parameters may be falsely increased in case of dehydration. Commercial sulfate turbidity tests are available but not as sensitive as the SRID.[14]

Prophylactic Treatments of the Newborn

The most common infections of newborn camelids in the first few days of life are clostridial diseases caused by *Escherichia coli* and coronavirus. Clostridiosis caused by *Clostridium perfringens* type A has been reported to cause severe losses among some populations of crias.[18,19] Protection of the cria against these infections is provided by adequate ingestion of colostrum and absorption of IgG from a vaccinated dam. Although several practitioners prefer not to vaccinate pregnant females, we have not seen any problems with administration of *C. perfringens* type C and D and *C. tetani* vaccine in the last 6 weeks of pregnancy. Administration of clostridial antitoxin should be considered if the cria did not have adequate passive transfer. Vaccination of crias in their first week of life has been proposed by some authors. Autologous vaccines may help reduce the effects of *C. perfringens* type A.

Recently, alpaca breeders have been giving bovine modified live vaccines against coronavirus orally in the first 24 hours of life. This strategy has been reported to be effective in controlling coronavirus outbreaks, probably because of the close genetic relationship between the strains isolated from alpacas and those from cattle.[20] The use of these vaccines in pregnant camelids to enhance colostral protection has not been fully investigated in alpacas and llamas. Abortion has been reported following parenteral use of these vaccines.

Crias should receive vitamins A, D, and E, as well as selenium, in areas where needed. Crias that do not receive vitamin D supplementation have reduced growth rate during winter and may show clinical signs of rickets if born in winter.[21] If local conditions justify, a dose of 1000 international units (IU) of D_3 per kilogram of body weight subcutaneously has been suggested for crias in late autumn and again in midwinter and to adult females in midwinter to prevent vitamin D inadequacy.[22]

It has become a standard practice in North America to test all newborn SACs for BVDV (polymerase chain reaction [PCR] test) for early identification of persistently infected (PI) crias so that they can be separated from the herd.[23–25]

Identification of High-Risk Neonates

Early identification and intensive care of at-risk crias is very important, as many will develop failure of passive transfer and septicemia.[6–9] Factors for high risk in neonates include dams with previous problems, prematurity, dystocia, prolonged obstetric manipulations, delivery by cesarean section, placentitis, premature placental separation, and prolonged pregnancy.

Premature birth may be a consequence of severe illness in the dam (i.e. severe respiratory syndrome) or uterine pathology (i.e., placentitis or placental insufficiency).[3] Premature camelids display specific phenotypic characteristics. These include a birth weight significantly (more than 20%) lower than average for the farm and a thick epidermal membrane firmly attached to the footpads and the mucocutaneous junctions (Figure 25-1). "Floppy" syndrome is often seen in premature camelids and is characterized by inability to rise, hold the head up, or maintain sternal recumbency, as well as floppy ears caused by immaturity of the cartilage (Figure 25-2). The coat appears silky, and the limbs are overextended at the carpus and fetlock because of laxity of tendon and poor muscle tone. The incisors are not erupted, and a lack of or poor suckling reflex is seen (Figure 25-3).

Excessively long gestation (>370 days) has been associated with increased risk for dysmaturity and neonatal deaths in the first 3 days of life in our experience. These neonates usually are presented with similar biophysical characteristics as the premature neonates except that they may have normal body development.[26]

Emergencies in First 24 Hours of Life

Emergencies seen in the first 24 hours of life are often associated with dystocia, abnormal pregnancy, failure of passive transfer, congenital abnormalities, digestive (meconium retention) or urinary (urine retention) problems, environmental thermal exposure or malnutrition. Sepsis is a major concern in neonates particularly in the first 10 days of life. Predisposing factors for sepsis are similar to those described in other species, namely prematurity, placentitis, and/or contaminated or crowded birthing facilities, dystocia, failure of passive transfer and lack or improper care of the umbilical stump. In one study the median age at presentation of crias with sepsis was 2 days.[6] Presence of hypopion, uveitis, and/or conjunctivitis indicate early septicemia. Septic neonates may also develop respiratory problems, diarrhea, meningoencephalitis and septic arthritis.[5,6] The most common isolates include *Escherichia coli, Enterococcus spp., Listeria monocytogenes, and Citrobacter spp*. The

