

**MULTIPLE LIGAMENTOUS INJURIES OF THE
CANINE STIFLE JOINT: A LITERATURE REVIEW
AND CLINICAL CASE STUDY**

by

Warrick John Bruce
BVSc (Massey) Cert SAO MRCVS

A thesis presented to
the Faculty of Veterinary Clinical Studies
University of Glasgow
for the Degree of Master of Veterinary Medicine

September 1995

©

Warrick John Bruce

ProQuest Number: 13834040

All rights reserved

INFORMATION TO ALL USERS

The quality of this reproduction is dependent upon the quality of the copy submitted.

In the unlikely event that the author did not send a complete manuscript and there are missing pages, these will be noted. Also, if material had to be removed, a note will indicate the deletion.



ProQuest 13834040

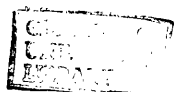
Published by ProQuest LLC (2019). Copyright of the Dissertation is held by the Author.

All rights reserved.

This work is protected against unauthorized copying under Title 17, United States Code
Microform Edition © ProQuest LLC.

ProQuest LLC.
789 East Eisenhower Parkway
P.O. Box 1346
Ann Arbor, MI 48106 – 1346

Thesis
10211
Copy 1



SUMMARY

The aim of this thesis is to detail the clinical, ancillary, surgical and postoperative findings in eleven cases of multiple ligamentous injuries of the canine stifle joint. All cases were presented to the University of Glasgow Veterinary School during the period October 1992 to August 1995.

Severe trauma was required to produced multiple ligamentous injury of the stifle joint in nine of the dogs in this series. This resulted from an injury occurring when running at speed, catching the limb in a fence or gate, or from a road traffic accident. In the remaining two dogs, injury occurred without obvious trauma and in these cases there was evidence of chronic stifle joint infection. Multiple ligamentous stifle joint injuries occurred most frequently in adult, male, working or sporting dogs and resulted in a non-weight bearing lameness and stifle joint subluxation. Complete dislocation of the joint occurred in two cases. The cranial cruciate ligament was always injured and ten of the cases involved the caudal cruciate ligament, as well as one, or both, of the collateral ligaments. The most frequent combination was injury to the cranial and caudal cruciate ligaments and the lateral collateral ligament and this injury occurred most often after catching the limb in a fence or gate. Injury to the menisci was a common finding.

The surgical method employed produced good to excellent results for long-term limb function in ten cases. There was no significant decrease in thigh muscle mass or reduction in stifle joint range of motion in nine of the cases at long-term follow-up. Radiographic changes such as osteophytic development, joint effusion and soft tissue swelling occurred in all cases postoperatively.

These findings are discussed in light of a broad review of the literature pertaining to multiple ligamentous injuries of the stifle joint in dogs and the relevant comparative aspects in man.

DECLARATION

I, Warrick John Bruce, do hereby declare that the work carried out in this thesis is original, was carried out by myself or with due acknowledgement, and has not been presented for the award of a degree at any other University.

signed:

date:

In loving memory of my grandmother
Grace Isobel Bruce
who always supported me in my quest to enter veterinary science.

**“The awareness of the ambiguity of one's highest
achievements (as well as one's deepest failures) is
a definite symptom of maturity.”**

Paul Tillich, Time, 1963.

TABLE OF CONTENTS

SUMMARY	I
DECLARATION.....	II
DEDICATION.....	III
TABLE OF CONTENTS.....	IV
TABLE OF FIGURES.....	IX
TABLE OF TABLES.....	XII
ACKNOWLEDGEMENTS.....	XIII
INTRODUCTION.....	XIV
SECTION 1: BASIC SCIENCE.....	1
PART 1: THE STABILISING STRUCTURES OF THE CANINE	
STIFLE.....	2
INTRODUCTION.....	2
PRIMARY JOINT RESTRAINTS	3
The Cranial and Caudal Cruciate Ligaments	3
Anatomy	3
Cruciate Ligament Dynamics.....	4
The Medial and Lateral Collateral Ligaments.....	9
Anatomy	9
Collateral Ligament Dynamics.....	9
SECONDARY JOINT RESTRAINTS	13
The Lateral and Medial Menisci	13
Anatomy	13
Meniscal Dynamics	15
The Patella and Patellar Ligament.....	15
Anatomy	15
Patella and Patellar Ligament Dynamics.....	16
The Joint Capsule	16
Anatomy	16
OTHER SUPPORTING STRUCTURES.....	18
Muscles and Tendons of the Stifle Joint	18
PART 2: MOVEMENT AND STABILISING MECHANISMS OF	
THE STIFLE JOINT.....	20
STIFLE KINESIOLOGY	20
THE CRUCIATE LIGAMENTS	23
THE COLLATERAL LIGAMENTS	24
THE MENISCI	24
THE PATELLA AND PATELLAR LIGAMENT.....	26
THE JOINT CAPSULE	26
MUSCLES AND TENDONS	26

PART 3: PATHOPHYSIOLOGICAL CONSIDERATIONS.....	27
LIGAMENT PROPERTIES	27
CLASSIFICATION OF LIGAMENT INJURY	27
Sprain.....	27
First-degree Sprain	27
Physical and Radiographic Findings	27
Second-degree Sprain.....	27
Physical and Radiographic Findings	28
Third-degree Sprain.....	28
Physical and Radiographic Findings	28
MECHANISMS OF LIGAMENT FAILURE.....	28
Mechanical Properties	28
Ligament Failure Modes	30
INJURY TO THE MENISCI.....	31
Surface Fibrillation.....	32
Peripheral Avulsion.....	32
Compression Injury	32
Axial Fringe Tear	32
Transverse (Radial) Tear	32
Full Thickness Longitudinal Tear	32
Atrophied, Proliferative, and Discoid Menisci	32
PART 4: LIGAMENT HEALING AND TREATMENT OF LIGAMENT INJURIES	33
THE REPARATIVE PROCESS	33
The Substrate Phase.....	33
The Repair Phase.....	33
The Maturation Phase.....	34
FACTORS INFLUENCING LIGAMENT HEALING	34
Anatomy of the Ligament.....	34
Severity of Injury.....	34
Surgical Repair	35
Apposition of the Ligament Ends.....	35
Tension on the Wound	35
Timing of Surgical Repair	35
Exercise and Immobilisation	35
TREATMENT OF LIGAMENT INJURIES	37
NON-SURGICAL TREATMENT	37

SURGICAL TREATMENT	38
Interstitial Failure	38
Primary Repair	38
Repair Augmentation	40
Ligament-Bone Failure.....	40
Ligament-Bone Avulsion	41
Ligament Replacement.....	41
Autogenous Tissues.....	41
Synthetic Materials.....	42
PART 5: MENISCAL HEALING AND TREATMENT OF MENISCAL INJURIES.....	43
MENISCAL HEALING AND BLOOD SUPPLY	43
HEALING ENHANCEMENT TECHNIQUES.....	43
TREATMENT OF MENISCAL INJURIES.....	44
Primary Repair	44
Total Meniscectomy and Partial Meniscectomy	44
SECTION 2: MULTIPLE LIGAMENTOUS INJURIES OF THE CANINE STIFLE JOINT.....	46
PART 1: MULTIPLE LIGAMENTOUS INJURIES OF THE CANINE STIFLE JOINT	47
INCIDENCE AND PREVALENCE.....	47
SIGNALMENT	47
AETIOLOGY	48
CLASSIFICATION.....	49
Subluxation/Dislocation	49
Combinations of Ligament Injuries.....	49
OTHER ASSOCIATED INJURIES	50
PART 2: THE CLINICAL AND DIAGNOSTIC FEATURES OF MULTIPLE LIGAMENTOUS INJURIES OF THE CANINE STIFLE JOINT.....	51
GENERAL CLINICAL FINDINGS	51
ASSESSMENT OF STIFLE JOINT STABILITY.....	51
Craniocaudal Stability	52
Angular Stability	53
Rotational Stability.....	53
ANCILLARY DIAGNOSTIC TECHNIQUES	55
Radiography	55
Surgery	55

PART 3: MANAGEMENT OF MULTIPLE LIGAMENTOUS INJURIES OF THE CANINE STIFLE JOINT	56
NON-SURGICAL TREATMENT	56
GENERAL PRINCIPLES FOR SURGICAL TREATMENT	56
SURGICAL TECHNIQUES	58
Primary Repair and Ligament Reconstruction	58
Extra-articular Suture Stabilisation	58
Trans-articular Pinning	59
Stifle Arthrodesis	59
Amputation	59
POSTOPERATIVE MANAGEMENT	60
SECTION 3: CLINICAL CASES	61
PART 1: MATERIALS AND METHODS	62
CASE SELECTION	62
SURGICAL TECHNIQUE	62
POSTOPERATIVE MANAGEMENT	66
IMMOBILISATION AND EXERCISE	66
POSTOPERATIVE ASSESSMENT	67
PART 2: CASE SUMMARIES	72
CASE 1: No. 124018	72
CASE 2: No. 122315	74
CASE 3: No. 126109	75
CASE 4: No. 125193	77
CASE 5: No. 126408	78
CASE 6: No. 123011	80
CASE 7: No. 122697	81
CASE 8: No. 123611	82
CASE 9: No. 115198	84
CASE 10: No. 124690	85
CASE 11: No. 123050	87
PART 3: RESULTS	90
PREOPERATIVE AND OPERATIVE DATA	90
Incidence and Prevalence	90
Patient Data	90
Clinical Findings	90
Radiographic Findings	91
Surgical Findings	91
Combinations of Ligament Injuries	91
Modes of Ligament Failure	91
Meniscal Injuries	91
Joint Capsule Rupture	91

POSTOPERATIVE DATA	92
EARLY POSTOPERATIVE EXAMINATION.....	92
LONG-TERM POSTOPERATIVE EXAMINATION.....	95
PART 4: DISCUSSION	102
PART 5: CONCLUSION	109
APPENDIX 1	110
SEDATION	110
GENERAL ANAESTHESIA	110
Premedication.....	110
Induction.....	110
Maintenance	110
Fluids	110
APPENDIX 2	111
ANALGESIA.....	111
ANTIBIOTIC THERAPY	111
REFERENCES.....	112

TABLE OF FIGURES

Figure 1	Origin and insertion of the right cranial cruciate ligament.....	5
Figure 2	Origin and insertion of the caudal cruciate ligament.....	5
Figure 3	A frontal view of a dissected specimen of the right stifle joint.....	6
Figure 4	Side view of the right stifle to show the shape, orientation and tension of the cranial cruciate ligament in extension.....	7
Figure 5	Side view of the right stifle to show change in shape, orientation and tension of the cranial cruciate ligament in flexion.....	7
Figure 6	Side view of the right stifle to show the shape, orientation and tension of the caudal cruciate ligament in extension.....	8
Figure 7	Side view of the right stifle to show the shape, orientation and tension of the caudal cruciate ligament in flexion.....	8
Figure 8	Origin and insertion of the medial collateral ligament.....	10
Figure 9	Origin and insertion of the lateral collateral ligament.....	10
Figure 10	Lateral view of stifle to show the shape, tension and orientation of the lateral collateral ligament in extension.....	11
Figure 11	Lateral view of stifle to show the change in shape, tension and orientation of the lateral collateral ligament in flexion.....	11
Figure 12	Medial view of stifle to show the shape, tension and orientation of the medial collateral ligament in extension.....	12
Figure 13	Medial view of stifle to show the change in shape, tension and orientation of the medial collateral ligament in flexion.....	12
Figure 14	View of the proximal articular surface of the tibia showing the menisci and their attachments.....	14
Figures 15a-d	Cranial, lateral, caudal and medial views of the right stifle illustrating the joint capsule attachments and associated structures.....	17
Figure 16	Schematic drawing of the stifle joint of the dog illustrating the three axes of motion (x,y, and z) and their orientation.....	21
Figure 17	Diagram illustrating the determination of the instant centre of motion.....	22
Figure 18	Diagram illustrating the compressive and translational force vectors acting about the cranial cruciate ligament.....	25
Figure 19	An example of the typical shape of a load-deformation curve for a tension test to failure of a bone/ligament/bone preparation.....	29

Figures 20a-k	Diagrams illustrating the types of meniscal injury.	31
Figure 21	Diagram illustrating the locking-loop suture pattern.	39
Figure 22	Diagram illustrating the three-loop pulley suture pattern.	39
Figure 23	Fascial graft used in cruciate ligament reconstruction.	63
Figure 24	Primary repair of the medial collateral ligament.	63
Figure 25	Augmented medial collateral ligament repair.	63
Figure 26	Fascial graft sutured to the lateral femoral condyle.	63
Figure 27	Medial view to show position of screws and figure-8 suture used in medial collateral ligament repair.	64
Figure 28	Lateral view to show position of screw and figure-8 suture used in lateral collateral ligament repair.	64
Figure 29	Measurement of mid-thigh muscle mass circumference.	68
Figure 30	Measurement of range of motion, zero point.	68
Figure 31	Measurement of range of motion, maximum flexion.	68
Figure 32	Measurement of range of motion, maximum extension.	68
Figure 33	Lateral view of the stifle joint illustrating the scoring system used to identify pathological changes.	70
Figure 34	Craniocaudal view of the stifle joint illustrating the scoring system used to identify pathological changes.	71
Figures 35a&b	Case 1. Preoperative lateral and craniocaudal radiographs of the left stifle joint.	73
Figures 36a&b	Case 1. Immediate postoperative lateral and craniocaudal radiographs of the left stifle joint.	73
Figures 37a&b	Case 3. Preoperative lateral and craniocaudal radiographs of the right stifle joint.	76
Figures 38a&b	Case 3. Three month postoperative lateral and craniocaudal radiographs of right stifle joint.	76
Figure 39	Case 5. Preoperative craniocaudal radiograph of the right stifle joint.	79
Figure 40	Case 5. Immediate postoperative craniocaudal radiograph of the right stifle joint.	79
Figures 41a&b	Case 8. Preoperative lateral and craniocaudal radiographs of the left stifle joint.	83
Figures 42a&b	Case 8. Immediate postoperative lateral and craniocaudal radiographs of the left stifle joint.	83
Figures 43a&b	Case 10. Preoperative lateral and craniocaudal radiographs of the left stifle joint.	86
Figures 44a&b	Case 10. Immediate postoperative lateral and craniocaudal radiographs of the left stifle joint.	86

Figures 45a&b	Case 11. Preoperative lateral and craniocaudal radiographs of the right stifle joint.	88
Figures 46a&b	Case 11. Immediate postoperative lateral and craniocaudal radiographs of the right stifle joint.....	88
Figure 47	Case 9. Twelve week postoperative craniocaudal radiograph of the left stifle joint.	94
Figures 48a&b	Case 1. Sixty-seven-and-a-half week postoperative lateral and craniocaudal radiographs of the left stifle joint.....	100
Figures 49a&b	Case 11. Thirty-three week postoperative lateral and craniocaudal radiographs of the right stifle joint.	100
Figure 50	Case 4. Thirty-two week craniocaudal radiograph of the right stifle joint.....	101

TABLE OF TABLES

Table 1	Increase in Joint Motion Following Severance of the Femorotibial Ligaments in Dogs.....	54
Table 2	Summary of Patient Data..	89
Table 3	Summary of Findings at Early Postoperative Examination.	93
Table 4	Summary of Findings at Long-Term Postoperative Examination.	96
Table 5	Summary of Findings at Long-Term Postoperative Examination contd.....	97
Table 6	Preoperative and Long-Term Postoperative Lateral Radiograph Scores.....	98
Table 7	Preoperative and Long-Term Postoperative Craniocaudal Radiograph Scores.....	99

ACKNOWLEDGEMENTS

I am grateful to my supervisor Dr. Mike Harvey for enabling me to pursue this study in the Department of Veterinary Surgery and for his support during the period of the project. I would also like to thank Professors Andrew Nash and Ian Griffiths for their helpful comments on the manuscript.

