REVIEW ARTICLE

A review of Neonatal mortality in Dogs

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ABSTRACT

Neonatal period within veterinary medicine is considered to be the transitional phase from fetal to adult life during which there is changes in structural and functional systems of the animal. It is also the period when the systems of the animal move from the physical, chemically, and microbiologically protected environment of the uterus to face the adaptive requirement for survival in a complex environment. Neonatal mortality in dogs has caused a lot of loss among dog breeders and has always posed threats on the survival of young dogs especially exotic breeds. The causes of neonatal mortality in dogs are multi-factorial. Some of the risk factors may be classified as fetal, maternal, environmental and scoring risk factors. Proper knowledge on the associated risk factors will help to reduce the rate of neonatal mortality in dog thereby improving on dog breeding.

Key words: Bacterial contamination, Dog, Milk, Neonatal mortality, Review

INTRODUCTION

Pup mortality, both during parturition and in the neonatal period, is a significant clinical problem that is poorly documented in the veterinary literature. Mortality rates of 10-30% by weaning (Lawler, 1989) and 15-40% in the first 12weeks of life (Sturgess, 1998) are reported. The majority of pup losses are stillbirths and deaths within the first week of life, that is, perinatal mortality (Gill, 2001). Lawler (1989) specified 65% of the losses occurred in the first week with about half of these being stillbirth. Pup mortality has been attributed to a wide variety of causes according to Gill (2001) which include dystocia, stillbirths, congenital defects, low birth weight or runting, trauma and fading puppy syndrome. But Lawler (1989) specified dystocia and prolonged labour as significant causes of early death. Fifty-five percent (55%) of deaths in Blunden (1986) study were attributed to unknown causes and were ascribed to fading puppy syndrome. Undetermined causes were also

prominent finding in both Andersen's (1957) and Potkay and Bacher's (1977) reports.

DEFINITIONS

In previous studies there has been no consistency in the definition of what constitutes a stillbirth or the neonatal period, nor in the classification of mortality. The following definitions were used in this study:

1. **Stillborn**. A stillborn pup is defined as any pup born dead and included mummified pups and fully developed pups that had died prior to birth (autolysis present indicating death prior to the commencement of whelping).

2. **Neonatal Mortality**. Neonatal Mortality applies to those liveborn pups dying within the first six weeks after birth. A pup was judged liveborn if either respiratory effort or cardiac activity could be detected.

3. **Early Neonatal Mortality**. Early Neonatal Mortality applies to those liveborn pups dying in the first seven days after birth.

4. **Late Neonatal Mortality**. Late Neonatal Mortality applies to those liveborn pups dying between eight and 42 days of age.

5. **Total Mortality**. Total Mortality is defined as the combined loss due to Stillbirths and Neonatal Mortality.

6. **Perinatal Mortality**. Perinatal Mortality is defined as the combined loss due to Stillbirths and Early Neonatal Deaths.

CLINICAL FINDINGS IN NEONATAL DISEASES

Fox (1963) described the following clinical course in neonatal dogs that died between one and three days of age. 'There was a rapid decrease in weight after birth, with persistent hypothermia and slowing of the heart rate. Respiration also slowed and near the time of death prolonged spells of apnoea lasting as long as 60seconds occurred. Spasticity and postural flexor dominance (foetal posture) were characteristic of the syndrome in early stages. Titanic rigours with hyperextension of forelimbs and spine occurred shortly before death and often during or just prior to respiratory arrest. Other signs were similar to those observed in human infants and ascribed to cerebral anoxia; muscle flaccidity followed by titanic rigours and tonic-clonic ''walking'' with the hind and forelimbs. Post-mortem examination revealed various states of cardiac failure and circulatory arrest. This clinical syndrome of hypothermia and cardiopulmonary failure that develops and regardless of the cause is characteristically fatal. This characteristic clinical course is the result of the immaturity on the neonatal pup.

FOETAL RISK FACTORS

The newborn puppy is an immature animal, dependent on its dam for survival in the first three weeks and as a consequence the aetiology of neonatal death is frequently complex and often undetermined (Chandler, 1990; Blunden, 1998).

Fox (1963 and 1965) recognised that immaturity of the homeostatic cardiovascular mechanism in newborn puppies could result in their death from a cardiopulmonary failure syndrome. Rather than being the primary factor, cardiopulmonary failure is frequently the final common pathway for a puppy that is compromised by the other factors with or without the complications of infections (Chandler, 1990).

