CLINICAL EVALUATION OF AFFECTIONS OF THE CANINE SHOULDER, ELBOW AND STIFLE JOINTS BY ARTHROSCOPY

MALA SHAMMI
DPV 98021 (SUR)

DEPARTMENT OF VETERINARY SURGERY AND RADIOLOGY
MADRAS VETERINARY COLLEGE
TAMIL NADU VETERINARY AND ANIMAL SCIENCES UNIVERSITY
CHENNAI - 600 051

2006

CLINICAL EVALUATION OF AFFECTIONS OF THE CANINE SHOULDER, ELBOW AND STIFLE JOINTS BY ARTHROSCOPY

MALA SHAMMI
DPV 98021 (SUR)

Thesis submitted in partial fulfilment of the requirement for the degree of

DOCTOR OF PHILOSOPHY
in
VETERINARY SURGERY AND RADIOLOGY

to the

TAMIL NADU VETERINARY AND ANIMAL SCIENCES UNIVERSITY
CHENNAI - 600 051

DEPARTMENT OF VETERINARY SURGERY AND RADIOLOGY
MADRAS VETERINARY COLLEGE
TAMIL NADU VETERINARY AND ANIMAL SCIENCES UNIVERSITY
CHENNAI - 600 051

2006

CERTIFICATE

This is to certify that the thesis entitled **"CLINICAL EVALUATION OF AFFECTIONS OF THE CANINE SHOULDER, ELBOW AND STIFLE JOINTS BY ARTHROSCOPY"** submitted in partial fulfilment of the requirements of the degree of **DOCTOR OF PHILOSOPHY** in **VETERINARY SURGERY AND RADIOLOGY** to the **TAMIL NADU VETERINARY AND ANIMAL SCIENCES UNIVERSITY,** Chennai - 600 051, is a bonafide research work carried out by **MALA SHAMMI, DPV 98021 (SUR)** under my supervision and guidance and that no part of this thesis has been submitted for the award of any other degree, diploma, fellowship or other similar titles or prizes and that the work has not been published in part or full in any scientific journal or popular journal or magazine.

29/01/06

Place : Chennai – 7.
Date : 29/01/06

Dr.R.SURESH KUMAR, Ph.D.,
(Chairman)

APPROVED

CHAIRMAN:

02/05/06

Dr.R.SURESHKUMAR, Ph.D.,

MEMBERS:

1. **Dr. K.AMEERJAN, Ph.D.,** 02/5/06.
2. **Dr. B. MURALI MANOHAR, Ph.D.,** 2506
3. **Dr. S. PRATHABAN, Ph.D.,**

DATE : 2/5/06

EXTERNAL EXAMINER 2/5/06

Dr. K. RATANKUTTY
Assistant Professor
Dept. of Veterinary Surgery
College of Vety & Animal Sciences

CURRICULUM VITAE

Name of the candidate	:	**MALA SHAMMI**
Date of birth	:	**11.09.1960**
Place of birth	:	**Thane, Mumbai**
Major field of specialisation	:	**Veterinary Surgery and Radiology**
Educational status	:	**M.V.Sc.(Surgery)**
Professional status	:	**Assistant Professor (S.G), Department of Clinics, Madras Veterinary College, Tamil Nadu Veterinary and Animal Sciences University Chennai 600 007, Tamil Nadu**
Marital status	:	**Married**
Permanent Address	:	**AA2, Tarangini Apartments, Mogappair West, Chennai - 600 058**
Publications	:	**Eight**
Membership of professional Societies	:	**1. Indian Society for Veterinary Surgery 2. Tamil Nadu Veterinary Council**

ACKNOWLEDGEMENT

I am extremely grateful and deeply indebted to **Dr.R.Sureshkumar, Ph.D.,** Professor and Head, Department of Veterinary Surgery and Radiology, Madras Veterinary College and Chairman of the Advisory Committee, for his meticulous guidance, unstinted moral support and tremendous understanding, without which this work would not have been possible.

I am sincerely thankful to **Dr.K.Ameerjan, Ph.D.,** Director, Directorate of Clinics, Madras Veterinary College and Member Advisory Committee for sharing his abundant experience, valuable suggestions, constructive criticism and help rendered during the study.

I am extremely grateful and deeply indebted to **Dr.B.Murali Manohar, Ph.D.,** Professor and Head, Department of Veterinary Pathology and Member, Advisory Committee, for the meticulous guidance, generous moral support, patient understanding and critical review of works done.

I am sincerely thankful to **Dr.S.Prathaban, Ph.D.**, Professor and Head, Department of Clinics, Madras Veterinary College and Member, Advisory Committee, for his valuable suggestions, constructive criticism and help rendered during the study.

I express my sincere gratitude to **Dr. B. Justin William, Dr. R.Jayaprakash, Dr.L.Nagarajan, Dr.G.D. Rao, Dr. K. Ramanujam, Dr.C.Ramani, Dr. Shafi** and **Dr. Jayakumar** for their valuable suggestions , moral support and help rendered during the period of study.

I express my sincere thanks to **Dr. R. Ganesh, Dr. T.N. Ganesh, Dr. Ravi Sunder George , Dr. S. Ayyappan ,** for their moral support and encouragement .

I express my gratitude to **Dr. L.N.Mathuram, Dr. R. Geetha, Dr. Cecilia Joseph,** and **Dr. Radhakrishnan** for their support throughout the period of study.

Special thanks is due to **Dr. J.Sophash** for the statistical analysis done for the study. I am deeply indebted to **Dr. R. Ramanathan** and **Dr. Puskinraj** for their invaluable help.

I am deeply indebted to **Dr. R. Uma Rani** who was with me through the loneliest hours of this work.

I thank the post graduate students of department of surgery for their help, co-operation and understanding through the period of study.

I express my heartfelt thanks to my husband **Shammi.R**, and my children **S. Parvesh Gautam** and **S. Karan Deepak** who put up with me and my idiosyncrasies over these long years.

A very special thanks is due to a group of people, who like a much needed storm, took over the course of my struggling ship and swept me to a safe shore.

Most importantly I express my most sincere gratitude to **Dr.R.Kathirvel,** Former Vice Chancellor, TANUVAS without whom I would never have had the opportunity to do my Ph.D.

I express my deepest gratitude to **Dr. P. Thangaraj, Ph.D.,** Dean, Madras Veterinary College for his understanding and kindness.

Last, but not the least I thank all the staff at **Student Xerox,** Kellys, for their patience and meticulous work in the preparation of the thesis.

Abstract

ABSTRACT

CLINICAL EVALUATION OF AFFECTIONS OF THE CANINE SHOULDER, ELBOW AND STIFLE JOINTS BY ARTHROSCOPY

Name	:	**MALA SHAMMI**
Degree of which submitted	:	Ph.D.,
Chairman	:	**Dr.R.SURESHKUMAR, Ph.D.,** Professor and Head, Department of Veterinary Surgery and Radiology, Madras Veterinary College, Chennai – 600 007.
University	:	Tamil Nadu Veterinary and Animal Sciences University, Chennai.
Year	:	2006

The efficacy and advantages of arthroscopy as a diagnostic aid was studied after clinical evaluation of shoulder, elbow and stifle joint affections. Thirty six dogs suffering from shoulder, elbow and stifle joint lameness were divided into three groups of 12 animals each and subjected to arthroscopic examination after physical, clinical and radiographic examination.

Physical examination revealed pain on hyperextension in all cases of shoulder joint lameness. Thigh muscle wastage was predominant in three dogs with stifle joint lameness. Clinical examination revealed higher grade of lameness in osteochondritis dissecans, ligament injury and synovitis.

Dogs below two years of age and dogs two-six years of age group showed, higher grade of lameness in all the three joint affections. Incidence of all the three joints affections were higher in large sized breed dogs. Males were mostly affected.

The haematological study revealed no significant changes between disease conditions and pre-and post-arthroscopically in the values of haemoglobin, packed cell volume, total erythrocyte count, total leukocyte count, neutrophils and lymphocytes. Significant decrease in erythrocyte sedimentation rate in osteochondritis dissecans was seen in both pre and post-arthroscopic period and in degenerative joint disease, ligament injury and synovitis during post arthroscopic period alone.

Biochemical study of the blood revealed significantly higher values of serum albumin in ligament injury and synovitis. The serum globulin level was higher in ligament injury and synovitis, which decreased post arthroscopically. Serum alkaline phosphatase was significantly higher in degenerative joint disease, which decreased during post arthroscopic period. No significant change in mean blood glucose level was found in the conditions studied and between pre and post-arthroscopic period.

No significant change in the mean volume of synovial fluid was noticed in the conditions studied pre-arthroscopically. However, significant reduction in volume was seen in degenerative joint disease, osteochondritis dissecans and synovitis during the post arthroscopic period.

Cytological examination of synovial fluid revealed significant decrease in mean leukocyte count in ligament injury, synovitis, osteochondritis dissecans and degenerative joint disease. Significant reduction in polymorphonuclear cells was seen in osteochondritis dissecans, degenerative joint disease and ligament injury during post arthroscopic period, whereas significant increase in mean mononuclear cells was noticed in degenerative joint disease, osteochondritis dissecans and ligament injury during post arthroscopic period.

Values of total protein, albumin, globulin level in synovial fluid were significantly reduced in ligament injury. Significant increase in alkaline phosphatase levels in synovial fluid was noticed in degenerative joint disease and osteochondritis dissecans pre-arthroscopically which decreased post-arthroscopically.

All the cases of degenerative joint disease, two cases of osteochondritis dissecans and two cases of cranial cruciate ligament rupture were diagnosed radiographically.

Arthroscopy of the shoulder joint lameness visualized cartilage lesions of osteochondritis dissecans like cartilage flaps, fissure, chondromalacia and lesions of degenerative joint disease, biceps tenditinitis, partial or complete tear of the biceps brachii, bicipital tenosynovitis and glenohumeral ligament rupture.

Arthroscopy of elbow joint lameness demonstrated fragmented coronoid process, osteochrondritis dissecans of humeral condyle, ununited anconeal process, articular anomaly and joint incongruity.

Arthroscopy of stifle joint lameness demonstrated osteochondritis dissecans of the femoral condyles, meniscal injuries, cruciate ligament injuries and degenerative lesions.

Contents

CONTENTS

List of Tables

LIST OF TABLES

List of Figures

LIST OF FIGURES

List of Plates

LIST OF PLATES

Introduction

CHAPTER – I

INTRODUCTION

Arthroscopy is a valuable diagnostic tool for detecting intra-articular joint derangements, which cannot be assessed by clinical examination and other routine diagnostic methods. The advantages of arthroscopy over conventional arthrotomy in the visualization of the interiors of joints are minimal invasiveness, early diagnosis, decreased pain, early return to function, good cosmetic result due to minimal scarring and most importantly, precise and accurate treatment with minimum post-operative care.

Although the concept of arthroscopy was first initiated by Tagaki in 1918 (Miller and Presnell, 1985 and Van Ryssen *et al.,* 2003), the first arthroscopic study in human beings was demonstrated by Watanabe *et al.* (1969). Veterinary arthroscopy was first performed in horses in 1970 (Hall and Keeran, 1975) and arthroscopy in the dog was first reported by Siemering (1978). Shoulder joint arthroscopy was first performed by Person (1985) for the diagnosis of osteochondrosis dissecans.

Arthroscopy offers many advantages over arthrotomy. Visualization of the joint is typically better, as it allows inspection of most of the areas of the joint through a single port that are not visible unless multiple arthrotomies are performed. Using arthroscopy as an exploratory procedure may avoid the need for an arthrotomy, especially in cases where a surgically treatable lesion is not obviously found.

Arthroscopy allows examination of intra-articular structures of the joints with minimal invasiveness and negligible morbidity with short recovery time as compared to exploratory arthrotomy. In addition, arthroscopy allows direct viewing of the intra-articular structures and their pathological changes. The considerable magnification coupled with the fluid irrigation of the joint makes it possible to appreciate the articular cartilage and synovium in detail as the synovial villi are suspended in the fluid irrigation medium and project into the joint cavity. Details of the synovial membrane and articular cartilage damage may not be readily apparent during arthrotomy as these lesions collapse and cling to the underlying surface. Repeated arthroscopic examination of a joint referred to as "second-look" arthroscopy can be done for evaluation of the progress of a pathological condition or the follow up of a treatment procedure as it involves minimal invasiveness of the technique, minimal post operative care and minimal post procedural complications.

Arthroscopy of the shoulder joint enables more detailed examination of the articular cartilage than positive arthrography and survey radiography. Arthroscopy demonstrates several types of cartilage lesions, cartilage flaps or fissure, lesions resembling chondromalacia and indentation. Various joint conditions that can be diagnosed and treated by arthroscopy in the shoulder are osteochondritis dissecans (OCD), degenerative joint disease (DJD), biceps tendinitis, partial or complete tear of the biceps brachii, bicipital tenosynovitis, joint mice, septic arthritis and partial or complete glenohumeral ligament rupture.

Elbow lameness caused by fragmented coronoid process, osteochondritis dissecans, ununited anconeal process, articular anomaly or joint incongruity is a major problem among young rapidly growing dogs. Early primary lesions of the elbow joint can be diagnosed by arthroscopy without radiographic signs with minimal trauma and without post procedural complications. In the elbow, indications for diagnostic or surgical arthroscopy include fragmented coronoid process (FCP), osteochondritis dissecans (OCD) of the humeral condyle, joint mice, arthritis and synovectomy.

Arthroscopy of the stifle joint is useful for the diagnosis and treatment of osteochondritis dissecans of the femoral condyles, precise evaluation and treatment of meniscal injuries, diagnosis and treatment of cruciate ligament tears or injuries and evaluation of primary and secondary degenerative lesions.

Currently, diagnostic and surgical arthroscopy remains as a challenging task in canine species. However, the superiority of arthroscopy in the diagnosis of the joint disorders especially in early stages and the increasing number of conditions which cannot be diagnosed by conventional methods has led to an increased awareness of the advantages of arthroscopy. Hence the present study was undertaken in canines with the following objectives.

1. To standardise the technique of arthroscopy of the canine shoulder, elbow and stifle joints.

2. To study the changes in the synovial fluid composition and haemato-biochemical parameters in joint affections in canines.

3. To clinically evaluate the affections of the shoulder, elbow and stifle joints of canines by arthroscopy.

Review of Literature

CHAPTER – II

REVIEW OF LITERATURE

2.1 History

Hall and Keeran (1975) were the first to describe the use of an arthroscope in horses and concluded the usefulness of arthroscopy in the diagnosis of joint pathology which radiography failed to show.

Siemering (1978) was the first to describe the arthroscopic findings of the canine stifle and found that arthroscopy was useful in the diagnosis of diseases of the canine stifle joint.

Kivumbi and Bennett (1981) examined 59 canine stifle joints, both normal and diseased, and described the anatomical structures visualized through the arthroscope. The authors concluded that arthroscopy was a potential diagnostic tool in the recognition of articular diseases of animals.

Person (1985) reported the technique of arthroscopy of the canine stifle, shoulder, and coxofemoral joints and described the first successful use of arthroscopy in the diagnosis and treatment of osteochondritis dissecans of the shoulder joint.

Van Ryssen *et al.* (2003) mentioned that the application of endoscopy to human joints was pioneered by Tagaki of Japan, who used a cystoscope to examine the human knee joint in an effort to improve the diagnosis of tuberculosis of the knee in the early 1900s. His success in this field led him to develop the first true arthroscope.

2.2 Biomechanics

2.2.1 Shoulder joint

Vasseur *et al.* (1982) studied the canine shoulder joint stability attributed by the tendons of the supraspinous, infraspinous, teres minor and subscapular muscles. The authors found that the joint capsule and the glenohumeral ligaments were equally important in maintaining joint stability along with the cuff muscles.

Bardet (1998) opined that glenohumeral stability of the shoulder joint resulted from a hierarchy of mechanisms, including those that do not require energy (passive mechanism) and those that do require energy (active mechanism). The author also reported that insertions of the "cuff" muscles were considered responsible for maintaining joint integrity and the joint capsule and collateral ligaments of the joint were found to play significant roles in stability.

Mitchell and Innes (2000) stated that canine gleno humeral joint was stabilized by several mechanisms broadly divided into active and passive. Passive stabilizing mechanisms included the lateral and medial gleno humeral ligaments on the lateral and medial surfaces of the joint respectively.

2.2.2 Elbow joint

Putz and Muller-Gerbl (1988) reported that the distribution of the hyaline articular cartilage and the density of the underlying bone were morphological reflections of the stresses acting upon the joint.

Thomsen *et al.* (2001) stated that the elbow was a very stable functional unit due to its bony tracking and self stabilising ability especially in the humero-radial joint.

2.2.3 Stifle joint

Arnoczky and Marshall (1977) studied 50 canine stifles to assess the anatomy and function of the cruciate ligaments. The authors found that cranial and caudal cruciate ligaments were composed of two component parts and their geometry of the femoral attachments were responsible for a reciprocal loosening and tightening of these components through a range of motion. The authors reported that transection of one or both of the cruciate ligaments resulted in joint instability.

Dejardin (2002) described the functional anatomy and biomechanics of the stifle joint in dogs. The cruciate and collateral ligaments were found to be the major static stabilizers of the stifle, although menisci, joint capsule and femoral musculature also contribute to joint stability. Normal stifle motion is a combination of flexion-extension about a medio-lateral axis with internal-external tibial rotation about a dorso-ventral axis. Hyperextension is prevented by progressive tensioning of the caudo-lateral band and to a lesser extent by cranial cruciate ligament impingement on the inter condylar notch and by the collateral ligaments. Internal tibial rotation occurs during flexion due to lateral collateral ligament relaxation and is limited by the twisting of the cruciate ligaments about one another. Because of its spatial orientation and anatomical structure, the cranial cruciate ligament also prevents cranial tibial translation throughout rotational outward movement (ROM). Cranial cruciate ligament

failure allows for hyperextension, excessive tibial internal rotation, and cranial tibial translation. Such abnormal movements cannot be limited by secondary constraints (collateral ligaments, menisci) and often induces abnormal stresses to these structures leading to secondary injuries such as meniscal tears.

2.3 Incidence of joint affections

Alexander *et al.* (1981) stated that the incidence of osteochrondritis dissecans (OCD) in the elbow of dog was more in large and giant breeds of dogs.

Fallon (1990) documented osteochrondritis dissecans of stifle mostly in large breeds of dogs and that the condition manifested itself at about six months of age. Males were more affected than females and bilateral lesions were common.

Manely *et al.* (1990) recorded that occurrence of OCD of the shoulder was almost exclusively in medium to large sized dogs between four and eight months of age.

Van Ryssen *et al.* (1993[a]) observed that the forelimb lameness in young dogs of large and giant breeds were more common in males than in females and that the most commonly affected joint was the shoulder.

Van Ryssen and Van Bree (1997) conducted a study in 100 dogs with elbow lameness and found that 74 were males, 52 of them below 12 months of age and 79 dogs were more than 30 kg body weight. The authors also reported that the rapidly growing young dogs of large breeds were affected.

Bardet (1998) reported that the shoulder joint affections mainly occurred in hyperactive medium and large breed dogs. The author recorded that out of 47 shoulder joints 57 per cent of the cases had degenerative joint diseases (DJD). Among the 46 dogs with shoulder instability 30 were males and 16 were females and their ages ranged from eighteen months to thirteen years.

Van Bree and Van Ryssen (1998) observed lesions of fragmented coronoid processes in 19 dogs and opined that the incidence was more in the dogs under 12 months of age. The authors also observed stifle lameness in young dogs due to osteochondrosis dissecans and that in 90 per cent of the cases the lesion was located at the medial aspect of the lateral femoral condyle.

Neaas *et al.* (2002) examined 48 stifles with cruciate ligament rupture. Complete rupture was diagnosed in 30 joints and the rest were affected with partial cruciate ligament rupture.

Gortz *et al.* (2004) reported that elbow incongruity was an important etiological factor in fragmented coronoid processes. Fragmentation was caused by mechanical overload possibly in the presence of a weak bone or cartilage. Fragmented coronoid processes could also be the result of sub chondral bone fracture with ineffective fibrous repair.

2.4 Etiology of joint diseases

Wind (1986), in a study of the canine elbow joint, reported that incongruity of the elbow was a common reason for fragmented coronoid process, osteochondritis dissecans of the humeral condyle and ununited anconeal process.

Wind and Packard (1986) carried out radiographic studies using the lateral view of the elbow to study the affections of the elbow joint. The authors reported that young dogs of heavy and large sized breeds showed a greater predilection for elbow affections like fragmented coronoid process, oesteochondritis dissecans of the humeral condyle and ununited anconeal process, especially in the growing stage of the dogs.

Van Ryssen *et al.* (1993[b]) observed that the osteochondrosis was the common cause for the forelimb lameness in young dogs of large and giant breeds. The authors also reported that the condition is seen more commonly in male dogs and the common predilection site being the caudal humeral head.

Van Ryssen and Van Bree (1997) reported the etiological causes that included mechanical overload, mechanical stress due to abnormal development of the trochlear notch resulting in elbow incongruity and as a change secondary to premature closure of one of the radial physis, and trauma. The authors opined that common causes for forelimb lameness especially in rapidly growing dogs of large breeds were conditions involving the elbow joint.

Bardet (1998) described that the shoulder instability to be the common cause of forelimb lameness in dogs. High demand, repetitive use of the shoulder and repetitive microtrauma were also implicated in the etiology of gleno-humeral instability.

Long and Nyland (1999) stated that forelimb lameness may be caused by degenerative changes or traumatic conditions involving the shoulder joint. Bicipital tendonitis was a common cause for lameness especially in large to medium sized breeds of dogs.

Hayashi *et al.* (2003) reported that cranial cruciate ligament rupture with concurrent development of osteoarthritis was one of the most common affections of the stifle joint in dogs. The age, body weight and breed were found to be predisposing factors with the large breeds of dogs being affected even at a younger age.

Lopez *et al.* (2003) stated that cranial cruciate ligament rupture and associated degenerative changes was a common cause for hind limb lameness in dogs.

2.5 Clinical evaluation

2.5.1 Clinical symptoms

Alexander *et al.* (1981) reported that the OCD of elbow joint revealed crepitation, pain upon flexion and extension of the elbow and that gross joint distension was usually not a feature of OCD of elbow joint.

Fallon (1990) found that dogs affected with OCD of the stifle exhibited signs of slight cranial drawer sign not associated with cranial cruciate ligament (CCL) rupture and thigh muscle wastage due to disuse atrophy. The joint was found to be painful on extension and had a decreased range of extension with crepitus.

Van Ryssen *et al.* (1993[b]) stated that dogs showing osteochondrosis of the shoulder exhibited signs of obvious forelimb lameness, and pain was elicited on extension and flexion of the joint. All the dogs demonstrated minor to moderate muscular wastage of the infraspinatus and deltoideus muscles.

Van Ryssen and Van Bree (1997) examined 100 dogs with lameness related to the elbow and recorded clinical symptoms of pain on flexion and or extension of the joint, endo rotation of the joint and periarticular swelling.

Bardet (1998) observed that pain on hyperextension of the shoulder joint was present in forty out of forty-seven shoulders examined and the common clinical presentation was permanent foreleg lameness.

Mitchell and Innes (2000) reported three cases of lateral gleno-humeral ligament rupture showing signs of chronic forelimb lameness. Other symptoms included mild to severe supra spinatus muscle atrophy, pain on palpation of the bicipital groove and extension as well as internal and external rotation of the glenohumeral joint.

Scharf *et al.* (2004) reported that dogs with bicipital tendinitis showed marked resentment to flexion or extension of the glenohumeral joint. The dogs also exhibited progressive or intermittent weight bearing lameness of one or both forelimbs. The condition could result from direct trauma or strain of the bicipital tendon or entrapment of joint mice within the tendon sheath.

2.5.2 Grading of lameness

Miller and Presnell (1985) advocated a score of 0 to 3 for the grading of lameness in 24 stifles of 19 dogs showing stifle involvement. 0 = no lameness, 1 = mild to moderate post exercise lameness, 2 = moderate to severe post exercise lameness and 3 = severe or non weight bearing lameness.

Lewis *et al.* (1987) reported the following grading for lameness evaluation: 0 = normal, I = intermittent weight bearing lameness, II = persistent weight bearing lameness, III = intermittent non weight bearing lameness, and IV = persistent non weight bearing lameness.

Bertrand *et al.* (1997) assigned lameness scores for evaluating lameness in dogs with hind limb lameness from grade 0 to 5. 0 = no lameness, 1 = subtle, intermittent weight bearing lameness, 2 = subtle, consistent weight bearing lameness, 3 = obvious weight bearing lameness, 4 = intermittent, non weight bearing lameness and 5 = consistent, non weight bearing lameness.

2.6 Synovial fluid analysis

2.6.1 Collection

Kivumbi and Bennett (1981) stressed the importance of the aspiration of the synovial fluid by arthrocentesis and reported that excess aspiration of the synovial fluid could cause cloudiness during arthroscopy.

Coles (1986) reported that the serum biochemistry and biochemical evaluation of the synovial fluid should be done simultaneously in order to compare and to correlate the changes with different pathological conditions. The synovial fluid changed with disease and hence could provide valuable information concerning the degree and type of change occurring within the joints.

Kumar and Agarwal (2001) opined that the greatest value of synovial fluid analysis was in differentiating inflammatory from non-inflammatory diseases. In addition, it might provide information to obtain a specific diagnosis of septic, crystalline and immune mediated arthritis, with few drops of synovial fluid. Nucleated cell count, differential cell count and a gross evaluation might provide useful information for prognosis and treatment of joint diseases.

2.6.2 Physical properties

2.6.2.1 Volume

Fernandez *et al.* (1983) stated that synovial fluid volume of normal dogs was 0.35 ml ± 0.25 ranging from 0.2 to 1.0 ml.

Lipowitz *et al.* (1985) reported that the normal volume of synovial fluid varied from joint to joint. In the dog the average was 0.24 ml, ranging from 0.01 ml to 1.0 ml. The author stated that in degenerative joint disease the volume of the synovial fluid might not be appreciably increased. However with increased inflammatory sign the amount of synovial fluid in joint was also increased.

Lewis *et al.* (1987) studied degenerative joint disease induced experimentally by transection of cranial cruciate ligament in ten dogs. The volume of the synovial fluid was found to be increased in six out of the ten dogs studied, with volumes ranging from 5-20 ml.

Houlton (1994) stated that the volume of synovial fluid obtained would vary from joint to joint. Volumes ranged from 0.01 to 1.0 ml. The author stated

that extent of joint effusion was easier to appreciate clinically, rather than by measuring the fluid obtained by synoviocentesis. The author emphasised that the volume should be recorded and that when significantly less fluid was obtained it might be due to the presence of other intraarticular material such as pannus or fibrin.

Mitchell and Innes (2000) examined three dogs with glenohumeral ligament rupture and on arthrocentesis obtained synovial fluid of 4 ml in one dog.

2.6.2.2 Physical appearance

Fernandez *et al.* (1983) described normal synovial fluid as a clear, colourless to straw coloured fluid. Cloudiness or turbidity was attributed to inflammation and a change in colour was suggestive of pathological condition or contamination with blood.