I am indebted to Dr. Martin Sullivan for assessing the radiographs in this study; and Dr. Stewart Reid for his practical help, and invaluable comments regarding the statistical analysis of the results. I would like to express my thanks to my colleagues Mr. Andrew Miller and Mr. Jim Anderson who were always willing to share their knowledge and experience. In particular, I am indebted to Jim, for his patience and practical advice in helping me come to grips with my computer to prepare this thesis.

I was assisted in the investigation of these cases by the technical staff of the Departments of Veterinary Surgery, Haematology and Pathology who analysed the synovial fluid samples, and by Miss Leslie Crawford and Miss Dawn Stewart who took most of the radiographs. I would also like to thank the many people who were involved in the care of these patients including: anaesthetists, clinical scholars, nurses and students. The contribution of the owners and their pets is deeply appreciated, especially those owners who went to extraordinary lengths to allow long-term evaluation.

The superb photography throughout this thesis was taken by Mr. Allan May to whom I am truly indebted. Also, my sincere thanks to Miss Susan Cain and Mr Alan Reid of the Veterinary Anatomy Department, for their help in the procurement, and advice in the preparation of the cadaver specimens.

Finally, I would like to thank Mrs. Sheila Wardlaw, and Miss Sara Wardlaw for proof-reading the manuscript; and Sara for being there with encouragement and moral support.

INTRODUCTION

The simultaneous rupture of two or more ligaments of the stifle joint is an uncommon injury in the dog (Hulse and Shires 1986). It is generally agreed that severe, high-energy trauma, imparted directly to the stifle joint, is required to cause multiple ligamentous injury; as the forces must overcome the multiple ligaments, menisci, tendons and muscles which interact to provide joint stability. The injury is most frequently reported occurring secondary to road traffic accidents (Aron 1988, Hulse and Shires 1986, Denny and Minter 1973).

Stifle ligament injuries occur in a variety of combinations and can result in varying degrees of gross joint instability. Clinically, the injuries usually result in a non-weight bearing lameness with subluxation or complete dislocation of the stifle joint. The cruciate and medial collateral ligaments are most commonly injured, perhaps because the point of impact of trauma on the lateral aspect generates tensile forces medially (Hulse and Shires 1986). Frequently, there are also other associated soft tissue and orthopaedic injuries (Aron 1988, Hulse and Shires 1986, Welches and Scavelli 1990). The diagnosis of multiple ligamentous stifle injuries is based on clinical and radiographic signs of joint instability as well as direct observation and stress palpation at the time of surgery (Aron 1988).

Treatment should be aimed at stabilising the stifle joint to allow the dog to regain effective use of the limb. Non-surgical treatment generally yields poor functional results and is not recommended (Laing 1993, Smith 1995). Several methods of surgical treatment have been reported including: primary repair or reconstruction of involved joint restraints (Hulse and Shires 1986), extra-articular stabilisation (Aron 1988, Smith 1995), and trans-articular pinning (Welches and Scavelli 1990). Good functional results have been achieved with all of these methods, however, reduction in stifle joint range of motion is a consistent postoperative finding. Stifle joint arthrodesis or limb amputation are options for chronically unstable or painful joints (Brinker and others 1990).

Successful treatment of multiple ligamentous stifle injuries requires a thorough understanding of all stifle supporting structures, their functional role and dynamics in joint stabilisation, and their response to injury. This information is summarised in a broad review of the literature and is presented as an introduction to the clinical case study. It will be noted that the findings presented in this clinical case study differ in a number of ways from that which has been previously reported in the literature.

SECTION 1

BASIC SCIENCE

SECTION 1: PART 1

THE STABILISING STRUCTURES OF THE CANINE STIFLE

INTRODUCTION

The stifle joint is a complex diarthrodial condylar joint, representing a moveable link between the ends of the femur and the tibia. It is supported by soft tissues and the free motion of the femur relative to the tibia is limited by the restraints of ligaments, joint geometry, weight-bearing compressive forces across the joint, and by the active restraints generated by the muscles (Noyes and others 1984).

Traumatic injuries to the stifle joint are common and often occur as a result of overloading or insufficiency of the collagenous supporting structures. Muscle reaction can potentially provide protection to a joint by limiting its displacement; however, traumatic injury to the stifle often occurs at high speed, and muscle reaction time is frequently too long to enable muscle protection (Noyes and others 1984). It is, therefore, the collagenous tissues that play a major role in stabilising the stifle joint.

The stabilising structures of the stifle joint have been classified as either primary or secondary joint restraints in accordance with their order of importance in maintaining joint stability (Noyes and others 1980). This classification system determines the primary "workers" from the secondary "helpers" in resisting a certain joint displacement and is based on the percentage of total restraining force that each of the supporting structures provide the joint. For example, in man the cranial cruciate ligament is the primary restraint against cranial translation of the tibia with respect to the femur and provides 85% of the total resisting force. All other ligaments and capsular structures provide the remaining secondary restraint; each typically less than 3% (Butler and others 1980).

PRIMARY JOINT RESTRAINTS

The primary joint restraints of the stifle are: the cranial cruciate ligament, caudal cruciate ligament, medial collateral ligament, and the lateral collateral ligament.

The Cranial and Caudal Cruciate Ligaments

Anatomy

The cranial and caudal cruciate ligaments are dynamic intra-articular structures composed primarily of longitudinal bundles of collagen and reticular fibres, fibroblasts and fibrocytes embedded in an amorphous matrix of proteoglycan. Occasionally histiocytes, fat cells, pigment cells, lymphocytes and polymorphs may be present (Bennett 1990). They are morphologically and biomechanically undistinguished from tendons or other joint ligaments (Leach and Jacobs 1990). Each cruciate ligament is covered by a well vascularised synovial membrane which is continuous with that of the femorotibial joint space (Arnoczky and others 1979).

The paraligamentous blood supply of the cruciate ligaments arises from the synovial membrane and this communicates with the intrinsic vessels of each ligament. Very few intraligamentous vessels anastomose with endosteal vessels of the femur or tibia (Arnoczky and others 1979). The cruciate ligaments have an abundant nerve supply which have important mechanoreceptive and proprioceptive functions (Yahia and others 1992).

The following anatomical description is based on that of Arnoczky and Marshall (1977).

The cranial cruciate ligament (CrCL) is composed of two component parts; the craniomedial band (CrMB) which consists of a smaller band of fibres, and the caudolateral part (CaLP) which forms the bulk of the ligament. Most fibres originate from a fossa on the caudal aspect of the medial side of the lateral femoral condyle. The femoral attachment of the CrCL is in the form of a segment of a circle with its long axis in the vertical orientation (Figure 1, page 5). The CrMB originates from the craniodorsal aspect of this area with some fibres from the caudal lateral aspect of the intercondylar area.

From its femoral attachment the CrCL courses cranially, medially, and distally across the intercondylar fossa and attaches to the cranial intercondyloid area of the tibia. The tibial attachment is comma-shaped and has a general craniocaudal orientation (Figure 1, page 5). Fibres that make up the CrMB insert on the craniomedial aspect of the tibial attachment. From origin to insertion, the fibres of the CrCL have a proximal-to-distal outward spiral of about 90 degrees and as the stifle is flexed the CrCL becomes wound and twisted.

The caudal cruciate ligament (CaCL) is composed of two component parts; the caudal band (CaB) which consists of a smaller band of fibres, and the cranial part (CrP) which forms the bulk of the ligament. Both parts arise from an ellipsoid area at a fossa in the distal aspect of the axial side of the medial femoral condyle (Figure 2, page 5). The most cranial part of the femoral attachment reaches the articular surface of the femoral trochlea. In almost two thirds of specimens examined by Arnoczky and Marshall (1977), the femoral attachment of the CaCL also contained fibres of the femoral ligament of the lateral meniscus.

From its femoral attachment, the CaCL passes caudodistally to the medial side of the popliteal notch (Figure 2, page 5). The orientation of the femoral and tibial attachments of the CaCL cause it to spiral slightly in the opposite direction to that of the cranial cruciate ligament. Not only is the CaCL slightly longer and broader than the CrCL it is medial to it and crosses it (Figure 3, page 6).

Cruciate Ligament Dynamics

The two component parts of the cranial and caudal cruciate ligaments function independently of one another in flexion and extension. Each part of the cranial cruciate ligament is taut in extension. In flexion, however, the CrMB remains taut as the ligament twists and the CaLP becomes loose (Figures 4 & 5, page 7). The CrP of the caudal cruciate ligament is taut in flexion and loose in extension, whilst the CaB is loose in flexion and taut in extension (Figures 6 & 7, page 8).

The changes in the component parts of the cruciate ligaments is explained by the changes of the position of the points of attachment during flexion and extension. The femoral attachment of both cruciate ligaments is behind the axis of flexion, whereas only the tibial attachment of the cranial cruciate ligament is in front of it. As the stifle is flexed, the vertical femoral attachment of the cranial cruciate ligament becomes horizontal bringing the fibres of the CaLP closer to their tibial attachment and so the fibres become relaxed. However, the fibres of the CrMB remain taut during flexion, as their femoral point of attachment moves caudoventrally rather than cranially.

The horizontal femoral attachment of the caudal cruciate ligament assumes a vertical orientation as the stifle is flexed moving the CrP of the ligament cranially and away from its tibial attachment. These fibres become taut in flexion, whereas the origin of the CaB moves ventrally, closer to the tibial attachment and becomes loose.

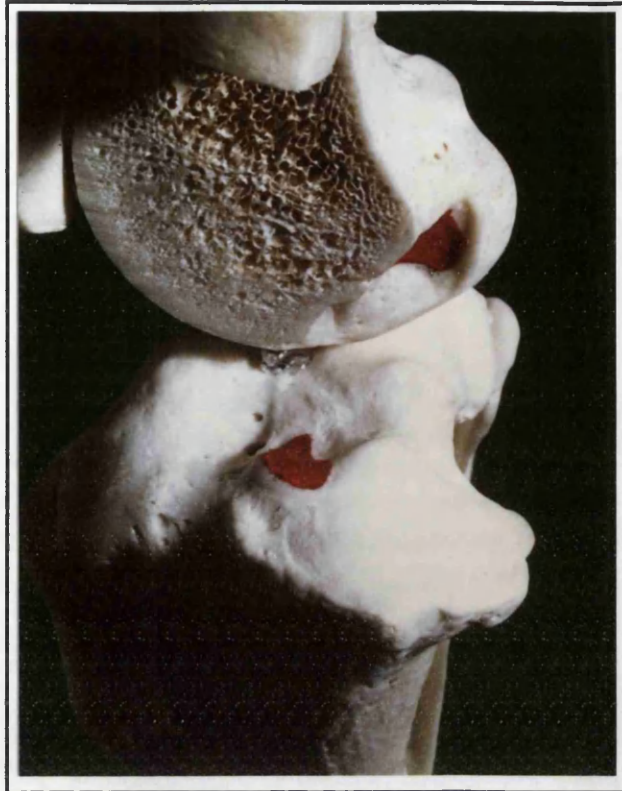


Figure 1 Origin and insertion of the right cranial cruciate ligament. The medial femoral condyle has been removed.

