**Canine Neonatology**

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**The Normal Neonate**

**Birth to weaning**

Immediately after birth, puppies will cry, have pink mucous membranes, have strong heart beats and move vigorously. At this stage all the umbilical cords should have been tied off and disinfected using chlorhexidene, iodine or other appropriate disinfectant. Normal puppies will nurse about every 2 hours. Puppies which are healthy will drink and then sleep quietly with the occasional jerky movement. Puppies which cry and move about continuously have some form of discomfort. This may be: hunger, coldness, isolation from the bitch or litter mates, the need to defecate or urinate or burping, bloating, pain or illness. The crying stops as soon as the cause of discomfort has been rectified. If the cause is not rectified, the puppy will in time stop crying, appear limp, have poor muscle tone, become weak, quiet, and finally fall into a coma and die soon afterwards. It is easy to distinguish the latter puppy from a healthy puppy. Healthy puppies which lie still because they are sleeping are very responsive to physical stimulation and will cry and move vigorously whereas unhealthy puppies will not respond to touch. Another easy way to evaluate the wellbeing of a healthy puppy is to evaluate the suckling reflex. This is done by inserting your finger into the puppy’s mouth and a healthy strong puppy will immediately suck on it whereas the weak one will either not suck at all or suck weakly. Weight gain is the most reliable and objective parameter to measure progress in neonates. In the first 24 hours however, puppies may either not gain weight or lose some weight (less than 10% of birth weight). Following that, the weight should increase steadily. Their weight should double in the first 8-12 days. The exact amount of weight gain varies a lot depending on breed and milk production of the dam and many other factors. A rough estimate of normal daily weight gain is 1-1.5 grams per day for every Kg of anticipated adult weight. Thus if the anticipated adult weight is 35 kilograms, the daily weight gain should be around 35–50 grams per day. The growth curve changes with age. Breeders will in time get their own weight gain curves for their breed. As long as there is gain from day to day, things are probably going well. In contrast, puppies which fail to gain weight are either sick or not getting enough milk for whatever reason and will die. The umbilical cord dries and falls off within 2-3 days following birth. For the first 5 days the puppies have extensor dominance meaning that they keep all their limbs forward. The eyes and ears open around day 10-14 and they can usually start standing a few days later. New-born puppies do not have controlled excretion and cannot defecate and urinate spontaneously without stimulation from the dam. The bitch will lick the puppy’s perineum (anus area) to stimulate excretion until controlled urination and defecation occurs at around 15-21 days. The puppies’ milk (deciduous) teeth erupt between 3-6 weeks but in some toy breeds this may be delayed by a week or two. Under normal circumstances, if the puppies are growing well, the puppies can be left with the dam to suckle until weaning age. If the dam is not able to provide adequate milk, supplementary feeding of milk formula and creep feeding should be considered. It is important to notice that if creep feeding is practiced, the puppies should be fed water as well in a shallow bowl.

**13.2. Dewclaw removal**

The dewclaw is the fifth inside facing claw. Dewclaw removal involves cutting off the dewclaw as close to the leg as possible, disinfecting it and applying some pressure on it to prevent bleeding. Cauterising tincture of ferrous chloride may also be used to stop haemorrhage. When performed by a veterinary surgeon, they may normally cauterise the small vessels severed using electronic devices and even insert small stitches. Dewclaw removal may be performed without anaesthesia by some veterinary surgeons whilst other elect to infiltrate some local anaesthetic. Some veterinary surgeons maintain that the needle to infiltrate the local anaesthesia is as painful as the removal of the dewclaw. Most breeds will have a dewclaw on the front feet whilst only some will have dewclaws on their back feet as well. Some members of the giant breeds may have dewclaws which are duplicated or even triplicated in their hind feet. These traits appear to be strongly familial. Dewclaws in dogs appear to be non-functional as they do not bear weight and neither are they able to hold onto prey as is the case in felines. Their removal therefore is non-consequential. There are breed differences with regards to their presence and desirability to remove them or not. In many breeds either or both of the front or back dewclaws are considered a nuisance and their removal is recommended. The nuisance factor is that in some dogs they do not show natural abrasion and wear down and grow almost 360° into themselves and cause infection. In other dogs they may hook onto objects, tear and bleed. In cases where removal of the dewclaws is desired, some recommendations are made. New-born puppies do not have adequate clotting mechanisms until they are about 72 hours old and before then they are at risk of serious haemorrhaging. Therefore it is best to delay the removal of dewclaws till after the third day but not later than the fifth day. This is because some breeds grow very quickly and by 5 days of age, the dewclaw has grown so large that removal is more difficult, bleeds more and removal is also likely to be more painful for the puppy.

There is another very important reason for the delay in time before removal of the dewclaws in puppies. As puppies are born with no immunity as explained in 7.1, the puppy is more likely to develop an infection if the skin is broken and the puppy has not yet received or absorbed all its antibodies.

**Tail docking**

Tail docking is a practice which has been performed for many years. In more recent years however, tail docking has become condemned as an unnecessary procedure which mutilates the dog. Others insist that if performed correctly, it is a perfectly humane procedure which may prevent far more distress than it causes. The reasons for tail docking include prevention of tail injuries in gundog breeds working in thick vegetation, increased manoeuvrability in underground holes in terrier breeds, prevention of soiling of perineum in long haired and thick coated breeds and maintaining breed standards. Despite possible advantages, tail docking has been banned in various countries or become frowned upon in others. This is a trend that is likely to spread. The ethical debate surrounding tail docking will not be discussed in this document. It is considered a minor procedure by some, which causes minimal discomfort and it does not appear to adversely affect puppies’ health in any way. Others maintain that it is an unnecessary painful procedure. It is important for the breeder to familiarise themselves with their local rules and regulations regarding legality of tail docking. In countries where tail docking remains legal, it is advised that breeders consult their veterinary surgeons to perform the procedure or at the very least show them how to perform it. The age of the puppies at the time of tail docking is important for similar reasons as explained as for dewclaw removal.

**Rearing orphan puppies**

The best and easiest way to raise puppies is to have a good dam which does it, as nature intended. They will feed them; care for them, clean them, stimulate excretion and keep them warm. If it appears that the mother has poor milk production but is otherwise a good mother, it should still be considered to have the bitch care for them and only supplement feed the puppies. Poor milk production should be suspected when there is insufficient weight gain and hungry cries from the puppies. There may be a problem with supplement feeding puppies when the dam also still nurses the puppies. When the bitch is the primary feeder of the puppies but the breeder needs to supplement feed them, the trick is to know how much to feed them extra. This is because the breeder has no idea how much milk they are already getting from the dam. The main danger of supplement feeding under these circumstances is over-feeding which may cause severe diarrhoea. Over feeding is particularly likely if the method of milk formula supplementation is tube feeding. The best way to manage this dilemma is to avoid tube feeding and supplement by bottle feed. This way the puppy can regulate its own intake. Foster mothers, when available, are the second best choice. Only when neither of this is possible, should hand rearing be practiced. Thus, only hand rear when there is no other option!

Puppies may become orphaned because the dam may be dead, sick, unable or unsuitable to adequately care for her puppies. Poor milk production and poor mothers are the most common reasons for fostering or hand rearing puppies. Irrespective of method of raising the puppies, the breeder should ensure that the puppies have adequate colostrum uptake. Puppies cannot control their body temperature (thermo-regulate) during the first 3 weeks of life and the environmental temperature must therefore be controlled for them. Normal puppies will crawl towards their mother and litter mates when cold and disperse when hot. For orphaned puppies, the ideal temperatures during the first, second, third and following weeks are 35°C, 31°C, 28°C and 26°C respectively. This can be achieved by using incubators, heaters or lamps. It is important to make sure that the puppies must have the ability to escape heat if the temperature becomes too hot. During the first 3 weeks the breeder should, after feeding the puppies, stimulate excretion by wiping the perineum and prepuce with a moist piece of cotton wool and drying the skin again afterwards.