Coles (1986) described normal synovial fluid to be a transparent, pale yellow fluid. In degenerative joint disease the presence of cartilagenous material caused the appearance of the synovial fluid to vary from a pale yellow and transparent to pale yellow and opaque. A definite flocculate appearance was observed in such conditions. Dark yellow to amber colour of the synovial fluid was observed in chronic traumatic arthritis and usually indicated a low grade prolonged haemorrhage.

Houlton (1994) stated that dark yellow to xanthochromic synovial fluid indicated chronic haemorrhage. Turbid samples of varied colour was associated with inflammatory joint disease, usually due to the presence of cells, fibrin or

other debris in the fluid. Also intraarticular ligament rupture would produce small amounts of haemorrhage and a serosanguinous appearance to the synovial fluid. Traumatic joint disease like cranial cruciate ligament rupture progressing to a chronic state would give results similar to degenerative joint disease.

2.6.2.3 Mucin clot test

Coles (1986) found that the mucin clot test of the synovial fluid of animals with traumatic or degenerative joint disease were usually normal with the viscosity within the normal limits. In articular disease, mucin clot was of poor quality with variable degree of flocculation appearance in a cloudy solution. The author also reported that in degenerative joint diseases, cartilagenous material might give the synovial fluid a definite flocculent appearance. In such cases the samples vary from pale yellow and transparent to pale yellow and opaque. Occasionally blood was observed in the sample and it became important to determine whether haemorrhage occurred prior to or during the aspiration. In acute traumatic arthritis the samples were markedly haemorrhagic, whereas samples that were streaked with whole blood usually indicated haemorrhage at the time of aspiration. Dark yellow to amber samples were seen in chronic traumatic arthritis and usually indicated a prolonged low-grade haemorrhage or massive haemorrhage that occurred prior to collection. Fluid collected from joints with acute to subacute arthritis and acute to chronic septic or infectious arthritis tended to clot rapidly.

Doxy (1983) stated that the synovial mucin was an acid glycoprotein that was demonstrated by the addition of acetic acid to synovial fluid. Mucin

clot quality was a representative indication of the viscous property and quality of hyaluronic acid present in synovial fluid. In the synovial fluid from animals with traumatic or degenerative joint lesions, the mucin concentration the viscosity and the mucin clot quality were normal. An abnormal result in the acetic acid test usually occurred when the mucin concentration and viscosity were lowered due to bacterial degeneration of mucin.

Lewis *et al.* (1987), on examination of the physical properties of the synovial fluid in ten adult dogs with degenerative joint disease, rated the mucin clot test to be poor in all the samples.

Houlton (1994) found that when the mucin clot was normal, a tight ropy clump formed in a clear solution and this mucin clot was graded as good. A softer mass with some shreds in solution was graded as fair and poor clot results showed shreds and small soft masses in a turbid solution. Fluids that produced a few clump flakes of mucin suspended in a cloudy solution were classified as very poor. Inflammatory fluids often had fair to poor quality mucin. Both septic and nonseptic inflammatory joint diseases might show poor quality mucin clot due to lysosomal enzymes released from degenerating neutrophils, whereas traumatic or degenerative osteoarthropathies often had a good mucin clot.

2.6.3 Cytological examination

Fernandez *et al.* (1983) reported that the normal synovial fluid contained 0-2,900 WBC/mm^3 and few red blood cells. The primary cell types seen on a smear were mononuclear cells (65% to 90%). Monocytes and macrophages

predominated while lymphocytes and synovial lining cells were less common. Monocytes were frequently round in synovial fluid smears and resembled cleaved lymphocytes. Polymophonuclear cells were rarely seen in normal synovial fluid (less than 10%) and indicated blood contamination.

Miller and Presnell (1985) reported that cytological examination of synovial fluid revealed less initial deviation and more rapid return to pre-surgical values in dogs that had an arthroscopy when compared with that of an arthrotomy. The authors also found increased number of polymorphonuclear cells indicating damage to periarticular vasculature, or altered permeability and chemotaxis associated with inflammation, while return to normal values was also slower in inflammatory conditions.

Coles (1986) stated that in degenerative joint disease normal to increased number of macrophages were found and showed enhanced phagocytic activity. Synoviocytes, chondrocytes, pieces of cartilage, osteoblasts and osteoclasts might be seen in fluid from joints in which there was exposed bone. In traumatic arthritis, haemosiderin-laden macrophages, some foreign body gaint cells and occasionally fat globules might be seen.

Lewis *et al.* (1987) reported that increased synovial fluid volume was from 5 to 20 ml in experimentally induced degenerative joint disease, leucocyte counts ranged from less than 1000 cells/dl to 12000 cells/dl with an increased percentage of neutrophils and mononuclear cells.

Fernandez *et al.* (1983) found that in disease conditions, whether DJD or early immunologic disease, the monocyte–macrophage compartment showed

increased numbers, increased phagocytic activity and reactive cell types. Gross evaluation and cytological examination of the synovial fluid might not always provide specific diagnosis but was useful in categorizing joint conditions into major groups such as reactive and inflammatory reactive conditions. Inflammatory conditions showed exudative reactions such as those seen in infectious and immune mediated joint diseases.

Hoelzler *et al.* (2004) observed a trend towards lowered differential polymorphonuclear cell count in the synovial fluid and a faster return to normal values for dogs that had undergone arthroscopy. The authors reported that polymorphonuclear cells were less than 10% in the absence of inflammation. Values between 10% and 30% indicated mild inflammation, 30% to 50% indicated moderate inflammation and above 50% indicated severe inflammation.

O'Neill and Innes (2004), on examination of the synovial fluid of a three year old male Springer spaniel showing signs of shoulder lameness, diagnosed medial gleno-humural ligament rupture with increased total nucleated blood cell count of 3.62x 10^9 per litre with 97% of the cells being mononuclear which was indicative of a chronic inflammatory process consistent with osteo-arthritis.

2.6.4 Biochemical evaluation

Coles (1986) established that the total protein values in acute non infectious inflammations increased with a decrease in the percentage of albumin and an increase in the percentage of alpha globulin. Chronic non-

infectious joint inflammation resulted in increased beta globulins. There was an overall increase in total protein in inflammatory joint conditions. The glucose levels of the synovial fluid were considered to be of diagnostic value. Due to the release of glycolytic enzymes by increased polymorphonuclear cells, the glucose levels were found to decrease in inflammatory joint conditions. The synovial fluid glucose values decreased when compared to serum glucose levels with increasing degree of inflammation. Alkaline phosphatase levels in normal synovial fluid were below that of serum levels. In inflammatory conditions with increased joint effusion the synovial fluid alkaline phosphatase increased approaching those of normal serum values.

Doxy (1983) stated that the mean synovial fluid sugar concentration proved to be higher than the serum sugar concentration. With increased inflammation, synovial fluid sugar values fell below those of the simultaneously measured serum sugar and sometimes even approached a value of zero.

McIlwraith and Fessler (1978) reported a close correlation between the activities of alkaline phosphatase, aspartate amino transferase and lactic dehydrogenase in synovial fluids and clinical severity of joint disease. The increased enzyme activity in the joint fluid might be due to the release of enzymes from leukocytes, necrotic or inflammed synovial tissue or production and release of enzymes by altered synovial tissue.

Tayal *et al.* (2001) reported that the protein levels (total protein, globulin and albumin) should normally be minimum (less than 2.5 g/L). The presence of plasma-derived proteins was associated with inflammation and usually paralleled by changes in serum levels.

2.7 Radiography

Alexander *et al.* (1981) reported that although radiographic evidence of osteochondrosis dessicans of the stifle varied from flattening of the affected femoral condyle to the appearance of radiolucent concave defect of the articular aspect, positive arthrography was often required to confirm diagnosis or to detect a cartilaginous flap or radiolucent loose bodies.

Milton *et al.* (1981), in a survey of 109 Grey hound specimens, reported that higher incidence of lesions of osteochondrosis dessicans occurred in the glenoid cavity and the lesions were not evident with standard radiography.

Boudrieau *et al.* (1982) reported that osteochondrosis dessicans of the elbow is not visualised radiographically before seven months of age even though symptoms of forelimb lameness may be present from two months onwards. Definitive radiographic lesions may also not be visible with the standard cranio caudal or lateral projections.

Lenehen and Van Sickle (1985) advocated a medial lateral radiograph with the affected shoulder down and the affected limb pulled cranioventrally while the head and neck were dorsiflexed and the upper leg and chest rotated away from the table surface. The most common radiographic finding of osteochondrosis dissecans of the shoulder was an irregular, radiolucent subchondral defect usually involving the caudal aspect of the humeral head. Subchondral sclerosis, calcified cartilage flap and joint mice was be seen depending on the progression of the lesion. Some demonstrating subchondral and articular defects were not visible on the plain radiograph.

Burk and Ackermann (1986) described cranial cruciate ligament rupture with displacement of the proximal tibia cranially in relation to the femur and distal displacement of the popliteal sesamoid bone radiographically. The authors also recorded the radiographic features of degenerative joint disease to show periosteal proliferation at the site of the joint capsule and at the margins of the articular cartilage. This proliferation was usually smooth and uniformly mineralized with well – defined margins. The subchondral bone was thinned, thickened, dense or irregular. Intra-articular bone densities were also be seen. Malalignment of articular surfaces or joint subluxation was seen in specific conditions. In most cases, however, radiographic findings were less extensive than those observed at surgery.

Presnell (1990) stated that osteochondrosis dissecans lesions of the elbow like fragmented coronoid process (FCP) were difficult to visualize radiographically. A cranio caudal view combined with two oblique cranio-caudal views were advocated and osteochondrosis dissecans was seen as a small defect at the medial condyle of the humerus.

Van Bree *et al.* (1993) stated that the positive contrast arthrography was useful in the evaluation of the status of the articular cartilage in shoulder osteochondrosis. False negative results also occurred in the determination of rupture and fragmentation of the articular cartilage and in the detection of joint mice. Diagnostic arthroscopy of the canine shoulder allowed good visualisation of intra-articular structures and pathologic changes of synovium and articular cartilage than conventional arthrotomy.

Van Ryssen *et al.* (1993[a]) reported that a common radiographic finding in osteochondrosis dessicans of the shoulder was a subchondral defect on the caudal aspect of the humeral head. The authors stated that plain radiographs could not be used to assess either the status of the articular cartilage or the presence of non-radiopaque loose bodies.

Anderson (1994) opined that osteochondrosis dessicans of the stifle is not very common and is often overlooked as the affected animal usually suffers from hip dysplasia as well.

Butterworth (1994) stated that a medio-lateral view of osteochondrosis dessicans of the shoulder usually revealed subchondral defect with flattening of the caudal humeral head, sclerotic margin of the defect, cartilage flaps and joint mice, visible only if mineralised. Plain radiographs were of limited value in bicipital tendinitis.

Lavelle (1994) stated osteochondrosis dessecans of the elbow would show defect in the subchondral bone in the caudal part of the humeral head. In the elbow due to the number of other conditions that comprise osteochondrosis dessecans of the elbow, radiographic diagnosis is not easy. Multiple views were required for a complete radiographic examination and usually revealed a large asymmetrical radiolucent area in the metaphysis of the distal ulna. Flexed medial to lateral views were advocated.

May (1994) reported the presence of osteophytes in cases of degenerater joint disease (DJD) at characteristic sites varying according to the joint affected and cartilage erosion, if severe could be appreciated. Narrowing of the joint space may not be visible unless a weight bearing view is taken and even then

may be difficult to assess. The author also stated that the main aid to diagnosis of DJD was radiography. However, the formation of osteophytes occurred early on progression of the disease condition.

Bertrand *et al.* (1997) reported that the radiographic lesions were identified arthroscopically in 75 per cent of the stifles examined. The authors opined that positioning of the stifle must be precise for subchondral defects to be visible in a radiograph and that the oblique cranio-caudal view increased the sensitivity of radiography in identifying subchondral defects.

Bardet (1998) radiographically examined forty five dogs with shoulder lameness using medio lateral, cranio caudal and stress medio lateral views. The first sign of degenerative joint disease was a small osteophyte on the caudal margin of the humeral head.

Van Bree and Van Ryssen (1998) found that plain radiographs of the shoulder were not helpful to assess the status of the articular cartilage or the presence of non radio-opaque loose bodies.

Bardet (1999), in a retrospective study of 23 dogs with shoulder lameness, advocated mediolateral, craniocaudal and mediolateral stress radiographs for the diagnosis of lesions of the biceps tendon. The radiographic examination of the shoulders revealed four normal shoulders (16%) and osteoarthritis in twenty one cases (84%).

Mitchell and Innes (2000) found that the traditional imaging methods including radiography, arthrography and ultrasonography were unrewarding in evaluating chronic forelimb lameness in dogs. Radiography was essential for

an early diagnosis for osteochondrosis dissecans of the stifle. A standard cranio-caudal view was advocated which showed irregularity in the contour of the femoral condyles. Medial aspect of lateral condyle was most often affected and the lesion appeared as a translucent concave defect with sclerotic margin. The lesions could also be seen on the medial condyle.

Gortz *et al.* (2004) described a case of fragmented coronoid process in a 10 month old Rottweiler in which radiographic, computed tomographic, arthroscopic and histologic findings were recorded. The authors found that by routine radiography of medio-lateral and antero-posterior projections of the elbow, fragments could not be detected and extended medio-lateral view showed an poorly defined medial coronoid process but fragments could still not be detected.

O'Neill and Innes (2004) opined that plain radiography might not always reveal lesions of the shoulder joint especially those related to the soft tissue structures in and around the joint.

2.8 Instrumentation

2.8.1 Telescope

Van Bree *et al.* (1992) used a 2.7mm 25^0 fore oblique arthroscope in a 4mm outer diameter sleeve for arthroscopic examination of oestrochondrosis lesion in 20 shoulder joints.

Van Ryssen *et al.* (1993[b]) arthroscopically treated twelve dogs with osteochondrosis of the shoulder using a 2.7mm, 25^0 fore oblique arthroscope.

Bardet (1998) performed arthroscopy of the shoulder joint in 45 dogs with a 2.7 mm 30^0 fore oblique arthroscope with a 3.5 mm outer sleeve diameter.

Rochat (2001) stated that a 2.7 mm diameter arthroscope was best suited for medium and large dogs. The 30^o fore oblique angle view was found to be most useful as it allowed a wide field of vision and rotation of the arthroscope making possible to view a large percentage of the joint through a single port.

Martini *et al.* (2002) performed diagnostic and surgical arthroscopy of the shoulders of 27 dogs with a 2.7mm 25^0 or more oblique arthroscope in a 4mm sleeve. A 150 Watt halogen light source with a fibreoptic cable and camera were used. Pressurized lactated Ringer's solution was used as the irrigating fluid.

Neaas *et al.* (2002) performed arthroscopy of the stifle in large and medium breeds of dogs using a 2.7mm 30^0 angle arthroscope with a 4mm diameter sleeve and blunt obturator. In small breeds the authors used a 1.9mm 30^0angled arthroscope.

Gortz *et al.* (2004) performed arthroscopy of the elbow using a 2.4 mm arthroscope in a 3.5 mm diameter sleeve through medial approach with lactated Ringer solution as irrigating fluid.

O'Neill and Innes (2004) used a 2.4mm 30^0 or fore oblique arthroscope for arthroscopy of the shoulder of a dog for diagnostic and therapeutic applications.

2.8.2 Light source

Van Bree *et al.* (1992) during arthroscopy of the shoulder joint used a 150 W light source with electronic flash generator.

Van Ryssen *et al.* (1993[c]) used a 150 W light source with an electronic flash generator.

Bardet (1998) used a xenon light source for performing arthroscopy of the shoulder joint in 45 dogs.

Van Bree and Van Ryssen (1998) found a simple light source of 100 to 150 W sufficient for diagnostic arthroscopy.

Rochat (2001) reported that the xenon light source with wattage of 400 provided brighter and whiter light, minimized eye strain and facilitated accurate identification of normal and pathologic intra articular architectures.

Neaas *et al.* (2002) performed arthroscopy of the stifle in large and medium breeds of dogs using a xenon bulb of 100 W as the light source.

2.8.3 Camera

Van Bree *et al.* (1992) carried out photographic documentation of shoulder arthoscopy with a 105mm objective, a 200 ASA film and an exposure time of 1/60 or 1/30 sec or a video camera.

Van Ryssen *et al.* (1993[a]) photographically documented arthroscopic treatment of 12 shoulder joints using a single reflex camera with at 105mm

objective, a 200 ASA film with an exposure time of 1/60 or 1/30 second or a video camera.

2.8.4 Irrigation solution

Reagan *et al.*, (1983) recommended the use of lactated Ringer's solution as an ideal irrigating solution for arthroscopic procedures. In comparison with other solutions like normal saline, phosphate buffered saline, acetated Ringer's solution and plasmalyte, lactated Ringer's solution enhanced cartilage metabolism by supporting normal synthesis of proteoglycan by the chondrocytes.

Bardet (1998) examined arthroscopically forty seven shoulder joints of dogs and used 10-15 ml of lactated Ringer's solution to distend the joint.

Van Bree and Van Ryssen (1998) used pressurized fluid bags of 0.9% sodium chloride for joint irrigation during an arthroscopic procedure of the shoulder. The authors reported that lactated Ringer's solution proved to be less harmful to the cartilage cells and safe fluid pressure within the joint was found to vary from 15mm to 150mm of Hg.

Rochat (2001) reported that lactated Ringer's solution as the fluid of choice for joint irrigation and recommended a pressure of 50 to 100 mm Hg was sufficient for proper joint distension without excessive fluid extravasation.

Martini *et al.* (2002) performed diagnostic and surgical arthroscopy of the shoulders of 27 dogs using pressurized lactated Ringer's solution as the irrigating fluid.

Gortz *et al.* (2004) performed arthroscopy of the elbow using lactated Ringer's solution as irrigation fluid.

2.9 Advantages of arthroscopy

McIlwraith and Fessler (1978) opined that the arthroscope offered a means by which the state of both the articular cartilage and synovial membrane could be conveniently and quickly ascertained.

Kivumbi and Bennett (1981) found that repeated arthroscopic examinations could be done with minimal joint damage. Follow-up examinations after joint lavaging could also be carried out due to the minimally invasive nature of the technique.

Van Gestel (1985) found that physical and radiographic examination of the stifle had limited diagnostic accuracy. In cases involving pathology of the medial meniscus and in severe cases involving pathology of the anterior cruciate ligament, arthroscopical findings were directly opposed to the clinical and radiographic diagnosis.

Lewis *et al.* (1987) established that arthroscopy allowed examination of intra articular structures of the joint with minimal invasiveness and negligible morbidity. The authors reported that dogs undergoing arthroscopy had a shorter recovery time compared to arthrotomy.

Bardet (1998) opined that arthroscopy offered the advantage of recognizing all the intra-articular pathologies due to magnification, which were not recognized in the past even after arthrotomy.

Mitchell and Innes (2000) recorded that arthroscopic examination of the shoulder joint allowed visualization of the glenohumeral ligament, synovia and tendon of origin of biceps brachii.

Rochat (2001) found that recovery from the arthroscopic procedure was more rapid than with arthrotomy, but the time for recovery from the underlying disease process itself was relatively unchanged.

Trumble *et al.* (2001) described a technique for the transection of the cranial cruciate ligament by arthroscopy. The authors opined that the procedure was minimally invasive with less incisional morbidity, thereby allowing quicker rehabilitation. Arthroscopy allowed direct visualization for the transection of the cranial cruciate ligament with minimal damage to the articular cartilage or caudal cruciate ligament.

Neass *et al.* (2002) cited that excellent visualization, minimal joint trauma, decreased operative time and lowered patient morbidity were the positive aspects of arthroscopy.

Hoelzler *et al.* (2004) recorded that arthroscopy required a smaller skin incision and that it created less surgical trauma compared to an arthrotomy. Improved joint observation, less tissue disruption, reduced scarring and a faster return to function were other reported benefits.

2.10 Disadvantages of arthroscopy

Kivumbi and Bennett (1981) reported unsatisfactory arthroscopic examinations due to difficulty in introducing the arthroscope into the joint

because of insufficient distension of the joint capsule, gross joint fibrosis and adhesions and inadvertent pericapsular infiltration of the lavage solution. Intraarticular haemorrhage and covering of the end of the arthroscope with infra patellar fat were other difficulties encountered.

2.11 Indications

Van Ryssen *et al.* (2003) opined that the most common indication for diagnostic and surgical arthroscopy of the shoulder joint was osteochondritis dissecans. The most common disorders of the elbow joint that indicated arthroscopic intervention were fragmented coronoid process (FCP), osteochondritis dissecans of the medial humeral condyle and ununited anconeal process.

Rochat (2001) reported that osteochondritis dissecans of the shoulder was the primary indication for arthroscopic examination. Other conditions included tears of the gleno-humeral ligament, glenoid labrum tears, bicipital teno-synovitis, bicipital tendon rupture, articular fracture and incomplete ossification of the caudal glenoid. In the elbow joint, osteochondritis dissecans and fragmented medial coronoid process were common conditions that indicated arthroscopy. The common lesions of the stifle indicating arthroscopy were osteochondrosis dissecans (OCD), cranial and caudal cruciate ligament rupture and meniscal damages.

Van Ryssen *et al.* (2003) reported that the indications for stifle arthroscopy were diagnosis of complete or partial cranial cruciate ligament rupture especially in the early stages before radiographic changes occurred, diagnosis and treatment of osteochondrities dissecans of the femoral condyles,

debridement of septic osteomyelitis, precise evaluation and treatment of meniscal injuries.

Gortz *et al.* (2004) reported decreased lameness after elbow arthroscopy for retrieval of fragmented medial coronoid process.

2.12 Anaesthesia

Kivumbi and Bennett (1981) administered general anaesthesia in all the dogs and recommended the same for the comfort of the patient, production of proper muscle relaxation, to avoid damage to the arthroscope, and to allow lateral and medial rotation of the joint.

Van Ryssen *et al.* (2003) recommended general anaesthesia for the arthroscopic procedure in dogs due to the impractibility of local block sedation and risk of damage to equipment or iatrogenic injury to the patient associated with movement.

2.13 Arthroscopy

2.13.1 Shoulder joint

Various authors reported that a stab incision made 1 cm distal to the acromion and immediately cranial to the acromial head of the deltoideus muscle for the arthroscope and with the egress needle dorsomedial to the greater tubercle afforded the best viewing of the structures of the shoulder joint. (Person (1989) Van Ryssen *et al* (1993[a]) Van Bree and Van Ryssen (1998) and Rochat (2001))

Medial gleno-humeral ligament rupture of the shoulder was diagnosed and treated arthroscopically using a standard lateral arthroscopic port (Van Ryssen *et al.* (1993[b]) and O'Neill and Innes (2004)).

It has been recorded that the understanding and diagnosis of shoulder pain in the adult dog had been greatly enhanced by arthroscopy (Van Ryssen *et al.* (2003) Bardet (1998) and Mitchell and Innes (2000)).

O'Neill and Innes (2004) used a standard lateral arthroscopic portal for the diagnosis and treatment of medial gleno-humeral ligament rupture of the shoulder joint.

2.13.1.1 Technique

Van Ryssen *et al.* (1993[a]) described successful arthroscopic treatment of shoulder osteochondrosis in 12 dogs. Synovitis was visible in all the affected joints.

Van Bree and Van Ryssen (1998) described the technique for shoulder arthroscopy in dogs. The dogs were placed in lateral recumbency with the affected limb upper most. The surgical field was prepared and draped aseptically allowing for full mobility of the leg during the procedure. The joint was held in a neutral position with the scapula and humerus at a 160^0 angle. A 19 gauge needle was used to puncture the joint cranio laterally between the acromion and the caudal part of the greater tubercle in a caudo medial direction. The joint was distended with 10 to 15ml of lactated Ringer's solution. A stab incision was made 1cm caudal to and 1 cm distal to the acromion for the

insertion of the arthroscope. The instrument portal was 2 to 4 cm caudal to and 1cm distal to the arthroscopic portal.

Van Ryssen *et al.* (2003) performed arthroscopy of the shoulder joint using a 2.7mm arthroscope with the dog in lateral recumbency with the affected limb upper most. The lateral approach was considered as the most common approach with access to inspect both the cranial and caudal compartments. The joint was punctured with a needle between the acromion and the caudal part of the greater tubercle in a caudomedial direction. The synovial fluid was aspirated and 8 to 10ml of irrigating fluid injected for joint distension. The arthroscope was inserted in a joint space between the glenoid and the humeral head, 1cm distal to the middle of the acromion.

Martini *et al.* (2002) described a modified technique for shoulder arthroscopy which allowed for a complete access of the scapulo-humeral joint through two entry ports instead of three.

Lehmann and Lehmann (2004) described a modified triangulation technique for arthroscopy of the cranial shoulder using a new target device which was faster and simpler and could be performed with more success and fewer complications.

2.13.1.2 Lesions

Van Bree *et al.* (1992) established the value of arthroscopy of the shoulder joint in demonstrating several types of cartilage lesions such as flaps or fissures, chondromalacia and indentation. "Kissing lesions" on the articular surface of the glenoid cavity opposite to the osteochondritis lesion that were

not visible by arthrography or arthrotomy could be demonstrated by arthroscopy. Also it was more accurate in detecting joint mice than arthrography. The arthroscopic evaluation of synovial inflammation correlated well with the histological findings.

Van Ryssen and Van Bree (1997) stated that the most commonly diagnosed conditions of elbow were fragmented coronoid process, osteochondritis dissecans of the medial side of the humeral condyle and disunited anconeal process. A combination of different conditions within the same elbow joint was described and the term "elbow dysplasia" was suggested.

Bardet (1998) reported that out of 47 shoulder joints 57% of the cases had degenerative joint diseases (DJD).

Van Bree and Van Ryssen (1998) stated that in 19 out of 150 dogs, fragmented coronoid process (FCP) was diagnosed arthroscopically without radiographic signs of arthrosis and that most of the dogs were under 12 months of age and opined that arthroscopy was the preferred procedure in diagnosing fragmented coronoid process (FCP) in an earlier stage before any joint damage had developed.

O'Neill and Innes (2004) found that medial gleno humeral ligament injury was associated with canine shoulder lameness and gleno humeral instability. Osteoarthritis, osteophyte formation, cartilage erosion on the humeral head and glenoid cavity.

Van Ryssen *et al.* (2003) described the normal shoulder arthroscopically. The biceps tendon was intact, with well delineated tendinous

structures, covered with a thin layer of synovial membrane with minimal vessel and villi. The subscapcularis tendon and the glenohumeral ligament was smooth and easily visible. Flexion of the joint enabled inspection of the caudal pouch.

2.13.2 Elbow joint

2.13.2.1. Technique

Bardet (1997[a]) and (1997[b]) found that the medial approach was advantageous in the diagnosis and treatment of fragmented medial coronoid process and osteochondritis dissecans of the elbow because of direct access over the lesions. The author also described the cranio-lateral approach for visualization of the humero-ulnar joint, medial coronoid process, collateral ligaments, synovial membrane, anconeal process, olecranon fossa, and the cranial recess of the elbow joint.

McCarthy (1999) described the medial and lateral approaches for arthoscopic evaluation of the elbow joint. The author preferred the medial approach due to easier joint entry and placement of instruments and also because of better visibility of the humeral condyle and other medial joint structures.

Rochat (2001) described the caudomedial, medial and craniolateral approaches for viewing the medial aspect of the joint. The medial approach had been used for diagnosing and treating osteochondrosis dissecans and fragmented coronoid process. The lateral aspect of the elbow was observed through three portals: lateral, caudolateral and caudodorsal portal and useful in evaluating condylar fractures, anconeal process and synovial biopsies.

