STUDIES ON PERCUTANEOUS TRANSFEMORO-ACETABULAR NYLON ZAP STRAP APPLICATION FOR THE REPAIR OF SUBLUXATION / LUXATION IN HIP DYSPLASTIC DOGS

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JULY, 2016

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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 SURGERY AND RADIOLOGY

By

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CERTIFICATE

This is to certify that the thesis entitled "***STUDIES ON PERCUTANEOUS TRANSFEMORO-ACETABULAR NYLON ZAP STRAP APPLICATION FOR THE REPAIR OF SUBLUXATION / LUXATION IN HIP DYSPLASTIC DOGS***" submitted by **Mr. AMITH, N. G., MVHK 1445** in partial fulfillment of the requirements for the award of **MASTER OF VETERINARY SCIENCE in VETERINARY SURGERY AND RADIOLOGY** of the Karnataka Veterinary, Animal & Fisheries 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
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Affectionately dedicated to

Parents

and

Teachers

ACKNOWLEDGEMENT

Firstly, I am very much thankful to the Department of Surgery and Radiology for providing sufficient facilities in completing the work successfully. I am also thankful to dog owners who have supported me in all the matters that I was in need for completing this work.

I extend my sincere thanks to **Dr. B.N. Nagaraja**, Professor, Department of Surgery and Radiology, Chairperson, straight forward kindhearted gentleman who has given much attention towards me, for his scholarly guidance in finishing my research and thesis work. I express my sincere gratitude to him for his kind hearted help and support throughout my study period and in extending my professional knowledge.

I wish to express deep sense of gratitude to **Dr. M.S. Vasanth,** Dean, veterinary college, Hassan and advisory committee member for his guidance, inspiration, advice and concern towards me throughout my postgraduate programme.

Dr. L. Ranganath, Professor and Head, Department of Surgery and Radiology, advisory committee member, must be appreciated for his guidance, discipline, advice and moral support in completing research, thesis and improving me professionally. I am very much thankful to him for his support during my stay in the department.

Dr. Dhoolappa Melinamani, Assistant Professor, Department of Anatomy and Histology, and another member of my Advisory Committee, helped me a lot in my research and thesis work. I am very much thankful to him for his support towards me.

I owe special thanks to **Dr. Sreenivasa Murthy** and **Dr. V Mahesh** for faithful and kindhearted help in extending my professional knowledge.

I wish to convey my heartful thanks to **Dr. S. Prabhudev, Dr. H.V. Veerabhadraiah** and **Dr. Kemparaju** working as contract teachers, Department of VSR, for there kindhearted help and co-operation during my stay in the department for postgraduate programme.

I am equally thankful to all my seniors **Ravikumar S, Avinash, Shivkumar, Manjunath, Sachin, Estella** for their kind support in the department.

My Heartfelt thanks to **Dr. Amitha** and **Dr. Chethan Kumar** for their support and helping nature throughout my degree programme

I am also thankful to post graduates colleagues **Parameshwara** and **Pramodh** for their kind support in completing this work.

With affection I would like to thank all department staff members, **Mrs. Nandini, Mr. Narayana, Mr. Krishnamurthy, Mr. Lakshman** and **Mr. Sridhara** for their extra concern towards me, their assistance and help during P.G programme.

There are many others who supported me in various ways; I express my sincere thanks to them.

Bangalore

July, 2016 **(AMITH, N. G.)**

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LIST OF ABBREVIATIONS/SYMBOLS

%	Per cent
@	At the rate of
ALT	Alanine aminotransferase
AST	Aspartate Aminotransferase
Cm	Centimetre
° C	Degree Centigrade
° F	Degree Fahrenheit
dL	Decilitre
EDTA	Ethylene diamine tetra acetic acid
hr	Hour
IU/ L	International Units per Litre
Kg	Kilogram
lb	Pound
mg	Milligrams
mg/dL	Milligrams per decilitre
mg/kg	Milligrams per kilogram
mm	Millimetre
mmHg	Millimetres of mercury
Min	Minute
SE	Standard error
viz.	Namely
ROM	Range of motion

Introduction

I. INTRODUCTION

Dogs were first domesticated more than 10,000 years ago although exactly when and where was still debated. Since then, humans have enjoyed a long parallel history with dogs during our own progression from hunter-gatherers and then farmers, to modern city dwellers. Historically dogs lived in close proximity to humans and were used as working animals to herd livestock, hunt, and guard the home, and it is only recently that dogs have made the shift towards companion animals. Humans and dogs have co-existed for thousands of years. Increasingly, over the last few centuries, many pedigree breeds have been generated based on selection for particular physical and/or behavioral characteristics, which have been fixed and maintained by inbreeding within closed familial lines.

The development of such pedigree dog breeds can be both a blessing and a curse: desirable features are rigidly retained, but sometimes, undesirable disease-causing genes can be inadvertently fixed within the breed. Such diseases can reveal themselves only when two copies of the faulty version of the gene are inherited (recessive). Furthermore, if a Champion Sire is carrying such a disease gene, it can quickly spread across the whole breed. Similarly, if a breed is expanded from a small number of founder dogs, and one or more of these carry disease genes, again the disease frequency is likely to increase in the growing population. Sadly, some extreme forms of breed characteristics with a genetic basis can also contribute to issues of health and welfare. Due to this indiscriminate inbreeding, many of the developmental inherited diseases like hip and elbow dysplasia are now affecting large number of dog population.

Genetic predisposition and bad management practices are affecting number of canines. Canine Hip dysplasia (HD) was a common and potentially debilitating orthopaedic disease in dogs and characterized by laxity of the coxofemoral joint leading to secondary osteoarthritis, pain, and reduction in joint function (Dennis, 2012). The condition is manifested by decreased congruence between femoral head and acetabulum resulting in degenerative damage to the joint (Skurkova and Ledecky, 2009). Hip joint laxity was considered a major risk factor leading to abnormal weight-bearing forces and subsequent development of osteoarthrosis (Riser, 1975 and Frost, 1989). Dogs that are affected with osteoarthrosis due to hip dysplasia show laxity, not all dogs that show laxity develop osteoarthrosis (Smith, 1992).

Although the etiology of this condition was not fully understood, and it was a complex polygenic disease due to the small additive effect of many genes (Zhu *et al.*, 2009). Environmental factors such as sex, age, and body weight have been reported to influence the expression and severity of the disease (Manley *et al.*, 2007). In addition, diet has been shown to have a significant effect on the development of HD in dogs predisposed to the disease and on the prevalence, severity, and clinical signs of osteoarthritis (Nouh *et al.*, 2014). Environmental influences such as obesity, injury at a young age, overexertion on hip joint or round ligament tear at a young age, repetitive motion on forming coxofemoral joint, and excess dietary calcium/ vitamin D are said to play key roles. HD primarily affects medium-sized and large-breed of dogs and has high heritability of up to 95% (Molano *et al.*, 2015). Osteoarthritic hip joints can benefit from early detection and subsequent treatment. Physical therapists have long utilized manual testing techniques and clinical reasoning to diagnose early-onset joint osteoarthritis and therapeutic treatments consisting of correcting muscle dysfunctions, relieving pain, joint mobilizations, and advisement on lifestyle modifications could be equally beneficial to the canine patient.

In humans as well as in dogs, the diagnosis can be done by palpation, radiographic evaluation or by ultra-sound (Adams *et al*., 2000). In dogs, the radiographic examination was routinely used with well defined standardization of analysis (Haan *et al*., 1993).

Treatment is recommended when both joint laxity and pain are exhibited. Treatment in these dogs can be conservative or surgical. Conservative management consists of the administration of analgesics or chondroprotective agents, weight reduction, and exercise restriction. Options for surgical treatments include pectineal myectomy, lengthening of the femoral neck, and corrective osteotomies. (Raghuvir *et al*., 2013)

Conservative treatment in dysplastic young dogs was directed towards alleviation of pain. Most clinically dysplastic young dogs without, and even with, osteoarthrosis will respond to conservative management (Smith, 1992). Analgesics, chondroprotective agents, weight reduction, and exercise restriction have been used to decrease the clinical signs of pain (Palmoski and Brandt, 1979). As conservative management does not cure hip dysplasia, the development of debilitating osteoarthrosis may still occur (Raghuvir *et al*., 2013).

Surgical intervention was currently advocated by many surgeons who argue that progression of clinical dysplastic disease was inevitable with conservative management and that early operative treatment was required to return the joint to normal weight-bearing forces and prevent osteoarthritic changes (Schrader, 1982; Slocum and Devine, 1990).

Surgical technique includes pectineal myectomy, lengthening of the femoral neck, intertrochanteric osteotomy, and triple pelvic osteotomy (Marx *et al.,* 2014). Technique selection was influenced by the function and age of the animal, financial considerations, preference of the surgeon, the degree of osteoarthrosis present, and the conformation of the femur and acetabulum.

Complications associated with total hip replacement include infection (1% to5%), dislocation (5%), noninfected component loosening (6%), fractures (2%), and sciatic neuropraxia (2%) (Raghuvir *et al*., 2013). These treatments demand too long to exert beneficial effects were extremely invasive (Marx *et al*., 2014).

The control and Management of canine hip dysplasia was difficult due inbreeding and early detection of the disease since the young age group less than one year is affected more severely. There was a need to decrease the clinical signs of pain, increase the weight-bearing forces, to prevent joint instability and to maintain the range of hip motion and to develop an inexpensive surgical technique.

Based on the above facts, the Percutaneous transfemoro-acetabular nylon zap Strap application was carried out in hip dysplastic dogs, with the following objectives:

1) To study and to record the occurrence of subluxation and luxation predisposed by hip dysplasia in dogs.

2) To standardize the technique of Percutaneous transfemoro–acetabular nylon zap strap for the treatment of hip subluxation / luxation in hip dysplastic dogs.

3) To study clinical and gross changes at the surgical site after stabilization of the hip joint.

4) To study and to record the hemato-biochemical changes associated with the effect of Percutaneous trans femoro-acetabular nylon zap strap, before and after stabilization of hip subluxation / luxation in hip dysplastic dogs.

5) To study Radiographic changes associated with the effect of Percutaneous trans femoro–acetabular nylon zap strap application after the stabilization of hip subluxation / luxation in hip dysplastic dogs.

Review of Literature

II. REVIEW OF LITERATURE

Canine hip dysplasia (CHD) was a hereditary developmental disorder of the coxofemoral joint was first described in 1935 by Schnelle. Hip dysplasia has been reported in man and in most domestic animals (Olsson, 1978). In some breeds of dogs and cats, it was the most common cause of osteoarthritis (Allan, 2000). Dogs of almost all breeds can develop CHD, but it occurs more often in larger breeds. Irregular exercise pattern, excessive feeding and obesity, are prone to early progression of the disease. The identification of affected dogs can only be made by radiological examination, because clinical signs of CHD were unspecific and not all affected dogs show them (Olsson 1971). Both conservative and surgical treatment options are available. Depending on the severity of the disease, techniques that modulate the progression of the disease and help alleviate the pain are selected for management of hip dysplasia.

Available literature on percutaneous transfemoro-acetabular nylon zaps strap application for the repair of subluxation / luxation in hip dysplastic dogs are reviewed under the following headings.

2.1 Occurrence

- 2.1.1 Species
- 2.1.2 Breeds
- 2.1.3 Age
- 2.1.4 Sex

2.2 Etiology

2.3 Diagnoses

2.3.1 Clinical examination

2.3.2 Radiography

2.3.3 Ultrasonography

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2.3.5 Magnetic resonance imaging

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2.5 Treatment

2.5.1 Conservative management/ Non surgical treatment

2.5.2 Surgical treatment

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2.8.1 Ligaments of hip joint

2.8.2 Muscles of hip joint

2.8.3 Vascular supply of hip joint

2.8.4 Nerve supply to hip joint

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2.1 OCCURRENCE

Lust *et al.* (1973) stated that Hip dysplasia was one of the most common orthopaedic diseases in dogs, can lead to chronic pain and functional impairment.

Riser (1975) stated that canine hip dysplasia was common trait mainly in large and giant breeds, in pet and working dogs; with prevalence higher than 50% in some breeds and Clinical signs were more evident in dogs younger than one year of age due to hip instability, or in adult dogs with chronic pain from osteoarthritis

Riser *et al.* (1985) observed that larger breeds have a higher frequency of hip dysplasia compared to small breeds and Those with the highest frequency have loose skin, a heavy, rounded, stocky conformation, less developed muscles, and more than 5% to 10% subcutaneous fat.

Brass (1989) reported that Hip dysplasia was a developmental orthopaedic disorder characterised by the formation of a dysmorphic, lax (loose) coxofemoral (hip) joint and seen particularly in larger and giant breeds, the malformation and laxity lead to the abnormal wearing of bone surfaces and the appearance of the osteoarthritic signs of degenerative joint disease (DJD).

Fries and Remedios (1995) found that dogs affected by hip dysplasia wer born with normal hips, but quickly develop subluxation of the femoral head

Kaneene *et al.* (1997) canine hip dysplasia (CHD) may affect any dogs; small, medium or large breed, however it is estimated that more than 50 % of some breeds are affected.

Todhunter *et al.* (2003) found that Canine hip dysplasia (CHD) was a common developmental trait that affects primarily large breed dogs and was characterized by poor hip joint congruity, functional subluxation, and development of debilitating secondary hip osteoarthritis and Heritability estimates for CHD range from 0.11 to 0.68.

Rausch *et al.* (2004) stated that HD was one of the most common orthopaedic disorders in dogs, representing approximately 30% of the total orthopaedic cases and occurs in all breeds, however the higher prevalence were seen in middle and large size breeds, and in breeds with rapid growth.

Lewis *et al.* (2010) stated that hip dysplasia was developmental orthopaedic disorder characterised by the formation of a loose, ill-fitting coxofemoral (hip) joint and the malformation of joint leads to abnormal wearing of bone surfaces and the appearance of the osteoarthiritic signs of degenerative joint disease (DJD) .Since the osteoarthritis that develops was irreversible, hip dysplasia was often impossible to treat and so the only way to improve dog welfare through reducing the prevalence, was through genetic selection.

Roberts and McGreevy (2010) stated that the hip dysplasia affects essentially all breeds, with an estimated prevalence ranging from 1% to 80% according to the Orthopedic Foundation for Animals and it appears to occur at a relatively high rate in large-bodied and brachycephalic dogs as well as those with high body length to height ratios.

Stock *et al.* (2011) stated that the prevalence of hip dysplasia (HD) ranges from 0 to 74% (OFA) within the different breeds and heritability estimates have been reported ranging from 0.1 to 0.6.

Lavrijsen *et al.* (2014) opined HD was observed in all sizes of dogs, they are especially frequent in large breed dogs, which have a relatively high rate of longitudinal bone growth.

Ginja *et al.* (2015) reported that Canine hip dysplasia (CHD) was the most common inherited polygenic orthopaedic trait in dogs with the phenotype influenced also by environmental factors and Heritability estimates for CHD vary from 0.1 to 0.83 due to different pedigrees, methods used to calculate the heritability, and the hip phenotypes analyzed.

Molano *et al.* (2015) found that Canine Hip Dysplasia was complex disease that entails deformation of the hip joint, leading to secondary osteoarthritis and chronic cartilage degeneration and primarily affects medium-sized and large-breed of dogs and has high heritability of up to 95%.

2.1.1 SPECIES

Johnston (1992) reported that hip dysplasia was a common orthopaedic problem in dogs affecting most of the large breeds of dogs. The indiscriminate breeding of predisposed dogs had led the diseases to be more prevalent in the dogs than in other species of animals.

Keller *et al.* (2000) reviewed 684 radiograph of hip from 12 breeds of cats and indicated the frequency of the feline hip dysplasia in the feline population study was to be about 6.6%. Breeds like Persian and Maine coon were more predisposed to hip dysplasia.

Bo *et al.* (2012) opined that there was recognised variation in the incidence rate of DDH in humans worldwide ranging from 0.1 to10 % and Highest incidence populations can be found in Finland, Croatia and Canada (5–195 per 1,000) and with very low incidences amongst populations in sub-Saharan Africa and Hong Kong (0–0.1 per 1,000).

2.1.2 BREEDS

Martin *et al.* (1980) found that the rate of hip dysplasia varied widely among the dog breeds and commonly seen in English Bulldog (83.3%), Saint Bernard (73.3%), Kuvasz (71.4%), Newfoundland (63.8%), Golden Retriever (55.7%), Great Dane (16.1%), Afghan Hound (10.9%,), Siberian Husky (5.3%).

Fox *et al.* (1987) found that the frequency of the occurrence disease varies among breeds from as high as 70.5% in bulldogs and 48.2% in St. Bernard's to a low of 1.9% in borzois.

Leighton (1997) found that the prevalence of hip dysplasia in a breed like German Shepherds was about 50 to 55 %.

Swenson *et al.* (1997) who observed higher incidence of hip joint dysplasia in bitches of German shepherds, golden retrievers and Bern sheep-dogs, but who failed to detect such a relationship in Labradors, Retrievers, Rottweilers and Newfoundland dogs.

Allan (2000) reported that the hip dysplasia was also common in heavier breeds of cats like Persian, Maine coon and Scottish fold with prevalence of 0.34% in the feline populations of United Kingdom.

Chalmers *et al.* (2006) observed that Greyhounds, a breed with one of the lowest incidences of CHD.

Ranganath and Subin (2006) carried out hip scoring in 50 dogs of recognized breeds and observed that the incidence of hip dysplasia was highest in the Labrador Retrievers (51.72 %).

Coopman *et al.* (2008) reviewed that hip dysplasia commonly seen in Labrador Retrievers with prevalence was about 25–40% in UK.

Kaneene *et al.* (2009) reported that the prevalence of CHD was particularly high among larger breeds of dogs; estimates of around 20% have been found in Labrador retrievers, one of the most popular breeds in the world, and up to 70% in Saint Bernards.

Shiju Simon *et al.* (2010) Found that The breed wise incidence of the hip dysplasia was more common in Labrador Retriever (36.76 %) followed by Alsatian (25.60 %), Great Dane (9.19 %), spitz (6.61 %), Golden Retriever (4.77 %), Rottweiler (4.41 %), Doberman Pinscher (2.94 %), non-descript dogs (2.20 %), St. Bernard's and Lapsopso (1.83 % each), Neopolitian Mastiff (1.47 %), Dachshund (1.10 %) and others (1 %).

Stanin *et al.* (2011) found that HD was most frequently diagnosed in English Bulldogs (81.33%) and least frequently in Rhodesian Ridgebacks (3.33%).

Fels and Distl (2014) reported that breed wise prevalence of canine hip dysplasia vary widely from 1% to 75% and in German shepherd dogs, prevalence of CHD was estimated at 35%.

Lavrijsen *et al.* (2014) total dogs of 214 breeds were screened for HD, among these breeds highest prevalence was seen in the Bullmastiff (51.9%) followed by Italian Corso Dog (32.8%), Boxer (26.8%), Rhodesian Ridgeback (6.4%) and Belgian Shepherd Dog varieties (4–6%).

Molano *et al.* (2015) stated that hip dysplasia was an example of a complex disorder and high prevalence seen in breeds of large size, including the Labrador retriever, the German shepherd and the golden retriever.

Kimeli *et al*. (2015) found that German shepherd and Golden Retriever appear to be the most affected breeds

2.1.3 AGE

Henricson *et al.* (1972) Found that dogs over 24 months of age to have a higher incidence of CHD.

Riser (1993) found that the highest incidence of hip dysplasia seen in above three months to one year.

Fries and Remedios (1995) reported that increased in the frequency and severity of Developmental Dysplasia of Hip in genetically susceptible dogs, most markedly seen in first 6 months of life.

Citi *et al.* (2005) reviewed pelvic radiographs of 891 dogs in a retrospective study to determine the incidence of Unilateral Canine Hip Dysplasia (UCHD) and found out that 149 (16.7%) dogs had UCHD. The highest incidence of 37.6% was in dogs less than

12 months of age, with 22.8% in dogs between 12-24 months of age and 14.1% in dogs older than 72 months.

Manley *et al.* (2007) found that the dogs presented with clinical signs of hip dysplasia are younger than one year of age with hip instability.

Genevois *et al.* (2008) found that the incidence of Non arthrotic dysplasia among dysplastic dogs aged between 12 and 24 months was 68.6 % which was higher when compared to other age groups.

Krontveit *et al.* (2010) studied the occurrence of hip dysplasia in different breeds and the study revealed that Newfoundland was the breed with the highest occurrence of CHD with an18 months incidence risk of 36% and in Labrador retriever and Irish Wolf hound the 12 months incidence risks were 20%, and 10%, respectively.

Shiju Simon *et al.* (2010) stated that the highest incidence of hip dysplasia was found in the age group of over three months to one year (52.94 %), followed by over three year to six years(17.27 %), over one year to three years (14.70 %), over six year to nine years (7.35 %) and above nine years(7.35 %) respectively.

Smith *et al.* (2012) in their study observed that radiographic hip dysplasia prevalence figures from the Labrador Retrievers were 31% at 1 year of age, 42% at 3 years, and 73% at the end of life. This age dependency of CHD was indicated by the linear increase in the OA component of CHD over the life of the dogs.

Greene *et al.* (2013) Hip joint extension was one degree lower for each year of age, and osteophyte or enthesophyte size was 1 mm larger with each 3-year increase in age.

2.1.4 SEX

Martin *et al.* (1980) found that there was a significant difference in the risk of occurrence of CHD by sex and males had a 1.2 times higher risk of CHD than females.

Lust (1993) Found that Males and females are affected with equal frequency, in contrast to the disease in humans, where 80% of cases are female.

Fries and Remedios (1995) opined that in dogs, males and females are equally affected with hip dysplasia.

Wood *et al.* (2002) observed that the hip dysplasia scores were higher in males than in females in Labrador Retrievers.

Stanin *et al.* (2011) found that female (60.5%) dogs were found to be more affected than male (39.5%) dogs.

Hou *et al.* (2010) observed no difference in prevalence of HD among males and females in Labrador Retrievers by sex.

Jayaprakash *et al.* (2007) and Shiju Simon *et al.* (2010) found that Male dogs were found to be more affected (59.55 %) than female dogs.

Coopman *et al.* (2008) in a study among different dog breeds of Belgium found that the median and mean ages of the dogs was 560 days and 698 days, respectively, and 60 per cent of the affected were female.

Lavrijsen *et al.* (2014) revealed that Golden Retrievers had significantly higher prevalence of HD in females than in males with a male to female ratio of 1:1.3.

Kimeli *et al*. (2015) found that females were more affected than males.

2.2 ETIOLOGY

Hutt (1967) stated Hip dysplasia was neither a dominant character nor a recessive one, but was polygenic (quantitative) in nature, i.e. induced by an undetermined number of genes.

Hedhammer *et al*. (1979) reported that CHD was considered as an inherited, developmental disease with a polygenic mode of inheritance and the factors influences to develop this condition were complex genetic (25% to 85%), environmental, nutritional status, rapid growth dietary anion gap and inutero endocrine .

Shepherd (1986) found that Hip dysplasia was a polygenic trait caused by the interaction of hundreds of genes, each contributing a small part to the disease.

Fries and Remedios (1995) observed that excess energy consumption, dietary calcium and vitamin D were increase the frequency and severity of hip dysplasia in genetically predisposed dogs.

Leighton (1997) found that the polygenetic trait of HD was the reason why the reduction in the incidence of this disease was slow.

Maki *et al* (2000) reported that animals with a normal individual radiographic phenotype can still be carriers of CHD genes, which will be transmitted to their offspring and maintained in the population.

Todhunter and Lust (2003) stated that Dog with nondysplastic hips may have one or more alleles at loci that promote expression of CHD and when they recombine with other alleles from a mate during subsequent meiosis at fertilization Then CHD will result in the offspring

Todhunter and Lust (2003) stated that excessive laxity of the hip joint, abnormal endochondral ossification and many environmental factors could influence the development of the hip and the interaction of these factors leads to a development of HD condition in dogs.

Chase *et al.* (2004) opined that the aetiology of hip dysplasia was not fully understood and it was assumed that genetic factors play a decisive role and disease intensity and early development of clinical symptoms are amplified by environmental factors.

Ledecky *et al.* (2004) In dogs, HD was characterised as a manifestation of a quantitative hereditary traits and these traits are those which change by themselves and pass from one individual to another to a varying degree. These traits were affected by more than 2 pairs of genes and change according to factors in the outer environment.

Manley *et al*. (2007) reported that Environmental factors such as sex, age, and body weight have been reported to influence the expression and severity of the HD.

Zhu *et al*. (2009) reviewed that Canine HD was a complex polygenic disease due to the small additive effect of many genes.

Ginja *et al.* (2010) found that CHD was a genetic complex trait with a polygenic inheritance pattern influenced by environmental factors and both dominant and recessive modes of inheritance have been proposed.

Lewis *et al*. (2010) reported that Hip dysplasia was an important and complex genetic disease in dogs and has both genetic and environmental influences with evidence of gene effects at multiple loci confirming complex underlying genetics.

Zhou *et al.* (2010) reported several specific single nucleotide polymorphisms and positional candidate genes in dogs with CHD have been found to correlate with genes associated with the expression of OA and developmental dysplasia of the hip.

Friedenberg *et al*. (2011) observed the one mutation in the *FBN2* gene on CFA11 chromosome was significantly associated with CHD in Labrador Retrievers.

Lewis *et al*. (2011) stated that Hip dysplasia was considered as complex diseases with multiple genes as well as environmental factors influencing susceptibility to this disorder.

Wilson *et al*. (2013) opined that the mode of inheritance of hip dysplasia was considered to be multifactorial with both suite of genes and environmental factors contributing to the occurrence and severity of the disease.

Fels and Distl (2014) identified a few promising quantitative trait loci for OA associated with CHD and the CHD phenotype in German shepherds recently.

Nouth *et al*. (2014) reported that diet has been shown to have a significant effect on the development, prevalence, severity and clinical signs of osteoarthritis in HD in dogs.

Rhodes and Clarke (2014) reported that Nutrition plays a major role in the development of canine Developmental dysplasia of the hip (DDH) and In genetically predisposed dogs, a number of dietary excesses were believed to increase the frequency and severity of hip dysplasia and Excess energy consumption or 'overnutrition' was associated with rapid growth rates and relative overloading of the skeleton leads to development of hip dysplasia.

Ginja *et al*. (2015) reported that phenotypic expression of CHD was modified by environmental factors and dogs with a normal phenotype can be carriers of some mutations and transmit these genes to their offspring.

Kimeli *et al*. (2015) reported that aetiology of canine hip dysplasia was not fully understood, environmental influences such as obesity, injury at a young age, overexertion on hip joint or round ligament tear at a young age, repetitive motion on forming coxofemoral joint, and excess dietary calcium/ vitamin D are said to play key roles in development

2.3 DIAGNOSIS

Crawford and Pharr (1975) reported that the optimum age for diagnosis of hip dysplasia was believed to be 24 months of age.

Fry and Clark (1992); Adams *et al.* (2000) reported that in humans as well as in dogs, the diagnosis of the hip dysplasia can be done by palpation, radiographic evaluation or by ultra-sound examination of the hip joint.

Madsen *et al.* (1993) observed Procollagen type III aminoterminal peptide was a molecule liberated into extracellular fluid during active capsular fibrosis and Serum levels were higher in the synovial fluid of dysplastic dogs

Hielm-Bjorkman *et al.* (2003) stated that there are numerous descriptions of multifactorial systems, with numeric, visual analog, and descriptive scales to reproducibly evaluate joint pain associated with canine hip dysplasia.

Fluckiger (2007) reported that internationally, three scoring modes were widely use in diagnosis of canine hip dysplasia and they are The Federation Cynologique Internationale (FCI), the Orthopaedic Foundation for Animals (OFA), and the British Veterinary Association/the Kennel Club (BVA/KC).

Raghuvir *et al.* (2013) opined that physical therapists have long utilized manual testing techniques and clinical reasoning to diagnose early-onset joint osteoarthritis in hip dysplasia.

Biabecki *et al.* (2014) reported that diagnosis was based on symptoms observed during the clinical examination like ortolani test, the barlow manoeuvre, barden's manoeuvre, evaluation of the animal's posture, pelvic shape, and gait and imaging studies.

Bartolome *et al.* (2015) found that based on genetic and radiographic information, they developed an accurate predictive genetic test for early diagnosis of hip dysplasia in Labrador Retrievers.

Ginja *et al.* (2015) opined that commercial DNA test has been available for Labrador Retrievers using a blood sample and provides a probability for development of CHD but we await evidence that this test reduces the incidence or severity of CHD.