Figure 25-1 Premature Crias with Thick Epidermal Membrane. A, Adhered to the mucocutaneous junctions of the lips. **B,** Adhesions of the epidermal membrane to the foot pad. **C,** Thick epidermal membrane. *(Reprinted with permission, Tibary A and A Anouassi A, Theriogenology in* Camelidae*, second edition, Actes Editions, Institut Agronomique et Veterinarie Hassan II, Rabat, Morocco.)*

Figure 25-2 Premature cria delivered by cesarean section; note the non-erect (floppy) ears. *(Reprinted with permission, Tibary A and A Anouassi A, Theriogenology in* Camelidae*, second edition, Actes Editions, Institut Agronomique et Veterinarie Hassan II, Rabat, Morocco.)*

antibiotics of choice for camelids at high risk of sepsis include the following combinations: Enrofloxacin and PPG, Enrofloxacin and ceftiofur, ceftiofur and gentamicin.[6,7,9] Gentamicin should be used with care, as it can be extremely nephrotoxic to severely dehydrated newborn camelids or if there is already evidence of renal dysfunction. Crias presented with anemia should be checked for *Mycoplasma hemolamae* infection.[27] The outcome of medical management of septic neonates is generally favorable if there is no gastrointestinal or central nervous system involvement.[6] Total or partial parenteral nutrition should be considered in severely depressed crias that are unable to nurse.[28,29]

Failure of Passive Transfer (FPT)

Failure of passive transfer is a major cause of neonatal mortality in camelids.[10,15,16] In cases of FPT hyperimmune warm (37°C) plasma should be given IV or intraperitoneally at a dose of 15 to 25 ml/kg. A rate of 100 to 200 mL/hr. has been suggested for IV administration in crias. However faster rates have been anecdotally reported by practitioners. The volume of pre-warmed plasma to be administered varies from 300 mL for alpacas to 500 mL for llamas. Crias that are weak or unable

Figure 25-3 **Premature Cria. A,** Lack of incisors eruption. **B,** Tendon laxity.

TABLE 25-3 Common Congenital Defects That May Have a Genetic Basis

System	Defects
Musculoskeletal	Choanal atresia, hernias, angular limb deformities, polydactyly, syndactyly, crooked tail, choanal atresia, campylognathia ("wry face"), cleft palate, dwarfism, jaw misalignments
Visceral	Atresia ani, atresia coli, ventricular septal defect
Reproductive	Segmental aplasia of reproductive organs: segmental aplasia of the uterus (uterus unicornis), epididymal cysts; gonadal hypoplasia: ovarian and testicular, testicular cysts, persistent frenulum, varicocele, supernumerary teats, teat agenesis
Sensorial	Cataract, blue eyes (deafness), nasolacrimal duct aplasia, gopher ears

to suckle should receive colostrum even if they have received plasma transfusion.

Orphaned Crias

Crias may be orphaned or simply may require hand raising because of lack of milk production or very rarely dam rejection. Goat or cow milk has been used effectively as a replacement. Some practitioners advise adding 15 mL of plain yogurt to 240 mL of cow milk to increase the caloric density. For long-term feeding, it is preferable to use goat or goat milk replacements. Human contact with crias, particularly males, should be limited to avoid subsequent abnormal behavior (i.e Berserk syndrome).

Congenital Abnormalities

Many congenital abnormalities have been described in alpacas and llamas and seem to occur more frequently in these species than in other domestic animals (Table 25-3).[30–34] The most common and potentially lethal congenital abnormalities that affect camelid neonates are choanal atresia, atresia ani or coli, atresia vulvi, severe heart defects, and severe urinary system abnormalities. Most commonly, affected animals will suffer from severe respiratory, circulatory, or metabolic complications. Heart defects (i.e., ventricular septal defect [VSD], tetralogy of Fallot [ToF]) may be very severe and lead to death of the cria within a few hours, but most will survive for a few days to months, with the only abnormality being failure to grow normally. We have observed episodes of syncope after exercise in crias with severe heart defects. Several factors, including genetics, teratogenic viral infections, or teratogenic drug or plant intoxication, as well as abnormal uterine environment, may contribute to the development of congenital abnormalities. Often the practitioner is faced with four main questions: (1) Is the defect genetic? (2) Is it lethal? (3) Will the defect affect the welfare of the animal? (4) Will the defect be exacerbated by reproduction (i.e., pregnancy)?[34] Unfortunately, available data do not always answer these questions. It is essential that clients be educated about the necessity to perform a necropsy on stillborn or dead neonates to determine exactly the cause of the death, if possible. In many cases, more than one abnormality is found to be present. Without proper diagnosis, no action can be taken to understand the genetic nature of some of these abnormalities.