Van Ryssen *et al.* (2003) described the technique with dog positioned in lateral recumbency with the affected limb down and the upper limb retracted caudally. The medial aspect of the elbow joint was routinely prepared and the elbow placed over the edge of the table to enable abduction. The point of entry for the inflow – outflow needle was located proximally between the medial aspect of the humeral condyle and the most proximal part of the olecrenon and directed toward the supracondylar foramen and the arthroscopic portal 1.5 to 2cm caudo distal to the medial epicondyle of the humerus. The instrument portal was 1 to 1.5cm distal to the medial humeral epicondyle and 1.5 to 3 cm cranial to the arthroscope.

2.13.2.2. Lesions

Van Bree and Van Ryssen (1998) recorded different types of lesions in the area of the medial coronoid process ranging from full fragmentation to minimal fissure formation. Chondromalacia – like lesions could also be identified. These lesions appeared as soft spongy cartilage without cleft formation . Associated with the findings of chondromalacia, synovitis was also arthroscopically diagnosed.

Van Ryssen *et al.* (2003) described arthroscopy of the normal elbow joint. Inspection of the joint started in the lateral compartment where the lateral portion of the humeral condyle, the caudal part of the radial head and the lateral coronoid process could be visualized. The arthroscope was then moved

proximally thereby visualizing and the border of the lateral portion of the humeral condyle, the lateral part of the joint capsule, the trochlear notch of the ulna and in the most proximal position, the anconeal process could be inspected. The arthroscope was moved in a cranio medial direction to observe the radial head, medial portion of the humeral condyle and the coronoid process. In the most cranial and medial position the medial collateral ligaments could be seen. The cartilage appeared bluish white and smooth, and the synovial villi thin and small.

Gortz *et al.* (2004) reported a case of a 10 month old male Rottweiler with traumatic fracture of the medial coronoid process. Arthroscopy was performed via medial approach , the fragmented medial coronoid process was visualized and removed arthroscopically.

2.13.3 Stifle joint

2.13.3.1 Technique

The medial or lateral para-patellar approach was suggested for stifle arthroscopy for visualization and treatment of ruptured cruciate ligament and menisci, joint mice, osteochondrosis dessicans and synovial biopsy (Kivumbi and Bennett (1981), Miller and Presnell (1985) and Rochat (2001)).

Miller and Presnell (1985) arthroscopically examined the stifles of 18 dogs. The lateral para-patellar approach was followed and sterile lactated Ringer's solution was used as the irrigating solution. The intrarticular structures examined in order were, the suprapatellar pouch, synovial membrane, the femoro-patellar joint, trochlear ridges, medial femoral condyle, cranial aspect of the medial meniscus, caudal medial pouch and caudal

meniscal horn, the inter condylar area, cruciate ligaments, lateral femorotibial joint and lateral menicus.

Person (1985) described the arthroscopic technique for examination of the canine stifle. The technique was developed through 168 arthroscopic examinations. Lactated Ringers' solution was used as the irrigating solution. Pathological changes visualized included synovial villi proliferation and hyperemia, osteophytes, flaking and fibrillated cartilage and meniscial tears or degeneration.

Lewis *et al.* (1987) used the lateral para-patellar approach for arthroscopy of the canine stifle, since it provided the best visualization of suprapatellar pouch, medial patellar pouch, lateral patellar, femoro-tibial articulation, medial meniscus, inter-condylar area, anterior and posterior cruciate ligaments, lateral meniscus and the origin of the long digital extension tendon.

Van Bree and Van Ryseen (1998) described the technique for stifle arthroscopy in dogs. The dogs were placed in dorsal recumbency and the cranial aspect of the stifle joint was prepared for aseptic surgery. The arthroscopic portal was through a stab incision, half way between the tibial tuberosity and the distal aspect of the patella into the joint space lateral or medial to the straight patellar ligament. Joint distension and irrigation was performed through a needle inserted in the suprapatellar pouch.

Van Ryssen *et al.* (2003) recommended the use of a 2.7mm short, 30^0 fore oblique arthroscope for stifle arthroscopy. The arthroscopic portal was made with a stab incision into the joint capsule just proximal to the lateral tibial prominence and just lateral to the patella. The authors used proximal medial

port for the outflow. The scope was advanced below the patella till the tip rested in the proximal part of the superior pouch.

Hoelzler *et al* (2004) studied the surgical management of cranial cruciate ligament injury in dogs by arthroscopy. The arthroscopy was performed by a lateral para-patellar portal which offered maximum visualization.

2.13.3.2 Lesions

Kivumbi and Bennett (1981) carried out a detailed study on 59 both normal and diseased canine stifle joints. The authors reported that all the intra articular structures namely the supra patellar pouch, femoro-patellar joint, medial compartment, intra-condylar notch and the lateral compartment could be identified using a single lateral intra patellar approach. The technique also allowed an assessment of the non-osseous structures of the joint. Pathological changes namely hypertrophy of the synovial membrane, articular cartilage fibrillation and erosion, meniscal degeneration and osteophyte development were appreciated.

2.14 Post arthroscopic evaluation

Miller and Presnell (1985) evaluated post operatively the gaits of the 18 dogs that underwent stifle arthroscopy on alternate post-operative days with a grading scale of 0 through 3 and the dogs' weight ranged from 7.5 kg to 61 kg.

Van Bree and Van Ryssen (1998) observed that following arthroscopy of the shoulder joint most dogs used the treated leg within an hour after

surgery. Lameness decreased remarkably within 10 days postoperatively and resolved completely after three weeks.

Van Ryssen *et al.* (2003) recorded that dogs treated arthroscopically for fragmented coronoid process (FCP) and osteochondrosis dissecans of the elbow usually showed aggravated lameness 1 to 3 days after surgery. Long term results reported 60% excellent, 35% good to fair and 5% unsatisfactory response to arthroscopic treatment.

2.15 Post operative complications

Kivumbi and Bennett (1981) observed no postarthroscopic secondary infection.

Van Ryssen *et al.* (1993[b]) found that the peri-articular swelling subsequent to arthroscopy due to leakage of irrigation fluid from the joint into the muscles and subcutaneous tissue reduced within 24 hours.

Bertrand *et al.* (1997) recorded complications arising from arthroscopy such as iatrogenic lesion of the lateral meniscus and subcutaneous accumulation of lavage solution in four stifles, which resolved within 24 hours of the procedure.

Van Ryssen and Van Bree (1997) arthroscopically examined 100 dogs with elbow lameness (148 elbow joints) and recorded that in 30% of the joints small iatrogenic lesions occurred, which however did not give rise to clinical symptoms. Also after the procedure periarticular fluid accumulated in some animals but resolved within 24 hours.

Van Bree and Van Ryssen (1998) found that none of the dogs showed any post surgical complications such as seroma formation following

arthroscopic evaluation and treatment of osteochondrosis dissecans lesions of the shoulder.

Martini *et al.* (2002) found that the peri-articular effusion of the lactated Ringer's solution proportional to the duration of the arthroscopic procedure caused no problem and spontaneously disappeared within 24 hours of the arthroscopy.

Van Ryssen *et al* (2003) reported that iatrogenic cartilage lesions were common and almost unavoidable but had minimal clinical significance. Oedema of the periarticular tissues and loss of an osteochondral fragment were other complications. Infections and neurovascular damage were rare.

Materials &Methods

CHAPTER - III

MATERIALS AND METHODS

3.1 Selection of cases

Dogs presented to the small animal orthopaedic unit of the Madras Veterinary College Hospital, Chennai with a history of lameness formed the source of animals for the study. Dogs with forelimb or hind limb lameness were subjected to detailed physical and clinical examination which included palpation of the affected joint, passive extension and flexion of the joint at rest. Animals showing involvement of the shoulder, elbow and stifle were selected for the study irrespective of their age, sex, breed and nature of joint affection.

3.2 Design of the study

Based on the clinical presentation, the dogs selected were divided into three groups for arthroscopic examination.

Group I: Twelve dogs exhibiting shoulder lameness

Group II: Twelve dogs exhibiting elbow lameness

Group III: Twelve dogs exhibiting stifle lameness

3. 3 Radiography

Each dog was subjected to plain radiography before arthroscopy. Lateral view for shoulder and joint, latero-medial and antero-posterior views for elbow joint and stifle joints were taken.

3.4 Arthroscopy

3.4.1 Premedication and anaesthesia

Premedication consisted of intramuscular administration of atropine sulphate at the dose rate of 0.04 mg/kg body weight and xylazine hydrochloride at the dose rate of 1 mg/kg bodyweight. General anesthesia was induced with an intramuscular injection of ketamine hydrochloride at the dose rate of 10mg/kg bodyweight. Sufficient muscle relaxation was achieved by intravenous infusion of diazepam at the dose rate of 0.25 mg/kg bodyweight. Anaesthesia was maintained by the intravenous administration of incremental doses of ketamine and diazepam.

3.4.2 Materials used

An arthroscopy unit (Karl Storz, Germany) with a rigid endoscope and a flexible fiberoptic cable was used for visualisation of the joints. This versatile unit was equipped with a complete range of accessories including monitor, telescopes, video camera, arthro pump and light source. (Plate 1).

Plate - 1
Arthroscopic Unit (Karl Storz - Model 20)

3.4.2.1 Telescope and arthroscopic sleeve

A 2.7 mm 30° forward oblique Hopkins telescope was used for the study. The fiberoptic cable was attached to the telescope with a screw on adaptor. The telescope was used in combination with a sleeve into which the telescope fitted in precisely. The sleeve had two parts, one was used for fluid ingress and other for egress (Plate 2). The arthropump controlled the rate of flow and the pressure of the irrigation fluid. (Plate 3).

3.4.2.2 Camera

The Endovision TELECAM-DX, an endoscopic video camera with attachments for rigid and flexible endoscopes was used. White balance reset the camera system's chrominance controls to conform to the colour temperature of the light source in use. (Plate 5).

3.4.2.3 Light source

The halogen light source was a built-in cold light fountain with a halogen lamp (24 V/250 W) used in conjunction with the Karl Storz ENDOVISION camera.

3.4.2.4 Xenon light source

The 300W xenon lamp corresponded to the colour temperature of sunlight and therefore produced exceptionally brilliant illumination. Full light intensity was reached as soon as the lamp was switched on. The brightness could be regulated from 0-100 % through microprocessor controlled opto mechanical dimmer. An antifog air pump prevented misting of the lens.

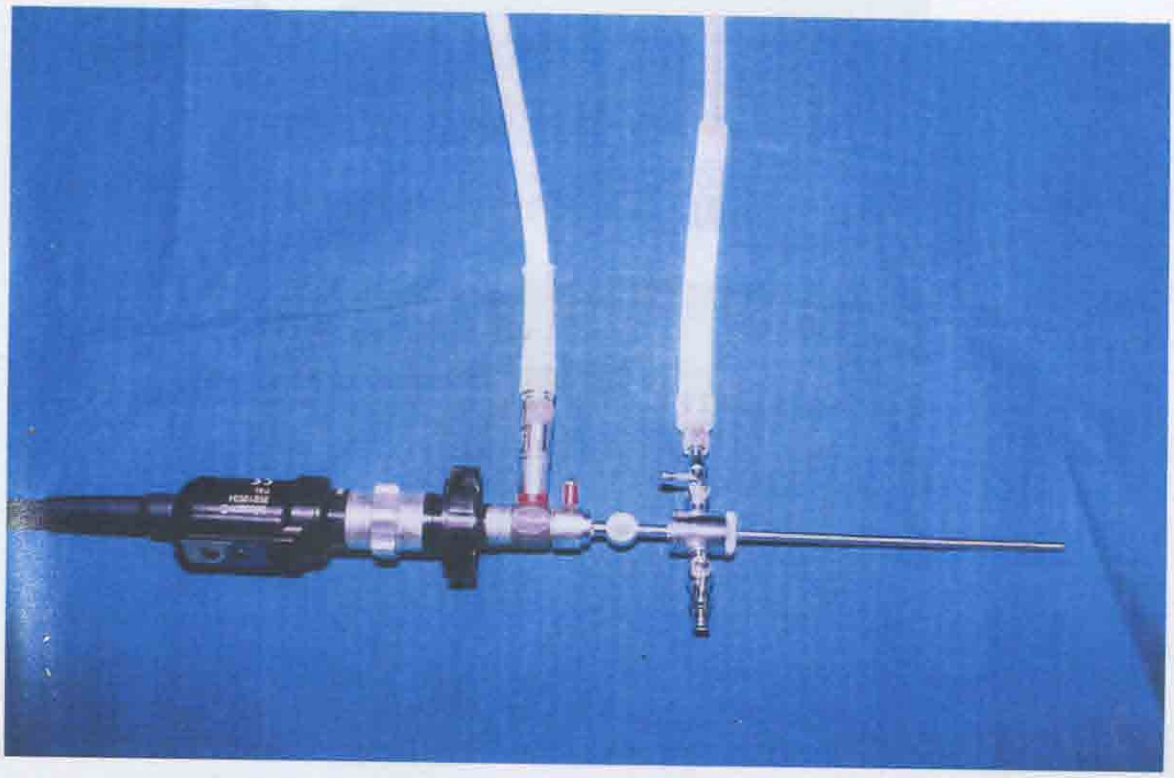


Plate - 2
Telescope (Hopkins, 2.7mm) and arthroscopic sleeve (3.0mm)

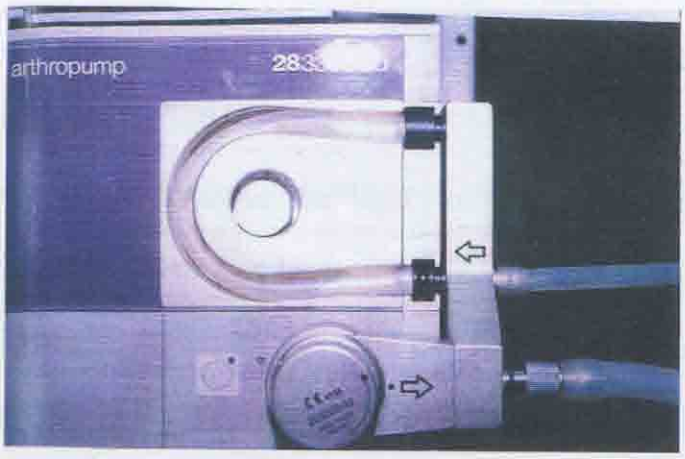


Plate - 3 Arthropump (Karl Storz)

Plate - 4 Fiberoptic cable (Karl Storz)

Plate - 5 Endocamera with cable (Karl Storz)

3.4.2.5 Fiberoptic light cable

Fiberoptic light cables contained a large number of single glass fibers and was used to transmit the light from its source to the camera. Fiberoptic light cables were opaque to ultraviolet light. Light absorption was approximately 10% per meter, the radiation loss was about 40% and about one third of the light struck the cable at the end of a two meter long fiberoptic light cable (Plate 4).

3.4.3 Techniques of arthroscopy

3.4.3.1 Portals for arthroscope

The portals for the egress and arthroscope are shown in table 1.

3.4.3.1.1 Shoulder joint

The egress port was one to two centimetre cranial to the acromian in a caudomedial direction and the arthroscopic portal was one centimetre distal to the acromion. (Person, 1985) (Fig. 1).

3.4.3.1.2 Elbow joint

The egress port was midway between the medial humeral condyle and the most proximal part of the olecranon and the arthroscopic portal was one centimeter distal to and 0.5 centimeter caudal to the medial epicondyle of the humerus (Rochat, 2001) (Fig. 2).

TABLE - 1

Arthroscopic portals of shoulder, elbow and stifle joints in dogs

Sl. No.	Joint	Egress	Telescope
1.	Shoulder	1-2 cm cranial to the acromion in a caudomedial direction	1 cm distal to the acromion
2.	Elbow	Between the medial humeral condyle and the most proximal part of the olecranon	1 cm distal and 0.5 cm caudal to the medial epicondyle of the humerus
3.	Stifle	Medial to patellar ligament mid way between the patella and the tibial tuberosity	Lateral to patellar ligament mid way between the patella and the tibial tuberosity

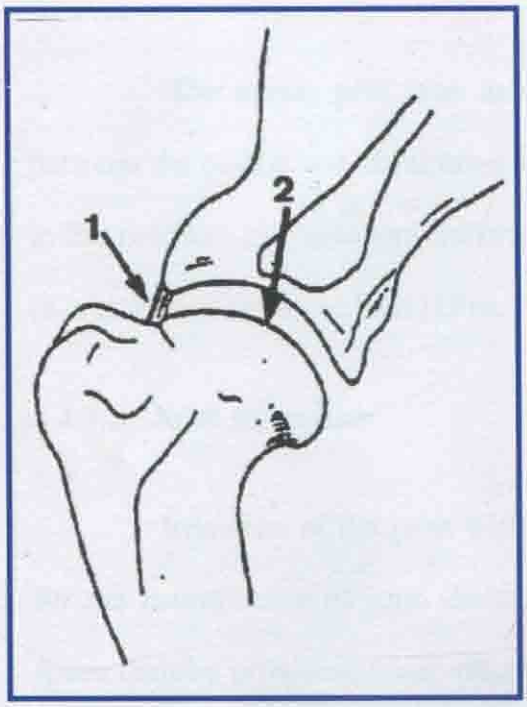


Fig.1. Shoulder joint - lateral view- portals of entry
1. Needle 2. Arthroscope

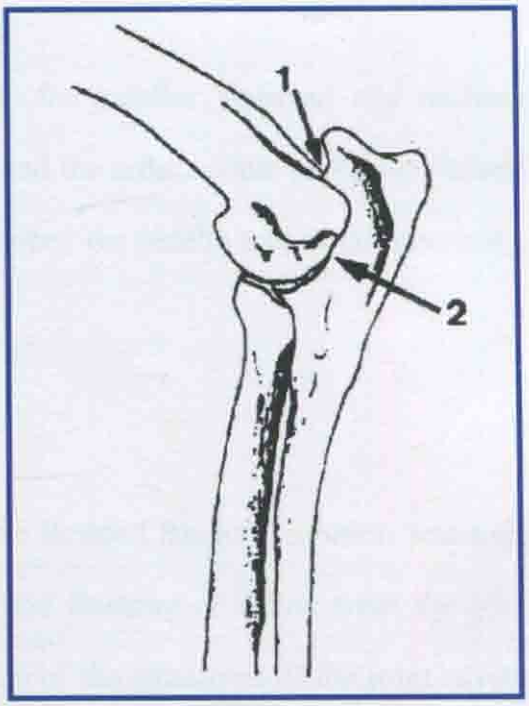


Fig.2. Elbow joint - medial view - portals of entry
1. Needle 2. Arthroscope

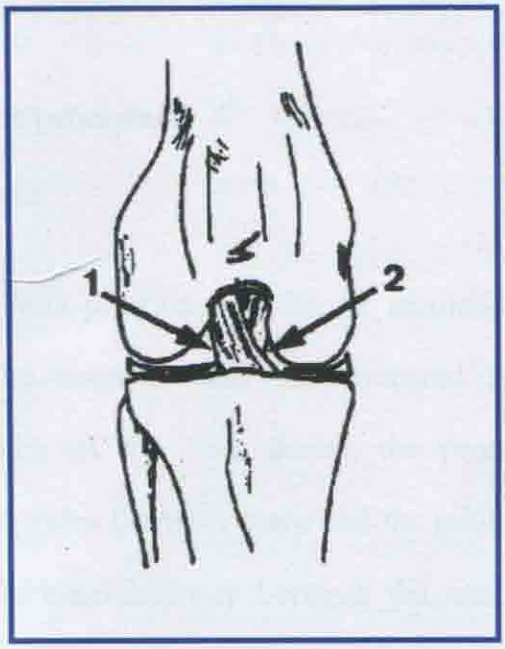


Fig.3. Stifle joint - cranial view - portals of entry
1. Needle 2. Arthroscope

3.4.3.1.3 Stifle joint

The egress port was medial to the patellar ligament and midway between the patella and tibial tuberosity and the arthroscopic portal was lateral to the patellar ligament and midway between the patella and tibial tuberosity. (Kivumbi and Bennett ,1981) (Fig. 3).

3.4.3.2 Joint irrigation

Irrigation of the joint with sterile lactated Ringer's solution was used for the maintenance of joint distension and flushing of debris from the joint space thereby providing clear visualisation of the structures of the joint cavity. The fluid entered the joint through the cannula and was drained through the egress needle. The infusion fluid passed through the arthropump which maintained the flow rate and pressure between 50 mm Hg to 75 mm Hg depending on the size of the joint. (Reagan *et al.,* 1983).

3.4.4 Arthroscopic procedure

3.4.4.1 Shoulder joint

The patient was positioned in lateral recumbency with the affected limb uppermost. The surgical field was prepared and draped aseptically allowing full mobility of the limb during the procedure. The limb was tractioned distally to widen the joint space and the joint was punctured with an 1.5 inch 18G needle craniolaterally between the caudolateral aspect of the greater tubercle and the distal aspect of the acromion process. The needle was directed in a caudomedial direction and synovial fluid was aspirated when the needle entered the joint with 2-3 ml disposable syringe. The joint was

distended with 10 to 15 ml of lactated Ringer's solution. A stab incision was then made 1 cm caudally and 1 cm distally to the acromion process. (Van Bree and Van Ryssen, 1998). The joint capsule was penetrated using the sharp trocar and arthroscopic sleeve. Once the joint was entered the sharp trocar was replaced by the arthroscope. An egress needle was placed dorsomedially to the greater tubercle taking care to avoid the bicipital tendon.

3.4.4.2 Elbow joint

The patient was positioned in lateral recumbency with the affected limb on the table. The medial side of the elbow was prepared for aseptic surgery. A 1.5 inch 18G needle was inserted in the caudodorsal portion of the joint between the anconeal process and the olecranon fossa. Synovial fluid was aspirated and the joint subsequently distended with 10 to 15 ml of lactated Ringer's solution. The arthroscopic portal for medial approach was then made along the arc of the trochlear notch approximately 1 cm distal to the prominence of the epicondyle. A stab incision was made at this point with a No. 11 Bard Parker blade and the sleeve with sharp trocar was inserted through the incision. Following entry into the joint the trocar was removed and the telescope was coupled with the cannula (Bardet, 1997[a]).

3.4.4.3 Stifle joint

The patient was positioned in dorsal recumbency with the pelvis placed at the end of the operating table to facilitate unobstructed access to both sides of the stifle. The cranial portion of the stifle was prepared for aseptic surgery and draped. A cranio-lateral approach was chosen. An 18G hypodermic needle

was inserted into the supra-patellar pouch and synovial fluid withdrawn. The joint was then distended with 10 to 20 ml of lactated Ringer's solution. A stab incision was made lateral to the patellar tendon mid way between the distal aspect of the patella and the tibial crest. The cannula with the sharp trocar was directed from distally cranio-lateral to proximally cranio-medial into the joint. As soon as the joint capsule was penetrated, the sharp trocar was replaced with the blunt obturator. The joint was extended and the blunt trocar was directed parallel to the trochlear groove into the patello-femoral joint. The trocar was then replaced with the arthroscope. The egress needle was inserted into the proximal patellar joint space parallel to the patella.

3.5 Parameters

3.5.1 Incidence

Breed, age and sex wise distribution of affections of the shoulder, elbow and stifle were recorded.

3.5.2 Physical and clinical examination

3.5.2.1 Pain on extension and flexion

The affected joints were subjected to extension and flexion to assess pain and resistance to the extension and flexion and compared with the contra lateral limb.

3.5.2.2 Grading of lameness

The lameness of the affected limb was graded as described by Lewis *et al.* (1987) pre arthroscopically.

3.5.3 Haematological evaluation

The following haematological tests were carried out pre and postoperatively and estimated as described by Chauhan (1995).

3.5.3.1 Packed cell volume (PCV)

Packed cell volume was estimated in percentage by microhaematocrit method.

3.5.3.2 Haemoglobin (Hb)

Haemoglobin was estimated in g/dl by Hellige-Sahli haemoglobinometer method.

3.5.3.3 Total erythrocyte count

Erythrocyte count in million per cubic millilmeter (10^6/cumm) was estimated using Hayem's fluid as diluent and haemocytometer.

3.5.3.4 Total leucocyte count

The total leucocyte count per cubic millimeters was counted by standard dilution technique using Thomas fluid and haemocytometer.

3.5.3.5 Differential count

Blood smear obtained was stained with Leishman's stain for differential count.

3.5.3.6 Erythrocyte sedimentation rate (ESR)

Erythrocyte sedimentation rate was estimated using Wintrobe's tube.

3.5.4 Serum biochemistry

Blood samples were collected for serum biochemistry pre operatively and post operatively. Biochemical estimations were done in a semi autoanalyser.

3.5.4.1 Serum glucose

Serum glucose was estimated by the GOD/POD method (Trinder, 1969).

3.5.4.2 Serum alkaline phosphatase

Serum alkaline phosphatase was estimated by kinetic photometric test (Tietz *et al.,* 1983).

3.5.4.3 Serum total protein and albumin

Serum total protein and albumin concentrations were estimated by modified Biuret method (Doumas *et al.,* 1971).

3.5.4.4 Serum globulin

Serum globulin level was estimated by subtracting albumin content from the total protein level (Benjamin, 1985).

3.5.5 Synovial fluid

Synovial fluid collection and analysis were done simultaneously with blood biochemistry in order to facilitate comparison (Coles, 1986). Owner's compliance was a limiting factor in deciding the post operative collection of synovial fluid. The collected synovial fluid was subjected to physical, biochemical and cytological test.

3.5.5.1 Physical tests

The following physical properties of synovial fluid were analysed and interpreted as per the procedures described by Coles (1986).

- Volume
- Colour
- Turbidity
- Viscosity
- Mucin clot test

3.5.5.2 Biochemical tests

The synovial fluid collected was subjected to biochemical tests for estimating the glucose, alkaline phosphatase, total protein, albumin and globulin.

Glucose

The glucose content in synovial fluid was estimated by GOD/POD method (Trinder, 1969).

Alkaline phosphatase

The alkaline phosphatase content in synovial fluid was estimated by Kinetic Photometric test (Tietz *et al.*, 1983).

Total protein

The total protein content in synovial fluid was estimated by modified Biuret method (Doumas *et al.*, 1971).

Globulin

The globulin content in synovial fluid was estimated by subtracting albumin content from the total protein level (Benjamin, 1985).

Cytology

Total nucleated cell count was performed with samples diluted with Thomas fluid in a WBC pipette, using a haemocytometer. Cytological examination along with differential count was performed using smears stained with Leishman's stain.

3.6 Statistical analysis

The data obtained were analysed statistically by unequal completely ramdomised design and paired `t' test as described by Snedecor and Cochran (1994).

Results

CHAPTER - IV

RESULTS

4.1 Incidence

Breed, sex and age wise incidence of shoulder, elbow and stifle joint affections are presented in table 3.

Among the twelve shoulder joint affections the breed wise incidence was three German Shepherd Dogs, five Great Danes, two Labradors, one Boxer, one Dobermann Pinscher and one Spitz. All the German Shepherd dogs were females, all the Great Dane dogs were males, Boxer and the Spitz were also males and the Dobermann Pinscher was a female. Amongst the males, six were in the age group below 2 years and two were between 2 and 6 years of age. Two females were below 2 years of age and one was between 2 and 6 years and one was above 6 years (Fig.7).