Fostering puppies is much easier and more successful than hand rearing if one has a good foster mother. Bitches which are kept together often have synchronized cycles and hence are likely to whelp at more or less the same time. This offers the opportunity to swap some puppies around if there is a particularly large litter. The breeder may even have one bitch rear the entire litter of another when required. The stage of lactation of the foster mother is important. The ideal is that the foster mother is at the identical stage of lactation as the mother of the puppies. This is however seldom possible. Irrespective of stage, the foster mother should have enough milk to ensure adequate weight gain. If insufficient milk production is suspected, supplement feeding may be required. Foster mothers need not necessarily be of the same breed but should preferably be of similar size. Not all bitches are suited as foster mothers. Puppies must therefore be introduced to the foster mother under careful supervision. If the foster mother has puppies of similar age and size, it is best to take them all away and rub them against each other so that they acquire the same odour before reintroducing them. If the size differences of the puppies are substantial, it may be necessary to remove the bigger puppies to give the smaller puppies’ uncontested access to the teats and let the puppies of different sizes and or ages drink at separate times. Breeders should make sure that when fostering puppies that they accurately identify the puppies to avoid incorrect registering. Though rare, pseudopregnant bitches and even spayed bitches with strong maternal instincts may start lactating when exposed to sufficient stimulation of hungry puppies. In some cases, foster mothers may even be of another species.

***Milk replacers during the first 3 weeks***

Commercial puppy milk formulas are available and are nutritionally balanced to meet the needs of orphan puppies. Homemade milk formula recipes are also available but are not advised unless the breeder is able to fully balance them. Ordinary cow’s milk should not be used. The constituents of milk vary widely from species to species. The major constituents which may differ between species are the percentages of respectively, fat, protein, lactose and water. The latter in dog’s milk, given in the same order are: 8.3, 9.5, 3.9 and 79%. Only enough milk replacer formula should ideally be made for one feed but if there is any surplus, it should be cooled and rewarmed for the next feed. It should not be stored for longer than 4 hours (till next feed) and not be rewarmed more than once. The quality of milk replacers differ. It is the author’s experience that milk replacers which do not clump easily, are superior to those that do. Poor quality milk formula is one of many causes of diarrhoea and bloating in puppies. Bloating may still be a problem with the best milk formula or homemade formulas. Adding additional fat in the form of cream (5 ml per every 100 ml formula) or tinned puppy starter mouse or high calorie supplement (also at 5 ml per 100 ml formula) may all decrease the incidence of bloating and increase weight gain. The formula should be made exactly adhering to the instructions. If the amount of water added to the milk powder is too much, the puppy will not receive the correct amount of nutrients for its weight and fail to thrive and grow well. It will remain hungry and cry despite getting correct volumes of milk and feed intervals. If the amount of water is too little, it will dehydrate and die. This is because the concentration of the solids in the milk formula is then too high and an osmotic imbalance occurs. This then leads to so-called osmotic diarrhoea. Simple over feeding of milk formula may also cause diarrhoea. Milk formula must be warmed to body temperature (38°C) prior to feeding. Feeding the correct amount and at correct intervals is crucial. Instructions in some milk replacer formulas are sometimes vague with regards to amounts which have to be fed. The total requirement of already made up milk formula which the puppy requires is 20 % of its bodyweight over a 24 hour period. The calculated amount can then be divided into 6 meals. So assuming the puppy weighs 300 grams, the puppy needs to consume 60 ml over 24 hours divided into 6 meals of 10 ml each. These guidelines may be used when bottle feeding. Puppies which demand more may be given more. However, if the puppy drinks substantially less than the calculated amount (20% less) the breeder should be alarmed and consider tube feeding. This feeding protocol is a tested one which serves many breeders well. Many variations thereof exist. During the first week, some breeders wish to feed smaller meals more often (2-3 hour intervals round the clock). This they do because it closer resembles the feeding frequency of puppies at that age. Following the first week they follow the conventional feeding protocol as above. The need to accurately weigh the puppies cannot be overemphasized.

All the principles which apply to human baby feeding bottles should be respected. This involves keeping bottles, teats, feeding tubes and syringes clean and dry as well as using disinfectants and cleaning agents safe for this purpose. Puppies may be bottle fed or tube fed. In order to maintain a proper suckling reflex it is best to bottle feed as far as possible. Bottle feeding offers the opportunity for the puppy to regulate their own intake and decreases the possibility of tube feeding into the lungs or feeding incorrect amounts. Tube feeding is convenient because it is not so time consuming and it ensures that the puppy receives the required amount. In cases where the puppy is too weak to suckle, tube feeding is the only option. Breeders having or wishing to tube feed should acquaint themselves with the procedure first having their veterinary surgeon demonstrate it on a puppy. If the correct tube and technique is used, it may be a helpful adjunct to rearing puppies. If breeders notice that the puppy regurgitates the meal they should stop immediately and bring this to their veterinary surgeon’s attention. They may have fed the puppy too much or its stomach contents have not been emptied from the previous meal. Also, tube feeding in excess of the stomach capacity will lead to regurgitation and possible death by aspiration or pneumonia. Normally, puppies would suckle for 5-6 weeks but it is possible to wean hand reared puppies earlier at 3-4 weeks of age by introducing specially formulated puppy rations (creep feed) designed for this purpose.

***Creep feeding***

Creep feeding is the concept of feeding puppies semisolids at the earliest age which they can consume them. This age varies somewhat but is usually started in puppies from day seventeen. Although still messy, at 21 days most puppies can consume semisolids and by day 24, most will eat well. At about this time, puppies will start walking as well. Top quality creep feeds in the form of porridge, small kibbles or tinned wet food are commercially available. Human baby food is not an acceptable alternative. Creep feeding has many advantages. It ensures good weight gain in cases where milk production is starting to wane and accustoms puppies to solids in preparation for weaning and rehoming. It is invaluable in cases where early weaning is necessary. Creep feeding is much cheaper and less time consuming and labour intensive than feeding milk replacers. Creep feed may be introduced from 17-21 days onwards and offered 3-5 times per day. Creep feeding is also practiced in nature by wild dogs. African wild dogs will regurgitate their partially digested meat meal in response to the begging of their puppies. In exceptional cases, domesticated dogs have been seen to regurgitate food in response to the whining hungry puppies.

**Weaning puppies**

Weaning refers to the process by which the puppies become independent of their mothers and start meeting their caloric requirements by eating only. The best weaning age for dam reared puppies is 6 weeks and for hand reared puppies it is around 3 weeks. The latter is referred to as early weaning. There are instances where early weaning is advised. This is because some bitches may become agitated, neglect their puppies or even show aggressive behaviour and injure them. Normally, the dam’s milk will start decreasing from the fifth week or so, but this may vary widely depending on stimulation from puppies, nutrition of dam and individual differences between dams. Most breeders will introduce creep feeding (13.4.3) from 3 weeks onwards and take the puppies away at around 6 weeks. The best way to wean puppies at this age is to abruptly remove them from the dam. It is generally recommended that when switching food, it is done gradually by slowly increasing the percentage of new food in the original food. This is more important in young puppies post weaning, at point of sale, than in adult dogs. Weaning is a particularly stressful time in a puppy’s life and at that age they are vulnerable to many causes of diarrhoea. Intestinal upsets are more common in sudden changes. Puppies sold are subjected to numerous stressors, the magnitude of which should not be underestimated. These stressors include, separation from the dam, separation from their litter mates, separation from familiar human contact, adaptation to a new environment, introduction to new family, introduction to new pets, introduction to the bottom of a new pecking order, sudden milk deprivation and introduction to new diets. It is well documented that stress mediated through endocrine and other pathways compromise the immune system. Weaning time also coincides with the time that the maternally derived antibodies start waning. In combination, all these changes lead to a significant reduction in resistance and this opens the way for both opportunistic and specific diseases in this critical period to invade and cause disease. Diarrhoea is a common manifestation during this time but more severe ailments are also possible. Because puppies are vulnerable to so many problems at the time of weaning, some breeders elect to wean the puppy themselves and only sell the puppy at a slightly older age (9-10 weeks). During the 2-3 weeks that they keep the puppy longer, they accustom the puppy to its new diet and start with the socialization process. Puppies will then also have had more than one vaccination making them less disease prone. Some breeders send the puppy to its new home with a week’s or so supply of the food it was on. It is interesting to note the large number of “well trusted” recipes which breeders have as a weaning formula. This formula may contain a wide variety of ingredients including cottage cheese, rice, porridges of all sorts, vegetables, probiotics and many more. Frequently, breeders will have an instruction list which is sold with the puppy. This usually has recommended vaccination programs, deworming schedules and feeding instructions. All too often these feeding instructions contain any number of ingredients followed by a complex homemade recipe. Although the breeder might have the very best intentions and is convinced that it works very well for them, the new puppy owner is unlikely to comply with the instructions. Also, the new owner’s veterinary surgeon is unlikely to agree with the feeding instructions given by the breeder, all leaving the new puppy owner very confused. Therefore, it is advised that these feeding instructions be simple and preferably refer to easily obtainable commercial products.

