STUDIES ON UTERINE CONTRACTION PATTERN AND CERTAIN BIOCHEMICAL PARAMETERS IN PRIMARY UTERINE INERTIA IN FEMALE DOGS

PRASHANTKUMAR

DEPARTMENT OF VETERINARY GYNAECOLOGY AND OBSTETRICS

VETERINARY COLLEGE, HEBBAL, BANGALORE

KARNATAKA VETERINARY, ANIMAL AND FISHERIES SCIENCES UNIVERSITY, BIDAR- 585 401

JUNE, 2013

STUDIES ON UTERINE CONTRACTION PATTERN AND CERTAIN BIOCHEMICAL PARAMETERS IN PRIMARY UTERINE INERTIA IN FEMALE DOGS

Thesis submitted to the

KARNATAKA VETERINARY, ANIMAL AND FISHERIES SCIENCES UNIVERSITY, BIDAR

in partial fulfillment of the requirements

for the award of the degree of

MASTER OF VETERINARY SCIENCE

IN

VETERINARY GYNAECOLOGY AND OBSTETRICS

By

PRASHANTKUMAR

DEPARTMENT OF VETERINARY GYNAECOLOGY AND OBSTETRICS

VETERINARY COLLEGE, HEBBAL, BANGALORE

KARNATAKA VETERINARY, ANIMAL AND FISHERIES SCIENCES UNIVERSITY, BIDAR- 585 401

JUNE, 2013

KARNATAKA VETERINARY, ANIMAL AND FISHERIES SCIENCES UNIVERSITY, BIDAR

DEPARTMENT OF VETERINARY GYNAECOLOGY AND OBSTETRICS

VETERINARY COLLEGE, HEBBAL, BANGALORE

CERTIFICATE

This is to certify that the thesis entitled "***STUDIES ON UTERINE CONTRACTION PATTERN AND CERTAIN BIOCHEMICAL PARAMETERS IN PRIMARY UTERINE INERTIA IN FEMALE DOGS"*** submitted by **Mr. PRASHANTKUMAR, ID No. MVHK-1134** in partial fulfilment of the requirements for the award of degree of **MASTER OF VETERINARY SCIENCE** in **VETERINARY GYNAECOLOGY AND OBSTETRICS** of the Karnataka Veterinary, Animal and Fishers Sciences University, Bidar is a record of bonafide research work carried out by him during the period of his study in this University under my guidance and supervision. And the thesis has not previously formed the basis for the award of any degree, diploma, associateship, fellowship or other similar titles.

Bangalore
June, 2013

Dr. A. KRISHNASWAMY
Major Advisor

Approved by :

Chairman : _____
(Dr. A. KRISHNASWAMY)

Members : 1. _____
(Dr. T. G. HONNAPPA)

2. _____
(Dr. V. C. MURTHY)

3. _____
(Dr. M. NARAYANA BHAT)

4. _____
(Dr. L. RANGANATH)

This thesis is Dedicated to

my Parents, Brother, Sister, Grandmother

and family for their love, endless support

and encouragement.

ACKNOWLEDGEMENT

The realization of this work was only possible due to the several people's collaboration, to which I desire to express my gratefulness.

Foremost, I would like to express my sincere gratitude to my advisor ***Dr. A. Krishnaswamy****, Professor and Head, Department of Veterinary Gynaecology and Obstetrics, for the continuous support of my M.V.Sc study and research, for his patience, motivation, enthusiasm, and immense knowledge. His guidance helped me in all the time of research and writing of this thesis. I could not have imagined having a better advisor and mentor for my M.V.Sc study.*

Besides my advisor, I would like to thank the rest of my thesis committee: ***Dr. T. G. Honnappa****, Associate Professor, Department of Gynaecology and Obstetrics,* ***Dr. V. Chandrashekara Murthy****, Associate Professor, Department of Gynaecology and Obstetrics,* ***Dr. M. Narayana Bhat****, Director, Institute of Wildlife Veterinary Research and* ***Dr. L. Ranganath****, Department of Veterinary Surgery and Radiology for their encouragement, insightful comments and hard questions.*

My special thanks to ***Dr. G. Sudha****, Assistant Professor,* ***Dr. Ravindranatha B. M.,*** *Assistant Professor and* ***Dr. Navya M.****, Assistant Professor Department of Gynaecology and Obstetrics for their constant support.*

My special and unforgetable thanks go to the senior colleagues ***Drs. Shwetha, Vibha, Navya, Kavya , Ashwartha, Hakim*** *and* ***Dost,*** *my fellow colleagues* ***Ashok, Harsha, Sowmya, Yathish,*** *and* ***Chithra,*** *Ph.D scholars* ***Drs. Shwetha, Mahesh Dodamani, Jayakumar, Kantharaj, and Lakshmikanth*** *and my junior colleagues* ***Sajjan, Abhignya, Yugandhar,***

Mallu, Premkumar, Prashanth C and Suja *who were always there with their cordial help and cooperation.*

I would like to extend my wishes to ***Vijayalakshmi, Siddram, Rajanna, Satish, Manjunath*** *and* ***Family*** *and other non- teaching staff who have helped me in completion of my thesis; either directly or indirectly.*

My special thanks to ***Dr. Shantkumar, Dr. Shwetharani, Dr. Shwetha K S, Dr. Narasimha Murthy, Dr. Amaregouda Patil, Dr. Anand Patil, Dr. Dattatreya, Dr. Tippanna, Dr. Vishwanath*** *and* ***Dr. Somashekhar H.***

A big hug to ***Team SNAPS, Team BCDC, Guru, Ashok Walikar*** *and* ***2011 Bidar Vets.***

Words can hardly express my feeling & a warmest gratitude goes to my friends for inspiring me with positive attitude, encouragement, knowledge, energy, generous help in carrying out my research work and for making my stay comfortable during the entire period of my study. I deeply express my sincere thanks to my all time friends, ***Shivaleela, Shiva, Avinash, Nagalambika, Gurunath, Dattu, Pradeep Mulawad, Basavaraj Hiremath, Prashant Bel, Ashok, Holagi, Sharanagouda, Nikhil, Mahendra, Tangolli, Shridhar, Sachin, Vinay, Sarvo, Gopa, Shashi, Sumanth, Swamy, Raju, Javed, Bandu, and Jagu.***

It is like a drop in the ocean of words that can never reach its mark to acknowledge infinite love, blessings, sacrifices and constant encouragement of my beloved ***Parents*** *and* ***family.***

Bangalore, **PRASHANTKUMAR**

June, 2013

CONTENTS

LIST OF TABLES

LIST OF PLATES

Introduction

I. INTRODUCTION

The ultimate goal in the breeding programme in the bitch is obtaining healthy offsprings while maintaining the health of the mother. As veterinary care has advanced, so too, the demand for specialised veterinary services in dogs, including management of pregnancy and whelping. Consequently, there are considerable efforts being made to develop methods to optimize the outcome for each puppy and bitch.

For the neonate, labour represents the most critical phase contributing to the first minutes after birth and the act of parturition is the most anxious time for the dog breeder, as the puppy survival rate and the future reproductive status of the dam are influenced by the events at this stage. In a study by Darvelid and Forsberg (1994), the frequency of puppy deaths up to 3 weeks of age following normal parturition was found to be 11.9 per cent. In comparison, the frequency of pups born dead or dying during abnormal parturition was 22.3 per cent.

Dystocia defined as difficult birth or inability to expel the foetus/ foetuses through the birth canal without assistance constitutes a serious problem in the bitch. Although, in a majority of the animals, parturition is usually a natural process, but there are occasions when normal birth is difficult or impossible and dystocia occurs as a result of unusual prolongation of the first or especially the second stage of labour. While dystocia occurs in less than 5 per cent of canine pregnancies (Jackson, 2004), it's a true obstetrical emergency and of great economic loss to the breeder (Davol, 2000).

Dystocia has conveniently been described as being maternal or foetal in origin and there is overwhelming evidence that in the bitch, maternal dystocia is encountered more frequently (Gaudet, 1985; Darvelid and Linde-Forsberg, 1994). There is also evidence that the most common cause of maternal dystocia is uterine inertia, representing 40 per cent (Gaudet, 1985) to 75.3 per cent (Darvelid and Linde-Forsberg, 1994) of all dystocia attributed to the dam. The most common form of uterine inertia in bitches is primary uterine inertia, which has been further classified as complete or partial (Van Den Weijden and Taverne, 1994). In complete primary uterine inertia, the bitch does not start labour. In partial primary uterine inertia, the bitch starts to deliver her puppies, but the labour ends prematurely, despite the presence of a patent birth canal (Bergstrom *et al.*, 2006[a]).

In view of the importance of primary uterine inertia as a highly significant cause of maternal dystocia, there is a need to identify the factors influencing its incidence. Similarly, there is very little or no information on the influence of such factors as the breed, size of the breed, age, parity and litter size on the incidence of primary uterine inertia in dogs, particularly from the Indian subcontinent.

The cause of primary uterine inertia is unknown, although a disturbance in the sequence of hormonal events required for normal labour may represent one possible cause (Bergstrom *et al.,* 2010). Labour is the physiological process by which a foetus is expelled from the uterus through the vagina and is characterized as regular uterine contractions accompanied by cervical effacement and dilatation (Maul *et al*., 2003) and it is commonly believed that uterine inertia is associated with complete absence, weak or

asynchronous uterine contractions. The greatest impediment to understanding normal labour, besides recognizing its onset (Pates *et al*., 2007), is related to the evaluation of the quality of the uterine contractions, in term of strength, duration, and frequency, since they are not appreciable externally. To quantitatively evaluate the uterine activity, a tocodynamometric method has been developed based on recording of uterine contractions across the abdominal surface (Maul *et al*., 2003). Tocodynamometry is a new approach in canine obstetrics to diagnose delivery problems. Foetal disturbances and hypoxic conditions can be suspected earlier with the possibility of immediate intervention, as in human obstetrics.

The suggested causes for primary uterine inertia include a deficiency of Oxytocin (Bergstrom *et al.,* 2010), Serum Calcium (Gaudet, 1985 and Jones and Joshua, 1988) and Blood Glucose (Jones and Joshua, 1988 and Linde-Forsberg and Eneroth, 2000). Therefore, many of the medical protocols used for treatment of primary uterine inertia have centered on intravenous infusion of oxytocin, glucose and calcium, either alone or in combination (Arthur *et al.,* 1989; Wallace and Davidson, 1995 and Bergstorm *et al*., 2006[a]). The efficacy of many medical protocols commonly used by the veterinarians to relieve dystocia due to uterine inertia has not been subjected to a thorough scrutiny. Therefore, the present study was designed to document the factors influencing the incidence of uterine inertia in dogs with particular reference to the evaluation of uterine activity. In addition, studies were also conducted to evaluate the efficacy of certain treatment protocols for augmenting uterine contractions in confirmed cases of primary uterine inertia. The specific objectives of the study were,

1. To record the prevalence of primary uterine inertia as a cause of dystocia in female dogs.
2. To estimate the concentration of blood glucose, calcium and magnesium in cases of primary uterine inertia in female dogs.
3. To determine the uterine contraction pattern in cases of primary uterine inertia.
4. To determine the effect of intravenous supplementation of oxytocin, calcium and glucose on the pattern of uterine contraction in cases primary uterine inertia female dogs.

Review of Literature

II. REVIEW OF LITERATURE

2.1 Normal course of parturition

It is essential that a Veterinarian shall be perfectly familiar with the normal course of parturition in the bitch he is called upon to attend, in order that he shall recognize at once when the process has ceased to be physiological and has become pathological. It is customary in any species to define the classical three stages of parturition and this system can be adhered in the bitch with regard, at least to the first two stages. From this point, the third stage is brief and nearly continuous with the second stage, the second and third stages are then reported. The various stages of parturition has been well described by Freak (1962), Freak (1975), Buckner (1979), Johnston (1986), Mosier (1986), Arthur (1986), Burke (1986), Arthur *et al.* (1989), Wallace and Davidson (1995), Johnston *et al.* (2001).

2.1.1 First stage of parturition

The first stage of parturition is described as the stage of relaxation and dilatation of the cervix and soft tissue of the vagina. It is also the stage of intermittent uterine contractions without the visible effort on the part of the bitch (Burke, 1986; Roberts, 1986 and Johnston *et al.*, 2001). During this stage, the dam's general activity may become intense, the bitch may glance occasionally at her flank area and may appear to be uncomfortable (Burke, 1986). Johnston (1986) described anorexia, restlessness, apprehension, panting, shivering, occasional vomiting and seclusion as the typical clinical signs of first stage of parturition. Concannon *et al.* (1989) stated that obsessive nesting behaviour may be observed shortly before the birth of individual pups.

Freak (1975) reported that the duration of first stage in bitch is most commonly within the range of 6 to 12 hours, but may be prolonged up to 36 hours in a nervous primigravida to an unnoticeable period in an easy whelping multigravida bitch. Burke (1986) also stated that the first stage can last from 1 to 36 hours and it averaged 6 to 12 hours. First stage in bitch occupies about 12 hours. Roberts (1986) reported that the first stage usually lasts for 2 to 12 hours.

Mosier (1986) stated that active contractions of both longitudinal and circular muscle fibres of the uterus are most intense just anterior to the most caudal foetus. Dilation of the cervix begins at the internal os, the pelvic ligaments relax and mucoid secretions pass through the cervix during the 12 hours preceding the end of first stage. Uterine contractions then occur at progressively closer intervals. Near the end of first stage, the most caudal foetus rotates on its long axis and extends its head, neck, and limbs. Four hours before the end of first stage, the dams cervix dilates to approximately 8 mm in diameter (Smith and McDonald, 1974). The allanto-chorionic membrane appears in the vagina near the end of first stage and its rupture results in discharge of fluids. Shortly after the rupture of allanto-chorionic membrane, the amniotic sac passes through the vagina and protruded as water bag between the lips of the vulva signalling the end of first stage.

2.1.2 Second stage of parturition

The second stage of parturition is the stage of propulsion and expulsion of pup or of straining and delivery (Freak, 1975).

Johnston (1986) stated that, abdominal contractions, and or presence of foetal membranes at the vulva are the signs of the onset of second stage of labour. A dam will usually be on her side during this stage, although, it is not uncommon for her to stand intermittently, strain and occasionally deliver a foetus while standing (Mosier, 1986).

The intermittent uterine contractions of the first stage start to propel the foetus towards the cervix. As the foetus is forced into the cervix and mechanically dilates it, neurohormonal reflex (Ferguson's reflex) results in further increase in circulating oxytocin and intensifies uterine contractions (Ferguson, 1941). As the foetus approaches the pelvis, the outer layer of the foetal membrane allantochorion, reaches the vulva and ruptures or is torn by the bitch (Roberts, 1971; Freak, 1975 and Roberts, 1986). The rather tougher layer of the amnion passes into the pelvis. At this moment, the foetus is engaging the pelvic inlet in an already rotated position and extended posture (Freak, 1975). Freak (1962) also stated that the foetus which dies before reaching the pelvic inlet almost invariably remains unrotated. As the foetal head in a normal anterior presentation engages fully in the pelvis, its pressure stimulates a simple neural reflex which brings forceful abdominal straining into play to assist propulsion of head and shoulders through the pelvis. In primigravid, the lips of the vulva may offer slight resistance and may require greatest expulsive effort (Roberts, 1971; Freak, 1975 and Mosier, 1986). Usually once the head is through the vulva, the foetus slides out easily, the bitch rips open the amnion with her incisors, cleans the nose of the pup and by licking pushes the membrane posteriorly over its abdomen until the pup lies free, breathing normally but still attached by the cord. At this point the umbilical cord is usually severed by the bitch biting it through her teeth.