Maxillofacial Abnormalities

Choanal atresia is a defect characterized by absence of communication between the nasal and pharyngeal cavities. It may be unilateral or bilateral and partial or complete. The condition is suspected if the newborn has some respiratory difficulties, particularly when attempting to feed. In complete bilateral choanal atresia, the cria may demonstrate severe dyspnea and open-mouth breathing. Verification of air flow can be achieved by placing a dry glass slide or a mirror under the nostril and checking for fogging. Another option is to pass a soft Foley catheter up each nostril to detect any obstruction at the level of the medial canthus. Diagnosis is confirmed by mouth-to-nose artificial breathing or by using contrast radiography after

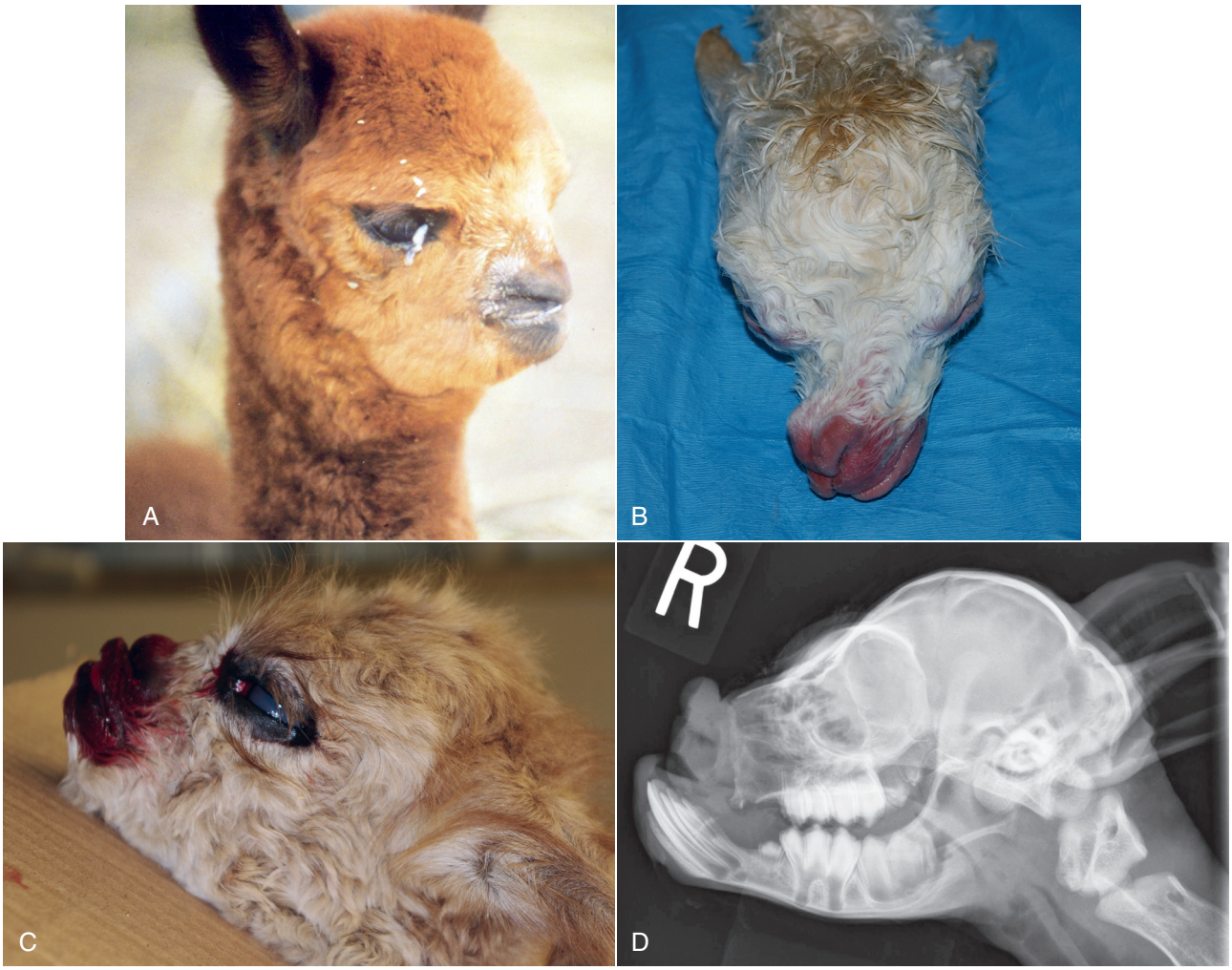

Figure 25-4 Maxillofacial Defects. A and **B,** Campylognathia (wry face) crias. **C and D,** Severe maxillofacial defects, palate agenesis. *(Reprinted with permission, Tibary A and A Anouassi A, Theriogenology in* Camelidae*, second edition, Actes Editions, Institut Agronomique et Veterinarie Hassan II, Rabat, Morocco.)*

injection of a radiopaque substance in the nasal cavity or by using computed tomography of the head to demonstrate the presence of a membranous or osseous separation between the nasal and pharyngeal cavities at the level of the choanae.[35–37] Surgical correction of choanal atresia has been attempted, but euthanasia should be considered because of the high rate of complications and their effects on quality of life. Maxillofacial agenesis or dysgenesis ("wry face") may be associated with choanal atresia as part of a complex genetic disorder (Figure 25-4).[38]

Gastrointestinal and Abdominal Abnormalities