Prior to two weeks of age the heart is not responsive to atropine indicating the immaturity of the vagus nerve. The baroreceptors are not functional until four days after birth (Fox, 1965). The pulmonary response is also minimal in newborn puppies (Bright and Holmberg, 1990). This immaturity of the homeostatic cardiovascular mechanism can cause a vicious cycle of collapse and inadequate response that becomes irreversible (Chandler, 1990).

An important factor affecting the mean arterial blood pressure (MABP) in the perinatal period is blood glucose concentration. One study demonstrated an approximately 50% decrease in MABP in pups with severe hypoglycaemia (Hernandez *et al.,* 1980). This was believed to be related to the inhibition of contractile processes in the myocardium and/or smooth muscle in the peripheral vasculature. The newborn puppy is particularly vulnerable because of four major factors. These are, their thermoregulatory mechanism is poorly developed, and there is a risk of

dehydration, a risk of hypoglycaemia and immunological immaturity (Blunden, 1998).

a. Neonatal Thermoregulation

In all systems of whelping management, the newborn pup is introduced at birth into a cooling environment, i.e., an ambient temperature of less than 80 degrees F (26.2° C). Cooling is enhanced because the animal is wet. Deep body temperature falls during the first 20 minutes. The degree of hypothermia that develops depends on the speed at which the dam cleans and dries the puppy and on the environmental temperature in which the dam has whelped. The pup gradually regains a higher body temperature by staying in close contact with the mammary glands of the dam. Once whelping starts the surface temperature of the mammae is only a degree or so less than the deep rectal temperature of the mother (Crighton, 1968). In the face of cooling, or even the threat of cooling, the puppy seeks shelter. If this fails to arrest the cooling it then searches for a source of external heat which it is able to absorb to compensate for heat loss. Thus, provided a source of heat is immediately available, it can maintain a relatively stable rectal temperature despite a cooling environment. It is also suggested that the newborn puppy is a true homeotherm that substitutes compensatory thermal conduction for its lack of compensatory thermogenesis. The shivering reflex and vasoconstriction mechanism are not operant in the newborn (Crighton, 1968). Poor maternal instinct may result in neglect of puppies at the time of birth so that the initial hypothermia is not corrected. Weak or premature pups may be unable to establish the necessary physical contact with the dam to achieve normothermia. If the periods of contact are intermittent, moderate hypothermia may persist (Crighton, 1968).

b. Neonatal Glycogen Reserves

The newborn relies almost exclusively on hepatic glycogen for energy for the first 24 hours. Hepatic glycogen stores may be low at birth owing to intrauterine malnutrition associated with excessive multiple pregnancy or maternal malnutrition. Within 8-12 hours after birth most hepatic glycogen has undergone glycogenolysis and the newborn is forced to rely on nutritional intake to maintain euglycaemia. It is during this interval that the newborn is exquisitely susceptible to the development of hypoglycaemia (Center *et al.,* 1990). Failure to suck results in rapid depletion of the liver reserve of glycogen and the development of hypoglycaemia by the second day.

c. Neonatal Ontogeny

The endothelio-chorial placenta in dogs is advanced in intimacy and allows for *in-utero* transfer to the foetus of from 1 - 20% of total newborn serum antibody concentration (Greene, 1984a). Since IgA is large and is not transported across the placenta most of the immunoglobulin transferred *in utero* is IgG. In the absence of ingestion of colostrum the puppy is probably protected for at least one week due to a small amount of *in utero* transfer of immunoglobulins (Appel and Gillespie, 1972).

There is little information available in regard to the period during which the pup is able to absorb immunoglobulins intact across the intestinal wall. Work by Gillette and Filkins (1966) indicated that absorption appeared optimal if antibody was fed to the puppies eight hours after birth, was almost complete 15 hours after feeding and was not detectable if fed 24 hours after birth.