The first pup is usually born within four hours after the onset of second stage of labour (Johnston, 1986 and Wallace and Davidson, 1995). On the other hand, Mosier (1986) reported that, the first foetus is usually delivered within 20 to 30 minutes after the onset of second stage of labour.

Freak (1962) noticed that the time period between the deliveries in bitch is extremely variable. In the normal easy whelping breeds, 10 to 30 minutes is considered as the usual period. In a very large litter, a group may be born at this rate, followed by a rest of even up to 4 hours, before a final one, two or three followed. The author opines after a regular straining has begun, the first foetus will not be live for more than 6 hours with relatively shorter period of about 2 hours allowed on each following.

Roberts (1971) also stated that, straining may begin ½ to 1 hour or more and before the second pup is expelled. The average total time for the second stage of the parturition is 3 to 6 hours and 12 hours is considered to be maximum (Roberts, 1986).

Buckner (1979) recommends the examination of the bitch, if she has whelped 1 or more puppies and then fail to deliver others present in the uterus during the next hour and that a bitch should not be allowed to labour vigorously at the onset of parturition for more than 2 hours without delivering a puppy.

Bennett (1980) considers the following criteria for diagnosing dystocia in a bitch (a) Strong and persistent expulsive effort failing to deliver a puppy within 20 to 30 minutes. (b) Weak infrequent expulsive effort to produce a pup within 2 hours and (c) more than 4 hours having elapsed since the birth of the puppy with no evidence of labour.

Roberts (1986) stated that, it may take about 1 hour of the labour for the first pup to be born. Following the delivery of the first pup the dam may rest for a variable period of time, the rate of expulsion of subsequent pups varies irregularly but progressively shorter.

Johnston (1986) also stated that subsequent puppies are generally delivered at interval of less than 2 hours each and that many bitches will deliver 2 puppies within minutes of each other and then rests, while some bitches will deliver entire litter in 2 to 4 hours, others may take 6 to 12 hours. The author concludes that, failure to progress is generally defined temporarily as occurring when the bitch had been in the second stage of labour for more than 2 hours prior to birth of her first pup or for more than 2 hours between pups.

Arthur *et al.* (1989) reported that the total time occupied by the second stage will depend chiefly on the number of foetuses, but as a general rule when the litter is within the usual limits of 4 to 8 it occupies about 6 hours and 12 hours is the maximum time it may occupy when the number of foetuses are very high and that it is improbable that the puppies born after this time even without assistance will be alive.

2.1.3 Third stage of parturition

The third stage of parturition consists of delivery of the foetal membrane and the partial involution of the uterus after delivery of each puppy (Burke, 1986). The foetal membranes maybe expelled along with the new born or retained for a variable period. And sometimes 2 foetal membranes maybe expelled after delivery of two puppies. And in

any event, the membrane should be eliminated within 45 minutes after the birth of the new born.

2.2 Incidence of dystocia

Details concerning the frequency of dystocia in the dogs are few which are because of the wide variations between breeds and the tendency for breeders to intervene, in some cases prematurely and unnecessarily. In addition, there are some breeds which are achondroplastic and brachycephalic, where normal birth rarely if ever occurs, and elective caesarean operations are the routine (Arthur, 2001). A retrospective study by Walett-Darvelid and Linde-Forsberg (1994) of 182 cases of dystocia found that 42 per cent of bitches that had whelped before had previously suffered from dystocia. The overall incidence of dystocia in the bitch is stated to be less than 5 per cent (Root *et al*., 1995; Linde- Forsberg and Eneroth, 2000; Gill, 2002; and Sparkes *et al*., 2006). However, the incidence of dystocia was almost 100 per cent in some breeds of dogs, especially those of achondroplastic type and those selected for large heads. Gill (2002) found that the frequency of dystocia varied from 9.1 per cent in Golden Retrievers to 85.7 per cent in Pekingese. Recently, Bergstorm *et al*. (2006[b]) using data from insurance claimed records of almost 2 lakhs insured bitches during the period between 1995 and 2002, estimated the overall incidence of dystocia to be around 16 per cent.

Bergstorm (2009) found the overall incidence of dystocia in bitch to be 5.7 per cent.

2.3 Causes of dystocia

Traditionally, according to its cause, dystocia is considered to be foetal or maternal in origin (Bennet, 1974; Smith, 1974; Arthur, 1975; Buckner, 1979; Johnston, 1986; Arthur *et al*., 1989; Darvelid and Forsberg, 1994 and Wallace and Davidson, 1995).

Smith (1974) stated that, foetal dystocia are more common than maternal and is caused by abnormalities in the size, conformation or presentation of one or more puppies at the same time.

Gaudet (1985), in a retrospective study of 128 cases of dystocia in bitches reported that 57(60 %) of the cases were deemed to be due to primarily to a maternal anatomical or physiological abnormalities, while 40 per cent had foetal causes.

In a study of 182 bitches of different breeds that were brought to a Veterinary Hospital of dystocia, Darvelid and Linde-Forseberg (1994) found that 75.3 per cent of the cases of dystocia had a maternal cause while 24.7 per cent are of foetal origin.

Bennur (1999) reported that, maternal causes of dystocia were encountered 6 times more frequently than foetal causes of dystocia. In his study involving 84 cases of dystocia, 72 cases (86 %) were considered to be of maternal origin and 12 cases (14 %) to be of foetal origin.

Polster (2006) in their analysis 698 cases treated for obstetrical disorders found maternal dystocia (57.6 %) outnumbers the foetal dystocia (42.4 %).

In a study involving 240 cases of dystocia in bitch, 63.34 per cent were determined to be of maternal origin and the rest (36.66 %) as foetal in origin (Narasimha Murthy, 2011).

In a study involving 442 cases of dystocia in bitch, 61.09 per cent were determined to be of maternal origin and the rest (38.91 %) as foetal in origin (Vibha, 2012).

2.4 Factors influencing maternal causes of dystocia

2.4.1 Breed

Breed of the dam has been reported by several authors to have significant influence in the course of parturition in the bitch (Freak, 1948; Freak, 1962; Heath, 1962; Bennett, 1974; Donovan, 1980; Shille, 1983; Jones and Joshua, 1988; Polster, 2006; Catharina and Gunilla, 2007 and Chatdarong *et al.*, 2007). The boxers suffer a high frequency of dystocia mainly due to uterine inertia but also due to foetal malpresentation (Catharina and Gunilla, 2007).

The Welsh Corgi shows extreme variation in the size of the pup and hence absolute foetal oversize as also in the majority of miniature breeds (Wright, 1939 and Freak, 1948). Primary uterine inertia is also reported to be more common in many Terriers and Cocker spaniels (Freak, 1948). Freak (1962) reported that, out of 222 cases of dystocia, 128 occurred in Scottish terrier and 6 occurred in miniature and Toy Poodles. Of the other breeds involved, Mongrels had the next highest incidence of 16 cases. Smith

(1965) stated that in bulldogs, a cord like structure crossing the lumen of the vagina dorsoventrally just caudal to the cervix was observed as a cause of dystocia.

Smith (1974) stated that, a relatively small pelvis is the contributing factor for dystocia in Boston terriers. Christiansen (1984) claimed that, dystocia mainly occurred in the miniature breeds because their pups are bigger in relation to the bitch compared with the case in medium and large breeds.

A retrospective study by Gaudet (1985) involving 128 cases of canine dystocia revealed Chihuahua, Dachshunds, Pekingese, Yorkshire terrier, Miniature Poodles and Pomeranian to have significantly increased risk of dystocia. However, the study did not support the reported high incidence of dystocia for brachycephalic breeds.

Robert (1986) stated that, the highest incidence of dystocia are encountered in Boston Terrier, Scotch Terrier, Pekingese and other small brachiocephalic breeds and lowest in the more natural breeds such as Hounds and Mongrels. He considered the disproportion between foetal size and maternal pelvic diameter as the primary reason for higher incidence of dystocia in toy and achondroplastic breeds.

Arthur *et al*. (1989) noted that Dachshunds and Aberdeen terrier to be particularly prone to primary uterine inertia. The Corgi showed extreme variation in the size of its puppies and hence absolute and relative oversize leading to dystocia. Brachiocephalic breeds together with Scottish terrier were said to be more prone to obstructive dystocia due to foetuses having comparatively large head and the dam having narrow pelvis.

Greyhound breed showed a high proportion of dystocia case due to arrested foetal development and foetal death (Freak, 1948) and dystocia's are rare in Greyhound (Sweeney, 1972). Bennett (1974) stated that, Bull dogs sometimes have slack abdominal musculature making it impossible for abdominal straining to lift the foetuses up to the pelvic cavity.

Moon *et al.* (1998) in their study to determine perioperative management and to calculate survival proportions in dogs undergoing caesarean section in the United states and Canada found that the most common breeds of dogs that underwent emergency surgery were Bulldog, Labrador Retriever, Boxer, Corgi's and Chihuahua and the most common breeds of dogs that underwent elective surgery were Bulldog, Labrador Retriever, Mastiff, Golden Retriever, and Yorkshire Terrier.

Bennur (1999) reported a significant effect of the breed of the dam on the incidence of dystocia in canines. In 75 cases of dystocia, 29.3 per cent were in German Shepherds, followed by Pomeranian (17.3 %), Boxer (13.3 %) and Dachshund (10.7 %).

Narasimha Murthy (2011) reported that in his study, dystocia was encountered in 18 different breeds and 18.12 per cent of all cases of dystocia were observed in Labrador retriever, followed by 12 per cent in German Shepherds, 9.4 per cent in Great Dane, 9.06 per cent in Spitz and 8.40 per cent in Dachshunds.

Vibha (2012) reported that uterine inertia was encountered in 17 different breeds and 16 per cent of all cases of uterine inertia were in Dachshund, followed by 14 per cent

in Labrador retriever, 12 per cent in German Shepherds, 10 per cent in Pug, 10 per cent in Saint Bernard and 08 per cent in Great Dane.

Shwetha (2012) reported that in her study, foetal dystocia was encountered in 20 different breeds and 23.81 per cent in Pug, followed by 18.01 per cent in Labrador retriever, 7.55 per cent in Dachshund, 7.00 per cent in Spitz and 6.40 per cent in Beagle.

2.4.2 Size

Bennur (1999) reported that 32 per cent of all dystocia cases encountered in his study were in small sized breeds and 68 per cent in medium and large breeds.

Polster (2006) stated that the possibility of dystocia was much higher in small breeds and reported that miniature breeds were more frequently affected in comparison to medium or large sized breeds.

Narasimha Murthy (2011) analysed the influence of the size of the breed on the incidence of dystocia in the bitch. Among 1236 cases of dystocia analysed, 422 cases (34.14 %) were in large sized breeds, and 360 cases (29.12 %) in small sized breeds. The number of animals with dystocia in medium and giant sized breeds were 228 (18.44 %) and 226 (18.28 %).

Shwetha (2012) analysed the influence of the size of the breed on the incidence of foetal dystocia in the bitch. Among 172 cases of foetal dystocia analysed, 86 cases (50 %) were in small sized breeds, and 73 cases (42.44 %) in medium sized breeds. The number of animals with foetal dystocia in giant and large sized breeds were 11 (6.40 %) and 2 (1.16 %).

Vibha (2012) analysed the influence of the size of the breed on the incidence of uterine inertia in the bitch. Among 50 cases uterine inertia analysed, 17 cases (34 %) were in large sized breeds and 14 (28 %) cases in medium sized breeds. The number of animals with uterine inertia in giant and small sized breeds were 10 (20 %) and 09 (18 %).

2.4.3 Age

Freak (1962) reported that, in 16 cases of dystocia in Mongrels 12 of them were over 5 years of age and 11 of which showed inertia and oversized foetus associated with low fecundity. Freak (1975) reported that, the dams aged 5 years and above were prone for complete primary uterine inertia. Smith (1965) also stated that, older bitches particularly those which are oversized and improperly exercised are particularly prone to development of uterine inertia.

Gaudet (1985) found that, no single age of the dystocia patients varied significantly when compared with overall intact female hospital population. In a total of 128 cases of dystocia, 24 were less than two years, 60 between two and four years, 25 between five and seven years, 10 between eight and ten years and three over ten years, with the age being unknown in 6 other dams.

Darvelid and Linde- Forsberg (1994) could not clearly establish the influence of age on the incidence of dystocia in bitches. In their study, 39 per cent were between two and eight years of age.

Bennur (1999) stated that there was a significant influence of age on the frequency distribution of dystocia in bitches. The incidence of dystocia was highest in bitches aged 2-4 years (50.7 %), and it was least in bitches aged over 8 years (6 %).

Polster (2006) reported that, 4.4 years is the average age of the obstetrically treated bitches and also stated that the average age of patients in relation to the causes of dystocia are higher in the following: Weak contractions (maternal) 4.5 years and single whelps (foetal) 5.2 years.

Narasimha Murthy (2011) reported that in his study, the highest number of dystocia cases was encountered in animals aged 2- 4 years (32.36 %), and that it was least in bitches aged over 8 years (11.18 %). The incidence of dystocia in bitches aged less than 2 years, 4- 6 years and 6-8 years were 24.78 per cent, 17.96 per cent and 13.75 per cent respectively.

Shwetha (2012) reported the highest number of foetal dystocia cases was encountered in animals aged 2-4 years (46.52 %), and that was least in bitches aged over 8 years (1.16 %). The incidence of foetal dystocia in bitches aged less than 2 years, 4-6 years and 6-8 years were 41.86 per cent, 8.72 per cent and 1.74 per cent respectively.

Vibha (2012) reported the highest number of uterine inertia cases was encountered in animals aged less than 2 years (30 %), and that was least in bitches aged over 8 years (10 %). The incidence of uterine inertia in bitches aged 2-4 years, 4-6 years and 6-8 years were 28.00 per cent, 18.00 per cent and 14.00 per cent respectively.

2.4.4 Parity

In retrospective study of 128 cases of canine dystocia, Gaudet (1985) found that, 37 per cent of the animals presented were primigravid.

Darvelid and Linde- Forsberg (1994) reported that, 28 per cent of the bitches experiencing dystocia had not littered before, 15 per cent had one litter, 15 per cent had two litters and 2 per cent had more than two litters. Further, 42 per cent of bitches which had whelped before also had problems during the previous parturition.

Bennur (1999) did not observe any significant influence of the parity of the bitch on the frequency distribution of dystocia in bitch. In his report, dystocia was observed in 39.3 per cent of animals during their first delivery and the rest were in plurigravida.

Polster (2006) in their study reported that 41.4 per cent of the patients needed obstetrical treatments were bearing their first litter. They also noted that chances of dystocia were lower as the number of birth per pups was higher.

Narasimha Murthy (2011) reported the incidence of dystocia as 31.01 per cent during first parity, 27.02 per cent during second or third parity, 22.98 per cent during fourth and fifth parity and 18.93 per cent after the fifth parity.

Shwetha (2012) reported the incidence of foetal dystocia as 43.61 per cent during first parity, 32.55 per cent during second or third parity, 14.53 per cent during fourth and fifth parity and 9.31 per cent after the fifth parity.

Vibha (2012) reported the incidence of uterine inertia as 54.00 per cent in animals during their first delivery and 46.00 per cent in plurigravida.