Abnormalities of the gastrointestinal tract include segmental aplasia (atresia ani or coli) as well as hernias. Atresia ani and atresia coli result in the blockage of the intestinal transit and accumulation of fluid in the gastrointestinal tract (Figure 25-5). The neonate may have normal activity and appearance in the first few hours but becomes progressively bloated and depressed after feeding. Ultrasonographic and radiologic examinations of the abdominal cavity confirm the diagnosis. Atresia coli may be mistaken for meconium retention. In the female, these abnormalities may involve the genital tract. Surgical correction of atresia ani has been described.[39]

Umbilical hernias could occur as an emergency in the immediate postpartum period. Small umbilical hernias (less than 2 cm) tend to resolve spontaneously with advancing age. Larger hernias should be corrected surgically if they do not respond well to management with bandage to avoid any complications.

Congenital diaphragmatic hernias may cause sudden death or severe respiratory distress immediately after birth. We have seen several cases of abnormalities of the respiratory system or diaphragmatic hernias that have been mistakenly "diagnosed" by the owner as choanal atresia.

Cardiac Abnormalities

The most common heart defect in alpacas and llamas is VSD. Other more severe defects that have been described include auricular septal defect, ToF, transposition of the great vessels, persistent right aortic arch, and patent ductus arteriosus.

Skeletal Defects

Skeletal defects are generally non–life threatening. They are dominated by angular limb deformities. The most common abnormality is carpus valgus. Less severe cases will improve with time. Severe cases (>10 degrees) require surgical correction.[40,41]

Polydactyly (additional digits) in alpacas is presumed to be caused by an autosomal dominant gene (Figure 25-6). Syndactyly (fused digits) is also thought to be hereditary (Figure 25-7).

Vertebral defects may be lethal (spina bifida) or may severely compromise the cria's ability to feed. Various sacrococcygeal abnormalities have been described in the offspring of a male with a coccygeal defect (kinked tail).[42]

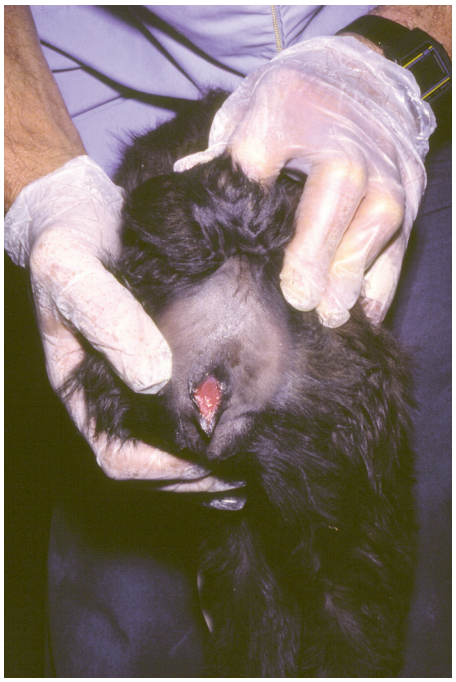

Figure 25-5 Atresia ani.