Secretory IgA present in colostrum and milk is important because of the protection that it provides against mucosal pathogens in the newborn animal. Secretory IgA provides continuous protection against gastrointestinal infection as long as nursing continues (Greene, 1984a). Neonatal puppies (like neonates of other species) are born germ free into a contaminated environment. They lack the indigenous population of microorganisms on skin and mucosal surfaces that is characteristic of the older neonate and adult animals. They acquire their own population from contact with their dam and other animals, from milk, food and other contaminated fomites (Greene, 1984a). Within the first week of life newborn animals are more susceptible to infection than older animals because they lack a well-developed microflora and because they have a non-selective protein transport mechanism that facilitates the absorption of immunoglobulins. This protein transport mechanism may also transport pathogenic microorganisms across the mucosal barrier. Maternal immunity is essential in providing protection during this critical period (Greene, 1984a). The normal microflora is important in protecting body surfaces from colonisation by foreign bacteria through several established mechanisms. They produce antibacterial substances (bacteriocins) and toxic metabolites,

compete for nutrients and sites of adherence and degrade ingested and locally produced toxins (Greene, 1984a). Indirectly their presence enhances the function of existing immunologic mechanisms.

d. Neonatal Renal Function

The kidney of the newborn contains an outer zone of non-differentiated tissue in the cortex which requires 2-3 weeks to undergo nephrogenesis and become functional (Mosier, 1978).

Functional tests of normal puppies have established that the glomerular filtration rate at birth ranges from 21-50% that of the adult and the tubular secretion rate at eight weeks of age ranges from 12-15% that of the adult. This leads to a slow clearance of fluids, increased sodium loss and inability to conserve fluids. There is a positive correlation of glomerular filtration rate and mean arterial blood pressure (Robinson, 1983).

Neonates have a greater extracellular water and total water compartment than adults (Jones, 1987). This difference is due mostly to extracellular fluid. As 82% of bodyweight is water and kidney function is immature, the neonate is particularly susceptible to dehydration (Blunden, 1998). Water turnover rate is twice that of the adult (Mosier, 1978). Neonatal puppy fluid maintenance requirements are approximately 132 - 220 mls/kg/day. What this means to the veterinarian is that nearly all sick pups clinically look the same. When presented, they are usually hypothermic, hypoglycaemic and dehydrated. It is then difficult to do a detailed clinical examination, without first correcting this potentially fatal cycle. In practice, supportive therapy is often given too late to alter survival outcome, and there is often insufficient investigation of neonatal mortalities to assess the cause of breeding loss accurately (Blunden, 1998).

e. Neonatal Diseases

i. Fading Puppy Complex (Syndrome)

The fading puppy syndrome has been a recurring problem for breeders and veterinary surgeons for decades. Various theories have been formulated to account for this syndrome and some have doubted that it is a genuine disease syndrome (Blunden, 1998). One cause of confusion has been to incorporate all conditions leading to poor weight gain and ill-thrift in the first months of life under this one syndrome. A whole range of aetiological possibilities have been considered including maternally related factors such as poor mothering, inadequate lactation, trauma and inadequate nutrition of the dam in pregnancy, congenital anomalies, neonatal isoerythrolysis, low birth weight, thymic atrophy and infectious agents including bacteria, viruses, protozoa and parasites. Abnormal lung surfactant has also been suggested (Blunden, 1998) Fading puppies are defined by Blunden (1986) as those puppies where no obvious cause of death is found after careful post mortem examination, including bacteriology and histopathology and in the context of good knowledge of the clinical history and management practices of the kennel. These puppies are apparently born healthy but fail to thrive and usually die in the first seven days. In Blunden's study of 114 puppies, examination of the growth plate at the costochondral junction revealed that marked growth arrest had occurred in 75% of fading puppies that had died by day three. This suggested that at least a number of puppies were not thriving from a very early stage in neonatal life and it was possible that growth retardation had commenced in late pregnancy. Certain clinical and pathological findings were common in this group. Signs of illness included general lassitude and weak sucking responses from day one or two and restlessness, plaintive and persistent crying, lateral recumbency with limb paddling, inability to stay on the teat and occasionally rigours with progression to signs of generalised weakness and death.

Sturgess (1998) defined fading puppy syndrome as a clinical description rather than a diagnosis and covered a multitude of infectious and non-infectious conditions of the neonate which cause animals born apparently healthy to gradually become inactive, lose their suckle reflex and die in the first two weeks of life. Blunden (1998) identified a tendency for certain dams within particular kennels to have successive fading litters.

ii. **Neonatal Infections**

Infectious diseases accounts for only a relative small percentage of deaths (Sturgess, 1998) and the majority of these deaths occurred in the late neonatal period. This time frame for neonatal infections may be related to the fact that in the absence of the ingestion of colostrum the puppy is probably protected for at least one week by the *in utero* transfer of immunoglobulins.