Among the twelve elbow joint affections the breed wise incidence was three German Shepherds, three Great Danes, three Dobermann Pinchers, one Rajapalayam and two Labradors. Of the German Shepherd Dogs two were males and one was female, of the Great Danes all three were males and the Rajapalayam was male. Among the Dobermanns one was male and two were females, and both Labradors were males. The male German Shepherd Dogs were both below 2 years of age and the female German Shepherd Dogs was in the 2 and 6 years age group. The three male Great Danes were all below 2 years of age as also the two

male Labradors. The single Doberman male and the single Rajapalayam male were also below two years of age (Fig.8).

Among the twelve stifle joint affections, the breed wise distribution was 5 German Shepherds, 1 Dobermann, one Boxer, 2 Rajaypalayams and 3 Spitz. Among the German Shepherds two were males and three females, the two Rajapalayams were males, among the Spitz one was male and two were females the Boxer and the Dobermann were males. The age wise incidence among the German Shepherd males one was below 2 and one was between 2 and 6 years of age, the German Shepherds females were below 2 years of age. One male Boxer one Dobermann and two Rajapalayam males were below 2 years of age and one male Spitz was in the age group of 2 to 6 and two females were above 6 years of age (Fig.9).

The present study revealed that shoulder joint affections were higher in large breeds of dogs weighing over 25kg body weight comprising 83.33% Medium and small breeds accounted for 8.33% each.

In elbow joint affections the incidence was higher in dogs of large breeds comprising 91.67%, whereas medium sized breeds represented 8.33% of the cases

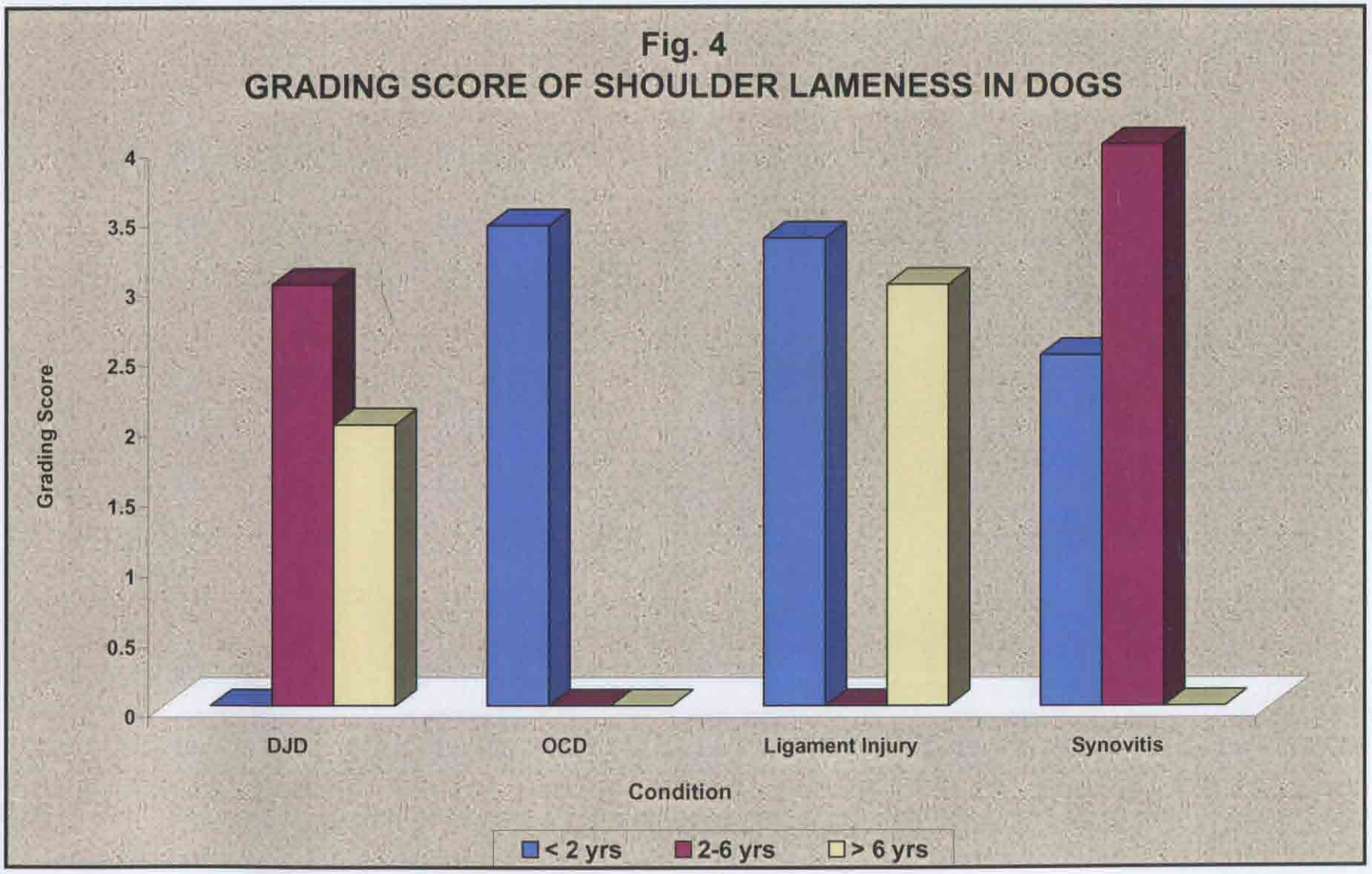
In the stifle joint affections the incidence was higher in large breed dogs comprising 50%, whereas medium and small sized breeds represented 25% each.

TABLE - 2

Mean ± S.E grading score of lameness due to shoulder, elbow and stifle affection in dogs

Condition	Shoulder			Elbow			Stifle			Total Mean ± S.E.
	<2 years	2-6 years	>6 years	<2 years	2-6 years	>6 years	<2 years	2-6 years	>6 years	
DJD	-	3.00 ± 0.00	2.00 ± 0.00	-	2.50 ± 0.36	2.00 ± 0.50	-	3.33± 0.58	3.00 ± 0.00	2.64 ± 0.51
OCD	3.42 ± 0.53	-	-	3.50 ± 0.50	-	-	3.00 ± 0.00	-	-	3.31± 0.22
Ligament Injury	3.33 ± 0.58	-	3.00 ± 0.00	-	-	-	-	3.33± 0.58	-	3.22 ± 0.16
Synovitis	2.50 ± 0.36	4.00 ± 0.00	-	3.33 ± 0.58	-	-	-	3.00 ± 0.00	-	3.21 ± 0.54
Total mean ± S.E.	3.08 ± 0.41	3.50 ± 0.50	2.50 ± 0.50	3.42 ± 0.09	2.50 ± 0.36	2.00 ± 0.50	3.00 ± 0.00	3.22 ± 0.16	3.20 ± 0.00	

Fig. 4


GRADING SCORE OF SHOULDER LAMENESS IN DOGS
Grading Score
4
3.5
3
2.5
2
1.5
1
0.5
0
DJD
OCD
Ligament Injury
Synovitis
Condition
< 2 yrs
2-6 yrs
> 6 yrs

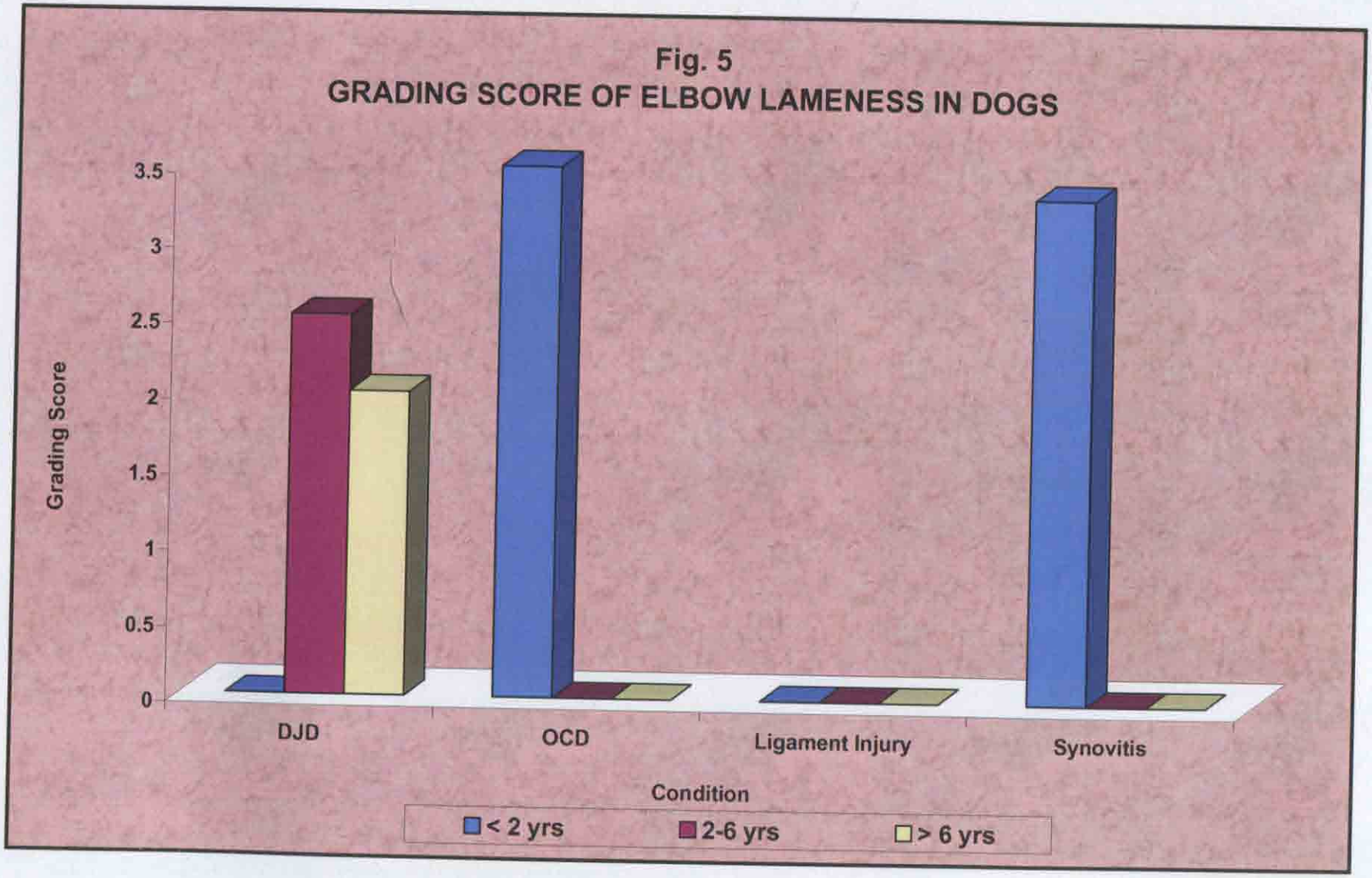


Fig. 5
GRADING SCORE OF ELBOW LAMENESS IN DOGS
Grading Score
3.5
3
2.5
2
1.5
1
0.5
0
DJD
OCD
Ligament Injury
Synovitis
Condition
< 2 yrs
2-6 yrs
> 6 yrs

Fig. 6

GRADING SCORE OF STIFLE LAMENESS IN DOGS

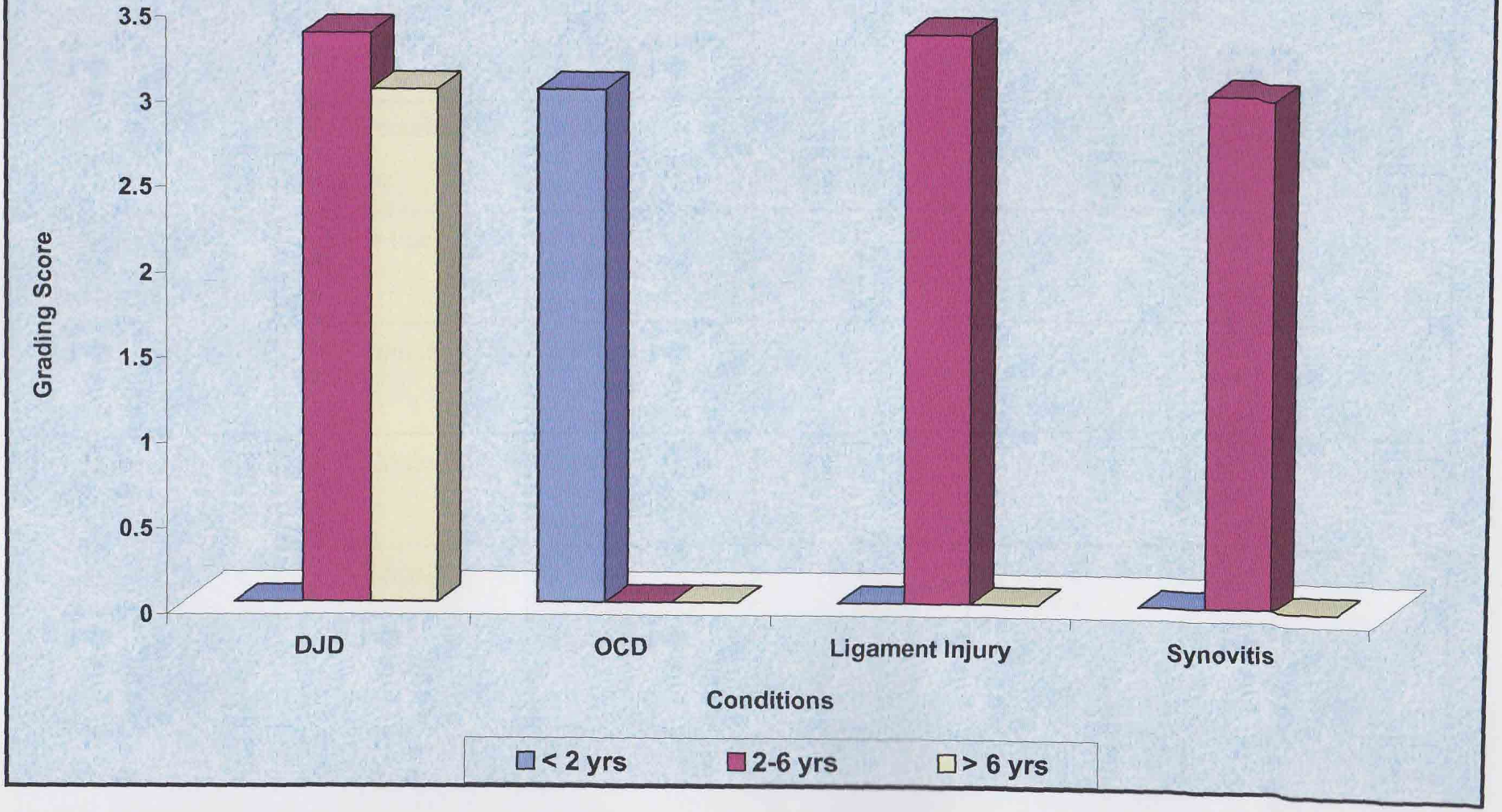


TABLE – 3

Breed, sex and age wise incidence of shoulders, elbow and stifle affections in dogs

Breed	Sex	Age	Shoulder	Elbow	Stifle
German Shepherd	Male	<2 years	-	2	1
		2-6 years	-	-	1
		>6 years	-	-	-
	Female	<2 years	1	-	3
		2-6 years	1	1	-
		>6 years	1	-	-
Great Dane	Male	<2 years	4	3	-
		2-6 years	1	-	-
		>6 years	-	-	-
	Female	<2 years	-	-	-
		2-6 years	-	-	-
		>6 years	-	-	-
Doberman	Male	<2 years	-	1	1
		2-6 years	-	-	-
		>6 years	-	-	-
	Female	<2 years	1	2	-
		2-6 years	-	-	-
		>6 years	-	-	-

Table 3 Contd

Breed	Sex	Age	Shoulder	Elbow	Stifle
Labrador	Male	<2 years	1	2	-
		2-6 years	-	-	-
		>6 years	-	-	-
	Female	<2 years	-	-	-
		2-6 years	-	-	-
		>6 years	-	-	-
Boxer	Male	<2 years	1	-	1
		2-6 years	-	-	-
		>6 years	-	-	-
	Female	<2 years	-	-	-
		2-6 years	-	-	-
		>6 years	-	-	-
Spitz	Male	<2 years	-	-	-
		2-6 years	1	-	1
		>6 years	-	-	-
	Female	<2 years	-	-	-
		2-6 years	-	-	-
		>6 years	-	-	2
Rajapalayam	Male	<2 years	-	1	2
		2-6 years	-	-	-
		>6 years	-	-	-
	Female	<2 years	-	-	-
		2-6 years	-	-	-
		>6 years	-	-	-

Fig. 7

BREED WISE INCIDENCE OF SHOULDER, ELBOW AND STIFLE JOINT AFFECTIONS IN DOGS

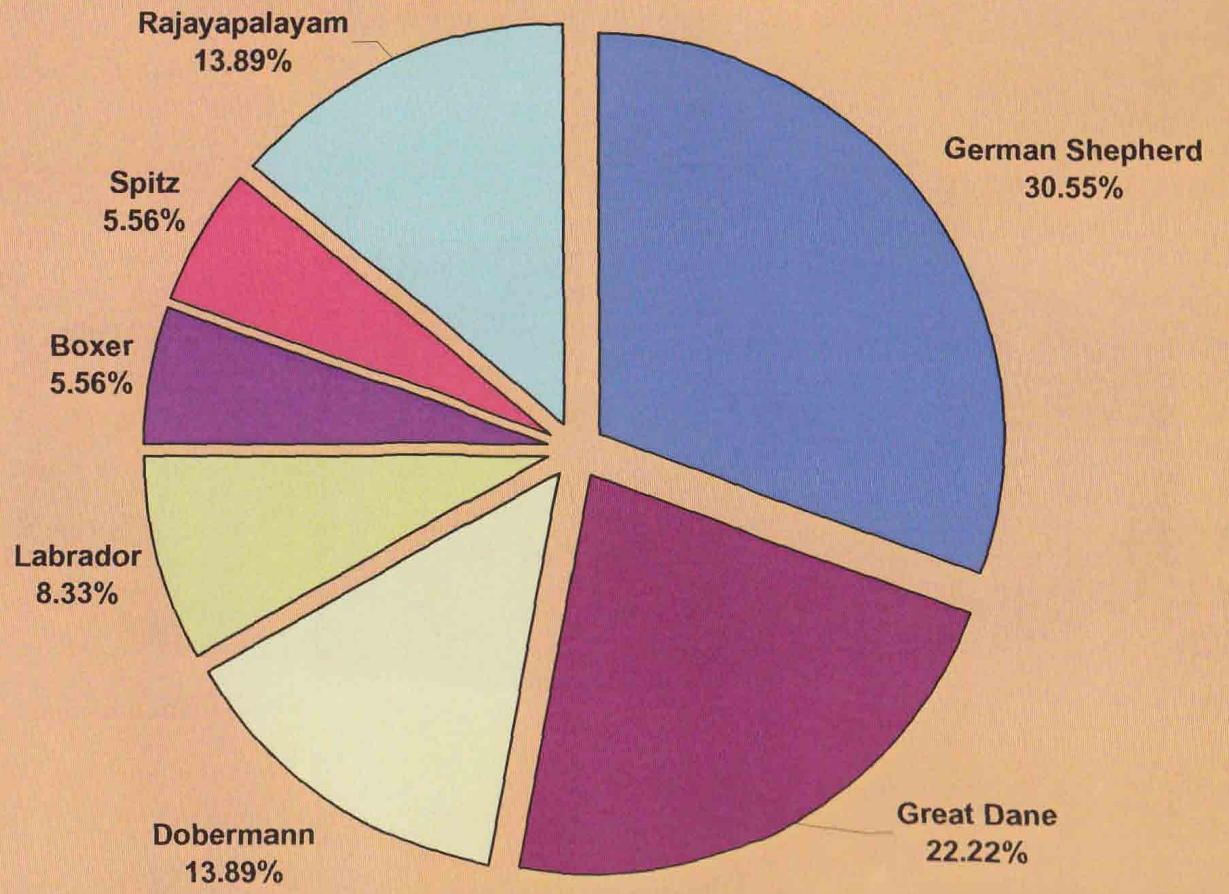
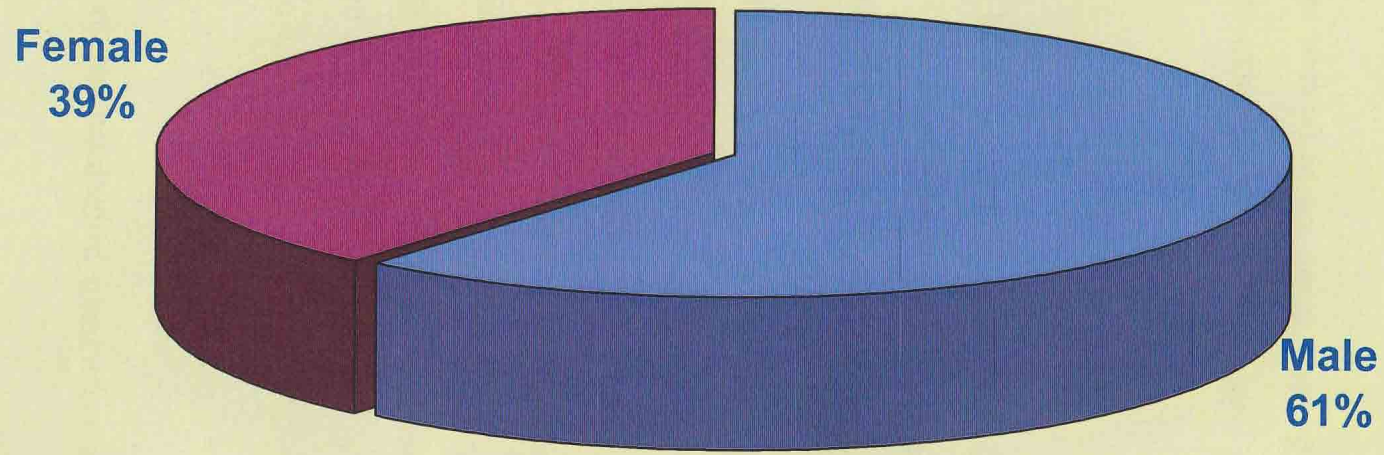


Fig. 8

SEX WISE INCIDENCE OF SHOULDER, ELBOW AND STIFLE JOINT AFFECTIONS IN DOGS



4.2 Pain on extension and flexion

Among the twelve animals with shoulder joint affections, all dogs evinced pain on hyperextension. Among the twelve elbow affections, pain-on flexion and extension of the elbow was noticed in eight cases and crepitation in four cases.

Gross joint distension was noticed in six cases. Among the twelve stifle joints examined all the animals exhibited pain on extension and thigh muscle wastage was seen in three dogs.

4.3 Grading of lameness

The total mean ± SE of lameness grading was 2.64 ± 0.51, 3.31 ± 0.22, 3.22 ± 0.16 and 3.21 ± 0.54 in degenerative joint disease, osteochondritis dissecans , ligament injury and synovitis respectively. (Table 2)

The total mean ± SE lameness grading in the dogs less than two years of age, 2-6 years and above six years 3.08 ±0.41, 3.50 ±0.50 and 2.50± 0.50 in shoulder lameness, 3.42 ±0.09, 2.50 ±0.36 and 2.00 ±0.50 in elbow lameness, 3.00± 0.00, 3.22± 0.16 and 3.00± 0.00 in stifle lameness respectively. The overall mean revealed higher lameness grade in osteochondritis dissecans, ligament injury and synovitis. Young dogs below two years of age and those dogs between two and six years of age showed a higher grade of lameness in shoulder, elbow and stifle joint affections.(Figure 4,5 and 6).

4.4 Haematological changes

The mean ± SE values of haematological parameters are presented in table 4.

4.4.1 Haemoglobin

The mean ± SE of haemoglobin in gms/ dl pre and post arthroscopically in degenerative joint disease was 10.06 ± 0.50 and 10.50 ± 0.31, osteochondrosis

Fig. 9

AGE WISE INCIDENCE OF SHOULDER, ELBOW AND STIFLE JOINT AFFECTIONS IN DOGS

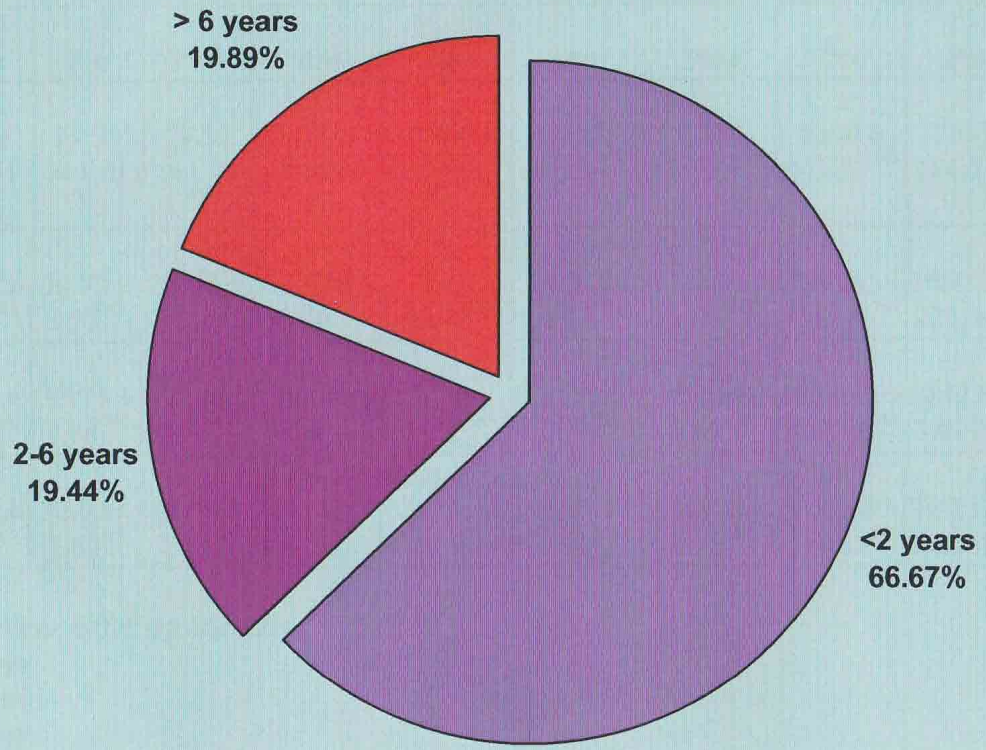


TABLE – 4

Mean ± SE values of haematological parameters

Condition/ Parameter	Haemoglobin g/dl		Packed cell volume%		Erythrocyte sedimentation rate mm/hr		Red blood cells 10^6/cumm		White blood cells 10^3/cu.mm		Neutrophils %		Lymphocytes %	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
DJD	10.06 ± 0.50 ^{NS}	10.50 ± 0.31 ^{NS}	29.33 ± 1.61 ^{NS}	29.33 ± 1.16 ^{NS}	6.78 ± 0.36^{a}	3.00 ± 0.29^{b}	4.99 ± 0.26 ^{NS}	5.24 ± 0.12 ^{NS}	8500 ± 941.22 ^{NS}	7744 ± 484.51 ^{NS}	61.11 ± 3.84^{NS}	64.00 ± 3.90^{NS}	35.44 ± 4.06^{NS}	35.19 ± 1.81^{NS}
OCD	10.16 ± 0.30^{NS}	10.35 ± 0.27^{NS}	30.92 ± 1.07^{NS}	29.92 ± 1.02^{NS}	2.62 ± 0.18^{NS}	2.69 ±0.17^{NS}	5.04 ± 0.17^{NS}	5.15 ± 0.09 ^{NS}	7584.61 ± 174.99 ^{NS}	7650.00 ± 223.46 ^{NS}	59.23 ± 1.79^{NS}	56.61 ± 1.54 ^{NS}	39.69 ± 4.89 ^{NS}	41.85 ± 1.70^{NS}
Ligament Injury	10.79 ± 0.51 ^{NS}	10.57 ± 0.29 ^{NS}	30.71 ± 1.78 ^{NS}	30.28 ± 1.48^{NS}	6.57 ± 0.43^{a}	3.57 ± 0.42 ^{B}	5.26 ± 0.23^{NS}	5.22 ± 0.15 ^{NS}	6828.57 ± 309.73 ^{NS}	6842.86 ± 346.99 ^{NS}	58.00 ± 1.73 ^{NS}	56.28 ± 1.92 ^{NS}	41.14 ± 1.56 ^{NS}	42.57 ± 2.13 ^{NS}
Synovitis	10.00 ± 0.53^{b}	10.85 ± 0.39^{a}	32.00 ± 1.57^{NS}	32.29 ± 1.34^{NS}	6.71 ± 0.36^{a}	3.29 ± 0.42^{B}	4.89 ± 0.40^{NS}	5.14 ± 0.18^{NS}	7850.00 ± 364.82^{NS}	7935.00 ± 322.69 ^{NS}	57.71 ± 3.68 ^{NS}	62.57 ± 1.89 ^{NS}	40.14 ± 4.44 ^{NS}	36.00 ± 1.23 ^{NS}

Means bearing different superscripts differ significantly.
Post arthoroscopic – after 10 days
DJD – Degenerative joint disease.
OCD – Osteochondritis dissecans.
Pre – Pre arthroscopically
Post – Post arthroscopically

dissecans was 10.16 ± 0.30 and 10.35 ±0.27, ligament rupture was 10.79 ± 0.51 and 10.57 ± 0.29 and synovitis was 10.00 ± 0.53 and 10.85 ±0.39 respectively.