Schachner and Lopez (2015) opined that worldwide, there are five popular, standardized evaluation systems with distinct metrics that are used to grade canine radiographic coxofemoral joint conformation and degenerative changes in HD. Also added that CT and magnetic resonance imaging become more readily available and affordable, use of three-dimensional imaging methodologies will likely become an integral part of diagnosis and assessment of canine hip dysplasia.

2.3.1 CLINICAL EXAMINATION

Chalman and Butler (1985) reported that Ortolani test was used as subjective evaluation of coxofemoral joint laxity originally designed for diagnosis of human congenital hip dislocation and also used as a CHD screening test in dogs.

Fry and Clark (1992) observed the two general behaviours often attributed to CHD, including lameness in young dogs less than one year of age that increases with activity or trauma, and gait abnormalities and hind limb muscle atrophy in older dogs.

Vezzoni *et al.* (2005) reported that common clinical signs associated with hip dysplasia were slight to moderate lameness; gait and running abnormalities, such as shortened stride length and bunny hopping; difficulty in rising and reluctance to climb stairs.

Ginja *et al.* (2008) stated that the Ortolani test is the most common and popular physical maneuver that was used in veterinary medicine to diagnose HJL in young dogs aged about 4–12 months.

Ginja *et al.* (2010) reported that Bardens' test was an examination technique designed to evaluate the hips of babies aged younger than six months and was thought to be more sensitive for detecting coxofemoral joint laxity and/or shallow acetabulum in puppies 6–8 weeks of age.

2.3.2 RADIOGRAPHY

Henry (1992) reported that OFA score was measured from the ventrodorsal, extended-hip position and It was classified into 7 grades score and were

1. Grade 1 (Excellent) - Superior conformation was present with a very tight joint space and almost completes coverage of the femoral head by the socket.

2. Grade 2 (Good) - Most of the socket covers the femoral head and there was a congruent joint space.

3. Grade 3 (Fair) - Slightly incongruent (subluxated) joint space with the persistence of good femoral head coverage by the socket.

4. Grade 4 (Borderline) - There was usually more incongruency present than what occurs in the minor amount found in a fair but there were no arthritic changes present that definitively diagnose the hip joint being dysplastic. Most dogs with this grade (over 50%) show no change in hip conformation over time and receive a normal hip rating; usually a fair hip phenotype.

5. Grade 5 (Mild) - The joint was obviously incongruent or subluxated. Usually there was a shallow socket only partially covering the femoral head.

6. Grade 6 (Moderate) - There was significant subluxation present where the ball was barely seated into a shallow socket causing joint incongruency. There were secondary arthritic bone changes usually along the femoral neck and head (termed remodeling), acetabular rim changes (termed osteophytes or bone spurs) and various degrees of trabecular bone pattern changes called sclerosis.

7. Grade 7 (Severe) - There was a shallow socket only partially covering the femoral head. There were pronounced arthritic changes at the joint.

Haan *et al.* (1993) reported that the radiographic examination was routinely used with well defined standardization of analysis for diagnosis of hip dysplasia in dogs.

Lust *et al.* (1993) observed that radiographically there was incongruence between the articular surfaces, with razing of the acetabulum, flattening of the femur head, coxofemoral sub-laxity or laxity, and secondary osteoarthritis alterations in hip dysplastic dogs and radiographic finds are not always compatible with the clinical signs. They also reported that University of Pennsylvania researchers developed a quantitative method to evaluate canine hip conformation and the primary distinction of this method was that passive hip joint laxity was measured in addition to subjective radiographic conformation. Three radiographic views were evaluated by PennHIP includes a standard hip-extended view for evidence of degenerative joint disease; a compression view for congruity between the femoral head and acetabulum; and a distraction view, for joint laxity. The closer the score is to 0, the better the fit, i.e., minimal femoral distraction, but a score of 1 indicates severe laxity and associated femoral distraction.

Smith *et al.* (1993) reported that the Orthopaedic Foundation for Animals evaluation was performed on hip-extended radiographs under heavy sedation or general and conformation was categorized as excellent, good, fair, borderline, mild, moderate, or severe. The first three categories are considered to be normal while the last three are dysplastic.

Willis (1997) reported that In UK, the British Veterinary Association and Kennel Club (BVA/KC) screening method was performed to diagnose canine hip dysplasia (CHD). Under this method, the pelvic area of dogs older than one year was radiographically screened for nine different categorical traits, and their sum for both hips provides a hip score (HS) between 0 and 106, with 0 being a perfect unaffected hip and 106 the maximum degree of CHD.

Farese *et al.* (1998) reported that dorsolateral subluxation was used to quantify joint laxity in a position to simulate weight-bearing. During general anaesthesia, pressure was applied to the femur at the level of the stifle while imaging the dog in ventral recumbency. Joints with less than 45% coverage of the femoral head by the lateral aspect of the cranial acetabular rim have an increased chance of developing joint changes and OA over time compared with those with a higher percentage (55%) of coverage.

Fluckiger *et al.* (1999) opined that the radiographic information on HJL in hip dysplasia was obtained using radiographic techniques such as PennHIP, dorsolateral subluxation (DLS) and half-axial position methods. Signs of DJD are evaluated using the standard ventrodorsal hip-extended view (SVDV).

Torres and Silva (2001) reported that for the conventional radiographic method (CRM), 30x40cm radiographic films were used and the animals were placed in dorsal decubit, with the posterior members extended, internally rotated, with the patella overlaying the sagital plan of the femur, the femurs parallel between them and in relation to the vertebral column and the pelvis in diagnosis of hip dysplasia in dogs.

Malm *et al.* (2007) observed that variation in the degree of muscle relaxation associated with sedation or anaesthesia during radiograph of hip can influence the ability to identify joint abnormalities by as much as 50%.

Rocha and Torres (2007) stated that the distraction radiographic method was more efficient in detecting passive articular laxity when compared to the conventional radiographic method, which was more efficient in evaluating osteoarthritis alterations in hip dysplastic dogs. They also repoted that for the ultrasonographic examination, the puppies were manually held. For the radiographic examination the animals received 0.044mg/kg atropine sulphate, subcutaneously; 10 minutes later (0.5mg/kg) xylazine chlorhydrate intramuscularly, and 10 minutes later (0.5mg/kg) diazepam intravenously.

Comhaire *et al.* (2009) opined that the Federation Cynologique Internationale (FCI) was one of the largest canine organizations in the world and includes kennel clubs from across Europe, Asia, Africa, and South America. Extended hip and abducted hind limb radiographs performed at 1 year of age (18 months for large breed dogs) were scored according to the official FCI system by radiologists approved by breed-specific kennel clubs. Each joint was assigned a grade of A–E, with A representing healthy and E representing severe dysplasia.

Risler *et al.* (2009); Szabo *et al.* (2007) reported that radiography has long been the gold standard to assess and quantify joint changes associated with CHD joint remodelling.

Bartolome *et al.* (2015) reviewed that a standard ventro-dorsal hip X-ray of each dog was taken and X-rays were evaluated according to the FCI (Federation Cynologique Internationale) official scale for hip dysplasia and graded as:

A = no signs of CHD,

B = near normal hips,

C = mild signs of CHD,

D = moderate signs of CHD,

E = severe CHD.

Ginja *et al.* (2015) reported that the definitive CHD diagnosis based on radiographic examination involves the exposure to ionizing radiation under general anaesthesia or heavy sedation to evaluate the signs of degenerative joint disease, incongruence, and/or passive hip joint laxity but the image does not reveal the underlying genetic quality of the dog.

Kimeli *et al.* (2015) reported that radiographic techniques have been developed and was widely used to identify hip dysplasia in dogs for purposes of breeding.

2.3.3 ULTRASONOGRAPHY

Greshake and Ackerman (1992) visualized acetabulum without the interference of ossification of the femur head in 6- week old dogs using ultrasonography.

Gerscovich (1997) opined that Ultrasonography in human neonates is the reference technique for the definitive diagnosis of developmental hip dysplasia.

O'Brien *et al.* (1997) reported that Dynamic ultrasonography was used in puppies at 8–16 weeks of age, to quantify HJL in hip dysplasia.

Rocha and Torres (2007) stated that the static ultrasonography did not prove to be a sensitive method to earlier detection of passive laxity of coxofemoral joints in dogs aging 14 and 15 day-old.

2.3.4 COMPUTED TOMOGRAPHY

Fujiki *et al.* (2007) reported that numerous measures were performed on pelvic CT scans for beagles and mixed breed dogs at various time points between the ages of 2 months and 1 year to assess the relationship of the measures with joint laxity and the dorsal acetabular rim angle and center distance index were found to be good indicators of joint laxity and dysplastic changes.

Schachner and Lopez (2015) recently used the three-dimensional images which were generated from computed tomography scans predicted the development of OA changes associated with CHD.

2.3.5 MAGNETIC RESONANCE IMAGING

Ginja *et al.* (2008) opined that increased synovial fluid volumes in hip joints detected by magnetic resonance imaging in 8-week-old puppies were correlated with later HJL and CHD.

Ginja *et al.* (2009) opined that magnetic resonance imaging was used to evaluate the three-dimensional structure of human articular soft tissues, and recently, canine articular soft tissues for early prediction of canine hip laxity and dysplasia.

2.4 HAEMATO-BIOCHEMICAL PARAMETERS

Benoni *et al.* (1984) reported an increased level of serum aspartate amino transferase in human patients up to fourth postoperative day following total hip arthroplasty.

Nagaraja (1994) observed an increase in the total leukocyte count following repair of avulsed gastrocnemius tendon in dogs and attributed to excitement, surgical stress, inflammatory process and physiological exercise post-operatively.

Ranganath and Subin (2006) in their study mentioned that no significant difference was observed in the rectal temperature, heart rate and respiratory rate in dogs with and without hip dysplasia.

Ranganath and Subin (2006) in their study mentioned that no significant difference was observed in the haematological parameters like total erythrocyte count, total leucocytes count, differential leukocytes count and hemoglobin.

Singh *et al.* (2008) conducted the study on correcting angular deformities after wedge osteotomy and stabilized with circular external skeletal fixator. Haemoglobin, Total Protein, Packed Cell Volume and Calcium were recorded in their study and it did not show any significant variation from the base values and they noticed an increase in Phosphorous and Alkaline Phosphatase level in the plasma in dogs.

Smitha (2014) analyzed haematological and biochemical parameters in dogs subjected for the repair of traumatic hip luxation and observed that decreased haemoglobin , packed cell volume, and total erythrocyte count and increased value of the total leukocyte count, serum ALT and AST on 1^{st} , 3^{rd} and 5^{th} postoperative day there after receded back to normal by the 15^{th} postoperative day. Serum creatinine level increased on 7^{th} postoperative day. The variations were minimal and statistically non-significant.

2.5 TREATMENT

Anderson (2011) reported two approaches of canine HD management, which include conservative management and surgery.

Raghuvir *et al.* (2013) reported that treatment for canine hip dysplasia was recommended when both joint laxity and pain are exhibited and therapeutic treatments consisting of correcting muscle dysfunctions, relieving pain, joint mobilizations, and advisement on lifestyle modifications could be equally beneficial to the canine patient.

Raghuvir *et al.* (2013) reported that treatment in hip dysplastic dogs can be conservative or surgical. Conservative management consists of the administration of analgesics or chondroprotective agents, weight reduction, and exercise restriction and surgical treatments include pectineal myectomy, lengthening of the femoral neck, and corrective osteotomies.

Ginja *et al.* (2015) reported that there was no ideal medical or surgical treatment for hip dysplasia in dogs so prevention based on controlled breeding was the optimal approach.

2.5.1 CONSERVATIVE MANAGEMENT/ NON SURGICAL TREATMENT

Belfield (1976) found that feeding high doses of vitamin C to pregnant bitches and their offspring until 2 year of age was reported to eliminate hip dysplasia.

Palmoski and Brandt (1979) observed that analgesics, chondroprotective agents, weight reduction, and exercise restriction have been used to decrease the clinical signs of pain in hip dysplasia.

Palmoski and Brandt (1979) opined that restricted exercise was also important in decreasing trauma-induced inflammation and injury in dysplastic joints in dogs.

Olsson *et al.* (1979) found that body weight management was important in decreasing weight-bearing stresses on joints and supporting soft tissues. Obese puppies with hip dysplasia had more degenerative joint disease than did those whose diet was restricted.

Moskowitz *et al.* (1980) reported that chronic parenteral and intra-articular administration of corticosteroids causes cartilage matrix degeneration by inhibiting proteoglycan and cartilage biosynthesis by chondrocytes, thereby enhancing the rate of joint deterioration. Intraarticular administration of corticosteroids can also result in iatrogenic septic arthritis. As such, corticosteroids should be used only as a last resort in the treatment of hip dysplasia.

Palmoski *et al.* (1980) reported that most of the NSAID compounds actually accelerate cartilage degeneration by suppression of chondrocyte and proteoglycan synthesis in the osteoarthritic environment.

Burkhart and Ghosh (1987) stated that some nonsteroidal drugs, such as piroxicam, have no adverse effects on cartilage synthesis.

Burhardt and Ghosh (1987) stated that Chondroprotective agents, including polysulfated glycosaminoglycans (PSGAGs), pentosan polysulfate, and chondrotin sulfate and glucosamine, have been used to treat cartilage injuries in dogs and these agents are thought to stimulate proteoglycan and hyaluronic acid synthesis, and inhibit proteases in the synovial fluid.

Wallace *et al.*,(1990); Jones *et al.*, (1992) observed that the occurrence and severity of side effects of NAIDS was dependent on the type of medication, the dose given, and the frequency of administration and Side effects of NSAIDs were infrequent but potentially devastating. Gastritis, nephrotoxicity, decreased platelet aggregation, and gastrointestinal ulceration may occur.

Lust *et al.* (1992) opined that PSGAGs have been used prophylactically in puppies susceptible to hip dysplasia. Puppies treated 2x per wk with IM injections of PSGAGs showed less subluxation than did untreated animals.

Smith (1992) reported that with conservative treatment alone, young dogs with joint laxity and pain have a 72% probability of returning to a comfortable and functional state after 18 month of age.

Lipowitz (1993) carried a study of 68 dogs diagnosed with hip dysplasia when they were immature, showed that there were minimal clinical signs of osteoarthrosis following conservative management when the dogs were checked 2 years later.

Kealy *et al*. (2000) stated that the main conservative management recommendations were based on limiting food consumption and controlled weight-bearing activity to prevent obesity and develop muscular tissues.

Impellizeri *et al*. (2001) opined that maintenance of optimum body weight has long been considered one of the most effective methods for reducing the signs associated with OA in hip dysplasia.

Kealy *et al*. (2000); Impellizeri *et al*. (2001); smith *et al*. (2006) stated that numerous studies indicate that achieving and maintaining a healthy body weight contributes to delayed onset and reduced clinical signs of pain associated with hip dysplasia in dogs .

Mlacnik *et al*. (2006) opined that weight loss in conjunction with physiotherapy that included transcutaneous electrical nerve stimulation improved the clinical outcome for obese dogs with radiographic signs of OA associated with HD.

Pollmeier *et al.* (2006) reported that Non steroidal anti-inflammatory drugs were commonly used for pain associated with severely arthritic joints in dogs.

Smith *et al*. (2006) observed that a lifelong dietary restriction of 25% delayed the appearance of OA as well as the intensity of clinical signs in Labrador Retrievers compared with feeding ad libitum.

Farrell *et al*. (2007) reported that conservative and nonsurgical management includes weight control, reduced exercise, and use of analgesics in 74 dogs over the span of 13 years did not improve quality of life as anticipated from previous reports.

Jaeger *et al.* (2007) reported alternative method for the treatment of painful CHD joints includes acupuncture and gold bead implantation. The implantation of gold beads at acupuncture points will show clinical improvement and decreases the pain.

Kirkby and Lewis (2012) stated that conservative management of CHD generally consists of a combination of mechanisms to reduce progression of joint damage and alleviate discomfort.

Malik *et al.* (2013) isolated and characterized canine ASCs, which were then successfully used to treat HD and paraplegic patients and dogs recovered well and were able to move freely one month after treatment.

Raghuvir *et al.* (2013) reported that conservative treatment in dysplastic young dogs were directed towards alleviation of pain and most clinically dysplastic young dogs without, and even with, osteoarthrosis will respond to conservative management.

Raghuvir *et al.* (2013) opined that as conservative management does not cure hip dysplasia, the development of debilitating osteoarthrosis may still occur.

Raghuvir *et al.* (2013) found that controlled nonconcussive exercise, such as swimming was beneficial in maintaining cartilage nutrition, range of joint motion, muscle strength, and cardiovascular function.

Rialland *et al.* (2013) reported that various food supplements was used to alleviate signs of coxofemoral joint pain associated with OA in hip dysplasia range from green-lipped mussels (*Perna canaliculus*) to fish oil and Polysulfated glycosaminoglycan supplements and injections have been recommended for prevention and treatment of OA in hip dysplastic dogs.

Cuervo *et al.* (2014) reported that randomized comparison between a single intra-articular injection of adipose-derived stem cells and plasma rich in growth factors were reduced behaviour associated with pain associated with OA in hip dysplasia but that the adipose-derived stem cells appeared to be more effective for up to 6 months post-treatment based upon owner assessments.

Marx *et al.* (2014) showed that autologous stromal vascular fraction or allogeneic cultured adipose-derived stem cells can be safely used in acupoint injection for treating hip dysplasia in dogs and On 15 day and 30 day after treatment, all dogs showed improvement in range of motion, lameness at trot, and pain on manipulation of the joints and it represent an important therapeutic alternative for this type of pathology.

Panigrahi (2014) opined that conservative management has been achieved by a combination of exercise restriction, weight control, analgesics, and physical therapies.

Vilar *et al.* (2014) reported intra-articular injection of adipose-derived stem cells into affected joint has been found to be a safe therapeutic approach for the treatment of symptoms associated with OA in HD and which reduces clinical signs of hip pain and lameness based on subjective clinical evaluations and force platform gait analysis.

Schachner and Lopez (2015) stated that conservative management was often employed to manage signs of CHD, with lifelong maintenance of body mass as one of the most promising methods.

2.5.2 SURGICAL TREATMENT

Hohn and Janes (1969) reported that Triple pelvic osteotomy involves a transverse osteotomy of the ilium, together with ischial and pubic osteotomies and the acetabular segment was then rotated, according to predetermined angles of reduction and subluxation, to increase coverage of the femoral head in Hip Dysplasia.

Bowen (1972) opined that Pectineal myectomy does not improve joint stability, osteoarthritic changes still occur and pain relief is temporary.

Cardinet *et al.* (1974) observed that pectineal myectomy may actually worsen the severity of osteoarthrosis in hip dysplastic dogs.

Berzon *et al.* (1980); Off and Matis (2010) stated that femoral head and neck ostectomy (FHNO) was a surgical procedure typically indicated for hip joint problems such as chronic hip dysplasia, luxation, fractures, and avascular necrosis.

Walker and Prieur (1987) stated that corrective osteotomies used in the treatment of hip dysplasia include intertrochanteric and triple pelvic osteotomy and the goals of both procedures were to re-establish joint congruency, maintain the range of hip motion, increase the weight-bearing forces, and prevent the development of degenerative joint disease.

Braden *et al.* (1990) stated that corrective osteotomies were also recommended in young dogs less than one year of age with clinical signs of lameness, were subluxated, and do not have radiographic signs of osteoarthrosis.

Brinker *et al.* (1990) reported that the toggle pin technique for surgical management of coxofemoral luxation relies on a prosthetic replacement for the ligament of head of femur to maintain joint reduction. The ligament prosthesis was not intended to remain intact indefinitely, but rather until periarticular fibrous tissue has matured sufficiently to maintain reduction of the joint.

Wallace (1992) reported that some surgeons argue that recurrence of clinical signs following pectineal myectomy was unpredictable and that surgery may temporarily alleviate pain, most surgeons no longer recommend pectineal myectomy for treatment of hip dysplasia.

Wallace (1992) stated that Pectineal myectomy has been advocated as a palliative procedure in the treatment of hip dysplasia and release of the pectineus muscle decreases the upward force impacting the femoral head into the acetabulum, thereby releasing tension on the joint capsule, decreasing muscle tension and pain, and allowing better coverage of the femoral head within the acetabulum.

Devine and Slocum (1995) observed that lengthening of the femoral neck was used to redirect and seat the femoral head into the acetabulum in hip dysplasia and this procedure was viewed with caution by some veterinary surgeons, as complications, such as femoral fractures, may occur and long-term scientific studies of its efficacy were lacking.

Dueland *et al.* (2001); Patricelli *et al.* (2002) stated that both juvenile pubic symphysiodesis and triple pelvic osteotomy were designed to increase femoral head coverage by ventrolateral rotation of the acetabulum in the treatment of hip dysplastic dogs and the juvenile pubic symphysiodesis procedure involves premature closure of the pubic symphysis resulting in reduction in the pelvic inlet width causes ventrolateral rotation of the acetabulum during pelvic growth, and was thought to result in a 40%–46% improvement in acetabular and dorsal acetabular rim angles.

Filippo *et al.* (2001) studied on extra articular stabilization of coxo-femoral luxation using multifilament absorbable suture (polyglactin-910) in dogs and stated that suture material was strong to maintain articular stability during the period of scar tissue formation with less complication compared to non absorbable suture material.

Patricelli *et al.* (2002) reported that JPS was a surgical treatment used on puppies at 14–20 weeks of age and at risk of developing CHD with greater improvements achieved when surgery was performed at 15 weeks of age and invasive procedure based on induction of thermal necrosis in chondrocytes of the growth plate of the pubis.

Manley *et al.* (2007); Dueland *et al.* (2010) reported some of the surgical procedures designed to prevent onset and development of OA in hips joint in hip dysplasia includes double and triple pelvic osteotomy, acetabular shelf and excision arthroplasty, femoral osteotomy, and juvenile pubic symphysiodesis.

Dueland *et al.* (2010) opined that juvenile pubic symphysiodesis appears to have the best outcomes when performed in puppies that are 12–16 weeks old.

Gemmill *et al.* 2010 reported that a hybrid system of a cementless acetabular cup and a cemented femoral implant has been successfully applied in dogs recently.

Ash *et al.* (2012) reported the clinical use of the Arthrex Mini TightRope and TightRope technique for traumatic craniodorsal coxofemoral luxation and stated it as a novel application as short-term results were at least comparable to existing surgical techniques and recommended long-term follow-up studies.

Marino *et al.* (2012) reported total hip replacement was commonly used and widely accepted as a surgical procedure for painful, irreversible, developmental, or acquired conditions of the coxofemoral joint, especially in large dogs. This method associated with high success rates (92– 98%) and relatively low complication rates (7.8–20%) based on both owner assessment and clinical evaluation of pain status and functionality.

Rose *et al.* (2012) opined that Triple pelvic osteotomy was a much more extensive procedure, and involves osteotomies of the ilium, pubis, and ischium to allow manual rotation of the acetabulum for better femoral head coverage and this procedure was generally recommended for young dogs without irreversible or with mild degeneration of the coxofemoral joint.

Raghuvir *et al.* (2013) reported that surgical treatment for canine hip dysplasia includes pectineal myectomy, lengthening of the femoral neck, intertrochanteric osteotomy, and triple pelvic osteotomy and technique selection is influenced by the function and age of the animal, financial considerations, preference of the surgeon, the degree of osteoarthrosis present, and the conformation of the femur and acetabulum.

Bergh and Budsberg *et al.* (2014) reported that despite of the prevalence of CHD, a gold standard surgical procedure has yet to be identified. As such, there are numerous surgeries to prevent progression of degenerative joint changes or alleviate pain and restore joint function.

BiaBecki *et al.* (2014) reported that femoral head and neck excision was a procedure intended to significantly reduce pain associated with movement of an injured or diseased coxofemoral joint like hip dysplasia.

Fitzpatrick *et al.* (2014) reported Total hip replacement was often applied in advanced cases of joint degeneration and was considered a salvage procedure and there are no clear guidelines for the best time to implement total hip replacement, but the average time between onset of signs and surgery was 10 months.

Heo *et al.* (2015) stated that Total hip replacement (THR) was a surgical technique in which weight-bearing surfaces of the hip joint are replaced with prosthetic implants and THR can improve quality of life by relieving pain and improving joint motion.

Ginja *et al.* (2015) reported that femoral head and neck excision reduces the pain produced by abnormal bone to bone hip joint contact, but it does not effectively maintain the full range of hip motion and limb function.

Kimeli *et al.* (2015) reported surgery aims to prevent/limit the development of HD or reduce/eliminate pain through salvage and surgical techniques that have been used with success include juvenile pubic symphysiodesis, pelvic osteotomy, pelvic ostectomy, denervation of hip joint capsule, shelf arthroplasty, intertrochanteric femoral osteotomy, excision arthroplasty, and total hip replacement.

Schachner and Lopez (2015) stated that surgical intervention to treat hip dysplasia was often employed to prevent joint changes or restore joint function, but there are no gold standards for either goal.

2.6 POST OPERATIVE CARE AND MANAGEMENT

Denny and Butterworth (2000) reviewed on the outcome of femoral head and neck excision and opined that many authors recommend passive flexion and extension exercises in the immediate postoperative period to maximize the range of motion in the developing false joint.

Martini *et al.* (2001) reported use of carprofen @ 4 mg/kg orally to manage post operative pain once daily for 5 days after surgery in dogs with coxofemoral luxation which were stabilized by extra-articular sutures.

Ozaydin *et al.* (2003) reported not to bandage the hip for two reasons: (1) A bandage such as an Ehmer sling may increase risk of separation of the ligament from the bone because of tension associated with limb flexion, and (2) the inherent strength of the ligamentum sacrotuberale and the reliability of the interference screw, and capsulorrhaphy should mitigate the need for extra support in dogs treated for hip luxation by transposition of ligamentum sacrotuberale

Greg (2004) found that active use of the limb was the most beneficial form of rehabilitation in minimizing muscle atrophy and promoting the formation of the pseudoarthrosis. Slow walking in the early postoperative period can be encouraged with the help of nonsteroidal antiinflammatory medications, if necessary.

Cetinkaya and Olcay (2010) reported use of amoxicillin-clavulanic acid medication post-operatively. Immediately after surgery meloxicam was administered to the cat as a single subcutaneous injection (0.2 mg/kg of body weight) to alleviate postoperative pain. Carprofen was administered to the dog for one week (2.2 mg/kg of body weight twice a day) for control of post operative pain in dogs and cats subjected for Modified Knowles toggle pin technique with nylon monofilament suture material for treatment of two caudoventral hip luxation.

Ash *et al.* (2012) reported radiographic follow up performed six weeks postoperatively included medio-lateral and ventrodorsal projections and found confirmed consistant and unchanged quality of hip reduction, toggle position and absence of periarticular osteophytosis or coxofemoral remodeling.

Aarnes *et al.* (2014) sedation with acepromazine and morphine, administration of an epidural containing morphine and eopivacaine, and intraoperative sedation with medetomidine,morphine,lidocaine and ketamine were suitable for femoral head and neck ostectomy , postoperatively, carprofen was administered once subcutaneously for 7 days

2.7 POST OPERATIVE COMPLICATIONS

Beale *et al.* (1991) reported that reluxation following surgical reduction of coxofemoral luxation has been reported in dogs with hip dysplasia. Although it has been reported that the presence of hip dysplasia does not increase the rate of recurrence following reduction, dogs with hip dysplasia and DJD are thought to do poorly with open or closed hip reduction.

Devine and Slocum, (1995) observed that most common complication associated with Lengthening of the femoral neck procedure in hip dysplasia was femoral head and neck fractures.

Hosgood and Lewis (1993) reported that complications of triple pelvic osteotomy include implant failure (13% to 36%), incisional problems (14%), acetabular fracture (12%), and transient sciatic neuropraxia (3%).

Sukhiani *et al.* (1994) stated that Pelvic canal narrowing considered as a significant complication associated with triple pelvic osteotomy.

Rawson *et al.* (2005) reported that common complications associated with femoral head and neck excision were noted by owners and on clinical examination, include pain caused by the femoral neck mechanically irritating the acetabulum , lameness associated with limb shortening, patellar luxation, sciatic neurapraxia, and also severe muscle atrophy which results in limited hip motion range.