2.5 Maternal causes of dystocia

According to Arthur (1975), dystocia's which arise in the mother are due to either constriction of birth canal or due to a deficiency of expulsive forces. The constrictive forms most frequently encountered are inadequate pelvis, incomplete dilatation of cervix, uterine torsion and congenital malformation of the birth canal.

2.5.1 Complete primary uterine inertia

Complete primary uterine inertia has been recognised as one of the principle cause of dystocia in bitch. The condition is characterized by the failure of uterine muscle to expel normal sized foetus through the birth canal which is normal, except perhaps for an incompletely dilated cervix and characterized by contraction which are either completely absent, weak or infrequent.

Freak (1962) reported 25 cases of complete primary uterine inertia in a series of 222 cases of dystocia handled over a period of 15 years. The commonest single cause was low fecundity. The author suggested that low fecundity and is accompanying low hormonal influence appears to result in simple and complete failure to initiate whelping. However, complete primary uterine inertia was also recorded in 3 cases with high fecundity presumably due to uterine distension. Since most cases of primary complete uterine inertia were recorded in Scottish terrier, the author believed that, at least in this breed there is a hereditary predisposition. He has also reported nervous voluntary

inhibition of labour in 17 of 222 cases of canine dystocia. Jackson (1972) reported 37 per cent cases of primary complete uterine inertia out of 200 cases treated in Veterinary practise in England.

The condition has been reportedly seen more frequently in bitches of 5 years and above, often of low fecundity with 3 or fewer foetuses (Freak, 1975) and particularly among the first litter bitches which were taken away from their familiar environment. Among the factors suggested to be responsible for voluntary inhibition of labour include unfamiliar surroundings and lack of reassurance of an owner's presence (Freak, 1975). Psychic disturbances are also said to be an important cause of uterine inertia in bitches. Obesity and lack of exercise have also been suggested to be one of the causes of primary complete uterine inertia (Buckner, 1979).

Gaudet (1985) reported that primary complete uterine inertia accounted for 7.9 per cent of all maternal causes of dystocia.

Johnston (1986) described the diagnosis of primary complete uterine inertia in detail. The bitch with primary complete uterine inertia was generally bright and alert with partial to complete cervical dilatation. And exhibit a typical green tinged vaginal discharge with weak uterine contractions. The author also stated that these patients were generally normocalcemic and show no response to the administration of calcium and oxytocin and that it may recur at subsequent pregnancy.

The cause of primary complete uterine inertia appears to be multiple and include an inherent weakness in the uterine muscle such as in Scottish Terrier breed.

Overstretching of myometrium by an excessively large foetus, hydro allantois or unusually large number of foetuses, toxic degeneration due to bacterial infection, fatty infiltration of myometrium and senility (Arthur *et al*., 1989).

Darvelid and Linde- Forsberg (1994) reported that, primary complete uterine inertia accounted for 68 per cent of all maternal causes of dystocia in bitch. Further, nearly 90 per cent of the bitches with primary complete uterine inertia had only 1 or 2 pups. Similarly, in other studies several other authors have also demonstrated that the small litter size is an important cause of primary complete uterine inertia in bitch (Freak, 1962; Jones and Joshua, 1988 and Arthur *et al.,* 1989).

Bennur (1999) stated that primary complete uterine inertia was the most common maternal cause of dystocia in bitch. The condition was diagnosed in 50 (69.4 %) out of 72 animals with maternal dystocia.

Catharina and Gunilla (2007) reported that the most common reasons for dystocia are primary uterine inertia (60 %) and malpresentation of the foetus (26 %).

Narasimha Murthy (2011) reported that the incidence of complete primary uterine inertia in bitches with maternal dystocia as 63.15 per cent.

Vibha (2012) reported that the incidence of complete primary uterine inertia in bitches with maternal dystocia as 42.00 per cent.

2.5.2 Partial primary uterine inertia

Gaudet (1985) defined partial primary uterine inertia as those in which the second stage of labour begins, few pups are delivered but uterine contractions diminish or become ineffective prior to the delivery of the entire litter. In his studies involving 95 cases of dystocia's, partial primary uterine inertia was encountered in 33 dams.

Darvelid and Linde- Forsberg (1994) encountered partial primary uterine inertia in 42 of 182 cases of dystocia in bitch.

Bennur (1999) encountered partial primary uterine inertia in 20 (27.8 %) out of 72 cases of maternal dystocia.

In the reports of Narasimha Murthy (2011), primary partial uterine inertia accounted for 28.95 per cent of cases of maternal dystocia.

Vibha (2012) encountered partial primary uterine inertia in 27 (54.00 %) out of 50 cases of maternal dystocia.

2.5.3 Abnormalities of the maternal birth canal

2.5.3.1 Pelvic inadequacy

The area and the shape of the dams' pelvic inlet and volume of the pelvic cavity constitute an important group of factors necessary for unassisted delivery (Sloss and Dufty, 1980). In an analysis of various causes of dystocia in bitch, Freak (1962) observed that abnormalities of maternal pelvis, in only 1 of the 222 cases of dystocia treated over a period of 15 years. However, the author also reported 77 cases of dystocia due to relative

oversize of the foetus, 64 of which were in Scottish Terriers. The author conclude that, in this breed dystocia is partly caused by the pelvic diameter of the bitch, which is common with that in other achondroplastic breeds, is greater in the horizontal plane than in vertical plane.

Smith (1974) agreed that, a small pelvis as a contributing factor for dystocia in Boston Terriers. He also states that the most common abnormalities of the pelvis is due malunion of fracture which reduces the size of the pelvic canal and alters the contour of the pelvic girdle. The author also opined that obstructive malformations of the pelvis resulting from bone disease are relatively rare in canines.

Freak (1975) also stated that, abnormal bony pelvis was fairly common in Scottish terrier and makes the posteriorly presented foetus a hazard in this breed. Gaudet (1985) reports in a retrospective study involving 128 cases of canine dystocia, 5 cases were attributable to maternal anatomical abnormalities. In a similar retrospective study Darvelid and Linde-Forsberg (1994) reported that a narrow birth canal accounted for 1.1 per cent of all maternal causes of dystocia.

Compton (1987) clinically categorized all pelvises into adequate, questionable, and too small. He stated that the latter group is the least common and generally includes the congenitally or developmentally abnormal pelvis and in most cases caesarean section should be the mode of delivery. In all other pelvises with a vertex presentation, a trial of labour is indicated because the foetal head is an excellent pelvimeter. With proper foetal monitoring with an intrauterine pressure catheter, with the use of a pantograph to assist in

the diagnosis of an active- phase arrest, followed by a caesarean section at the appropriate time, there is no increase in foetal or maternal morbidity.

2.5.3.2 Abnormal maternal soft tissue structure

Persistence of Mullerian duct in the shape of a pillar of tissue running from vaginal roof to floor that may constitute an obstruction causing dystocia (Freak, 1962; Herr, 1978 and Darvelid and Linde- Forsberg, 1994). The other rare forms of maternal tissue abnormalities causing dystocia include vaginal prolapse (Wilson and Rajendran, 1961; Schutte, 1967; Troger, 1970 and Memon *et al*., 1993), abscess and neoplasm (Gaudet, 1985), a cord like structure crossing the lumen of the vagina dorsoventrally just caudal to the cervix and other congenital abnormalities (Smith, 1965).

2.5.3.3 Incomplete dilatation of cervix

Insufficient dilatation of cervix is reportedly not an important entity on its own, but seen commonly as a part of primary uterine inertia (Bennett, 1974). The abnormalities of uterus that cause dystocia in canines although less common include uterine rupture (Ficus and Hallenberg, 1971) and uterine torsion (Kulkarni *et al.,* 1965; Smith, 1965; Tompsett, 1971; Brown, 1974 and Prabhakar *et al.,* 1995). Dover (1966) described an abnormality of uterus, where the body and the first inch of both the horns were formed by thin fibrous tubes causing failure to deliver the pups and Kneen (1966) recorded single case of dystocia caused by aberrant round ligament encircling each uterine horn. Gaudet (1985) reported dystocia in bitch due to severe uterine adhesions from previous caesarean section. The presence of gravid uterine horn in an inguinal

region is a rare cause of dystocia has been published in several case reports (Heath, 1962; Ellet and Archibald, 1965 and Arthur *et al*., 1989).

2.6 Temperature, pulse, respiratory changes in animals with dystocia

Bennur (1999) recorded the temperature, pulse and respiration in 40 bitches with dystocia. The rectal temperature ranged from 99 ^{0}F to 104^0 F with a mean of 101.45± 0.31^{0}F. The pulse rate ranged from 56 – 90 per minute, with a mean of 75.36± 4.72 per minute. The respiratory rate varied from 17 to 60 per minute with a mean of 33.2±6.

Shwetha (2012) recorded the temperature, pulse and respiration in 66 bitches with foetal dystocia. The rectal temperature ranged from 98.9 ^{0}F to 104.2 ^{0} F with a mean of 101.58 ± 0.20^{0}F. The pulse rate ranged from 49 – 95 per minute, with a mean of 66.09 ± 1.72 per minute. The respiratory rate varied from 20 to 40 per minute with a mean of 29.63±0.87.

Vibha (2012) recorded the temperature, pulse and respiration in 22 bitches with complete primary uterine inertia. The rectal temperature ranged from 99.8 ^{0}F to 102.6 ^{0} F with a mean of 101.32 ± 0.16^{0}F. The pulse rate ranged from 77 – 88 per minute, with a mean of 81.26 ± 0.96 per minute. The respiratory rate varied from 16 to 24 per minute with a mean of 21.00 ± 0.70 and she recorded the temperature, pulse and respiration in 28 bitches with partial primary uterine inertia. The rectal temperature ranged from 100.1 ^{0}F to 102.7 ^{0} F with a mean of 101.17 ± 0.25^{0}F. The pulse rate ranged from 75 – 89 per minute, with a mean of 83.17 ± 0.13 per minute. The respiratory rate varied from 17 to 26 per minute with a mean of 24.00 ± 0.50.

2.7 Colour of vaginal discharges

Bennur (1999) reported that the most frequent colour of the discharge observed in the study was greenish (71.7 %). In 15.2 per cent of the cases, the discharge was brownish and also foul smelling. Haemorrhagic discharge was reported in 2 cases.

Johnston *et al*. (2001) stated that the presence of lochia or uteroverdin (greenish-blackish vulvar discharge) indicate that placental separation had occurred for at least one pup, and that it is a reliable sign that whelping should begin within 1 to 2 hours in a term bitch and a failure to do so signifies potential dystocia. They also stated that sanguineous vulvar discharge near term maybe caused by a traumatic birth, uterine torsion, or inadequate clotting factors.

In the reports of Narasimha Murthy (2011), there was neither any history nor any visible evidence of vaginal discharges in 4.16 per cent cases of dystocia. The discharge was greenish / blackish- green in 75 per cent, haemorrhagic in 4.16 per cent and brownish and foul smelling in 16.66 per cent of animals with dystocia.

In the reports of Vibha (2012), the colour of vaginal discharges observed in animals with uterine inertia in majority of animals had a greenish mucoid vaginal discharge in 82 per cent while the discharge was thick and blackish in 14 per cent of animals and reddish black in 4 per cent of the animals.

2.8 Duration of maternal dystocia in animals diagnosed as cases of uterine inertia

Bennur (1999) reported, in his study, only 5 per cent of the cases were presented between 2 to 4 hours after the onset of labour. The percentage of animals with dystocia presented between 4 to 6, 6 to 8, and more than 8 hours after the onset of labour were 12.5 per cent, 17.5 per cent, and 65 per cent respectively.

Narasimha Murthy (2011) reported the interval from onset of labour to its referral for treatment in different types of dystocia. The duration of dystocia was reported to be of 1 to 2 hours duration, 2 to 6 hours and more than 6 hours in 3.33, 1.67 and 25 per cent of cases of dystocia due to primary uterine inertia. Fifteen per cent of dystocia due to partial primary uterine inertia were presented between 4 to 8 hours after the onset of labour and another 20 per cent the duration of partial primary uterine inertia was more than 8 hours.

Vibha (2012) reported the duration of complete primary uterine inertia was between 4-6 hours and 6-8 hours in 9.09 per cent and 18.18 per cent of animals respectively. A majority of animals with complete primary uterine inertia were presented with a history of maternal dystocia of at least 8 hours of duration.

In three animals (10.71 per cent), the duration of dystocia due to partial primary uterine inertia was considered to be less than four hours in duration, while it was 46.42 per cent between 4-6 hours in 13 animals. Five animals were diagnosed as cases of partial primary uterine inertia of 6-8 hours in duration and another 7 animals of 8-12 hours in duration.

2.9 Evaluation of treatment protocols for primary uterine inertia

2.9.1 Oxytocin

McDonald (1965) opined that posterior pituitary extracts remains a useful agent to reinforce weak uterine contractions provided the cervix is open and the uterine muscles are not under tension. Prior to administering oxytocin it is recommended that the clinician should ascertain the position and presentation of the most caudal foetus, that there are no pelvic, vaginal and vulvar abnormalities and that the cervix is dilated (Mosier, 1986).

Various workers have frequently employed oxytocin either as posterior pituitary extract or in a synthetic form to augment uterine contractions. In such cases oxytocin has been commonly injected in doses of 5- 20 IU subcutaneously or intramuscularly (Freak, 1962 and Mosier, 1986), 3- 20 IU intramuscular (Johnston, 1986), 5- 15 IU intramuscular (Arthur *et al.,* 1989) or 0.5- 1 IU per lb body weight intramuscularly (Wallace and Davidson, 1995).

Freak (1962) stated that, while oxytocin is effective in early stages to overcome partial inertia, the uterus tends to lose its sensitivity towards the end of the prolonged parturition and its effect is relatively lower to deliver the final pup in large litter (Arthur, 1975).

Johnston (1986) recommended an interval of 30 minutes between oxytocin injections and if there is no progress even after 3 injections, caesarean section is recommended. He also noted that the possible adverse effect of oxytocin therapy such as premature placental separation may occur. The author stated that the most domestic females become refractory to oxytocin after repeated injections.

Mosier (1986) reported that, the effect of single dose will last approximately 15 minutes and recommended that when repeated doses were given there should be minimum interval of 30 minutes between the injections.

A recent study indicated that low plasma oxytocin concentration is one of the cause of primary uterine inertia in bitches with normal serum calcium concentrations and the condition aggravate in bitches with low calcium levels (Bergstorm *et al*., 2006[a]). This explains the interrelationship between oxytocin and calcium in the medical management of dystocia. Their study also showed that only about one-third of the bitches responded to oxytocin alone (Bergstorm *et al*., 2006[a]), indicating that calcium may be very beneficial in many cases of dystocia in which medical management is appropriate.

When oxytocin has been used alone, doses have historically been reported as high as 5-20 IU administered IM in the dog. However, studies by Davidson (2001) suggest that doses as low as 0.5-2 IU are more effective in increasing the frequency and quality of the contraction. Initial doses of 0.1 IU/kg are recommended and the dose can be increased incrementally to a maximum of 2 IU/kg (never exceed 20 U/dog in any breed) are recommended. Johnston *et al*. (2001) advocate oxytocin administration at 30-40 min intervals.

Bergstorm *et al*. (2006[a]) in their experiment to evaluate two treatment methods in bitches with primary uterine inertia in relation to blood concentrations of oxytocin, calcium and glucose reported that before treatment, plasma oxytocin concentrations are 35±15 pmol/1 in-group treatment with combination of intravenous calcium solutions and oxytocin and 30±15 pmol/1 in group treated with oxytocin only.