Many of the attempts to reproduce bacterial diseases in puppies have been unsuccessful which may mean that, although the bacteria are involved in the aetiology of neonatal mortality, some triggering or precipitating factor is also needed (Evans, 1978).

Possible routes of infection are oral, via the umbilicus, by the ingestion of vaginal discharge during the process of parturition or from the infected environment, by inhalation and across the placenta (Evans, 1978). There is reasonable evidence to suggest that at least three types of bacteria are involved in neonatal mortality (Evans, 1978). These are *Haemolytic streptococci, Escherichia coli* and *Brucella* species (Jones, 1987).

Clostridial perfringens Type A was recovered in greater frequency from vaginal swabs of bitches with a history of neonatal losses than from normal breeding bitches (Blunden, 1983). *Cl. perfringens* can appear in the faeces of apparently normal puppies at 24-28 hours after birth. The possibility remains that some factors such as incorrect feeding, hypothermia or the use of antibiotic could affect the natural gut flora while it is becoming established, allowing the organism to become established with an ensuing enterotoxaemia (Blunden, 1983).

iii. Viral Infections

Infectious canine hepatitis (ICH) is caused by canine adenovirus 1 (Greene, 1984b). The young pup infected with CAV-1 may present in acute collapse after a short illness. Clinical signs are usually vague and may include dullness, inactivity, reluctance to nurse and coma. Some puppies proceed to coma and death within just a few hours (Center *et al.,* 1990). Newborn puppies can acquire canine herpes virus infection (CHV) infection from passage through the birth canal or from contact with litter mates, oronasal secretions of the dam, or fomites. *In utero* infection has been documented. CHV in neonatal puppies is associated with an acute fatal illness occurring between 1-3 weeks of age (Greene, 1984b). The narrow range of susceptibility of neonates may relate to *in utero* exposure or may be attributed to exposure to the virus during or soon after birth. Additional predisposing factors include the lack of thermoregulatory mechanisms during the first week of life and an incubation period of four to six days. Myocardial disease associated with canine parvovirus infection develops following *in utero* or early neonatal infection of puppies (less than eight weeks) from non-immune bitches. Virus can persist in cardiac fibres in a latent form until myocytes multiply into multi nucleated cell types between three and eight weeks of age.

iv. Primary Immunodeficiency Diseases

Primary immunodeficiency is a congenital failure of the immune system that has a proven or suspect genetic basis and that predisposes an animal to disease (Greene, 1984a). Primary immunodeficiency diseases should be considered in the neonatal pup when non-colostrum deprived animals have recurrent, prolonged infection that responds poorly to treatment and neonatal deaths in litters of common parentage (Guilford, 1987).

v. Parasitic infection

Andersen (1957) found that parasites caused a few mortalities. These occurred at 4-5 weeks of age. It is considered possible that puppy deaths after the fourth day can be due to worm infestations and that the migrating ascarid *Toxocara canis* in particular is involved (Evans, 1978). As many as 90% of puppies are affected prenatally with *T. canis*. Infestation may also occur via the colostrum (Evans, 1978). In contrast to the situation for *T. canis*, intrauterine infection with the canine hookworm, *Ancylostoma caninum*, is of minor significance. Suckling pups acquire *A. caninum* larvae from colostrum and milk as well transcutaneously and by ingestion of other contaminated material (Evans, 1978). Postnatal infections with *Toxoplasma Gondii* may occur as acute or chronic clinical entities (Greene and Prestwood, 1984). Acute postnatal infection is rapidly progressive and disseminated toxoplasmosis may occur when large numbers of oocysts or bradyzoites are ingested.

vi. Congenital Defects

Congenital defects are defined as abnormalities of a structure or function present at birth. Many, but not all, congenital defects are genetically caused (Leipold, 1978). Congenital disorders may be lethal to the neonate at birth or shortly after, or result in euthanasia of the animal. Many of the defects present at birth are not identifiable without clinical or pathological identification (such as ocular) and do not contribute to neonatal mortality. Many structural abnormalities develop as clinical entities later in life, e.g. portosystemic shunts or only become evident when the animal becomes ambulatory (Leipold, 1978).