Statistical analysis revealed no significant changes between disease conditions and pre and post arthroscopically.

4.4.2 Packed cell volume

The mean ± SE of packed cell volume in per cent pre and post arthroscopically in degenerative joint disease was 29.33 ± 1.61 and 29.33 ± 1.61, osteochondrosis dissecans was 30.92 ± 1.07 and 29.92 ± 1.02, ligament rupture was 30.71 ± 1.78 and 30.28 ± 1.48 and synovitis was 32.00 ± 1.7 and 32.29 ± 1.34 respectively.

Statistical analysis revealed no significant changes between disease conditions and pre and post arthroscopically.

4.4.3 Erythrocyte sedimentation rate

The mean ± SE of erythrocyte sedimentation rate in mm/ 30 minutes pre and post arthroscopically in degenerative joint disease was 6.78 ± 0.36 and 3.00 ± 0.29, osteochondrosis dissecans was 2.62 ± 0.18 and 2.69 ± 0.17, ligament rupture was 6.57 ± 0.43 and 3.57 ± 0.42 and synovitis was 6.71± 0.36 and 3.29 ± 0.42 respectively.

Statistical analysis revealed significant decrease in erythrocyte sedimentation rate in osteochondrosis dissecans ($P<0.05$). However between pre and post arthroscopic procedures in mean erythrocyte sedimentation rate values

decreased significantly during post arthroscopic period in degenerative joint disease, ligament injury and synovitis ($P<0.05$).

4.4.4 Total erythrocyte count

The mean ± SE of total erythrocyte count in 10^6/ ml post arthroscopically in degenerative joint disease was 4.99 ± 0.26 and 5.24± 0.12, osteochondrosis dissecans was 5.04± 0.17 and 5.15 ± 0.09, ligament rupture was 5.26 ± 0.23 and 5.22 ± 0.15 and synovitis was 4.89 ± 0.40 and 5.14 ± 0.18 respectively.

Statistical analysis revealed no significant changes between disease conditions and pre and post arthroscopically.

4.4.5 Total leucocyte count

The mean ± SE of total leucocyte count, in no./ml pre and post arthroscopically in degenerative joint disease was 8500 ± 941.22 and 7744 ±484.51, osteochondrosis dissecans was 7584.61 ± 174.99 and 7650.00 ±223.46 ligament rupture was 6828.57 ±309.73 and 6842.86 ±346.99, and synovitis was 7850.00 ±364.82 and 7935 ±322.69 respectively.

Statistical analysis revealed no significant changes between disease conditions and pre and post arthroscopically.

4.4.6 Neutrophils

The mean ± SE of neutrophils in percentage pre and post arthroscopically in degenerative joint disease was 61.11 ±3.84 and 64.00 ±3.90, osteochondrosis

dissecans was 59.23 ±1.79 and 56.61 ±1.54, ligament rupture was 58.00 ±1.73 and 56.28 ±1.92, and synovitis was 57.71 ±3.68 and 62.57 ±1.89 respectively.

Statistical analysis revealed no significant changes between disease conditions and pre and post arthroscopically.

4.4.7 Lymphocytes

The mean ± SE of lymphocytes in percentage pre and post arthroscopically in degenerative joint disease was 35.44 ±4.06 and 35.17 ±1.81, osteochondrosis dissecans was 39.69 ±4.89 and 41.85 ±1.70 ,ligament rupture was 41.14 ±1.56 and 42.57 ±2.13, and synovitis was 40.14 ±4.44 and 36.00 ±1.23 respectively.

Statistical analysis revealed no significant changes between disease conditions and pre and post arthroscopically.

4.5. Biochemical changes

The mean ± SE values of biochemical parameters are presented in table 5.

4.5.1 Total serum protein

The mean ± SE of total protein in g % pre and post arthroscopically in degenerative joint disease was 5.86 ±0.15 and 5.49 ±0.07, osteochondrosis dissecans was 5.62 ±0.09 and 5.50 ±0.07, ligament rupture was 7.77 ±0.13 and 6.46 ±0.04, and synovitis was 7.61 ±0.16 and 6.06 ±0.28 respectively.

TABLE - 5

Mean ± SE values of biochemical parameters

Condition/ Parameter	Total Protein g/dl		Albumin g/dl		Globulin g/dl		Glucose mg/dl		Alkaline phosphatase U/L	
	Pre	Post	Pre	Post	Pre	Post	Pre^{+}	Post^{+}	Pre	Post
DJD	5.86 ± 0.15^{qa}	5.49 ± 0.07^{rb}	2.7 ± 0.07^{q} NS	2.63 ± 0.10^{r} NS	3.17 ± 0.12^{qA}	2.86 ± 0.10^{pB}	86.56 ± 3.65^{NS}	87.89 ± 3.49^{NS}	73.78 ± 8.35^{q} NS	71.44 ± 7.22^{q} NS
OCD	5.62 ± 0.09^{q} NS	5.50 ± 0.07^{r} NS	2.76 ± 0.05^{q} NS	2.89 ± 0.06^{qr} NS	2.85 ± 0.07^{rA}	2.58 ± 0.07^{rq}	95.23 ± 2.59^{A}	89.85 ± 1.44^{B}	169.62 ± 16.73^{PA}	143.00 ± 16.35^{PB}
Ligament Injury	7.77 ± 0.13^{PA}	6.46 ± 0.04^{PB}	3.94 ± 0.19^{PNS}	4.06 ± 0.14^{PNS}	3.83 ± 0.08^{PA}	2.36 ± 0.13^{qB}	95.00 ± 2.60^{NS}	95.85 ± 1.08^{NS}	62.71 ± 8.26^{q} NS	65.85 ± 8.42^{q} NS
Synovitis	7.61 ± 0.16^{PA}	6.06 ± 0.28 qB	3.64 ± 0.15^{p} NS	3.19 ± 0.13^{q} NS	3.97 ± 0.04^{PA}	2.76 ± 0.26^{pB}	95.29 ± 2.93^{NS}	92.86 ± 1.58^{NS}	70.00 ± 5.91^{qNS}	60.00 ± 3.93^{qNS}

Means bearing different superscripts for a parameter differ significantly.
Post arthoroscopic – after 10 days
DJD – Degenerative joint disease.
OCD – Osteochondritis dissecans.
Pre – Pre arthroscopically
Post – Post arthroscopically

Significant increase in total serum protein was observed in ligament rupture and synovitis pre arthroscopically ($P<0.05$). However the serum total protein decreased significantly post arthroscopically in degenerative joint disease and ligament injury ($P<0.05$).

4.5.2 Serum albumin

The mean ± SE of albumin in g % pre and post arthroscopically in degenerative joint disease was 2.7 ±0.07 and 2.63 ±0.10, osteochondrosis dissecans was 2.76 ±0.05 and 2.89 ±0.06, ligament rupture was 3.94 ±0.19 and 4.06 ±0.14 and synovitis was 3.64 ±0.15 and 3.19 ±0.13 respectively.

The serum albumin level was significantly higher in ligament injury ($P<0.05$) but no significant difference could be observed in synovitis between pre and post arthroscopically.

4.5.3 Serum globulin

The mean ± SE of globulin in g % pre and post arthroscopically in degenerative joint disease was 3.17 ±0.12 and 2.86 ±0.10, osteochondrosis dissecans was 2.85 ±0.07 and 2.58 ±0.07, ligament rupture was 3.83 ±0.08 and 2.36 ±0.13, and synovitis was 3.97 ±0.04 and 2.76 ±0.26 respectively.

The serum globulin level was higher in ligament injury and synovitis ($P<0.05$). However globulin level decreased post arthroscopically ($P<0.05$).

4.5.4 Blood gluclose

The mean ± SE of glucose in mg/100mL pre and post arthroscopically in degenerative joint disease was 86.56±3.65 and 87.89±3.49, osteochondrosis

dissecans was 95.23 ± 2.59 and 89.85 ± 1.44, ligament rupture was 95.00 ± 2.60 and 95.85 ± 1.08, and synovitis was 95.29 ± 2.93 and 92.86 ± 1.58 respectively.

The mean blood glucose level did not vary significantly between conditions and between pre-and post-arthroscopically.

4.5.5 Serum alkaline phosphatase

The mean ± SE of serum alkaline phosphatase in U/ L, pre and post arthroscopically in degenerative joint disease was 73.78 ± 8.35 and 71.44 ± 7.22 osteochondrosis dissecans was 169.62 ± 16.73 and 143.00 ± 16.35, ligament rupture was 62.71 ± 8.26 and 65.85 ± 8.42, and synovitis was 70.00 ± 5.91 and 60.00 ± 3.73 respectively.

The serum alkaline phosphatase was significantly higher in OCD, when compared with other conditions during pre-arthroscopically ($P<0.05$). In osteochondrosis dissecans post-arthroscopically the mean decreased significantly when compared to pre-arthroscopic values ($P<0.05$) but in other conditions the means did not vary.

4.6 Synovial fluid

The mean ± SE values of physical parameters of synovial fluid are presented in table 6 and 7.

TABLE - 6

Physical appearance of synovial fluid in different disease conditions in dogs

Condition	Colour	No. of Cases		Turbidity	No. of Cases		Viscosity	No. of Cases		Mucin Clot Test		
		Pre Op.	Post Op.		Pre Op.	Post. Op		Pre. Op	Post. Op	Grading	Pre. Op	Post. Op
Degenerative Joint Disease	Colourless	5	-	Clear	9	9	4 +	4	9	Good	4	8
	Pale yellow	2	9	Flocculent	-	-	3+	5	-	Fair	5	1
	Yellow	2	-	Turbid	-	-	2+			Poor		
							1+					
Osteochondritis dissecans	Colourless	3	3	Clear	13	13	4 +	10	13	Good	7	13
	Pale yellow	3	10	Flocculent	-	-	3+	3		Fair	6	
	Yellow	7	-	Turbid	-	-				Poor		
Ligament Inuury	Colourless	-	-	Clear	2	3	4+	-	3	Good	5	6
	Pale yellow	-	2	Flocculent	5	4	3+	5	4	Fair	2	1
	Yellow	2	5				2+	2	-	Poor	-	-
	Blood Tinged	5	-				1+	-	-			
Synovitis	Colourless	-		Clear	4	5	4+	2	2	Good	4	4
	Pale yellow	3	5	Flocculent	3	2	3+	5	5	Fair	3	3
	Yellow	4	2				2+			Poor	-	-
	Blood Tinged						1+					

4.6.1 Physical properties

4.6.1.1 Degenerative joint disease

Among the nine cases showing degenerative joint disease the colour of the synovial fluid in five cases was colourless, two were pale yellow and two were yellow preoperatively. Post –operatively all nine were pale yellow. The fluid was clear both preoperatively and post operatively. Viscosity was normal in four and slightly reduced in five cases out of nine pre-operatively and all cases were normal post operatively. The mucin clot test was grade as good in four cases and fair in five cases preoperatively and good in eight and fair in one post operatively.

Among the thirteen cases of osteochondritis dissecans the synovial fluid colour was colourless in three, pale yellow in three, and yellow in seven pre operatively and colourless in three pale yellow in ten post operatively.

4.6.1.2 Osteochondritis dissecans

All the thirteen cases were clear showing no signs of turbidity both preoperatively and postoperatively. Viscosity was good in ten cases and slightly reduced in three cases preoperatively and all were normal postoperatively. The mucin clot test was rated good in seven cases and fair in six cases preoperatively and good in thirteen cases.

4.6.1.3 Ligament injury

Among the seven cases of ligament injury, the colour of the synovial fluid was yellow in two cases and blood tinged in five cases preoperatively and

pale yellow in two and yellow in five post-operatively. The fluid was clear in two and flocculence was seen in five cases pre-operatively and three were clear and four were flocculent post operatively. Viscosity was reduced mildly in five cases and reduced comparatively in two cases pre-operatively and three were normal and four were slightly reduced post-operatively. The mucin clot test was rated good in five and fair in two pre-operatively and good in six and fair in one post-operatively.

4.6.1.4 Synovitis

Among the seven cases of synovitis, the colour of the synovial fluid was pale yellow in three cases and yellow in four cases pre operatively, and pale yellow in five and yellow in two post operatively. The synovial fluid was clear in four and flocculence was seen in three cases pre operatively and five were clear and two flocculent post operatively. Viscosity was reduced in five cases and normal in two pre operatively and post operatively. Mucin clot test was rated to be good in four cases and fair in three pre operatively and post operatively.

4.6.2 Cytological examination

The mean ± SE values of synovial fluid cytology are presented in table 7.

4.6.2.1 Volume

The mean ± SE of synovial fluid volume in ml pre and post arthroscopically in degenerative joint disease was 0.67 ± 0.07 and 0.22 ± 0.009, osteochondrosis dissecans was 0.87 ± 0.78 and 0.21 ±0.009, ligament rupture was

TABLE – 7

Volume and cytology of synovial fluid of in dogs in different disease conditions in dogs

Condition/ Parameter	Volume ml		White Blood cells 10^3/cmm		Polymorphonuclear cells %		Mononuclear cells %	
	Pre	Post	Pre	Post	Pre$^+$	Post	Pre$^+$	Post
DJD	0.67 ± 0.07^{b}	0.22 ± 0.009^{c}	$3588.85 \pm 312^{AP\ NS}$	$486.66 \pm 26.28^{rB\ NS}$	8.33 ± 0.79^{APS}	3.89 ± 0.39^{q}	92.56 ± 0.78^{Bq}	96.11 ± 0.39^{pA}
OCD	0.87 ± 0.78^{b}	0.21 ± 0.009^{c}	2830.79 ± 115.68^{qNS}	416.15 ± 25.05^{rNS}	10.23 ± 0.74^{A}	3.54 ± 0.42^{qB}	89.77 ± 1.20^{B}	96.46 ± 0.42^{pA}
Ligament Injury	1.93 ± 0.11^{a}	0.96 ± 0.08^{b}	2057.14 ± 188.80^{rA}	1128.57 ± 87.19^{qB}	10.71 ± 1.08^{A}	8.71 ± 0.10^{pB}	89.29 ± 1.08^{B}	91.29 ± 0.99^{qA}
Synovitis	1.91 ± 0.27^{a}	1.61 ± 0.21^{a}	2728.57 ± 486.83^{qA}	2157.14 ± 643.69^{pB}	11.14 ± 1.39^{NS}	9.71 ± 1.86^{NS}	88.86 ± 1.39^{NS}	86.00 ± 1.91^{pNS}

Means bearing different superscripts in a parameters differ significantly.
DJD – Degenerative joint disease.
OCD – Osteochondritis dissecans.
Pre – Pre arthroscopically
Post – Post arthroscopically

1.93 ±0.11 and 0.96 ±0.08 and synovitis was 1.91 ±0.27 and 1.61 ±0.21 respectively.

The mean volume of synovial fluid did not vary between the conditions pre-arthroscopically. However significant reduction could be noticed during the post-arthroscopic period in degenerative joint disease, osteochondrosis dissecans and synovitis ($P<0.05$).

4.6.2.2 Total leucocyte count

The mean ± SE of synovial fluid total leucocyte count in number per ml pre and post arthroscopically in degenerative joint disease was 3588.85 ± 312.00 and 486.66 ± 26.28, osteochondrosis dissecans was 2830.79 ± 115.68 and 416.15 ± 25.05, ligament rupture was 2057.14 ± 188.80 and 1128.57 ± 87.19 and synovitis was 2728.57 ± 486.83 and 2157.14 ± 643.69 respectively.

The mean white blood cell count decreased significantly in ligament injury followed by synovitis, osteochondrosis dissecans and degenerative joint disease ($P<0.05$). After arthroscopy, the mean white blood cell count decreased in ligament rupture and synovitis.

4.6.2.3 Polymorphonuclear cells

The mean ± SE of polymorphonuclear cells of synovial fluid in percentage pre and post arthroscopically in degenerative joint disease was 8.33 ±0.79 and 3.89 ± 0.39, osteochondrosis dissecans was 10.23 ± 0.74 and 3.54 ± 0.42, ligament rupture was 10.71 ± 1.08 and 8.71 ± 0.10 and synovitis was 11.14 ± 1.39 and 9.71 ± 1.86 respectively.

The polymorphonuclear cells increased significantly in synovitis when compared with ligament injury, degenerative joint disease and osteochondrosis dissecans ($P \leq 0.05$). Following athroscopy polymorphonuclear cells decreased significantly in degenerative joint disease, osteochondrosis dissecans and ligament injury ($P \leq 0.05$).

4.6.2.4 Mononuclear cells

The mean ±SE of mononuclear cells in the synovial fluid in percentage pre and post arthroscopically in degenerative joint disease was 92.56 ± 0.78 and 96.11 ± 0.39, osteochondrosis dissecans was 89.77 ±1.20 and 96.46 ±0.42, ligament rupture was 89.29 ±1.08 and 91.29 ±0.99 and synovitis was 88.86 ±1.39 and 86.00 ±1.91 respectively.

The mean mononuclear cells significantly increased following arthrosocpy in DJD, OCD, and ligament injury ($P<0.05$).

4.7 Synovial fluid - biochemical parameters

The mean ± SE values of biochemical parameters of synovial fluid are presented in table 8.

4.7.1 Total protein

The mean ±SE of total protein of synovial fluid in g per dl pre and post arthroscopically in degenerative joint disease was 3.16 ±0.19 and 1.87 ±0.15, osteochondrosis dissecans was 3.57 ±0.21 and 2.04 ±0.10, ligament rupture was 2.96 ±0.21 and 2.24 ±0.10 and synovitis was 3.80 ±0.23 and 3.03 ±0.37 respectively.

TABLE – 8

Biochemistry of synovial fluid in different disease conditions in dogs

Condition/ Parameter	Total Protein g/dl		Albumin g/dl		*Globulin g/dl*		Glucose mg/dl		Alkaline Phosphatase U/L	
	Pre	Post	Pre^{+}	Post	Pre^{+}	Post	Pre^{+}	Post	Pre^{+}	Post
DJD	3.16 ± 0.19^{pq}	1.87 ± 0.15^{q}	1.22 ± 0.12^{NS}	1.09 ± 0.09^{NS}	2.26 ± 0.12^{A}	0.78 ± 0.08^{Bq}	78.11 ± 3.16^{A}	116.00 ± 5.05^{Bpq}	147.78 ± 17.78^{Ap}	33.11 ± 3.35^{Bq}
OCD	3.57 ± 0.21^{Pq}	2.04 ± 0.10^{q}	1.39 ± 0.14^{NS}	1.18 ± 0.06^{NS} qr	2.10 ± 0.09^{A}	0.86 ± 0.05^{Bq}	83.31 ± 2.85^{A}	129.77 ± 4.72^{Bp}	169.62 ± 16.74^{Ap}	32.00 ± 2.65^{Bq}
Ligament Injury	2.96 ± 0.21^{q}	2.24 ± 0.10^{q}	1.04 ± 0.10^{A}	1.37 ± 0.06^{B} pq	1.89 ± 0.18^{A}	0.87 ± 0.09^{Bq}	77.00 ± 4.19^{A}	106.14 ± 4.61^{Bq}	86.00 ± 8.89^{Aq}	45.43 ± 4.16^{BPq}
Synovitis	3.80 ± 0.23^{p}	3.03 ± 0.37^{p}	1.40 ± 0.16^{NS}	1.60 ± 0.87^{NSp}	2.27 ± 0.12^{A}	1.43 ± 0.31^{Bp}	79.57 ± 3.26^{A}	101.14 ± 6.56^{Bq}	99.14 ± 9.15^{Aq}	60.29 ± 16.51^{Bp}

Means bearing different superscripts differ significantly.
DJD – Degenerative joint disease.
OCD – Osteochondritis dissecans.
Pre – Pre arthroscopically
Post – Post arthroscopically

The synovial fluid total protein significantly reduced in ligament injury during pre arthroscopic period ($P \leq 0.05$). However no variation could be noticed during pre and post arthroscopic procedure.

4.7.2 Albumin

The mean ± SE of albumin of synovial fluid pre and post arthroscopically in degenerative joint disease was 1.22 ± 0.12 and 1.09 ± 0.09, osteochondrosis dissecans was 1.39 ± 0.14 and 1.18 ± 0.06, ligament rupture was 1.04 ± 0.10 and 1.37 ± 0.06 and synovitis was 1.40 ±0.16 and 1.60 ±0.87 respectively.

The mean albumin level was significantly less in ligament injury when compared with the other three conditions ($P \leq 0.05$). However the means did not vary between pre and post arthroscopically except in ligament injury wherein significant increase was noticed ($P \leq 0.05$).

4.7.3 Globulin

The mean ± SE of globulin of synovial fluid in g / dl pre and post arthroscopically in degenerative joint disease was 2.26 ± 0.12 and 0.78 ± 0.08, osteochondrosis dissecans was 2.10 ± 0.09 and 0.86 ± 0.05, ligament rupture was 1.89 ± 0.18 and 0.87 ± 0.09 and synovitis was 2.27 ± 0.12 and 1.43 ± 0.31 respectively.

The mean globulin level was significantly less in ligament injury when compared with the other three conditions ($P \leq 0.05$). However, the means did not vary between pre and post arthroscopically except in ligament injury where significant increase was noticed ($P \leq 0.05$).

4.7.4 Glucose

The mean ± SE of glucose of synovial fluid in mg / dl pre and post arthroscopically in degenerative joint disease was 78.11 ± 3.16 and 116.00 ± 5.05, osteochondrosis dissecans was 83.31 ± 2.85 and 129.77 ± 4.72, ligament rupture was 77.00 ± 4.19 and 106.14 ± 4.61 and synovitis was 79.57 ± 3.26 and 101.14 ± 6.56 respectively.

No variations were noticed pre-arthroscopically in any of the four conditions. But the mean glucose level significantly increased post-arthroscopically in all the four conditions ($P \leq 0.05$).

4.7.5 Alkaline phosphatase

The mean ± SE of alkaline phosphatase of synovial fluid in U/L pre and post arthroscopically in degenerative joint disease was 147.78 ± 17.78 and 33.11 ± 3.35, osteochondrosis dissecans was 169.62 ± 16.74 and 32.00 ± 2.65, ligament rupture was 86.00 ± 8.89 and 45.43 ± 4.16 and synovitis was 99.14 ± 9.15 and 60.29 ± 16.51 respectively.

Significant increase in alkaline phosphatase was noticed in degenerative joint disease and osteochondrosis dissecans pre arthroscopically ($P \leq 0.05$). But the mean values decreased post arthroscopically in degenerative joint disease, osteochondrosis dissecans and ligament injury ($P \leq 0.05$).

4.8 Instrumentation

4.8.1 Shoulder joint

Arthroscopic evaluation with 2.7 mm 30 forward oblique arthroscope with a 3.5 mm outside diameter sleeve with xenon light source of 300 W was

found to be useful in the visualization of the intra articular structures of the shoulder joint. The arthroscopic portal was 1 cm distal to the acromoan process. Irrigation of the joint with sterile lactated Ringer's solution through the arthropump which maintained the pressure of 5 to 75 mmHg was carried out. The structures visualized were glenoid articular surface and labrum, humeral head, greater and lesser tubercles and the intertubercular groove, tendon of origin of the biceps brachii, acromoan process, medial and caudal synovium and the cranial and caudal recess. The bicipital tendon could be observed for most of its link by directing the arthroscope cranially or rotating the limb externally as required. The caudal humeral head and joint pouch could be viewed by directing the arthroscope caudally and manipulating the joint through various ranges of flexion, extension and internal and external rotation.

4.8.2 Elbow joint

To visualize the elbow joint, the arthroscope and arthroscopic sleeve were of the same specifications. The egress port was midway between the humeral condyle and the most proximal part of the olecranon and the arthroscoic portal was 1 cm distal to and 0.5 cm caudal to the medial epicondyle of the humerus. The structures viewed were trochlea, medial half of the trochlear notch, anconeal process, coronoid process, medial aspect of the radial head and the synovium of the medial joint space. Internal rotation and abduction of the distal limb maximized examination of the joint.

4.8.3 Stifle joint

For arthroscopioc evaluation of the stifle joint the same standard telescope, light source and camera were used and the arthroscoic portal was

lateral to the patellar ligament and midway between the patellar and tibial tuberosity. The structures viewed were the medial and lateral recesses, trochlear ridges, articular surface of the patella, trochlear groove, femoral condyles, menesci, cranial and caudal cruciate ligament, long digital extensor and a limited portion of the tibial condylar articular surface.

4.9 Radiographic lesions

4.9.1 Shoulder joint

Out of the twelve shoulder joints studied radiographic lesions revealed periarticular and articular bony proliferation of glenoid cavity and humeral head suggestive of osteoarthritic changes in two cases. Radiolucent depression within the subchondral bone of the cranial articular surface of the humerus suggestive of chondromalacia was seen in one case. In one case flattening of humeral head with osteochondral defect (Plate 6) and a large calcified cartilage flap over the area in the caudal aspect of articular surface was seen (Plate 7). No radiographic lesion was noticed in other cases.

