FROM CLEFT PALATES TO CLEFT LIPS:
MANAGING LITTER AND BREED RISK

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Abnormalities in neonates are always a concern for breeders, particularly new breeders. While fetal abnormalities are not the norm, they do occur. A class of abnormality sometimes seen in neonates is a midline defect. Midline defects in puppies include, but are not limited to, cleft palates, umbilical hernias, open abdominal walls, open fontanel, spina bifida, anything that happens down the center of the patient (e.g., skull, spinal column and tail).

This article focuses on a specific midline defect: cleft palates [i.e., primary cleft palate, secondary cleft palate, and cleft lip (also called harelip)]. Cleft palates occur in all breeds and mixed breeds, but brachycephalic breeds (short, flattened facial features) seem particularly prone to them. Purebreds are alleged to have a higher incidence than mixed breeds. As of January 2025, the Berner-Garde Foundation Health Database documented 75 Bernese Mountain Dogs (BMDs) with cleft palates, most of whom were stillborn.

WHAT IS A CLEFT PALATE OR CLEFT LIP?

The parts of a dog's oral cavity relevant to a cleft palate discussion include the primary or hard palate, the secondary or soft palate, and the lip. The primary palate is the bony front portion of the roof of the mouth. The secondary palate is the muscular rear portion of the roof of the mouth. Lips are divided into two halves, the upper and lower lip. Lips are comprised of skin, muscle, tendon, glands and oral mucosa.

A cleft palate is a congenital birth defect where there is an opening in the roof of the mouth (palate). This opening can occur in the front part of the palate (hard palate) or the back part (soft palate). The opening may be the result of tissues not completely connecting to each other during fetal development. In severe cases, the hole opens from the palate to the nasal passages. A cleft lip is a defect of the lip that can extend to the underlying bone and/or hard palate.

Veterinarians classify cleft palate defects as ***primary*** or ***secondary*** clefts. A primary cleft involves the hard palate. The secondary cleft, which is more common, involves the hard and/or soft palate. These secondary clefts can go unnoticed until clinical signs begin to appear such as stunted growth, difficulty nursing, coughing, sneezing, pneumonia or chronic rhinitis.

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WHAT CAUSES A CLEFT PALATE OR CLEFT LIP IN NEONATES?

While the exact cause of cleft palates is unknown, cleft palates in dogs are hypothesized to result from any of several causes, in isolation or in combination:

- genetic (inherited from the parents)
- toxins
  - environmental and/or
  - pharmaceutical
- maternal
  - nutritional excess or deficiency
  - exposure to viruses

GENETICS

Currently there is a BMDCA-sponsored study in progress to investigate a possible genetic cause of clefts in Bernese Mountain Dogs. The goal is to design a DNA test for this disorder.

There is evidence of cleft-causing mutations in two genes (SAMTS20 and DLX6) circulating in different parts of the Nova Scotia Duck Toller population (Wolf et al., 2014 and 2015). Each of these mutations has a recessive mode of inheritance and can cause clefts independent of each other. Currently, genetic tests for these two mutations are commercially available

to breeders and dog owners, however, they may not be relevant for all breeds. There is evidence from four Staffordshire Bull Terriers with this deformity showing no mutations in these two genes, indicating that their occurrence was due to a mutation in a different gene or a non-genetic factor (Ruszkowski et al. 2023).

Geneticist Dr. Lambertus Klei, a frequent speaker at Berner University, suggested that it would be of interest to test DNA from BMDs with clefts to see if the same gene mutations (SAMTS20 and DLX6) are also occurring in Berners. At a minimum it would rule out that these mutations are the cause of clefts in the BMDs tested.

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Dr. Klei points out that not only do the mutations appear to be breed-specific, but they also show evidence of being family-specific. This will make it difficult to develop a general genetic test for these palate and lip malformations.

Evidence from human studies also points to a heterogeneous background. Oral and palatal clefts occur in many human disorders and are often accompanied by other skeletal malformations, pointing to a diverse genetic background.