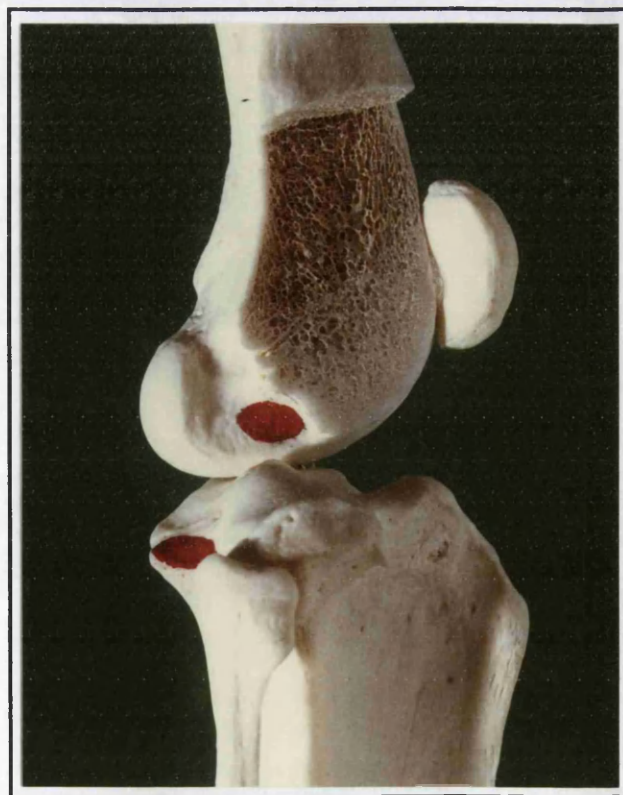


Figure 2 Origin and insertion of the right caudal cruciate ligament. The lateral femoral condyle has been removed.

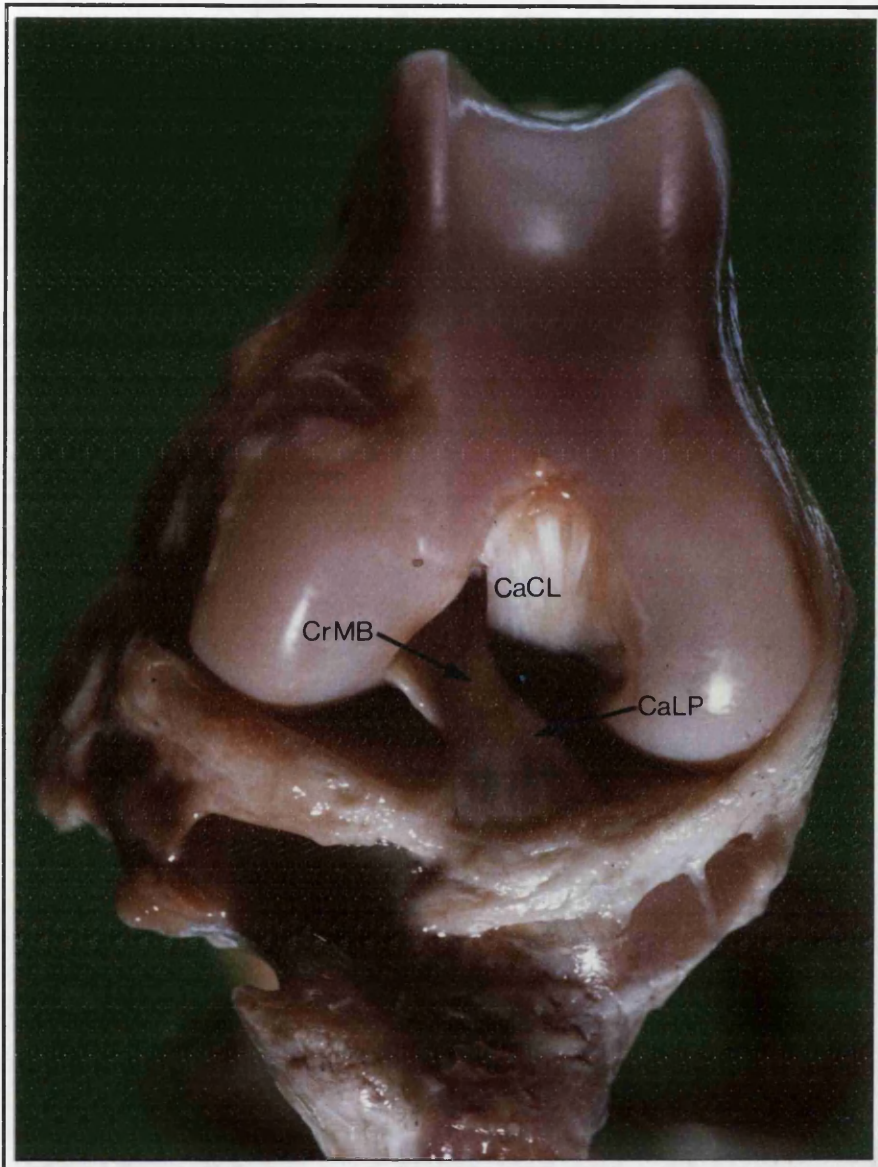


Figure 3 A frontal view of a dissected specimen of the right stifle joint. The patella, patellar ligament, infra-patellar fat pad and long digital extensor muscle have been removed. CaCL = caudal cruciate ligament, CrMB = craniomedial band of the cranial cruciate ligament, CaLP = caudolateral part of the cranial cruciate ligament.

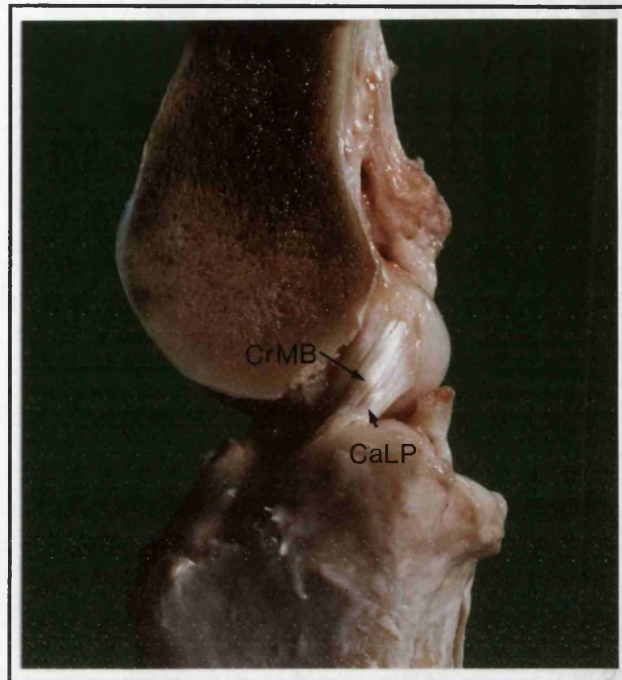


Figure 4 Side view of the right stifle to show the shape, orientation and tension of the cranial cruciate ligament in extension. The medial femoral condyle has been removed. Both the CrMB and the CaLP are taut. CrMB = craniomedial band, CaLP = caudolateral part.

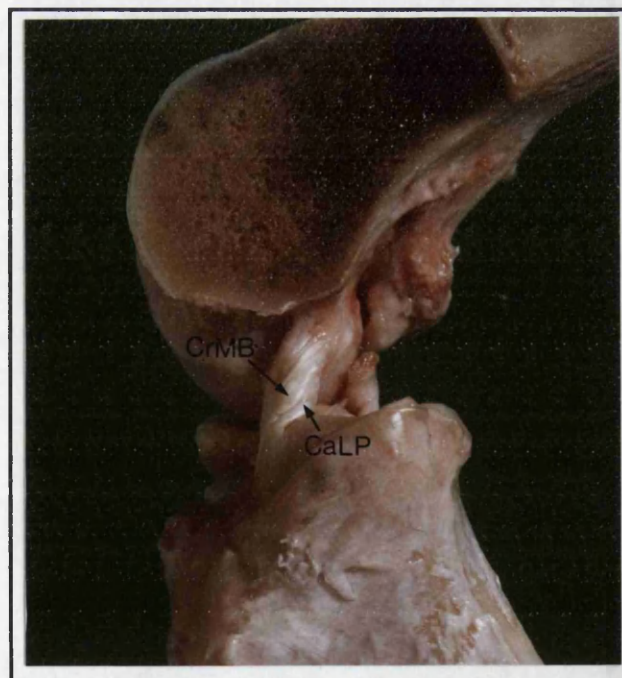


Figure 5 Side view of the right stifle to show the shape, orientation and tension of the cranial cruciate ligament in flexion. The medial femoral condyle has been removed. The CrMB is taut and the CaLP is loose. CrMB = craniomedial band, CaLP = caudolateral part.

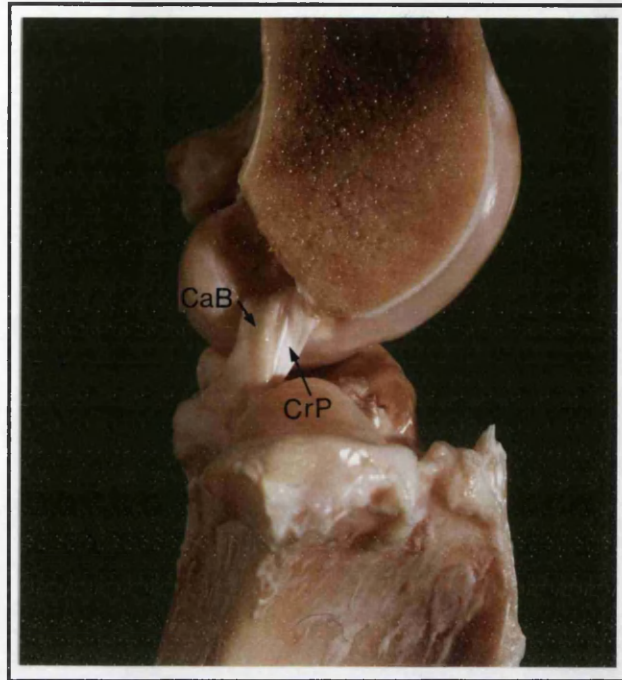


Figure 6 Side view of the right stifle to show the shape, orientation and tension of the caudal cruciate ligament in extension. The medial femoral condyle and fibula have been removed. The CrP is loose and the CaB is taut. CrP = cranial part, CaB = caudal band.

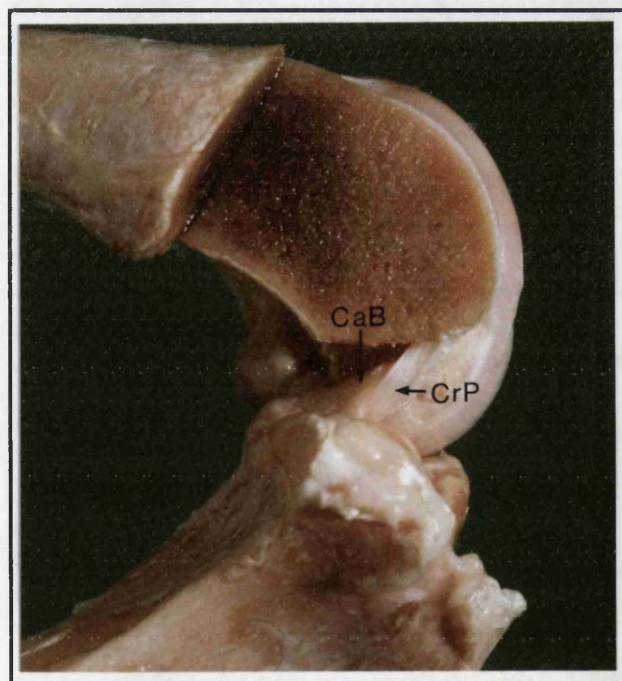


Figure 7 Side view of the right stifle to show the shape, orientation and tension of the caudal cruciate ligament in flexion. The medial femoral condyle and fibula have been removed. The CrP is taut and the CaB is loose. CrP = cranial part, CaB = caudal band.

The Medial and Lateral Collateral Ligaments

Anatomy

The collateral ligaments are strong extra-articular ligaments with close associations with the joint capsule. They are not covered with synovial membrane and their nerve and paraligamentous vascular supply is chiefly from the joint capsule and surrounding connective tissues (Rudy 1974).

The following anatomical description is based on that of Vasseur and Arnoczky (1981).

The origin of the medial collateral ligament is from an oval area on the medial femoral epicondyle. The ligament extends distally and blends with the joint capsule, forming a strong attachment to the joint capsule and the medial meniscus. The fibres of the ligament are longitudinally orientated and maintain a uniform width. As the ligament extends distally across the medial tibial condyle, it passes superficial to the tibial insertion of the semimembranosus muscle and inserts over a large rectangular area of the proximal medial tibia (Figure 8, page 10). A fluid-filled bursa lies between the ligament and the tibia.

The origin of the lateral collateral ligament is from a discrete oval roughened area, just proximal to the tendon of origin of the popliteal muscle on the lateral femoral epicondyle. The ligament passes superficial to the popliteal tendon and extends caudodistally as a strong fibrous band to insert on the fibular head (Figure 9, page 10). The fibres of the ligament are orientated longitudinally and maintain a constant width. Loose connective tissue joins the ligament to the joint capsule but there is no attachment to the lateral meniscus. The lateral collateral ligament has a superficial component that arises from the area of the lateral femorofabellar ligament and contributes to the caudal border of the ligament. This superficial band merges with the major component of the ligament as it crosses the joint surface and then separates to insert diffusely over the fascia of the fibularis longus muscle (Figure 10, page 11).

Collateral Ligament Dynamics

Both ligaments are taut in extension. In flexion, the medial collateral ligament remains taut and the lateral collateral ligament becomes loose with its entire bulk folded. The caudodistal orientation of the lateral collateral ligament means that, as the joint is flexed, the points of origin and insertion become closer together and the ligament loosens (Figures 10 & 11, page 11). In contrast, the orientation of the medial collateral ligament is such that, in extension, the caudal margin of the ligament is taut and, in flexion, the cranial margin of the ligament is taut (Figures 12 & 13, page 12).