2.9.2 Dextrose

Freak (1962) reported a clinical case of hypoglycaemia associated with parturition in the bitch which was first taken as eclampsia and only true state of affairs was diagnosed, when there was a failure to respond to calcium, but, responded to 10 ml of 20 per cent glucose given intravenously.

Hypoglycaemia has also been reported to cause uterine inertia during parturition (Buckner, 1979). It has been reported that hypoglycaemia often mimics a clinical picture similar to hypocalcaemia in bitch and deferential diagnosis between hypoglycaemia and other causes of uterine inertia requires blood sugar tests (Buckner, 1979).

Successful correction of uterine inertia due to hypoglycaemia by intravenous glucose administration has also been reported in bitch (Bennett, 1974; Greiner, 1974 and Jones and Joshua, 1988).

Linde-Forsberg and Eneroth (2000) have proposed hypoglycaemia as a cause of primary inertia, especially in toy breeds of dogs. But Johnston *et al*. (2001) have stated that hypoglycaemia is uncommon in canine dystocia. Bergstorm *et al*. (2006[b]) also reported that many bitches had hyperglycaemia during dystocia, and this was thought to be secondary to high cortisol concentrations, which have been measured during normal labour in dogs (Olsson *et al*., 2003).

Lucio *et al*. (2008) reported that bitches exhibited normal glycaemia independent of the obstetric management. In their study on peripartum hemodynamic status of bitches under distinct obstetric conditions and also considered Labour is a stressful condition for

any female, signed by reflex release of cortisol and relative hyperglycaemia. However, an endocrine control through an acute release of insulin maintains glucose level at the normal range.

Bergstorm *et al*. (2006[a]) evaluated two treatment methods in bitches with primary uterine inertia in relation to blood concentrations of oxytocin, calcium and glucose and reported that before treatment, blood glucose values were 95.0 +/- 0.5mmol/l in group treated with combination of intravenous calcium solutions and oxytocin and 7.3 +/- 1.4mmol/l in group treated with oxytocin only.

2.9.3 Calcium

For proper function and response, neuromuscular tissues are dependent upon a normal balance of electrolytes within the body. In particular, uterine contractions are dependent upon adequate levels of calcium (Pamela, 2001). In cases where calcium metabolism has been comprised (i.e. by inadequate diet, by dietary supplementation of a nutritionally balanced diet exogenous calcium during pregnancy, or by extended periods of uterine contractions as seen in long deliveries), mildly depleted levels of serum calcium within a whelping bitch may inhibit the normal progression of delivery by interfering with uterine contractions (Pamela, 2001). Although hypocalcaemia is usually a problem in mid lactation, the condition can arise as a parturient complication**.** Freak (1975) observed that, some subclinical hypocalcemic bitches may not show the typical tremors and incoordination but, may exhibit restlessness coupled with uterine inertia.

Gaudet and Kitchell (1985) opined that, although dystocia due to hypocalcaemia is rarely confirmed by laboratory analysis it should be assumed to be present in cases of

uterine inertia that fail to respond to oxytocin administration. Their study documented at least 16 cases in which propulsive uterine contractions recorded following calcium therapy which failed to respond to oxytocin administration.

Darvelid and Forsberg (1994) reported that calcium and or oxytocin treatment was successful in relieving dystocia due to uterine inertia in 44 of 181 cases (24.3 %). In hypocalcemic bitch, slow intravenous injection of calcium borogluconate is usually recommended (Buckner, 1979; Bennett, 1980 and Johnston, 1986).

Several other studies have also documented the beneficial effect of calcium administration in bitch with dystocia due to uterine inertia (Smith, 1965; Bennett, 1974; Donovan, 1980; Shille, 1983; Gaudet, 1985 and Jones and Joshua, 1988).

The administration of calcium gluconate to treat dystocia can be directed and tailored based on the results of monitoring. Generally, the administration of calcium increases the strength of myometrial activity. When ineffective weak uterine contractions are detected calcium gluconate as 10 per cent solution (0.465 mEqCa^{++}/mm) can be given subcutaneously at the rate of 1 mL per 4.5 kg/ 10 lb body weight (Davidson, 2003).

Calcium therapy has shown to be useful in cases where bitches have failed to respond initially to oxytocin alone, and is a helpful therapy in the medical management of dystocia (Gaudet, 1985). A complicating factor is that many bitches primary inertia have serum calcium contractions that are similar to those with normal myometric contractions, making diagnosis of hypocalcaemia difficult and less ionized calcium is available diagnostically (Jackson, 1995).

Johnston *et al*. (2001) compared blood calcium levels between eutocia and dystocia bitches and concluded that there was no indication that blood calcium deficiency is the cause of uterine inertia in 17 of 26 animals' diagnosed dystocia because of uterine inertia. Several preparations of calcium are commercially available and 10 per cent calcium gluconate is one of the commonly used in bitches at a dose of 0.2ml per kg body weight by intravenous route or 5 ml per kg body weight subcutaneous route. Care should be exercised while administering calcium intravenously since cardiac arrhythmia is often observed and thus, it may require a concurrent chest auscultation.

2.10 Blood biochemical studies

There has been limited investigation of parturition in the bitches and there is little information published on clinical and obstetrical examination other than opinion and anecdote. While there are substantial data on hemodynamic and vascular changes during normal parturition in humans, little is known about the physiological events in the dog.

2.10.1 Blood glucose concentrations

Studies on the maternal hemodynamic changes occurring during normal parturition and its modifications in bitches with dystocia by Lucio *et al*. (2009) revealed that the blood glucose concentrations in bitches throughout the pregnancy were within the normal range, although pre-partum concentrations were significantly lower than many of the other time periods.

Some authors have proposed hypoglycemia as a cause of primary inertia, especially in toy breeds of dogs (Eneroth *et al*., 2000). Others reported that hypoglycemia

was uncommon in canine dystocia (Johnston *et al*., 2001). One study (Bergstrom *et al*., 2006[a]) reported that many bitches had hyperglycemia during dystocia, and this was thought to be secondary to high cortisol concentrations, which have been measured during normal labor in dogs (Olsson *et al*., 2003)

2.10.2 Serum calcium concentrations

Lucio *et al*. (2009) measured blood serum calcium concentrations in bitches with normal parturition, those with dystocia corrected by manipulative assistance or caesarean operation and those with uterine inertia treated by oxytocin administration.

Contraction of skeletal, cardiac, and smooth muscle cells (i.e. myometrium) requires ATP and calcium (Cunningham, 1992). In medical management of dystocia, the administration of calcium increased their strength (Davidson, 2001). Calcium therapy has been shown to work in cases where bitches have failed to respond initially with oxytocin alone, and was a helpful therapy in the medical management of dystocia (Gaudet, 1985). A complicating factor is that many bitches with primary inertia have serum calcium concentrations that are similar to those with normal myometrial contractions, making diagnosis of hypocalcemia difficult unless the level of ionized calcium available (Kraus and Schwab, 1990 ; Jackson, 1995). Several salts of calcium are commercially available, and 10 per cent calcium gluconate is commonly used in bitches at a dose of 0.2 ml/kg IV or 1–5 ml per dog SC (Johnston *et al*., 2001).

Emily *et al*. (2006) reported that serum biochemical concentrations remained within normal limits even when adjusted to account for hemodilution and also stated that,

hematological and serum biochemical profiles in pregnant bitches do not differ significantly from those found in normal adult dogs.

2.10.3 Serum magnesium concentrations

Magnesium infusion is used to prevent preterm labour in humans. Magnesium causes both smooth muscle relaxation and inhibition of myometrial contraction by both intracellular and extracellular mechanisms (Fomin *et al*., 2006; Popper *et al*., 1989). Therefore, magnesium may be of importance when evaluating causes of dystocia. The myometrial intracellular phosphorous and potassium concentrations have been compared in myometrial strips obtained from women in normal labour and in labour that was oxytocin resistant. The concentration of phosphorous was significantly higher in the normal group (Rezapour *et al*., 1996). The authors suggest that a dysfunction in the sodium-potassium pump can result in dystocia.

Magnesium, potassium and phosphorous were analysed in bitches with primary uterine inertia, they were within normal reference range with no signs of abnormality. It is unlikely that the serum concentration of the electrolytes is a cause to uterine inertia in the bitch. To evaluate this further, intracellular studies in the bitch may be required (Bergstorm, 2009).

2.11 Use of tocodynomometry for monitoring the whelping

A novel approach to veterinary obstetrical monitoring is the use of external monitoring devices using tocodynomometry and a hand held doppler to detect and record uterine activity and foetal heart rates (Davidson, 2001; Davidson, 2003). The use of

uterine and foetal monitors allows the veterinary clinician to observe and monitor labour, as well as manage labour medically or surgically with insight instead of guess work (Davidson *et al.,* 2003). The uterine sensor detects changes in intrauterine and intra amniotic pressures. Directing the Doppler perpendicularly over a foetus results in a characteristic amplification of the foetal heart sounds, distinct from maternal arterial or cardiac sounds, which enables determination of foetal heart rates (Davidson, 2001; Davidson, 2011).

The canine uterus each have characteristic patterns of contractility, varying in frequency and strength before and during different the stages of labour (Wallace, 1994; Davidson, 2003). Serial tocodynamometry in the bitch and queen permits evaluation of the progression of labour (Davidson *et al.*, 2003). During late term, the uterus may contract once or twice an hour before actual stage I labour is initiated. During stage I and II labour, uterine contractions vary in frequency from 0 to 12 per hour, and in strength from 15 to 40 mm Hg, with spikes up to 60 mm Hg. Contractions during active labour can last 2 to 5 minutes in duration. Recognizable patterns exist during pre labour and active (stages 1-3) labour (Wallace, 1994; Davidson, 2001). Further abnormal, dysfunctional labour patterns can be weak or prolonged, and often are associated with foetal distress and the completion of labour can be evaluated via tocodynamometry (Davidson, 2003; Davidson *et al*., 2003).

Davidson *et al*., (2003) reported the overall still birth rate in female dogs declined from 9.2 per cent to 2.5 per cent with incorporation of uterine and foetal monitoring into the whelping process. On other hand Medical therapy for dystocia, based on the

administration of oxytocin and calcium gluconate, can be directed and tailored based on the results of monitoring (Davidson, 2001; Davidson *et al*., 2003). Generally, the administration of oxytocin increases the frequency of uterine contractions, while the administration of calcium increases their strength. However the frequency of oxytocin administration is dictated by the labor pattern, and it is generally not given more frequently than hourly (Wallace, 1994; Davidson *et al*., 2003). Calcium gluconate 10 per cent solution with 0.465 mEqCa++/ml is given SC at 1 ml/5.5 kg BW as indicated by the strength of uterine contractions, generally no more frequently than every 4-6 hours (Davidson, 2001; Davidson, 2003 and Davidson, 2011).

Further Surgical intervention (caesarean section) is indicated if a bitch or queen fails to respond to medical management, or if foetal distress is evidenced despite adequate to increased uterine contractility (suggesting mismatch of maternal birth canal to foetal size, or foetal malposition or malposture incompatible with vaginal delivery), or if aberrant contractile patterns are noted by uterine monitoring (Davidson *et al.*, 2003; Davidson, 2011).

Materials and Methods

III. MATERIALS AND METHODS

3.1 Retrospective studies

The case records of canine dystocia presented to the obstetrical clinics between January 2007 to March 2012 were analysed to determine the incidence of foetal and maternal dystocia in dogs. The case sheets of bitches which had been diagnosed as cases of complete primary uterine inertia were further screened to evaluate the influence of various factors associated with complete primary uterine inertia. The influence in factors analysed were as here under.

3.1.1 Breed

The breed of the animal was recorded to determine the influence of breed on the incidence of complete uterine inertia in bitches.

3.1.2 Size of the breed

Based on the recommended body weights of the breed of the animal, dogs with complete primary uterine inertia were categorised into the following groups in an attempt to assess the relationship of the size of the bitch with the incidence of dystocia.

a. Toy and Small sized breeds (body weight less than 10 kg)

b. Medium sized breeds ((body weight between 10- 25 kg)

c. Large sized breeds (body weight between 25- 45 kg)

d. Giant sized breeds (body weight more than 45 kg)

3.1.3 Age of the dam

The age of the bitch diagnosed as uterine inertia was recorded during the course of the present study period, and based on the age of the animals; they were grouped into less than 2 years, 2 - 4 years, 4-6 years, 6 - 8 years and more than 8 years. The frequency distribution of dystocia among different age groups was compared to assess the possibility of predisposition of age of the animal to dystocia.

3.1.4 Parity of the dam

Animals with complete primary uterine inertia were categorised into either primipara or pluripara to analyse the influence of parity on the incidence of uterine inertia.

3.1.5 Litter size

An association of litter size on the prevalence of complete primary uterine inertia was evaluated in clinical cases and based on the litter size, animals were categorised into the following groups:

a) Animals carrying a single pup.

b) Animals with a litter size of 2-4.

c) Animals with a litter size of 4-8.

d) Animals with a litter size of more than 8.

3.2 Prospective studies

Prospective studies were carried out on 40 clinical cases of dystocia diagnosed to be due to complete primary uterine inertia. A diagnosis that dystocia due to Complete Primary Uterine Inertia was made, if the animal presented had a history and ultrasonic evidence of completion of pregnancy term, complete absence or the presence of very weak signs of first stage of labour, and the presence of greenish or blackish-green lochia on the perineum, vulva or vestibule for at least two hours. In some cases when the history was suggestive of complete primary uterine inertia, but there was no evidence of lochia in the perineum, the anterior vagina was further examined using a rigid vaginoscope to identify the presence of discharges or cervical opening (Mosier, 1986).

3.2.1 Clinical evaluation of female dogs with complete primary uterine inertia

A detailed clinical evaluation was carried out in 40 cases of dystocia diagnosed to be due to complete primary uterine inertia during the course of the present investigation. The parameters considered for clinical evaluation included the recording of temperature, pulse and respiration, the colour of vaginal discharges, duration of maternal dystocia, presence or absence of Ferguson's reflex and the assessment of vagina for relaxation. In addition, blood samples were also obtained from each animal before administration of drugs for determination of serum calcium, serum magnesium and blood glucose concentration.

3.2.1.1 Temperature, pulse and respiration

The temperature was recorded in Fahrenheit using a digital clinical thermometer. The pulse and respiratory rate was recorded as number per minute.

3.2.1.2 Colour of vaginal discharges

The colour of the discharge exhibited by the animals with complete primary uterine inertia was recorded by obtaining the history from the owner, visual inspection of the perineum and vulva and endoscopic examination of the vaginal lumen which was carried out using a rigid fibroptic vaginoscope (STORZ, KARL STORZ-ENDOSCOPY). The endoscopy also enabled the presence or absence of water bag in the vaginal lumen which could not be identified by digital examination of the vagina and the patency of the cervix.

Based on the nature of the vaginal discharges, animals with uterine inertia were categorized into,

a. Animals exhibiting thick black vaginal discharges.

b. Animals exhibiting greenish and mucoid vaginal discharges.

c. Animals exhibiting reddish black vaginal discharges.

3.2.1.3 Duration of maternal dystocia in animals diagnosed as cases of complete primary uterine inertia

The information regarding the approximate duration of maternal dystocia was obtained in all clinical cases of complete primary uterine inertia. In animals diagnosed as cases of complete primary uterine inertia, the duration of dystocia was determined as the interval from the time when the owner first observed vaginal discharges to the time of its presentation to the obstetrical clinic for the further treatment.