Reported pup losses associated with grossly recognised congenital defects varies. Potkay and Bacher (1977) recorded a 1.0% loss, Blunden (1986) 1.4% and Andersen (1957) 2.2%. Functional defects may lead to neonatal death and are being diagnosed with increasing frequency. Inherited coagulation disorders and metabolic defects are two such entities.
Some inherited coagulation disorders reported to cause neonatal death are Factor II, Factor VIII and Factor X deficiencies (Fogh and Fogh, 1988).

Metabolic diseases of the dog and cat were investigated by Jezyk (1983). A variety of disorders were identified in a relatively small population and concluded that if it is even a rough estimate of the prevalence of such disorders in the general population then these diseases may represent a significant cause of death. That such a disorder might be present must therefore be suspected by the clinician. Often the basis for this suspicion is epidemiologic in nature, such as repeated losses from successive litters by the same bitch or unusual patterns of puppy mortality in related bitches (Lawler, 1989).

vii. Low Birth Weight (Runting)

Pup mortality attributed to runting varied from 1.4% (Andersen, 1957) to 2.1% (Blunden, 1986). There were no criteria, e.g. specific birth weight, by which runts or "small for date" pups were recognised.
Puppies which are more than 25% below the average birth weight are reported to have a higher mortality rate (Mosier, 1978). Weight is a reflection of the relative maturity of the organism. Low birth weight puppies are physiologically immature when compared to litter mates of average birth weights. They are also at greater risk from hypothermia and cannot compete well for milk against their larger litter mates. In human medicine the term Intrauterine Growth Retardation (IUGR) is used and this describes the clinical entity, that is, low birth weight for gestational age. The magnitude of the problem of IUGR is second only to prematurity as a cause of perinatal mortality in the infant. While prematurity causes primarily an increased neonatal mortality, IUGR causes a vastly increased intrauterine foetal death rate. Death from intrapartum asphyxia alone is 10 times higher than for appropriately grown infants with 14% of all stillbirths and 6% of all neonatal deaths occurring in infants whose birth weights are less than the third percentile for gestational age (Renfield, 1975). In the vast majority of small-for-date infants there is no identifiable cause to explain their small size. Two major factors influence foetal growth. These are the inherent growth potential of the foetus and the growth support it receives by way of the `supply line' - the placenta and maternal organism (Renfield, 1975). Acute perinatal distress, while not affecting foetal growth per se, is characteristic of IUGR. Hypoxic insults to the deprived infant may occur during any stage of gestation. The limited nutritional and circulatory reserves of such a foetus make even normal labour and delivery, in itself a hypoxic stimulus, a more stressful event for the infant. The higher rate of antepartum and intrapartum stillbirths and low one minute Apgar score in this group attests to this concept (Renfield, 1975). The extent of growth retardation and its contribution to perinatal loss in the dog requires further investigation. For this, normal birth weight and the standard deviation from the mean must be identified for each breed of dog and mortality relative to the birth weight variation investigated.

MATERNAL RISK FACTORS

The outcome of pregnancy depends, largely, on the overall health of the dam. Nutrition and management of the dam during pregnancy, whelping and the neonatal period greatly influence the outcome of the breeding program. Many factors have predictable effects on pregnancy and neonatal survival. Selection of animals with metabolic abnormalities or the genetic predisposition to develop them should be avoided (Johnson *et al*., 1987). This broad statement is inadequately documented in the veterinary literature. Similarly, maternal cardiac and renal disease and diabetes mellitus are all reported to affect the health of the foetus but again is poorly documented.

a. Maternal Age

Age has a profound influence on the reproductive performance. Andersen (1965) studied a colony of 65 Beagle bitches. The number of pups weaned was greatest (4.28 pups) and puppy mortality was lowest (18.5%) when dams were two years old. In another group of 17 Beagles the number of pups weaned was highest (4.19) and neonatal mortality was lowest (15%) when dams were 3.5 years old. By five years of age conception failure was more than 50%. When these same bitches were seven years old, an average of 1.22 pups were weaned and puppy mortality was

38.9%. By nine years of age, only 0.6 pups were weaned per litter and mortality was 76.3%.

b. Maternal Infections

Maternal infections are well known causes of perinatal morbidity and mortality (Johnson et al., 1987). The dam may show no clinical signs and yet the infectious agent may have profound effects on the foetus or neonate. The type of disease that results from *in utero* infections depends more on the stage of gestation at which infection occurs than on the aetiological agent (Greene, 1984a).