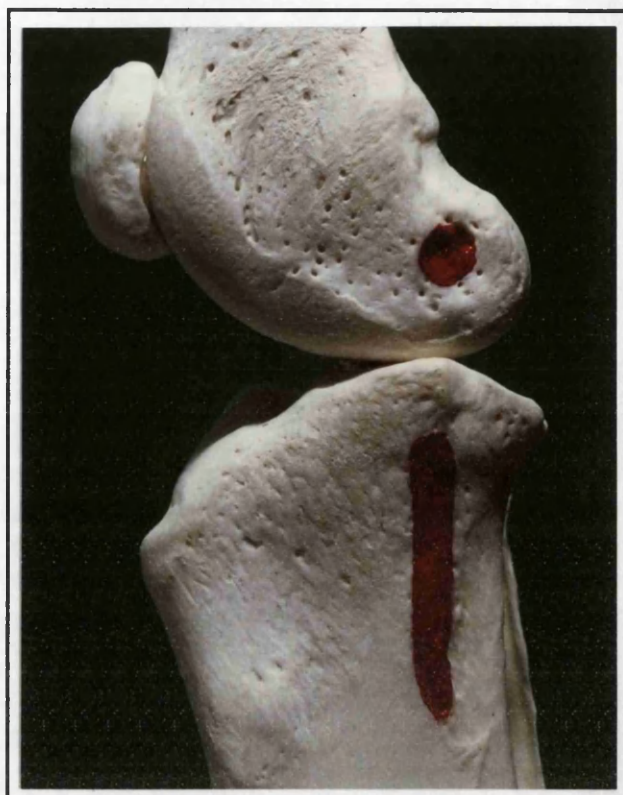


Figure 8 Origin and insertion of the medial collateral ligament.

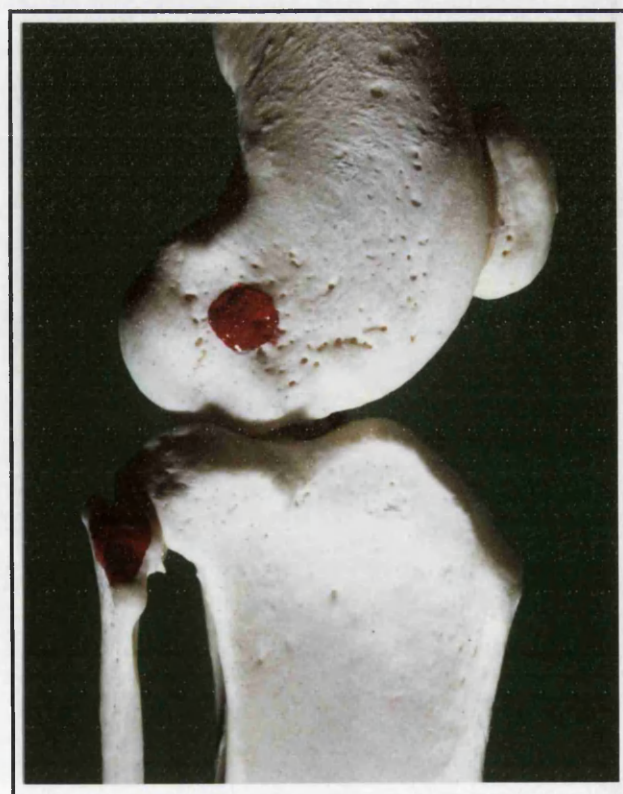


Figure 9 Origin and insertion of the lateral collateral ligament.

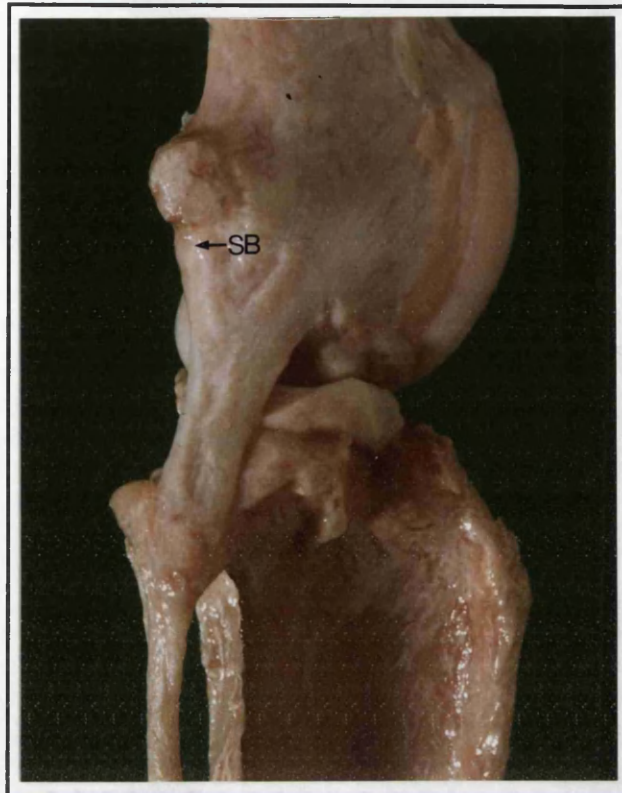


Figure 10 Lateral view of stifle to show the shape, tension and orientation of the lateral collateral ligament in extension. The entire ligament is taut. SB = superficial band.

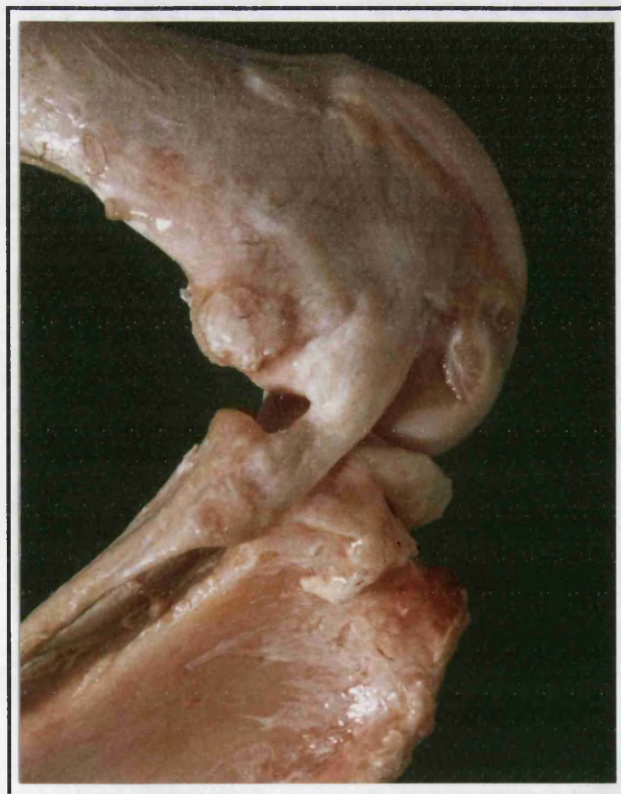


Figure 11 Lateral view of stifle to show the change in shape, tension and orientation of the lateral collateral ligament in flexion. The entire ligament is loose.

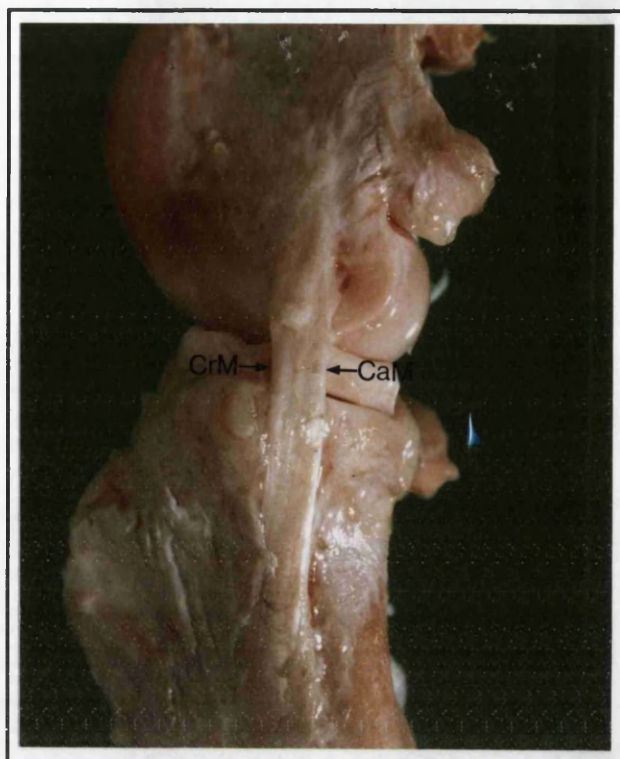


Figure 12 Medial view of stifle to show the shape, tension and orientation of the medial collateral ligament in extension. The CrM is loose and the CaM is taut. CrM = cranial margin, CaM = caudal margin.



Figure 13 Medial view of stifle to show the change in shape, tension and orientation of the medial collateral ligament in flexion. The CrM is taut and the CaM is loose. CrM = cranial margin, CaM = caudal margin.

SECONDARY JOINT RESTRAINTS

The secondary joint restraints of the stifle are the lateral and medial menisci, the joint capsule, the patella, and the patellar ligament.

The Lateral and Medial Menisci

Anatomy

The menisci are two biconcave, C-shaped discs located laterally and medially in the femorotibial joint space (Evans and Christensen 1979). Each meniscus lies adjacent to the articular cartilage of the corresponding femoral condyle proximally and the tibial condyle distally; a thin film of synovial fluid is interposed between the menisci and these articular surfaces. The menisci are wedge-shaped, in cross section, and each is attached by its thick convex peripheral border to the joint capsule; its thin concave inner border is directed axially. The lateral meniscus is slightly longer and narrower than the medial meniscus.

Normal menisci consist of dense fibrous tissue comprised largely of collagen with varying proportions of elastin, and in some cases, occasional chondrocytes. The collagen fibres are arranged in concentric layers running parallel to the abaxial border. In adults, only the thick abaxial portion of the menisci has a significant blood supply and the thin, translucent axial portion is essentially avascular (Tirgari 1978).

The menisci are held in position by six meniscal ligaments (Figure 14, page 14). The lateral meniscus is attached to the tibia by cranial and caudal meniscotibial ligaments. The caudal horn of the lateral meniscus is also attached to the femoral condyle by the meniscofemoral ligament. The medial meniscus is attached to the tibia by cranial and caudal meniscotibial ligaments. An intermeniscal ligament connects the caudal aspect of the cranial meniscotibial ligament of the medial meniscus to the cranial aspect of the cranial meniscotibial ligament of the lateral meniscus. The medial meniscus also has a fibrous attachment to the medial collateral ligament, and its peripheral margins are attached to the joint capsule by coronary ligaments. In contrast, the lateral meniscus has no collateral ligament attachment, and its loose joint capsule attachments are interrupted caudolaterally where the intra-capsular tendon of the popliteus muscle passes from its origin on the femur.

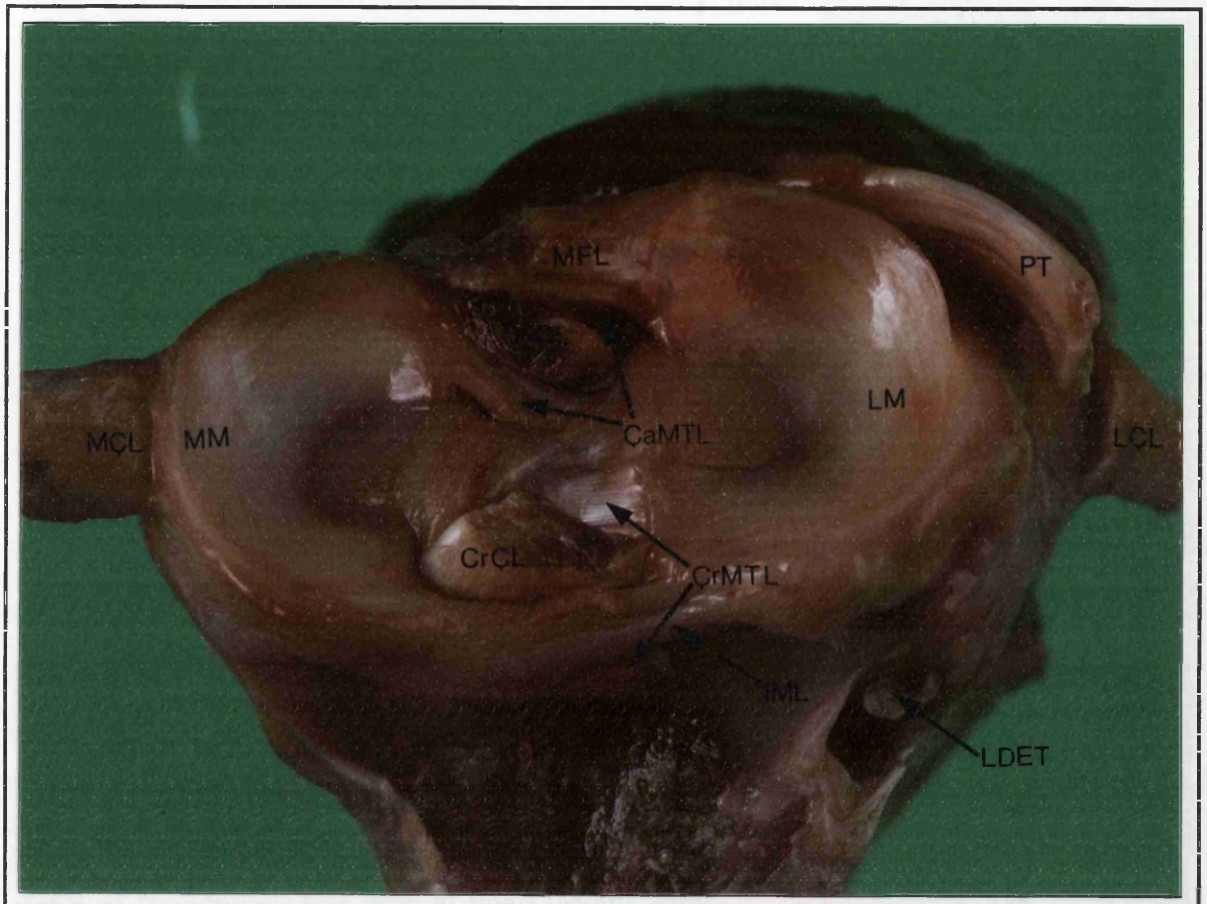


Figure 14 View of the proximal articular surface of the tibia showing the menisci and their attachments. CrCL = cranial cruciate ligament, CaCL = caudal cruciate ligament, MCL = medial collateral ligament, LCL = lateral collateral ligament, LDET = long digital extensor tendon, PT = popliteus muscle tendon, MM = medial meniscus, LM = lateral meniscus, CrMTL = cranial meniscotibial ligaments, CaMTL = caudal meniscotibial ligaments, MFL = menisiofemoral ligament, IML = intermeniscal ligament.