Defects of the Urogenital System

Defects of the urogenital system include lethal defects such as bilateral renal agenesis, bladder agenesis, and complete vulvar opening aplasia (lethal if not detected early).[43] Vulvar aplasia is easy to correct surgically; however, it is important to rule out other malformations such as ectopic ureters that may be associated with it. Other abnormalities that may be detected in crias include ambiguous sexual differentiation and hypospadia in males. Congenital abnormalities of the mammary gland are abnormal conformation of the teat (partial separation) and supernumerary teats (polythelia). Supernumerary teats may be associated with functional glandular tissue (Figure 25-8).

Abnormalities of Sensory Organs

Several ocular and ear abnormalities include cataracts, deafness associated with blue eyes, nasolacrimal duct aplasia, and gopher ears (Figure 25-9).[33,44] Congenital blindness associated with different ocular defects impact neonate behavior and wellness.[45–47]

Meconium Impaction

Meconium passage is usually observed within 18 to 24 hours after birth. In normal active neonates, meconium passage will start within 6 hours after the first feeding. Great variation of color and consistency of meconium is seen in camelids. It

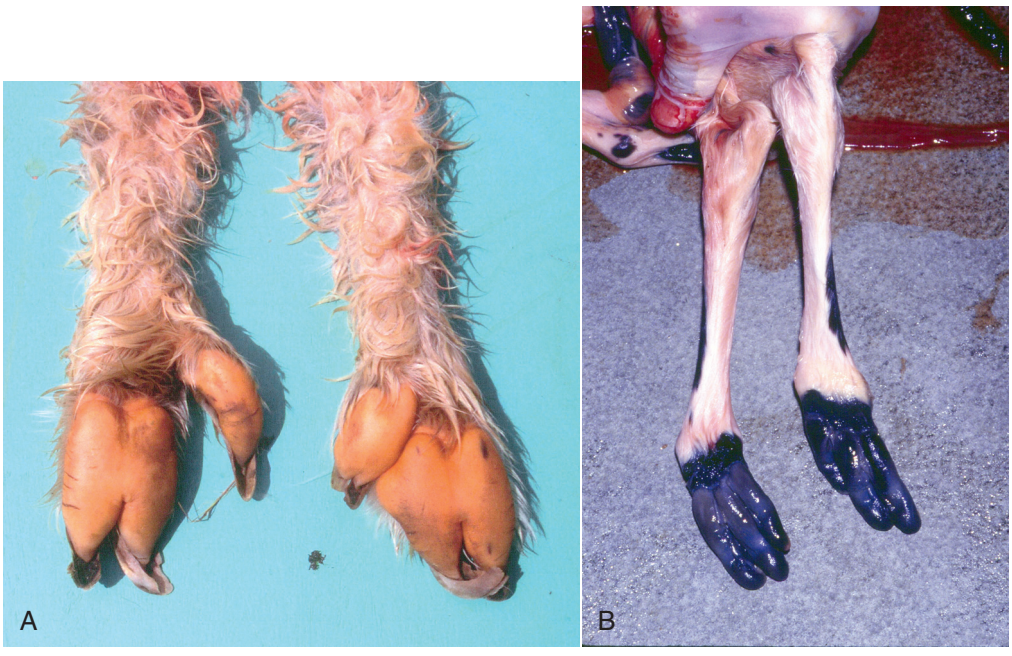

Figure 25-6 **A and B,** Polydactyly.

Figure 25-7 Syndactyly.

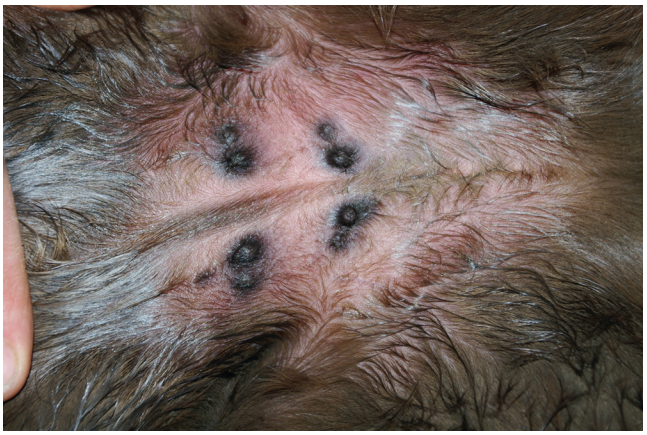

Figure 25-8 Supernumerary teats.