**Neonatal Disease**

The neonatal period in dogs is not as well defined in the literature as it is in humans. It could be fair to define the neonatal period starting at birth until 8 weeks of age. This is because at that stage the organ functions have matured and more closely resemble adult function. For the purposes of this discussion, the puppy’s development is divided in three separate phases. The first is the first 3 weeks of life, second from 3 weeks to weaning (around 6-8 weeks) and third from 8 weeks till adulthood. This is done because as a rule, many conditions present themselves in these distinct age groups. It is however true that slight overlap is possible.

Canine neonatology and paediatrics are respectively the fields of study involving conditions of the neonate and conditions of the puppy before adulthood (puberty). These fields of study are neglected in veterinary science. As a result, veterinary surgeons are generally poorly equipped to deal with sick neonates. Furthermore, disease in the puppy especially below 3 weeks of age can be very frustrating. This is because even when the breeder recognises the early symptoms of disease, their veterinary surgeon is usually unable to change fatal outcome irrespective of their interventions. The breeders’ frustration is further exacerbated by the veterinary surgeons’ inability to diagnose accurately and propose preventative strategies. Furthermore, post mortems and exhaustive tests may be unrewarding, leaving the breeder with more veterinary costs, more dead puppies and still no answers. Also, lack of experience on behalf of the veterinary surgeon results in their reluctance to encourage breeders to present puppies in the first place. For these reasons, breeders may have become despondent and fail to consult veterinary surgeons regarding sick puppies. This unfortunate state of affairs is counterproductive as it does not improve skill in care of sick puppies and creates the incorrect perception by breeders that nothing can be done in any case. Some breeders have also become “used to” an unacceptably high percentage of puppy losses. Despite all these difficulties, veterinary surgeons and breeders should realise that it is in their interest to present and examine as many as possible sick, dying and dead puppies for the establishment of accurate diagnoses. In the hands of progressive veterinary surgeons, this more enthusiastic approach leads to higher success rates in diagnoses and survival rates but most importantly, proposing of preventative protocols.

It is important that breeders realise the effect of early intervention on outcome in case of neonatal disease. In the case of adults the time interval from the start of first clinical signs of disease until death may in some cases be in the order of days. In stark contrast to this, in many cases in neonates, this period may be in the order of hours. This is because neonates, relative to adults, have low energy reserves, lower resistance to disease, poor thermoregulation, poor resistance to dehydration and generally poor ability to maintain homeostasis in adverse conditions. Sick puppies should therefore be treated as soon as possible. Dead puppies presented for post mortem examination should preferably be presented on the day of death and kept cool on ice in the interim or kept in refrigeration. They should not be frozen as this spoils the opportunity to perform a post mortem and some laboratory tests on them.

**Therapeutic principles in puppies**

Irrespective of cause of disease, the therapeutic principles in all these puppies remain the same. In neonates, the veterinary surgeon must address hypoxia, hypoglycaemia, hypothermia and hydration using the paediatric treatment principles. These principles are to:

• Ensure delivery of oxygen

• Administer energy in form of glucose solution

• Provide heat

• Administer balanced electrolyte fluids to correct hydration status

The doses of therapeutic agents should be tailored to suit the neonate’s metabolism. Fluids and drugs can be administered orally but in cases where the puppy is in shock the fluids should be administered intravenously in the jugular vein, intraperitoneally (in the abdominal cavity) or subcutaneously. To the breeder it is important to make sure that the temperature of the puppy is normal before milk replacers are fed to the puppies. This is because at a lower temperature, the enzyme systems are not as active, the peristaltic movements of the gut halts (termed ileus) and the puppies are left incapable of digesting the milk. This leads to fermentation, bloating and later regurgitation and aspiration on follow up feeds. It is a good idea for breeders to have at hand an electrolyte solution containing some glucose which they can administer orally to sick puppies. This mixture should be heated to body temperature before administration.

**14.2. Neonatal disease and conditions in puppies from birth to 3 weeks of age**

There are a host of conditions which are lumped together under this heading. This is because to the breeder all these conditions appear to be “one syndrome”. In young neonates, irrespective of cause, all the puppies show the same symptoms. The clinical signs of sick puppies under 3 weeks of age are generally poor suckling reflex, weight loss, slight diarrhoea, bloating, slight cyanosis of mucous membranes and tummy skin (turning bluish) and continuous crying of puppies. The bloat probably accounts for the apparent display of abdominal pain when handled. It is also important to realise that a compromised puppy, bigger or smaller than its littermates, irrespective of cause, will have difficulty in competing for feeding space on the dam’s teats. Also they may have poor sucking or no sucking ability and therefore intervention via tube feeding is indicated. Maternal neglect is usually pronounced and deliberate in weak puppies’ calling for intervention.

***Low birth-mass puppies***

Low birth-mass puppies should be identified at birth. Puppies which were of normal birth weight can appear to look smaller in comparison to its littermates at 5 days of age, due to failure to gain weight. Low birth-mass puppies have decreased ability to breathe and maintain metabolic function. They are also more susceptible to infection, hypothermia, hypoglycaemia and generally have a decreased chance for survival. These compromised puppies usually die within the first few days after birth. There is usually a spread of birth weights in big litters, but puppies weighing 25% below average for the breed are considered low birth-mass. There are many causes for low birth-mass. In very large litters, limited uterine space and placental insufficiency may lead to impaired nutrition of the foetus. Herpesvirus infection may also be a contributing factor in susceptible bitches. In some species, the critical birth-mass below which an individual neonate cannot or is very unlikely to survive, irrespective of intervention, has been established. These values are not known for dog breeds. The significance of this discussion is that if a puppy died which was suspected to have been a low birth mass puppy, the cost of diagnostic work ups are probably not warranted because it is less likely to give conclusive answers. Due to their poor prognosis, some breeders elect to euthanize them early on. This is different from cases where entire litters are affected or apparently normal puppies abruptly die. In these cases proper work ups and interventions are more likely to be fruitful.

***Weak puppies***

Weak puppies are discussed separately from the others because if the weakness is the only clinical sign in absence of other pathology, it may be easily reversed using the general paediatric principles of intervention. The puppy may have become temporarily hypoglycaemic or hypothermic and if corrected the puppy quickly regains strength, and its suckling reflex. The main cause for weak puppies is dystocia and prolonged birth. Unfortunately a number of these puppies will develop respiratory infections due to the aspiration of meconium prior to birth. Dams or their owners neglecting to remove meconium from puppies may lead to bacterial growth on new-born leading to neonatal dermatitis. Maternal neglect and poor mothering may lead to trauma to the puppy and weakness. There is the common notion that a dam has the ability to identify underlying problems with puppies and therefore selectively neglect those individuals hence deliberate neglect. Breeders should not fully trust this “instinctive ability” as many puppies which turn out perfectly normal can be saved from this neglect. When the weakness is a result of another underlying, yet unidentified cause, the recovery will not be that prompt. In these cases the underlying cause must be identified.

***Neonatal septicaemia***

Puppies have immature immune systems during the first 2 weeks of life, making them very vulnerable to infections. Colostrum deprived puppies are at an even increased risk for infections at this age. Puppies which were subject to a difficult birth, or were exposed to chilling are also at increased risk. The incidence of septicaemia also increases in puppies of dams suffering from urogenital or mammary gland infections. The value of clean whelping quarters and the cleansing of bitches have previously been discussed and may help prevent infections. It is very likely that once the first puppy presents with the symptoms others will soon follow. Neonatal septicaemia is caused when the blood is invaded by bacteria which cause an infection in all the organ systems and blood. The layman term is blood poisoning. The bacteria may enter the body through the umbilical cord but entry through other routes is also possible. Umbilical hygiene soon after birth cannot be overemphasized. Omphalitis is infection of the umbilical site and is characterised by redness and wetness of the umbilical area. This may progress and spread to cause septicaemia. The source of the bacteria may be the environment or the dam. In addition to the normal clinical signs of illness in puppies, septicaemic puppies may show respiratory distress from fluid build-up in lungs.