Meniscal Dynamics

Flexion and extension of the stifle joint causes the menisci to glide caudally and cranially over the tibial and femoral condylar articular surfaces. Significantly more movement occurs between the femur and the menisci than between the tibia and the menisci. The secure attachments of the medial meniscus to the medial collateral ligament and the joint capsule render it less mobile than the lateral meniscus. When the stifle is extended the menisci glide cranially until, in extreme extension, they reach the cranial border of the articular surface of the tibia. In flexion, the menisci glide caudally on the tibia and almost reach the caudal border of the tibial condyles. Further flexion results in sliding movement between the femur and the menisci only.

Flexion of the stifle joint is also associated with a degree of internal rotation of the tibia. During flexion, the medial meniscus remains relatively stationary near the axis of rotation through the medial condyle; whereas, the lateral meniscus is forced caudally on the lateral tibial condyle as the tibia rotates medially.

The menisci are subject to a certain amount of distortion during motion: in extreme flexion the cranial horns are stretched cranio-caudally while the caudal horns are rounded; in extension the distortion is reversed and the cranial horns are rounded and the caudal horns elongated (Dyce and others 1952).

The Patella and Patellar Ligament

Anatomy

The patella is the largest sesamoid bone in the canine skeleton and is intercalated in the tendon of insertion of the quadriceps femoris muscle which covers the cranial, medial, and lateral surfaces of the femoral shaft. The medial, intermediate, and lateral heads of the quadriceps femoris muscle originate on the proximal end and shaft of the femur. The cranial head (rectus femoris) originates by a strong tendon from the iliopubic eminence of the ilium. The quadriceps femoris muscle inserts on the tibial tuberosity as a strong tendon and the portion of this tendon running from the patella to the tibial tuberosity is called the patellar ligament.

The patella normally rides within the trochlear groove of the femur. It is held in this position by a strong fascial layer that surrounds the patella and patellar ligament and blends with the aponeurosis of the femoral fascia (fascia lata) and biceps femoris muscle on the lateral, medial, and cranial aspects of the stifle. The fascia lata extends medially as the crural fascia and is attached laterally and cranially to the quadriceps, tensor fascia lata, biceps femoris, and cranial sartorius muscles. The fascia lata has attachments to the collateral ligaments. In addition, patella stability is aided by delicate medial and lateral femoropatellar ligaments which are narrow bands of fibres that partially blend with the overlying fascia. These ligaments course between the patella and the medial epicondyle, and the patella and the lateral fabella. The patellar ligament is separated from the joint capsule by a large quantity of fat: the infra-patellar fat pad.

Patella and Patellar Ligament Dynamics

The quadriceps group of muscles gives cranioproximal traction to the tibia via the patella and patellar ligament and contraction of this muscle group results in extension of the stifle joint. To oppose the forces of gravity and bear weight, the dog must contract the quadriceps muscles which force the patella caudally into the trochlear groove transferring the load to the femur. When the stifle is extended the patella glides proximally within the trochlear groove. Flexion of the stifle results in the patella gliding distally within the trochlear groove and requires relaxation of the quadriceps muscles and contraction of the stifle flexor muscles (hamstring group). During flexion and extension, the fascia lata and the patella and patellar ligament move as one unit.

The Joint Capsule

Anatomy

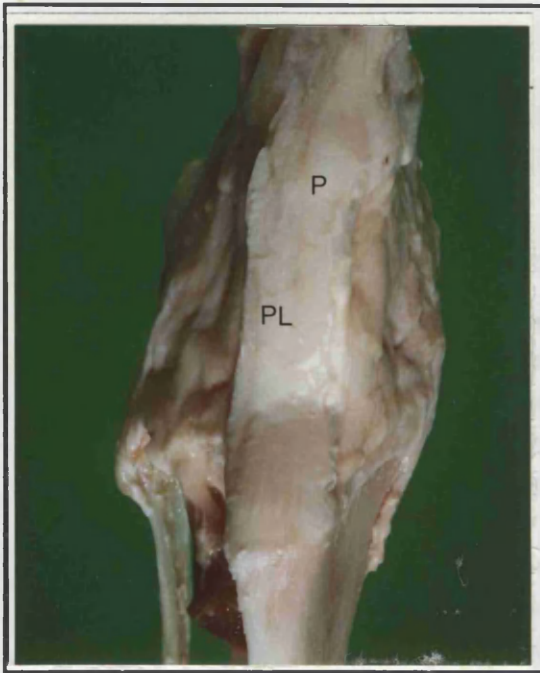
The joint capsule of the stifle is the largest in the body and contains between 0.2 and 2.0 ml of synovial fluid in a normal adult dog (Robins 1990). It is composed of two layers: an inner synovial membrane and an outer fibrous capsule.

The synovial membrane is composed of an intimal layer consisting of cells called synoviocytes, and an underlying subintimal layer of well vascularised connective tissue. The synovial membrane blends with the periosteum at the periphery of the joint and covers all intra-articular structures except the articular cartilage and the articulating surfaces of the menisci. It is responsible for the production of joint fluid and the removal of debris from the joint.

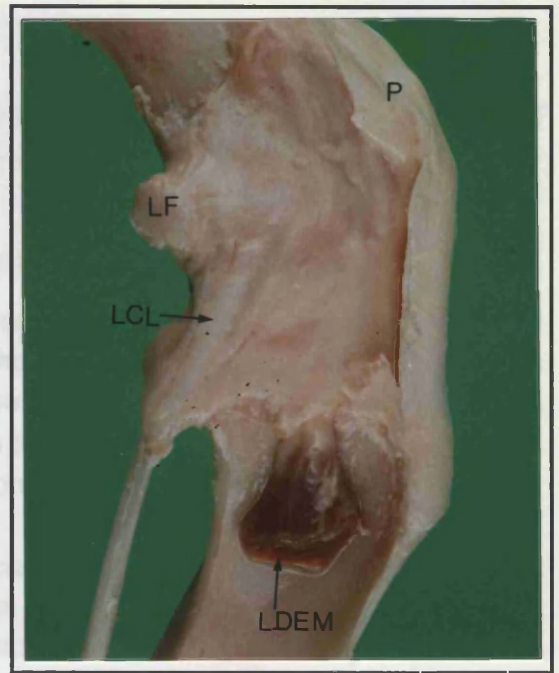
The fibrous capsule consists of dense, inelastic, fibrous connective tissue that is in close contact with the synovial membrane; except in the area distal to the patella where the infra-patellar fat pad is interposed between the two layers cranial to the intermeniscal ligament. The fibrous capsule is firmly attached to the periphery of both menisci, the margins of the articular surfaces of the patella and sesamoid bones, the femoral trochlea and condyles, the tibial condyles, and the head of the fibula.

The joint capsule forms three sacs that intercommunicate freely. The largest sac is beneath the patella where it extends laterally, medially, and proximally. Two smaller sacs are situated between the femoral and tibial condyles and are partially divided by the menisci and cruciate ligaments. These sacs have sub-pouches that extend to the lateral and medial fabellae, the tibiofibular joint, the extensor sulcus, (where it ensheaths the origin of the long digital extensor tendon), and between the tendon of the popliteus muscle and the lateral epicondyle of the femur (Evans and Christensen 1979).

The joint capsule attachments and associated structures are illustrated in Figures 15a-d, page 17.



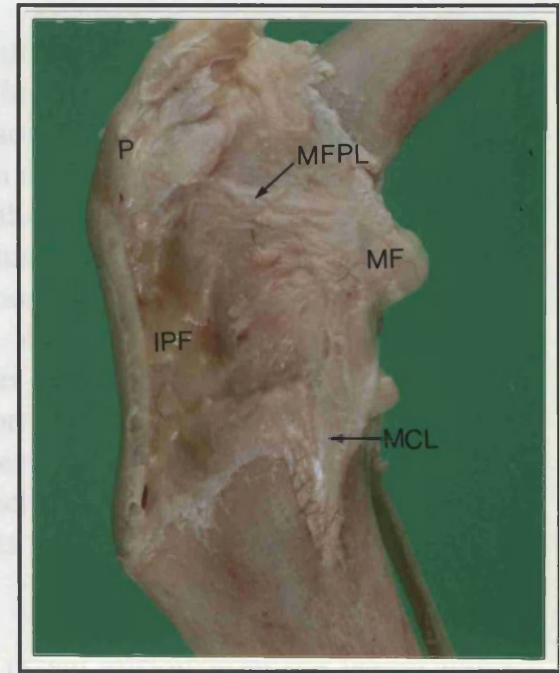
a.



b.



c.



d.

Figures 15a-d Cranial, lateral, caudal and medial views of the right stifle illustrating the joint capsule attachments and associated structures. P = patella, PL = patellar ligament, LF = lateral fabella, MF = medial fabella, LCL = lateral collateral ligament, MCL = medial collateral ligament, MFPL = medial femoropatellar ligament, LDEM = long digital extensor muscle, IPF = infra-patellar fat pad. N.B. The lateral femoropatellar ligament has been removed.

OTHER SUPPORTING STRUCTURES

In addition to the primary and secondary joint restraints, the integrated function of muscles and their tendons play a vital role in normal joint movement and stability (Rudy 1974).

Muscles and Tendons of the Stifle Joint

The cranial belly of the sartorius muscle inserts onto the fascia lata proximally and to the patella caudomedially. The caudal belly of the sartorius muscle forms an aponeurosis with the gracilis and semitendinosus muscles and inserts on the medial aspect of the proximal tibia. These muscles support the medial aspect of the joint, flex it, and cause a limited degree of adduction and medial axial rotation of the tibia.

The biceps femoris muscle is a large superficial muscle that covers the lateral aspect of the thigh. It inserts onto the fascia lata and exerts force in a proximal and caudolateral direction. This muscle contributes to rotational stability of the stifle during weight bearing, and the caudal part assists in flexion, outward rotation, and elevation of the crus when the limb is not bearing weight.

The gastrocnemius muscle arises from the distal caudal femoral shaft as two distinct medial and lateral tendons. Each tendon has an associated sesamoid bone, the fabellae, which articulates with a femoral facet just proximal to the corresponding femoral condyle. The lateral tendon of origin arises from a small lateral prominence of the distal femoral shaft just proximal to the lateral condyle. At this level it is related superficially to the biceps femoris muscle. Some of the tendon fibres from the lateral fabella pass cranially over the femoral attachment of the lateral collateral ligament and insert on the lateral edge of the patella to form the lateral femoropatellar ligament. The medial tendon of origin arises from a small medial prominence of the distal femoral shaft just proximal to the medial femoral condyle and is related to the adductor muscle superficially. The cranial portion of the semimembranosus muscle has extensive attachments to the caudomedial surface of the gastrocnemius muscle, and fibres from its tendon course cranially to the patella to form the medial femoropatellar ligament.

The popliteus muscle has a long tendon of origin arising from the lateral femoral condyle just cranial and distal to the origin of the lateral collateral ligament. It passes caudodistally beneath the lateral collateral ligament and curves medially and caudally over the tibial condyle to form a broad fanlike insertion on the caudomedial surface of the proximal tibial shaft. The tendon is in intimate contact with the peripheral edge of the lateral meniscus and contains a small sesamoid bone where it curves over the lateral tibial condyle. This muscle assists in flexion of the stifle, but it acts principally to rotate the tibia inward during flexion.

The long digital extensor muscle arises from the extensor fossa of the femur just cranial and proximal to the origin of the popliteus tendon. Its tendon of origin is covered by synovial membrane and courses intra-articularly through the sulcus muscularis of the tibia. A pouch of joint capsule forms a synovial sheath around the tendon as it emerges from the joint for three to four centimetres. The principal action of the long digital extensor muscle is to extend the digits and flex the tarsus, but it also acts as a minor extensor of the stifle and provides some lateral support to the joint (Evans and Christensen 1979).

SECTION 1: PART 2

MOVEMENT AND STABILISING MECHANISMS OF THE STIFLE JOINT

STIFLE KINESIOLOGY

Free motion of the femur relative to the tibia can occur about or along three mutual orthogonal axes and results in six basic movements of the stifle joint (Figure 16, page 21). While each motion (*e.g.*, flexion-extension, varus-valgus angulation, axial rotation, cranio-caudal translation) may be present to some degree in normal stifle joint function, individual movements on or about a specific axis are controlled by femoral condylar geometry and soft tissue constraints (Arnoczky 1985).

In normal movement the stifle has a combined motion in three planes. Flexion and extension takes place about the x (or transverse) axis, while rotatory movement occurs about the y (or longitudinal) axis. The average normal stifle range of motion about the x axis in the dog has been recorded as being 110 degrees, from 40 degrees in flexion to 150 degrees in full extension (Rudy 1974). With passive movement Newton and Nunamaker (1985) recorded the normal stifle range of motion as 130 to 150 degrees. Rotatory movement changes with flexion and extension. In full extension, an average of six degrees of internal rotation and five degrees of external rotation can be obtained and in 90 degrees of flexion, an average of 19 degrees of internal rotation and eight degrees of external rotation may be obtained (Arnoczky and Marshall 1977). Flexion and extension motion predominates during the weight bearing phase of the gait, whereas axial rotation is most pronounced during flexion without weight bearing and allows the leg to fold beneath the body.