Based on the duration of dystocia, animals diagnosed as cases of complete primary uterine inertia were categorized into the following subgroups.

a. Animals in dystocia for two to four hours.

b. Animals in dystocia for four to six hours.

c. Animals in dystocia for six to eight hours.

d. Animals in dystocia for more than eight hours.

3.2.1.4 Blood glucose concentration

In all cases of complete primary uterine inertia, blood samples were drawn into EDTA coated vacutainers and the blood samples were immediately subjected for estimation of glucose. Glucose concentration was estimated using GOD/POD (Glucose Oxidase/ Peroxidase) method and the concentration was expressed as mg/dl. On the basis of blood glucose concentration animals were categorized as normoglycemic when the blood glucose was between 65 to 120 mg/dl, hypoglycaemic when it was less than 40 mg/ml and hyperglycaemic when it exceeded 120 mg/ml (Michael *et al.,* 1994).

3.2.1.5 Serum calcium and magnesium concentration

Blood samples from cases of complete primary uterine inertia were drawn into serum separating vacutainers. The blood samples were allowed to clot for six to eight hours and then subjected to centrifugation at 1500-3000 rpm for 15 minutes. The supernatant was then aspirated into serum storage vials and the collected serum samples were subjected for determination of calcium (mg/dl) and magnesium concentration (mg/dl) using the method of Monoreagent Endpoint.

On the basis of serum calcium concentration, the animals were considered as normocalcemic when the serum calcium concentration was between 9.2 to 11 mg/dl, hypercalcemic when it was greater than 11 mg/dl and hypocalcemic when it was less than 9 mg/dl (Michael *et al.,* 1994).

3.2.1.6 Recording of uterine tocodynomometry

The uterine contraction patterns were recorded in 40 cases of complete primary uterine inertia using a tocodynamometer*. The tocodynamometer used has been designed for human patients and the same was used in the present study as tocodynamometers developed for use in veterinary practice are not available in this country.

The tocodynamometer used consisted of a uterine sensor, a recorder, and a modem. (Plate 1). The lateral abdomen of the animal was clipped, and the animal was placed on an animal examination table in the lateral recumbency (Plate 2). The uterine sensor was placed and kept pressed on the abdominal skin and was secured with abdominal belts. During uterine monitoring, the bitches were kept quite avoiding any movements. Uterine contractions were recorded for 30 minutes. The uterine tocodynamometer displays the uterine activity both in terms of percentage as well as in the form of graph displaying the frequency of uterine contractions. In the present study, contractions less than 10 per cent were considered to be baseline uterine pressure and contractions in excess of 80 per cent were considered as nearly optimal.

*Tocodynamometer – SONICAID TEAM

The number of uterine contractions over a period of 30 minutes was recorded in each bitch with complete primary uterine inertia and this was done before initiation of any treatment for the relief of dystocia. The animal was considered to be in active labour if the tocodynamometer identified 8-10 contractions over a period of 30 minutes with each contractions lasting for 3-5 minutes.

3.3 Medical management of complete primary uterine inertia

Medical management of cases of complete primary uterine inertia under uterine tocodynamometric monitoring was carried out using oxytocin, dextrose and oxytocin or calcium and oxytocin. Each treatment was evaluated on a group of 10 animals to determine the most effective treatment for the relief of complete primary uterine inertia.

3.3.1 Efficacy of oxytocin

Ten animals diagnosed as cases of dystocia due to complete primary uterine inertia were injected with a single injection of one unit of oxytocin (Syntocinon ®, Novartis India Limited) intravenously. Following intravenous injection of oxytocin, the animal was given thirty minutes time to deliver a puppy. If a puppy was not delivered within thirty minutes, the treatment was considered a failure and no further injection of oxytocin was given. Uterine tocodynamometric studies were carried out up to 30 minutes after oxytocin injection.

3.3.2 Efficacy of dextrose and oxytocin

Ten animals with complete primary uterine inertia were infused with 25% dextrose at a dose of 1 g/kg body weight. Following completion of dextrose infusion, the

animals received 1 unit oxytocin injection intravenously. If a puppy was not delivered within thirty minutes after oxytocin infusion, the treatment was considered a failure and no further injection of oxytocin was given. Uterine tocodynamometric studies were carried out up to 30 minutes after oxytocin injection.

3.3.3 Efficacy of calcium and oxytocin

Ten cases of complete primary uterine inertia were infused with a solution containing 50 mg/ml of calcium gluconate and 87.5 mg/ml of calcium lactobionate equivalent to 9 mg of elemental calcium (Calcium-Sandoz®, Novartis India Limited). Calcium was slowly infused intravenously over a period of ten minutes at the rate of 1 ml/kg body weight, not exceeding 20 ml. This was immediately followed by intravenous infusion of oxytocin at the rate of 1unit/ kg body weight. If a puppy was not delivered within thirty minutes after oxytocin infusion, the treatment was considered a failure and no further injection of oxytocin was given. Uterine tocodynamometric studies were carried out up to 30 minutes after oxytocin injection.

Results

IV. RESULTS

4.1 Incidence of dystocia

A retrospective analysis of clinical records maintained in the Department of Veterinary Gynaecology and Obstetrics, Veterinary College, Bangalore revealed that a total of 20840 female dogs were presented for treatment of various reproductive disorders between January 2007 to March 2012. Out of these cases 1355 female dogs were presented with the complaint of dystocia. Thus, the overall incidence of dystocia in female dogs was determined as 6.50 per cent (Table 1).

4.2 Incidence of maternal and foetal dystocia in bitches

Table 2 presents the incidence of maternal and foetal dystocia in 1355 cases of dystocia that were presented between January 2007 and March 2012. Out of 1355 cases, 880 (64.94 per cent) were determined as maternal in origin and the rest (475, 35.06 %) as of foetal origin.

4.3 Maternal causes of dystocia

Table 3 presents the different types of maternal dystocia recorded in 880 bitches. In the present study, Complete Primary Uterine Inertia and Partial Primary Uterine Inertia together accounted for over 99 per cent of maternal dystocia. Partial Primary Uterine Inertia was the most frequently diagnosed maternal cause of dystocia (60.80 %), which was diagnosed in 535 animals. Complete primary uterine inertia was encountered in 342 (38.86 %) animals. Although, uterine torsion and vaginal septum as a cause of maternal

dystocia were also encountered in the present study, they were diagnosed in a fewer number of animals.

4.4 Factors influencing the incidence of complete primary uterine inertia in bitches

The various factors influencing the incidence of complete primary uterine inertia were analysed from the case records of 342 cases

4.4.1 Breed

Table 4 presents the frequency distribution of the incidence of complete primary uterine inertia encountered in the retrospective analysis. In the present study, complete primary uterine inertia was diagnosed more frequently in Dachshunds which accounted for 15.78 per cent of the cases. It was also diagnosed more frequently in Labrador Retrievers (13.74 %), German Shepherds (11.98 %) and Pugs (9.94 %). In other breeds, dystocia due to complete primary uterine inertia was diagnosed infrequently and its incidence as a cause of dystocia was less than 4 per cent.

4.4.2 Size of the breed

In the retrospective analysis carried out, complete primary uterine inertia as a cause of dystocia was more frequently identified in large (34.21 %) and medium (27.78 %) sized breeds (Table 5). The lowest incidence was recorded in toy breeds (18.13 %).

4.4.3 Age of the dam

In the retrospective analysis carried out, the age of the dam appeared to influence the frequency of complete primary uterine inertia. The frequency of complete primary

uterine inertia was highest (30.12 %) in animals less than 2 years of age. The incidence, then progressively declined with advancing age. Its incidence was recorded as 28.07, 17.84, 14.03 and 9.94 per cent in animals of the age group of 2-4, 4-6 , 6-8 and over 8 years respectively (Table 6).

4.3.4 Parity of the dam

Retrospective studies revealed that the frequency of occurrence of complete primary uterine inertia was about 8 per cent higher in primiparous bitches than pluriparous bitches. Its incidence was recorded as 54.09 per cent in primiparous bitches and 45.91 per cent in pluriparous bitches (Table 7).

4.3.5 Litter size

Table 8 presents the influence of litter size on the incidence of complete primary uterine inertia. Dams carrying a single foetus appeared to be more prone for the development of complete primary uterine inertia. 40 per cent of all cases of complete primary uterine inertia was associated with a single puppy syndrome. Further, bitches carrying a very large number of puppies also appeared to be more prone for complete primary uterine inertia (26.02 %).

4.4 Prospective studies

The prospective studies were carried out on 40 clinical cases of complete primary uterine inertia presented during the course of study in the Department of Veterinary Gynaecology and Obstetrics, Veterinary College, Hebbal, Bangalore. These animals were

utilized for the clinical evaluation of the animals with complete primary uterine inertia as well as for the evaluation of certain treatment protocols.

4.4.1 Rectal temperature, pulse and respiration

The rectal temperature in 40 animals with Complete Primary Uterine Inertia ranged between 99.9 and 102.2^0F and averaged 101.56 ± 0.18^0 F. (Table 9). The pulse rate ranged between 77 to 88 beats per minute and the average pulse rate was determined as 71.28 ± 0.76. Table 9 also shows the respiratory rate in 40 animals with complete primary uterine inertia. The respiratory rate ranged between 14 and 23 per minute and averaged 19.20 ± 0.52 per minute.

4.4.2 Colour of vaginal discharges

40 bitches diagnosed as cases of complete primary uterine inertia were examined and the colour of vaginal discharges were noted and the same is presented in Table 10. A majority of animals (80 %) had a greenish mucoid vaginal discharge, while the discharge was thick and blackish in 15 per cent of animals and reddish black in 5 per cent of the animals.

4.4.3 Duration of complete primary uterine inertia

In the present study, threefourths of the cases of complete primary uterine inertia were presented by the owners with the history of either greenish and mucoid, thick and black or reddish black vaginal discharges for a period in excess of 8 hours without any progress in the delivery (Table 11). None of the cases of complete primary uterine inertia

were presented within 2-4 hours after the initiation of labour. The rest of the cases were presented between 4-8 hours after the onset of labour

4.4.4 Blood glucose concentration (mg/dl)

The blood glucose concentration (mg/dl) was found to be within the physiological limits in 75 per cent of animals diagnosed as cases of complete primary uterine inertia. In these animals, the blood glucose concentration ranged between 81 and 117 mg/ dl and the mean blood glucose concentration was recorded as 110.74 ±5.82 mg/dl (Table 12). In 25 per cent of animals, the blood glucose concentration was determined to be higher than the physiological level and ranged between 134 and 157 mg/dl and averaged 141.82 ± 7.85. None of the animals exhibited any evidence of hypoglycaemia (< 40mg/dl).

4.4.5 Serum calcium concentration (mg/dl)

The serum calcium levels were determined to be within the normal physiological concentration in all the 40 animals with complete primary uterine inertia. The serum calcium level ranged between 9.4 and 10.9 mg/dl with a mean of 10.12 ± 0.48 mg/dl (Table 13).

4.4.6 Serum magnesium concentration (mg/dl)

The serum magnesium concentration was also determined in all 40 cases of complete primary uterine inertia. Its concentration was found to range from 1.82 to 2.28 mg/dl and the mean concentration was determined as 2.04 ± 0.17 mg/dl (Table 14).

4.4.7 Uterine tocodynamometric studies

In the present study, 40 clinical cases of dystocia diagnosed to be due to complete primary uterine inertia were evaluated for the pattern of uterine contractions using a uterine tocodynamometer. The strength of uterine as well as the frequency of uterine contractions were determined over a period of 30 minutes.

In every case of complete primary uterine inertia the strength of uterine contractions was considered to be poor and in most cases, it remained around 10 per cent (Table 15). Further, the contractions in every animal was also mild and infrequent (Table 16).

4.5 Evaluation of treatment protocols

4.5.1 Efficacy of oxytocin

The efficacy of oxytocin injections in cases of dystocia due to complete primary uterine inertia is presented in Table 17. The efficacy of oxytocin in relieving complete primary uterine inertia was evaluated in 10 animals and none of the animals responded to the treatment. Following injection of oxytocin, the strength of uterine contractions was observed to immediately increased to over 80 per cent, the contractions remained over 80 per cent for less than 2 minutes. However, the contractions were not associated with the expulsion of the pup and the uterine contractions and rapidly returned to the levels recorded prior to treatment. There were no further increases in the strength of the uterine contractions over the next 30 minutes and the recording of the uterine contractions were discontinued and the animal was subjected for caesarean section.

Similarly, injection of oxytocin was associated with an immediate peak of uterine contractions and the peaks gradually declined in height over the next few minutes.

4.5.2 Efficacy of dextrose and oxytocin

Ten animals diagnosed as cases of complete primary uterine inertia were infused with dextrose followed by oxytocin injection. Uterine tocodynamometric studies continued during the course of dextrose injection and also up to 30 minutes after oxytocin injection. The treatment protocol employed failed to relive dystocia in any of the animals (Table 18). Further, the strength and frequency of uterine contractions remained unaffected during dextrose infusion and its pattern after oxytocin therapy was similar to the pattern observed with the previous treatment protocol.

4.5.3 Efficacy of calcium and oxytocin

The efficacy of intravenous infusion of calcium, followed by oxytocin was evaluated in 10 animals with complete primary uterine inertia and the response observed is presented in Table 19. No positive response was observed in any of the treated animals with complete primary uterine inertia. Infusion of calcium did not influence the pattern of uterine contraction but the subsequent administration of oxytocin did brings about a change for a transient period.