Ireifej *et al.* (2012) Reported complications associated with Total hip replacement in dogs include luxation/dislocation, septic loosening, aseptic loosening of both cemented and cementless components, improper implant positioning, periprosthetic femoral fractures, sciatic neurapraxia, patella luxation, extraosseous cement granuloma formation, and femoralmedullary infarction

BiaBecki *et al.* (2014) reported complications after Triple pelvic osteotomy were related to pelvic anatomy, excessive stress placed on the implants, or both and Frequent deleterious effects were femoral dysplasia, obstipation, dysuria, and neurological abnormalities. Poor surgical technique may lead to complications such as iatrogenic injury to the sciatic nerve, progression of degenerative joint disease, and infection.

2.8 SURGICAL ANATOMY

In the coxofemoral joint, the hemispheric convex caput femoris articulates with the concave facies lunata of the acetabulum. The caput femoris (femur head) of the dog was clearly shouldered by the collum ossis femoris from the rest of the femur. The acetabulum formed by the parts of os ilium, os ischii and os pubis. The corpus ossis ilii forms the craniolateral part and the corpus ossis ischii forms the caudolateral part of the acetabulum. Most of the dogs additionally have an os acetabuli in the center of the other bones. In the course of the ossification of the epiphyseal cartilage these bones aggregate to the hemispheric concave acetabulum. In the center of the acetabulum was the fossa acetabuli which was the attachment site of the ligamentum capitis ossis femoris. The ligamentum ossis femoris ends in the fovea capitis femoris. The rim of the acetabulum is interrupted by the incisura acetabuli, which is closed by the ligamentum transversum acetabuli. The rim of the acetabulum was supplemented by the labrum acetabulare, making the hip joint become a cotyloid joint. The agitation of the hip joint is restricted by the surrounding muscles. The capsula articularis reaches from the rim of the acetabulum to the rim of the articular surface of the femur.

Initially, the hip joint was a cartilage template that grows and models with development. Ossification of the bones forming the hip joint has only started at birth and proceeds until about four to seven months of age. Formation of a normal hip joint depends upon the femoral head being held firmly within the acetabulum, because the femoral head determines the development of the acetabulum.

Lust *et al.* (1980) observed that the joint capsule completely encloses the hip joint and plays an important role in maintaining the femoral head in acetabular socket. The capsule and the joint fluid present in it produce a hydrostatic stability factor that prevents the lateral translation of the femoral head. The mean joint fluid volume is 0.80 ml in dogs with normal hip conformation

2.8.1 Ligaments of hip joint

Wadsworth (1993) stated that at some stage, before the pelvis hits the ground, the knee makes contact with the ground and as the pelvis moves ventrally the hip begins to rotate externally. If the force is of sufficient magnitude the teres ligament and the joint capsule rupture and the tension in the gluteal muscles results in luxation of the femoral head in a craniodorsal direction

a) Round ligament – The fovea was a point of attachment for the ligament of the head of the femur also known as the round ligament or teres ligament. The ligament that attaches the head of the femur to the acetabulum. It was non weight bearing ligament covered by a synovial membrane. In hip the heavy muscles that transverse the joints are more responsible for holding the femur in place than the ligament. A rupture of this ligament may lead to a common injury known as Coxofemoral luxation.

b) Capsular ligament – attached to the rim of cotyloid cavity and surrounds the fibrocartilage above and below to the rim around head of the femur. Both medial surface and lateral surface are lined by synovial membrane of the joint. Its lateral surface comes in contact with rectus femoris, in front of gamelli, obturater internus behind, obturater externus, medially tendon of illacus and psoas major.

2.8.2 Muscles of hip joint

Smith *et al.* (1990) stated that primary stability of the joint comes from the joint capsule, which extends from the acetabulum to the neck of the femur. Secondary support for the hip joint comes from the ligament of the head of the femur, which runs from the fovea capitis of the femur to the acetabular fossa and also numerous surrounding muscles contributing support and their function as flexors, extensors, abductors, adductors and internal rotators of the limbs.

The muscles of the hip are grouped according to function. The flexors of the hip include the iliopsoas, the tensor fascia lata, the articularis coxae, the rectus femoris and sortorius muscles. The extensors of the hip provide the largest muscle in the hip and include the gluteals, quadratus femoris, piriformis, biceps femoris, semitendinosus, semimembranosus, gracilis and adductor muscles. The external rotators include the internal obturater, external obturater, gemelli, quadratus femoris and iliopsoas. The internal rotators and abductors of the femur include the gluteal muscle and tensor fascia lata. The adductors of the femur include the adductor longus, adductor magnus bravis, pectineus and gracilis muscles.

2.8.3 Vascular supply of hip joint

Kaderly *et al.* (1982) stated that the blood supply to the femoral head and neck arises from the lateral circumflex femoral artery, the medial circumflex femoral artery, and to a limited extent from a branch of the caudal gluteal artery.

2.8.4 Nerve supply to hip joint

Evans and Christensen (1979) described that lumbosacral trunk was the most important part of the lumbosacral plexus (L_4-S_2) and it was continued outside the pelvis as sciatic nerve. The lumbosacral trunk primarily from the sixth and seventh lumbar nerves with a small contribution from the first cord and occasionally the second sacral nerves. The lumbosarcral trunk has two medium sized branches, cranial and caudal gluteal nerves. It became the sciatic nerve after the last sacral branches enters it at the greater ischiatic foramen.

The sciatic nerve was the largest nerve in the body. It is a continuation of the lumbosacral trunk, and the extra pelvic part of the trunk was regarded as the sciatic nerve. It consists of two nerves, the tibial and peroneal nerve. The sciatic nerve passes caudally over the hip medial to the greater trochanter and then distally, caudal to the femur on the lateral side of adductor muscle. A branch leaves the nerves at the level of hip and innervates the biceps femoris, semitendinosus and semimembranosus muscle. The sciatic nerve terminates in the thigh as the common peroneal and tibial nerves.

2.9 DYSPLASTIC HIP JOINT

Lust and Summers (1981) and Shepherd (1986) reported that pups genetically predisposed to hip dysplasia were normal at birth. Stretching of the joint capsule and ligament of the femoral head was observed as early as two weeks of age. Mild proliferative, nonsuppurative synovitis, edema, and fibroplasia of the ligament of the femoral head, as well as joint effusion, were present at 4^{th} week. By 12^{th} week, affected individuals had changes in both the synovium and the articular cartilage grossly with flaking and fissuring of the surface cartilage and microscopically, surface chondrocytes were lost and changes in the matrix's proteoglycan content and collagen fibril network had occurred.

Lust *et al.* (1985) reported that cartilage degeneration, joint capsule thickening, stretching or rupture of the ligament of the femoral head, proliferation of the dorsal acetabular rim and thickening of the femoral neck and atrophy of local muscle was characterized in advanced hip dysplasia. Further he stated that at this point, joint stability might improve or progress to complete luxation. The rate and degree of disease progression varied with the individual and the amount of joint instability present.

Alexander (1992) reported that hip dysplasia was a biomechanical disease where hip instability in the young dog altered the concentration of forces on the growing femoral head and acetabulum. This affected the bone growth and remodeling, resulting in abnormal joint conformation and secondary degenerative joint disease. Abnormal weight bearing forces caused microfractures in the subchondral bone of the dorsal acetabular rim and femoral head. With healing, the bone became harder and less able to absorb shock.

More force was transmitted to the overlying cartilage, increasing its degeneration at these sites. Cartilage on the medial aspect of the femoral head and dorsal acetabular rim was gradually worn away, exposing the subchondral bone. The subchondral bone became sclerotic and eburnated. Sharpey's fibers were torn, causing osteophytes to form along the joint capsule's attachment to the acetabulum and femoral neck.

Kealy *et al.* (1993) observed that dysplastic dogs had higher synovial fluid osmolalities than did normal dogs due to differences in synovial fluid electrolyte concentrations of sodium, potassium and chloride.

Smith (1998) stated that synovial fluid volume has been implicated in the pathogenesis of hip dysplasia through its effect on joint laxity. When normal synovial fluid volumes were present, displacement of the femur created negative intra articular pressure that tended to pull the femoral head back into the acetabulum. This mechanism was lost when joint effusion was present.

Materials and Methods

III. MATERIALS AND METHODS

The occurrence of subluxation or luxation among the dogs with hip dysplasia presented to Veterinary College Hospital was recorded for a period of twelve months (May 2015 to April 2016). The study was carried out among six dogs presented to the Department of Surgery and Radiology, Veterinary College Hospital, Hebbal, Bangalore with gait and running abnormalities of hind limb, difficulty in rising and reluctance to climb stairs, joint instability, pain and slight to moderate lameness in hind limbs and those which have been diagnosed with canine hip dysplasia. All the dogs were subjected for radiography, physiological, haemato-biochemical tests to assess their fitness for the surgery. Only dogs found fit with normal body parameters were chosen for "Percutaneous transfemero-acetabular nylon zap strap application" for the repair of subluxation/luxation in Hip dysplastic dogs.

3.1 Occurrence

The occurrence of subluxation/ luxation among the dogs with hip dysplasia with regard to age, breed and sex of dogs were recorded in percentage for a period of one year.

3.2 Case history

The cases in which hip dysplasia was suspected, details regarding anamnesis like age, breed, body weight, signalment and owner observations were recorded.

3.3 Pre surgical evaluation.

3.3.1 Clinical examination

Physiological parameters like rectal temperature (^{0}F), respiratory rate (per minute) and heart rate (per minute) were recorded prior to surgery.

Clinical weight bearing pattern of limb will be observed and recorded before surgery.

3.3.2 Radiographical examination

The suspected animals were subjected to radiography of affected hip by ventro-dorsal view to identify subluxation/ luxation of hip dysplastic dogs under general anaesthesia.

3.3.3 Blood examination.

3.3.3.1 Haematological studies

All the dogs were subjected for haematological evaluation *viz.*, Total erythrocyte count (millions/cmm), Hemoglobin (g %), Total Leucocyte count (thousands/cmm), Differential leucocyte count (%) prior to surgery and post operative values were recorded.

3.3.3.2 Biochemical evaluation

All the dogs were subjected for biochemical estimation *viz.*, Serum creatinine (mg/dl), Alanine amino transferase (ALT) (IU/L) and Aspartate amino transferase (AST) (IU/L) prior to surgery and post operative values were recorded.

3.4 Surgical treatment

3.4.1 Pre-operative preparations

3.4.1.1 Preparation of surgical instruments and implants

In addition to the general surgical instrument set (Plate-1) and orthopedic set (Plate-2), K-wires and nylon zap strap of different sizes were taken and sterilized by autoclaving at 121^0C under 15lb pressure for 21minutes prior to surgery.

3.4.1.2 Preparation of materials

For stabilization of coxo-femoral subluxation/ luxation in dysplastic dogs, the nylon zap strap was used, which was sterilized by autoclaving at 121^0C under 15 lb pressures for 21 minutes prior to surgery.

3.4.1.3 Patient preparation

The surgical site was prepared for surgery. Surgical site for standard cranio-dorsal approach to hip joint was made to the severely affected hip. All the selected dogs were fasted for twelve hours and water was withheld for six hours prior to surgery. The area around coxofemoral joint was shaved. The site of surgery was prepared aseptically (Plate-3) by washing with soap initially, followed by scrubbing with Chlorhexidine solution, then swabbing with surgical spirit and then finally painting with Povidone Iodine.

Plate 1 : Photograph showing standard surgical set

Plate 2: Photograph showing orthopedic surgical set

3.4.1.4 Premedication and Anaesthesia

The surgery was performed under general anaesthesia. The dogs were pre-medicated with

- Inj. Atropine sulphate (Atropine sulphate Injection IP-. Superb Drugs Private Ltd., Kolkata) given at the rate of 0.04 mg / kg subcutaneously.
- Inj. Diazepam (Calmpose®, Ranbaxy Laboratories, Panota Sahib, H.P) at the rate of 1 mg / kg intravenously.
- Inj. Ceftriaxone (Intacef®, Intas pharmaceutical Ltd, Allahabad) at 20 mg /kg intravenously.
- Inj. Pentazocine (Fortwin®, Ranbaxy Laboratories, Ahmedabad) at the rate of 1mg/kg intramuscularly was given prior to surgery.

Ringer's Lactate (RL®, Claris Life science) was infused at the rate of 10ml/Kg/hour throughout the surgical procedure.

Subsequently they were anaesthetized with Thiopentone sodium (Thiosol®, Neon Laboratories, Mumbai) calculated at the rate of 12.5 mg / kg IV and given 'to effect' and maintained with 2% Isoflurane (Sosrane®, Metrix Pharmaceutical, Mumbai) inhalant anaesthesia.

3.4.1.5 Animal positioning

For surgery, dogs were positioned on lateral recumbency on the operation table with affected limb upwards. Dogs were draped with sterile drapes covering the dog completely except the surgical site and the head (Plate-4).

Plate 3 : Photograph showing aseptically prepared Surgical site

Plate 4: Photograph showing Patient positioned in the lateral recumbency with the affected pelvic limb upwards

3.4.1.6 Surgical technique

A cranio-dorsal approach was made to hip joint, a curvy linear incision (plate-5) was made separating skin and subcutaneous through blunt dissection of middle and deep gluteal muscle, hip joint was palpated with index finger. Coxo-femoral joint was reduced if luxated .Greater trochanter of femur was located and a temporary marking was done. A cannulated bit designed to guide the nylon zap strap of appropriate size was inserted at the marked point and drilled cranio-dorsally passing from femoral head to acetabulum in and through it (Plate-6). Bit was confirmed in position by digital palpation through the surgical approach made. Conformational radiograph of cannulated bit in appropriate position was done under conventional fluoroscopy. After confirmation of bit in position, (plate-7) nylon zap strap of appropriate size was inserted through cannula of the bit guiding it through the acetabular fossa and out of it (Plate-8). The nylon zap strap was grasped through the cranio-dorsal approach to hip and brought back through the subcutaneous tissue near the incision (Plate- 9 &10) made to pass the cannulated bit. Bit was removed manually and the nylon zap strap was applied (Plate-11). Excess of nylon zap strap was cut. A thick cotton gauze pad was provided under the knot.

Approach made for hip was closed surgically. Middle and Deep gluteal muscles were opposed using Chromic catgut (0) (Trugut®, Suture India., Bangalore) by simple interrupted suture pattern. Skin was closed using monofilament polyamide suture (0) (Trulon® Suture India., Bangalore) by horizontal mattress suture pattern. Wound dressing was done. No external immobilization was done postoperatively (Plate-12).

Plate 5: Photograph showing Curvilinear incision at the surgical site

Plate 6: Photograph showing positioning of cannulated bit into the acetabular fossa

Plate 7: Photograph showing correct positioning of cannulated bit to guide nylon zap strap

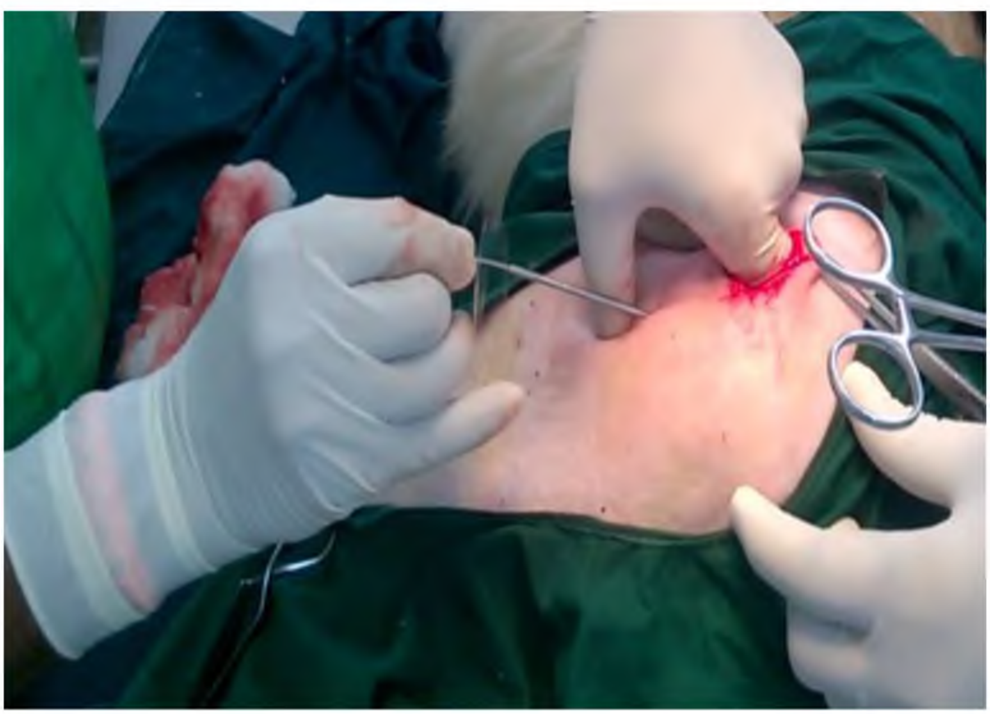

Plate 8: Photograph showing introduction of nylon zap strap into the cannulated bit

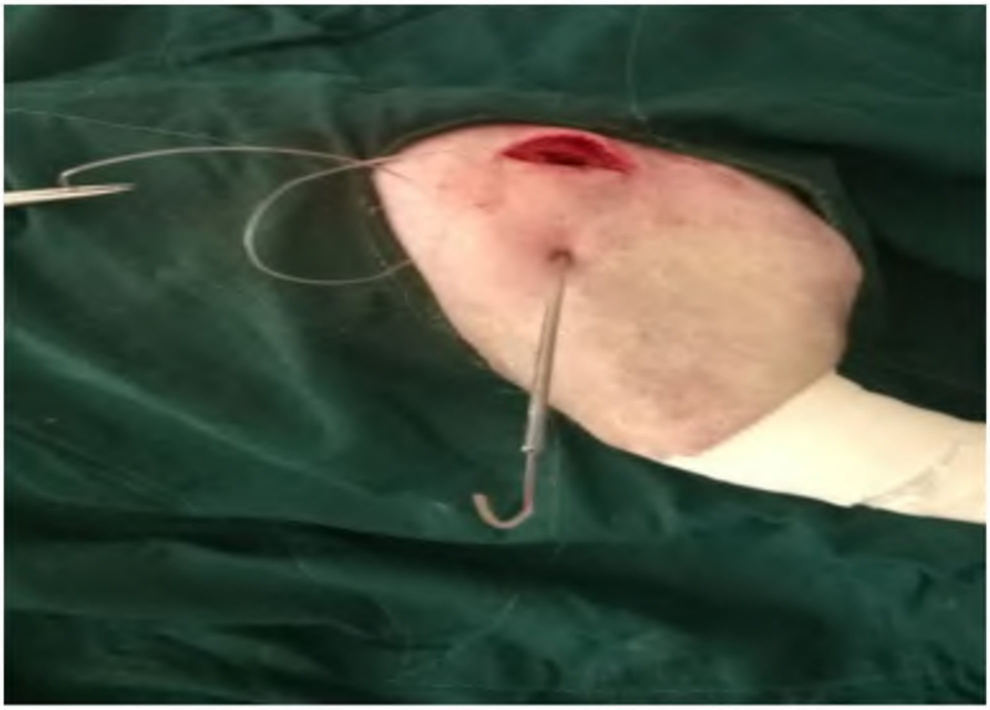

Plate 9: Photograph showing after introduction of nylon zap strap into the bit

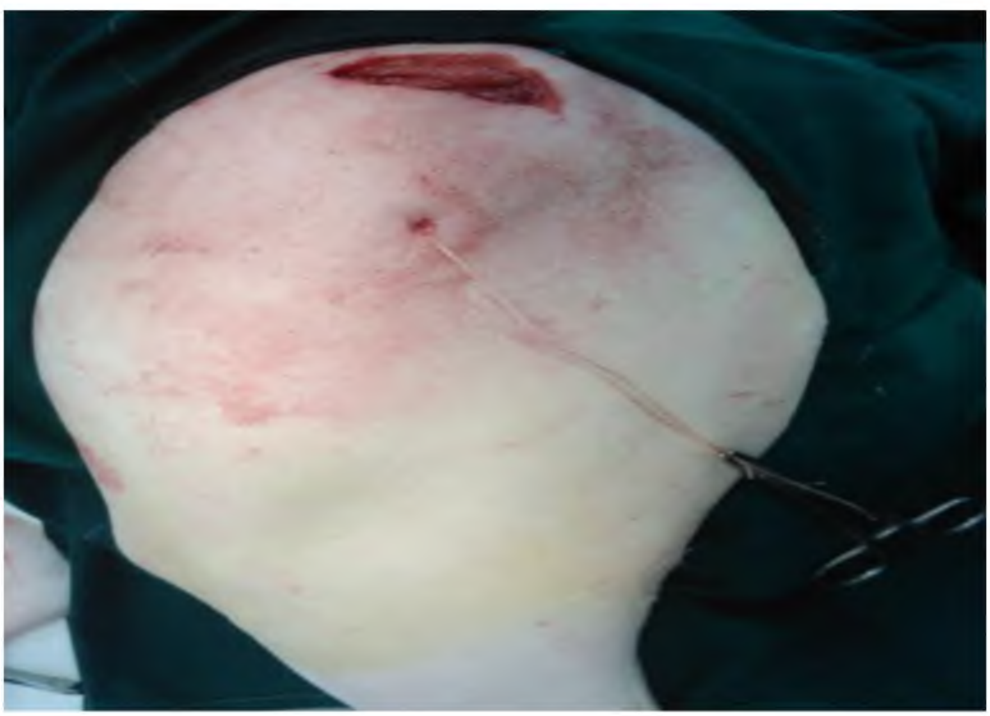

Plate 10: Photograph showing both the ends of the nylon zap strap on the skin

Plate 11: After Application of nylon zap strap and closing skin by horizontal mattress suture pattern

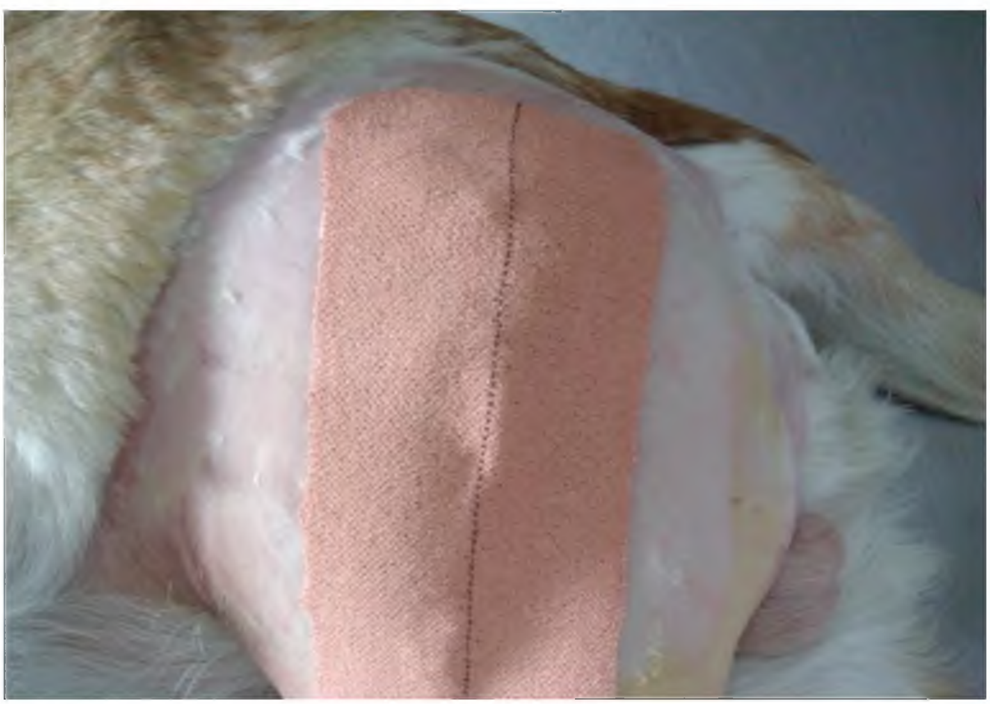

Plate 12: Photograph showing operated limb without any external immobilization Post-operatively

3.4.1.7 Postoperative care and management

Postoperatively antibiotic Ceftriaxone (Intacef® Inj 500 mg vial, Intas Pharmaceticals Ltd, Ahamedabad) @ 20 mg/kg body weight was administered. Tramadol (Supridol®, 50mg/ml ampoule, Neon laboratories limited, Mumbai) was administered at 2mg/kg body weight, post-operatively for 3 days as an analgesic.

Alternative day surgical wound dressing was done using antiseptics and elastic adhesive bandage. No external immobilization was applied to stabilise the operated hip joint.

3.5 Post-surgical evaluation

Physiological parameters *viz.*, temperature, respiration and heart rate were recorded before, immediately after surgery and on 3rd, 5th, 7th, 15th, 30th, 45th and 60th post operative days.

Radiographs of ventro -dorsal hip was taken to assess the position of femoral head in the acetabulum, before, immediately after surgery and on 7th, 15th, 30th, 45th and 60th post operative days.

For haematological evaluation, two ml of blood was collected in vials containing EDTA and subjected to haematological parameters estimation *viz.*, Total erythrocyte count (millions/cmm), Hemoglobin (g %), Total Leucocyte count (thousands/cmm), Differential leucocyte count (%) were estimated before, immediately after surgery and on 0, 3rd, 5th ,7th, 15th, 30th, 45th and 60th day.

Two ml of blood sample was collected in a vial and allowed to clot at room temperature for separation of serum. Biochemical parameters *viz.,* Serum creatinine (mg/dl), Alanine amino transferase (ALT) (IU/L) and Aspartate amino transferase (AST) (IU/L) were estimated before and on 0, 3rd, 5th, 7th, 15th, 30th, 45th and 60th day were estimated using auto analyzer.

Clinical parameters *viz.,* evaluation of weight bearing pattern was done, before, immediately after surgery and on 0, 3rd, 5th, 7th, 15th, 30th, 45th and 60thpost operative days.

Nylon zap strap was left in place varying from 55-60 days post-operatively in all the cases and removed later on.

3.6 Statistical analysis

All the results of clinical, haematological and biochemical parameters were statistically analyzed using one way Analysis of variance with more than one observation per cell and means for the respective periods were tested by Dunnets post hoc test as described by Snedecor and Cochran (1996).

Results

IV. RESULTS

The present study was undertaken on six clinical cases of dogs with coxo-femoral luxation/subluxation suffering from severe hip dysplasia, irrespective of age, breed and sex, presented to the Department of Veterinary Surgery and Radiology, Veterinary College, Hebbal, Bangalore, between May 2015 to April 2016 and the results of the study are presented under the following headings.

4.1 Occurrence of canine hip dysplasia with coxo-femoral luxation/subluxation

The total number of cases presented to the Veterinary College Hospital, Hebbal Bangalore was 19854 during the period from May 2015 to April 2016. Out of these, 15725 were canines, among these dogs that were presented with hip joint laxity and confirmed hip dysplastic under standard ventrodorsal radiography of hip were 81.