4.9.2 Elbow joint

Out of three cases of elbow degenerative joint disease, the radiographic findings were oblitertion of joint space and osteophyte formation of humeral condyles, radio-ulnar articular surface, coronoid process and humeral epicondylar region (Plate 8). In the two cases, increased soft tissue density of the elbow joint could be noticed. Remaining seven dogs revealed no appreciable radiographic lesions in the affected joint.

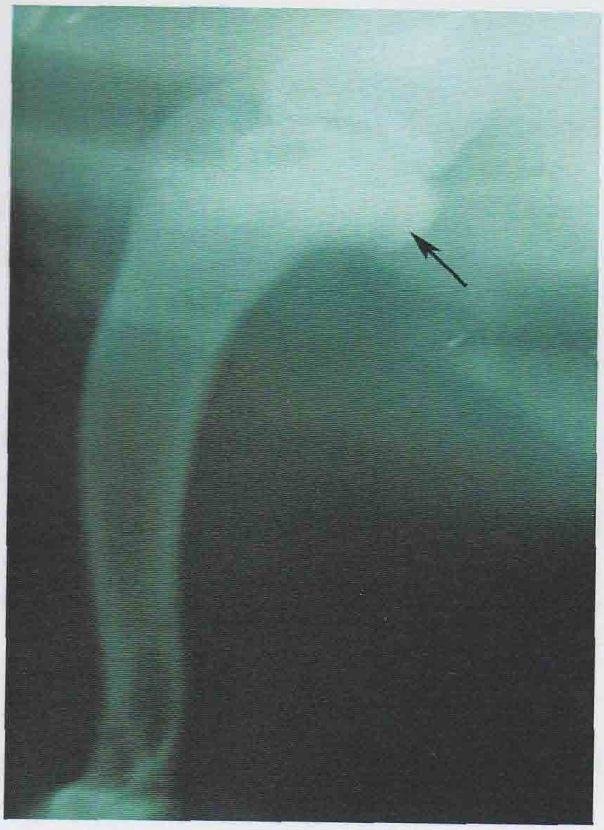


Plate - 6 : Skiagram of the shoulder joint of a dog showing osteochondritis dissecans (cartilage flap)

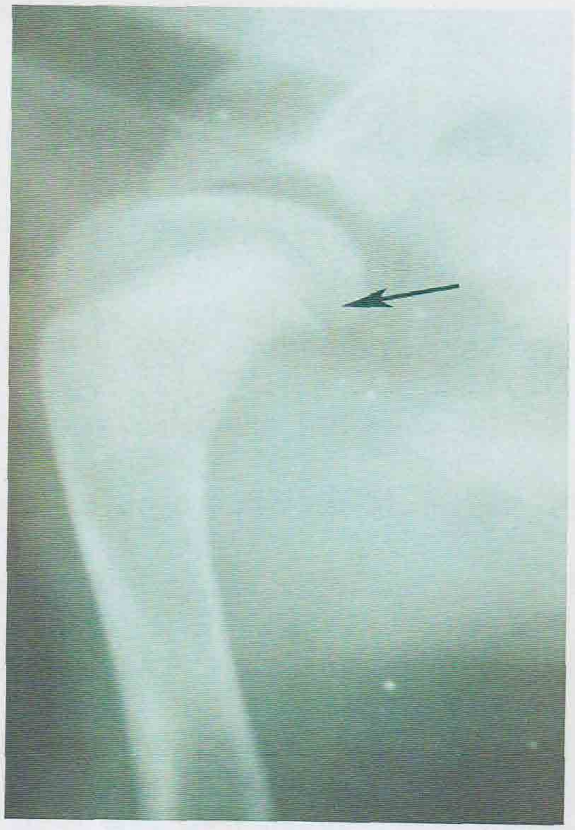


Plate - 7 : Skiagram of the shoulder joint of a dog showing osteochondritis dissecans

4.9.3 Stifle joint

In the stifle joint, all the four cases of degenerative joint disease could be diagnosed radiographically. Periarticular and periosteal proliferation with well defined margin at the joint capsule were noticed on the proximal aspect of the femoral trochlea, distal aspect of the patella, femoral condyles and proximal tibia (Plate 9 and 10). Narrowing and collapse of the joint space, malalignment of articular surface (Plate 11) were seen in two of the four cases. Cranial cruciate ligament rupture were seen in two cases and characterised by mild soft tissue swelling around the stifle joint with malalignment of proximal tibia and distal femur with proximal tibia displaced cranially in relation to the distal femur. There were no bony lesions seen in both cases. Soft tissue swelling with increased soft tissue density around the joint was evident in three cases which proved to be synovitis later.

4.10 Arthroscopic lesions

4.10.1 Shoulder joint

Among the twelve dogs exhibiting signs of shoulder lameness arthroscopic examination revealed the following lesions. Two dogs showed osteoarthritis suggestive of degenerative joint disease (Plate 12). Both dogs showed lesions of osteoarthritic changes of the joint with bony proliferation and osteophyte formation involving humeral head, glenoid cavity, and periarticular areas and acromion process. Fibrillation and flaking of articular cartilage of humeral head was seen in one case. Synovial proliferation and hyperaemia were seen in both the cases.

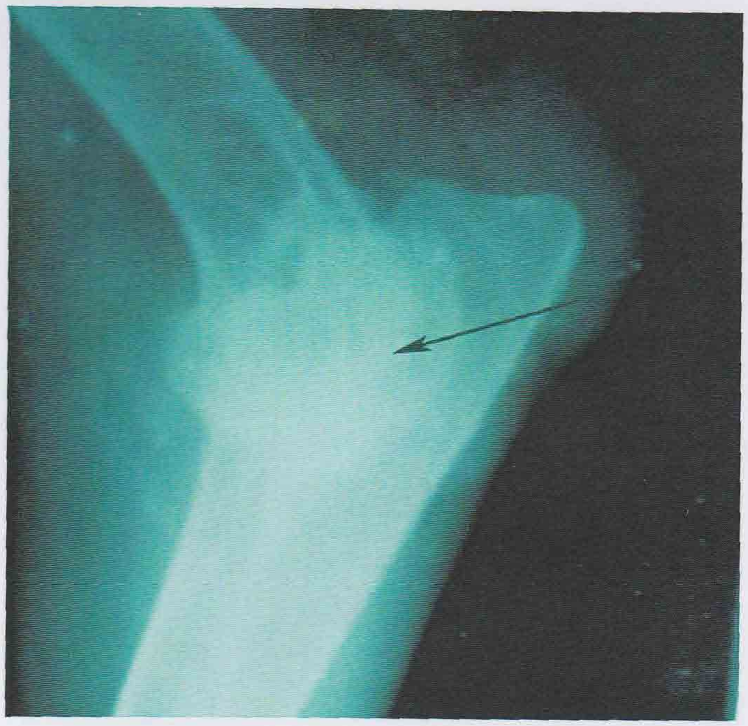


Plate - 8 : Skiagram of the elbow joint of a dog showing degenerative joint disease

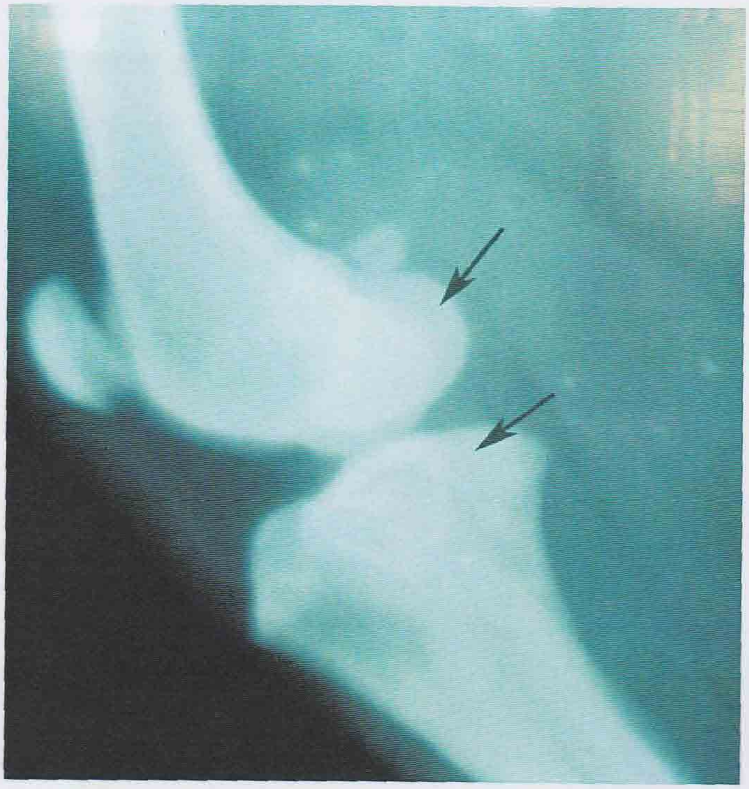


Plate - 9 : Skiagram of the stifle joint of a dog showing degenerative joint disease

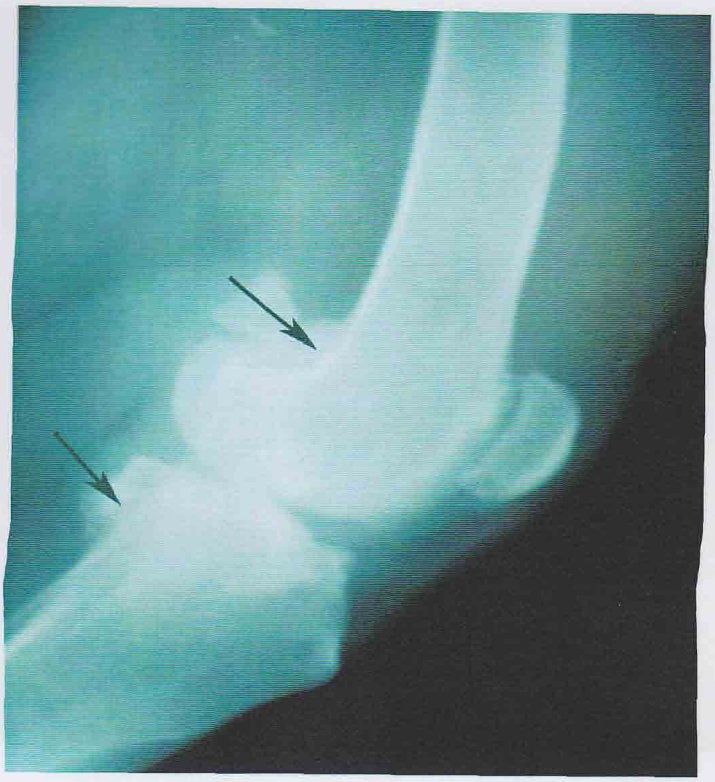


Plate - 10 : Skiagram of the stifle joint of a dog showing degenerative joint disease

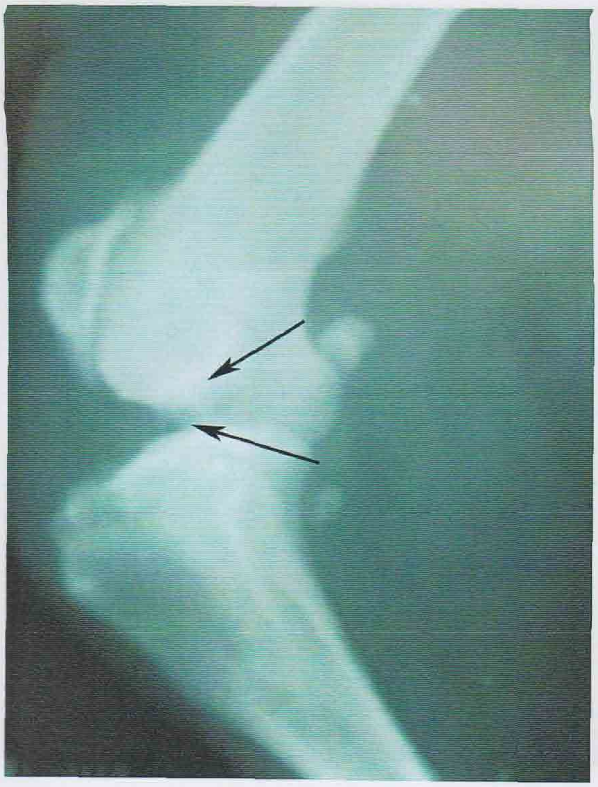


Plate - 11 : Skiagram of the stifle joint of a dog showing degenerative joint disease

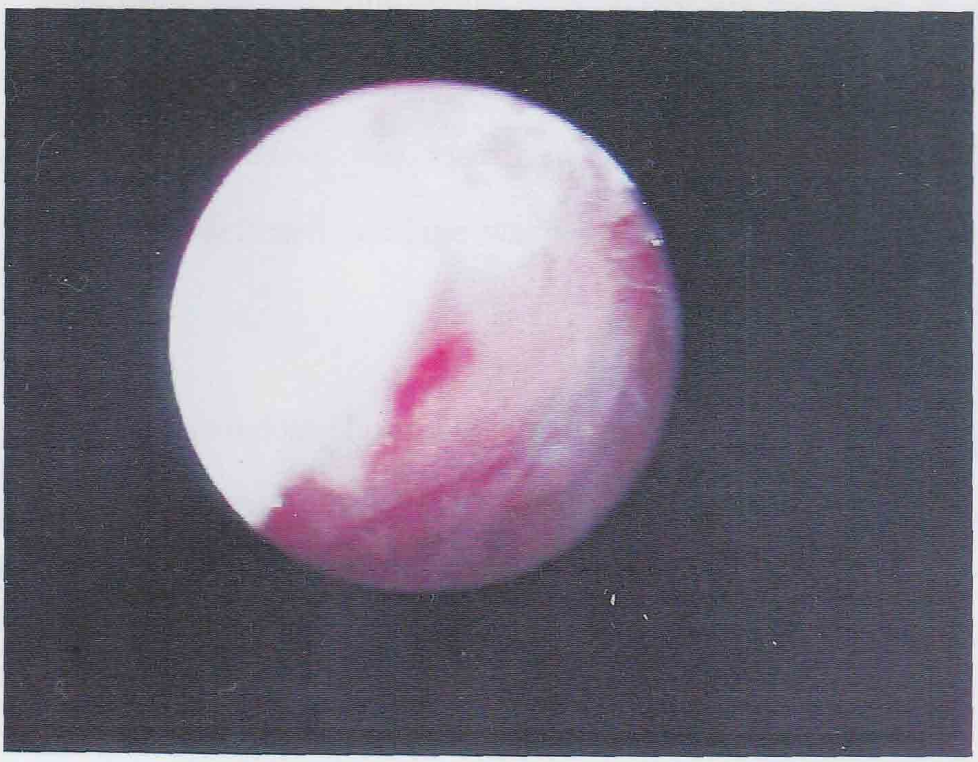


Plate - 12 : Arthroscopic picture of the shoulder joint of dog showing degenerative joint disease (osteoarthritis)

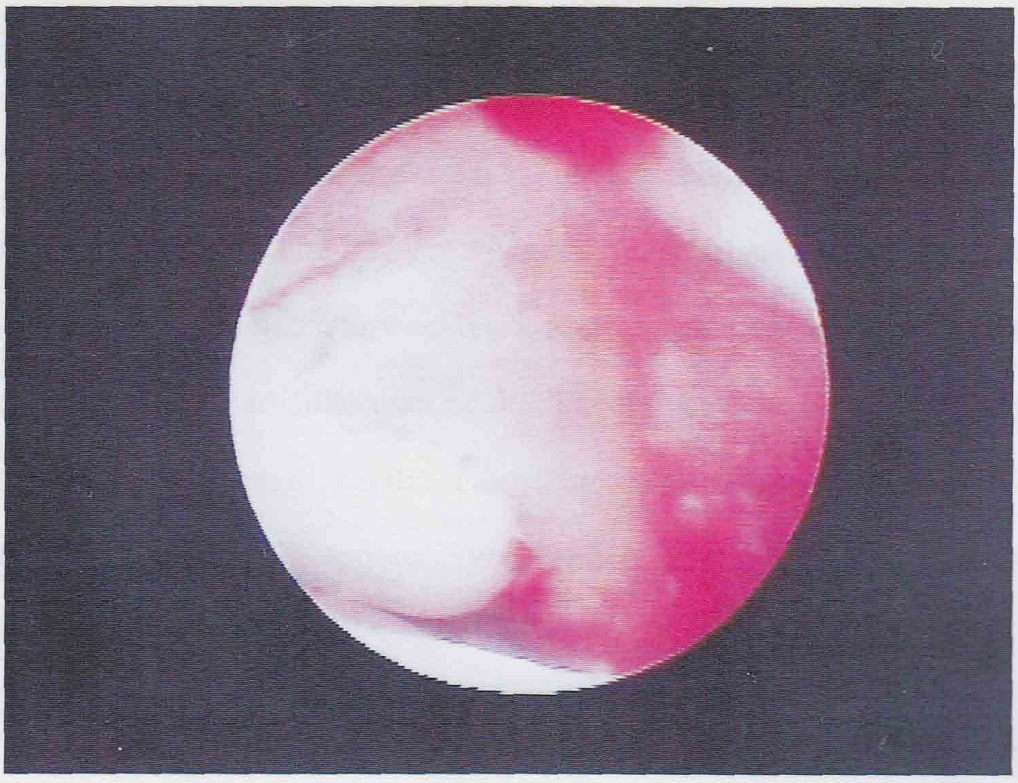


Plate - 13 : Arthroscopic picture of the shoulder joint of a dog showing osteochondritis dissecans (chondromalacia)

Four cases showed arthroscopic osteochondrosis dissecans. In one case, soft and spongy cartilage was visualised in which synovial proliferation in the cranial articular surface was visualised. In another case small irregular shallow, spongy, softened cartilage was seen on the caudal articular surface (Plate 13).

The other two dogs showed loosened and detached cartilage flaps (stage II of cartilage lesion) with synovial hyperemia and erosion of the articular surface (Plate 14).

Four dogs of this group showed ligament involvement of varying degrees. Of the four, one dog showed medial glenohumeral ligament rupture. The synovial membrane and villi were hyperemic and hyperplastic. Erosion of the articular surface was also observed (Plate 15). One dog showed rupture of the biciptal tendon with severe haemorrhage in the joint space.

In one case a large cartilagenous flap on the caudal aspect of the humeral head exposing a larger area of subchondral bone was seen. Calcification of the flap was visualised. A large calcified cartilagenous flap was seen in the caudal articular area of the humeral surface distal to the acromion process of the glenoid cavity. Fibrillation and fissures were seen on the cartilage adjacent to the lesions. Synovial hyperaemia and mild hyperplasia of synovium were seen.

In one case rupture of medial glenohumeral ligament was visualised at its origin at the glenoid cavity and the associated lesions were hyperaemia and hyperplasia of the synovium and erosion of the articular surface of the caudal

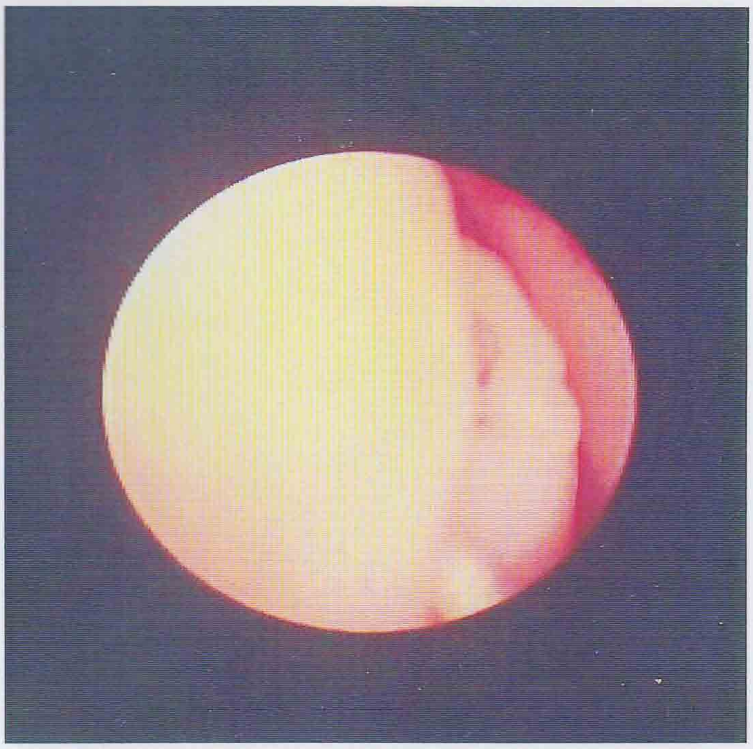


Plate - 14 : Arthroscopic picture of the shoulder joint of dog showing osteochondritis dissecans (detached cartilage flap)

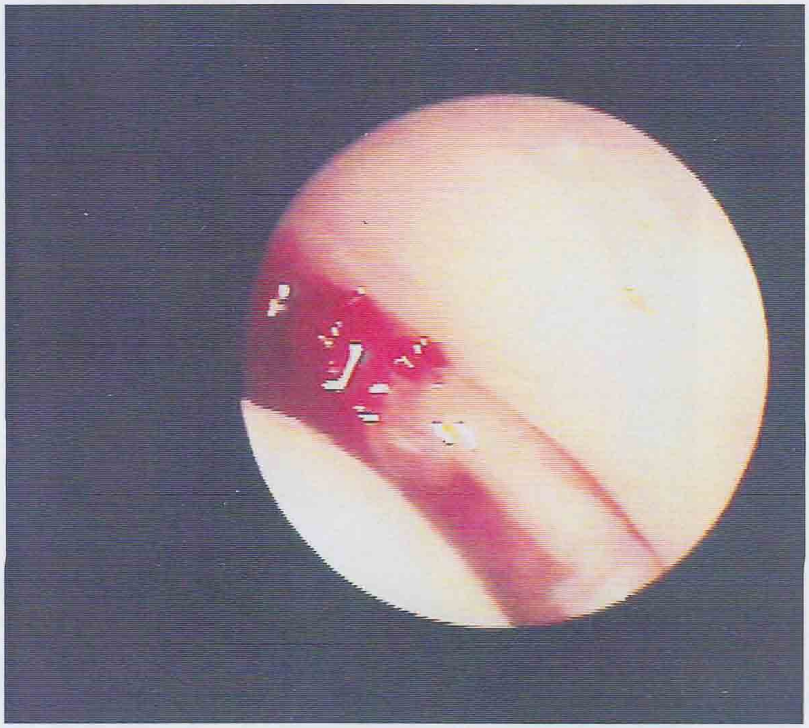


Plate - 15 : Arthroscopic picture of the shoulder joint of a dogshowing medial glenohumeral ligament rupture

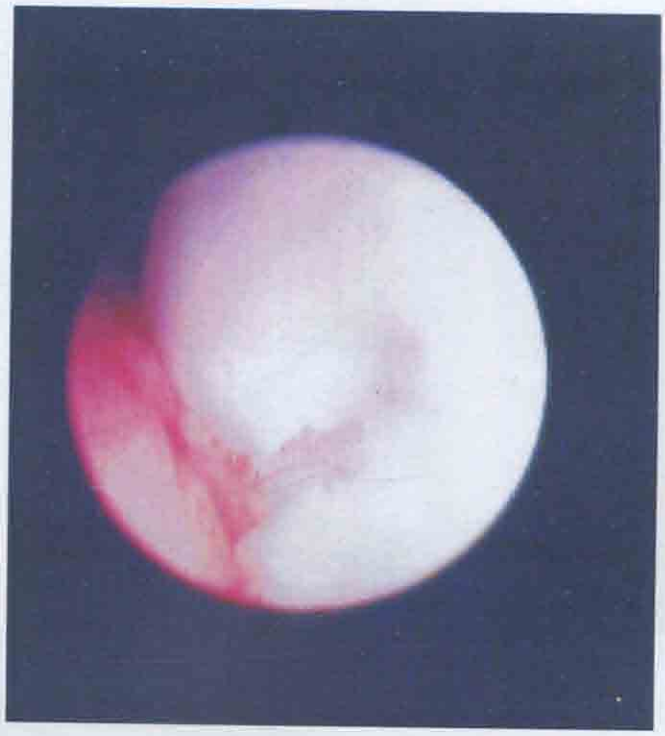


Plate - 16 : Arthroscopic picture of the shoulder joint of a dog showing bicipital tendinitis

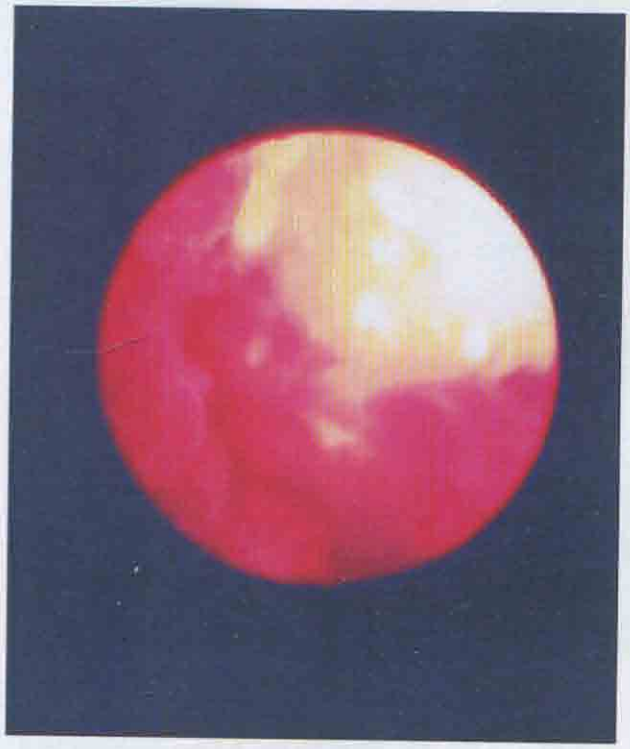


Plate - 17 : Arthroscopic picture of the shoulder joint of a dog showing bicipital tendon rupture

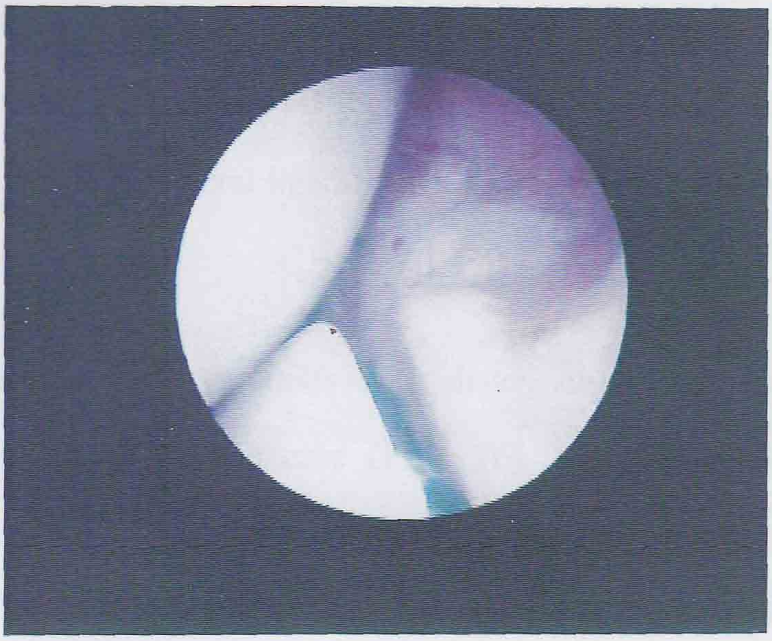


Plate - 18 : Arthroscopic picture of the elbow joint of a dog showing degenerative joint disease (osteoarthritis)

Plate - 19 : Arthroscopic picture of the elbow joint of a dog showing osteochondritis dissecans (chondromalacia)

humeral head exposing subchondral bone of the glenoid cavity together with softening and pitting of the articular cartilage. Both the cranial and caudal bands of the medial glenohumeral ligament were ruptured. Generalised distension of medioglenohumeral ligament with inflammation and hyperaemia was visualised in one case.