Table 1: Overall incidence of dystocia in canines

No. of female dogs presented between January 2007 to March 2012	No. of cases of dystocia	Incidence (%)
20840	1355	6.50

Table 2: Incidence of maternal and foetal dystocia in bitches

Type of dystocia	No. of cases	Incidence (%)
Maternal	880	64.94
Foetal	475	35.06
Total	**1355**	**100**

Table 3: Maternal causes of dystocia in bitches

Type of cause	No. of cases	Incidence (%)
Primary complete uterine inertia	342	38.86
Primary partial uterine inertia	535	60.80
Uterine torsion	02	0.23
Vaginal septum	01	0.11
Total	**880**	**100**

Table 4: Breed wise incidence of complete primary uterine inertia

Breed	No. of cases	Incidence (%)
Dachshund	54	15.79
Labrador Retriever	47	13.74
German Shepherd Dog	41	11.98
Pug	34	9.94
Saint Bernard	34	9.94
Great Dane	27	7.90
Bull dog	14	4.09
Rottweiler	14	4.09
Golden Retriever	14	4.09
Beagle	14	4.09
Pekingese	7	2.05
Siberian Husky	7	2.05
Cocker Spaniel	7	2.05
Boxer	7	2.05
Dalmatian	7	2.05
Poodle	7	2.05
Mastiff	7	2.05
Total	**342**	**100**

Table 5: Incidence of complete primary uterine inertia based on the size of the breed

Size	No. of cases	Incidence (%)
Toy and small breed (Body weight < 10kg)	62	18.13
Medium breed (Body weight 10-25kg)	95	27.78
Large breed (Body weight 25-45 kg)	117	34.21
Giant breed (Body weight> 45 kg)	68	19.88
Total	**342**	**100**

Table 6: Age wise incidence of complete primary uterine inertia in bitches

Age (years)	No. of cases	Incidence (%)
<2	103	30.12
2-4	96	28.07
4-6	61	17.84
6-8	48	14.03
>8	34	9.94
Total	**342**	**100**

Table 7: Incidence of complete primary uterine inertia in bitches based on parity

Parity	No. of cases	Incidence (%)
Primipara	185	54.09
Pluripara	157	45.91
Total	**342**	**100**

Table 8: Incidence of complete primary uterine inertia in bitches based on litter size

Litter size	No. of animals	Incidence (%)
1	137	40.06
2-4	62	18.13
4-8	54	15.79
>8	89	26.02
Total	**342**	**100**

Table 9: Mean temperature, pulse and respiration in bitches (n= 40) with complete primary uterine inertia

Clinical parameters	Complete primary uterine inertia
Temperature (^{0}F)	101.56 ± 0.18
Pulse/minute	71.28 ± 0.96
Respiration rate/minute	19.20 ± 0.52

Table 10: Colour of vaginal discharges in bitches (n=40) with complete primary uterine inertia

Nature of discharge	No. of cases	Percentage (%)
Thick and black	11	15
Greenish and mucoid	27	80
Reddish black	02	05

Table 11: Duration of dystocia in bitches (n=40) with complete primary uterine inertia

No. of hours	No. of cases	Incidence (%)
2-4	00	00
4-6	08	6.25
6-8	15	18.75
>8	17	75.00
Total	**40**	**100**

Table 12: Blood glucose concentration in bitches (n=40) diagnosed as cases of dystocia due to Complete Primary Uterine Inertia

Blood glucose status	No. of cases	Percentage	Mean blood glucose concentration (mg/dl)
Normoglycemic (65-120 mg/dl)	34	75	110.74 ± 5.82
Hyperglycaemic (>120 mg/dl)	06	25	141.82 ± 7.85
Total	**40**	**100**	

Table 13: Serum calcium concentration in bitches (n=40) diagnosed as cases of dystocia due to Complete Primary Uterine Inertia

Serum calcium status	No. of cases	Incidence (%)	Mean serum calcium concentration (mg/dl)
Normocalcemic (9.0-11mg/dl)	40	100	10.12 ± 0.48
Total	**40**	**100**	

Table 14: Serum magnesium concentration in bitches (n=40) diagnosed as cases of dystocia due to Complete Primary Uterine Inertia

Serum magnesium status	No. of cases	Incidence (%)	Mean serum magnesium concentration (mg/dl)
Normomagnesaemic (1.8-3.0 mg/dl)	40	100	2.04±0.17
Total	**40**	**100**	

Table 15: Strength of uterine contraction pattern in cases of complete primary uterine inertia (n=40)

Strength of uterine contractions (%)	**No. of cases**	**Percentage**
Poor uterine contractions (less than 50%)	40	100
Moderate uterine contractions (50-80%)	00	00
Optimum uterine contractions (more than 80%)	00	00
Total	**40**	**100**

Table 16: Frequency of uterine contractions in cases of complete primary uterine inertia

Frequency	**No. of cases**	**Percentage**
Mild and infrequent	40	100
Mild and moderately frequent	00	00
Strong and frequent uterine contractions	00	00
Total	**40**	**100**

Table 17: Efficacy of oxytocin injection in cases of complete primary uterine inertia

No. of cases treated	No. of animals responding to treatment	% efficacy
10	00	00

Table 18: Efficacy of dextrose and oxytocin infusion in cases of complete primary uterine inertia

No. of cases treated	No. of animals responding to treatment	% efficacy
10	00	00

Table 19: Efficacy of calcium and oxytocin infusion in cases of complete primary uterine inertia

No. of cases treated	No. of animals responding to treatment	% efficacy
10	00	00

Plate 1 : Uterine tocodynamometer

Plate 2: Uterine tocodynamometric studies being carried out in an animal placed on lateral recumbency

Discussion

V. DISCUSSION

5.1 Incidence of dystocia

In the present study, the overall incidence of dystocia in female dogs was determined as 6.50 per cent (Table 1). The overall incidence of dystocia in the bitch is reported to be less than 5 per cent. (Root *et al*., 1995; Linde- Forsberg and Eneroth, 2000; Gill, 2002; and Sparkes *et al*., 2006). Bergstorm et al., 2009 also stated that the overall incidence of dystocia in the bitch as 5.7 per cent. The results of the present study are in close agreement with the above reports. However, A retrospective study by Walett-Darvelid and Linde-Forsberg (1994) of 182 cases of dystocia found that 42% of bitches that had whelped before had previously suffered from dystocia. The incidence of dystocia was almost 100 per cent in some breeds of dogs, especially those of achondroplastic type and those selected for large heads. Gill (2002) found that the frequency of dystocia varied from 9.1 per cent in Golden Retrievers to 85.7 per cent in Pekingese. Recently, Bergstorm *et al*. (2006[b]) using data from insurance claimed records of almost 2 lakhs insured bitches during the period between 1995 and 2002, estimated the overall incidence of dystocia to be around 16 per cent. In addition, there are some breeds which are achondroplastic and brachycephalic, where normal birth rarely if ever occurs, and elective caesarean operations are the routine (Arthur, 2001). Therefore, the reported variation in the frequency of dystocia in female dogs is because of the wide variation in the incidence of dystocia between different breeds, the tendency for the breeders to intervene, in some cases, prematurely and unnecessarily, the number of animals screened and the method of study used for arriving at the incidence.

5.2 Incidence of maternal dystocia

Dystocia is usually considered to be either of maternal or foetal in origin, and on the basis of this classification, in the present study, dystocia due to maternal origin were diagnosed much more frequently than of foetal origin. In 1355 cases of dystocia considered during retrospective analysis, dystocia due to maternal causes were diagnosed in 64.94 per cent of the cases (Table 2). The incidence of maternal dystocia observed in the present study is in close agreement with the incidence (61.09 %) reported by Vibha, (2012). Similarly, A higher incidence of maternal dystocia as compared to foetal causes has also been reported by Gaudet (1985); Darvelid and Linde- Forsberg (1994); Bennur (1999) and Narasimha Murthy (2011). The higher incidence of maternal dystocia observed in the bitch may be related to the prolonged duration of second stage of labour, temperament of the bitch and managemental practices such as nutrition and exercise.

5.3 Causes of maternal dystocia

In the present study, primary uterine inertia accounted for nearly 99 per cent of maternal dystocia (Table 3). Further, uterine inertia was classified as either complete or partial (Bergstrom *et al.,* 2010) and partial primary uterine inertia was diagnosed more frequently than complete primary uterine inertia. Uterine inertia has been previously reported as the most common cause of maternal dystocia in bitches (Gaudet, 1985; Darvelid and Linde- Forsberg, 1994; Polster, 2006; Narasimha Murthy, 2011; Vibha, 2012).

In the present study, vaginal septum and uterine torsion were also diagnosed as rare causes of maternal dystocia. Uterine torsion as a cause of maternal dystocia has been

reported to be rare in canines (Brown, 1974; Bennett, 1980; Jones and Joshua, 1988; Arthur *et al*., 1989). Vaginal septum as a cause of maternal dystocia has also been reported by Freak (1962); Herr (1978) and Darvelid and Forsberg (1994). Other reported causes of primary complete uterine inertia include over-stretching of the myometrium due to hydro allantois or large litters, toxic degeneration of the uterus, senility, obesity and lack of exercise, uterine torsion and trauma (Bennett, 1974; Freak, 1975; Buckner, 1979; Arthur *et al*., 1989).

5.4 Factors influencing dystocia

The observation that uterine inertia was the most frequent cause of maternal dystocia in the bitch necessiated a need for further investigation into the various factors influencing the incidence of uterine inertia. The factors investigated were the breed, age, parity and litter size in animals with uterine inertia. However, the study was restricted to only the factors influencing the incidence of complete primary uterine inertia as this type of case of uterine inertia is much more difficult to handle than the primary partial uterine inertia.

5.4.1 Breed

Complete primary uterine inertia was encountered in seventeen different breeds of female dogs (Table 4). Dachshund, Labrodor Retrivers and German Shepherds accounted for a little over 40 per cent of all cases of complete primary uterine inertia. Freak, 1948 reported that Dachshunds are particularly prone for primary uterine inertia. A similar opinion was expressed by Narasimha Murthy, (2011) and Vibha, (2012). It is also stated

that Scottish Terriers have a higher incidence of maternal dystocia and, in particular, primary uterine inertia which is believed to be of hereditary origin (Freak, 1962).

In the present study, it was surprising to note that complete primary uterine inertia was encountered frequently in Labrador Retrievers and German Shepherds. These two breeds have not been reported to be particularly susceptible for maternal dystocia. It is probable that a higher frequency of uterine inertia recorded in these breeds may be due to a higher population of these breeds in and around Bangalore city, rather than due to breed disposition. Similarly, few cases of uterine inertia were recorded in some of the breeds and are perhaps due to the fact that their numbers are relatively few in Bangalore.

5.4.2 Size of the breed

In the present study, complete primary uterine inertia was more frequently identified in medium and large sized breeds (Table 5). These breeds accounted for nearly 62 per cent of all cases of complete primary uterine inertia. Narasimha Murthy, (2011); Vibha, (2012) have also reported that the incidence of primary uterine inertia was more frequently observed in medium and large sized breeds. In the present study, medium and large sized breeds were represented by 10 different breeds which also happen to be the preferred breeds among animal lovers in Bangalore city. Therefore the population of medium and large breeds such as Labrador Retrivers and German Shepherds are much higher in Bangalore city and therefore, the higher number of uterine inertia in medium and large breeds may be more because of their higher population. Studies of Darvelid and Forsberg (1994) also could not clearly establish a relationship between the body weight of the bitch and the incidence of dystocia.

5.4.3 Age of the dam

In the present study, the highest incidence of complete primary uterine inertia was diagnosed in female dogs aged less than 2 years (30.12 %). Its incidence in animals of the age group of 2-4 years was 28.07 per cent (Table 6). Together, nearly 58 per cent of all complete primary uterine inertia was observed in animals less than 4 years of age. Similar observations have been made by Narasimha Murthy, (2011) and Vibha, (2012). Contrary to the above observation, Freak (1962), Smith (1974) and Freak (1975) reported that animals aged 5 years and above are more prone for dystocia, particularly due to uterine inertia. The low occurrence of inertia in animals aged 4 years and above, observed in the present study may be due to the fact that most canine owners prefer not to breed aged animals.

5.4.4 Parity

The incidence of complete primary uterine inertia was about 8 per cent higher in female dogs delivering for the first time as compared to the incidence in pluriparous bitches. The incidence in complete primary uterine inertia was observed as 54.09 per cent and 45.91 per cent in primiparous and pluriparous bitches respectively (Table 7). It was also observed that the incidence of complete primary uterine inertia decreased with increase in parity. A similar observation has been made by vibha, (2012). A higher incidence of complete primary uterine inertia in young female dogs is probably due to anxiety, fear, excessive interference of the owner, or change in certain managemental practices such as shifting the dog to unaccustomed area for the purpose of delivery.

5.4.5 Litter size

In the present study, female dogs carrying a single foetus or those with a litter size in excess of 8 foetuses appeared to be at high risk for complete primary uterine inertia (Table 8). Freak (1962) reported that the commonest single cause observed in animals with primary complete uterine inertia was low fecundity. The author suggested that low fecundity and its accompanying low hormonal influence appeared to result in simple and complete failure to initiate whelping. He also recorded a higher incidence of uterine inertia in animals with high fecundity and attributed the cause of uterine inertia in such animals to be due to uterine distension. Arthur *et al.* (1989) agreed that overstretching of the myometrium by an excessively large litter size was an important cause of uterine inertia in dogs.

5.5 Clinical evaluation of female dogs with complete primary uterine inertia

5.5.1 Rectal temperature, pulse and respiration

In the present study, the temperature, pulse and respiratory rates were within normal limits (Table 9) and none of the animals exhibited any clinical evidence of toxaemia or septicaemia, perhaps due to the fact that they were presented within a reasonably short time after the onset of delivery. However, In a dystocia patient, it is important to evaluate the patient for signs such as toxaemia, septicaemia or dehydration and this is reflected by changes in rectal temperature, pulse or respiratory rate. It is also reasonable to assume that respiration and pulse rate in dystocia patients may be affected by a variety of extraneous factors such as anxiety, transport, hospital environment and

handling and therefore changes in temperature, pulse and respiratory rate must be interpreted with caution.

5.5.2 Colour of vaginal discharges in bitches with complete primary uterine inertia

In the present study, 80 per cent of the animals diagnosed with complete primary uterine inertia exhibited a copious greenish mucoid vaginal discharges at the time of presentation (Table 10), suggesting that dystocia had been existing for a shorter period of time. It was also observed in the present study that the animals tended to exhibit a mucoid and greenish coloured discharge up to 8 hours after the onset of labour and invariably, live foetuses were encountered on ultrasound examination. Roberts, (1986) stated that the greenish nature of the vaginal discharges was due to placental separation. The discharge was thick and black in 15 per cent of animals with complete primary uterine inertia indicating complete placental separation of one or more foetuses. Further, this kind of discharge may indicate the possibility of dystocia existing for a longer period of time.

The vaginal discharges were reddish black in 5 per cent of animals with complete primary uterine inertia (Table 10) indicating that dystocia had been existing for a considerable period of time and the possibility of most of the foetus being invariably dead. Ultrasonographic examination of 2 animals with reddish black discharges failed to reveal any evidence of foetal viability. The results of the present study seem to suggest that the colour of the vaginal discharges can be used by the obstetrician to approximately determine the duration of dystocia as well as the chances of delivering viable puppies and it can be used as a prognostic indication of foetal viability.

5.5.3 Duration of complete primary uterine inertia

The viability of the foetus is dictated by the length of time for which complete primary uterine inertia has been existing and it is reasonable to presume that with increased duration of complete primary uterine inertia, a progressive detachment of the placenta occurs and the viability of the foetuses reduced. In the present study nearly 3/4th of the cases of complete primary uterine inertia were presented at a considerably advanced stage of uterine inertia (Table 11). Further, none of the cases were presented at an early stage. This observation indicates that in cases of complete primary uterine inertia, most owners are not aware as to how long they have to wait for normal delivery and when to consider the case as abnormal and present the animal for treatment.

5.5.4 Blood glucose concentration (mg/dl) in animals with complete primary uterine inertia

In the present study, the blood glucose concentration was within normal limits in 75 per cent of animals with complete primary uterine inertia (Table 12). The rest of the animals exhibited hyperglycaemia and none of the animals exhibited any evidence of hypoglycaemia as a cause of dystocia. The results of the present study are in agreement with the reports of Linde- Forsberg and Eneroth (2000) and Bergstrom *et al.* (2006[a]) that animals with uterine inertia may not be hypoglycaemic as it is commonly believed. On the other hand, hyperglycaemia may be observed in some animals secondary to high cortisol concentration. Regardless, blood glucose is something that can be rapidly measured to determine if hypoglycaemia is present and requires treatment.

Hypoglycaemia is commonly believed to be a major cause of uterine inertia and many of the clinicians routinely use dextrose infusion in an attempt to relieve uterine inertia. Linde- Forsberg and Eneroth (2000) proposed hypoglycaemia as a cause of uterine inertia, especially in toy breeds. However, Johnston *et al*. (2001) state that hypoglycaemia is uncommon in canine dystocia. One study, (Bergstrom *et al.,* 2006[a]) reported that many bitches had hyperglycaemia during dystocia, and this was thought to be secondary to high cortisol concentration, which have been measured during normal labour in dogs (Olsson *et al*., 2003).