Table 1: Total number of cases presented to Department of TVCC from May 2015 to April 2016

Total number of cases presented	**19854**
Total number of dogs presented	**15725(79.20%)**
Total number of dogs with hip dysplasia	**81(0.51%)**

Table 2: Total number of subluxation and luxation in hip dysplastic dogs

Subluxation of hip	**59(0.37%)**
Luxation of hip	**22(0.13%)**

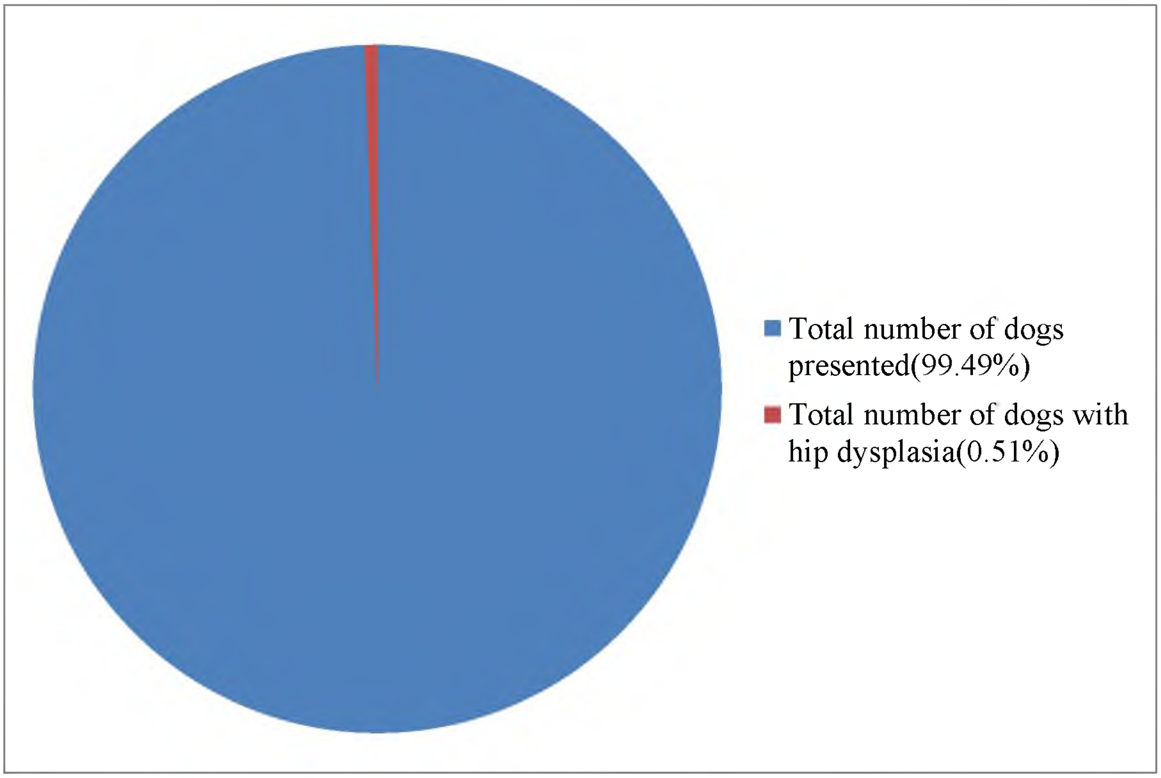

Fig. 1: Pie diagram showing occurrence of Hip Dysplasia in dogs (May 2015 to April 2016)

4.2 Breed, Age and Gender wise occurrence of hip dysplasia with coxo-femoral luxation/subluxation

4.2.1 Breed-wise occurrence of hip dysplasia in dogs

Breed-wise occurrence of hip dysplasia in dogs were – Labrador retriever 18 (22.22%), German shepherd dog 12 (14.81%), Saint Bernard 12 (14.81%), Rottweiler's 11 (13.58%), Golden Retriever 10 (12.34%), Pugs 9 (11.11%), Great Dane 6 (7.40%), Non-Descriptive breed 2 (2.46%) and Doberman 1 (1.23%).

Occurrence of subluxation of hip in dogs were – Labrador retriever, Saint Bernard, German shepherd , Rottweiler's, Golden Retriever, Pugs, Great Dane, Non-Descriptive breed and Doberman breeds were 20.33% (12), 16.94% (10), 15.25% (9), 11.86% (7), 11.86% (7), 11.86% (7), 8.47% (5), 1.69% (1), 1.69% (1) respectively (table 3 and fig 2).

Occurrence of luxation of hip in dogs were – Labrador retriever, Rottweiler's, German shepherd, Golden Retriever, Saint Bernard, Pugs, Great Dane and Non-Descriptive breeds were 27.27% (6), 18.18% (4), 13.63% (3), 13.63% (3), 9.09% (2), 9.09% (2), 4,54% (1), 4.54% (1) respectively (table 3 and fig 2).

4.2.2 Age - wise occurrence of hip dysplasia in dogs

Age-wise occurrence of hip dysplasia in dogs less than one year old, one to two years, two to three years, three to four years, four to five years , five to six years and more than six years were 60.49% (49), 8.64% (7), 8.64% (7), 3.70% (3), 6.17% (5), 3.70% (3),8.64% (7) respectively (table 4 and fig 3).

Table 3: Breed-wise occurrence of hip dysplasia in dogs

Breeds	Hip dysplasia	Subluxation of hip	Luxation of hip
Labrador retriever	22.22%(18)	20.33%(12)	27.27%(6)
German shepherd	14.81%(12)	15.25%(9)	13.63%(3)
Saint Bernard	14.81%(12)	16.94%(10)	9.09%(2)
Rottweiler's	13.58%(11)	11.86%(7)	18.18%(4)
Golden Retriever	12.34%(10)	11.86%(7)	13.63%(3)
Pugs	11.11%(9)	11.86%(7)	9.09%(2)
Great Dane	7.40%(6)	8.47%(5)	4,54%(1)
Non-Descriptive breed	2.46%(2)	1.69%(1)	4,54%(1)
Doberman	1.23%(1)	1.69%(1)	-

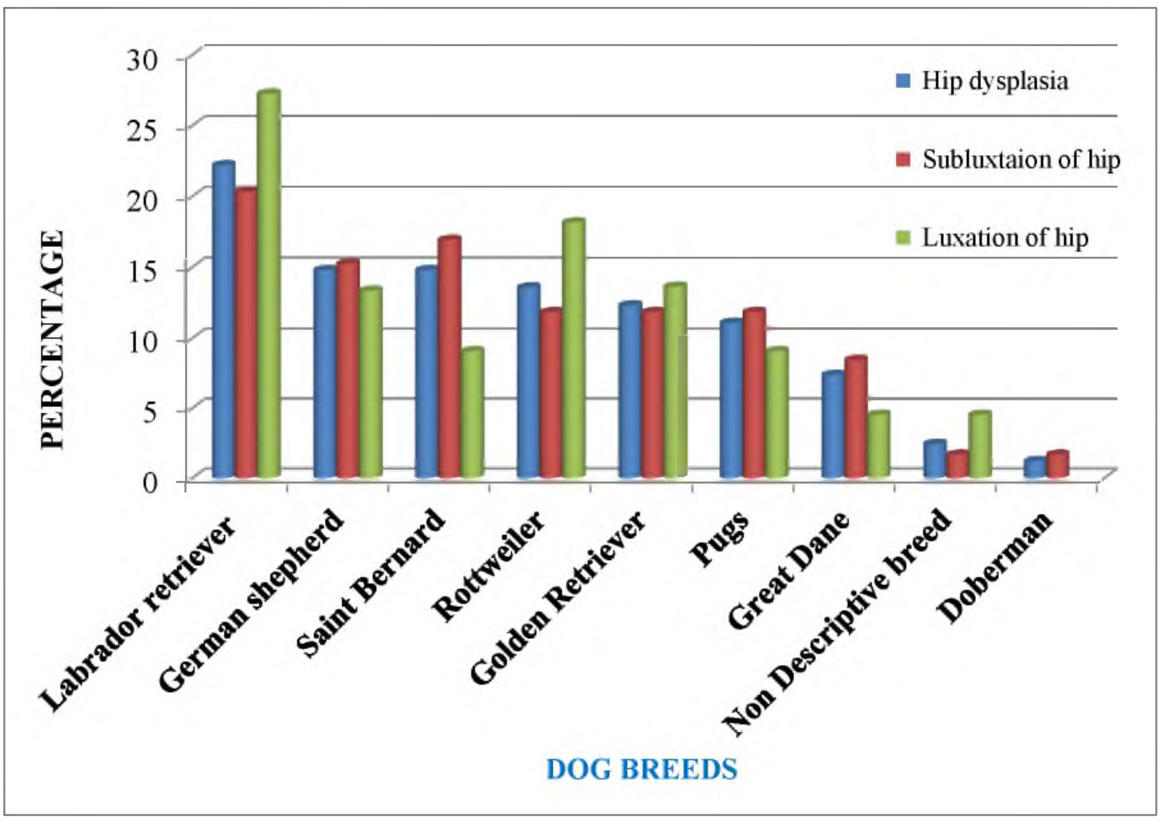

Fig. 2: Graph showing breed wise occurrence of Hip Dysplasia in dogs

Occurrence of subluxation of hip in dogs less than one year old, one to two years, two to three years, three to four years, four to five years , five to six years and more than six years were 64.40% (38), 8.47% (5), 6.77% (4), 5.08% (3), 6.77% (4), 1.64% (1), 6.77% (4) respectively (table 4 and fig 3).

Occurrence of luxation of hip in dogs less than one year old, one to two years, two to three years, three to four years, four to five years , five to six years and more than six years were 50%% (11), 9.09%% (2), 13.63% (3), 0% (0), 4.54% (1), 9.09% (2), 13.63% (3) respectively (table 4 and fig 3).

4.2.3 Gender - wise occurrence of hip dysplasia in dogs

Out of the 81 dogs represented with hip dysplasia 57 (70.37%) dogs were males and 24 (29.62%) were females. (Table 5 and fig 4).

Occurrence of subluxation of hip in male and female dysplastic dogs were 72.88 % (43) and 27.11 % (16) respectively (table 5 and fig 5).

Occurrence of luxation of hip in male and female dysplastic dogs were 63.63 % (14) and 36.36% (8) respectively (table 5 and fig 5).

4.3 Premedication and anaesthesia

Premedication was done with Atropine sulphate at the dose rate of 0.04 mg per kg body weight sub-cutaneously and Diazepam at the dose rate of 1 mg per kg body weight administered intravenously. After ten minute general anaesthesia was induced with 2.5% Thiopentone sodium intravenously to effect and maintained with Isoflurane, which

Table 4: Age - wise occurrence of hip dysplasia in dogs

Age	Hip dysplasia	Subluxation of hip	Luxation of hip
Less than one year old	60.49%(49)	64.40%(38)	50%%(11)
One to Two years	8.64%(7)	8.47%(5)	9.09%%(2)
Two to Three years	8.64%(7)	6.77%(4)	13.63%(3)
Three to Four years	3.70%(3)	5.08%(3)	-
Four to Five years	6.17%(5)	6.77%(4)	4.54%(1)
Five to six years	3.70%(3)	1.64%(1)	9.09%(2)
More than Six years	8.64%(7)	6.77%(4)	13.63%(3)

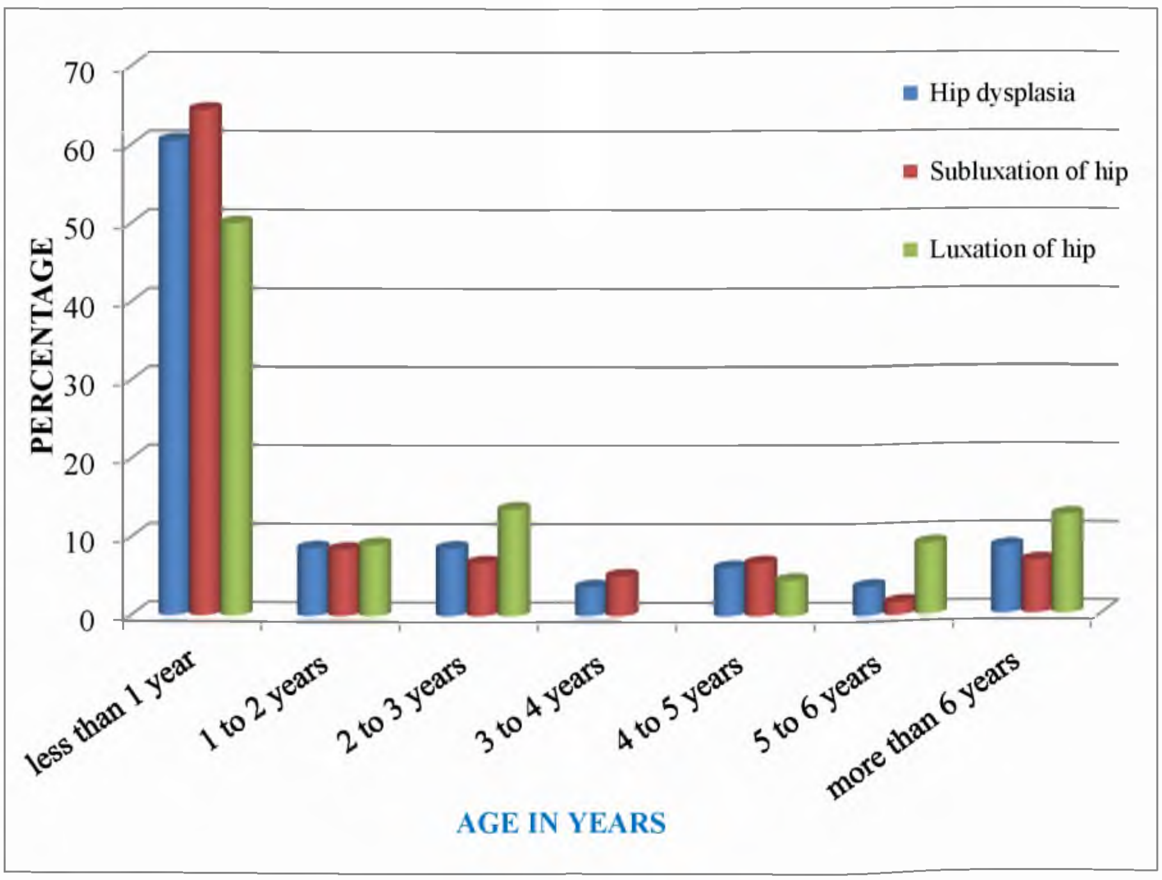

Fig. 3: Graph showing age wise occurrence of Hip Dysplasia in dogs

Table 5: Gender - wise occurrence of hip dysplasia in dogs

Gender	Hip dysplasia	Subluxation of hip	Luxation of hip
Male	57(70.37%)	43(72.88%)	14(63.63%)
Female	24 (29.62%)	16(27.11%)	8(36.36%)

Table 6: Details of dysplastic dogs with coxofemoral luxation/subluxation selected for the study

CASE No.	BREED	AGE	SEX	BODY WEIGHT (Kg)	Radiological interpretation	LIMB AFFECTED
1	Golden Retriever	6 months	Female	19	Bilateral canine hip dysplasia	Bilateral severe hip dysplasia
2	Labrador Retriever	9 months	Male	28	unilateral canine hip dysplasia	Right hind leg subluxation
3	Pug	4 months	Female	5.2	Bilateral canine hip dysplasia	Left hind leg subluxation
4	Golden Retriever	4.5 months	Female	10	Moderate bilateral canine hip dysplasia	Right hind leg luxation
5	Golden Retriever	9 months	Female	23	Bilateral canine hip dysplasia	Right hind leg luxation
6	Labrador Retriever	9 months	Male	34.5	Bilateral canine hip dysplasia	Subluxation of Right hind leg and luxation of the left hind leg

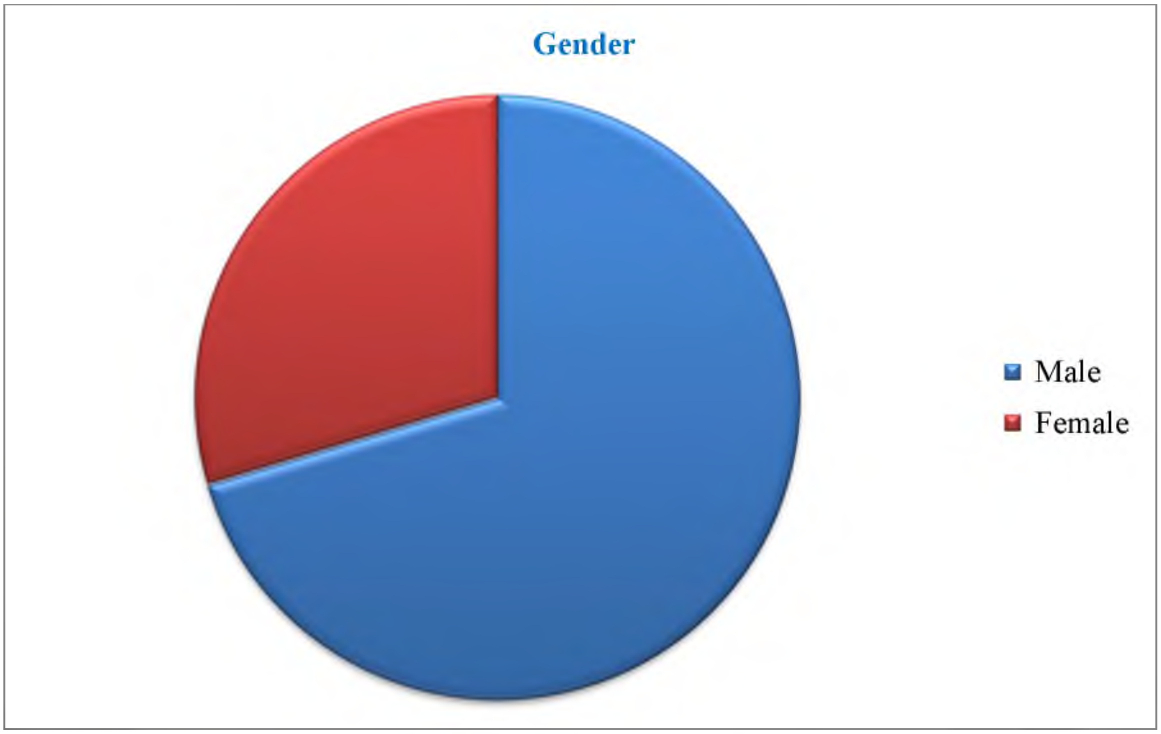

Fig. 4: Pie diagram showing Gender wise occurrence of Hip Dysplasia in dogs

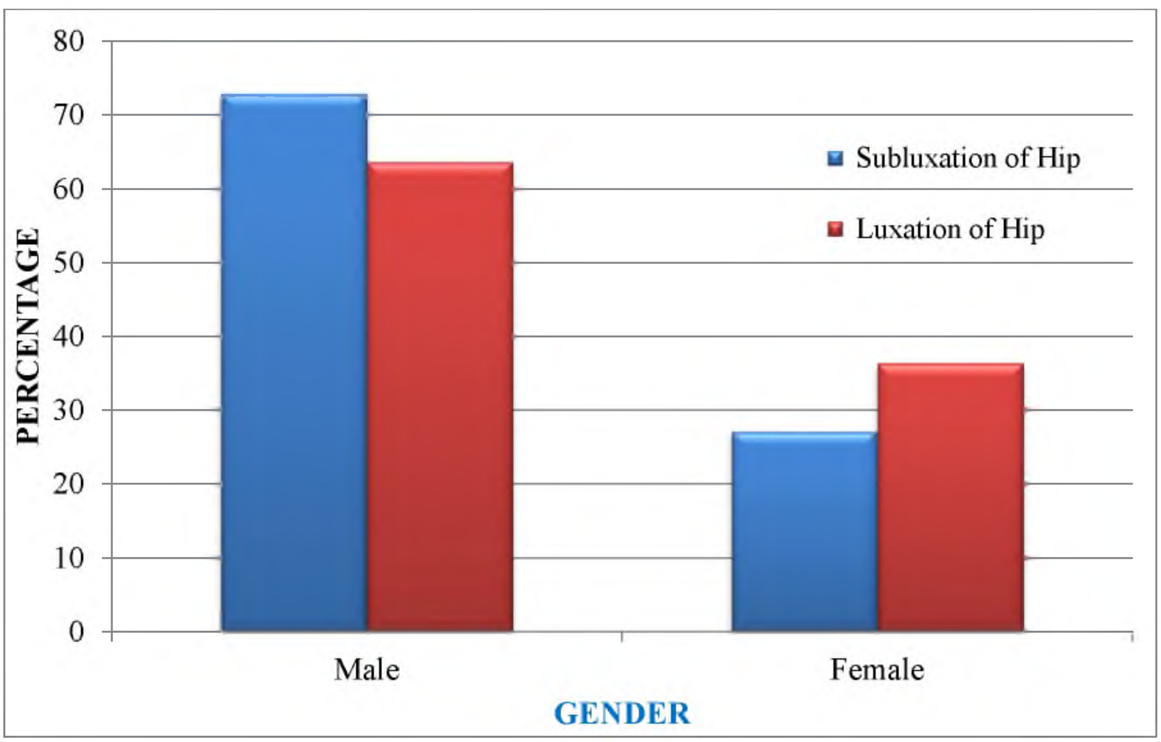

Fig. 5: Graph showing Gender - wise occurrence of subluxation and luxation in hip dysplastic dogs

provided adequate surgical plane of anesthesia for successful completion of surgery without any complication in all the cases.

Administration of Atropine sulphate reduced the volume of salivation. Preanaesthesia with diazepam provided good sedative effects along with muscle relaxation. The induction of anaesthesia was smooth and uneventful in all the cases. No intra-operative anaesthetic complications were noticed in any of the cases. All the dogs had a smooth recovery from the anaesthesia.

4.4 Antimicrobial prophylaxis and analgesics

In the present study, for all the animals, preoperatively Ceftriaxone at the dose rate of 20 mg per kg body weight was administered intravenously. Prophylaxis with Ceftriaxone, a broad spectrum antibiotic, provided satisfactory antimicrobial coverage postoperatively.

Pentazocine is a non-opiod analgesic at 1mg/kg body weight intramuscularly, post-operatively was satisfactory in providing sufficient analgesic effect without any side-effects in all the six operated dysplastic dogs for effective pain management.

4.5 Clinical evaluation

4.5.1 Weight bearing

Weight bearing was assessed and graded either as Poor, Fair, Good or Excellent. All the dogs were graded as good on the first postoperative day and animals showed excellent weight bearing on 30th post operative day onwards.(Photo 13-20) (Table 7).

Table 7: Weight bearing pattern in dogs subjected to application of the nylon zap strap for the repair of coxofemoral subluxation/luxation in hip dysplastic dogs.

Animal No.	Days						
	1st	**3rd**	**7th**	**15th**	**30th**	**45th**	**60th**
1	Good	Good	Good	Good	Excellent	Excellent	Excellent
2	poor	Good	Good	Good	Good	Excellent	Excellent
3	Good	Good	Good	Good	Excellent	Excellent	Excellent
4	Poor	Good	Good	Good	Good	Excellent	Excellent
5	Good	Good	Good	Good	Good	Excellent	Excellent
6	Poor	Good	Good	Good	Excellent	Excellent	Excellent

CLINICAL WEIGHT BEARING PATTERN

Plate 13: Preoperative photograph showing weight bearing pattern graded as poor

Plate 14: Photograph showing weight bearing pattern graded as good on 0 post-operative day

Plate 15: Photograph showing weight bearing pattern graded as good on 3th post-operative day

Plate 16: Photograph showing weight bearing pattern graded as good on 7th post-operative day

Plate 17: Photograph showing weight bearing pattern graded as excellent on 15th post-operative day

Plate 18: Photograph showing weight bearing pattern graded as excellent on 30th post-operative day

Plate 19: Photograph showing weight bearing pattern graded as excellent on 45th post-operative day

Plate 20: Photograph showing weight bearing pattern graded as excellent on 60th post operative day

4.5.2 Radiographic evaluation of Hip Dysplasia in dogs after stabilization with nylon zap strap

The assessment of coxofemoral luxation/subluxation in hip dysplastic dogs following stabilization with percutaneous transfemoro-acetabular zap strap appication was done by pre and post operative radiographs.

Day 0: The dysplastic dogs having coxofemoral luxation/subluxation (Plate 21) were stabilized with nylon zap strap. Immediate post operative radiography revealed normal anatomical alignment of coxofemoral joint in all six cases after stabilization (Plate 22).

Since, the nylon zap strap is radiolucent the opposition of femoral head to acetabular socket was assessed for the nylon zap strap in position.

Day 7: Radiographs on 7th post operative day revealed no change in alignment of coxofemoral luxation/subluxation in dysplastic dogs. Since, the nylon is radiolucent the opposition of femoral head to acetabular socket was assessed for the nylon zap strap in position. (Plate 23).

Day 15: Radiographs on 15th post-operative day showed no changes in aligment and there were no arthritic changes noticed in the femoral head and the acetabulum. (Plate 24)

Day 30: Radiographs on 30th post-operative day showed good opposition with femoral head and acetabulum with no arthritic changes (Plate 25).

Day 45: Radiographs on 45th post-operative day showed good apposition between the head of the femur and acetabulum with no arthritic changes (Plate 26).

Day 60: The 60th post-operative day radiograph showed proper placement of the head of the femur into the acetabulum without any arthritic changes, no reluxation and nylon zap strap breakage. Nylon zap strap was removed on 60th post operative day (Plate 27).

RADIOGRAPHIC EVALUATION

Plate 21: Pre-operative radiograph of a dog suffering from hip dysplasia with luxated left coxo-femoral joint

Plate 22: Immediate post-operative radiograph of operated left limb in good opposition

Plate 23: Photograph of radiograph of femoral head in good opposition on 7th day

Plate 24: Photograph of radiograph of femoral head in good opposition on 15th day

Plate 25: Photograph of radiograph of femoral head in good opposition on 30th day

Plate 26: Photograph of radiograph of femoral head in good opposition on 45th day

Plate 27: Photograph of radiograph of femoral head in good opposition on 60th day

4.6 Physiological parameters

4.6.1 Rectal temperature (^{0} F)

The preoperative mean value of rectal temperature 100.53 ± 0.14 ^{0} F and post-operatively it ranged from100.51 ± 0.22 to 102.35 ± 0.11 ^{0}F and the values were within the normal range. The mean rectal temperatures were significantly elevated ($P \leq 0.05$) on 1st and 3rd postoperative days. However these values were within the normal physiological range throughout the study period. The changes in mean values of rectal temperature were statistically non significant (Table 8 and Fig. 6).

4.6.2 Respiratory rate (breaths per minute)

The preoperative mean value of respiratory rate was 36.33±1.20 breaths/min and post-operatively it ranged from 35.33 ±1.42 breaths/min to 37.66±2.04 breaths/min and the values were within the normal range (Table 8 and Fig. 7).

4.6.3 Heart rate (beats per minute)

The preoperative mean value of heart rate was 80.33±1.38 beats/min and post-operatively it ranged from 77.50±0.84 to 79.66±2.20 beats/min and the values were within the normal range. The mean heart rates were significantly elevated ($P \leq 0.05$) than preoperative values on 15th and 30th postoperative days. However these values were within the normal physiological range throughout the study period. Further, within the group the mean values of heart rate were statistically non-significant (Table 8 and Fig. 8).