An overview article on the incidence of orofacial clefts in purebred US dogs was recently published (Roman et al., 2019). While BMDs were not included in the study, several findings are of potential interest because of our breed's origins. The BMD of today traces back to the Mastiff type and the black-and-tan dogs used by Roman soldiers in the Alps over 2000 years ago. The most interesting general genetic and environmental findings possibly relevant to our breed include:

- Breeds in the Mastiff/Terrier genetic cluster were predisposed to clefts.
- Certain breeds in the ancient genetic cluster were at increased odds of orofacial clefts.
- Male purebred dogs were at increased odds of cleft palates.
- Geographic region appeared to be a relevant risk factor suggesting possible non-genetic factors (i.e., dogs in the Midwest had lower rates of clefts).
- Cleft puppies involve substantial financial losses for the purebred dog breeder (from increased veterinary and nursing care costs to lost sales).

The current effort by the BMDCA to collect DNA on puppies with cleft palates and/or lips is a good start. Dr. Klei suggests that it would be of help in the search for causal genetic variants if DNA were also collected on the parents and at least one unaffected full sibling.

TOXINS

Lacking a genetic test for cleft palates and lips, there are still a few things a breeder can do to manage the risk of cleft palates in their puppies. Both *environmental* and *pharmaceutical toxins* have been implicated in cleft palates and lips.

Normal neonates. Photo credit Nancy Melone.

ENVIRONMENTAL TOXINS

Common environmental toxins encountered by breeding dogs include chemicals used in residential lawn care and agricultural applications (e.g., pesticides, herbicides and fertilizers). An often-forgotten source of toxic exposure for breeding dogs are chemicals used in household cleaning and disinfecting (e.g., chlorine). For a more complete list of specific chemical toxins linked to cleft palates, see Buser & Pohl (2015).

PHARMACEUTICAL TOXINS

Drugs also pose cleft risks to developing fetuses. Studies have implicated some pharmaceutical products, including corticosteroids (e.g., prednisone), certain antibiotics (e.g., doxycycline), aspirin,

griseofulvin and some anti-seizure drugs. Some anti-parasitic drugs have also been associated with fetal deformities. Before breeding, transition breeding dogs to reproductively safer anti-parasitic products (e.g., Revolution has been tested safe on breeding dogs and lactating bitches, but according to some users in high tick areas, it is somewhat less effective at controlling ticks). Because the market is small, most other anti-parasitic products have not been tested on breeding dogs or lactating bitches.

Buser and Pohl (2015) have described "windows of susceptibility" for toxin exposure in humans and mice during fetal orofacial formation. They argue that the window of susceptibility to environmental toxins in the development of cleft palates is quite narrow and follows closely the window of palate development in the fetus of *any given species*. More recently, Freiberger et al., (2021) have supported the Buser and Pohl findings that palate development occurs in dogs in the steps described in humans and mice, but the actual palate *closure* occurs at an intermediate time later in gestation (~d35-44, with closure by d44).

Factors found to be important in assessing toxin deformity risk are:

- Dosage
- Timing of exposure
- Susceptibility of the species (i.e., the genetic makeup)
- The process by which the toxin interacts with the target cell to produce the toxic effect (i.e., the mechanism of action)

There is some debate among investigators as to whether dosage or timing of exposure is the more significant factor. The risk of deformity is increased with either higher dosage or toxin exposure during orofacial development.

The obvious recommendation is to minimize or eliminate a bitch/dog's exposure in the home to these chemicals and, equally important, to minimize a bitch/dog's exposure to them outside the home (e.g., avoid those nice green, weed-free lawns). Do not administer any drugs to brood bitches unless under the supervision of a theriogenologist (reproduction) specialist or a general practice veterinarian experienced in canine reproduction.

MATERNALLY-CENTERED CAUSES

During certain periods of orofacial development during gestation, maternally-centered causes have been associated with the occurrence of cleft palates and cleft lips in canine neonates. These include nutritional excesses and deficiencies as well as viral infections.

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NUTRITIONAL EXCESSES OR DEFICIENCIES

Nutritional causes of cleft palates can arise from both excesses and deficiencies. Raw-fed bitches are at higher risk for these nutritional imbalances as well as other imbalances during pregnancy. For example, studies at Whelp Wise indicate bitches fed raw have a higher rate of dystocia (difficulty in delivery), likely caused by nutritional imbalances related to uterine contractions.

Excess Vitamin A

Excess vitamin A has been associated with cleft palates in neonates. Vitamin A is fat soluble so it can be overdosed easily. If you supplement with any vitamin product or food, be careful you do not overdose. Some human foods high in vitamin A include liver, tuna, cod liver oil, dairy products, spinach, kale, pumpkin and carrots. Be mindful of your treats.