Broad spectrum treatment which includes the gram negative spectrum is indicated in these cases. However, treatment of advanced cases is usually futile as they die within 2 days. Appropriate antibiotic therapy of litter mates showing either no symptoms or early symptoms may help prevent further losses.

Neonatal tetanus is a very rare cause of death in puppies. Despite the occasional typical case (tetanic spams) seen by veterinary surgeons and breeders in puppies at the age of 3-5 days (younger than the 6-8 days in humans), it has not been well documented in the dog. The few cases the author has seen have all originated from warmer climates and premises where other livestock (especially horses and cattle) are kept.

***Colic puppies***

Colic refers to abdominal pain caused by accumulation of gas in the intestinal tract. The gas build-up may be caused by the milk formula or by gut stasis. Stasis of the gut may be the result of any number of diseases. Eventually all sick puppies will show some extent of abdominal distension and colic. Unlike babies, colic in puppies may be fatal and is difficult to treat. It may be argued that it is the underlying cause of the colic that will be the cause of death. It is however also true that those puppies which were previously perfectly normal and received milk replacer may develop colic and die. Inferior commercial milk replacers and inappropriate homemade milk replacers are more likely to cause colic but even the very best formulas may still cause bloat. Increasing the fat percentage appears to reduce the fermentative capacity of the milk and reduce incidence of bloat in the hands of the author.

At the first signs of colic, the milk formula can be withheld for one meal and a 5% glucose/electrolyte fluid can be fed orally. At the next meal the formula (best quality possible) should be used and its fat percentage be slightly increased as explained and mixed 50:50 with the glucose mix. The following meals the puppies can be fed the enriched milk formula only. The puppies can also be treated with antispasmodics, drugs to induce gut motility as well as prebiotics.

***Toxic milk syndrome***

Toxic milk syndrome is a poorly defined syndrome recognised in puppies nursing on a bitch which is presumed to secrete toxins in her milk as a result of an infection of the uterus or mammary glands. Signs suggesting toxic milk syndrome are if all the puppies in the litter show illness simultaneously and if the bitch has a low grade fever and is not eating that well. There need not be clear evidence of infection in the bitch externally. The puppies will bloat, cry, show diarrhoea and strain continuously. The suspicion of toxic milk syndrome can be confirmed if the puppies are removed from the bitch and show an immediate recovery. The bitch should also be treated. Once the bitch has fully recovered, it may be considered to reintroduce the puppies. Care must be taken to use drugs which are safe for the puppies. Unfortunately, in most of the cases the removal of the puppies combined with the illness in the dam may have affected her milk production completely or partly and continued supplementation of the puppies may be required. In some cases the uterus infection may be so stubborn that it requires either long term therapy or even sterilisation in bitches not intended for further breeding. Clearly in these cases, hand rearing is the only option. Toxic milk syndrome is fatal in puppies which are not removed from the dam.

***Congenital abnormalities***

It is by no means the intention of this book to cover all the possible congenital abnormalities in all the breeds. Only a few of the more common abnormalities are mentioned. There are good books available on the congenital defects of the dog in breed context. It is of importance that breeders familiarise themselves with the more common abnormalities in their breed. If they notice an abnormality in a puppy which is unfamiliar to them, it is recommended that they present it to their veterinary surgeon to at least get an indication of what it could be. This allows the breeder to put a name to the abnormalities for record keeping purposes. The significance is that when breeders get repeat occurrences of abnormalities, this may alert them to possible genetic problems.

**Cranial clefts**

Cranial clefts (also known as open heads) is a condition known to dog breeders characterised by a skull which has not closed properly during embryonic development and the resultant puppy is born with a cleft in its skull with the brains frequently protruding from this cleft. The eyes also seem to pop out of the skull. These puppies are mostly alive at birth. This defect seems to be part of the midline closure defects like spina bifida, hemi vertebrae and cleft palates. Cranial clefts appear to be reported more often in the Bull Terrier breed but the author has photographic evidence of this defect in numerous small and large breeds.

The condition is well documented in humans, laboratory animals and pigs. The condition in humans has been linked to folic acid metabolism and in animals to folic acid and manganese metabolism. It is not necessarily a diet deficient in manganese or folic acid that is implicated but rather a deficit in folic acid metabolism which increases the requirement of both folic acid and manganese. It is speculated that this defect folic acid metabolism is hereditary. It is not known whether the defect seen in dogs is primary a hereditary defect as is hemi vertebrae or whether a defective folic acid metabolism is involved. There is no scientific evidence to support whether folic acid supplementation can indeed prevent cranial clefts in dogs. We do know however that moderate supplementation of folic acid cannot harm the pregnant bitch. More research is obviously required regarding this mysterious syndrome. To the sceptics, it is true that the condition exists albeit rare (photographic evidence is available) and it occurs in dogs fed a balanced diet that are not suspected to be deficient in any nutrient whatsoever.

**Cleft Palate/Cleft Lip Complex**

Harelip and split palate (cleft lips and/or palates) are two conditions which result from failure of the maxillary buds to fuse in the canine foetus at around midterm-pregnancy. Cleft of the lower lip is rare and usually occurs on the midline. Clefts of the upper lip are usually just off the midline and usually unilateral. They may or may not be associated with clefts of the palate as well. The most common defect however is the split palate on its own.

Distinct breed incidences have been reported but this abnormality may occur at a low frequency in most breeds. In some cases, sporadic instances are due merely to accidents of development at the embryo stage. However, if a number of cases recur in the same strain or breed, a genetic basis should be suspected. Brachycephalic breeds can have up to a 30% risk factor. A specific gene has been identified that controls this event and plays a crucial role in the folate cycle. Work is in progress to identify a specific marker so carriers can be detected. Although the primary aetiology (cause) is thought to be hereditary, drug or chemical exposure, mechanical interferences with the foetus and some viral infections during pregnancy have also been implicated. In humans the maternal supplementation of folate very specifically during the first month of pregnancy reduces the risk of cleft palates in children of predisposed woman by up to 50%. Studies in Boston Terriers and French Bulldogs have shown the same. It is therefore justified to supplement folate in at-risk breeds at doses of around 5 mg folic acid per day from around mating till last quarter of pregnancy. Some breeders wish to continue till near parturition. The puppy born with a split palate is easily identified as milk pours from its nose whilst attempting to suckle and it soon loses weight and fades. This condition can in some cases be surgically corrected when the puppy is weaned (after extensive tube feeding). It is advised that this puppy be sterilized and certainly not bred from.

**Hernia of the umbilicus or inguinal regions**

A hernia is a failure of the muscular part of the abdominal wall to close but the skin overlying it is closed. When this happens it allows abdominal content to protrude through the defect in the abdominal wall and become visible as a bubble or lump of tissue underneath the skin. In case of an umbilical hernia, it is failure of the umbilical ring to close. The hernia is expressed as a soft lump in the middle of the abdomen. Umbilical hernias may vary in size from a few millimetres to a few centimetres. Some may extend up into the diaphragm. The incidence in the general population is low but it may vary between breeds and blood lines. This observation is highly suggestive of an underlying genetic role. Very often the breeder will blame the bitch for having pulled the umbilicus and torn the muscular wall leading to the hernia. In cases where the puppies were delivered by caesarean section the breeder may suspect that the veterinary surgeon may have pulled on the umbilical cord leading to the tearing and formation of a hernia. This is not true. It is true that some small hernias may “seal off” with a piece of omentum (net of fat in abdomen) and will never show any clinical sign associated with the hernia. This has led many breeders to believe that delayed closure of the umbilicus does happen in some dogs. The latter is probably untrue. Until proven otherwise, it is safer to assume that an umbilical hernia is of genetic origin. Hernias can be readily surgically corrected but excessive scarring is frequently encountered. Inguinal hernias are hernias of the inguinal ring. This is the region between the hind legs where the thigh and abdomen meet. The inguinal ring is a slit in the musculature in this region. If the ring is too lax it allows abdominal content (usually fat and intestine) to protrude through the muscle and become visible under the skin in that region. It is potentially fatal and requires early surgical correction. In both cases the correct thing to do is not to breed with affected animals and select against this defect.