Table 8: Rectal Temperature, Respiratory Rate and Heart Rate (Mean ± SE) in hip dysplastic dogs

Days	Rectal Temperature (°F)	Respiratory Rate (breaths / minute)	Heart Rate (beats / minute)
0 day	100.53±0.14	36.33±1.20	76.66±1.20
1th day	102.35±0.11	37.66±2.13	77.66±1.66
3th day	101.86±0.20	36.33±1.22	77.5±1.36
5th day	101.63±0.13	37.66±1.33	77.33±1.11
7th day	101.16±0.04	35.33±1.42	77.00±1.26
15th day	101.01±0.17	37.66±2.04	78.50±2.55
30th day	100.53±0.18	35.66±1.17	78.00±1.69
45th day	100.51±0.22	35.00±0.51	77.50±1.62
60th day	100.60±0.13	35.00±1.00	76.33±1.17

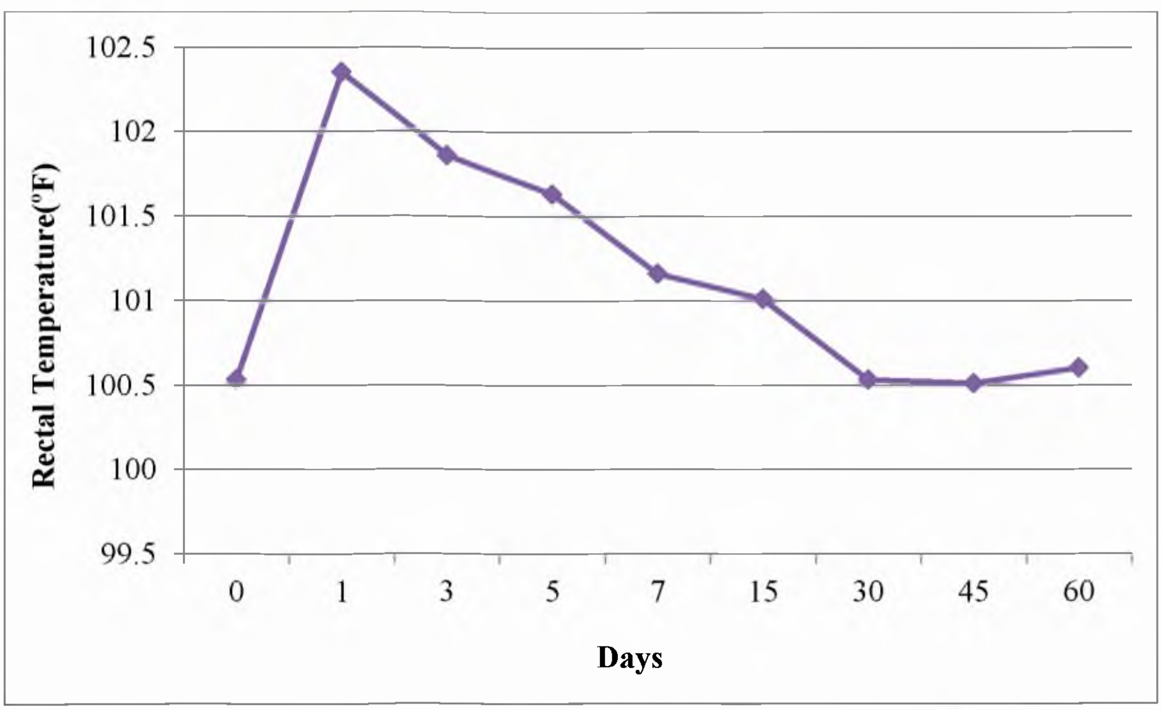

Fig. 6: Graph showing pre and post-operative rectal temperature (°F) (Mean±SE) in Hip dysplastic dogs

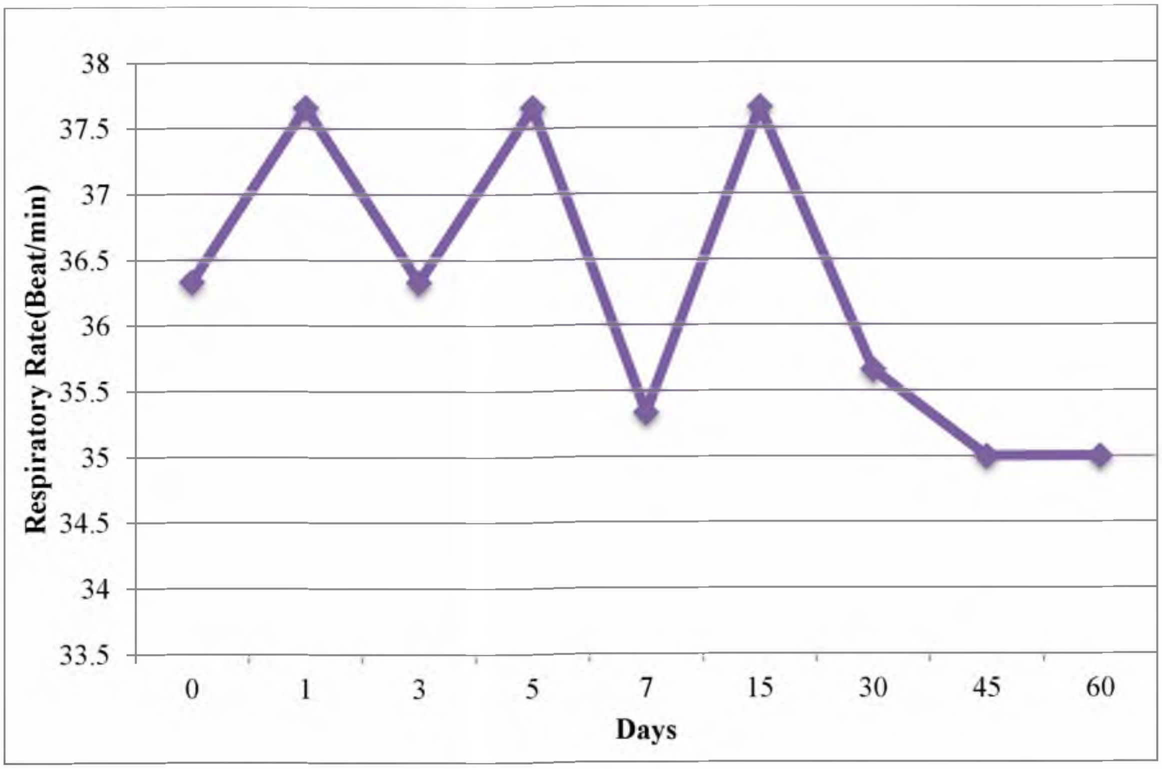

Fig. 7: Graph showing pre and post-operative respiratory rate (breaths / min) (Mean±SE) in hip dysplastic dogs.

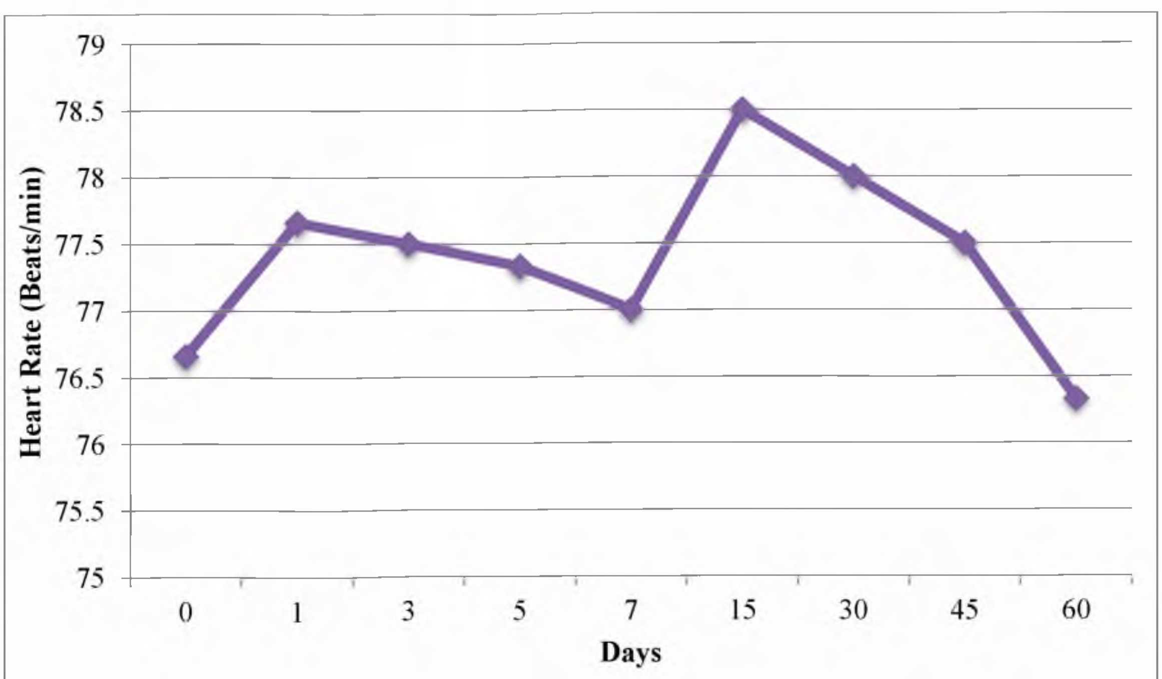

Fig. 8: Graph showing pre and post-operative heart rate (beats / min) (Mean±SE) in hip dysplastic dogs

4.7 Haematological studies

4.7.1 Haemoglobin (gm/dl)

The preoperative mean value of hemoglobin was 12.16±0.35 and post-operatively it ranged from 11.03±0.29 to 12.45±0.28 and the values were within the normal range.

There was an apparent decrease in hemoglobin from 3rd to 5th post-operative days. However the values were within the normal range. Further, within the group the changes in mean values of hemoglobin were statistically non significant (Table 9 and Fig. 9).

4.7.2 PCV (%)

The preoperative mean value of PCV (%) was 39.40±1.31 and post-operatively it ranged from 38.56±1.54 to 41.48±1.99 and the values were within the normal range.

There was an apparent decreased in packed cell volume on 7th post operative day and increased on 15th and 30th post-operative days. However the values were within the normal range. Further, within the group the changes in mean values of packed cell volume were statistically non significant (Table 9 and Fig. 10).

4.7.3 Total Erythrocyte Count (millions/cmm)

The preoperative mean value of total erythrocyte count level was 6.04±0.35x10^6 cells/cmm and post-operatively it ranged from 6.17±0.29 x10^6 cells/cmm to 6.37±0.71 x10^6 cells/mm^3 and the values were within the normal range.

There was an apparent decrease in total erythrocyte count on 1^{st}, 3rd and 5^{th} post-operative day and the values were within the normal range. Further, within the group the changes in mean values of Total Erythrocyte Count were statistically non significant (Table 9 and Fig. 11).

4.7.4 Total Leukocyte Count (thousand cells/cmm)

The preoperative mean value of total leukocyte count level was 10.616.67±314.55 cells/cmm and post-operatively it ranged from 9333.33±424.00 cells/cmm to 10816.67±569.45 cells/mm^3 and the values were within the normal range.

There was an apparent increased in total leukocyte count from first to third post-operative day and the values were within the normal range. Further, within the group the changes in mean values of total leukocyte count were statistically non-significant (Table 9 and Fig. 12).

4.8 Differential leukocyte count

4.8.1 Neutrophils

The preoperative mean neutrophilic count (%) was 78.50±0.88 and post-operatively it ranged from 78.33 ±0.66 to 82.83 ± 0.60 and the values were within the normal range.

There was an apparent increase in neutrophilic count from first to fifth post-operative day and the values were within the normal range. Further, within the group the changes in mean values of neutrophilic counts were statistically non-significant (Table 10 and Fig. 13).

Table 9: Haemoglobin (gm/dl), PCV (%), Total Erythrocyte Count (millions/ cmm) and Total Leukocyte Count (thousands/cmm) in (Mean±SE) with hip dysplasia before and after surgery

Days	Hemoglobin (gm/dl)	PCV (%)	Total Erythrocyte Count (millions/cmm)	Total Leukocyte Count (thousands/cmm)
0 day	12.16±0.35	39.40±1.31	6. 35±0.11	10333.33±98.88
1th day	11.58±0.35	40.46±0.84	6.31±0.21	10900.00±547.11
3rd day	11.03±0.29	40.60±1.50	6.17±0.16	10650.00±168.81
5th day	11.33±0.50	40.60±1.78	6.25±0.15	10216.67±245.51
7th day	12.11±0.71	38.56±1.54	6.37±0.28	10166.67±574.26
15th day	11.70±0.50	41.48±1.99	6.31±0.33	10266.67±304.04
30th day	11.85±0.78	42.30±1.90	6.35±0.32	10450.00±306.32
45th day	12.35±0.35	40.55±0.26	6.27±0.23	10316.67±278.58
60th day	12.45±0.28	39.96±1.23	6.31±0.08	10366.67±264.15

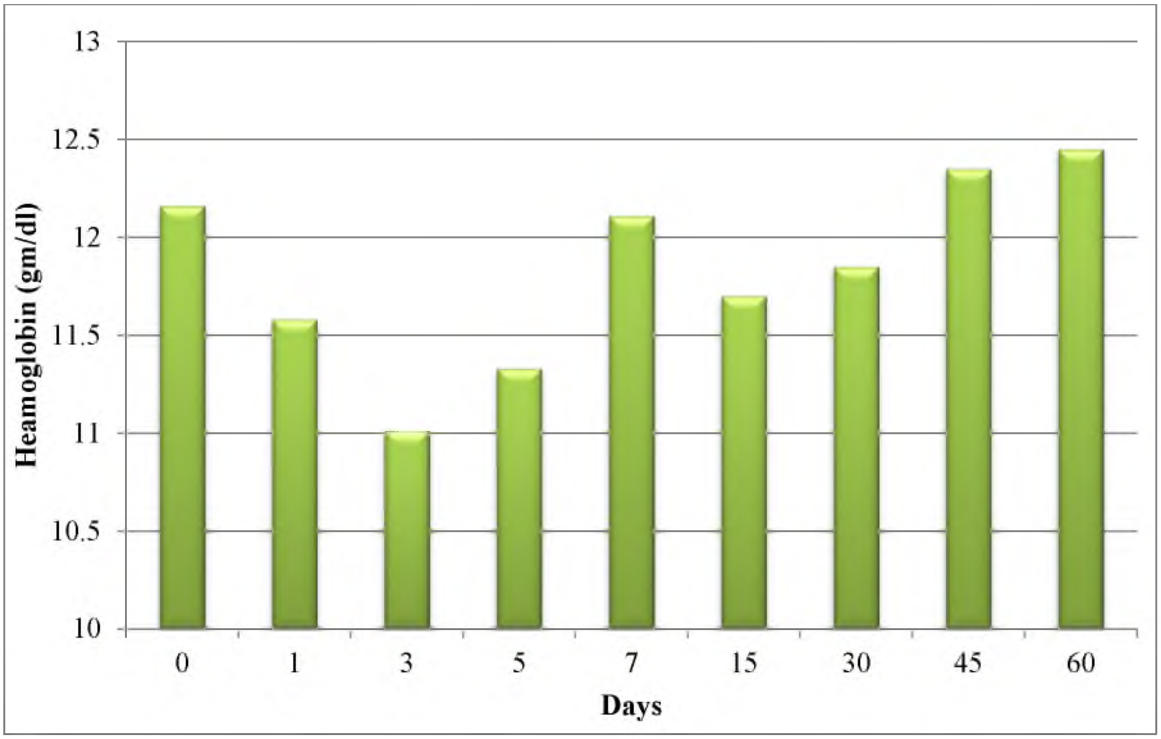

Fig. 9: Graph showing pre and post-operative haemoglobin values (gm/dL) (Mean±SE) in hip dysplastic dogs

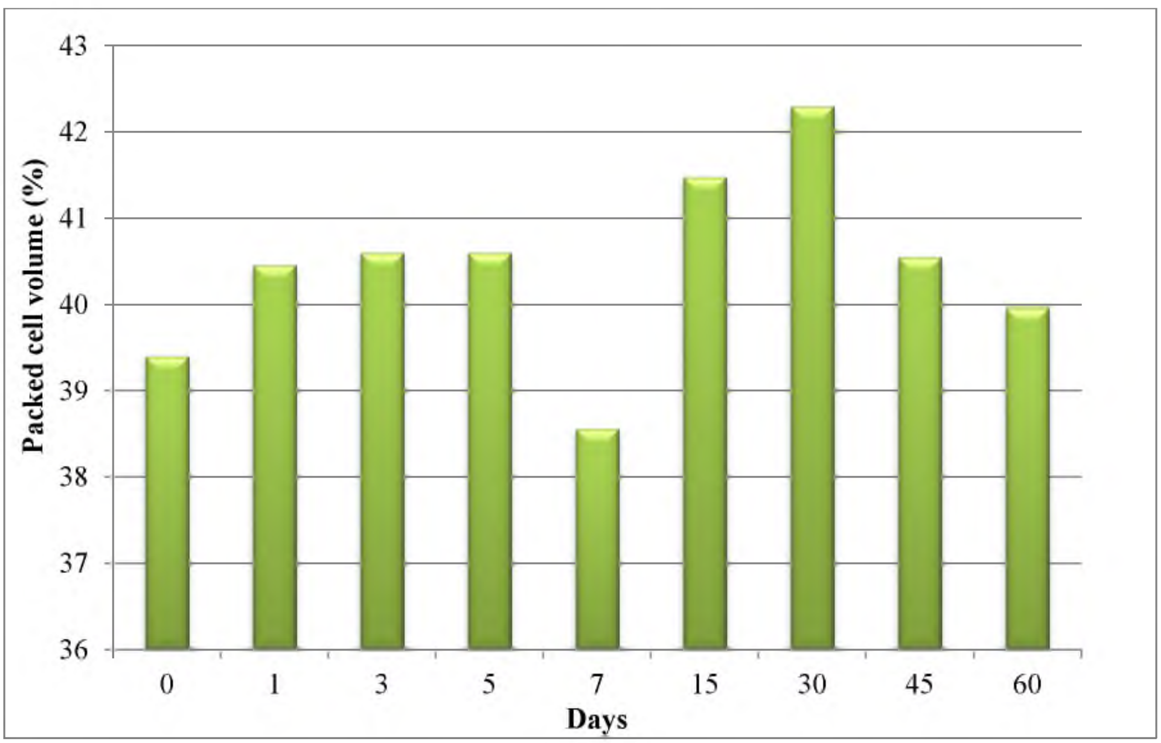

Fig. 10: Graph showing pre and post-operative PCV (%) (Mean±SE) in hip dysplastic dogs

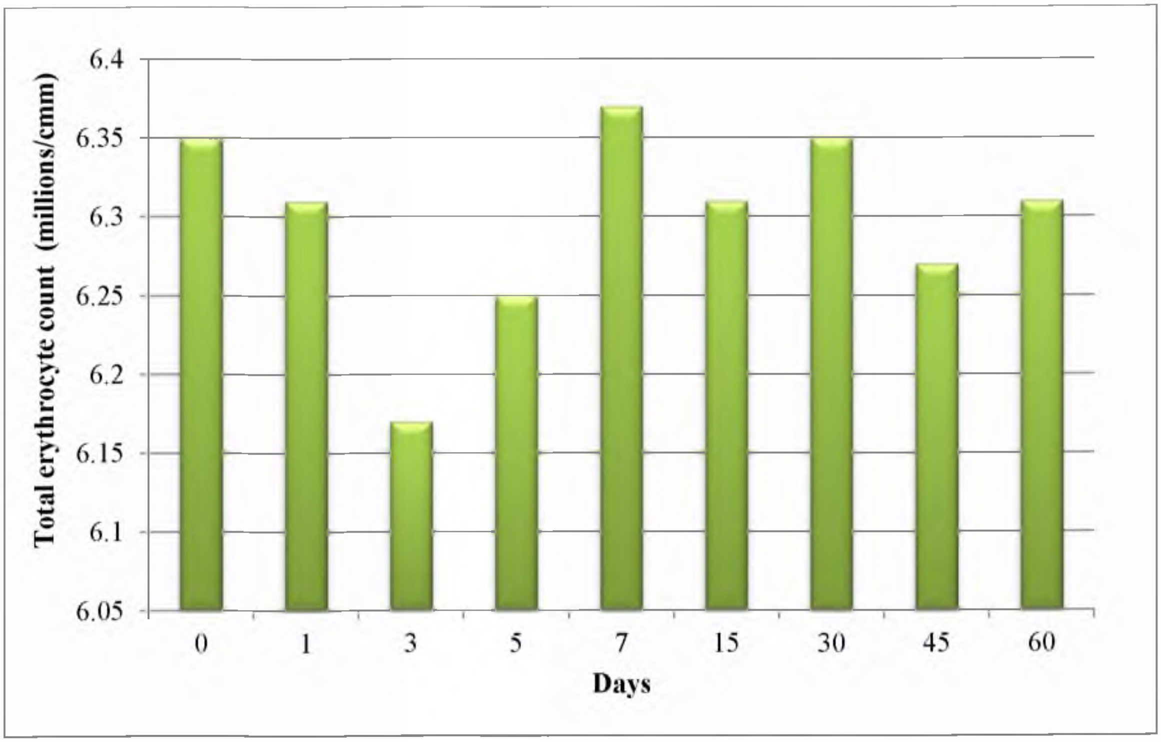

Fig. 11: Graph showing pre and post-operative TEC (millions/cmm) (Mean±SE) in hip dysplastic dogs

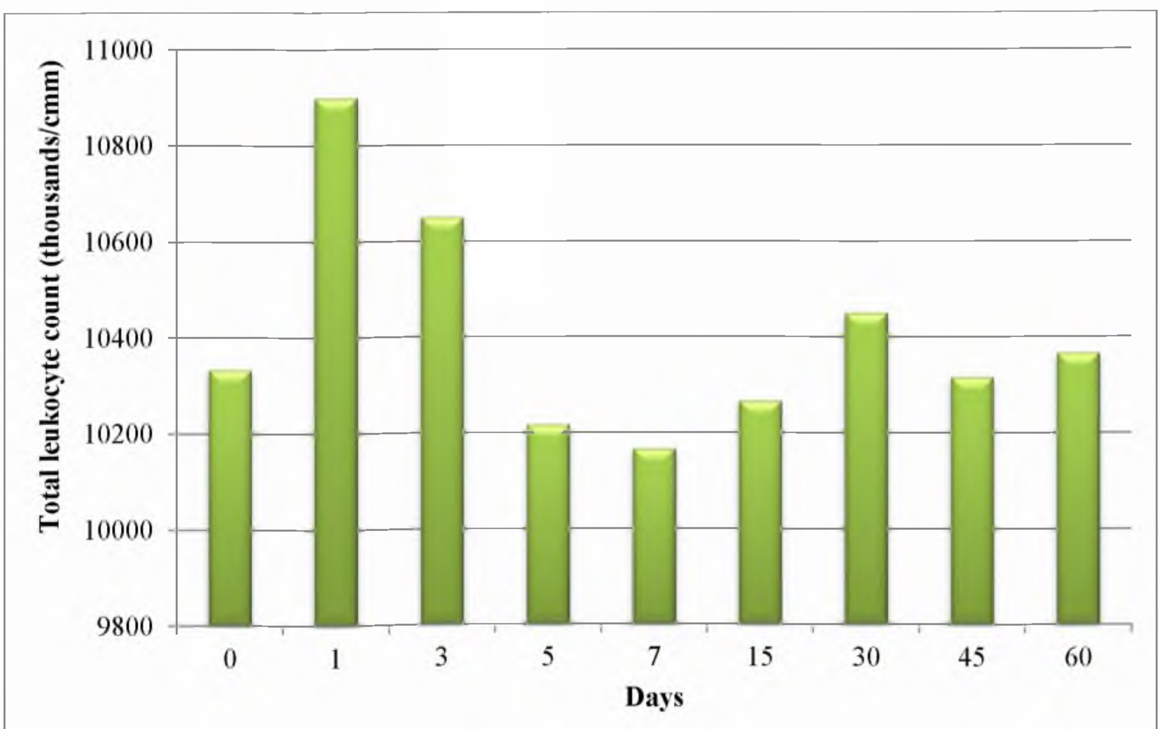

Fig. 12: Graph showing pre and post-operative TLC (thousands/cmm) (Mean±SE) in hip dysplastic dogs

4.8.2 Lymphocytes

The preoperative mean lymphocytic count (%) was 19.66±0.49 and post-operatively it ranged from 15.33 ±1.22 to 18.50 ± 1.05 and the values were within the normal range.

There was an apparent decrease in lymphocytic count on 30th post-operative day and the values were within the normal range. Further, within the group the changes in mean values of lymphocytic counts were statistically non-significant (Table 10 and Fig. 14).

4.8.3 Monocytes

The preoperative mean monocyte count (%) was 0.83±0.16 and post-operatively it ranged from 0.66 ± 0.21 to 1.00 ± 0.25 and the values were within the normal range. (Table 10 and Fig. 15).

4.8.4 Eosinophils

The preoperative mean eosinophil count (%) was 1.16±0.30 and post-operatively it ranged from 0.66 ± 0.21 to 1.50 ± 0.42 and the values were within the normal range.

There was an apparent increase in Eosinophil count on 5th and 45th post-operative day and the values were within the normal range. Further, within the group the changes in mean values of Eosinophilic counts were statistically non-significant (Table 10 and Fig. 16).

4.8.5 Basophils

The basophil counts were nil in both the pre and postoperative periods.

Table 10: Differential leukocyte count (Mean±SE) in hip dysplastic dogs before and after surgery

Days	Neutrophils %	Lymphocytes %	Monocytes %	Esinophils %
0 day	78.50±0.88	19.66±0.49	0.83±0.16	1.16±0.30
1^{th} day	82.83±0.65	15.83±1.53	1.00±0.25	1.33±0.42
3^{rd} day	82.66±0.71	16.66±1.62	0.83±0.30	0.83±0.16
5^{th} day	81.33±0.91	16.50±1.52	0.66±0.21	1.50±0.42
7^{th} day	79.83±1.07	17.66±1.42	0.66±0.21	1.16±0.40
15^{th} day	80.33±0.61	16.83±1.01	1.00±0.25	1.00±0.36
30^{th} day	79.83±1.19	15.33±1.22	0.66±0.33	1.00±0.25
45^{th} day	77.66±1.05	16.83±1.81	0.50±0.22	1.50±0.42
60^{th} day	78.33±0.66	18.50±1.05	0.83±0.30	0.66±0.21

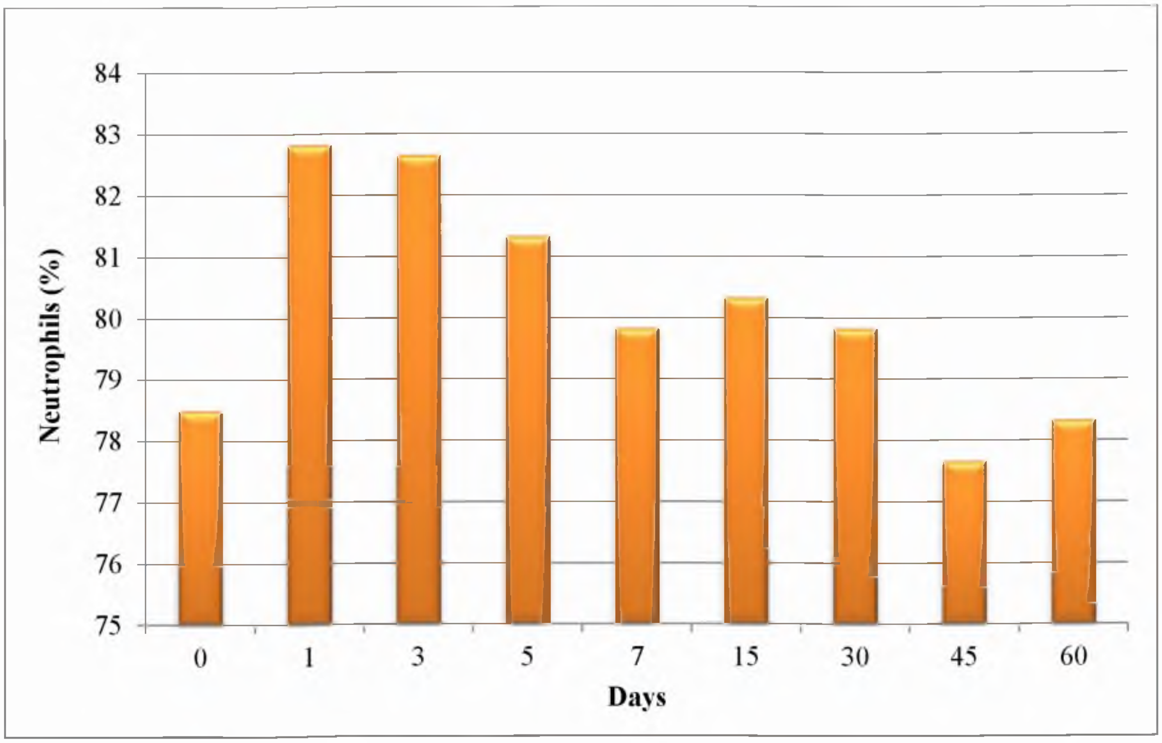

Fig. 13: Graph showing pre and post-operative Neutrophils (%) (Mean±SE) in hip dysplastic dogs

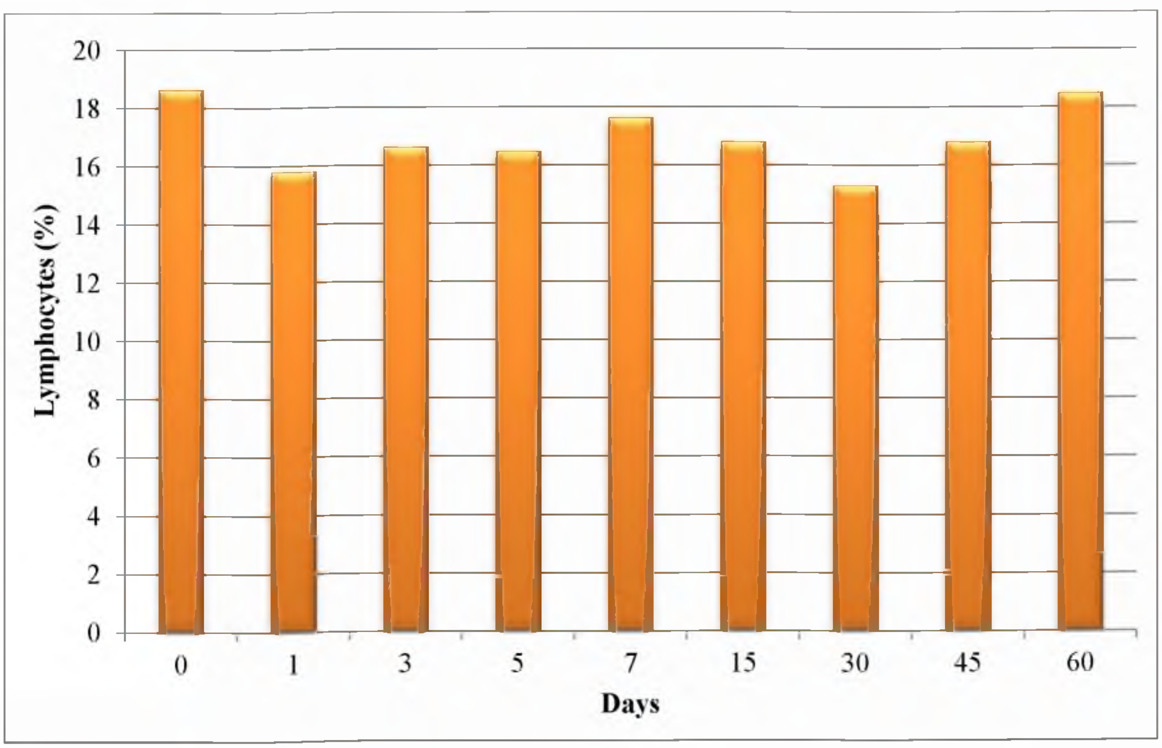

Fig. 14: Graph showing pre and post-operative Lymphocytes (%) (Mean±SE) in hip dysplastic dogs