5.5.5 Serum calcium concentration (mg/dl) in animals with complete primary uterine inertia

In the present study, none of the 40 dogs with complete primary uterine inertia had any evidence of hypocalcaemia (Table 13). The results of the present study is in agreement with the reports of Bergstrom *et al*. (2006[a]) and Vibha, (2012) who have similarly found that the serum calcium concentration was within the reference range in both bitches with normal parturition and bitches with primary uterine inertia. however, Barret (1949) stated that hypocalcaemia can be a cause of parturient complication. Gaudet and Kitchell (1985) opined that, although dystocia due to hypocalcaemia is rarely confirmed by laboratory analysis, it should be assumed to be present in cases of uterine inertia. On the other hand, Kraus and Schwab (1990) and Jackson (1995) are of the opinion that serum calcium concentrations in many bitches with primary inertia are similar to those with normal myometrial contractions, making a diagnosis of hypocalcaemia difficult unless ionized calcium level is available diagnostically.

5.5.6 Serum magnesium concentration (mg/dl) in animals with complete primary uterine inertia

The serum magnesium concentration in all 40 cases of complete primary uterine ineretia was ranged between 1.82 to 2.28 mg/dl and the mean concentration was determined as 2.04 ± 0.17 mg/dl (Table 14). Bergstorm, (2009) analysed the concentrations of magnesium, potassium and phosphorous in bitches with primary uterine inertia and found that they were within the normal reference range with no signs of abnormality. He concluded that serum it is unlikely that the serum concentration of electrolytes is a cause of uterine inertia in the bitch.

On the other hand, magnesium causes both smooth muscle relaxation and inhibition of myometrial contraction by both intracellular and extracellular mechanisms (Fomin *et al*., 2006; Popper *et al*., 1989). Therefore, magnesium may be of importance when evaluating causes of dystocia. The myometrial intracellular phosphorous and potassium concentrations have been compared in myometrial strips obtained from women in normal labour and in labour that was oxytocin resistant. The concentration of phosphorous was significantly higher in the normal group (Rezapour *et al*., 1996). The authors suggest that a dysfunction in the sodium-potassium pump can result in dystocia. Therefore, further intracellular studies in the bitch may be required to assess the relationship between the magnesium concentration and complete primary uterine inertia.

5.5.7 Uterine tocodynamometric studies

Labour is the physiological process by which foetus is expelled from the uterus through the vagina and is characterized as regular uterine contractions accompanied by

cervical softening and dilatation. The greatest impediment to understanding normal labour, besides recognizing its onset, is related to the evaluation of quality of uterine contractions, in terms of strength, duration and frequency, as they are not appreciable externally. To quantitatively evaluate the uterine activity, a tocodynamometric method has been developed based on recording of uterine contractions across the abdominal surface (Maul *et al*., 2003). The tocodynamometer do not require invasive probes, allowing them to be used for most pregnancies without risk to the mother or the foetus. External tocodynamometer monitoring devices are extensively used in human hospital births at present a tocodynamometer using specialized uterine pressure sensors suitable for use in canines has been developed allowing practitioners to objectively record uterine activity (Davidson, 2001). Tocodynamometry has become a new approach in canine obstetrics to diagnose delivery problems. However, its use in canine obstetrics even in some the developed countries appears to be limited.

Tocodynamometer designed for veterinary use is not available in India. It is also not known if the tocodynamometer designed for human obstetrical evaluation can be used even in canines. Due to non availability of veterinary uterine tocodynamometer, a uterine tocodynamometer designed for human obstetrics was used to evaluate the pattern of uterine contractions in female dogs diagnosed with complete primary uterine inertia.

The uterine contractions in cases of complete primary uterine inertia was monitored over a period of 15 minutes. Using a veterinary uterine tocodynamometer, Groppetti et al., (2010) considered that the bitch was in active labour when the uterine contractions were 10 mmHg in strength and frequency ranged from 4-12 contractions per

hour, each one of 2-5 minutes duration. After the onset of labour, the intrapartum detection of 0-3 mild contractions per 30 minutes without parturition, constituted recommendation for medical management of uterine inertia (Groppetti et al., 2010).

In the present study, the strength of the uterine contractions was less than 10 per cent (optimum - 80 % and above) for the entire duration 30 minutes of monitoring in all cases of complete primary uterine inertia (Table 15). Further, the number of strong uterine contractions were never more than 3 during the entire period of monitoring (Table 16). The uterine tocodynamometer was clearly useful in identifying that complete primary uterine inertia is associated with infrequent uterine contractions. This observation suggested that the animals need to be medically managed using ecbolics and therefore the animals were subsequently infused with either oxytocin, calcium or dextrose, either alone or in combination.

5.6 Evaluation of treatment protocols for complete primary uterine inertia

5.6.1 Efficacy of oxytocin

Various workers have frequently employed oxytocin either as posterior pituitary extract or as a synthetic form to augment uterine contractions. In such cases, oxytocin has been commonly injected in doses of 5- 20 units subcutaneously or intramuscularly (Freak, 1962; Mosier, 1986); 3- 20 units intramuscular (Johnston, 1986), 5-15 units intramuscular (Arthur, 1989) or 0.5- 1 unit per lb body weight intramuscularly (Wallace and Davidson, 1995). However, Davidson (2001) suggested that doses as low as 0.5- 2 units are more effective in increasing the frequency and quality of contraction and that

higher doses may bring about sustained contractions which may endanger the life of the foetus. In the present study therefore, oxytocin was used at a very low dose (1 unit).

In the present study all the animals with complete primary uterine inertia failed to respond to an intravenous infusion of oxytocin carried out in an effort to augment uterine contractions (Table 17). Uterine tocodynamometric studies revealed that infusion of oxytocin in a case of complete primary uterine inertia did result in uterine contractions, but the strength of uterine contraction in any of the animals did not exceed more than 50 per cent. Further, only one or two uterine contractions lasting less than a minute was identified following oxytocin infusion and the contractions returned to base line levels thereafter. It has been recommended by Groppetti et al.,2010 that physiological labour is possible when there are 12 contractions per hour, with each one of 2-5 minutes duration. The absence of clearly detectable uterine contractions following oxytocin injection explained why all cases of complete primary uterine inertia did not respond to oxytocin therapy in the studies of Vibha, (2012). It is possible that complete primary uterine inertia is associated with inherent defective oxytocin receptors in the uterus.

5.6.2 Efficacy of oxytocin and dextrose infusion

There are several reports in the literature suggesting hypoglycaemia as a cause of uterine inertia in bitches (Freak, 1962; Bennett, 1974; Griener, 1974; Buckner, 1979; Jones and Joshua, 1988; Linde Forsberg and Eneroth, 2000) and this forms the basis of using dextrose infusions as a line of medical treatment in cases of uterine inertia (Bennur, 1999; Narasimha Murthy, 2011).

In the present study, animals diagnosed with uterine inertia were first infused with dextrose, followed by intravenous infusion of oxytocin at the end of dextrose infusion. Dextrose was infused primarily with the objective of correcting hypoglycaemia if any and as a source of energy, and oxytocin to augment uterine contractions. It was observed in the present study that this line of treatment did not evoke any response in animals with complete uterine inertia (Table 18). Further, uterine tocodynamometric studies also revealed that infusion of dextrose prior to oxytocin infusion also did not augment the uterine contractions Further, uterine tocodynamometric studies also revealed that infusion of dextrose prior to oxytocin infusion also did not augment the uterine contractions.

5.6.3 Efficacy of calcium and oxytocin infusion

There are a number of reports which have documented successful treatment of uterine inertia with intravenous infusion of calcium (Smith, 1965; Bennett, 1974; Freak, 1975; Donovon, 1980; Shille, 1983; Gaudet and Kitchell, 1985 and Jones and Joshua, 1988). Generally, the administration of calcium increases the strength of myometrial activity and Davidson (2003) stated that when ineffective or weak uterine contraction are detected, calcium Gluconate as 10% solution should be given subcutaneously at the rate of 1ml/ 4.5 kg body weight. In the present study therefore, animals with complete primary uterine inertia were first treated with calcium in an effort to strengthen the uterine contractions followed by oxytocin to increase the frequency of uterine contractions.

In the present study, infusion of calcium and oxytocin failed to relieve uterine inertia in any of the cases where the inertia was diagnosed as complete (Table 19).

Johnston *et al*. (2001) concluded that there was no indication that serum calcium deficiency was a cause of uterine inertia. Bergstrom *et al*. (2010) also opined that in many cases of uterine inertia, calcium was not needed to initiate the labour and that oxytocin alone may be sufficient.

The present study clearly identified that maternal dystocia are overwhelmingly more common than foetal dystocia in bitch, and that uterine inertia is the principle cause of maternal dystocia. Uterine inertia appeared to be influenced by a significant number of factors notably, breed, age and litter size. The cause of uterine inertia appears to be multiple and include an inherent weakness in the uterine muscle, overstretching of the myometrium by an excessively large foetus, hydro allantois, unusually large number of foetuses, toxic degeneration of the uterine muscle, fatty infiltration of the myometrium, senility, obesity and lack of exercise and nervous voluntary inhibition (Freak, 1962; Buckner, 1979, Arthur *et al.,* 1989).

The present study did not try to elaborate the various causes of uterine inertia. In a clinical setup, it is perhaps also extremely difficult to pin point any one single cause as responsible for uterine inertia. This perhaps explains the reasons as to why a majority of the cases of complete primary uterine inertia failed to respond to medical protocol. The medical treatment aimed at augmenting uterine contractions is unlikely to be successful if there is a hereditary weakness of the uterine musculature. Similarly, complete uterine inertia due to toxic degeneration of uterine muscle or hydro allantois is also unlikely to respond to medical approaches. Therefore, a poor response obtained to medical therapy in cases of complete primary uterine inertia was on the expected lines. Further, when

obstetrician decides to try medical therapy in cases of complete primary uterine inertia, he should keep in mind that a high percentage of animals may have to be subjected for caesarean section. Further, in trying medical therapy, an obstetrician may be losing valuable time in terms of life of the puppy as oxytocin injections may induce enough contractions of the uterus to cause separation of the foetal membranes, but not the expulsion of the foetus, it therefore seems reasonable to suggest that caesarean section should be the first line of treatment for all cases of complete uterine inertia to maximize the foetal survival rate.

Summary

VI. SUMMARY

Retrospective studies were carried out to analyse the incidence of foetal and maternal dystocia in bitch and the influence of the breed of the dam, age, size of the breed, parity and litter size on the occurrence of complete primary uterine inertia in the bitch. In addition, a systematic evaluation of clinical evaluation of a patient diagnosed with primary complete uterine inertia as well as the efficacy of various medical protocols commonly employed for the treatment of primary uterine inertia was also investigated.

In the present study, dystocia due to maternal causes was encountered more frequently than foetal causes. It was also observed that complete primary and partial primary uterine inertias account for nearly 99 per cent of maternal causes of dystocia.

Dachshund, Labrador Retrievers and German Shepherds were the most frequent breeds in which dystocia were diagnosed to be due to complete primary uterine inertia. It was further observed that primary uterine inertia was much more common in animals under the age of 2 years and in older animals (> 8 years). The size of the breed appeared to have a significant effect on the occurrence of primary uterine inertia, with medium and large sized breeds being more commonly diagnosed as cases of primary uterine inertia. Primary uterine inertia was slightly more common in primiparous animals and animals carrying a single foetus or an excessively large litter were more frequently diagnosed as cases of primary uterine inertia.

The rectal temperature, pulse and respiratory rates were unaffected by the state of uterine inertia. Most cases of complete primary uterine inertia presented exhibited a

greenish mucoid vaginal discharge. Most animals with complete primary uterine inertia were presented at a significantly advanced stage of uterine inertia as compared to partial primary uterine inertia.

Hypoglycaemia as a cause of complete primary uterine inertia was not identified in any of the animals, while a few animals exhibited hyperglycaemia. Serum calcium and magnesium levels levels were also within the normal physiological range in every animal with complete primary uterine inertia and hypocalcaemia and hypomagnesaemia as a cause of complete primary uterine inertia could not be ascertained.

The uterine contractions were monitored using a human uterine tocodynamometrer to assess the strength, frequency and duration of uterine contractions in cases of complete primary uterine inertia. Complete primary uterine inertia was characterized by uterine contractions which were weak, occurred at a very low frequency and at very infrequent intervals.

The efficacy of oxytocin, dextrose and oxytocin and calcium and oxytocin were evaluated in cases of complete primary uterine inertia. The response of animals to any of the medical treatment protocol used was disappointing. Further, tocodynamometric studies revealed that oxytocin alone or in combination with dextrose or calcium did not restore the uterine contractions. It was concluded that cases of complete primary uterine inertia are best treated using the surgical option.

Bibliography

VII. BIBLIOGRAPHY

ARTHUR, G. H., 1975. Maternal dystocia. Veterinary Reproduction and Obstetrics, Edn4th, London Bailliére Tindall, 170-190

ARTHUR, G. H., 1986. The course of parturition: Physiology and maternal behaviour. *In*: Veterinary Reproduction and Obstetrics, Edn 4th, Bailliére Tindall, London, 134-139

ARTHUR, G. H., 2001. Maternal dystocia: causes and treatment. *In*: Veterinary Reproduction and Obstetrics, Edn 8th, W. B. Saunders Company. 229-244

ARTUHR, G. H., NOAKES, D. E. and PEARSON, H., 1989. Dystocia and other disorders associated with parturition. Veterinary Reproduction and Obstetrics, Edn 6th, Bailliére Tindall, London, 182-230

BARRET, E. P., 1949. A preliminary note on the treatment of uterine inertia in the bitch.*Vet. Rec*., **61**:783

BENNETT, D., 1974. Canine dystocia: a review of literature. *J. Small. Anim. Pract*., **15**: 101- 117

BENNETT, D., 1980. Normal and abnormal parturition.*In*: Current Therapy in Theriogenology: Diagnosis, treatment and prevention of reproductive disease in animals. D. A. Morrow (Hrsg.). Philadelphia: W. B. Saunders Company, 595- 606

BENNUR, S., 1999. A clinical study on etiology and treatment of canine dystocia. M.V.Sc thesis submitted to University of Agricultural Sciences, Bangalore.

BERGSTORM, A., NODTVEDT, A., LAGERSTEDT, A.S. and EGENVALL, A., 2006b. Incidence and breed predilection for dystocia and risk factors for caesarean section in a Swedish population of insured dogs. *Vet. Sur.,* **35**: 786-791

BERGSTRÖM, A., 2009. Dystocia in the Bitch: Epidemiology, aetiology and treatment. Doctoral Thesis, Swedish University of Agricultural Sciences, Uppsala

BERGSTROM, A., FRANSSON, B., LAGERSTEDT, A. S. and OLSSON, K., 2006[a]. Primary uterine inertia in 27 bitches: aetiology and treatment. *J. Small. Animal.Prac*., **47:**456-460

BERGSTRÖM, A., FRANSSON, B., LAGERSTEDT, A. S., KINDAHL, H. OLSSON, U. and OLSSON, K., 2010. Hormonal concentrations in bitches with primary uterine inertia.*Theriogenology*.,**73**: 1068-1075

BROWN, A. J., 1974. Torsion of the gravid uterus in a bitch. *Vet. Rec*., **87:** 202

BUCKNER, R. G., 1979. The genital system.*In*: Canine Medicine edited by Catcott, E. J. Edn 4th. Vol. 1 American Veterinary publication Drawer K.K. Santa Barbara California USA. 501-532

BURKE, T. J., 1986. Normal and abnormal parturition.*In*: Small Animal Reproduction and Fertility. A clinical approach to diagnosis and treatment edited by Burke, T. J. Lea and Febiger Philadelphia, USA. 335-345

CATHARINA, L. F. and GUNILLA, P., 2007. A survey of dystocia in the Boxer breed. *Acta. Vet. Scand*.,**49** (1): 8

CHATDARONG, K., TUMMARUK. P., SIRIVAIDYAPONG. S. and RAKSIL. S., 2007. Seasonal and breed effects on reproductive parameters in bitches in the tropics: a retrospective study. *J. Small. Anim. Pract*., **48**(8): 444- 448

CHRISTIANSEN, I. J., 1984. Reproduction in the dog and cat. Bailliére Tindall, London, 197-221

COMPTON, A. A., 1987. Soft tissue and pelvic dystocia. *Clin. Obstet. Gynecol*., **30**(1): 69-76

CONCANNON, P. W., MC CANN, J. P. and TEMPLE, M., 1989. Biology and endocrinology of ovulation, pregnancy and parturition in the dog. *J. Reprod. Fertil. Suppl*., **39**: 3-25

CUNNINGHAM, J.G., 1992. Fetal and neonatal oxygen transport, in Cunningham JG *Textbook of Veterinary physiologyt, ed 3. Philadelphia, Saunders.,* 516–521.