**Omphalocoels and gastroschisis**

Omphalocoels and gastroschisis refers to incomplete closure of the abdominal wall and skin, stretching over a large distance on the abdominal midline and unlike umbilical hernias, are not restricted to the area surrounding the umbilicus. In both these conditions the defect in the abdominal muscular wall and skin allows abdominal organs to protrude from the abdomen. It is usually the intestines which are protruding but liver and other organs may also be visible. If the extra-abdominal viscera (abdominal contents) are not covered by peritoneal membrane, the condition is termed gastroschisis and if it is, it is termed omphalocoel.

**Congenital hydrocephalus**

Hydrocephalus is the abnormal accumulation of fluid in the ventricular system inside the brain. There are numerous causes of hydrocephalus and it may be congenital or acquired. Congenital hydrocephalus is by far the most common and toy and brachycephalic breeds are most commonly affected. In some breeds a hereditary origin is suspected. Hydrocephalus can easily be diagnosed by virtue of the greatly enlarged head of the puppy relative to the rest of its body. The skull may also appear dome shaped. The dome may appear to have a soft spot where no skull bone can be felt. This is referred to as a fontanel. It is however important to note that some toy The latter is of academic interest only. What is of practical interest is that this defect may occur in any breed and may be of genetic origin. Breeders may suspect that the bitch caused the defect following birth. As with umbilical hernias, they may also suspect that the veterinary surgeon may have caused it by excessive traction on the umbilical cord during delivery by caesarean section. Affected puppies should be euthanized. Some breeds (mainly Chihuahuas) may have open fontanels and this may be perfectly normal in them. Unlike children, puppies of most breeds are not born with fontanels. Also, unlike children, in breeds where the puppies are born with an open fontanel, it does not close in time and remains a permanent feature of the dog in adulthood. Lastly, it appears that breeds in which open fontanels is a normal feature, are predisposed to hydrocephalus. Puppies suffering from hydrocephalus may be either symptomless or show an array of neurological signs varying from retardation, epilepsy, loss of balance and many other signs. It is usually advised that these puppies are euthanized.

Swimmer puppies are ones that have their limbs spread out sideways. They are unable to stand or walk. The cause is unknown but hereditary factors may be involved. Swimmers may occur in litters of any breed, although medium to large breeds are overrepresented. Environmental factors include slippery surfaces, excessive milk consumption, limited crawling and walking space or opportunity. Treatment involves taping the legs together (hobbling) and providing a non-slip surface to walk on. Severely affected puppies which do not respond to treatment may have to be euthanized.

**Limb defects at birth**

There are numerous defects of puppies born with deviation of limbs in any direction. Some are ascribed to developmental defects due to “positioning” or limited space in the uterus. Whatever the causes, many of these defects may spontaneously resolve before weaning age and such puppies should be given the benefit of time. Abnormal number of limbs has also been reported as a rare congenital defect named polymelia.

**Pectus excavatum (flat chest)**

This is a congenital malformation of the sternum and ribcage causing flattening of the chest. Some flat chested puppies are swimmers as well. These puppies may show respiratory or cardiovascular symptoms or be asymptomatic. As with swimmers, they will first be noted around 3-4 weeks of age. Severely affected puppies may have to be euthanized.

**Hydrops foetalis (anasarca puppies, oedematous puppies, walrus puppies, jelly puppy,**

**water puppy)**

This condition has numerous synonyms. It is a condition where the puppy has massive water accumulation present underneath the skin and this is clearly visible at birth. Most of these puppies are too big to allow for natural birth and have to be delivered by caesarean section. The exact cause remains unknown but a genetic cause is suspected. These puppies can easily be detected on ultrasound and will predict a caesarean section. Some mildly affected puppies may survive with careful nursing. Most of these puppies however, die within minutes after birth due to breathing difficulties caused by fluids on the lungs. The condition is by far the most common in Bulldogs but can occur in almost all other breeds.

***Fading puppy syndrome***

This “condition” is aptly dealt with last under this heading. The fading puppy syndrome is a layman term breeders use to describe puppies which were apparently born healthy, but within a few days of birth fail to thrive, become weak and die. Once dead, fading puppies usually have no clear discernible cause identifiable on post-mortem examination. This syndrome should not be considered a diagnosis or condition. The lack of knowledge of the true causes of most neonatal illnesses or death has led to this collective term. It is probable that any number of undiagnosed conditions may cause the syndrome. As mentioned before, irrespective of the initial cause of illness in these puppies, the clinical signs will end up being similar in all cases. These symptoms are; a puppy which gets weaker, loses weight, cries and dies. These puppies may have; an underlying infectious cause, suffered from shortcomings in animal husbandry, been subjected to traumatic cause or have congenital defects. This syndrome has very little significance, except for the fact that if breeders are plagued with an unacceptably high percentage of “fading puppies”, they should in consultation with their veterinary surgeon start looking for the underlying cause. More research is required on this important subject.

***Puppy strangles (juvenile cellulitis, juvenile pyoderma, submandibular abscesses)***

Puppy strangles is a poorly understood condition which usually occurs at the age of 2-4 weeks. Puppies with strangles may show one or more of the following clinical signs; huge fluctuating swelling under their chin filled with puss, swollen submandibular glands (glands under chin), pustules on chin, muzzle or ears or abscesses in one or more joints. The treatment involves lancing the abscesses combined with corticosteroid therapy. Despite the fact that most of these abscesses appear to be sterile, antibiotic therapy combined with corticosteroids makes perfect sense in these cases to prevent secondary infections. The fact that frequently more than one puppy in the litter is affected, suggests some infectious cause despite failure to isolate infectious organisms in most of these cases. Polyarthritis is sometimes associated with the sub-mandibular abscesses. Some anecdotal evidence suggests that preventative treatment with antibiotics and steroids for litter mates of puppies with puppy strangles is helpful. Treatment of affected puppies is mostly successful. In contrast, untreated cases frequently die.

***White scours***

White scours is a very apt name given to a form of diarrhoea seen in puppies 2-4 weeks of age that are still nursing. Unlike dogs, it is a well-recognised condition in domestic livestock. The condition is characterised by the sudden onset of profuse white diarrhoea (semi congealed) without mucus or blood. Based on bacterial culture of stool and response to treatment using gram negative spectrum antibiotics, gram negative bacteria such as *Escherichia coli* and others

**Neonatal disease and conditions in puppies from 3 weeks of age till weaning**

***Neonatal opthalmia (opthalmitis neonatorum)***

Neonatal opthalmia is a condition where the puppy develops build-up of puss behind its eyelids before they have even opened. The breeder may notice that the eyelids are bulging or that they have partially been opened with some puss emerging from them. In some cases the only clinical sign may be eyelids which appear pussy and are stuck to each other. It is thought to be caused by bacterial infection. It may occur in one or both eyes and generally when one puppy presents with the first signs it is highly probable that more if not the entire litter may develop it soon. The eyelids should be gently forced open and the eyes washed out with an appropriate eye wash.

***Infectious juvenile pneumonia***

This condition is often associated with large breeding kennels which host a large number of susceptible puppies at any given time. Typically these puppies will be 3-5 weeks but puppies as young as 1 week or as old as 7 weeks may be involved. Initially the condition will spread to puppies of similar age (seldom young adults and older). Initially, the condition appears benign and frequently a mild tracheobronchitis (Kennel Cough) is suspected. Later it becomes evident that the condition is poorly responsive to treatment and the dogs develop a full blown pneumonia and sinusitis with puss running from their noses. The exact cause is not known but is probably multifactorial including colostrum deprivation, chilling, herpesvirus infection, bordetellosis and other kennel cough infectious agents. Although some puppies may survive following treatment, many may resume coughing after apparent recovery. Many of the affected puppies are euthanized due to failure to respond to treatment and expense of the protracted treatment. Attempts at controlling an outbreak should include isolation of affected individuals, improving ventilation, raising environmental temperature and treatment of in contact littermates and litters. Including a Bordetella vaccine in your vaccination regimen (particularly breeding dams) may help prevention of the condition if bordetella is involved.