Generalised bicipital tendinitis with distension of the tendon with hyperaemia was noticed in one case (Plate 16). Bicipital tendon rupture intraarticularly associated with hyperaemia and hyperplastic synovial villi and haemorrhage in the joint space was noticed in one case (Plate 17). In two cases severe hyperaemia and hyperplasia of the synovium was seen suggestive of synovitis.

4.10.2. Elbow joint

Among the 12 dogs exhibiting signs of elbow lameness, arthroscopic examination revealed the following lesions. Three dogs showed osteoarthritis suggestive of degenerative joint disease. Osteophyte proliferation was seen in the articular and periarticular surface of the humeral condyle and radial and olecranon process of ulna. Erosive lesions of articular cartilage of humerus and radius with fibrillation and flaking of articular cartilage were observed (Plate 18). Excessive synovial proliferation with minimum sign of hyperaemia was noticed in all the three cases.

Six dogs showed lesions of osteochondritis dissecans. Of which two dogs showed lesions of chondromalacia. Soft, spongy cartilage was seen on the

medial condyle of the humerus with irregular articular surfaces. Synovial hyperaemia was seen in both cases (Plate 19).

In both cases of loose cartilaginous flap, severe hyperemia of the synovium and mild erosion of articular cartilage were seen on the medial condyle of the humerus. In another case, loose cartilage separated as a cartilage flap from the articular cartilage exposing the periosteum of cranial aspect of the medial condyle. Displaced coronoid process was seen in one case and the arthroscopic lesion noticed was the large displaced fragment of the top of the medial coronoid process. The fractured sites were sharp and haemorrhagic (Plate 20 and 21). The associated lesions were hyperplastic synovial villi and humeral head cartilage erosion.

Fragmented left medial coronoid process with cartilaginous erosion of the lateral rim of the medial coronoid process was seen in one case. Attachment of the fragments to the annular ligaments was visible. Mild hyperaemia of synovia was observed. In two cases, severe hyperemic synovium with hyperplasia of the synovial villi suggestive of synovitis was noticed (Plate 22).

4.10.3. Stifle joint

Among the 12 stifle joints four cases showed lesions of degenerative joint disease. The lesions were osteophyte formation, bony proliferation and discolouration of articular surface with fibrillation. Flaking of articular cartilage was also noticed on the articular and periarticular surface of femoral condyle, tibial and distal trochlea of the femur (Plate 23).

One case showed soft and spongy cartilage at the femoral condyle with hyperaemia of synovial villi suggestive of chondromalacia (Plate 24). In one

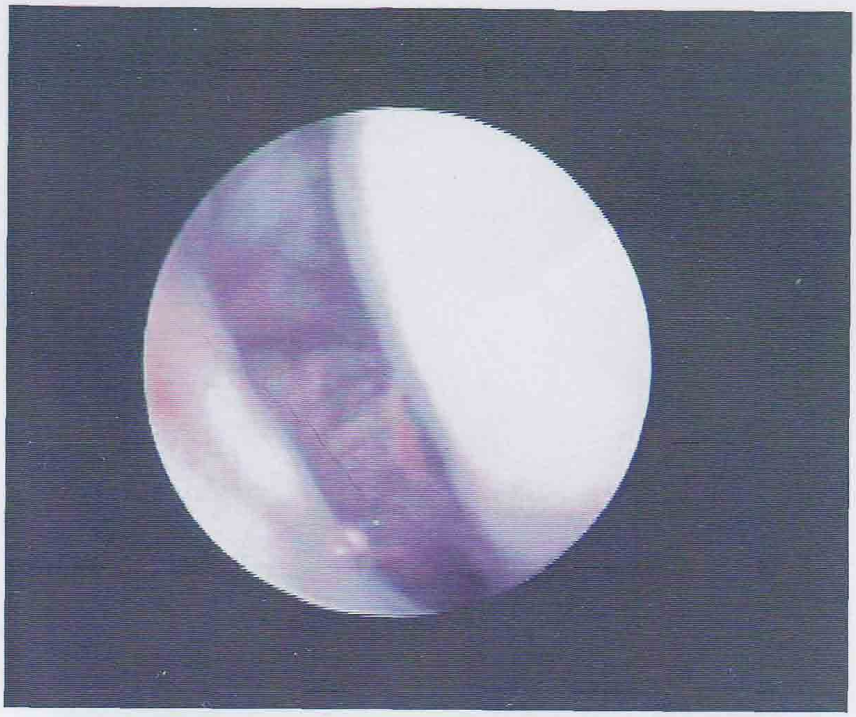


Plate - 20 : Arthroscopic picture of the elbow joint of a dog showing fragmented coronoid process

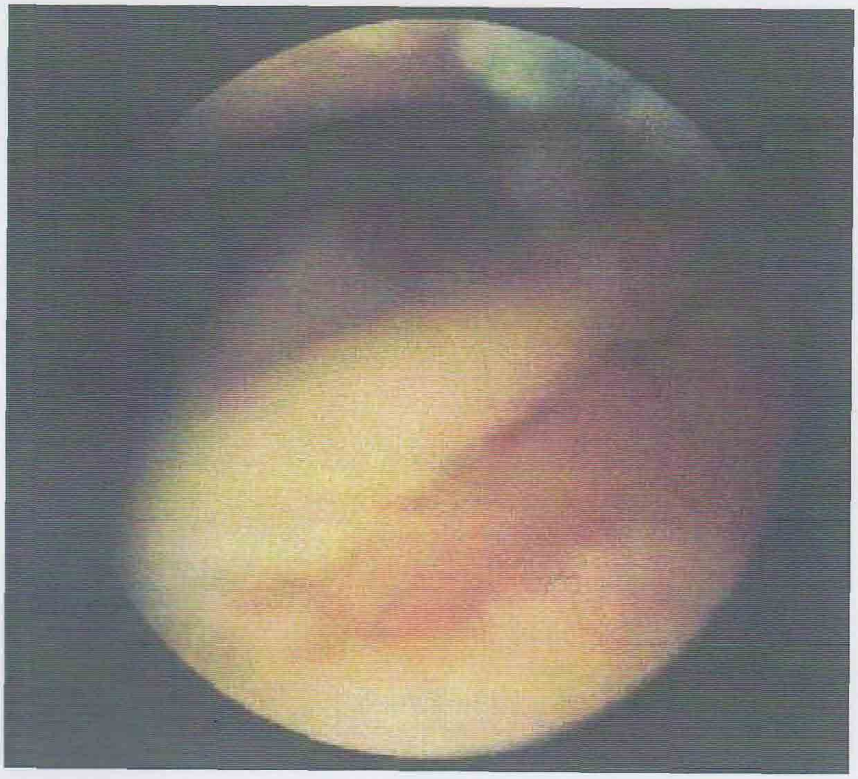


Plate - 21 : Arthroscopic picture of the elbow joint of a dog showing displaced fragment of the coronoid process

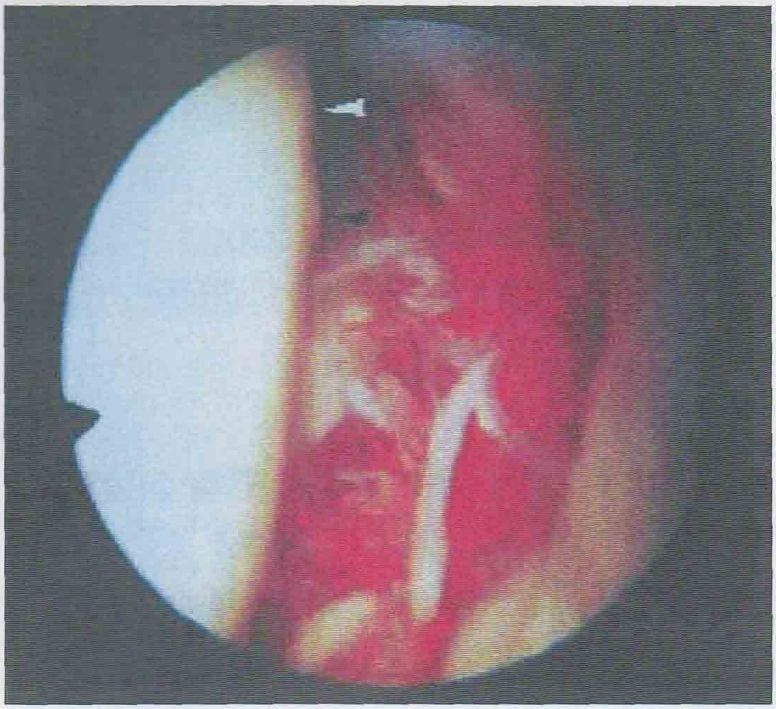


Plate - 22 : Arthroscopic picture of the elbow joint of a dog showing severe synovitis and small osteophytes

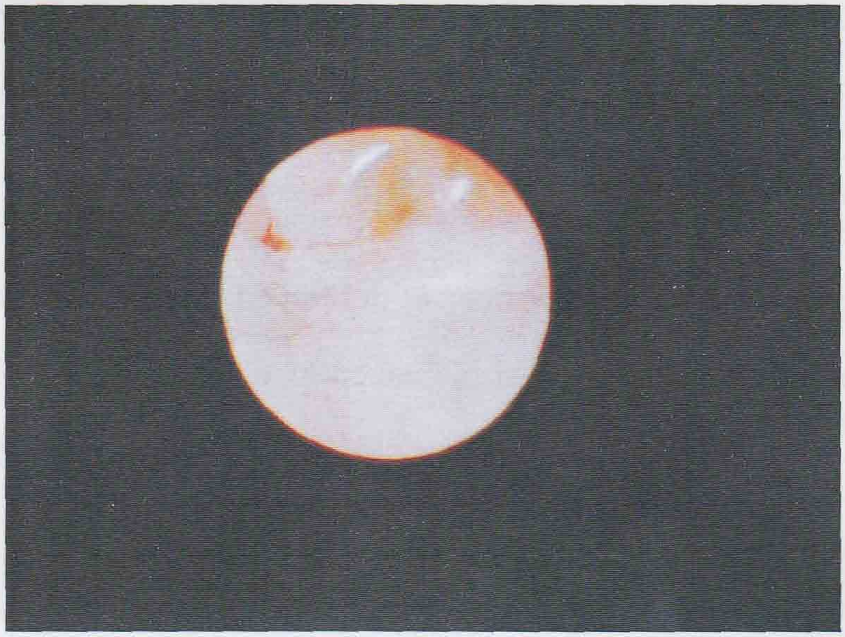


Plate - 23 : Arthroscopic picture of the stifle joint of a dog showing degenerative joint disease (osteoarthritis)

case, cartilage flap was noticed at the medial femoral condyle. The associated lesions were hyperaemic synovium and erosion of the articular synovium (Plate 25).

In two cases, cranial cruciate ligament rupture (Plate 26) was noticed with medial meniscial lesions and the accompanying arthroscopic lesions were haematoma at the site of rupture, mild hyperaemia of synovium, mild hyperplasia of synovial villi and haemorrhagic synovial fluid. A transverse tear of the medial meniscus was noticed in both cases. Fibrillation of meniscus adjoining to the tear was seen. Partial cranial cruciate ligament injury involving cranial medial band was seen with hyperemic and swollen cranial cruciate ligament at the site of injury. Mild fibrillation of medial meniscial was seen.

Severe hyperaemia with hyperplastic synovial villi suggestive of synovitis was noticed in three cases.

4.11 Post arthroscopic complications

Among the 36 joints studied, iatrogenic lesions of the menisci were seen in two cases. Swelling of the joint in the surrounding area was noticed in five cases, which subsided within 24 hours. No infection or neurovascular damage was noticed in any case.

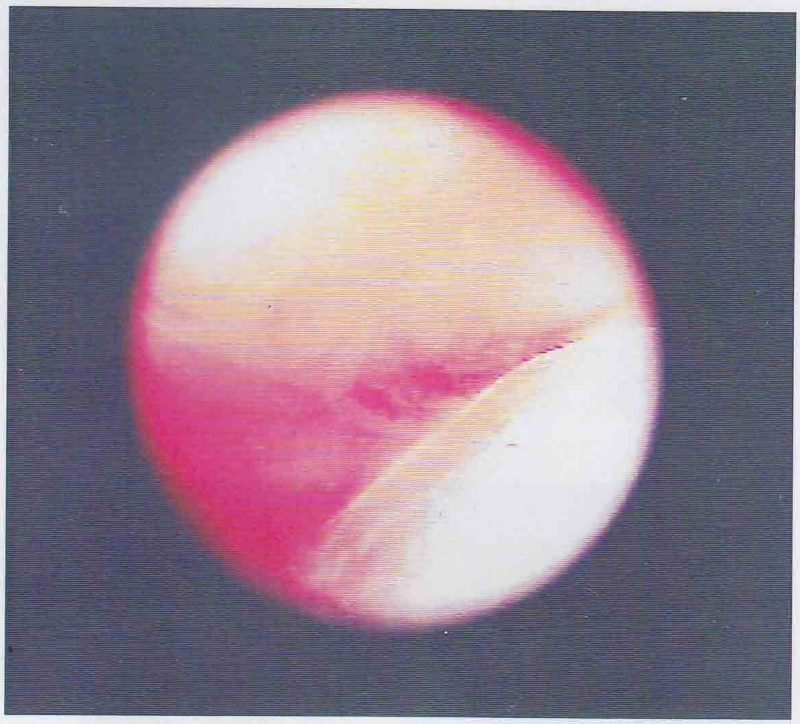


Plate - 24 : Arthroscopic picture of the stifle joint of a dog showing osteochondritis dissecans (chondromalacia)

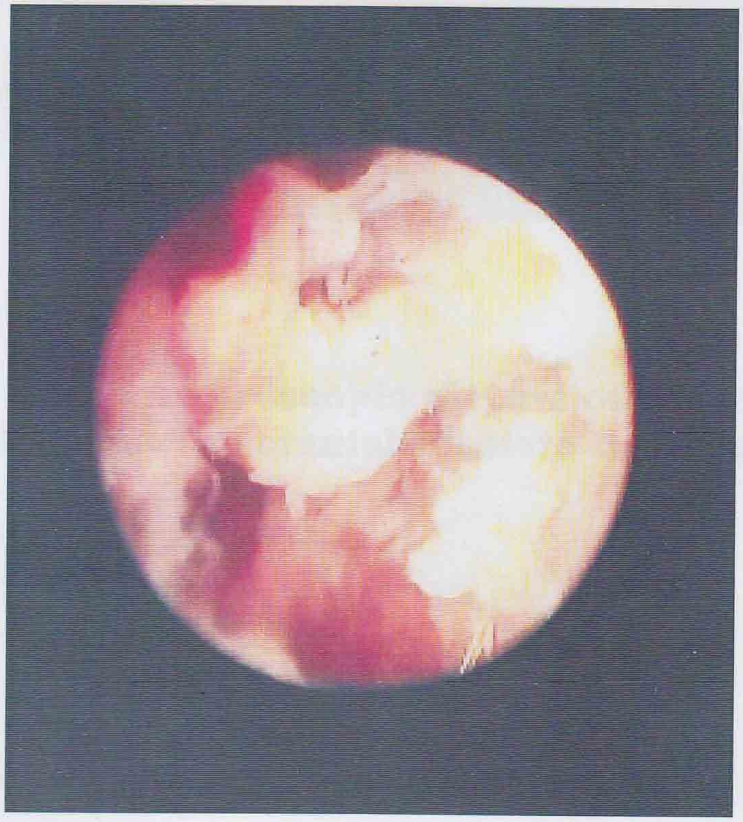


Plate - 25 : Arthroscopic picture of the stifle joint of a dog showing osteochondritis dissecans (detached cartilage flap)

Plate - 26 : Arthroscopic picture of the stifle joint of a dog showing cranial cruciate ligament rupture

Discussion

CHAPTER – V

DISCUSSION

5.1 Incidence

The breed wise incidence of shoulder, elbow and stifle joint affections revealed higher incidence in large size breeds of dogs (German Shepherd Dog, Great Dane, Boxer, Dobermann) followed by medium size breeds of dogs (Rajapalayam) and the least in small breeds of dogs (Spitz). The sex wise distribution revealed higher incidence of lameness in males (24) than females (12).

The age wise incidence revealed higher incidence of shoulder, elbow and stifle lameness in dogs less than two years of age (27) followed by two to six years of age (6) and above six years of age (3). This concurred with the findings of Van Ryssen *et al.* (1993[a]) who had reported that forelimb lameness was more in young dogs of large and giant breed of dogs and more common in males than in females. Higher incidence of forelimb lameness in hyperactive young male dogs of large breeds was observed (Van Ryssen and Van Bree (1997), and Bardet (1998)). Alexander *et al.* (1981) observed stifle lameness to affect the large or giant breeds of dogs between three and nine months of age.

Milton *et al.* (1981) reported higher incidence of osteochondritis dissecans of the shoulder in rapidly growing dogs of large and giant breeds with higher incidence in males.

The higher incidence of shoulder, elbow and stifle joint affections in young males of large breeds of dogs could be attributed to the faster rate of growth when compared to females which leads to developmental defects like osteochondrosis. Van Ryssen *et al.* (1993[a]), also reported that mechanical overload and stress due to abnormal development of the trochlear notch resulting in elbow incongruity contributed to the etiology. It also occurred as a change to secondary premature closure of one of the radial physis and trauma (Van Ryssen and Van Bree, 1997) and high demand, repetitive use of shoulder leading to gleno humeral instability and repetitive micro trauma (Bardet, 1998).

5.2 Pain on extension and flexion

The present study revealed pain on hyperextension in all the animals and it concurred with the findings of Bardet (1998) who had observed pain on hyperextension in 31 animals out of 47 cases and attributed the cause of lameness to the medial glenohumeral ligament distension with thickening or tearing. Mitchell and Innes (2000) also observed mild to severe supra and infra spinatous muscle atrophy, pain on palpation of the bicipital groove and extension as well as internal and external rotation of gleno humeral joint. The physical signs - pain on flexion and extension was noticed in all the cases. The present study revealed pain on flexion and extension in 9 animals and pain and crepitation in 4 animals. Findings of the present study concurred with those of Alexander *et al.* (1981) who observed crepitation, pain on flexion and extension. The authors also reported that gross distension of the elbow joint was not usually a feature for elbow affections and that osteochondritis dissecans of the elbow was the major cause for lameness.

Pain was noticed in all the 12 stifle joints along with thigh muscle wastage in three animals. Fallon (1990) also reported the same clinical signs and attributed osteochondritis dissecans of the stifle and cranial cruciate ligament rupture as the major causes for stifle joint lameness.

5.3 Grading of lameness

The low grade lameness in degenerative joint disease could be due to the chronic degenerative nature of the disease when compared with the other three conditions (Lewis *et al.,* 1987).

The low grade lameness in dogs above six years when compared to the other two age groups could be due to the higher incidence of degenerative joint disease in dogs above six years of age. (Lipowitz and Newton, 1985).

5.4 Haematological changes

The mean haemoglobin level, PCV, ESR, RBC, WBC, neutrophils and lymphocytes were within the normal clinical levels. These findings were also in concurrence with Lenehan and Van Sickle (1985) who found that in degenerative joint disease, haemogram and blood chemistry were usually within normal limits. However, variations could be observed among these parameters pre and post operatively in synovitis which could be attributed to the acute nature of the condition (Coles, 1986).

5.5 Biochemical changes

5.5.1 Serum total protein, albumin , globulin and glucose

The mean total protein, albumin and globulin significantly increased pre-arthroscopically in ligament injury and synovitis which could be attributed to the inflammatory response to the insult.

The mean serum glucose levels pre and post arthroscopically remained within the normal range revealing no correlation with the joint affections. The serum glucose level could be a tool to assess and compare the synovial fluid glucose levels as the latter has been proved to be higher than the serum glucose concentration (Coles, 1986).

5.5.2 Alkaline phosphatase

The mean serum alkaline phosphatase showed significant increase pre arthroscopically only in degenerative joint disease. However, the values remained within the normal range. The increase in serum alkaline phosphatase could be due to destruction of the bone and the formation of osteophytes in degenerative joint disease. (Coles, 1986).

5.6 Synovial fluid

5.6.1 Physical properties

5.6.1.1 Volume

The volume of synovial fluid did not vary significantly between any of the four disease conditions. However the pre arthroscopic volume was significantly higher than post arthroscopic volumes in all the dogs. The average

volume was 0.24 ml ranging from 0.01 ml to 1.00 ml (Lipowitz and Newton, 1985). Increase in the volume was reported by Lipowitz and Newton (1985) and Fernandez *et al.* (1983) during inflammatory conditions and by Mitchell and Innes, (2000) in gleno humeral ligament rupture. The findings of the present study concurred with those of Lewis *et al.* (1987) who also reported increase in volume of synovial fluid in experimentally induced degenerative joint disease.

5.6.1.2 Colour and turbidity

The colour of the normal synovial fluid of dog is clear, colourless to straw coloured fluid (Fernandez *et al.,* 1983 and Coles, 1986). The colour might vary from cloudy to pale yellow sand transparent to pale yellow and opaque in degenerative joint disease (Coles, 1986), dark yellow to xantho-chromic due to chronic haemorrhage and breakdown of erythrocytes (Houlton, 1994 and Fernandez *et al.,* 1983), dark yellow to amber coloured in traumatic arthritis (Coles, 1986).

In the present study the colour of the synovial fluid ranged from colourless to yellow in degenerative joint disease, osteochondritis dissecans and synovitis and colourless to blood tinged in ligament injury. The blood tinged synovial fluid could be attributed to the haemorrhage from associated structures of the joint space (Coles, 1986). Houlton (1994) also observed blood tinged synovial fluid in traumatic joint disease like cranial cruciate ligament rupture. In degenerative joint disease and osteochondritis dissecans the synovial fluid was clear. Turbidity was not present in ligament injury and in synovitis, the turbidity ranged from clear to flocculent. Fernandez *et al.* (1983)

reported that cloudiness or turbidity of synovial fluid was an indication of inflammation. The flocculent nature of the synovial fluid in ligament injury and synovitis could be attributed to the inflammatory joint disease due to the presence of cells, fibrin or other debris in the fluid (Houlton, 1994).

5.6.1.3 Viscosity and mucin clot test

The viscosity of synovial fluid was closer to normal in degenerative joint disease, osteochondrosis dissecans and synovitis. Whereas, in ligament injury the viscosity reduced to 2+. Decreased viscosity was noticed due to dilution of the fluid by bacterial hyaluronidase enzyme (Fernandez *et al.,* 1983) and due to the presence of lysozymal enzyme released by the degenerating neutrophils (Houlton, 1994). A fair viscosity in ligament injury could be attributed to the inflammatory reaction. The mucin clot test was fair to good in degenerative joint disease, osteochondrosis dissecans, ligament injury and synovitis. The mucin clot test was appraised as the quantity and quality of the mucin (Fernandez *et al,*1983). The inflammatory fluids affected the grade of mucin clot test. The findings of the present study were contrary to the findings of Lewis *et al.* (1987) who had observed poor quality mucin clot test in adult dogs with degenerative joint disease .

5.6.2 Cytological examination

The normal synovial fluid contained 0 to 2900 white blood cells/cmm and few red blood cells. Primary cell types seen in the synovial smear were 65% to 90% mononuclear cells. The monocytes and macrophages predominated while lymphocytes and synovial lining cells were less common

(Fernandez *et al.*,1983). In the present study the white blood cells significantly increased during pre-arthroscopic period in all the conditions when compared with that of post-arthroscopic values. The polymorphonuclear cells also showed higher levels during pre-arthroscopic period when compared to post-arthroscoic period . However, the decline in the mean was minimal in ligament injury and synovitis during post-arthroscopic period. The mononuclear cells ranged from 88.86 ±1.39% to 92.56 ± 0.78% pre-arthroscopically. The same trend was noticed during post-arthroscopic period. Coles (1986) stated that in degenerative joint disease, macrophages were increased in synovial fluid. Increase in the polymorphonuclear cells with reduction in mononuclear cells with increase in white blood cell count was observed in inflammatory disease and the same trend was noticed in the present study in the disease conditions like osteochondritis dissecans, ligament injury and synovitis. In degenerative joint disease, the polymorphonuclear cells were less than 10% and this concurred with the findings of Hoelzler *et al.* (2004).

5.7 Biochemical parameters

The mean total protein ranged from 2.96g/dl ± 0.21g/dl to 3.80g/dl ± 0.23g/dl pre arthroscopically. The synovial albumin level also ranged from 1.04g/dl ± 0.10g/dl to 1.40g/dl ± 0.16g/dl pre arthroscopically. The mean globulin level also ranged from 1.89g/dl ±0.18g/dl to 2.27g/dl ± 0.12g/dl pre arthroscopically. The total protein and globulin level were significantly higher in synovitis and osteochondritis dissecans when compared with the other two conditions. The increased total protein and globulin could be due to traumatic and degenerative conditions (Doxy,1983). Tayal *et al.* (2001) also observed changes in the total protein, globulin and albumin in inflammation and the present findings concurred with the previous findings.

The mean glucose level was significantly lesser pre arthrocopically in all the disease conditions. Doxy (1983) reported that the normal synovial fluid glucose concentration was higher than the serum glucose concentration and during inflammation synovial fluid glucose level decreased below those of the serum glucose. (Doxy,1983).

The mean synovial alkaline phosphatase level was higher in osteochondritis dissecans, degenerative joint disease when compared with ligament injury and synovitis pre-arthroscopically. Post-arthroscopically the values decreased significantly in all the four conditions. The decreased enzyme level in the joint could be attributed to the release of the enzyme from the necrotic or inflamed synovial tissue or the production and release of enzymes by altered synovial tissue (Mcllwraith and Fessler, 1978).

5.8 Instrumentation and structures visualized

5.8.1 Shoulder joint

Van Bree *et al.* (1992) and Van Ryssen *et al.* (1993[a]) used a 2.7 mm 25° forward oblique arthroscope for arthroscopic examination of shoulder joints in dogs. Whereas Bardet (1997[a]) used 2.7 mm 30° forward oblique arthroscope with a 3.5mm outer diameter sleeve, the light source was xenon 100 W.

The structures visualised concurred with the findings of Bardet (1997[a]) who had also visualised synovium, articular cartilage surfaces, glenohumeral ligament, labrum, tendon of biceps muscle, and joint capsule. Person (1989) and Bardet (1997[b]) used similar portals - egress port 2cm cranial to acromion in a caudo medial direction and arthroscopic portal 1cm distal to the acromion process as in the present study.

5.8.2 Elbow joint

Bardet (1997[a]) studied the elbow joint through a portal located 1.2 cm cranial and 1cm distal to the lateral humeral condyle.