Maternal infections with Canine Parvovirus, Herpes Virus, Distemper, *Brucella Canis* and toxoplasmosis are all reported to cause foetal and neonatal mortality (Percy *et al*., 1971; Carmichael, 1976; Krakowka *et al.,* 1977; Robinson *et al*., 1979; Higgins *et al*., 1981; Lenghaus and Studdert, 1982; Hashimoto *et al*., 1983; Greene and Kakuk, 1984; Greene, 1984c).

Mycoplasmas are common inhabitants of the canine vagina and prepuce but may cause reproductive disease if inoculated into the uterus (metritis) or vas deferens (orchitis). Their role in spontaneous embryonic death, resorption, abortion and stillbirth is unknown (Johnston and Raksil, 1987).

Campylobacter jejuni infection has been reported in association with premature labour and abortion. *C. jejuni* was cultured from the foetal liver and stomach (Bulgin *et al.,* 1984).

Escherichia coli is a common aerobic bacteria present in the vagina of the normal bitch. It is commonly cultured from the uterus of bitches with pyometra and from vaginal discharges following foetal death and abortion (Greene and Kakuk, 1984).

Salmonella bacteria have been isolated as a cause of abortion and foetal death in a Boxer. Their role in foetal death like that of *E. coli*, may be that of a primary pathogen or as a secondary ascending invader (Redwood and Bell, 1983).

Beta-haemolytic streptococci are common inhabitants of the vagina of the normal bitch. These bacteria have been isolated from the uterus of some bitches with pyometron and from some puppies with neonatal septicaemia. Mantovani *et al*. (1961) reported the presence of *beta-haemolytic streptococci* (type 1) from the vagina and lymph node aspirate of five collie bitches with a history of abortion, infertility and neonatal death.

c. Maternal Behaviour

The maternal behaviour post whelping can significantly affect pup mortality. In Andersen's study (1957), maternal factors and mismothering, which included trauma, excessive licking, lactational failure and cannibalism, caused the death of 6.8% of all pups born and was the principal cause of pup mortality. Maternal/management causes (trauma and lactation problems) accounted for the loss of 4.8% of pups in Blunden's study (1986).

The lack of effective maternal care allows environmental influences, particularly the environmental temperature, to affect the puppies (Crighton, 1968). Certain bitches refuse to relax and adopt lateral recumbency. They may sit or move away from the puppy. This creates a situation where an otherwise healthy puppy has an unstable rectal temperature (Crighton, 1968). Excessive maternal activity is displayed by other bitches. They spend too much time cleaning a litter and certain pups may have great difficulty in keeping in contact with the dam for lengthy periods. The widely recognised phenomenon where a bitch selects one or two puppies and then rejects them will result in hypothermia even if the rejects are healthy. Poor mammary gland development at the time of whelping may be one further factor in postpartum hypothermia in a litter (Crighton, 1968).

d. Dystocia

Dystocia or prolonged labour and the associated hypoxia or anoxia are very significant causes of early death in the dog (Lawler, 1989). Dystocia is defined as a difficult birth or inability to expel the foetus(es) from the birth canal at the time of parturition (Macintire, 1994).

There are, however, limited reports in the literature on the extent of the contribution of dystocia to pup mortality. Andersen (1957) reported the loss of 2.3% of all pups born due to dystocia.

Linde-Forsberg and Eneroth (1998) consider the true incidence of dystocia in the bitch to be around 5% overall, but it may amount to almost 100% in some breeds of dog, especially of the achondroplastic type and those selected for large heads. Traditionally,

dystocia is classified as being either of maternal or foetal origin, or a combination of both. Maternal causes are reported to account for 75.3% of dystocias occurring in the dog, with 48.9% due to primary complete inertia (maternal primary inertia) and 23.1% due to primary partial inertia (maternal secondary inertia). Foetal causes accounted for 24.7% of dystocias encountered, with malpresentations (15.4%) and foetal oversize (6.6%) the predominant forms occurring (Linde-Forsberg and Eneroth, 1998).