***Hypoglycaemia in toy breeds***

Hypoglycaemia means a low glucose (blood sugar) level. Glucose is the basic form of energy which the body uses for all its functions. It is formed following digestion of foods and is stored in the form of glycogen in liver and muscle. New-borns have very little fat and glycogen reserves. They also lack the metabolic capacity to generate glucose from precursors. Therefore neonates that are milk deprived for a couple of hours may develop hypoglycaemia. In contrast to the neonate, most puppies at post weaning age that are eating well and in good condition have adequate ability to quickly mobilise glucose from their stores and replenish these stores from food ingested. However, some puppies of the toy breeds appear to retain infantile glucose metabolism. As a result, they have a limited storage capacity of glucose and may very quickly develop hypoglycaemia if they skip as little as one meal or if they develop the mildest of illnesses. Most of these puppies will become less prone to hypoglycaemia when reaching adulthood but they never really fully outgrow it. Smaller individuals within the breed which struggle to maintain a good condition appear to be at increased risk. Hypoglycaemic puppies will only appear lethargic at first but later develop poor coordination, balance loss, seizures, opisthotonus (pulling back of head and neck) and finally coma and death. Breeders whom specialise in breeding of the so called “teacup” sized dogs are familiar with the risk of hypoglycaemia in their puppies. To avoid this they normally advise their new owners to feed the puppies (3-4) small meals per day and always keep a glucose solution at hand for emergency use.

**Neonatal disease and conditions in puppies from weaning onwards**

The stress associated with weaning is significant. Stress combined with infectious agents may path the way for a great number of conditions which in isolation would otherwise not have caused a problem at all.

***Concept of erosive and multifactorial disease***

An erosive disease is a disease which seldom seriously affects the animal on its own but needs many other factors or agents to simultaneously impact on the animal in order to cause more serious disease. For the purpose of explaining these concepts, worm infection (verminosis) and coccidiosis will be used. In puppies for instance, the mere presence of coccidia in the stool will have little effect on the well-being of the puppies. Neither will a mild worm infection. If the puppies are however stressed due to any number of the following; insufficient milk intake, weaning stress, cold weather, crowding and poor hygienic conditions, then the host animal will become somewhat immune compromised and the worm burden increases or coccidian oocyst counts in stool increase. Under these circumstances the gut wall becomes compromised and other protozoa (spirochaetes for instance) which are normally ordinary commensals become pathogenic and the damaged gut may become secondarily infected with bacteria as well. These agents (worms, coccidia, spirochaetes and nonspecific bacteria) now together cause an enteric disease characterised by maldigestion and malabsorption seen as diarrhoea with sometimes blood specs on it. Typically these puppies may have a stunted growth, eat poorly, be thin and be poor doers. Often the diet is erroneously blamed for this condition and diarrhoea in these puppies. Clearly these puppies are now even more immune-compromised. Therefore, even a small infective dose of an enterovirus (for instance parvovirus) or Giardia or both are needed to cause an absolutely devastating parvovirus or giardia outbreak. In this example, both the coccidia and worms had an erosive effect on the host and together with other adverse influences; a so called multifactorial disease complex was created. Many other combinations of infection may under these circumstances synergistically cause severe disease. Individually however they would not have had a serious impact. It is important that both the breeder and their veterinary surgeon understand the concept of multifactorial disease. Failure to recognise its existence leads to failure to control certain diseases. The prevention of multifactorial disease lies in anticipating it, knowledge of contributing factors, knowledge of identity of participating infectious agents and employment of routine preventative measures and therapies against a multitude of infectious agents. Addressing hygienic matters also reduces risk.

***Impact of crowding on disease***

Although any condition or disease may afflict dog populations of any size, there tends to be an exponential increase in the number of problems directly associated with the size of a breeding colony. This happens not only because the stress factors increase in number and intensity but also because crowded conditions favour organisms which can potentially cause disease. For the purposes of this discussion a large breeding population is four active breeding bitches or more. In kennels dogs tend to be kennelled to avoid fighting (particularly the males of most breeds). More dogs are kept per surface unit and there is closer contact between these individuals. Hygienic conditions are more difficult to maintain. There occurs an increase in pathogen build-up in the environment e.g. viruses, bacteria, worms, protozoa and fungi. The re-infestation rate of mentioned pathogens which we wish to control is much higher. There is also evidence to support that, crowding increases the ability of pathogens to induce disease via various mechanisms. In crowded situations the impact of carrier animals (carrying disease but not showing it themselves) is more significant. In actively breeding kennels, there is a continuous supply of more susceptible individuals in the form of new-born puppies. The end result is that pathogens which would otherwise be normal commensals (harmless residents) now become part of disease syndromes. Also the protection of dogs against the various diseases in kennels needs more complex measures than is the case in household situations with individually kept dogs.

***Non-specific diarrhoea at weaning age***

Diarrhoea is the most common complaint by new owners in recently sold puppies. In addition to the nutritional diarrhoea at weaning due to diet change already discussed in 13.5, nonspecific diarrhoea may act as a source of irritation to breeders. In these cases no specific cause of the diarrhoea can be established. In most cases this diarrhoea will be self-limiting and spontaneously disappear as mysteriously at it came. In some cases however, bacterial overgrowth of the small intestine may be diagnosed. Bacterial overgrowth may also have been induced by the breeders’ or new owners’ attempts at self-treatment using various antibiotics. Specific intestinal prescription diets for diarrhoeic puppies combined with probiotics and colostrum based therapies may help speed up recovery. In stubborn cases or cases where the diarrhoea is associated with vomiting, more serious causes should be suspected and veterinary attendance sought.

***Canine parvovirus***

Canine parvovirus is the most common cause of serious enteritis in young dogs. It is a highly contagious disease which occurs worldwide. It remains a serious problem in recently sold puppies and in breeding kennels. Most breeders have either heard of this dreaded disease known as “canine parvovirus” or may have had the misfortune of having lost puppies to it. Parvovirus infection is also known as “cat flu” in some countries, but this is a misnomer, as cats have little to do with the virus. The disease is characterised by lethargy, anorexia, vomition, severe diarrhoea, dehydration and death if not treated promptly.

Treatment involves lengthy hospitalisation including intensive fluid and antibiotic therapy. Other causes of gastro-enteritis may mimic parvovirus infection. Corona virus is commonly touted as culprit but plays a minimal role and may only be of importance if associated with parvovirus. It is important for the breeder to realise that parvovirus infection in a breeding kennel may present in a much more severe form as opposed to individual puppies which fall ill after sale at the new owner’s home. Puppies of breeders whom have several litters of similar age simultaneously are at increased risk of a serious outbreak, with increased mortalities. This is because the parvovirus may start spreading from one puppy to the next and from one litter to the next despite hygienic measures and isolation. What follows is an outbreak of parvovirus in the breeding kennel and a disaster in the making. During an outbreak of parvovirus infection in breeding establishments, a large number of puppies excrete virulent virus in unusually high numbers, resulting in very high doses of virus available to infect new susceptible puppies. This, in combination with stress factors associated with overcrowding, may explain the observation that the severity of parvovirus associated gastro-enteritis and mortality in breeding kennels, despite treatment, is far greater than that encountered in general practice using the same treatment protocol. Intensive treatment of an infected puppy will usually be successful in about 80% of cases when small numbers are involved. However, in contrast to this, only 20% of puppies may survive despite the same treatment in cases of a parvovirus outbreak. Unfortunately, the cost of treatment may surpass the value of the puppy and hence some breeders and owners may elect to euthanize affected puppies.