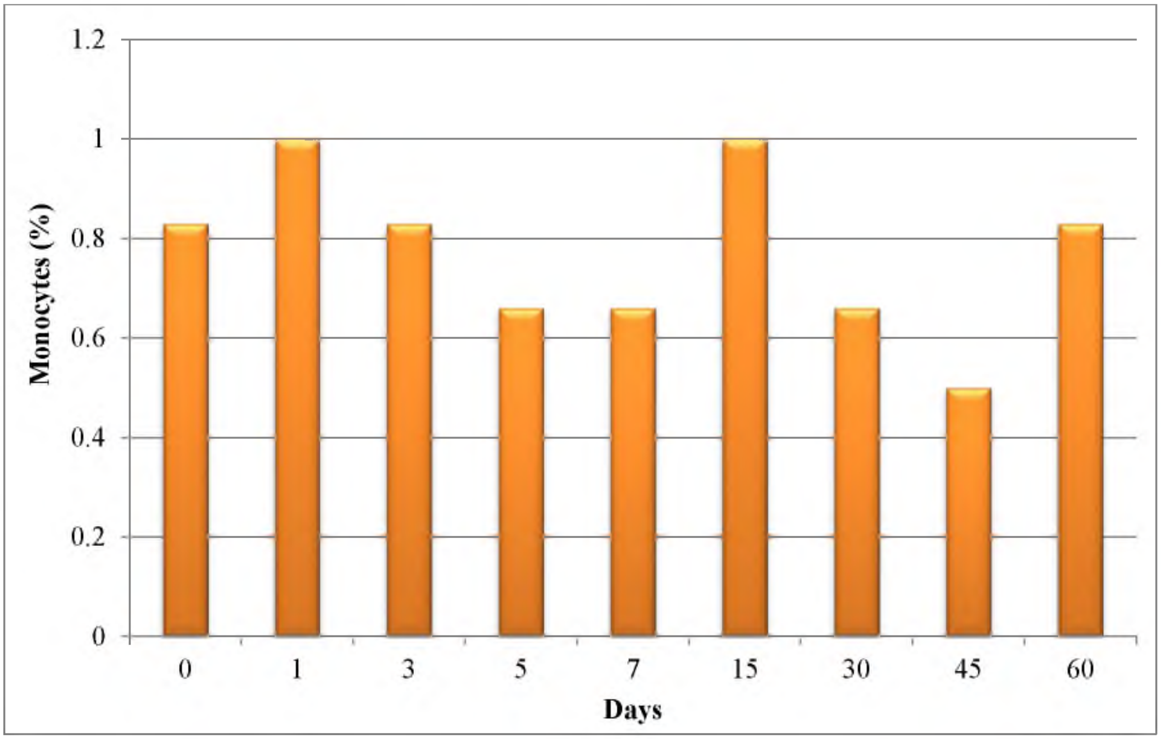

Fig. 15: Graph showing pre and post-operative Monocytes (%) (Mean±SE) in hip dysplastic dogs

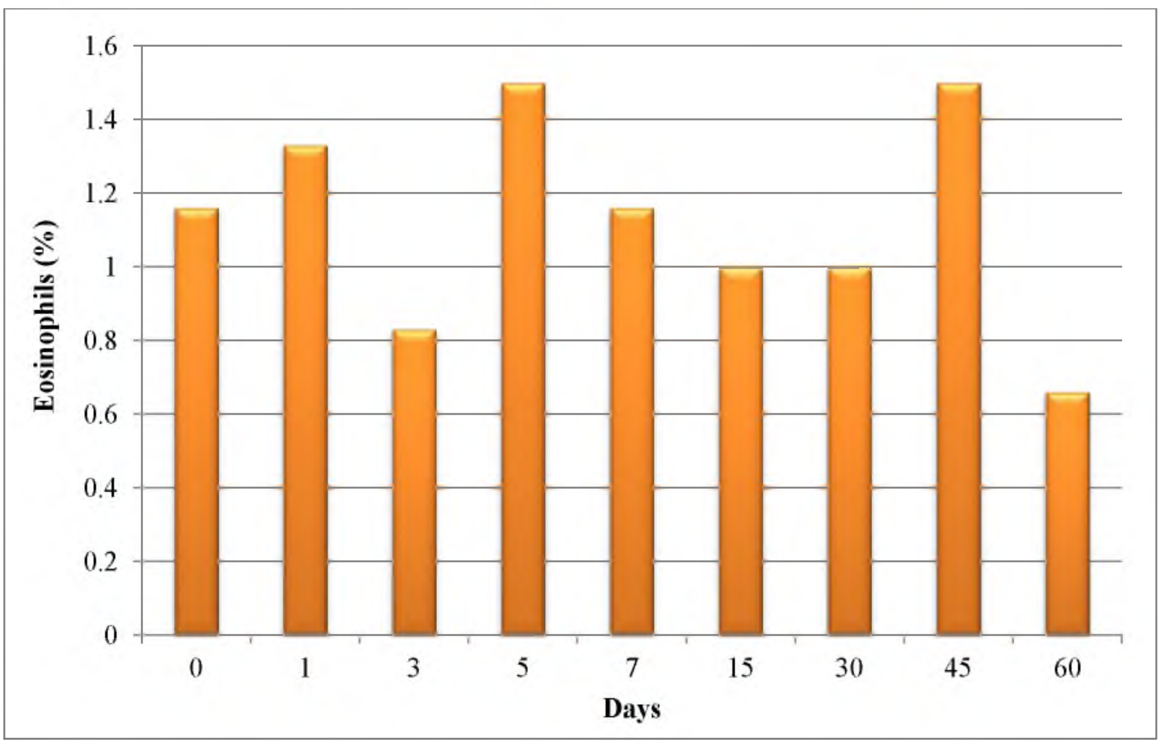

Fig. 16: Graph showing pre and post-operative Esinophils (%) (Mean±SE) in hip dysplastic dogs

4.9 Biochemical Studies

4.9.1 Serum alanine aminotransferase (IU/L)

The preoperative mean value of Serum alanine aminotransferase was 33.33 ±3.45 IU/L and post-operatively it ranged from 32.66±2.20 IU/L to 36.16±2.79 IU/L and the values were within the normal range.

There was an apparent increase in serum alanine aminotransferase upto 7th post-operative day which gradually reduced to normal by 60th post operative day and however the values were within the normal range. Further, within the group the changes in mean values of serum alanine aminotransferase were statistically non significant (Table. 11 and Fig. 17).

4.9.2 Serum Aspartate Aminotransferase (IU/L)

The preoperative mean value of Serum aspartate aminotransferase was 39.66±0.42 IU/L and post-operatively it ranged from 39.00±0.85 IU/L to 43.16±1.01 IU/L and the values were within the normal range.

There was an apparent increase in serum aspartate aminotransferase upto 7th post-operative day which gradually reduced to normal by 60th post operative day and however the values were within the normal range. Further, within the group the changes in mean values of serum alanine aminotransferase were statistically non significant (Table. 11 and Fig. 18).

4.9.3 Serum alkaline phosphatase (IU/L)

The preoperative mean value of serum alkaline phosphatase was 123.33±1.45 IU/L and post-operatively it ranged from 120.16±1.40 to 131.00±0.96 IU/L and the values were within the normal range.

There was an apparent increase in serum alkaline phosphatase upto 15th post-operative day and the values were within the normal range. Further, within the groups the changes in mean values of serum alkaline phosphatase were statistically non-significant (Table. 11 and Fig. 19).

4.9.4. Serum Creatinine (mg/dl)

The preoperative mean value of serum creatinine was 0.80±0.06 (mg/dl) and post-operatively it ranged from 0.73 ±0.09 mg/dl to 0.85±0.11 mg/dl and the values were within the normal range.

There was an apparent increase in serum creatinine upto 7thpost-operative day which gradually reduced to normal by 45th day. However the values were within the normal range. Further, within the group the changes in mean values of serum creatinine was statistically non-significant (Table. 11 and Fig. 20).

Table 11: Serum alanine amino transferase (IU/L), Serum alkaline phosphatase (IU/L) and serum creatinine(mg/dl) in dogs (Mean±SE) with hip dysplasia before and after surgery

Days	Serum Alanine amino Transferase (IU/L)	Serum Aspartate Aminotransferase (IU/L)	Serum Alkaline Phosphatase (IU/L)	Serum Creatinine (mg/dl)
0 day	33.33±3.45	39.66±0.42	123.33±1.45	0.80±0.06
1st day	34.33±2.52	40.83±0.65	131.00±0.96	0.78±0.06
3rd day	34.16±2.25	41.83±0.54	126.00±2.29	0.78±0.10
5th day	35.00±1.87	42.5±0.76	126.83±2.25	0.73±0.09
7th day	36.16±2.79	43.16±1.01	127.83±2.13	0.85±0.11
15th day	32.66±3.99	40.16±0.74	128.33±2.10	0.83±0.09
30th day	32.66±2.20	39.50±0,84	125.33±1.54	0.76±0.15
45th day	34.50±1.82	39.00±0.85	125.33±1.56	0.76±0.12
60th day	34.00±0.92	39.16±1.60	120.16±1.40	0.78±0.04

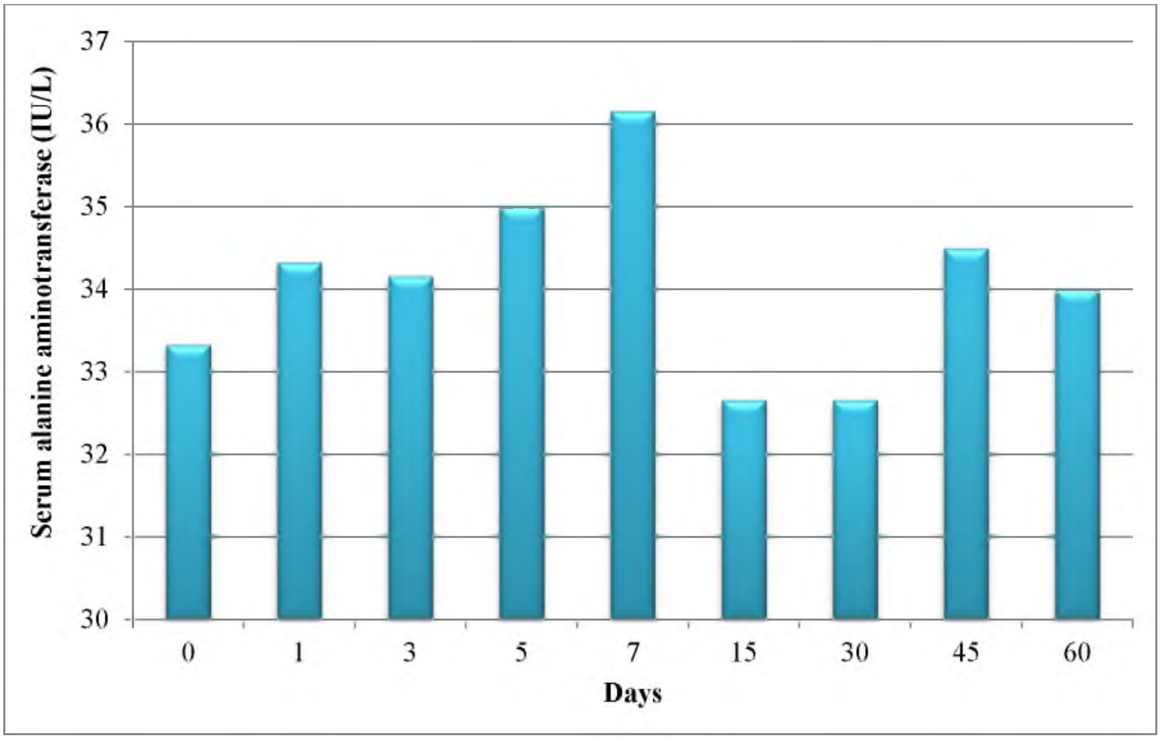

Fig. 17: Graph showing pre and post-operative serum alanine amino transferase (IU/L) (Mean±SE) in dogs with hip dysplasia

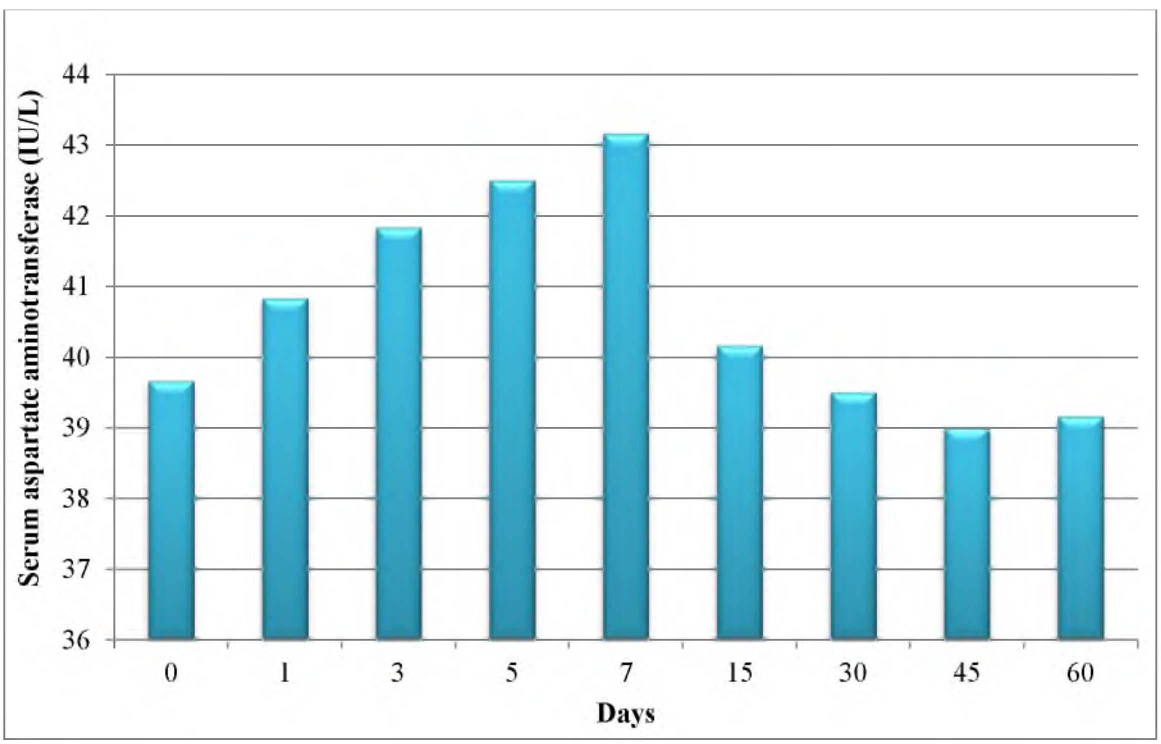

Fig. 18: Graph showing pre and post-operative Serum Aspartate Aminotransferase (IU/L) (Mean±SE) in dogs with hip dysplasia

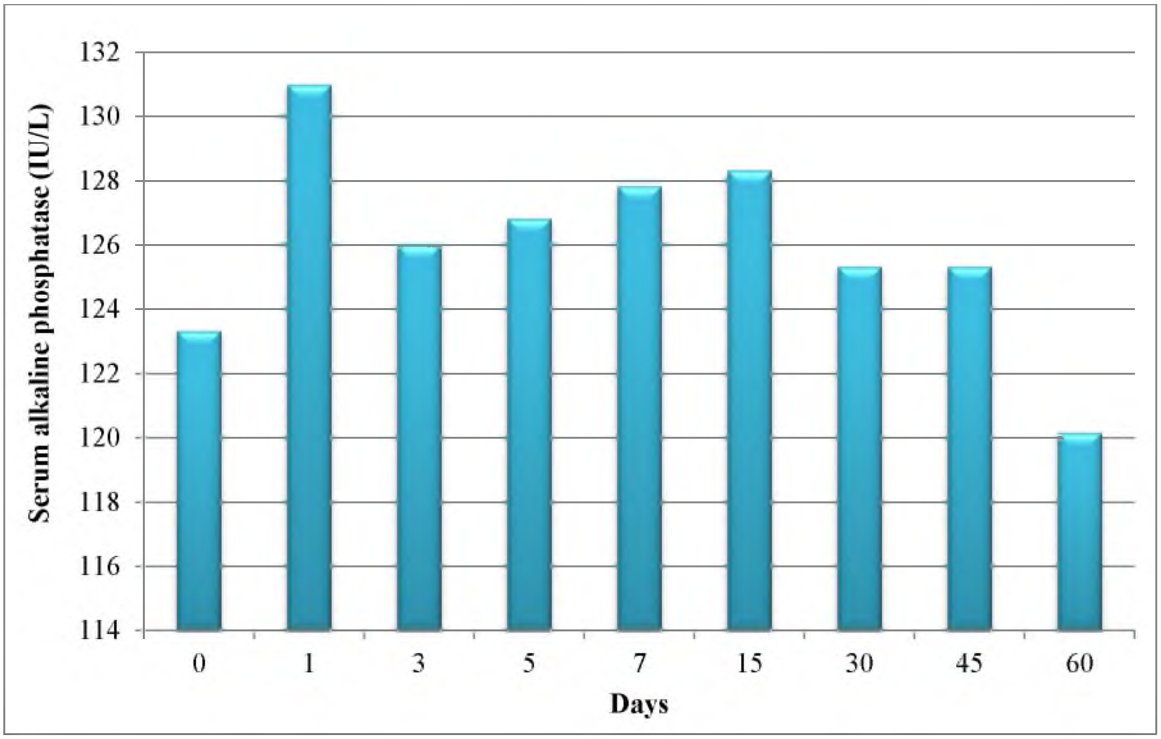

Fig. 19: Graph showing pre and post-operative serum alkaline phosphatase (IU/L) (Mean±SE) in dogs with hip dysplasia

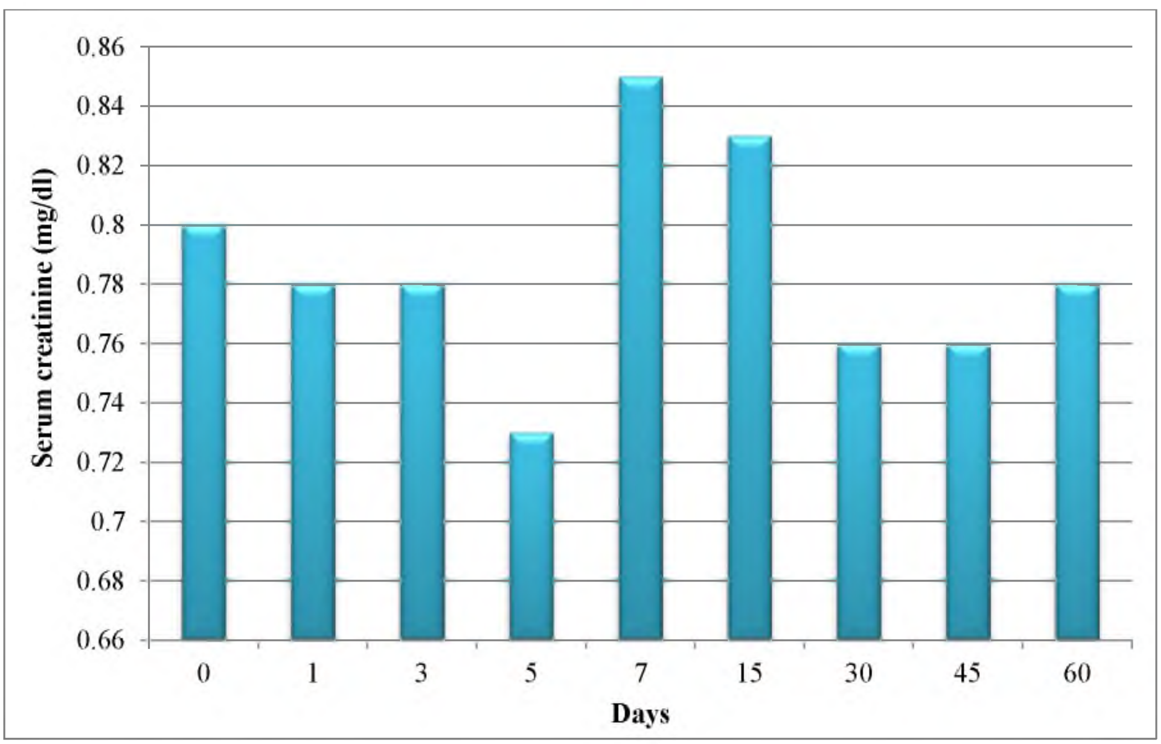

Fig. 20: Graph showing pre and post-operative serum creatinine (mg/dl) (Mean±SE) in dogs with hip dysplasia

Discussion

V. DISCUSSION

The present study was carried out to study the occurrence of coxofemoral subluxation/luxation in hip dysplastic dogs and to standardize the technique of Percutaneous transfemero-acetabular nylon zap strap application for the repair of subluxation/luxation in hip dysplastic dogs.

5.1 Occurrence

In the present study subluxation/luxation in hip dysplastic dogs recorded over one year of period (May 2015 to April 2016). Totally 81 cases of hip dysplastic dogs were reviewed. Among 81 cases, occurrence of subluxation of hip joint was 59(0.37%) and luxation of hip joint was 22(0.13%) dogs out of 13489 dogs represented. Canine hip dysplasia (CHD) was one of the most prevalent developmental orthopaedic diseases in dogs and it was characterized by an abnormal formation of the hip joint with different degrees of laxity and subluxation/luxation, which ultimately leads to secondary osteoarthritis (OA) and impaired animal welfare. The study was in accordance with (Ginja *et al*., 2010; Bartolome *et al.*, 2015).

In the present study, the highest incidence of hip dysplasia was more common in Labrador retriever breed 18 (22.22%) followed by German shepherd 12 (14.81%), Saint Bernard 12 (14.81%), Rottweiler's 11 (13.58%), Golden Retriever 10 (12.34%), Pugs 9 (11.11%), Great Dane 6 (7.40%), Non-Descriptive breed 2 (2.46%) and Doberman 1 (1.23%). Among hip dysplastic dogs, Incidence of subluxation of hip were more in Labrador retriever 20.33%(12) followed by Saint Bernard 16.94%(10), German shepherd 15.25%(9), Rottweiler's11.86%(7), Golden Retriever11.86%(7), Pugs11.86%(7), Great

Dane8.47%(5), Non-Descriptive breed 1.69%(1) and Doberman breeds 1.69%(1) and Incidence of luxation of hip were more seen in Labrador retriever 27.27%(6), Rottweiler's18.18%(4), German shepherd 13.63%(3), Golden Retriever 13.63%(3), Saint Bernard 9.09%(2), Pugs 9.09%(2), Great Dane 4,54%(1) and Non-Descriptive breeds 4.54%(1). The breed wise incidence of the hip dysplasia was more common in Labrador retriever and the large breeds of dogs most commonly affected with hip dysplasia because of heavy body weight, heavy pelvic muscle mass, rapid growth rate, loose skin, more than 5% to 10% subcutaneous fat and relative overloading of the skeleton. The study was in accordance with (Riser, 1985; Fries and Remedios, 1995; Ranganath and Subin, 2006; Coopman *et al*., 2008; Shiju Simon *et al*., 2010)

Age-wise distribution of hip dysplasia in dogs more commonly seen in the age group of less than one year old were about 49(60.49%) dogs followed by 7(8.64%) dogs were between one to two years, 7(8.64%) dogs were between two to three years, 3(3.70%) dogs were between three to four years, 5(6.17%) dogs were between four to five years , 3(3.70%) dogs were five to six years and 7(8.64%) dogs were more than six years.

Occurrence of subluxation of hip in dogs more commonly seen in the age group of less than one year old were about 38(64.40%) dogs followed by 5(8.47%) dogs were between one to two years, 4(6.77%) dogs were between two to three years, 3(5.08%) dogs were between three to four years, 4(6.77%) dogs were between four to five years , 1(1.64%) dog was five to six years and 4(6.77%) dogs were more than six years of age. Occurrence of luxation of hip in dogs more commonly seen in age group of less than one year old were about 11(50%) dogs followed by 2(9.09%) dogs were between one to two years, 3(13.63%) dogs were between two to three years, 0(0%) dogs were between three to four years, 1(4.54%) dogs were between four to five years , 2(9.09%) dogs were five to six years and 3(13.63%) dogs were more than six years of age. In the present study, the highest incidence of hip dysplasia in dogs was found in the age group less than one year. The study was in accordance with Manley *et al.* (2007) and Shiju Simon *et al.* (2010)

In the present study, the sex-wise distribution of hip dysplasia, the males 57(70.37 %) were commonly affected as compared to females 24 (29.62%). Among the hip dysplastic dogs, Occurrence of subluxation more seen in male 43(75.43 %) compared to females 16(66.66 %) and Occurrence of luxation of hip also common in male 14(24.56%) compared to females 8(33.33%) dogs. In the present study, the highest incidence of hip dysplasia was seen in males compared to females. Similar observations was made by Martin *et al.* (1980), Wood *et al.* (2002), Jayaprakash *et al.* (2007), Shiju Simon *et al.* (2010) and Stanin *et al.* (2011),

5.2 Etiology for coxofemoral subluxation/luxation in hip dysplastic dogs.

In the present study, the highest incidence of coxofemoral subluxation/luxation were recorded due to hip dysplasia in dogs. In dogs, hip dysplasia was developmental orthopaedic disorder characterised by the formation of a loose, ill-fitting coxofemoral (hip) joint and the malformation of joint leads to abnormal wearing of bone surfaces, excessive laxity of the hip joint with abnormal endochondral ossification leads to subluxation/luxation of hip joint and the appearance of the osteoarthiritic signs of degenerative joint disease (DJD). Similar observations was made by Todhunter and Lust, (2003); Lewis *et al*. (2010).

5.3 Premedication with anaesthesia.

The premedication of dogs with Atropine sulphate at a dose rate of 0.045 mg per kg was found to be satisfactory. The administration of Atropine sulphate reduced the salivary secretion and maintained normal heart rate. This is in agreement with Kumar *et al.* (2004).

Preanaesthesia with diazepam at a dose rate of 1 mg per kg body weight provided good sedation. The use of diazepam for premedication was also reported earlier by Yardimci *et al*. (2011).

Induction with 2.5 % Thiopentone sodium intravenously to the effect and maintenance by general anaesthesia with Isoflurane provided adequate surgical plane of anesthesia for successful completion of surgery without any complication in all cases. This had also been reported earlier by Aithal *et al.* (1999), Fazili *et al.* (2008).

5.4 Positioning of the animals

The animal was placed on lateral recumbency with the affected limb above. This positioning was satisfactory enough to visualise the subluxation/luxation site and successful reduction of coxofemoral subluxation/luxation and easy application of nylon zap strap to coxofemoral subluxation/luxation in hip dysplastic dogs. This was in accordance with Carmichael (1989).

5.5 Surgical technique

Surgical technique employed was adequate for satisfactory correction of hip subluxation/luxation and to pass the nylon zap strap through femoral head and acetabular axis. These results are in agreement with the suggestions of Fossum *et al.*, (1997), Smitha, (2014)

5.6 Weight bearing

All the dogs started bearing weight on affected limb as early on the first post operative day. This may be due to satisfactory placement of the femoral head into the acetabulum and this is in accordance with the observations of Anthony *et al.* (1990) and application of nylon zap strap provided excellent stability to coxofemoral subluxation/luxation there by allowing early weight bearing by the animal. The grading of functional limb usage in the post operative days was as per the method suggested by McCartney *et al*., (2011); Smitha (2014)

5.7 Radiological evaluation

The radiographs were obtained, preoperatively and immediately after operation to know the position of the femoral head in the acetabulum. Similar procedure was followed earlier by Margaret *et al., (*1987) and Pai and Kumar (1990).