Figure 25-9 **A and B,** Gopher ears.

may be pasty or stringy and tan to dark tarry in color. Clinical signs of meconium impaction include straining, squatting, and tail wagging. These signs progress to anorexia, bloating, and signs of abdominal discomfort. Initial treatment consists of one or two warm soapy water enemas (20 to 40 mL). If after two enemas, the meconium has not passed, intravenous fluids may be indicated. Multiple soapy water enemas may irritate the rectal mucosa and result in severe straining and rectal prolapse.[12] Various preparations, including magnesium sulfate, human phosphate preparations, and acetylcysteine-containing preparations, have been used. The last are particularly helpful when meconium is very dry and sticky. Neonates that have retained meconium may have other abnormalities and should be examined closely. The main differential diagnosis to pursue is atresia ani. Routine administration of enemas to every newborn cria should be discouraged. Severe electrolytes disturbance have occurred in crias after inadvertent transvaginal retroperitoneal administration of hypophosphate enemas.[48]

Urine Retention

Urine retention may be associated with congenital abnormalities of the urinary and genital tracts.[49] In males, urethral blockage (aplasia) results in bladder rupture. In females, vulvar agenesis or atresia vulvi cause an obvious bulging of a pouch in the perineal areas and are often painful because of the large quantity of urine in the uterus and abdominal distension.

Umbilical Abnormalities

Accidents to the umbilicus are not uncommon. The simplest form is persistent bleeding, which may be treated with hemostasis provided through a hemostat or umbilical tape. Umbilical hernias are relatively common, but most will resolve spontaneously or with belt (vet-wrap) management if less than 3 cm. Umbilical hernias that persist or continue to increase in size require surgical repair. We have seen umbilical hernia and rupture of the abdominal wall with evisceration following dystocia caused by a term uterine torsion, possibly from twisting of the cord around the fetus. These are easily repaired surgically. Omphalophlebitis may develop later in life in crias that were born in unsanitary conditions or had a failure of passive transfer. Patent urachus may be observed but is less common than in other species.

Exposure

Hypothermia and depression caused by exposure or starvation are probably the most common acquired problems in the newborn camelid in the first 24 hours of life. These problems