In addition, the age of affected puppies tend to be older (7-10 weeks) in pet homes than those of affected puppies in breeding kennels (5-7 weeks). Parvovirus is ubiquitous, hardy and may persist for long periods of time in the environment. Large breeding concerns have continuous movement of both animals and people on their premises. These cannot be considered closed isolated kennels. The implication is that the introduction of problems like verminosis, coccidiosis and other causes of gastro-enteritis cannot be adequately controlled. Keeping a closed kennel and maintaining hygiene are good principles which will decrease risk but certainly not eliminate it all together. Living with the threat of parvovirus may be a source of huge frustration to some breeders. This is because strict isolation and hygienic measures do not always adequately protect against the onslaught of parvovirus. This virus is so ubiquitous that it somehow slips into the kennel. Although direct transmission from dog to dog and indirect transmission via virus carried on shoes, clothing and hands are the most likely routes of transmission, fomite transmission of parvovirus is speculated to also be possible. Fomite transmission is where an infectious agent is carried on dust and other particulate matter in the air. This may explain why parvovirus associated enteritis is more common following the windy season in some countries. Another frustrating observation by breeders is the inexplicable occurrence of parvovirus in one kennel whereas the other remains unaffected despite the fact that the former may have superior management and hygiene. One explanation for this phenomenon may be the concept of herd immunity. The herd in this context refers to huge populations of dogs which reside in a geographic area. In areas where herd immunity has been achieved (when the percentage of vaccinated dogs exceeds 50%) this immunity will diminish the circulating virulent virus in that area and help protect many of the unvaccinated animals (domestic and wild) which are susceptible to the disease in question or unprotected animals (puppies which have not completed their vaccination program). This phenomenon may help explain why one breeder consistently reports great success in control of parvovirus and others fail in contaminated environments using identical vaccination protocols. Geographic pockets where herd immunity against parvovirus has been achieved are generally areas where cultural perceptions favour vaccination and other veterinary expenses on dogs. Breeders finding themselves in such geographic pocket where herd immunity is achieved, erroneously hold the view that they have the “correct recipe” to control parvovirus. If these breeders however, sometime and somehow, introduce parvovirus in their kennel, they will experience the full brunt of parvovirus. It has been speculated that certain breeds are at higher risk for parvovirus infection than others. Although Rottweilers, American Pit Bull Terriers, Doberman Pinschers and German Shepherd

Dogs and some other breeds are reputedly at increased risk, breeders of other breeds should fully understand that, in case of a serious outbreak, parvovirus does not discriminate against breed. Under these circumstances all young susceptible puppies, irrespective of breed are likely to be equally affected. The cornerstone of prevention of parvovirus infection in young dogs remains vaccination, but this has many shortcomings which require explanation. Puppies acquire immunity from their dam through ingestion of colostrum. If the dam was adequately vaccinated against parvovirus, this colostrum will contain antibodies against parvovirus. If not, the puppies will be susceptible to parvovirus almost immediately after birth. These antibodies protect the puppies against parvovirus for a period which varies for the first 6-10 weeks of life. However, the same antibodies may prevent response to vaccination. Scientists refer to this phenomenon as the interference of maternally derived antibodies with vaccination. Therefore numerous vaccinations, 3-4 weeks apart may be required to adequately immunise a puppy. This has some important implications. This phenomenon leaves the puppy vulnerable to parvovirus from the age of about 6-11 weeks, also called the window of susceptibility. Early studies following the emergence of parvovirus demonstrated significant interference by maternally-derived antibody to vaccination of puppies. This observation has aided to advance the practice of accepting 6 weeks as the earliest age of vaccination against parvovirus. However considering that parvovirus may affect puppies in breeding kennels at a younger age, it thus follows that it may be prudent to vaccinate puppies in infected breeding kennels at 4-5 weeks of age, rather than the standard 6 weeks in order to shorten the window of susceptibility. Early vaccination (4-5 weeks) against parvovirus is viable and recommended under these circumstances. Special parvovirus vaccines (so called high titre vaccines) registered for this purpose are available in most countries and are effective even in puppies with high levels of maternally-derived antibodies. Veterinary surgeons acting as consultants to large-scale breeders are well aware of the significance of parvovirus infections as a constant threat to susceptible dogs, resulting in high morbidity and mortality. They should acquaint themselves with the use of these vaccines and breeders at risk should enquire about them. These vaccines may save many puppies’ lives. As explained, puppies appear to be at their most vulnerable at around 6-11 weeks. This corresponds with the time that puppies are weaned, vaccinated, dewormed, relocated and exposed to the new owner’s environment and other pets. This is a very stressful period in a puppy’s life. It should therefore come as no surprise that recently sold puppies frequently become infected with parvovirus. This also explains why some breeders and many owners of newly acquired puppies erroneously hold the view that the parvovirus vaccination caused parvovirus rather than prevent it. All that indeed happened in the above scenario was that the puppy still had enough antibodies in its bloodstream to prevent a proper response to the vaccine but not enough to prevent infection from exposure to the real parvovirus as soon as it left the breeders premises. Puppies which fall ill soon after sale are a source of frustration to the breeder. This is because the new owners in these cases are likely to claim that the puppy left the breeder already ill or incubating the disease. The incubation period of a disease refers to the period that lapses between exposure of the puppy to an infectious agent and the onset of symptoms of the disease caused by the infectious agent. In case of parvovirus, the incubation period may be as short as 2-3 days or as long as 10 days. Breeders whom consistently have problems with parvovirus soon after sale should also consider earlier vaccination. Due to the myriad of problems which newly sold puppies experience and the frustration it brings to the breeder, some breeders elect to sell puppies way after this vulnerable age. In these cases the breeders take care of adaptation to new food, almost complete their deworming and vaccination programs and sells them at the age of approximately 11 weeks. This practice may not be practical for the larger breeder who will run out of space and hands to care for these puppies. This also exposes the breeder to increased risk of disease outbreak other than parvovirus. With regards to adult dogs, annual boosting against parvovirus is the norm. There are however vaccines which last longer in dogs which require boosting every third year only. For the time being, every third year vaccination or longer is not recommended in actively breeding bitches. This is because a dam may lose a significant portion of her antibody levels at sequential pregnancies with large litters, which may compromise her ability to adequately protect future litters against infectious disease. It is therefore suggested that, at least in active breeding bitches, annual vaccination against the core diseases not be abandoned until further research suggests that this is safe. There are small animal vaccines registered for use in pregnant and lactating dogs and they may be used if the bitch requires her annual booster whilst pregnant. The deliberate vaccination of pregnant bitches 2-3 weeks before whelping in addition to annual boosters is practiced by some breeders and advised by some veterinary surgeons to ensure good colostral immunity. This practice has no value in bitches which are vaccinated annually. Breeders whom have experienced parvovirus are quick to suspect that vaccine failure may have been the cause or indeed that the vaccine actually caused the problem in the first place. Vaccine failure may be defined as the inability of the vaccine to result in a protective immune response in the vaccinated animal. Although faulty vaccines have been reported, this is very rare indeed. It is true that some individual dogs may be poor responders to vaccines and fail to acquire immunity. If vaccine failure is suspected, large numbers of cases are expected to be involved and serological studies (studies of antibody levels following vaccination) are indicated and should be requested. Emergence of new parvovirus strains against which current vaccines do not protect is also a potential threat. Parvovirus is a common nosocomial (hospital acquired) disease. This has important implications for the breeder. Any animal hospital with the best intentions and management, may act as sources of infection to admitted patients. For this reason, extreme care should be taken in veterinary facilities to prevent transmission of disease to puppies when presented for vaccinations. When puppies are however admitted for any ailment, inadvertent transmission of nosocomial disease may be unavoidable. In these cases it is advised that the puppy either remains in the veterinary facility, boarding facility or at the very least, in the quarantine facility of the breeder until it has completed all of its vaccinations. It may also be homed straight from the veterinary surgeon to its new home where risk of disease spread is minimal. The surest way to trigger a parvovirus outbreak in a breeding kennel is to introduce a puppy shedding parvovirus from elsewhere. Translocated puppies are stressed, immune-compromised, more susceptible and thus more likely to, firstly become infected and secondly, shed sufficient virus to infect others.

In summary, it is strongly recommended that current vaccination guidelines for puppies and dogs be followed according to region. In high risk situations, earlier vaccination than the accepted norm, at 4 weeks, should be considered. Reduction, but not complete elimination of parvovirus-induced disease in large breeding kennels or in highly contaminated environments is a realistic expectation using this approach. The control of other pathogens in breeding kennels which act as immune-compromising factors may both aid in prevention of infectious disease and further reduce severity of disease and favour treatment outcome in puppies originating from breeding colonies.

***Nosocomial (hospital-acquired) disease***

Often an owner or breeder will object to an animal being hospitalised due to the possibility of a hospital-acquired disease. Usually with adult animals, this objection is not founded. This is because adult dogs which are fully vaccinated are very unlikely to contract infectious disease. In contrast, with puppies the risk of hospital acquired disease is real. This may leave the breeder with a dilemma. Leave the puppy at home and it is not treated properly and dies or infects other puppies anyway. Take it in the hospital and there is a small but real risk that it picks up another disease and brings it home. For this reason in puppies younger than 3 months, it is often advised that the hospitalised puppy is quarantined for a period of 2 weeks following hospital discharge. This quarantine may be at the hospital, at an isolated area at the breeder or at another location where there are no breeding activities and other puppies at risk.