As the stifle joint flexes and extends, the axis of flexion of the femur (x axis) relative to the tibia does not remain constant but moves along the z axis. At any one instant, however, there is a point on the femur that has zero velocity with respect to the femur. This point constitutes the instant centre of motion (Arnoczky and others 1977) (Figure 17, page 22). Joints that have the instant centre located on the articular surface have a rolling motion with minimal friction loss or wear. Where the instant centre lies on a line perpendicular to the surface at the point of joint contact, there is a sliding motion of the articular surfaces. The least resistance to sliding motion will occur when the direction of velocity of the surface point is at a tangent to the contact point surface. During normal flexion and extension of the stifle in the dog there is combined rolling-sliding motion (Arnoczky and others 1977).

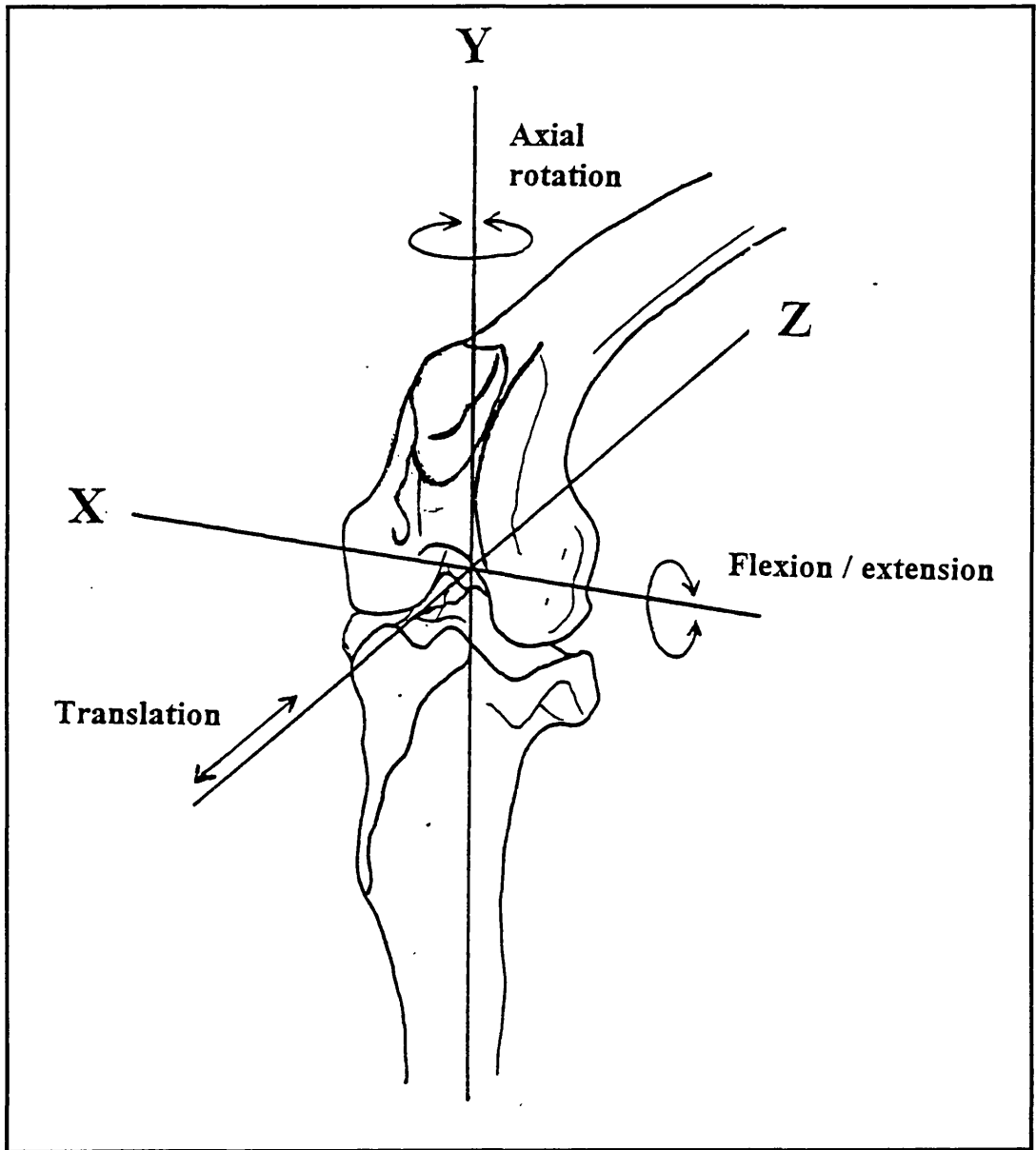


Figure 16 Schematic drawing of the stifle joint of the dog illustrating the three axes of motion (x,y, and z) and their orientation.

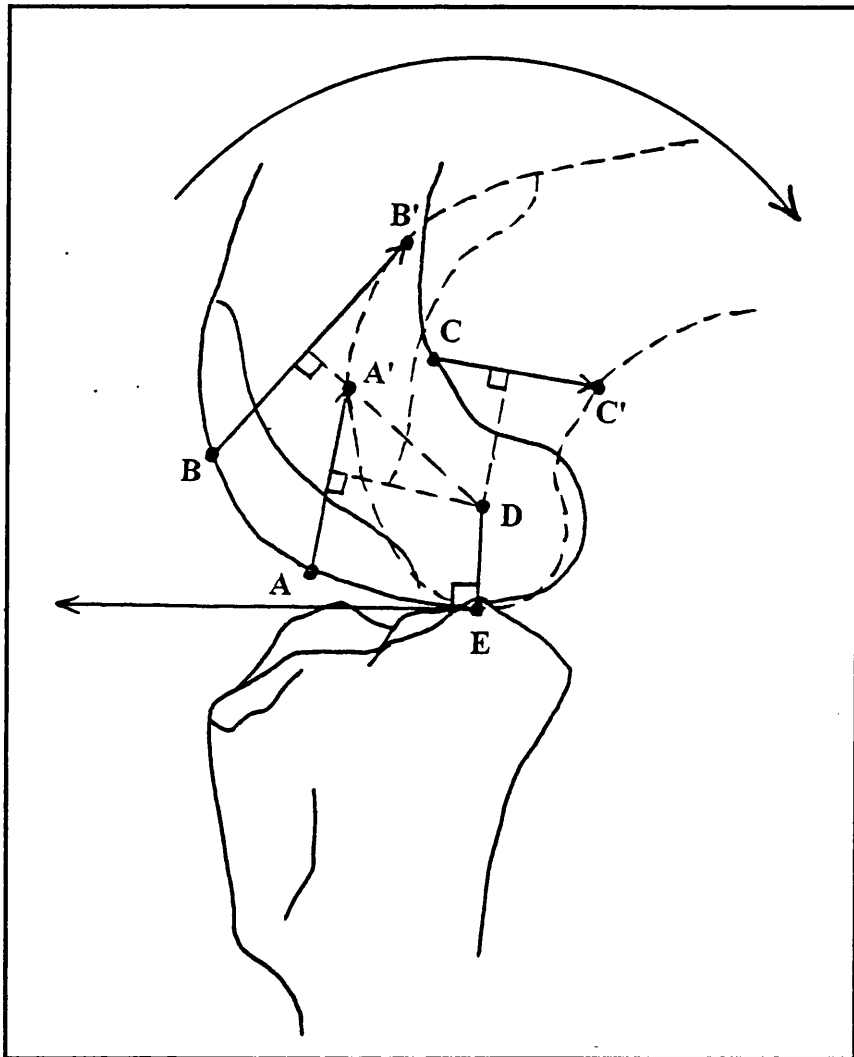


Figure 17 Diagram illustrating the determination of the instant centre of motion. During the femoral motion shown, the points A, B and C displace to A', B' and C' respectively represented by the lines AA', BB' and CC'. Perpendicular bisectors of these lines intersect at the instant centre of motion (D) for the displacement shown. The positioning of the instant centre on a line perpendicular to the articular surface at the point of joint contact (E), indicates that the articular surfaces will be sliding on each other with relatively free and normal action. (Based on illustrations from Arnoczky and others 1977).

THE CRUCIATE LIGAMENTS

The cruciate ligaments are the primary restraints for craniocaudal motion along the z axis. The cranial cruciate ligament primarily functions to prevent cranial displacement of the tibia relative to the femur (cranial drawer movement), whereas the caudal cruciate ligament prevents caudal movement of the tibia relative to the femur (caudal drawer movement). Injury to the cranial or caudal cruciate ligament results in an abnormal movement between the femur and the tibia during flexion and extension.

The cruciate ligaments are the primary restraints of excessive internal rotation in the flexed stifle. The cranial and caudal cruciate ligaments are able to provide axial stability due to their spatial orientation within the joint. As the stifle is flexed, the cruciate ligaments twist about each other to limit internal tibial rotation. Severance of the cranial cruciate ligament increases internal rotation by an average of nine degrees in extension and 26 degrees at 90 degrees of flexion (Arnoczky and Marshall 1977). Neither ligament alone significantly limits external rotation (Arnoczky and Marshall 1977).

The cranial cruciate ligament is the primary restraint limiting cranial rolling movement of the condyles during terminal extension. When the stifle is in full extension, the femoral condyles rock cranially on the tibial condyles placing the articular surfaces of the femur and the tibia at their most cranial point of contact and forcing the menisci to slide forward. Further cranial movement of the articular condyles is prevented by the cranial cruciate ligament. Transection of the cranial cruciate ligament alone resulted in an average increase of 12 degrees of hyperextension. When both cruciate ligaments were transected there was an average increase of 18 degrees of hyperextension (Arnoczky and Marshall 1977). The caudal cruciate ligament therefore acts as a secondary restraint to the cranial cruciate ligament in limiting hyperextension.

The cruciate ligaments are secondary restraints to the collateral ligaments in limiting abnormal valgus (lateral) and varus (medial) angulation. Severance of either collateral ligament yields only slight increases in varus or valgus angulation and further displacement is limited by the cruciate ligaments (Vasseur and Arnoczky 1981).

Both cruciate ligaments together play a role in limiting hyperflexion of the stifle. However, measuring this accurately is difficult and there is a considerable variation between individuals (Arnoczky and Marshall 1977).

THE COLLATERAL LIGAMENTS

In extension the collateral ligaments are the primary restraints against abnormal varus and valgus angulation.

When the stifle is in extension, both collateral ligaments are tight and they restrain the tibia against abnormal internal and external rotation. With flexion of the stifle the lateral collateral ligament begins to relax. This allows caudal displacement of the smaller lateral femoral condyle on the tibial plateau and results in internal rotation of the tibia on the femur. Conversely, as the stifle is extended, the lateral collateral ligament tightens and the lateral femoral condyle moves cranially on the tibial plateau causing external rotation of the tibia on the femur. In man, this motion has classically been described as the "screw-home" mechanism. The axis of rotation is approximately in the centre of the medial femoral condyle (Arnoczky and others, 1977).

The medial collateral ligament is important in stabilisation of the stifle joint because its fibres remain under tension in both flexion and extension. It is the only medial structure that limits lateral tibial displacement, angulation, or external rotation of the tibia.

THE MENISCI

The menisci contribute to the stability of the stifle joint by nature of their anatomy, forming moveable cups for each femoral condyle. They increase the depth of the articular surface of the proximal tibia and make up for the incongruity between the adjacent articular surfaces of the femur and tibia. The menisci are secondary restraints for abnormal craniocaudal, varovalgus and axial movement of the femur and tibia during flexion and extension (Seale and Haynes 1981). The stabilising effects of the menisci are enhanced in extension by weight bearing, and the function of the cranial cruciate ligament. During weight bearing the forces acting about the cranial cruciate ligament may be resolved into two components. The first force component resists cranial translation of the tibia with respect to the femur. The second is a compressive force that aids stability by driving the femoral condyles into the concave surfaces of the menisci (Figure 18, page 25). In this way the menisci act as energy wells, stabilising and centring the femur on the tibia (Smith 1995).

In addition, the menisci act as shock absorbers; they aid in the distribution of synovial fluid and, therefore, contribute to the nourishment and lubrication of articular cartilage. The nerve supply to the menisci may serve a sensory function enabling fine control of the joint (O'Connor and McConnaughey 1978).

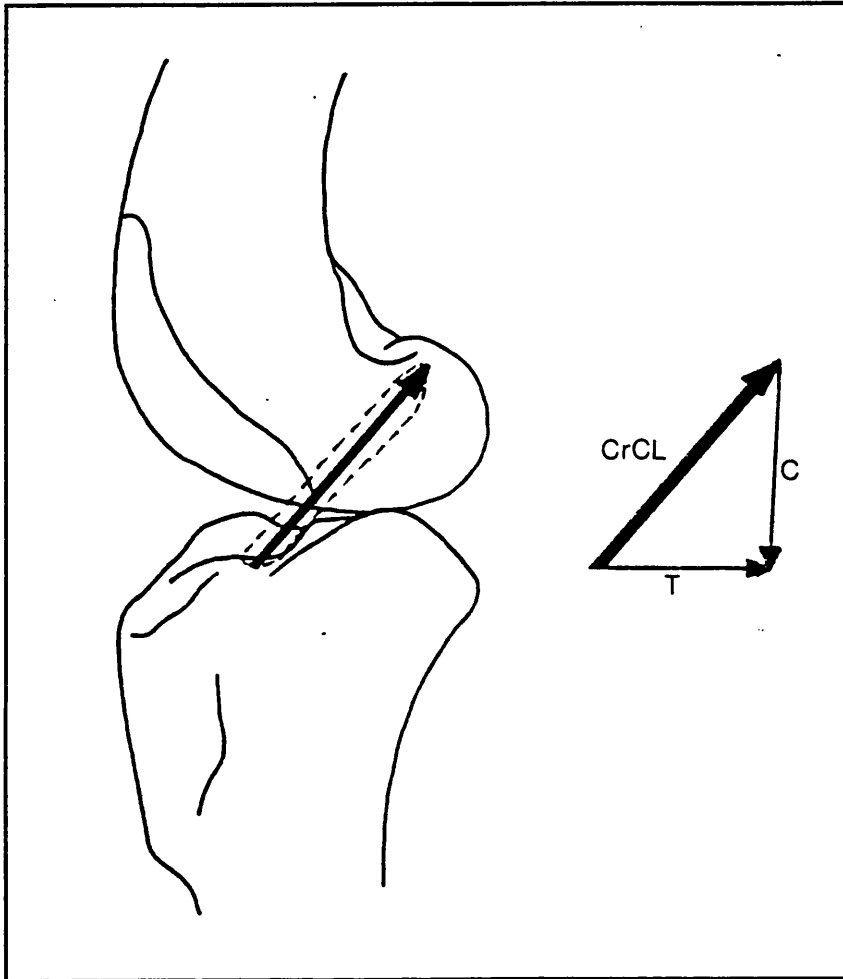


Figure 18 Diagram illustrating the compressive (C) and translational (T) force vectors acting about the cranial cruciate ligament (CrCL).