In the present study, radiographs were taken immediately after nylon zap strap application revealed good apposition of head of the femur and acetabulum.

Radiographs on 7th post operative day revealed, good alignment of femoral head into the acetabulum and In-situ nylon zap strap. Similar observations were also noticed by Bennett and Duff (1980) and Margaret *et al*., (1987).

Radiographs on 15^{h} post operative day revealed nylon zap strap in position and there were no arthritic changes noticed in the femoral head and the acetabulum. These observations are in line with those of Ron *et al*., (1994) and Ash *et al*., (2012).

Radiographs on 30th post operative day revealed good opposition with femoral head and acetabulum and no arthritic changes were noticed. Similar observations were also recorded by Rochereau and Bernarde*et al., (*2012).

Radiographs on 45th post operative day revealed, good apposition between the head of the femur and acetabulum. The nylon zap strap was in position. Similar observations were noticed by Candace *et al*., (1987).

Radiographs on 60th post operative day revealed proper placement of the head of the femur into the acetabulum without any arthritic changes, no reluxation and nylon zap strap breakage. Nylon zap strap was removed on 60th post operative day. Similar observations were noticed by David *et al*., (2006), Venzin and Montavon (2007) and smitha, (2014).

5.8 Post-operative observation

5.8.1 Clinical evaluation

5.8.1.1 Temperature, Heart rate and Respiratory rate

The mean ± S.E. of Rectal Temperature (^{0}F) values in dogs were recorded during pre and post-surgical evaluation period. The values were increased non- significantly on 1st and 3rd postoperative days. However these values were within the normal physiological range throughout the study period.

The mean ±S.E. of Respiratory rate (breaths/min) values in dogs were recorded during pre and post-surgical evaluation period. The values were increased non-significantly on 5th and 15th postoperative days. However these variations were within the normal physiological range throughout the study period.

The mean ± S.E of Heart rate (beats/min) values in dogs were recorded during pre and post-surgical evaluation. The values were increased non- significantly on 15th and 30th postoperative days. However these values were within the normal physiological range throughout the study period.

This variation in mean rectal temperature, heart rate and respiration rates may possibly be a manifestation by traumatic stress, surgical stress and inflammatory processes occurring at the surgical site. These observations were in accordance with Shashidhara (1997), Smitha (2014) and Shivakumar (2015).

5.8.1.2 Haemoglobin, Packed Cell Volume and Total Erythrocyte Count (TEC)

The mean ± S.E. Haemoglobin (g/dl) values in dogs recorded during pre and post surgical evaluation period. The values were decreased non- significantly on 3rd and 5th postoperative days. However these values were within the normal physiological range throughout the study period as there was minimal blood loss during peri-operative period.

The mean ± S.E. values of packed cell volume (%) in dogs recorded during pre and post surgical evaluation period. The values were increased non- significantly on 15rd and 30th post operative days and decreased on 7th post operative day. However these values were within the normal physiological range throughout the study period.

The mean ± S.E. values of total erythrocyte count (10^6cells/cmm) in dogs recorded during pre and post surgical evaluation period. The values were decreased non-significantly on 1st, 3rd and 5th postoperative days. However these variations were within the normal physiological range.

There was minimal bleeding during the surgical procedure; the variations in haematological parameters were statistically non significant. These observations are in lined with Coles (1974), Todd *et al*., (2000), Singh *et al*., (2008) and Smitha (2014).

5.8.1.3 Total Leukocyte Count

The mean ± S.E. values of total leukocyte count (thousands/cmm) in dogs were recorded during pre and post-surgical evaluation period. The values were elevated non-significantly on 1st, 3rd and 5rd postoperative days and there after receded back to normal by 15th post-operative day. This could be due to surgical stress on operated site (Nagaraja, 1996) and inflammatory changes (Shashidhara, 1997) and Smitha (2014).

5.8.1.4 Serum Alanine Aminotransferase (ALT)

The mean ± S.E. values of serum alanine aminotransferase values (U/L) in dogs recorded during pre and post surgical evaluation period were significantly increased than preoperative values on 1st, 3rd, 5th and 7th post operative days. However these variations were within the normal physiological range.

The non significant increase in ALT level was noticed in initial study period. Rise in ALT above normal physiological range may be due to trauma and surgical stress to the muscles and inflammation. It may also be due to stress of thiopentone sodium on liver.

According to Willard and Tvedten (2012), Common causes of increased serum ALT include hepatic anoxia, poor hepatic perfusion, spontaneous and muscle trauma. ALT is present in the cytosol, increased serum ALT reflects cell membrane damage and leakage. A similar non significant rise of ALT levels was observed by Shashidhara (1997), Gopalkrishna (1993) and Smitha (2014) and Shivakumar (2015)

5.8.1.5 Serum aspartate aminotransferase

The mean± S.E. values of serum alanine aminotransferase values (U/L) in dogs recorded during pre and post surgical evaluation period were significantly increased than preoperative values on 1st, 3rd, 5th and 7th post operative days. However these variations were within the normal physiological range.

The non significant AST level rise only in initial study period within normal physiological range may be due to trauma and surgical insult to the muscles and inflammation. It may also be due to stress of Thiopentone sodium on liver. According to Willard and Tvedten (2012), AST is present in significant quantities in many tissues, including liver, muscle and RBCs. AST is present in the mitochondria. AST increases tend to reflect more serious damage because the mitochondria are not damaged as readily as is the cell membrane.

5.8.1.6 Serum Alkaline Phosphatase (ALP)

A non significant increase in peak value of serum alkaline phosphatase level observed up to 15th postoperative day, later on the values lowered towards the end of the study period. The increase may be due to trauma to the muscle and surgical stress and adrenal hyper function as observed by Willard and Tvedten (2012). All the mean values were within the normal physiological range. The variations were minimal and statistically non significant. A similar non-significant rise of ALP levels were observed by Shashidhara (1997) Gopalkrishna (1993) and Smitha (2014).

5.8.1.7 Serum Creatinine

There was an apparent increase in serum creatinine level observed on 7^{th} and 15^{th} postoperative day, later on the values lowered towards the end of the study period. The variation in the serum creatinine values were within the normal range. Neither the anesthesia nor surgical procedure in the present study had any influence on the kidney function. These observations are in accordance with Smitha (2014) and Shivakumar (2015).

5.9 Post operative complications

None of the dogs showed complications associated with the technique or results. There was no reluxation or arthritic changes postoperatively. McCartney *et al*., (2011) stated osteoarthritis was most common complication in modified transarticular pinning technique. Similar observation was made by Adamiak (2012) in Shani Johnston Shahar technique but author reported reluxation of 12.5% in their technique. Ireifej *et al*. (2012) Reported complications associated with Total hip replacement in dogs include luxation/dislocation, septic loosening, aseptic loosening of both cemented and cementless components, improper implant positioning, periprosthetic femoral fractures, sciatic neurapraxia, patella luxation, extraosseous cement granuloma formation, and femoralmedullary infarction.

Summary

VI. SUMMARY

A study was conducted over a period of one year (May 2015-April 2016) to evaluate the technique of Percutaneous transfemoro-acetabular nylon zap Strap application for repair of coxofemoral luxation/subluxation in six clinical cases of dogs suffering from Hip Dysplasia belonging to different age, sex, breed and body weights.

1. Totally 19854 cases were presented to Veterinary College Hospital, Bangalore during May 2015 to April 2016. Out of 15725 canines presented, 81 dogs were suffering from Hip Dysplasia with coxo-femoral subluxation 59(0.37%) and luxation 22(0.13%) with an overall occurrence of 0.51%

2. Among 59 cases of subluxation of hip joint, 72.88 %(43) were males and 27.11 %(16) were female dogs. Out of 22 luxation cases of hip joint, 63.63 %(14) cases were male and 36.36%(8) were female dogs.

3. A total of 9 breeds of dogs were recorded among 81 cases of hip dysplasia. Breed-wise incidence of hip dysplasia in dogs were Labrador retriever 18 (22.22%), German shepherd dog 12 (14.81%), Saint Bernard 12 (14.81%), Rottweiler's 11 (13.58%), Golden Retriever 10 (12.34%), Pugs 9 (11.11%), Great Dane 6 (7.40%), Non-Descriptive breed 2 (2.46%) and Doberman 1 (1.23%). Among hip dysplasia, incidence of subluxation of hip in dogs were in following order - Labrador retriever, Saint Bernard, German shepherd , Rottweiler's, Golden Retriever, Pugs, Great Dane, Non-Descriptive breed and Doberman breeds were 20.33%(12), 16.94%(10), 15.25%(9), 11.86%(7), 11.86%(7), 11.86%(7), 8.47%(5), 1.69%(1), 1.69%(1)

respectively and incidence of luxation of hip in dogs were – Labrador retriever, Rottweiler's, German shepherd , Golden Retriever, Saint Bernard, Pugs, Great Dane and Non-Descriptive breeds were 27.27%(6), 18.18%(4), 13.63%(3), 13.63%(3), 9.09%(2), 9.09%(2), 4,54%(1), 4.54%(1) respectively.

4. Age-wise occurrence of hip dysplasia in dogs less than one year old, one to two years, two to three years, three to four years, four to five years , five to six years and more than six years were 60.49%(49), 8.64%(7), 8.64%(7), 3.70%(3), 6.17%(5), 3.70%(3),8.64%(7) respectively. among the 81 hip dysplastic dogs, incidence of subluxation of hip in dogs less than one year old, one to two years, two to three years, three to four years, four to five years , five to six years and more than six years were 64.40%(38), 8.47%(5), 6.77%(4), 5.08%(3), 6.77%(4), 1.64%(1), 6.77%(4) respectively and incidence of luxation of hip in dogs less than one year old, one to two years, two to three years, three to four years, four to five years , five to six years and more than six years were 50%%(11), 9.09%%(2), 13.63%(3), 0%(0), 4.54%(1), 9.09%(2), 13.63%(3) respectively.

5. Physical examination and standard ventro dorsal radiograph of hip was found to be adequate in determining the limb which had coxo-femoral luxation/subluxation in hip dysplastic dogs.

6. Atropine and Diazepam were useful in reducing salivation and enhancing muscular relaxation respectively during anaesthesia for the surgical procedure in dogs. Induction of Anaesthesia with Thiopental sodium followed by maintenance with Isoflurane was satisfactory for the present study.

7. Percutaneous transfemoro-acetabular nylon zap Strap application for repair of coxofemoral luxation/subluxation could be performed without any difficulty in all cases of dogs suffering from hip dysplasia.

8. Post operative care without external immobilization helped the animals for early recovery and prevented muscles atrophy.

9. Post operative radiography up to 60^{th} day revealed the position of nylon zap strap within the femoral head and the acetabulum. None of the cases had relaxation.

10. Variations in levels of clinical, haematological and biochemical parameters like haemoglobin, packed cell volume, total erythrocyte count, total leukocyte count, differential leukocyte count, Serum creatinine, Serum alkaline phosphatise, serum aspartate aminotransferase and Serum alanine aminotransferase were found to be within the normal physiological range in all dogs during the study period.

11. Excellent and complete recovery was seen in all the 6 dogs (100%) in the present study.

In conclusion, the technique of Percutaneous transfemoro-acetabular nylon zap Strap application is a simple and efficient technique with high success rate for treatment of coxofemoral luxation/subluxation in dysplastic dogs in providing joint stability and reduces the pain.

Bibliography

VII. BIBLIOGRAPHY

AARNES, T. K., HUBBELL, J. A., HILDRETH, B. E., 2014.Use of sedation and rupivacaine-morphine epidural for femoral head and neck ostectomy in a dog. *J. Small Anim Pract.*, 55(**6**): 334-6

ADAMIAK, Z. 2012. Treatment of bilateral hip luxation in dogs with the Shani-Johnston-Shahar technique: case report. *Revue. Med. Vet.*,**163(2):**76-78

ADAMS, W.M., DUELAND, R.T. and DANIELS, R., 2000. Comparison of two palpation, four radiographic and three ultrasound methods for early detection of mild to moderate canine hip dysplasia. *Vet. Rad. Ultras.*, **41:** 484-490

AITHAL, H. P., SINGH, G. R. and BISHT, G. S., 1999. Fractures in dogs : a survey of 402 cases. *Indian J. Vet. Surg.*, **20** : 15-21

ALEXANDER, J. W., 1992. The pathogenesis of canine hip dysplasia. *Vet. Clin. North. Am. Small Anim. Pract.*, **22**: 503-511

ALLAN, G.S., 2000. Radiographic features of feline joint diseases. *Vet. Clin. North. Am. Small. Anim. Pract.*, **30:** 281-302

ANDERSON, A., 2011. Treatment of hip dysplasia. *J. Small. Anim. Pract.*, **52:** 182-189

ANTHONY, P. P., GIE, G. A., HOWIE, C. R., and LING, R. S. M. (1990).Localised endosteal bone lysis in relation to the femoral components of cemented total hip arthroplasties. J. Bone Joint Surg.72B, 971–979

ASH, K., ROSSELLI, D., DANIELSKI, A., FARRELL, M., HAMILTON, M. and FITZPATRICK, N. 2012.Correction of craniodorsalcoxofemoral luxation in cats and small breed dogs using a modified Knowles technique with the braided polyblend Tight Rope™ systems. *Vet . Comp. Orthop. Traumatol.*,**25**: 54–60.

BARTOLOME, N., SEGARRA, S., ARTIEDA, M., FRANCINO, O., SANCHEZ, E., SZCZYPIORSKA, M., CASELLAS, J., TEJEDOR, D., CERDEIRA, J., MARTINEZ, A., VELASCO, A. and SANCHEZ, A., 2015. A Genetic Predictive Model for Canine Hip Dysplasia: Integration of Genome Wide Association Study (GWAS) and Candidate Gene Approaches. doi:10.1371/journal. pone.0122558

BEALE, B. S., LEWIS, D. D. and PARKER, R. B. 1991. Ischio-ilial pinning for stabilization of coxofemoral luxations in 21 dogs: a retrospective evaluation. *Vet Comp. Orthol. Traumatol.*, **38**:28-34

BELFIELD, W.O., 1976. Chronic subclinical scurvey and canine hip dysplasia. *Vet.Med. Small Anim. Clin.*, **74:** 1399-1401

BENNETT, D. and DUFF, S. R. (1980).Transarticular pinning as a treatment for hip luxation in the dog and cat.*J. Sm. Anim. Pract.*, **21**:373-379.

BENONI, G., JOHNELL, O.N. and ROSBERG, B., 1984. Postoperative course of serum aminotransferases after total hip arthroplasty. *Acta. Anaesthesiol. Scand.*, **28**:362-366

BERGH, M.S. and BUDSBERG, S.C., 2014. A systematic review of the literature describing the efficacy of surgical treatments for canine hip dysplasia. *Vet.Surg.*, **43:** 501–506

BERZON, J.L., HOWARD, P.E., COVELL, S.J., TROTTER, E.J. and DUELAND, R., 1980. A retrospective study of the efficacy of femoral head and neck excisions in 94 dogs and cats. *Vet. Surg.*, **9:** 88-92

BIABECKI, J., MAJCHRZYCKI, M., SZYMCZAK, A., KLIMOWICZ-BODYS, M.D., WIERZCHOV, E. and KOBOMECKI, K., 2014. Hip Joint Replacement Using Monofilament Polypropylene Surgical Mesh: An Animal Model. *Bio. Med. Res. Int.*, http://dx.doi.org/10.1155/2014/187320

BO, N., PENG, W. and XINGHONG, P., 2012. Early cartilage degeneration in a rat experimental model of developmental dysplasia of the hip. *Connect. Tis. Res.*, **53**(6): 513–520

BOWEN, J.M., LEWIS, R.E., KNELLER, S.K., WILSON, R.C. and ARNOLD, R.A., 1972. Progression of hip dysplasia in German shepherd dogs after unilateral pectineal myotomy. *J. Am. Vet. Med. Assoc.,* **161:** 899-904

BRADEN, T.D., PRIEUR, W.D. and KANEENE, J.B., 1990. Clinical evaluation of intertrochanteric osteotomy for treatment of dogs with early-stage hip dysplasia: 37 cases (1980-1987). *J. Am. Vet. Med. Assoc*., **196:** 337-341

BRASS, W., 1989. Hip dysplasia in dogs. *J. Small. Anim. Pract.,* **30:** 166–170

BRINKER, W. O., PIERMATTEI, D. L. and FLO, G. L., 1990.Diagnosis and treatment of orthopedic conditions of the hindlimb, in handbook of small animal orthopedics and fracture treatment (Ed 2). Philadelphia, PA, Saunders, *pp* 342-355

BURHARDT, D. and GHOSH, P., 1987. Laboratory evaluation of antiarthritic drugs as potential chondroprotective agents. *Sem. Art. Rheu.,* **17:** 3-34

CANDACE E. LAYTON, and H. RODNEY FERGUSON. 1987. Lameness associated with coxofemorai soft tissue masses in. six dogs. *Vet Surg*., **16**:21-24.

CARDINET, G.H., GUFFY, M.M. and WALLACE, L.J., 1974. Canine hip dysplasia; effects of pectineal tenotomy in the coxofemoral joints of German shepherd dogs. *J. Am. Vet. Med. Assoc.,* **164:** 591-598

CARMICHAEL, S., WHEELER, S. J. and VAUGHAN L.C., 1989**.** Single condylar fractures of the distal femur in the dog. *J. Small Anim. Pract.,* **30**:500–4.

CETINKAYA, M. A. and OLCAY, B. 2010. Modified knowles toggle pin technique with nylon monofilament suture material for treatment of two caudoventral hip luxation cases. *Vet. Comp. Orthop. Traumatol.*, **23**:114-118.

CHALMAN, J.A. and BUTLER, H.C., 1985. Coxofemoral joint laxity and the Ortolani sign. *J. Am. Anim. Hosp. Assoc.*, **21:** 671–676

CHALMERS, H.J., DYKES, N.L. and LUST, G., 2006. Assessment of bone mineral density of the femoral head in dogs with early osteoarthritis. *Am. J. Vet. Res.*, **67:** 796–800

CHASE, K., LAWLER, D. F., ADLER, F. R., OSTRANDER, E. A. and LARK, K.G., 2004. "Bilaterally asymmetric effects of quantitative trait loci (QTLs): QTLs that affect laxity in the right versus left coxofemoral (hip) joints of the dog (Canisfamiliaris),".*Amer. Jour. Med. Genet.,* **124**(3): 239–247

CITI, S., VIGNOLI , M., RONI , F. and MORGAN, J.P. 2005. A radiological study of the incidence of unilateral canine hip dysplasia. *Schwieiz. Arch . Tierheilkd.*, **147**: 173 -178

COLES, E. H. 1974. *Veterinary Clinical Pathology*.secondEdn, W. B. Saunders Company, Philiadelphia.

COMHAIRE, F.H., CRIEL, A.C.C., DASSY, C.A.A., GUEVAR, P.G.J., SNAPS, F.R., 2009. Precision, reproducibility, and clinical usefulness of measuring the Norberg angle by means of computerized image analysis. *Am. J. Vet. Res.*, **70:** 228–235

COOPMAN, F., VERHOEVEN, G., SAUNDERS, J., DUCHATEAU, L. and VAN BREE, H., 2008. Prevalence of hip dysplasia, elbow dysplasia and humeral head osteochondrosis in dog breeds in Belgium. *Vet. Rec.,* **163:**654–658

CRAWFORD, R.D. and PHARR, J.W., 1975. Recommendations on canine hip dysplasia - diagnosis, treatment and genetic select. *Can. Vet. J.,* **16**(10): 308-309

CUERVO, B., RUBIO, M. and SOPENA, J., 2014. Hip osteoarthritis in dogs: a randomized study using mesenchymal stem cells from adipose tissue and plasma rich in growth factors. *Int. J. Mol. Sci.*, **15:** 13437–13460

DAVID SPRANKLIN, STEVEN ELDER, CAROLYN BOYLE and RON MCLAUGHLIN, 2006. Comparison of a Suture Anchor and a toggle rod for use in toggle pin fixation of coxofemoral luxations. *J. Am. Anim. Hosp. Assoc.*, **42**:121-126

DENNIS, R., 2012. Interpretation and use of bva/kc hip scores in dogs. *Practice.*, **34:** 178-194

DENNY, H. R. and BUTTERWORTH, S. J., 2000.A guide to canine and feline orthopaedic surgery (Ed. 4). – Oxford, PA Blackwell Science, *pp*. 445–467.

DEVINE, T. and SLOCUM, B., 1995. Results of femoral neck lengthening procedure in 75 dogs (abstract). Proc. *Annu. Meet. Vet. Orthop. Soc.*, **3**:123-127

DUELAND, R.T., ADAMS, W.M., FIALKOWSKI, J.P., PATRICELLI, A.J., MATHEWS, K.G. and NORDHEIM, E.V., 2001. Effects of pubic symphysiodesis in dysplastic puppies. *Vet. Surg.*, **30:** 201–217

DUELAND, R.T., ADAMS, W.M., PATRICELLI, A.J., LINN, K.A. and CRUMP, P.M., 2010. Canine hip dysplasia treated by juvenile pubic symphysiodesis. Part I: two year results of computed tomography and distraction index. *Vet. Comp. Orthop. Traumatol.*, **23:** 306–317

EVANS, H. E and CHRISTENSEN, G. C. 1979. MILLRS anatomy of the dog. Second edition. W.B. saunders, philadephia. pp. 254-257, 374-392, 1006-1022.

FARESE, J.P., TODHUNTER, R.J., LUST, G., WILLIAMS, A.J., DYKES, N.L., 1998. Dorsolateral subluxation of hip joints in dogs measured in a weight-bearing position with radiography and computed tomography. *Vet. Surg.*, **27:** 393–405

FARRELL, M., CLEMENTS, D.N and MELLOR, D., 2007. Retrospective evaluation of the long-term outcome of non-surgical management of 74 dogs with clinical hip dysplasia. *Vet. Rec.*, **160:** 506–511

FAZILI, M. R., CHAWLA, S.K., SINGH, J., TAYAL, R. and BEHL, S.M., 2008. Behavioural alterations due to pain and analgesic role of Meloxicam and Rofecoxib in dogs undergoing long bone fracture repair. *Indian J. Vet. Surg.*, **29**: 77-81

FELS, L. and DISTL, O., 2014. Identification and validation of quantitative trait loci (QTL) for canine hip dysplasia (CHD) in German shepherd dogs**.** *PLoS. One.* **(9):**e96618

FILIPPO, M. M., SIMONAZZI and MAURIZIO, D. B., 2001.Extra-articular absorbable suture stabilization of coxofemoral luxation in dogs. *Vet Surg.* **30:** 468-475

FITZPATRICK, N., LAW, A.Y, BIELECKI, M. B. and GIRLING, S., 2014. Cementless total hip replacement in 20 juveniles using BFX™ arthroplasty. *Vet. Surg.*, **43:** 715–725

FLUCKIGER, M., 2007. Scoring radiographs for canine hip dysplasia - The big three organisations in the world. *Euro. J. Comp. Anim. Pract.*, **17**(2): 135-140

FLUCKIGER, M.A., FRIEDRICH, G.A. and BINDER, H., 1999. A radiographic stress technique for evaluation of coxofemoral joint laxity in dogs. *Vet. Surg.*, **28**(1): 1-9

FOSSUM, T. W. 1997. *Small Animal Surgery.* 1stEdn**.** Elsevier, Milano. 1316-1324.

FOX, S.M., BUMS, J. and BURT, J., 1987. The dysplastic hip: a crippling problem in dogs. *Vet. Med.,* **82:** 684-693

FRIEDENBERG, S.G, ZHU, L. and ZHANG, Z., 2011. Evaluation of a fibrillin 2 gene haplotype associated with hip dysplasia and incipient osteoarthritis in dogs. *Am. J. Vet. Res.*, **72**(4): 530–540

FRIES, C. and REMEDIOS, A., 1995. The pathogenesis and diagnosis of canine hip dysplasia: a review. *Can. Vet. J.*, **36:** 494–502

FROST, H.M., 1989. Pathogenesis of congenital hip dysplasia (CDH): A proposal. *Vet. Comp. Orthop. Trauma.*, **1:** 1-10

FRY, T.R. and CLARK, D.M., 1992. Canine hip dysplasia: clinical signs and physical diagnosis. *Vet. Clinc. N. Am. Anim.: Small An. Pract.*, **22:** 551-558

FUJIKI, M., KURIMA, Y., YAMANOKUCHI, K., MISUMI, K. and SAKAMOTO, H., 2007. Computed tomographic evaluation of growth-related changes in the hip joints of young dogs. *Am. J. Vet. Res.*, **68:**730–734

GEMMILL, T.J., PINK, J. and RENWICK, A., 2010. Hybrid cemented/cementless total hip replacement in dogs: seventy-eight consecutive joint replacements. *Vet. Surg.*, **40:** 621–630

GENEVOIS, J. P., REMY, D., VIGUIER, E., CAROZZO, C., COLLARD, F., CACHON, T., MAITRE, P., FAU, D., 2008. Prevalence of hip dysplasia according to official radiographic screening, among 31 breeds of dogs in France. *Vet Comp. Orthol. Traumatol.*,**21**: 21–24

GERSCOVICH, E.O.,1997. A radiologist's guide to the imaging in the diagnosis and treatment of developmental dysplasia of the hip and General considerations, physical examination as applied to real-time sonography and radiography. *Skeletal Radiol.*, **26**(7): 386–397

GINJA, M., GASPAR, A.R. and GINJA, C., 2015. Emerging insights into the genetic basis of canine hip dysplasia. *Vet. Med. Res. Rep.*, **6:**193–202

GINJA, M.M., GONZALO-ORDEN, J.M. and MELO-PINTO, P., 2008. Early hip laxity examination in predicting moderate and severe hip dysplasia in Estrela mountain dog. *J. Small. Anim. Pract.*, **49**(12): 641–646

GINJA, M.M.D., FERREIRA, A.J. and JESUS, S.S., 2009. Comparison of clinical, radiographic, computed tomographic and magnetic resonance imaging methods for early prediction of canine hip laxity and dysplasia. *Vet. Radiol. Ultrasound.*, **50:**135–143

GINJA, M.M.D., SILVESTRE, A.M., GONZALO-ORDEN, J.M. and FERREIRA, A.J., 2010. Diagnosis, genetic control and preventive management of canine hip dysplasia: a review. *Vet. J.*, **184:** 269–276

GOPALKRISHNA, B. L. 1993. Extra-articular sling stabilization for repair of hip dislocation in experimental dogs. *M.V.Sc. thesis,* University of Agricultural Sciences, Bidar, India.

GREENE, M. L., DENIS, J. M. and DUNCAN, B. L. 2013. Associations among exercise duration, lameness severity and hip joint range of motion in Labrador retrievers with hip dysplasia. *J. Am. Vet. Med. Assoc.*, 242: 1528-1533.