***Canine coccidiosis***

Many species of coccidia infect the intestinal tract of dogs. Although finding evidence of this parasite in the faeces is very common, disease caused by coccidiosis on its own is not. Infection usually is asymptomatic and self-limiting in puppies. In multifactorial circumstances, coccidiosis may become a clinical entity. The most common clinical signs then are severe diarrhoea (sometimes bloody or semisolid stool with slime and blood on it), weight loss and dehydration. Canine coccidiosis may be primary and in exceptional cases have fatal consequences. Usually, coccidiosis is associated with other infectious agents e.g. spirochetes; worms; Giardia, immunosuppression, and acts as an erosive disease as well as an opportunistic pathogen, always waiting to cause disease in the presence of a precipitating cause. The source of infection is usually the pregnant bitch which infects the puppies soon after birth. Typically the bitch will start excreting coccidia oocysts from 2 weeks before labour until 2 weeks or later after whelping. The coccidia infection in the bitch is usually asymptomatic and their only significance is as a source of infection to the puppies. Special therapeutic agents were designed for the control of coccidiosis in other species and these are also effective in its control in dogs. These anticoccidial drugs may be administered to the bitch 2 weeks before labour (and 5 days later again) and again 2 weeks after whelping (and 5 days later again) in order to control coccidia oocyst shedding, reduce environmental load and prevent spread to the puppies. The puppies may be treated at 2 weeks (and 5 days later again), and at 6 weeks (and 5 days later again). These drugs appear to be safe during pregnancy but the responsibility for the use thereof lies with the prescribing veterinary surgeon. This is because these drugs are not registered for use in dogs. Sanitation is important, especially in kennels where large numbers of animals are housed. Faeces should be removed frequently. Faecal contamination of feed and water should be prevented. Runs, cages, and utensils should be disinfected daily. Excessive wet conditions (due to cleaning or weather), with extended periods where all surfaces are moist, seem to favour survival of the pathogen. Desiccation and ultraviolet light radiation (from sunlight) are effective in the control of many pathogens.

***Giardiasis***

Giardiasis is the disease caused by *Giardia lamblia* which is an organism found in the intestinal tract of many animals. Transmission occurs via oral contact with organisms in faeces of infected individuals. In dogs it is more common in kennels. Stress may result in higher parasite counts in the faeces and increased severity of disease. It may also be part of an erosive multi factorial disease complex. The clinical signs may vary from subclinical disease (not visible) to very severe enteritis, inappetence and weight loss. Giardia, given the opportunity via stressors, has the potential for a severe outbreak of gastro enteritis in both young and adult dogs. The best way to confirm giardiasis is to submit faeces samples of various dogs for examination.

Giardia is susceptible to most disinfectants and to desiccation. Allowing the kennels to dry and avoidance of pooling of water helps a great deal in reducing the environmental load of the organism and risk of reinfection. Effective treatment is available but may need to be administered for up to 10 days and must be administered to all affected dogs and in contact dogs. In some countries a vaccine is available but this control measure should not be fully relied upon and should be used in conjunction with other control measures.

***Ringworm (dermatophytosis)***

Dermatophytes are fungi which grow on skin and hair of many animals including man. Fungi produce spores which are the infective vesicles (eggs) and means by which the fungi translocate from one animal to the next. This transmission may occur following direct contact between animals or indirectly via spores which become airborne. Spores may remain viable in the environment for up to 18 months. Puppies and young adults are more susceptible. Immunocompromised animals and animals suffering from other skin conditions may also be at increased risk of ringworm infection. Although warm weather and high humidity may predispose animals in warm climates, these conditions can also prevail in colder climates where animals are kept indoors. In the dog, ringworm normally has classical presentation, namely a round area on the skin which may be slightly raised and appear hairless. The lesions may be focal or multifocal and heal from the centre, enlarging peripherally. Ringworm may in some cases itch and become infected with bacteria as well. The discussion of ringworm is not only important because it has the potential of reaching outbreak proportions in breeding kennels but also because it may wreak havoc in the home of the new owners. Puppies sold with an existing ringworm lesion or from stock currently infected are bound to develop a lesion soon after sale. They may also infect the new owner’s other pets and or the people in the household. This may lead to serious discontent and complaints. Therefore breeders whom have the misfortune of ringworm infection in their puppies should strongly consider keeping the puppies back until they have the outbreak under control. The incubation period of ringworm is 1-3 weeks. The implication is that puppies might have to be kept back for at least this period (3 weeks). Topical treatments may lessen the spore load but are often ineffective in treating the deeper ringworm lesions. Treatment with oral medication for 30-40 days may be required. If it is impractical to keep the puppies back for so long, puppies may be sent to their new homes provided it is with full knowledge of the condition and appropriate instructions for treatment. This should consist of systemic treatment which typically involves oral intake of tablets for 30 days as well as topical treatments (medicinal shampoos and creams) to aid in curing of lesions but mainly to reduce risk of spread to humans and other pets by reducing spores on the skin.

In cases where the breeder experiences continued re-emergence of the ringworm in new puppies they should attempt to either identify carrier animals in their breeding stock or treat all the in-contact animals on the premises whether they appear infected or not irrespective of age. They may also consider treating the environment using disinfectants known to be effective against fungal spores.

***Encephalitozoon***

*Encephalitozoon cuniculi* is a widespread infection of rabbits and occasionally of rodents and dogs caused by a protozoan (microscopic organism). The condition is rare in dogs but nevertheless requires mention because it is more likely to be diagnosed in dog kennels as opposed to pet homes. The disease may be spread from rabbits or rodents via their urine to the dog and thereafter from dog to dog. Once infected, the dog may be totally asymptomatic but shed spores of this disease (microsporidia) into its urine for extended periods. They may ultimately shed the infection and demonstrate antibodies in their blood against the disease for variable periods. Other animals acquire the infection by the oronasal (mouth and nose) route, when an animal licks/sniffs the spore-infected urine of another animal. This disease is not venereally transmissible. There is generally a low potential for an Encephalitozoon outbreak but this may occur in unsanitary conditions. Infected dogs, irrespective of what age they were infected at, will usually show no clinical signs. Only puppies from infected dams will normally develop clinical signs. The puppies acquire the infection as foetuses in utero (when inside the uterus). In these cases the pregnancy and birth progresses normally and the clinical signs manifest in the puppies. In highly exceptional cases, immune-compromised nursing puppies may acquire the infection soon after birth. The age of manifestation varies and may be as early as 3-4 weeks of age or as late as 12 months. The normal age at manifestation is usually around 3 months of age. This may be a frustrating condition to the breeder as at that stage, the puppies are usually with their new owners. The clinical signs are failing to thrive, depression, weight loss, ataxia (unsteady gait), head tilt, head swaying, blindness, hind quarter paresis and seizures. The parasite mainly affects the brain, eyes and urinary tract, causing encephalitis, cataracts, kidney failure and death in all affected individuals. An accurate diagnosis can be made by post mortem and histopathologic demonstration of the organism in the organs. In the live animal a diagnosis can be made on urinalysis as well as blood tests. There is no treatment for infected dogs and no vaccine to prevent it. Studies show that between 8-38% of dogs have been infected. This high prevalence of antibodies in dogs indicates that the parasite is a lot more common than the low incidence of confirmed clinical signs seem to suggest. This is probably because clinical disease only seems to occur after the small window when bitches are pregnant and transplacentally infect their offspring. This also explains its significance in breeding kennels. There is absolutely no need to euthanize a dam or in-contact dog which has produced puppies which died from Encephalitozoon. A bitch which has had one litter affected with Encephalitozoon is unlikely to subsequently produce another infected litter. Generally in a kennel, only isolated cases will emerge. The spores are very sensitive to most disinfectants and simple sanitary principles will usually adequately control the problem.

Keeping bitches away from the general dog population during pregnancy and preventing contamination of drinking water and dog foods with rodent urine may also aid in its prevention. Dog breeders should preferably not keep pet rodents or rabbits in the vicinity of the dog breeding kennels and should adequately control pest rodents. *Encephalitozoon cuniculi* is also a potential zoonoses but human infection is very rare

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