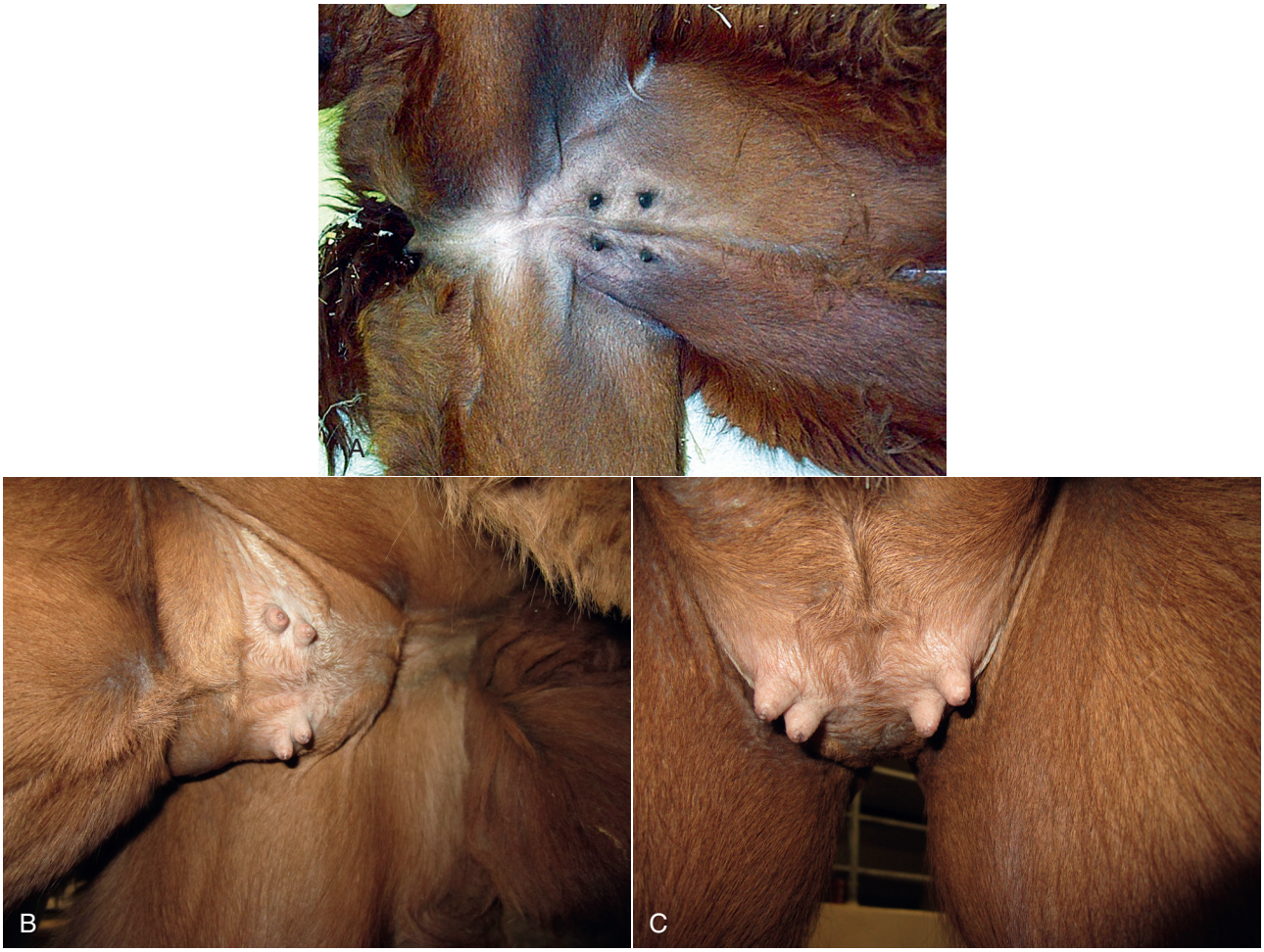

Figure 25-10 **Normal Udder Conformation in Alpacas. A,** Non. **B** and **C,** Lactating.

are often associated with crowding, primiparous females (poor mothering ability), unattended lengthy parturition, agalactia, or congenital abnormalities preventing the neonate from standing and nursing normally. They are easily prevented by closely monitoring term dams and providing a clean warm environment for parturition. Resuscitation of the compromised cria requires a warm environment and treatment of failure of passive transfer and dehydration. Hypothermia and depression may also be seen in crias with severe blood loss from the umbilicus or rupture.

Lactation

Mammary gland function and lactation remain poorly studied in alpacas and llamas, and reports in the English literature are scarce. However, some information is available on the anatomy of the mammary gland and milk production and characteristics.

Anatomy of the Mammary Gland

The mammary gland in alpacas and llamas is in the inguinal area and difficult to visualize particularly in nonlactating animals (Figure 25-10). Examination of the udder often requires restraint of the female in lateral recumbency or in the standing position, with one of the back legs flexed and held high. A detailed description on the anatomy of the llama mammary glands has been provided by Chavez et al. (Figure 25-11).[50]

Development of the Mammary Gland

Gross anatomic differentiation of the mammary gland is evident in the female fetus at about 4 month of gestation. The major growth of the gland occurs in the last month of gestation. In the last 2 to 3 weeks of gestation, the mammary gland size is about 5 to 6 times the size at the beginning of gestation.[51]

The major developmental abnormality of the mammary gland is the presence of supernumerary teats (polythelia), with an incidence of 17% in llamas and 6% in alpacas. Absence of teats (athelia) has been described in up to 0.9% of alpacas. Abnormal teat conformation, which is common, may compromise the ability of the cria to feed.