THE PATELLA AND PATELLAR LIGAMENT

In most dogs, the axis of the quadriceps muscle group, femoral trochlea, patella, patellar ligament, and tibial tuberosity is a straight line. At a normal standing angle the patella is forced against the femoral trochlea by the action of the quadriceps muscle group. In this position the patella exerts a stabilising force against axial rotation of the joint. The patellar ligament, by its insertion on the tibial tuberosity, normally exerts a constantly changing but continuous force in a proximal direction that tends to reduce tibial axial rotation in either direction. The combination of the cranial tibial traction via the patellar ligament and the caudal pressure on the femur by the patella is a biomechanical agonist to the cranial cruciate ligament (Henderson and Milton 1978). In full extension, the stabilising influence of the patella and patellar ligament is supplemented by the tensed collateral ligaments.

When the joint is not in full extension, and the extensor muscles of the stifle are relaxing or just beginning to contract then the patella is stabilised by the medial and lateral femoropatellar ligaments (Rudy 1974).

THE JOINT CAPSULE

The fibrous joint capsule acts as a secondary restraint for abnormal craniocaudal and axial movements and, together with the collateral ligaments, as a primary restraint against abnormal varovalgus angulation in flexion and extension (Rudy 1974).

MUSCLES AND TENDONS

Although the muscles of the stifle contribute to stifle stability, their major function is the movement of the components of the joint.

The quadriceps femoris, tensor fascia lata, cranial belly of the sartorius, and biceps femoris muscle all provide strong support to the cranial, medial, and lateral aspects of the stifle. Their dynamic action together with that of the popliteus muscle plays a role in controlling the axial rotation of the joint. The biceps femoris, gracilis, semimembranosus, and caudal belly of the sartorius muscles, by the direction of their fibres, all support the function of the cranial cruciate ligament. The long digital extensor tendon which crosses the cranial surface of the stifle weakly contributes to cranial and lateral stifle support (Henderson and Milton 1978).

During weight bearing there is continuous tension in the gastrocnemius muscle. The continuous stifle flexion force of this muscle counterbalances the action of the quadriceps femoris muscle and the fascia lata, and contributes strong caudal support to the joint (Henderson and Milton 1978).

The popliteus muscle supplies a strong caudal support to the lateral meniscus, and holds it against the lateral femoral condyle during inward rotation of the tibia (Rudy 1974).

SECTION 1: PART 3

PATHOPHYSIOLOGICAL CONSIDERATIONS

LIGAMENT PROPERTIES

A ligament is a dense, very stable band of relatively nonelastic tissue that unites two or more bones. It is composed predominantly of long concentric and parallel collagen fibres. Ligaments effectively maintain proper osseous spatial relationships by providing a limit to a given range of motion (Evans and Christensen 1979). They have a degree of flexibility and allow a certain amount of motion to occur at their origin and insertion points. However, their nonelastic properties means that they can be stretched only to a limited degree before they rupture or "tear" (Farrow and Newton 1985).

CLASSIFICATION OF LIGAMENT INJURY

Sprain

Direct or indirect trauma to a joint may be defined as a sprain. Traditional sprain classification schemes have focused on the qualitative aspects of the ligamentous injury and the integrity of the ligament fibres. Although this system is useful in categorising the gross severity of the ligamentous injury, it does not measure residual ligament strength and therefore may not always reflect the functional capacity of the injured ligament (Noyes and others 1984).

First-degree Sprain

A first-degree sprain or mild injury involves minimal tearing of the ligament fibres, as well as a varying degree of internal haemorrhage.

Physical and Radiographic Findings

Mild sprains are associated with minimal lameness. There is mild to moderate soft tissue swelling over the affected ligament and tenderness on palpation. There may be a variable amount of pain on manipulation of the joint.

Minimal regional soft tissue swelling may be the only detectable finding radiographically.

Second-degree Sprain

A second-degree sprain or moderate injury results in a definite structural breakdown following partial tearing of the ligament fibres. Haemorrhage is both internal and periligamentous, with moderately extensive inflammatory oedema.

Physical and Radiographic Findings

Moderate sprains are associated with obvious lameness and swelling. There is pain on palpation and this is readily elicited with minimal manipulation of the joint. There is no apparent instability of the joint.

Regional soft tissue swelling is detectable radiographically and this is usually evident both intra-capsularly and extra-capsularly. However, stressed radiographs fail to demonstrate spatial derangements in the joint.

Third-degree Sprain

A third-degree sprain or severe injury results in the loss of functional load-carrying ability which usually involves the complete rupture of the ligament and loss of continuity. Complete ligament rupture may occur within the ligament body, or at the ligament origin and insertion points, and may be associated with the avulsion of its bony attachment.

Physical and Radiographic Findings

These types of injuries are very painful and there is minimal or no weight bearing of the affected limb. Gross swelling is present and this can extend some distance from the site of injury. There is extreme pain on palpation or manipulation of the joint and this is frequently accompanied by crepitation and abnormal mobility.

Radiography reveals soft tissue swelling. Joint mal-alignment is often apparent and readily demonstrable with stressed radiographs. Avulsion fractures at the points of ligament origin and insertion may also be apparent.

MECHANISMS OF LIGAMENT FAILURE

Mechanical Properties

When ligaments and isolated collagen fibres are tested to failure under experimental conditions they produce similar shaped load-deformation curves. However, the absolute values of the x and y axes of the load-deformation curves differ between ligaments, and between ligaments and isolated collagen fibres. For example, when isolated collagen fibres are tested, collagen fibre failure begins at 7-8% strain, whereas, when the properties of whole ligaments are examined, ligament failure may begin at strain levels of 20 to 40% (Shah and others 1977, Noyes and others 1974). Some of the differences in these mechanical properties is related to differences in ligament microstructure, tissue dimensions, loading rates, clamping and loading configuration, and other sources of artefact in the testing technique (Noyes and others 1984).

The standard shape of the load-deformation curve to failure of a ligament is illustrated in Figure 19, page 29.

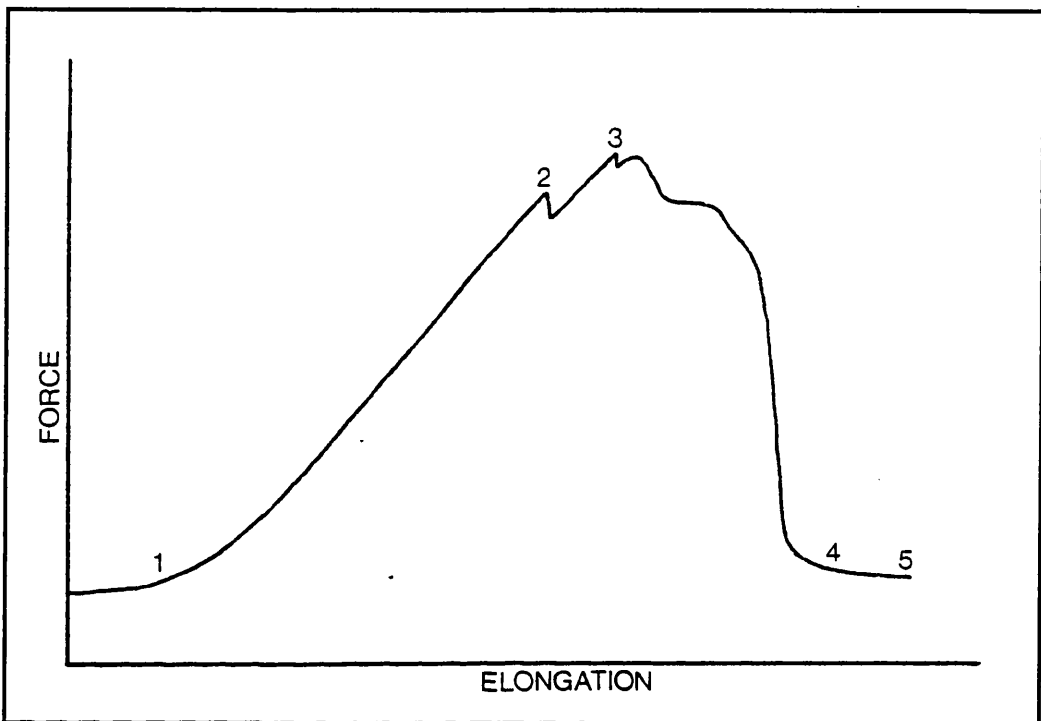


Figure 19 An example of the typical shape of a load-deformation curve for a tension test to failure of a bone/ligament/bone preparation. Based on tracings from Noyes and others (1974).

The initial concave portion of the curve (labelled 1) represents a structural change in fibril organisation from a condensed, undulating pattern to a more straight, parallel arrangement. In this region, little force is required to elongate the tissue and loading is viscoelastic so that removal of the force restores the undulating microscopic pattern and the initial length. Subsequently, the tissue becomes progressively more stiff, requiring greater increases in force to produce elongation. Further loading results in a near linear curve and the fibres are parallel. Near the end of the linear region, small force reductions occur representing sequential failure of a few greatly stretched fibres. This is termed the linear loading endpoint (labelled 2). Further elongation requires progressively less force and is accompanied by random progressive fibre bundle failure. The maximum load is attained (labelled 3) and complete failure occurs rapidly. The load-carrying ability of the ligament is lost (labelled 4) and this is followed by shear failure as the ligament fibres slide past each other until complete continuity of the ligament is lost (labelled 5).

The clinical significance of information gained by load-deformation curves is three fold. Firstly, visual inspection is not sufficiently sensitive to distinguish between normal structural continuity, microscopic failure, or residual elongation prior to ultimate failure. Secondly, a spectrum of partial ligament failure (second-degree sprain injury) exists, and each situation implies a significantly different capacity for the ligament to resist forces and provide joint stability. Thirdly, ligament continuity, as determined by arthrography, arthrotomy, or arthroscopy, is often an unreliable indicator of future ligament functional capacity (Noyes and others 1984).

Ligament Failure Modes

Noyes and others (1974) observed three principle failure modes in bone/ligament/bone preparations.

The first type is the most frequently encountered clinically and occurs as a result of failure through the substance of the ligament. As the ligament is stressed beyond its load carrying capacity, some fibre bundles become fully loaded and fail prior to others. Furthermore, different fibre bundles fail at different points along the length of the ligament. This phenomenon produces the classic "mop end" appearance of the interstitial ligament failure and involves injury to major portions of the collagenous structure throughout the ligament length. It is clinically important to recognise that future healing after surgical repair involves the ligament as a whole, and not just healing of the gross cleavage site.

The second and third types of failure occur at the ligament-bone attachment site. The attachment of ligaments to bone occurs via a transition from ligament to fibrocartilage, to mineralised fibrocartilage, and finally to lamellar bone (Cooper and Misol 1970). The second mode of failure occurs at the lamellar bone site and results in the not uncommon avulsion fracture.

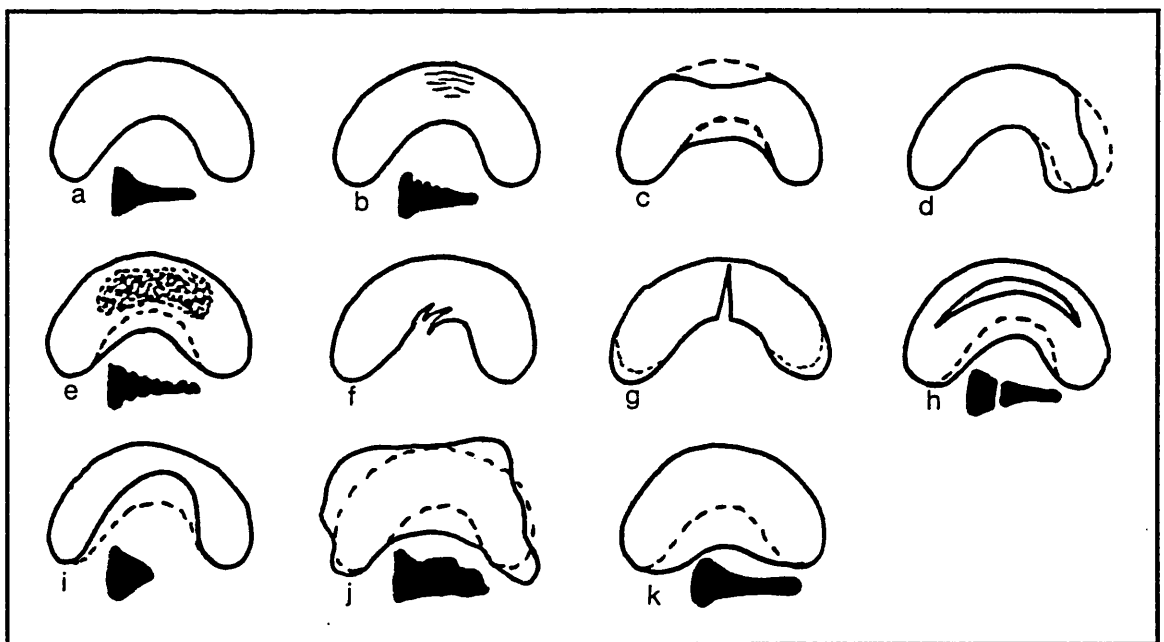
The third mode of failure is less common, produced by a cleavage at the ligament-bone interface. The level at which the cleavage occurs is variable, but is usually between the fibrocartilage and the mineralised fibrocartilage; or through the mineralised fibrocartilage itself.