DARVELID, A.W. and LINDE-FORSBERG, C., 1994. Dystocia in the bitch: A retrospective study of 182 cases. *J. Sm. Anim. Pract*., **35**: 402-407

DAVIDSON, A. P., 2001. Uterine and fetal monitoring in the bitch. Veterinary Clinics of North America: *Small. Anim. Pract.,* **31**(2): 305- 313

DAVIDSON, A. P., 2011. Dystocia management. In JD Bonagura (ed); *Kirk's Veterinary Therapy XIV*. WB Saunders Co, Philadelphia, PA, in press.

DAVIDSON, A. P., GRUNDY, S. A. and FOLEY, J. E., 2003. Successful medical management of neonatal canine herpesvirus: a case report. *Comm Therio*., 3(1) Doc 1

DAVIDSON, A., 2003. Obstetrical monitoring in dogs. *Vet. Med*., **6**: 508- 516

DAVOL, P. A., 2000. Medical management of complications affecting delivery (Whelping).http://www.labbies.com/reproduction3.jtm

DONOVAN, E. F., 1980. Dystocia. *In*: Current Veterinary Therapy, Edn 7th, R. W. Kirk, W. B. Saunders, Philadelphia, 1212-1214

DOVER, P. M., 1966**.** An unusual case of dystocia in the bitch. *N. Z. Vet. J*., **14**: 201

ELLET, E. W. and ARCHIBALD, J., 1965.*In:* Canine Surgery, Edn1st. Archibald. Am. Vet. Publications Inc. Wheaton, Illinois

EMILY T. KIMBERELY, MARGRET L. CASAL, PATRICIA A.O'DONNELL, MARK, E, and HASKINS, 2006. Effects of pregnancy on complete blood cell counts and serum biochemical profiles in dogs. *Theriogenology,* **66:** 663–687.

ENEROTH, A., UHLHORN, M., SWENSSON, L., LINDE-FORSBERG, C. and HALL M., 2000. Valpningsproblem hos fransk bulldogg, skotsk terrier & bostonterrier (Dystocia in the French Bulldog, Scottish terrier and Boston terrier). *Hundsport., 112*(**9):**38-41.

FERGUSON, J. K. W., 1941. A study of the motility of the intact uterus at term. *Surg. Gynecol. Obstet.,* **73**: 359-366

FICUS, H. J. and HALLENBERG, N., 1971.An illustrated report of a case of dystocia in an Alsatian dog. *Kleintier-Prax*., **16**:128-129

FOMIN, V. P., GIBBS, S. G., VANAM, R., MORIMIYA, A. and HURD, W. W., 2006. Effect of magnesium sulfate on contractile force and intracellular calcium concentration in pregnant human myometrium. Am. J. Obstet. and Gynecol. **194**(5): 1384-90

FREAK, M. J., 1948.The Whelping bitch. *Vet. Rec.,* **60**: 295-301

FREAK, M, J., 1962. Abnormal conditions associated with pregnancy and parturition in the bitch. *Vet. Rec.,* **74***:1323*-1339

FREAK, M. J., 1975.Practitioners- breeder's approach to canine parturition. *Vet. Rec*., **96**:303-308

GAUDET, D. A. and KITCHELL, B. E., 1985. Canine dystocia. *Compend. Contin. Educ. Pract. Vet.,* **7**(5): 406- 418

GAUDET, D. A. J., 1985. Retrospective study of 128 cases of canine dystocia. *Am. Anim. Hosp. Assoc*., **21**: 813–818

GILL, M. A., 2002. Perinatal and late neonatal mortality in the dog. *PhD thesis. University of Sydney*

GREINER, T. P., 1974. Genital emergencies. *In*: Current Veterinary Therapy, V. Kirk. R. W., ed. Philadelphia. W.B. Saunders. Co., 909-915

GROPPETTI, D., PECILE, A., DEL, A. P., COPLEY, K., MINERO, M. and CREMONESI, F., 2010. Evaluation of newborn canine viability by means of umbilical vein lactate measurement, apgar score and uterine Tocodynamometry. *Theriogenology*., **74**: 1187-1196

HEATH, J. S., 1962. Indications and complications in caesarean section in the dog and cat: a review, *J. Small. Anim. Pract.,* **4**: 289-292

HERR, S., 1978. Persistent post cervical band as a cause of dystocia in a bitch. *Vet. Med. and Small. Anim. Clin*., **73**: 401

JACKSON, P. G. G., 1972. *In*: Veterinary Reproduction and Obstetrics, *Edn* 6th. Bailliére Tindall, London, 204

JACKSON, P. G. G., 1995. Handbook of Veterinary Obstetrics. *Edn* 1st. London, Saunders

JACKSON, P. G. G., 2004. Dystocia in the dog. *In:* Handbook of Veterinary Obstetrics, Edn2nd. Saunders and imprint of Elsevier Ltd. Edinbourgh, 141-166

JOHNSTON, S. D., 1986. Parturition and Dystocia in the bitch. *In:* Current Therapy in Theriogenology*: Edt*. D. A. Morrow, W. B., Saunders Philadelphia, 500-501

JOHNSTON, S. D., KUSTRITZ. M. V. R. and OLSON. P. N. S., 2001. Disorders of the canine uterus and uterine tubes (oviducts) .*In:* Canine and Feline Theriogenology. W. B. Saunders Company, Philadelphia, 206- 224

JONES, D. E. and JOSHUA, J. O., 1988. Reproductive clinical problems in the dog, *Edn* 2nd., Wright, London, 80-112

KNEEN, L. E., 1966. An unusual dystocia in the bitch. *Vet. Rec.,***79:** 399

KRAUS, A. and SCHWAB, A**.,** 1990. The concentration of ionized and total calcium in the blood of female dogs with uterine inertia. *Tierarztl Prax.,* **18**(6):641-3.

KULKARNI, P. E., PARELKAR, A. D., VAIDYA, V. G. and JAMKHENDKAR, P. R., 1965. Torsion of uterus in bitches. *Indian. Vet. J*., **42**: 55-57

LINDE-FORSBERG, C. and ENEROTH, A., 2000. Abnormalities in pregnancy, parturition and the periparturient period, *In*: Textbook of Veterinary Internal Medicine, Ettinger SJ, Feldman. EC, Edn 5th . Philadelphia, Saunders, 1527- 1539

LÚCIO, C. F., SILVA, L. C., RODRIGUES, J. A., VEIGA, G. A. and VANNUCCHI, C. I., 2009. Peripartum haemodynamic status of bitches with normal birth or dystocia. *Reprod Domest Anim*., **44**(2):133-136.

LUCIO, NORMAN, E. J. WOLSKY, K. J. and MACKAY, G. A., 2008. Pregnancy-related diabetes mellitus in two dogs. *N.Z. Vet. J*., **54**(6): 360-364

MAUL, H., MANER, W. L., SAADE G. R. and GARFIELD, R. E., 2003. The physiology of uterine contractions. *Clin. Perinatol*., **30**: 665-676

McDONALD, L. E., 1965. Veterinary Pharmacology and Therapeutics, *Edn* 3rd. Lowa State, University Press, Ames

MEMON, M. A., PAVLETIC, M. M. and KUMAR, M. S., 1993. Chronic vaginal prolapse during pregnancy in a bitch. **202**(2): 295- 297

MICHAEL, D. W., HAROLD, T. and GRANT, H. T., 1994.Small Animal Clinical Diagnosis by Laboratory Methods. *Edn* 2nd. W.B Saunders Company. Philadelphia. 141-157

MOON, P. F., ERB, H. N., LUDDERS, J. W., GLEED, R. D. and PASCOE, P. J., 1998. Perioperative management and mortality rates of dogs undergoing caesarean section in the United States and Canada. *J. Am. Vet. Med. Assoc.,* **213**(3): 365-369

MOSIER, J. E., 1986. Normal and abnormal parturition. *In*: Small Animal Reproduction and Fertility. A clinical approach to diagnosis and treatment edited by Burke, T. J. Lea and Febiger. Philadelphia, USA. 335-345

NARASIMHA MURTHY., 2011. Comparative evaluation of different methods of management of dystocia in dogs. Ph.D thesis submitted to Karnataka Veterinary, Animal and Fisheries Sciences University, Bidar

OLSSON, K., BERGSTRÖM, A., KINDAHL, H. and LAGERDTEDT, A.S., 2003. Increased plasma concentrations of vasopressin, oxytocin, cortisol, and the prostaglandin F2 alpha metabolite during labour in the dog. *Acta. Physiol. Scand*., **179**: 1-7

PAMELA., 2001. Pregnancy Management in Dogs and Cats. Cornell University, Ithaca, NY, USA, University of Florida, 2Gainesville, FL, USA

PATES, J. A., MCINTIRE, D. D. and LEVENO, K. J. 2007. Uterine contractions preceding labour. *Obstet. Gynaecol*., **110**: 566-569

POLSTER, K. J., 2006.Research on the frequency, the causes and the treatment of dystocia in bitches. Doctoral Thesis, Department of Medicine, Berlin, Germany

POPPER, L. D., BATRA, S. C. AKERLUND, M. 1989. The effect of magnesium on calcium uptake and contractility in the human myometrium. Gynaecologic and Obstetric Investigation., **28**(2): 78-81

PRABHAKAR, S., KOCCHAR, H. P. S. and NANDA, A. S. 1995.Torsion of one uterine horn in a primigravid Great Dane bitch. *Indian. Vet. J*., **72**: 416-417

REZAPOUR, M., ROOMANS, G. M., BACKSTROM, T. and ULMSTEN, U., (1996). X-ray microanalysis of myometrium in parturient women at term. *Journal of Submicroscopic Cytology and Pathology* **28**(1): 75-80

ROBERTS, S. J., 1971. Veterinary Obstetrics and Genital Diseases. Edn1st. Cornell University, Ithaca, New York, 237-239

ROBERTS, S. J., 1986. Parturition. *In*: Veterinary Obstetrics and Genital Diseases. (Theriogenology), Edn 2nd, Cornell University, Ithaca, New York. 251-255

ROOT, M. V., JOHNSTON, S. D. and OLSON, P. N., 1995. Estrous length, pregnancy rate, gestation and parturition lengths, litter size, and juvenile mortality in the domestic cat. *J. Am. Anim. Hosp. Assoc.,* **31**(5): 429-433

SCHUTTE, A. P., 1967. Vaginal prolapse in the bitch. *J. S. Afr. Vet. Med. Assoc.*, **38**:197-203

SHILLE, D. E., 1983. Diagnosis and Management of Dystocia in the Bitch and Queen. *In*: Current Techniques in Small Animal Surgery, Bojrob, M. J. Ed. Philadelphia, Lea and Febiger, 338-346

SHWETHA., 2012. Studies on incidence, risk factors and clinical management of fetal dystocia in dogs. M.V.Sc thesis submitted to Karnataka Veterinary, Animal and Fisheries Sciences University, Bidar

SLOSS, V. and DUFTY. J. H., 1980. *In*: Handbook of Bovine Obstetrics. W. Williams and Wilkins Company, Baltimore, USA

SMITH, K. W., 1965. Female Genital Tract. *In*: Canine Surgery. Edn.1st, Archibald, J. editor, Santa Barbara, CA. *American. Vet. Pub. Inc*. 641-676

SMITH, K. W., 1974. Female genital system. *In*: Canine Surgery, edited by ARCHIBALD, J. *Edn* 2nd. *Am. Vet. Pub. Inc*. Drawer Santa Barbara California. 751- 782

SMITH, M. S. and MC DONALD, L. E., 1974. Serum levels of luteinizing hormone and progesterone during the estrus cycle, pseudopregnancy and pregnancy in the dog. *Endocrinol*., **94**: 404

SPARKES, A. H., RAGERS, K., HENLEY, W. E., GUNN-MOORE, D. A., MAY, J. M. and GRUFFYDD, J. T. J., 2006. A questionnaire-based study of gestation, parturition and neonatal mortality in pedigree breeding cats in the UK.*J. Feline. Med. Surg.,* **8**(3): 145-157

SWEENEY, P. A., 1972. Some observation on behaviour of greyhounds. *J. Small. Anim. Pract.,* **13:**679-685

TOMPSETT, J. W., 1971**.** Torsion of a dog's uterine horn. *Mod. Vet. Pract*.,**52**: 52

TROGER, C. P., 1970. Vaginal prolapse in the bitch. *Mod. Vet. Pract*.,**51**: 38-41

VAN DEN WEIJDEN, B. C. and TAVERNE, M. A. M., 1994.Aspects of obstetric care in the dog. *Vet. Q*., **16**:20-22

VIBHA., 2012. Medical management of primary uterine inertia in female dogs. M.V.Sc thesis submitted to Karnataka Veterinary, Animal and Fisheries Sciences University, Bidar

WALETT-DARVELID, A. and LINDE-FORSBERG, C., 1994. Dystocia in the bitch: A retrospective study of 182 cases. *J. Small Animal Practice*., **35**: 402- 407

WALLACE, M. S. and DAVIDSON, A. P., 1995. Abnormalities in pregnancy, parturition and periparturient period.*In*: Veterinary Internal Medicine. Ed. By Ettinger, S. J. and Feldman, E. C. Edn4th. Vol. II. W. B. Saunders Company Philadelphia, USA. 1614-1624

WALLACE, M. S. Management of parturition and problems of the periparturient period of dogs and cats. *Sem. Vet. Med. and Surg. (Sm. An.).,* **9**(1): 1994

WILSON, F. D. and RAJENDRAN, E.I., 1961. Antepartum partial prolapse of vagina in a bitch. *Vet. J*., 38: 100-102

Abstract

VIII. ABSTRACT

A retrospective analysis reveled the incidence of dystocia in female dogs to be 6.5 per cent and dystocia due to maternal causes to be more frequent than the foetal causes. Complete primary and partial primary uterine inertias were identified to be the most frequent cause of maternal dystocia. Factors such as breed, size of the dam, age, parity and litter size were found to be closely associated with complete primary uterine inertia. Cases of complete primary uterine inertia were physiologically normal as revealed by the normal rectal temperature, pulse and respiration. They were also normoglycaemic, normocalcaemic and normomagnasaemic. Uterine tocodynamometric studies revealed that complete primary uterine inertia was associated with weak and infrequent uterine contractions therapeutic intervention using oxytocin, oxytocin and dextrose or calcium and oxytocin failed to significantly augment the uterine contractions. It was concluded that cases of complete primary uterine inertia are best treated using the surgical option.