Lactation and Properties of Milk

The physiology of lactation has been investigated in few studies on milk production and milk characteristics particularly.[52–56] Initial milk production has been estimated at about 1600 mL in alpacas and 2000 mL in llamas. Peak

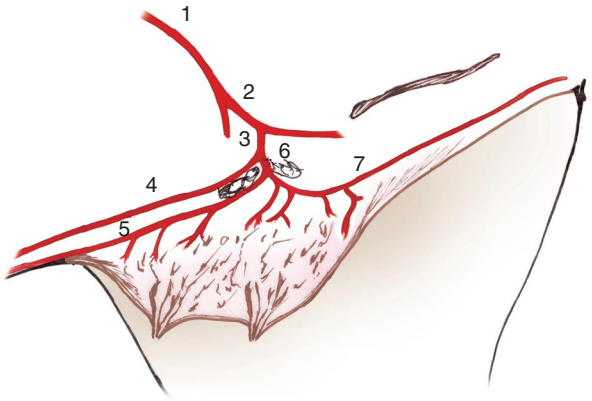

Figure 25-11 Schematic Drawing of the Mammary Gland and Its Blood Supply. A, External iliac artery. **B,** deep femoral artery. **C,** Pudendo-epigastric arterial trunk. **D,** Caudal epigastric artery. **E,** Caudal superficial epigastric (cranial mammary) artery. **F,** Caudal mammary artery.

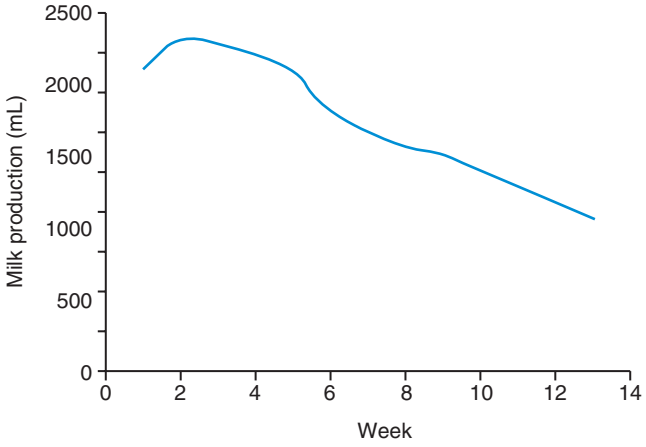

Figure 25-12 Typical Lactation Curve in Llamas. Alpacas' lactation curve will follow the same trend with lower, peak, and total production.

production is reached about 3 weeks after parturition (2325 ± 480 mL in llamas and 1938 ± 290 mL in alpacas).[51] Available information is limited, but lactation persistency and the general lactation curve resemble those of cattle (Figure 25-12). Following the peak period, milk production drops at a rate of 9% weekly. Peak and total milk production increase with the age of the dam. Weaning of crias takes place usually between ages 4 and 6 months. It is important to note that milk production in alpacas and llamas tends to dry up very quickly after weaning or loss of the cria.

The effects of lactation on the body condition score (BCS) and blood biochemistry of the dam have been studied in alpacas. Under South American grazing conditions, weight and BCS do not seem to be greatly affected by lactation. In one study, plasma urea nitrogen and creatinine were seen to be lower during lactation than at the end of pregnancy. Interestingly, in this study no change was observed in the serum level of nonesterified fatty acid (NEFA) during lactation. Blood chloride level decreased during lactation, whereas a significant increase of calcium occurred during lactation compared with the last few weeks of pregnancy. The authors concluded that metabolic adjustment is completed within the first week or two after parturition in alpacas. Lactational anestrus is rarely seen in alpacas and lamas. However, follicular wave dynamics and the quality of the corpus luteum may be affected particularly in thin animals.

A few studies have been performed on milk constituents. Llama milk is higher in lactose (6.5%) and lower in fat (2.7%) compared with cattle milk. However, other studies have reported higher fat content (4 to 4.5%).[52,55] The energy content of llama milk is lower than that of domestic ruminants (70 kilocalories per 100 grams [kcal/100 g]). Milk composition is not affected by stage of lactation and lactation number. Llama milk has a higher content in calcium and lower content in sodium, chloride, and potassium.[53] Trace mineral content is similar to that of ruminant milk. Alpaca milk composition was described in two locations (Andean high plateau [AHP] and Patagonia [P]).[56] Fat content was lower in both colostrum and milk in the P region compared with those in the AHP region. Overall mean fat content in milk was 3.8 ± 0.6 and 2.6 ± 0.5 for the AHP and P regions, respectively. Lactose content for alpaca milk was 4.4 ± 0.5 and 5.2 ± 0.5 for the AHP and P regions, respectively.[56] These regional differences in milk constituents were attributed by the authors to a difference in pasture quality and the energy expenditure from walking.

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