The diagnosis of dystocia and acceptable time constraints have been reported by Johnston (1988), Jones and Joshua (1982) and Macintire (1994). The diagnosis of dystocia is dependent on demonstrating failure to start labour on due date (use knowledge of ovulation date, rectal temperature drop), failure to progress normally in labour (more than four to five hours in stage two labour before delivery of the first pup, or more than two to three hours between puppies), foetal membranes in vulva for greater than 15 minutes, strong contractions lasting more than 30 minutes with no foetus. Jones and Joshua (1982) state that the first pup is usually born within one hour of the onset of meaningful straining, but often appears within as little as 20 minutes; up to 2 hours need not cause anxiety. By six hours placental separation is occurring and the life of the presenting puppy may be in jeopardy, thus six hours should be regarded as the maximal period permissible without investigation; even after two hours some separation may exist. The interval between birth comprises two parts - resting and straining. Rest periods vary from 5 minutes to 3 hours and occasionally even longer, although it is arguable if 4 hours rest falls within normal limits. Provided vigorous straining without progress does not occur, the longer intervals do not call for urgent attention. A common pattern is for a bitch to produce two or three puppies at short intervals e.g. ten to 30 minutes and then go into a rest phase of one to three hours before repeating the process. The birth of a large litter may occupy 24 hours or so but if there is no excessive effort needed for each birth the dam should not become unduly tired. From a veterinary standpoint one of the most difficult clinical decisions is the distinction between the end of normal labour and the beginning of abnormal labour. The above time constraints may be excessive and require review. Further, the specific contribution of dystocia to pup mortality and the post mortem and histopathological findings in these pups requires detailed investigation.

ENVIRONMENTAL RISK FACTORS

A low ambient temperature not only tends to produce hypothermia in a puppy but it may also cause a dam to adopt a posture of ventral recumbency so that radiant heat loss from the engorged mammary gland is minimised. Thus a puppy, already hypothermic, may be denied access to a source of heat (Crighton, 1968).
In a neonatal mortality study by Andersen (1957), monthly climatic variations did not coincide with fluctuations in puppy mortality. However, puppy deaths did coincide with sudden diurnal changes in climatic conditions. The highest losses were observed in the autumn which was characterised by rapid changes in temperature where a warm day may be followed by a cold night. Fatalities in puppies between eight and 21 days of age occurred in summer when the temperature rose to 108 degrees F. Evidently they attempted to seek a cooler area and were unable to return to their litter nest after direct exposure to the sun.

RISK FACTORS / RISK SCORING

Human obstetrical and parturient care revolves around "risk scoring" and foetal monitoring, with the ultimate aim of maintaining maternal health and delivery of healthy neonate. The scoring techniques and foetal evaluation used in human obstetrical care (Depp, 1986) are not adaptable to the canine. However, unless some accurate form of risk assessment during pregnancy and whelping can be developed it may not be possible to reduce the current high levels of pup mortality reported.

The influence of parturition on pup mortality has been inadequately documented. An increased risk of pup death with increasing maternal age has been documented by Andersen (1957). Pup mortality is also reported to vary between breeds (Lawler 1989). Other maternal risk factors such as parity and litter size are not documented. In contrast, the pig, which is also polyviviparous, has been extensively studied and is the only other domestic species that is of any comparative value (the data on the cat are as sparse as that published on the dog). Also, the mortality problem in the pig is apparently similar to that recorded in the dog. Glastonbury (1976) reported that 74% of all preweaning mortalities occurred before the fourth day of life and Sprecher *et al*. (1974) considered that a high

percentage of these deaths were associated with anoxia and hypoxia.

The risk factors identified in the pig that are associated with parturition are litter size, position in the birth order, parturition time and umbilical cord detachment (Dzuik and Harmon, 1969: Wrathall, 1971: Svendsen and Bille, 1981).

Risk factors such as bitch breed, age, parity, whelping time and litter size must be identified. The influence, if any, of pup birth weight, sex, presentation, placental attachment and litter position must be investigated. Such an investigation would necessitate a veterinarian to be present at the whelpings. Because of the large number of litters and different breeds required this becomes a practical and economical impossibility. Alternatively, a system that requires breeders to observe and record whelping data and clinically assess and monitor the pup and bitch is the best alternative available. The accuracy of the breeder's observations may be subject to error. With education and clear guidelines this error can be minimised and a large amount of relevant clinical information then becomes available for the investigation of pup mortality.

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