In the present study the arthroscopic portal was 1cm distal and 0.5 cm caudal to the medial epicondyle of the humerus which provided excellent working space and the findings concurred with Bardet (1997[a]) who had also used the same portal for visualization of the structures of the elbow joint.

Van Ryssen *et al.* (2003) also suggested the use of 2.7 cm long, 30^0 arthroscope for optimal viewing o the elbow joint.

Bardet (1997[a]), Bardet (1997[b]) and McCarthy (1999), suggested that the medial aspect of the joint could be approached through the caudo medial, medial or cranio lateral approach. Bardet (1997[a]) reported that the medial approach was usually employed for the diagnosis and treatment of osteochnondrosis dissecans and fragmentation of medial coronoid process. The lateral aspect of the elbow could be observed arthroscopically by three portals straight lateral, caudo lateral and caudodorso lateral portal (Bardet 1997[a] and Bardet 1997[b]). But these approaches were used for the identification and removal of loose bodies of the acromial process, evaluation of condylar fracture, synovial biopsy and other miscellaneous orthropathies of the elbow joint.

5.8.3 Stifle joint

Van Ryssen *et al.* (2003) reported that a 2.7 mm 300 forward oblique arthroscope was ideal for most conditions of the stifle joint. The authors also suggested cranial, lateral and proximal medial portal for exploration of subtrochlear pouch, lateral and medial pouches, however cruciate ligament and menisci were usually not attempted through this portal as it could be hindered by the hyper plastic synovium and fat pad.

The present approach, first lateral to the patellar ligament and midway between the patella and patellar tuberosity provided accurate visualisation and reproducibility of the medial and lateral recesses, trochlear ridges, articular surface of the patella, trochlear groove, menisci cranial and caudal cruciate ligaments, long digital extensor and a limited portion of tibial condylar surface. These findings concurred with those of Person (1989), Bertrand et al., (1997), Rochat (2001) and Hoelzler *et al.* (2004).

5.9 Correlation of radiographic and arthroscopic lesions

5.9.1 Degenerative joint disease

In two cases degenerative joint disease was diagnosed both radiographically and arthroscopically. The lesions visualised by arthroscopy showed greater details of osteoarthritic changes. The damage to the synovium and cartilage were more clearly visible by arthroscopy.

May (1994) appreciated the presence of osteophytes in cases of degenerative joint disease at characteristic sites varying according to the joint

affected and cartilage erosion could be appreciated in seven cases only. The author also stated that the diagnosis of degenerative joint disease radiographically could be done only after the formation of osteophytes which will usually occur early in the progression of degenerative joint disease. However, Burk and Ackerman (1986) reported that the radiographic findings were less extensive than those of observed at surgery. Findings of the present study concurred with the earlier reports.

Erosive lesions of the cartilage of the humeral condyles, fibrillation and flaking of the articular cartilage, osteophyte formation, synovial proliferation and hyperemia were consistent findings of degenerative joint disease arthroscopically. The arthroscopic findings of the present study concurred with the findings of Lewis *et al.*(1987).

Out of four cases of osteochondrosis dissecans of the shoulder joint, only two cases were appreciated radiographically.

5.9.2 Osteochondritis dissecans

Of the 13 cases of osteochondritis dissecans diagnosed arthroscopically, the lesions were as follows 2-chondromalacia of shoulder, 2- loose cartilage of shoulder, 2-chondromalacia of elbow, 2-loose cartilage of elbow, 1-displaced coronoid process, 1-fragmented coronoid process, 1-cartilage flap on the humeral condyle, 1-cartilage flap of stifle and 1-chondromalacia of stifle were the distribution of the lesion. Only one case of a loose flap of cartilage (second stage cartilage disease) could be diagnosed radiologically.

The arthroscopic lesions of osteochondritis dissecans due to chondromalacia in the shoulder, elbow and stifle joint were characterised by the soft spongy nature of the cartilage and in the early stage synovial proliferation on the articular surface was noticed. These arthroscopic lesions concurred with the findings of Alexander *et al.* (1981) and Boudrieau *et al.* (1982). Loose cartilage was noticed in two of shoulder joints and in two elbow joints. The characteristic arthroscopic lesions were irregular, shallow, spongy and softened cartilage on the articular surface in stage I and loosened detached cartilage flap in stage II with hyperemic synovia with erosion of articular surface. These observations concurred with the findings of Alexander *et al.* (1981) and Milton *et al.* (1981).

In cases with fragmented coronoid process, the lesions were large displaced fragment on the top of the medial coronoid process and in one case fragmented displaced coronoid process in the other. The associated lesions were hyperplasia and hyperemia of the synovium. In fragmented coronoid process, the fractured coronoid process was attached to the annular ligament. The present observations concurred with those of Gortz *et al.*(2004).

5.9.3 Ligament injury

Among the ligament injuries noticed arthroscopically, the lesions were medial glenohumeral ligament rupture-1, medial glenohumeral ligament distension-1, bicipital tendinitis-1, bicipital tendon rupture-1, cranial cruciate ligament rupture – 2 and cranial cruciate ligament injury-1.

Arthroscopic evaluation of medial glenohumeral ligament rupture led rupture of cranial and caudal band of glenohumeral ligament and the ciated lesions were hyperemia and hyperplasia of the synovium and sions of the articular surface.

In glenohumeral ligament distension, the main lesions were distension, nflammation and hyperemia of the ligament. Van Bree and Van Ryssen (1998) described the normal shoulder as having smooth cartilage, glenoid and humeral head. The tendinous structure was covered with synovial membrane and the synovial villi. The glenohumeral ligament was smooth and easily visible. The observations of the present study concurred with those of O'Neill and Innes (2004).

Bicipital tendinitis with distension of the tendon was noticed in one case and intra articular rupture of bicipital tendon was noticed in another case. The associated lesions were hyperplastic synovial villi, haemarthrosis and severe hyperemia and hyperplasia of the synovia (Bardet, 1999).

In cranial cruciate ligament rupture, the arthroscopic lesions were haematoma at the site of rupture, mild hyperaemia of synovium, mild hyperplasia of synovial villi and haemorrhagic synovial. These observations concurred with those of Gretchen (1983) and Lewis *et al.* (1987).

In cranial cruciate ligament rupture, malalignment of the proximal tibia and distal femur with proximal tibia displaced cranially in relation to the distal femur and popliteal sesamoid bone displacement distally were seen. The findings concurred with the observations of Burk and Ackermann (1986).

5.9.4 Synovitis

The characteristic arthroscopic signs of synovitis namely hyperemia, hyperplasia of the synovial membrane and distension of joint space were recorded in two shoulder joints, two elbow joints and three stifle joints.

Radiographically, only increased density of the joint space could be observed. The arthroscopic lesions observed were in accordance with the report of Van Ryssen *et al.* (2003).

5.10 Post arthroscopic complications

The swelling around the joint was due to the irrigating fluid that diffused into the periarticular area and muscles and subcutaneous tissue (Van Ryssen *et al.*, 1993[b] and Martini *et al.*, 2002) and the lesions subsided within 24 hours and the findings concurred with those of Van Ryssen *et al.* (1993[a]), Bertrand *et al.* (1997) and Van Ryssen and Van Bree (1997). Iatrogenic lesions of the menisci arising from arthroscopy was reported by Bertrand *et al.*(1997).

Incidence of seroma, infection and neurovascular damage was not noticed and Van Ryssen *et al.* (2003) reported that these complications were very rare.

Summary

CHAPTER – VI

SUMMARY

Shoulder, elbow and stifle joint affections in dogs were athroscopically assessed and its efficacy and advantages as diagnostic tool was studied.

Thirty six dogs suffering from shoulder, elbow and stifle joint lameness were divided into three groups of 12 animals each and subjected to arthroscopic examination after physical, clinical and radiographical examination.

Physical examination revealed pain on hyperextension in all cases shoulder joint lameness. Thigh muscle wastage was predominant in three dogs with stifle joint lameness.

Clinical examination revealed higher lameness grade in osteochondritis dissecans, ligament injury and synovitis.

Young dogs below two years of age and dogs two-six years of age group showed higher grade of lameness in all the three joint affections. Incidence of all the three joints affections were higher in large breed dogs. Males were mostly affected.

The haematological study revealed no significant changes between all four disease conditions studied. Pre and post arthroscopic values of haemoglobin, packed cell volume, total erythrocyte count, total leukocyte count, neutrophils and lymphocytes also showed no significant changes.

Significant decrease in erythrocyte sedimentation rate in osteochondritis dissecans was seen in both pre and post arthroscopic period. Significant decrease in erythrocyte sedimentation rate was noticed in degenerative joint disease, ligament injury and synovitis during post arthroscopic period only.

Biochemical study of the serum revealedsignificantly higher values of serum albumin in ligament injury and synovitis. The serum globulin level was higher in ligament injury and synovitis which decreased post arthroscopically. Serum alkaline phosphatase was significantly higher in degenerative joint disease, which decreased to normal values during post arthroscopic period. No significant change in mean blood glucose level was found in any conditions and between pre and post arthroscopically.

No significant change in mean volume of synovial fluid was noticed in any conditions prearthroscopically. However, significant reduction in volume was seen in degenerative joint disease, osteochondritis dissecans and synovitis post arthroscopically.

Cytological examination of synovial fluid revealed significant decrease in mean white blood cell count in ligament injury, synovitis, osteochondritis dissecans and degenerative joint disease. Significant reduction in polymorphonuclear cells was seen in osteochondritis dissecans, degenerative joint disease and ligament injury during the post arthroscopic period. Whereas, significant increase in mean mononuclear cells was noticed in degenerative joint disease, osteochondritis dissecans and ligament injury during the post arthroscopic period.

Total protein, albumin, globulin levels in synovial fluid were significantly reduced in ligament injury. Significant increase in alkaline phosphatase in synovial fluid was noticed in degenerative joint disease and osteochondritis dissecans pre-arthroscopically which decreased post arthroscopically to normal levels.

All the cases of degenerative joint disease, 2 cases of osteochondritis dissecans and 2 cases of cranial cruciate ligament rupture were diagnosed radiographically.

Arthroscopy of the shoulder joints revealed cartilage lesions of osteochondritis dissecans like cartilage flaps, fissure, chondromalacia and lesions of degenerative joint disease, biceps tenditinitis, partial or complete tear of the biceps brachii, bicipital tenosynovitis, and glenohumeral ligament rupture.

Arthroscopy of elbow joints demonstrated fragmented coronoid process, osteochrondritis dissecans of humeral condyle, ununited anconeal process, articular anomally and joint incongruity.

Arthroscopy of stifle joints demonstrated osteochondritis dissecans of the femoral condyle, meniscal injuries, cruciate ligament injuries and degenerative lesions.

Bibliography

BIBLIOGRAPHY

Alexander, J.M., D.C. Richerdson and B.A. Selcer, 1981. Osteochondritis dissecans of the elbow, stifle and hock – A review. *J. Am. Anim. Hosp. Assoc.*, **17**: 51-55.

Anderson, J., 1994. The Stifle. In: *Manual of Small Animal Arthrology*. (Eds.) J. Houlton and R. Collinson, British Small Animal Veterinary Association, Publications., **pp.** 267-280.

Arnoczky, S.P and J.L. Marshall, 1977. The cruciate ligaments of the canine stifle: An anatomical and functional analysis. *Am. J. Vet. Res.*, **38**: 1807 - 1814.

Bardet, J.F., 1997[a]. Arthroscopy of the elbow in dogs. Part I: The normal arthroscopic anatomy using the craniolateral portal. *Vet. Comp. Orthop .traumatol.*, **10**:1-5.

Bardet, J.F., 1997[b]. Arthroscopy of the elbow in dogs. Part II: The cranial portals in the diagnosis and treatment of the lesions of the coronoid process. *Vet. Comp. Orthop .traumatol.*, **10**:60-66.

Bardet, J.F., 1998. Diagnosis of shoulder instability in dogs and cats: A retrospective study. *J. Am. Anim. Hosp. Assoc.*, **34**: 42-54.

Bardet, J.F., 1999. Lesions of the biceps tendon diagnosis and classification *Vet.Comp.Orthop.traumatol.*, **12**: 188-195.

Benjamin, M.M., 1985. *Outline of Veterinary Clinical Pathology*. 3rd edn, Kalyani Publishers, New Delhi., **pp.** 233-354.

Bertrand, S.G., D.D. Lewis, J.B. Madison, J.H. Haan, W.P. Stubbs and J.T. Stallings, 1997. Arthroscopic examination and treatment of osteochondritis dissecans of the femoral condyle of six dogs. *J. Am. Anim. Hosp. Assoc.*, **33**: 451-455.

Boudrieau, R.J., R.B. Hohn and J.F. Bardet, 1982. Osteochondritis dissecans of the elbow in the dog. *J. Am. Anim. Hosp. Assoc.*, **19**: 627-635.

Burk, R.L and N. Ackerman, 1986. Appendicular Skeleton. In: *Small Animal Radiology,* Churchill Livingstone, New York., **pp** 235-309.

Butterworth, S., 1994. The Shoulder. In: *Manual of Small Animal Arthrology.* (Eds). J. Houlton and R. Collinson. British Small Animal Veterinary Association, England., **pp.**149-174.

Chauhan, R.S., 1995. *Text book of Veterinary Clinical and Laboratory Diagnosis*. Jaypee Brothers Medical Publishers (P) Ltd. New Delhi., **pp.** 167-174.

Coles, E.H., 1986. *Veterinary Clinical Pathology*. 4th edn., W.B.Saunders Company, Philadelphia., **pp.**256-260.

Dejardin, L.M., 2002. Cruciate disease: Pathophysiology and expectations. 2002 American College of Veterinary Surgeons Veterinary Symposium Equine and Small Animal Proceedings. Small Animal Seminar Arthrology Track.

Doumas, B.T., W.A. Watson and H.G. Biggs, 1971. In: *Practical Clinical Biochemistry*. 5th edn, (Eds), Arky, H., A.H. Crowenlock and M.Bell William Heinemann Medical Books Ltd., London., **pp.** 115-135

Doxy, D.O., 1983. *Clinical Pathology and Diagnostic Procedures*. 2nd edn., Balliere Tindall, London., **pp.** 225-227.

Fallon, R.K., 1990. Osteochondritis dissecans of the stifle. In: *Current Techniques in Small Animal Surgery*. 3rd edn. (Ed.) Bojrab, M.J., Lea and Febiger., **pp.** 721 –722.

Fernandez, F.R., C.B. Grindem, A.J. Lipowitz and V. Perman, 1983. Synovial fluid analysis: Preparation of smears for cytologic examination of canine synovial fluid. *J. Am. Anim. Hosp. Assoc.,* **19** : 727-734.

Gortz K., B. Van Ryssen, O. Taeymans, M. Van Heerden, I. Gielen and M. Risselada, 2004. Traumatic fracture of the medial coronoid process in a dog. *Vet. Comp. Orthop. Traumatol.*, **17**:159-162.

Gretchen, F., 1983. Classification of meniscal injuries in the canine stifle based upon gross pathological appearance. *J. Am. Anim. Hosp. Assoc.,* **19**:325-334.

Hall, M.E. and R.J. Keeran, 1975. Use of the arthroscope in the horse. *Vet. Med. Small Anim. Clin.*, **70**: 705.

Hayashi, K., J.D. Frank, C. Dubinsky, Z. Hao, M.D. Markel, P.A. Manley and P. Muir 2003. Histologic changes in ruptured canine cruciate ligament. *Vet. Surg.*, **32** : 269 -277.

Hoelzler, M.G., D.L. Millis, D.A. Francis and J.P. Weigel, 2004. Results of arthroscopic versus open arthrotomy for surgical management of cranial cruciate ligament deficiency in dogs. *Vet. Surg.,* **33**: 146-153.

Houlton, J. 1994. Ancillary aids to the diagnosis of joint disease. In: *Manual of Small Animal Arthrology*. (Eds.), Houlton, J and Collinson, R. British Small Animal Veterinary Association, England., **pp.** 22-38.

Kivumbi, C.W and D. Bennett, 1981. Arthroscopy of the canine stifle joint. *Vet. Rec.,* **109**: 241-249.

Kumar, M and K.B.P. Agarwal, 2001. Biochemical alterations of synovial fluid in induced traumatic arthritis in buffalo calves *(Bubalus bubalus). Indian. J. Vet .Surg.*, **22**: 87-91.

Lavelle, R., 1994. Osteochondrosis. In: *Manual of Small Animal Arthrology* (Eds.), Houlton, J and R. Collinson, British Small Animal Veterinary Association, Kingslay House, Church Lane, Shurdington, Cheltenham., **pp.**75-85.

Lehmann, M and K. Lehmann, 2004. Modification of the triangulation technique for arthroscopy of the canine shoulder joint using a new target device. *Vet.Comp.Orthop.Traumatol .*, **17**: 1- 8.

Lenehan, T.M. and D.C. Van Sickle, 1985. Canine osteochondrosis. In: *Text Book of Small Animal Orthopaedics*. (Ed.), Newton, C.D. and D.M. Nunamaker. J.B. Lippincott Company, Philadelphia., **pp.** 981- 998.

Lewis, D.D., R.L. Goring, R.B. Parker and P.A. Curasi, 1987. A comparison of diagnostic methods used in the evaluation of early degenerative joint disease in the dog. *J. Am. Anim. Hosp. Assoc.*, **23**: 305-315.

Lipowitz, A.J. and C.D. Newton, 1985. Degenerative joint disease and traumatic arthritis. In: *Text book of Small Animal Orthopaedics* (Eds.), Newton C.D. and D.M. Nunamaker. J.B. Lippincott company, Philadelphia., **pp.**1029-1046.

Long, C.D and T.G. Nyland, 1999. Ultrasonographic evaluation of the canine shoulder. *Vet. Radiol. and Ultrasound.*, **40**: 372-379.

Lopez, M.J., M.D. Markel, V. Kalscheur, Y. Lu and P.A. Manley, 2003. Hamstring graft techniques for stabilization of canine cranial cruciate ligament deficient stifles. *Vet. Surg.*, **32**: 399- 401.

Manley, P.A, P.E. Howard and G.M. Constantinesu, 1990. Surgery for osteochondrosis of the canine shoulder. In: *Current Techniques in Small Animal Surgery*. (Ed.), Bojrab, M.J 3rd edn. Lea and Febiger., **pp.** 749-756.

Martini, F.M., S. Pinna and M. Del Bue, 2002. A simplified technique for diagnostic and surgical arthroscopy of the shoulder joint in the dog. *J. Small Anim. Pract.*, **43**: 7-11.

May, S., 1994. Degenerative joint disease. In: *Manual of Small Animal Arthrology*. (Ed.) Houlton, J.E.F. British Small Animal Veterinary Association, Kingslay House, Church Lane, Shurdington, Cheltenham., **pp.** 62-74.

McCarthy, T.C, 1999. Arthroscopy. In: Veterinary Endoscopy. Freeman, L.J. 2nd edn, Mosby, St. Louis., **pp.** 237-250.

McIlwraith, C.W. and J.F. Fessler, 1978. Arthrosopy in the diagnosis of equine joint disease. *J. Am. Vet. Med. Assoc.*, **172**: 263-268.

Miller, C.W. and K.R Presnell, 1985. Examination of the canine stifle: arthroscopy versus arthrotomy. *J. Am. Anim. Hosp. Assoc.*, **21**: 623.

Milton, J.C., P.F. Rumph and A.D. Reed, 1981. Osteochondritis dissecans of the shoulder in the racing Grey hound: A report of two cases and a survey of 109 Greyhound anatomy specimens. *J. Am. Anim. Hosp. Assoc.*, **17** : 617-622.

Mitchell, R.A.S and J.F. Innes, 2000. Lateral glenohumeral ligament rupture in three dogs. *J. Small Anim. Pract.*, **41**: 511-514.

Neaas, A., R. Srnec and H. Kecova, 2002. Diagnostic realiability of stifle arthroscopy of pathological changes in cruciate deficient knee. *Acta Vet. Brno.,* **71**: 249-254.

O' Neill, T. and J.F. Innes, 2004. Treatment of shoulder instability caused by medial glenohumeral ligament rupture with thermal capsulorraphy. *J. Small. Anim. Pract.,* **45**: 521-524.

Person, M.W., 1985. A procedure for arthroscopic examination of the canine stifle joint. *J. Am. Anim. Hosp. Assoc.,* **21**: 179-186.

Person, M.W., 1989. Arthroscopic treatment of oteochondritis dissecans in the canine shoulder. *Vet. Surg.,* **18:** 175- 189.

Presnell, K.R., 1990. Surgery for Fragmented Medial Coronid Process. In: *Current Techniques of Small Animal Surgery.* (Ed.), Bojrab M.J., 3rd edn. Lea and Febiger., **pp.**778-782.

Putz, R and M. Muller-Gerbl., 1988. (Functional anatomy of the elbow joint) *Orthopade.,* **17**: 338-346.

Reagan, B.F, V.K. McIneray, B.V. Tread Well, B. Zarius and H.J. Mankin, 1983. Irrigating solutions for arthroscopy. A metabolic study. *J. Bone and Joint Surg.,* **65:** 629-630.

Rochat, M.C., 2001. Arthroscopy. *Vet. Clin. North Am: Small Anim. Pract.,* **31:** 761-787.

Scharf, G., S. Steinbuchl, M. Messmer and S.Oglerca, 2004. Glenoid dysplasia and bicipital tenosynovitis in Maine coon cat. *J. Small Anim. Pract.,* **45:** 515-520.

Siemering, G.H., 1978. Arthroscopy of dogs. *J. Am. Vet. Med. Assoc.*, **172:**575-577.

Snedecor, G.W. and W.G. Cochran, 1994. *Statistical Methods*. 8th edn. Oxford and IBH Publishing Company, Kolkata., **pp.** 60- 64.

Tayal, R., I.S. Chandra, B.A. Moulvi and J. Singh, 2001. Efficacy of phenylbutazone with analgin in the treatment of acute noninfectious arthritis in cow calves. *Indian J. Vet. Surg.* **22**: 119.

Thomsen, M., M. Loew and H. Nagere, 2001. Kinematics and biomechanics of the elbow joint, *Orthopade.*, **30**: 582-586.

Tietz, N.W., D. Rinker and L.M. Shaw, 1983. IFCC Method for alkaline phosphatase, *J. Clin. Chem. Clin. Bioche.*, **21** : 731-748.

Trumble, T.N, C.W. McIlwraith and R.C. Billinghurst, 2001. Techniques for arthroscopically guided transection of the cranial cruciate ligament as a model for osteoarthritis. *Vet. Surg.*, **30** : 508.

Van Bree, H., B. Van Ryssen and M. Desmidt, 1992. Osteochondrosis lesions of the canine shoulder: Correlation of positive contrast arthrography and arthroscopy. *Vet. Radiol. and Ultrasound.*, **33**: 342-347.

Van Bree, H, H. Degryse, B. Van Ryssen, F. Ramon and M. Desmidt, 1993. Pathologic correlations with magnetic reasonance images of the osteochondrosis lesions in canine shoulders. *J. Am. Vet. Med. Assoc.*, **202**: 1099-1105.

Van Bree, H.J., and B.Van Ryssen, 1998. Diagnostic and surgical arthroscopy in oesteochondrosis lesions. *Vet. Clin. North. Am. Small Anim. Pract.*, **28**: 161-189.

Van Gestel, M.A., 1985. Diagnostic accuracy of stifle arthroscopy in the dog. *J. Am. Anim. Hosp. Assoc.*, **21**: 759-763.

Van Ryssen, B. and H. Van Bree, 1997. Arthroscopic findings in 100 dogs with elbow lameness. *Vet. Rec.*, **140:** 360-362.

Van Ryssen, B., H. Van Bree and S. Missinne, 1993[a]. Successful arthroscopic treatment of shoulder osteochondrosis in the dog. *J. Small Anim. Pract.*, **34**: 521-528.

Van Ryssen, B, H. Van Bree and P. Simoens 1993[b]. Elbow arthroscopy in clinically normal dogs. *Am. J. Vet. Res.*, **54**: 191-198.

Van Ryssen, B., H. Van Bree and P. Vyt. 1993[c]. Arthroscopy of the canine hock joint. *J. Am. Anim. Hosp. Assoc.* **29**: 107-113.

Van Ryssen, B., H. Van Bree., W.O.Whitney and K.S. Schulz, 2003. Arthroscopy. In: *Text book of Small Animal Surgery.* 3rd edn, W.B. Saunders Company, Philadelphia, **pp.** 2285-2306.

Vasseur, P.B., D. Moore and S.A. Brown, 1982. Stability of the canine shoulder joint an *in vitro* analysis. *Am. J. Vet Res.*, **43**: 352-355.

Watanabe M., S. Takeda and H. Ikeuchi, 1969. *Atlas of Arthroscopy.* 2nd edn. Tokyo, Igaku Shrin Ltd., **pp.** 38-40.

Wind, A.P., 1986. Elbow incongruity and developmental elbow diseases in the dog. Part I. *J. Am. Anim. Hosp. Assoc.* **22**: 712-723.

Wind, A.P and M.E. Packard, 1986. Elbow incongruity and developmental elbow diseases. *J. Am. Anim. Hosp. Assoc.*, **22**: 725-730.

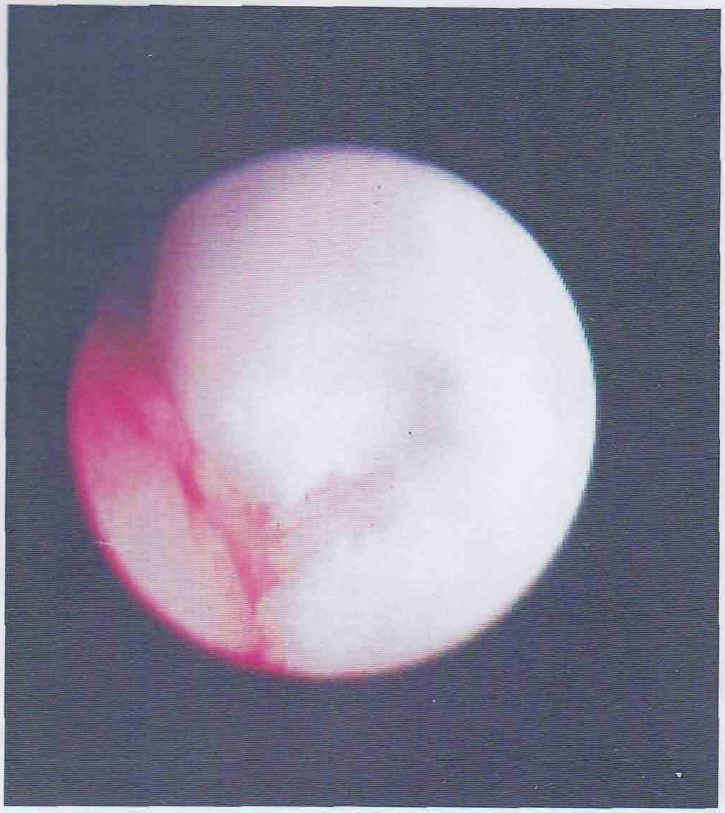


Plate - 16 : Arthroscopic picture of the shoulder joint of a dog showing bicipital tendinitis

Plate - 17 : Arthroscopic picture of the shoulder joint of a dog showing bicipital tendon rupture