Folic Acid Deficiency

In contrast to vitamin A excesses, folic acid (vitamin B9) deficiency has been associated with midline defects in humans and some dog breeds. Folic acid is water soluble so it is not easily overdosed, but more is not necessarily better. Recent research has shown that some breeds (i.e., pugs and chihuahuas) may benefit from folic acid supplements (Domosławska et al. 2013) and other breeds (i.e., Labrador Retrievers, Golden Retrievers and Labrador/Golden crosses) may not benefit (Gonzalas et al., 2021). It is believed that those breeds that do benefit may not metabolize folate well.

There is no published dose for dogs, but reproduction veterinarian, Marty Greer, DVM, JD recommends 400 mcg per dog per day until day 40 of the pregnancy. For maximal effect, it should be started at least one month prior to breeding.

VIRAL INFECTIONS

Finally, a bitch's exposure to certain viruses during certain stages of gestation has been implicated to cause clefts. There are multiple reasons to sequester a pregnant bitch from other dogs during pregnancy (e.g., canine herpes virus) and this appears to be another reason. The prudent breeder starts out with a healthy bitch, who is current on vaccines/titers before breeding and post breeding avoids dog shows and possibly showing any dog in the house during this time. Again, this is another area where a breeder's individual risk tolerance might be different across breeders. Some breeders may have a higher risk tolerance and choose to attend shows. Obviously, all vaccines should be done prior to the decision to breed. Do NOT vaccinate during pregnancy.

CAN CLEFT PALATES BE TREATED?

The goal of prevention remains elusive. As such, surgery, medical management or euthanasia offer secondary options. Surgical repair has been the recommended treatment for puppies with severe clefts. The best timing of the surgery is not well established. Typically, surgery is not recommended for puppies under 8 weeks because of the potential for tissue damage and injury to the blood supply that could damage development of the muzzle. Surgery after 5 months may be more difficult because of anecdotally reported widening of the cleft. On the positive side, the tissue has greater tensile strength in the older puppy.

Until recently surgery has been recommended as the only treatment for cleft repair. However, surgery can fail or require revision. Humane euthanasia has typically been the fallback recommendation if the repair could not be done or the prognosis for the repair was not good. Alternatives to surgery other than euthanasia were usually not proposed.

In 2025, researchers at the University of North Carolina State University sought to examine more conservative treatments (i.e., medical management) for the condition (Monck, et al., 2025). Specifically, they examined quality of life for dogs medically managed (i.e., no surgery) that survived transition to solid food. Previously, the common belief was that the size of the cleft was correlated with the prevalence of clinical signs. Because this had never been empirically established, dogs' clefts were classified by the size

> *Plan what you will do BEFORE you are faced with doing it!*

of the cleft: wide throughout, wide rostrally and slit throughout. Although neonatal death was high in the first 6 weeks (~41%) with the median age for death at 6.5 days in this study, results from the study indicated that for dogs surviving, sneezing was the most frequent clinical sign. Dogs with wide-throughout clefts had more material lodge in the opening requiring veterinary removal than dogs with slit-throughout openings. In contrast to conventional wisdom, *there was no correlation between cleft size and either clinical signs or quality of life; larger clefts were not associated with more clinical signs or lower quality of life*. Sixty-three percent of the dogs had a perfect score on a weighted scale for quality of life and 83.3% had a perfect score on a visual analog scale. Sixty-four of 65 owners said they would adopt another dog with a cleft palate. The conclusion of the researchers was that *conservative management appears to be a viable option for treatment of cleft palates in some dogs*.

WHAT IS A BREEDER TO DO?

The breeder of a cleft palate or cleft lipped neonate is faced with two decisions—one is immediate and the other can be made later. The first is deciding how they will handle the affected puppy. Depending on the answer, this decision may need to be made on the spot. The second decision, which can be made any time before breeding the pair again, is deciding how to handle the sire and dam that produced the affected puppy. The first decision pertains to the current litter. The second decision pertains to future litters and the future of the breed.

WHAT IF I HAVE A CLEFT PALATE PUPPY?

Plan what you will do BEFORE you are faced with doing it! Decisions made in the heat of the moment tend not to be the best ones. Have a strategy for the varying degrees of this deformity, from a cleft lip to severe palate deformities. What will you do if you have a neonate with a cleft lip? A cleft palate? These are decisions that are personal and can vary across breeders depending on one's view of life and death. Moreover, a decision often depends on the severity of the diagnosis and as such requires a case-by-case

evaluation. Regardless of the final decision, breeders should record the cleft palate or cleft lip neonate in the Berner-Garde Health Database.