GREG, H., 2004. The femoral head and neck ostectomy. *Can. Vet. J.*, **45**: 163-164

GRESHAKE, R. J. and ACKERMAN, N., 1992. Ultrasound evaluation of the coxofemoral joints of the canine neonate. *Vet. Ultras.*, **33:** 99-104

HAAN, J.J., VEALE, B.S. and PARKER, R.B., 1993. Diagnosis and treatment of canine hip dysplasia. *Can. Pract.*, **18:** 24-28

HEDHAMMER, A., OISON, S.E. and ANDERSON, S.A., 1979. *J. Am. Vet. Med. Assoc.*, **174:** 1012

HENRICSON. B., LJUNGGREN, G. and OLSSON, S.E., 1972. Canine hip dysplasia in Sweden: Incidence and genetics. *Acta. Radiol. Suppl.*, **319:** 175-180

HENRY, G.A., 1992. Radiographic development of canine hip dysplasia. *Vet. Clin. North. Am. Small. Anim. Pract.,* **22**(3): 559-578

HEO, S.Y., SEOL, J.W. and LEE, H.B., 2015. Total hip replacement in two dogs with unsuccessful femoral head ostectomy. *J. Vet. Sci.*, **16**(1): 131-134

HIELM-BJÖRKMAN, A.K., KUUSELA, E. and LIMAN, A., 2003. Evaluation of methods for assessment of pain associated with chronic osteoarthritis in dogs. *J. Am. Vet. Med. Assoc.*, **222:** 1552–1558

HOHN, R.B. and JANES, J.M., 1969. Pelvic osteotomy in the treatment of canine hip dysplasia. *Clin. Orthop.*, **62:** 70-78

HOSGOOD, G. and LEWIS, D.D., 1993. Retrospective evaluation of fixation complications of 40 pelvic osteotomies in 36 dogs. *J. Small. Anim. Pract.*, **34:** 123-130

HOU, Y., WANG, Y., LUST, G., ZHU, L., ZHANG, Z. and TODHUNTER, R.J., 2010. Retrospective analysis for genetic improvement of hip joints of cohort Labrador Retrievers in the United States: 1970–2007. *PLoS ONE*, **5**(2): e9410

HUTT, F.B., 1967. Genetic selection to reduce the incidence of hip dysplasia in dogs. *J. Am. Vet. Med. Assoc.*, **151:** 1041

IMPELLIZERI, J.A, TETRICK, M.A. and MUIR, P., 2001.Effect of weight reduction on clinical signs of lameness in dogs with hip osteoarthritis. *J. Am. Vet. Med. Assoc.*, **216:** 1089–1091

IREIFEJ, S., MARINO, D. and LOUGHIN, C., 2012. Nano total hip replacement In 12 dogs *Vet. Surg.*, **41**(1): 130–135

JAEGER, G.T., LARSEN, S., SOLI, N. and MOE, L., 2007. Two years follow-up study of the pain-relieving effect of gold bead implantation in dogs with hip-joint arthritis. *Acta. Vet. Scand.*, **49:** 9

JAYAPRAKASH, R., DHANALAKSHMI, N., SURESHKUMAR, R and GANESH. T.N. 2007. A retrospective study on incidence of canine hip dysplasia. *Ind. Vet. J.*, **84:** 519-520

JOHNSTON, S. A., 1992.Conservative and medical management of hip dysplasia. *Vet. Clin North. Am. Smal.l Anim. Pract.*, **22**(3): 595-606

JONES, R.D., BAYNES, R.E. and NIMITZ, C.T., 1992.Nonsteroidal anti-inflammatory drug toxicosis in dogs and cats: 240 cases (1989-1990). *J. Am .Vet. Med. Assoc.*, **201:** 475-47

KADERLY, R. E., ANDERSON, W. D. and ANDERSON, B. G. 1982. Extraosseous vascular supply to the mature dog´s coxofemoral joint. *Am. J. Vet. Res.*, **43**: 1208-1214

KANEENE, J.B., MOSTOSKY, U.V. and MILLER, R., 2009. Update of a retrospective cohort study of changes in hip joint phenotype of dogs evaluated by the OFA in the United States, 1989–2003. *Vet. Surg.*, **38**(3): 398–405

KANEENE, J.B., MOSTOSKY, U.V. and PADGETT, G.A., 1997. Retrospective cohort study of changes in hip joint phenotype of dogs in the United States. *J. Am. Vet. Med. Assoc.*, **211**(12): 1542-1544

KEALY, J.K., 1987. Diagnostic Radiology of dog and cat. 2nd Ed.W. B. Saunders Co., Philadelphia **p**.352.

KEALY, R. D., LAWLER, D. F. and MONTI, K. F., 1993. Effects of dietary electrolyte balance on subluxation of the femoral head in growing dogs. *Am. J. Vet. Res.*, **54**: 555-562

KEALY, R.D., LAWLER, D.F. and BALLAM, J.M., 2000. Evaluation of the effect of limited food consumption on radiographic evidence of osteoarthritis in dogs. *J. Am. Vet. Med. Assoc.*, **217**(11): 1678–1680

KELLER, G. G., REED, A. L., LATTIMER, J. C and CORLEY, A., 2000. Hip dysplasia : a feline population study. *Vet. Radiol. Ultra.*, **40**(5): 460-464

KIMELI, P., MBUGUA, S.W., CAP, R.M., KIRUI, G., ABUOM, T.O., MWANGI, W.E., KIPYEGON, A.N. and MANDE, J.D., 2015. A retrospective study on findings of canine hip dysplasia screening in Kenya. *Vet. World.,* **8**(11): 1326-1330

KIRKBY, K.A. and LEWIS, D.D., 2012. Canine hip dysplasia: reviewing the evidence for nonsurgical management. *Vet. Surg.*, **41:** 2–9

KRONTVEIT, R. I., ANE, N., BENTE, K. S., ERIK, R., HEGE, K. S. and CATHRINE, T., 2010. Prospective study on Canine Hip Dysplasia and growth in a cohort of four large breeds in Norway (1998–2001). *J. Prev. Vet. Med.,* **97**: 252–263

LAVRIJSEN, I.C.M., HEUVEN, H.C.M., MEIJ, B.P., THEYSE, L.F.H., NAP, R.C., LEEGWATER, P.A.J. and HAZEWINKEL, H.A.W., 2014. Prevalence and co-occurrence of hip dysplasia and elbowdysplasia in Dutch pure-bred dogs. *Pre. Vet. Med.,* **114:**114–122

LEDECKY, V., SEVCIK, A., PUZDER, M., CAPIK, I., HLUCHY, M., TRBOLOVA, A., NECAS, A. 2004. occurrence of hip joint dysplasia in some hunting dog breeds. *Vet. Arhiv.,* **74:** 417-425.

LEIGHTON, E., 1997. Genetics of canine hip dysplasia. *J. Am. Vet. Med. Assoc.,* **210**(10): 1474-1479

LEWIS, T.W., BLOTT, S.C. and WOOLLIAMS, J.A., 2010. Genetic Evaluation of Hip Score in UK Labrador Retrievers., *Journal. Pone,* 5 (10) e12797; doi:10.1371/. 0012797

LEWIS, T.W., ILSKA, J.J., BLOTT, S.C. and WOOLIAMS, J.A., 2011. Genetic evaluationof elbow scores and the relationship with hip scores in UK Labrador Retrievers. *Vet. J.,* **189:** 227–233

LIPOWITZ, A.J., 1993. Degenerative joint disease. *In:* Slatter D, ed. Textbook of Small Animal Surgery, 2nd ed. Philadelphia: WB Saunders., ***pp*** 1921-1927.

LUST G. Other orthopedic diseases: hip dysplasia in dogs. In: Slatter D, ed. Textbook of Small Animal Surgery, 2nd ed., vol 2. Philadelphia: WB Saunders, 1993: 1938-1944.

LUST, G. and SUMMERS, B. A., 1981. Early asymptomatic stage of degenerative joint disease in canine hip joints. *Am. J. Vet. Res.*, **42**: 1849-1855

LUST, G., BEILMAN, W. and DUELAND, D., 1980. Intra articular volume and hip joint instability in dogs with hip dysplasia. *J. Bone. Joint. Surg. Am.,* **62:** 576

LUST, G., GEARY, J.C. and SHEFFY, B.E., 1973.Development of hip dysplasia in dogs. *Am. J. Vet. Res.,* **34:** 87-91

LUST, G., RENDANO, V. T. and SUMMERS, B. A., 1985. Canine hip dysplasia: concepts and diagnosis. *J. Am. Vet. Med. Assoc.*, **187**: 638-640

LUST, G., WILLIAMS, A.J., BURTON-WURSTER, N., 1993. Joint laxity and its association with hip dysplasia in Labrador Retievers. *Am. J. Vet. Res.*, **54:** 1990-1999

LUST, G., WILLIAMS, A.J., BURTON-WURSTER, N., BECK, K.A. and RUBIN, G., 1992. Effects of intramuscular administration of glycosaminoglycan polysulfates on signs of incipient hip dysplasia in growing pups. *Am. J. Vet. Res.,* **53:** 1836-1843

MADSEN, J.S., JENSEN, L.T., STROM, H., HORSLEV-PETERSEN, K. and SVALASTOGA, E., 1993. Procollagen type-III aminoterminal peptide in serum and synovial fluid of dogs with hip dysplasia and coxarthrosis. *Am. J. Vet .Res.,* **51:** 1544-1546

MAKI, K., LIINAMO, A.E. and OJALA, M., 2000. Estimates of genetic parameters for hip and elbow dysplasia in Finnish Rottweilers. *J. Anim. Sci.*, **78**(5): 1141–1148

MALIK, H.N., DUBEY, A. and SINGHAL, D.K., 2013. Isolation, characterization, and differentiation of adipose tissue derived mesenchymal stem cells: an autologous transplantation to patients. *Repro. Fert. Dev.*, **26:** 216

MALM, S., STRANDBERG, E., DANELL, B., AUDELL, L., SWENSON, L. and HEDHAMMAR, A., 2007.I mpact of sedation method on the diagnosis of hip and elbow dysplasia in Swedish dogs. *Prev. Vet. Med.*, **78:** 196–209

MANLEY, P.A., ADAMS, W.M., DANIELSON, K.C., DUEL, R.T. and LINN, K.A., 2007. Long-term outcome of juvenile pubic symphysiodesis and triple pelvic osteotomy in dogs with hip dysplasia. *J. Am. Vet. Med. Assoc.*, **230**(2): 206-210

MARGARET, E., JOHNSON and TERRANCE D. BRADEN. 1987. A Retrospective Study of Prosthetic Capsule Technique for the Treatment of Problem Cases of Dislocated Hips. *Vet. Surg.*, **16**: 346-351.

MARINO, D.J., IREIFEJ, S.J. and LOUGHIN, C.A., 2012. "Micro total hip replacement in dogs and cats," *Vet. Surg.*, **41**(1): 121–129

MARTIN, S.W., KIRBY, K. and PENNOCK, P. W., 1980. Canine hip dysplasia: breed effects. *Can. Vet. J.*, **21:** 293-296

MARTINI, F. M., SIMONAZZI, B. and DELBUE, M., 2001.Extra-articular absorbable suture stabilization of coxofemoral luxation in dogs. *Vet. Surg.*, **30:** 468–475

MARX, C., SILVEIRA, M.D., SELBACH, I., SILVA, A.S.D., BRAGA, L.M.G.D.M., CAMASSOLA, M. and NARDI, N.B., 2014. Acupoint Injection of Autologous Stromal Vascular Fraction and Allogeneic Adipose-Derived Stem Cells to Treat Hip Dysplasia in Dogs., DOI.10.1155/391274

McCARTNEY, W., KISS, K. and MCGOVERN, F. 2011. Short Communications: Treatment of 70 dogs with traumatic hip luxation using a modified transarticular pinning technique. *Vet. Rec.*, **168:**355.

MLACNIK, E., BOCKSTAHLER, B.A., MÜLLER, M., TETRICK, M.A., NAP, R.C. and ZENTEK, J., 2006. Effects of caloric restriction and a moderate or intense physiotherapy program for treatment of lameness in overweight dogs with osteoarthritis. *J. Am. Vet. Med. Assoc.*, **229:** 1756–1760

MOLANO, S.E., WONG, P.R., CLEMENTS, D.N., BLOTT, S.C., WIENER, P. and WOOLLIAMS, J.A., 2015. Genomic prediction of traits related to canine dysplasia. *Front. Genet.*, **6:** 97

MOSKOWITZ, R.W., DAVIS, W., SAMMARCO, J., MAST, W. and CHASE, S.W., 1980. Experimentally induced corticosteroid arthropathy. *Art. Rheu.*, **13:** 236-243

NAGARAJA, B. N., 1994. Plastic implants for femoral fractures in experimental dogs. *M.V.Sc. thesis,* University of Agricultural Sciences, Bangalore, India.

NOUH, S.R., HOYDA, M.A., HAITHEM, A.F. and MOHAMED, M.S., 2014. A retrospective study on canine hip dysplasia in different breeds in Egypt. *Glob. Vet.*, **13**(4): 503-510

O'BRIEN, R.T., DUELAND, R.T., ADAMS, W.C. and MEINEN, J., 1997. Dynamic ultrasonographic measurement of passive coxofemoral joint laxity in puppies. *J. Am. Anim. Hosp. Assoc.*, **33**(3): 275–28

OFF, W. and MATIS, U., 2010. Excision arthroplasty of the hip joint in dogs and cats. Clinical, radiographic, and gait analysis findings from the Department of Surgery, Veterinary Faculty of the Ludwig-Maximilians-University of Munich, Germany. *Vet. Comp. Orthop. Trau.*, **23:** 297-305

OLSSON, S., HEDHANNER, A. and LASSHAM, H., 1979. Hip dysplasia and osteochondrosis in the dog. *Neth. Small. Anim. Vet. Assoc.*, **18:** 70-72

OLSSON, S.E., 1971. Physical evaluation and selection of military dogs. *J. Am. Vet. Med. Assoc.,* **159:** 1444-1446

OLSSON, S.E., 1978. Osteochondrosis in Domestic Animals. *Acta. Radiol. Suppl.,* **358:** 299-305

OZAYDIN, I., KILICE, B. and DEMIRKAN, I., 2003.Reduction and stabilization of hip luxation by transposition of the ligamentum sacrotuberale in dogs: an in vivo study. *Vet. Surg*., 32(**1**): 46-51

PAI, V.S. and KUMAR. 1990. Management of unreduced traumatic posterior dislocation of the hip: heavy traction and abduction method. *Brit. J. Accid. Surg*., **21**:225-227.

PALMOSKI, M.J. and BRANDT, K.D., 1979. Effect of salicylate on proteoglycan metabolism in normal canine articular cartilage in vitro. *Art. Rheum.,* **22:** 746-754

PALMOSKI, M.J., COLYER, R.A. and BRANDT, K.D., 1980. Marked suppression by salicylate of augmented proteoglycan synthesis of osteoarthritic cartilage. *Art. Rheu.,* **23:** 83-91

PANIGRAHI, P.N., 2014. Conservative treatment of hip dysplasia in a Labrador dog - A case study. *Vet. Res. Int.*, **2**(4): 105-107

PATRICELLI, A.J., DUELAND, R.T., ADAMS, W.M., FIALKOWSKI, J.P., LINN, K. A. and NORDHEIM, E.V., 2002. Juvenile pubic symphysiodesis in dysplastic puppies at 15 and 20 weeks of age. *Vet. Surg*., **31:** 435–444

POLLMEIER, M., TOULEMONDE, C., FLEISHMAN, C. and HANSON, P.D., 2006. Clinical evaluation of firocoxib and carprofen for the treatment of dogs with osteoarthritis. *Vet. Rec*., **159:** 547–551

RAGHUVIR, H.B., SHIVRAJSINH, K. J., DIPAK, N.S., HARIT, D.B., CHIRAG A. B. and NARESH, H. K., 2013. Treatment of Canine Hip Dysplasia: A Review. *J. Anim. Sci. Adv.,* **3**(12): 589-597

RANGANATH, L. and SUBIN, K., 2006. Hip scoring in dogs. *Ind. J. Vet. Surg*., 27(**2**): 116- 118

RAUSCH, S.F., MENDES, T.C. and CARAPETO, L.P., 2004. Displasia coxofemoral felina – relato de caso. *Hora. Vet*., **23:** 56-57

RAWSON, E.A., ARONSOHN, M.G. and BURK, R.L., 2005. "Simultaneous bilateral femoral head and neck ostectomy for the treatment of canine hip dysplasia," *J. Amer. Ani. Hosp. Asso.*, **41**(3)**:** 166–170

RHODES, A.M.L. and CLARKE, N.M.P., 2014. A review of environmental factors implicated in human developmental dysplasia of the hip. *J. Child. Orthop.,* **8:** 375–379

RIALLAND, P., BICHOT, S. and LUSSIER, B., 2013. Effect of a diet enriched with green-lipped mussel on pain behaviour and functioning in dogs with clinical osteoarthritis. *Can. J. Vet. Res*., **77:** 66–74

RISER, W.H, RHODES, W.H. and NEWTON, C.D., 1985. Hip dysplasia. *In:* Textbook of Small Animal Orthopedics. Newton CD, Nunamaker DM, eds. Philadelphia: JB Lippincott., ***pp*** 953-980.

RISER, W.H., 1975. The dog as a model for the study of hip dysplasia. *Vet. Pathol.,* **12:** 229-334

RISER, W.H., 1993. Canine hip dysplasia, *In* Bojrab, M.J. Disease mechanism in Small Animal Surgery, 2nd Ed. Philadelphia. Lea and Febiger, ***pp*** 797.

RISLER, A., KLAUER, J.M., KEULER, N.S. and ADAMS, W.M., 2009. Puppy line, metaphyseal sclerosis, and caudolateral curvilinear and circumferential femoral head osteophytes in early detection of canine hip dysplasia. *Vet. Radiol. Ultrasound.*, **50:** 157–166

ROBERTS, T. and MCGREEVY, P.D., 2010. Selection for breed-specific long-bodied phenotypes is associated with increased expression of canine hip dysplasia. *Vet. J.*, **183:** 266–272

ROCHA, B.D. and TORRES, R.C.S., 2007. Ultrasonic and radiographic study of laxity in hip joints of young dogs. *Arq. Bras. Med. Vet. Zootec.,* **59**(1): 90-96

ROCHEREAU, P. and BERNARDE, A. 2012.Stabilization of coxofemoral luxation using tenodesis of the deep gluteal muscle.*Vet. Comp. Orthop. Traumatol.*,**25**: 49–53.

RON M. MCLAUGHLIN, J.R. and D. MICHAEL TILLSON, 1994. Flexible external fixation for craniodorsal coxofemoral luxations in dogs. *Vet. Surg.*, **23**:21-30

ROSE, S.A., BRUECKER, K.A., PETERSEN, S.W. and UDDIN, N., 2012. Use of locking plate and screws for triple pelvic osteotomy. *Vet. Surg.*, **41:** 114–120

SCHACHNER, E.R. and LOPEZ, M.J., 2015. Diagnosis, prevention, and management of canine hip dysplasia: a review. *Vet. Med. Res. Rep.,* **6:**181–192

SCHNELLE, G.B. 1935. Some new diseases in the dog. *Am Kennel Gaz.,* **52**: 25-31

SCHRADER, S.C., 1982. Triple osteotomy of the pelvis as treatment for canine hip dysplasia. *J. Am. Vet. Med. Assoc.,* **178:** 39-44

SHASHIDHARA, N. S., 1997. Modified toggle for repair of hip dislocation in experimental dogs. *M.V.Sc. thesis,* University of Agricultural Sciences, Bangalore, India.

SHEPHERD, J., 1986. Canine hip dysplasia: Aetiology, pathogenesis and eradication. *Aust. Vet. Pract.*, **16:** 71-78

SHIJU SIMON, M., GANESH, R., AYYAPPAN, S., RAO, G.D., SURESH KUMAR, R., MANONMANI, M AND DAS, B.C., 2010. Incidence of Canine Hip Dysplasia : A Survey of 272 Cases. *Vet. World.,* **3**(5): 219-220

SHIVAKUMAR, E. 2015. studies on percutaneous transfemoro-acetabular nylon wiring for traumatic hip luxation in companion animals. *M.V.Sc. thesis,* Karnataka Veterinary, Animal and Fisheries Science University, Bidar, India.

SINGH, K., KINJAVDEKAR, P., AITHAL, H. P., GOPINATHAN, P., AMARPAL, PAWDE, A. M. and SINGH, G. R., 2008.Comparison of dynamic compression plate with circular external skeletal fixator for correcting angular deformity after wedge osteotomy of canine antebrachium. *Ind. J. Vet. Surg.*, **29**:87-92.

SKURKOVA, L. and LEDECKY, V., 2009. Early diagnosis of canine hip dysplasia. *Folia. Vet.*, **53**(2): 77-82

SLOCUM, B. and DEVINE, T., 1990. Pathomechanics of hip dysplasia. *In:* Canine Orthopedics. *Edt.* WHITTICK, W.G., *Edn. 2nd.*, Philadelphia: Lea & Febiger., *pp* 471-481.

SMITH, C., 1992. Treatments for hip dysplasia spark controversy. *J. Am. Vet. Med. Assoc.*, **201:** 205-209

SMITH, G. K., 1998. Canine hip dysplasia: pathogenesis, diagnosis, and genetic control. *Vet. Quat.*, **20**: 22–24

SMITH, G. K., DARRYL, N. B and GREGOR, T.P. 1990. New concept of coxofemoral joint stability and the development of a clinical stress- radiographic method of quantitating hip joint laxity in a dog. *J. Am. vet. Med. Assoc.*, 196: 59-70.

SMITH, G.K., GREGOR, T.P., RHODES, W.H. and BIERY, D.N., 1993.Coxofemoral joint laxity from distraction radiography and its contemporaneous and prospective correlation with laxity, subjective score and evidence of degenerative joint disease from conventional hip-extended radiography. *Am. J. Vet. Res.*, **54:** 1021–1042

SMITH, G.K., PASTER, E.R. and POWERS, M.Y., 2006. Lifelong diet restriction and radiographic evidence of osteoarthritis of the hip joint in dogs. *J. Am. Vet. Med. Assoc.*, **229:** 690–693

SMITH, K. G., DENNIS , F. L., DARRYL, N. B., MICHELLE, Y. P., FRANCES, S., THOMAS, P. G., GEORGA , T. K., MISCHA , B., LYNCH, M., RICHARD, H. E. and RICHARD , D. K., 2012.Chronology of Hip Dysplasia Development in a Cohort of 48 Labrador Retrievers followed for life. *Vet Surg.,* **41**: 20–33

SMITHA, S. 2014. Studies on trans-articular wiring for repair of coxofemoral luxation in dogs. *M.V.Sc. thesis,* Karnataka Veterinary, Animal and Fisheries Science University, Bidar, India.

SNEDECOR, C. W. and COCHRAN, W. G., 1996.In : Statistical Analysis. 8thEdn.Oxford and IBH publishing co. New Delhi, pp. 335-345

STANIN, D., PAVLAK, M., VRBANAC, Z. and POTOCNJAK, D., 2011. Prevalence of hip dysplasia in dogs according to official radiographic screening in Croatia. *Vet. Arhiv.,* **81**(2): 235-248

STOCK, K.F., KLEIN, S., TELLHELM, B. and DISTL, O., 2011. Genetic analyses of elbow and hip dysplasia in the German shepherd dog. *J. Anim. Breed. Genet.,* **128** (3): 219–229

SUKHIANI, H.R., HOMBERG, D.L. and HURTIG, M.B., 1994. Pelvic canal narrowing caused by triple pelvic osteotomy in the dog. Part 1: The effect of pubic remnant length and angle of acetabular rotation. *Vet. Comp. Orthop. Trauma.,* **7:** 110-113

SWENSON, L., AUDELL, L., HEDHAMMAR, A. 1997. Prevalence and inheritance of and selection for hip dysplasia in seven breeds of dogs in Sweden and benefit: cost analysis of a screening and control program. *J.A.V.M.A.*, **(210):** 207-214.

SZABO, S.D., BIERY, D.N. and LAWLER, D.F., 2007. Evaluation of a circumferential femoral head osteophyte as an early indicator of osteoarthritis characteristic of canine hip dysplasia in dogs. *J. Am. Vet. Med. Assoc.*, **231:** 889–892

TODD, C. T., JEFFREY, N. PECK. and JACEK, J. DEHAAN. 2000. Spontaneous bilateral coxofemoral luxation in four dogs. *J. Am. Anim. Hosp. Assoc.*, **36**:268–76.

TODHUNTER, R. J AND LUST, G. 2003. Canine hip dysplasia: pathogenesis, pp. 2009-2019. *In* D. Slatter, ed. Textbook of Small Animal Surgery. W.B. Saunders, USA.

TODHUNTER, R. J., BLISS, S.P., CASELLA, G., **Wu, R**., LUST, G., BURTON-WURSTER, N. I., WILLIAMS, A. J., GILBERT, R.O. and ACLAND, G.M., 2003. Genetic Structure of Susceptibility Traits for Hip Dysplasia and Microsatellite Informativeness of an Outcrossed Canine Pedigree. *J. Her.*, **94**(1): 39–48

TORRES, R.C.S. and SILVA, E.F., 2001. Displasia coxofemoral em cães – parte II avaliação radiográfica. *Rev. CFMV.*, **8:** 36-40

VENZIN, C. and MONTAVON, P. M. 2007.Augmentation of the transverse acetabular ligament in canine caudoventral hip luxation.*Vet. Comp. Orthop. Traumatol.*,**20**: 320–323.

VEZZONI, A., DRAVELLI, G. and CORBARI, A., 2005. The early diagnosis of canine hip dysplasia. *E.J.C.A.P.*, **15**(2): 173–184

VILAR, J.M., BATISTA, M. and MORALES, M., 2014. Assessment of the effect of intraarticular injection of autologous adipose-derived mesenchymal stem cells in osteoarthritic dogs using a double blinded force platform analysis. *BMC. Vet. Res.*, **10:** 143

WADSWORTH, P. L. 1993. Biomechanics of the luxation of joints. In: Pathophysiology in small animal surgery. Bojrab MJ (ed). Philadelphia: W.B. Saunders. 1048–1057.

WALLACE, L.J., 1992. Pectineus tendon surgery for the management of canine hip dysplasia. *Vet. Clin. North Am. Small Anim. Pract.*, **3:** 607-621

WALLACE, M.S., ZAWIE, D.A. and GARVEY, M.S., 1990. Gastric ulceration in the dog secondary to the use of nonsteroidal anti-inflammatory drugs. *J. Am. Anim. Hosp. Assoc.*, **26:** 467-472

WILLARD, M.D. and TVEDTEN, H. 2012. *Small animal clinical diagnosis by laboratory methods.* 5th Edn. Elseviers Saunders, Missouri. Pp: 124

WILLIS, M.B., 1997.A review of the progress in canine hip-dysplasia control in Britain. *J. Am. Vet. Med. Assoc.*, **210:** 1480–1482

WILSON, B.J., NICHOLAS, F.W., JAMES, J.W., WADE,C.M., HERMAN, W., RAADSMA, H.W. and THOMSON, P.C., 2013 .Genetic Correlations among Canine Hip Dysplasia Radiographic Traits in a Cohort of Australian German Shepherd Dogs, and Implications for the Design of a More Effective Genetic Control Program**.** *PLoS. ONE.*, **8** (11): e78929

WOOD, J.L.N., LAKHANI, K.H. and ROGERS, K., 2002.Heritability and epidemiology of canine hip dysplasia score and its components in Labrador retrievers in the United Kingdom. *Vet Med.*, **55**: 95-108

YARDIMCI, C., OZAK, A. and NISBET, H.O., 2011Management of distal humeral fractures indogs with unilateral semicircular external skeletal fixators: prospective clinical trial and results in twelve cases. *Revue Méd. Vét.,* **162**: 613-620.fa

ZHOU, Z., SHENG, X. and ZHANG, Z., 2010. Differential genetic regulation of canine hip dysplasia and osteoarthritis. *PLoS. ONE.*, **5:**e13219.

ZHU, L., ZHANG, Z. and FRIEDENBERG, S., 2009. The long (and winding) road to gene discovery for canine hip dysplasia. *Vet. J.*, **181**(2): 97-110

Abstract

VIII. ABSTRACT

A study on Percutaneous Transfemoro-acetabular nylon zap strap application technique for the repair of coxofemoral subluxation/luxation in 6 hip dysplastic dogs was carried out in a period of one year. Totally 81 cases of hip dysplastic dogs were reviewed and occurrence of hip dysplasia was 0.51% and the coxofemoral subluxation and luxation were about 0.37% and 0.13% respectively in hip dysplastic dogs. The higest incidence of hip dysplasia was seen in Labrador retriever and coxofemoral subluxation and luxation were about 20.33% and 27.27% respectively. Males had a highest incidence of hip subluxation (72.88%) and luxation (63.63%) in hip dysplastic dogs. Coxofemoral subluxation and luxation in Hip dysplastic dogs was most frequently found in young and large breeds and highest incidence was seen in the age group less than one year was about 64.40% and 50% respectively. Percutaneous Transfemoro-acetabular nylon zap strap application was performed in six clinical cases of dogs and various parameters were studied. There was statistically non-significant variation in physiological parameters (rectal temperature, respiratory rate, and heart rate), hematological parameters (Hb, TEC, PCV, TLC, DLC) and biochemical parameters (ALT, AST, ALP and creatinine). The radiographic findings showed good opposition with femoral head and acetabulum and Weight bearing was graded as good at the first postoperative day and excellent on 30^{th} postoperative day onwards in dogs. No intra or post-operative complication associated with surgical technique was noticed. The technique was found to a simple, practical and innovative technique requires no specialized equipment and found to be very successful in stabilization of coxofemoral joint in hip dysplastic dogs and long term pain management.

Key word: Hip dysplasia, Transfemoro-acetabular, physiological, hematological, biochemical.