Many pups with cleft lips with minimal palate involvement can live normal lives because the lip does not affect eating or drinking much. While closing the lip may not be needed for functionality, dog owners often choose to close the lip for cosmetic reasons. It is typically not a complicated surgery.

Cleft palates require more careful thought and may involve professional judgments on the severity and prognosis, if treated. These are the hard decisions, especially when the cleft is obvious at birth and a decision must be made. Cleft palates do not heal on their own. If left untreated, potentially fatal conditions can occur. So, you are faced with a hard decision. Do you resuscitate the cleft palate neonate? If not, can you find a home for it where it will be properly cared for either surgically or with medical management? If not, will you care for it?

The cleft palate neonate will likely require multiple surgeries as it grows. Typically, these surgeries should not be done until the neonate is 8-12 weeks old and can manage surgical risks better. As such, the neonate will need to be tube fed by the breeder or new owner to obtain adequate nutrition for growth and to avoid respiratory issues. Even then, these surgeries are no guarantee. On the other hand, if the surgery succeeds the prognosis is usually good.

A single surgery done by a dental surgeon or specialist can cost $5,000-$10,000. Insurance may or may not cover it. Recovery involves many weeks, with rechecks every couple of weeks. The big risk for these dogs is the surgical site breakdown caused by tissue tension or trauma during or after surgery. Puppies must be fed a soft semi-liquid diet until the site heals. Hard food or toys can easily break open the repaired site. Healing takes a minimum of six weeks.

Given recent research, conservative management can also work for some owners with dogs with more severe clefts. In those cases, there may be extra work for the breeder during the first eight weeks of life (e.g., tube feeding).

These situations require informed judgment based on both the severity of the puppy's diagnosis and the breeder/owner's personal beliefs and personal and financial situation. Breeding is hard. Deciding not to resuscitate a newborn with a cleft palate is one of the hardest decisions a breeder will ever make.

WHAT IF MY BITCH OR STUD DOG PRODUCES A CLEFT PALATE PUPPY?

Most veterinarians advise culling both dogs from further breeding if they produce a cleft puppy. Since the cause of cleft palates is still considered unknown and there are alternative explanations for some portion of these defects, this advice seems extreme when no genetic mutation has been found, and the mode of inheritance (if genetic) is unknown. In rare breeds with narrow gene pools and limited numbers of breeding dogs, such severe culling advice can carry its own risks to the breed.

A less drastic strategy would be to remove the sire from further breeding. The rationale here is that sires are likely to have more litters and offspring than dams and therefore sires have a higher chance of spreading the mutation (if there is one). Dr. Klei goes on to say that this strategy is not based on genetic theory but rather on calculated probabilities and the "bold" assumption that this is caused by a single recessive mutation. He is skeptical that all clefts are caused by the same simple recessive mutation.

Dr. Klei advises against test matings to determine whether the dog and/or bitch is a carrier of a causal mutation. So far, the evidence points to a heterogeneougenetic background making this a pointless exercise and likely to pointlessly produce pups with deformities.

QUOD ERAT DEMONSTRANDUM

Because it is unlikely that we breeders will or even can produce a genetically and conformationally perfect dog at this time, the goal in breeding responsibly is to use genetic testing, phenotypic testing and whatever other additional forms of information we possess to make better, rather than worse, breeding decisions. As Jerold Bell, DVM, Adjunct Professor, Clinical Genetics, Department of Clinical Sciences, Tufts University, has said, "Genetic tests do not determine which dogs get bred, only which dogs they get bred to."

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ABOUT THE AUTHOR

Nancy's articles, published internationally, have won numerous American Kennel Club (AKC) Publication Excellence Awards, Dog Writer's Association of America (DWAA) Maxwell Medallions and a Morris Animal Foundation Advances in Canine Veterinary Medicine Award. Currently she serves on the board of the Nederlandse Kooikerhondje Club of the USA (NKCUSA) and chairs their Health and Genetics Committee. She is Editor Emerita of The Alpenhorn and served on the boards of the Berner-Garde Foundation (BGF) and Bernese Auction Rescue Coalition (BARC). Her Ph.D. is in Information and Decision Science from the University of Minnesota.