The mode of the ligament failure is largely determined by the rate at which a force is applied. When the behaviour of ligaments is studied under high strain rates, typical of *in vivo* loading and trauma, there is a predominance of ligament substance tears. At slow strain rates, there is a predominance of premature bone avulsion, as bone is more rate sensitive and will fail at a lower load when the strain rate is slow (Noyes and others 1974).

INJURY TO THE MENISCI

Meniscal injury is common following injury to the primary stabilising structures of the stifle in dogs. Stifle joint instability associated with ligament injury results in abnormal compressive, shearing and rotational forces acting on the menisci. Meniscal injury prevalence of 48% (Flo 1975), 49% (Bennett and May 1991¹), 53% (Flo and DeYoung 1978) and 68% (Flo 1983) have been cited in association with partial or complete ruptures of the cranial cruciate ligament. The prevalence is thought to increase with body weight (Bennett and May 1991¹) and with the chronicity of joint instability (Arnoczky and others 1979¹). The medial meniscus is less mobile than the lateral meniscus as it is firmly attached to the tibia, joint capsule and medial collateral ligament. This renders it more susceptible to injury particularly in the cranial cruciate ligament deficient stifle. With cranial drawer instability and weight bearing, the caudal femoral condyle applies pressure directly onto the caudal rim of the medial meniscus and shears the longitudinal as well as radial fibres. The lateral meniscus, however, is less firmly attached to the tibia and is pulled caudally by its femoral ligament, so that the pressure of the femoral condyle is not concentrated on the meniscal rim (Flo and DeYoung 1978).

Meniscal injuries have been classified by Flo and others (1983), and Bennett and May (1991)¹ on the basis of their gross pathological appearance (Figures 20a-k, page 31).



Figures 20a-k Diagrams illustrating the types of meniscal injury. a = normal, b = surface fibrillation, c = abaxial peripheral avulsion, d = caudal horn peripheral avulsion, e = compression, f = axial fringe tear, g = transverse tear, h = longitudinal tear, i = atrophied meniscus, j = proliferative meniscus, k = discoid meniscus.

Surface fibrillation

This is characterised by a roughened femoral surface of the meniscus with tags of meniscus protruding from the surface. The tears do not traverse the entire meniscal thickness.

Peripheral avulsion

Peripheral avulsion of the caudal attachments, with cranial displacement and folding of the caudal horn of the medial meniscus, was the most common meniscal lesion seen by Bennett and May (1991)¹. The displacement is related to cranial movement of the tibia. Other types of peripheral avulsions are considered to be rare and usually occur with collateral ligament injury coupled with cruciate ligament rupture following severe trauma to the stifle (Flo and others 1983). With severe trauma, it is probable that the initial impact separates the meniscotibial and meniscofemoral capsular attachments (Hulse and Shires 1985).

Compression injury

This type of injury is seen when loss of stifle stability results in an abnormal concentration of forces on the menisci. Compression injuries are commonly seen following rupture of the cranial cruciate ligament where the femoral condyle repeatedly crushes the caudal horn of the medial meniscus during abnormal drawer movement (Flo and others 1983).

Axial fringe tear

Axial fringe tears are characterised by free tags of meniscus on the inner concave border. These are rarely seen in the dog and their significance for causing pain and cartilage degeneration is unknown (Flo and others 1983).

Transverse (radial) tear

This is a full thickness tear of the meniscus from the inner concave border and extending abaxially to varying degrees. Transverse tears occur very rarely in the dog (Flo and others 1983). They usually involve the medial meniscus and are secondary to abnormal rotatory forces resulting from cranial cruciate ligament injury. The abnormal displacement of the medial femoral condyle, coupled with the internal rotational instability of the ligament injury, results in excessive torsional forces on the meniscus (Hulse and Shires 1985).

Full thickness longitudinal tear

This injury may consist of single or parallel multiple splits in the meniscus, which traverse the entire meniscal length, and run parallel to the axial and abaxial borders. Flo (1983) considers this injury to be the most typical meniscal injury seen in the dog. The "bucket-handle tear" is a subset of this group and is seen when the inner concave portion of the meniscus becomes lax and displaced from the tibial surface giving the appearance of a "bucket-handle".

Atrophied, Proliferative, and Discoid menisci

These injuries are rarely seen in the dog (Flo and others 1983, Bennett and May 1991¹).

SECTION 1: PART 4

LIGAMENT HEALING AND TREATMENT OF LIGAMENT INJURIES

THE REPARATIVE PROCESS

Under optimum conditions the healing of ligaments follows a general pattern common to virtually all connective tissues (Miltner and others 1937). The reparative process has been divided into three phases; the substrate phase, the repair phase, and the maturation phase.

The Substrate Phase

Injury to a ligament, or to the vascular part of the meniscus, is followed by an inflammatory reaction which removes foreign and devitalised material and initiates the mechanism of repair. A haemorrhagic clot forms uniting the damaged edges of the ligament and, about six hours after injury, natural debridement commences. Initially neutrophils, and later monocytes, begin to migrate into the wound and remove and break down cellular debris, bacteria, and other foreign material. The inflammatory response that occurs following injury is directly related to the severity of the trauma and to the extent of injury to the normal tissue. This dictates whether a healthy inflammatory reaction can occur, and whether local tissues can effectively drain away accumulating fluid.

The Repair Phase

Repair processes commence almost immediately following injury and proceed concurrently with the inflammatory process. In uncomplicated wounds debridement is completed by the third to fifth day, at which time fibroblast proliferation and capillary infiltration can commence.

Fibroblasts arise from undifferentiated mesenchymal cells in nearby connective tissues and secrete the ground substances and collagen which form scar tissue. Initially it is the protein-polysaccharides and glycoproteins of the ground substance that is produced. At about the fourth or fifth day, collagen synthesis commences and continues at a very rapid rate and at the same time new capillaries originate as budlike structures from nearby blood vessels and infiltrate the healing area. This network of capillaries provides large quantities of oxygen for the cells which are actively synthesising protein. The fibroblastic phase of repair lasts two to four weeks, depending on the nature of the injury, during which time the capillaries regress and the number of synthesising fibroblasts diminishes.

The Maturation Phase

In a healing wound there is normally an over production of collagen fibres and a single scar is formed in the wounded ligament or meniscus and in surrounding structures. This is termed the "one wound" concept. The maturation phase of healing is aimed at reorganising the collagen fibres in the scar so that purposefully orientated collagen fibres increase and non purposefully orientated fibres are removed. This maturation and control is a result of a delicate balance between collagen production and degradation.

Once the collagen content of a wound has stabilised, the strength continues to increase as a result of cross-linking and reorientation of collagen fibres as some disorganisation of the collagen fibres persists after the maturation phase. There is an almost imperceptible gain in strength for at least two years after ligament healing; however, the strength of the scar never reaches that of normal ligament (Johnson 1985).

FACTORS INFLUENCING LIGAMENT HEALING

The orderly ligament healing process of necrosis and cellular infiltration, fibroblastic invasion and finally collagenisation described above is influenced dramatically by the individual ligaments that are affected, the nature of the injury, and the type and timing of the treatment given (O'Donoghue and others 1961).

Anatomy of the ligament

The reparative processes which occur following injury to the collateral ligaments differ from those of the cruciate ligaments. This is due to the different locations of the ligaments and the relationship to their blood supply.

The collateral ligaments are intimately related to the surrounding tissues; a relationship that makes possible a rich blood supply and the formation of scar tissue when the ligament is injured. In contrast, the cruciate ligaments extend across the joint as isolated bands, and are covered by a fold of synovial membrane from which they receive their blood supply. Following cruciate ligament injury and tearing of the synovial membrane, haematoma formation cannot occur between the ligament ends as it is washed away by the synovial fluid. It is therefore impossible for repair to take place by organisation of a haematoma (O'Donoghue and others 1966, Arnoczky and others 1979³). The damaged cruciate ligament may disappear within a few weeks of injury due to disruption in its blood supply and the absorptive action of the synovial fluid (O'Donoghue and others 1966).

Severity of injury

In first-degree sprains, where there is minimal tearing and disruption of the ligament fibres, there is microscopic evidence of complete healing in six weeks. In severe sprains healing takes much longer and the process of repair persists for at least ten weeks (Miltner and others 1937).

Surgical Repair

Apposition of the ligament ends

Surgically apposing the ligament ends provides the most favourable conditions for ligament healing. This has been shown to minimise scarring, accelerate repair, hasten collagen formation, and produce the most normal-appearing healed ligament (O'Donoghue and others 1961). Where the ends of the ruptured ligament are not in close approximation, then the aforementioned orderly process of ligament healing does not occur and healing is by diffuse and excessive scar formation (Jack 1950).

Successfully repaired cruciate and collateral ligaments of the canine stifle attain a normal histological appearance at about six to ten weeks (O'Donoghue and others 1961, O'Donoghue and others 1966).

Studies on the tensile strength of sutured stifle collateral ligaments showed that sutured ligaments were stronger than the unsutured controls at all stages of healing. After six weeks of healing, the suture line was no longer the weakest point of the tested ligament. In the unsutured controls, the area of healing remained the weakest point at nine weeks when the experiment was terminated (Clayton and Weir 1959).

Tension on the wound

When excessive tension is placed on a healing ligament, the ligament ends are distracted and the deficit is filled with irregular scar tissue devoid of orderly arrangement (Jack 1950). The result is an elongated ligament which is structurally weakened and functionally impaired.

Excessive suture tension sufficient to accordion-pleat the ligament will cause necrosis of the ligament end and failure of the repair (O'Donoghue and others 1966).

Timing of surgical repair

Surgical apposition of ligament ends without undue tension is easiest immediately after ligament rupture because of the inelastic nature of the tissue. After seven to ten days the ligament ends have retracted and the tissue is oedematous and friable; it is no longer possible to accurately appose the ends and achieve suture security (Jack 1950).

Exercise and Immobilisation

Immobilisation directly influences the healing potential of damaged ligaments. The ligaments of the stifle joint in the dog have been shown to heal more effectively and with greater mechanical strength when the stifle joint is not immobilised postoperatively (Piper and Whiteside 1980). Following repair of the medial collateral ligament, the collagen concentration and collagen fibre bundle diameter was significantly higher in exercised dogs, compared to non-exercised dogs and those which were immobilised by a cast (Tipton and others 1970).

Mason and Allen (1941) demonstrated that function and motion in the first two weeks of tendon healing lead to increased reaction and separation of the suture line. However, restricted use of the tendon from the fourteenth day and which continued for two weeks, lead to only slight increases in reaction and rapid increases in tensile strength. Active unguarded use of the tendon, even after three weeks, always lead to increased reaction and stretching of the suture line.

It has been shown that when dogs are immobilised or confined for six weeks or more, the strength of the attachment of ligament to bone is diminished (O'Donoghue and others 1961). This weakening is due to subperiosteal resorption which occurs with the disuse of caging and immobilisation (Laros and others 1971). Work with primates showed that after eight weeks of immobilisation, followed by five months of reconditioning, bone strength had returned to near normal levels. However, ligament strength required one year of reconditioning to reach pre-immobilisation levels (Noyes 1977).

TREATMENT OF LIGAMENT INJURIES

The ultimate goal in the treatment of ligamentous injuries is to eliminate joint instability so that functional use of the joint may return. There are a number of methods available to achieve this goal and these include: conservative treatment by joint immobilisation, primary repair of the damaged ligament, and replacement of the ligament.

NON-SURGICAL TREATMENT

Isolated injuries of intra-articular ligaments have been treated conservatively. Good success has been achieved by the non-operative management of cranial cruciate ligament ruptures in small dogs (<15 kg) (Vasseur 1984), and following caudal cruciate ligament transection (Harari and others 1987). Success following non-operative management has been attributed to the buttressing of the unstable joint by capsular fibrosis and osteophytosis.

The conservative treatment of extra-articular ligament injuries is generally reserved for isolated mild sprains of the collateral ligaments with minimal disruption of the collagen fibres. Injuries of this type require external coaptation in the form of a splint or cast for at least two to three weeks followed by two weeks in a firm Robert-Jones bandage (Brinker and others 1990). The joint should be stressed toward the side of the injured collateral ligament to afford the most protection to the healing ligament (Egger 1983).

More extensive damage and obvious joint instability implies that the ligament has either been stretched or has ruptured completely. Healing in this position will result in disorganised scar tissue formation together with a ligament which is functionally impaired and biomechanically weak (Jack 1950). Excellent results have been achieved in man by treating complete ruptures of the collateral ligaments of the stifle with external coaptation alone (Shelbourne and Patel 1995). This success has been attributed to the fact that the ligament ends have spontaneously become closely approximated together and it is thought it is this approximation, and not the stability afforded by sutures, that is the important factor (O'Donoghue and others 1961). It has been demonstrated by Jack (1950), however, that at the time of rupture, the torn ends of the ligament momentarily retract and direct contact between the ends is often not restored because they become enveloped by a loose areolar sheath.

Rigid external coaptation is required for a minimum of six to eight weeks to allow for complete healing (James 1980). O'Donoghue and others (1961) found it to be practically impossible to obtain rigid external fixation of the canine stifle joint without the use of small Steinmann pins placed transversely through the distal femur and proximal tibia. A well moulded cast which incorporated these pins was applied to the limb extending as high as possible on the thigh and distally to the foot.

Poor results following conservative treatment of severe collateral ligament injuries of the stifle joint may, therefore, be attributed to poor apposition of the ligament ends and lack of rigid external coaptation. Farrow and Newton (1985) reported only 25% success in conservatively treated collateral ligament injuries in dogs.

SURGICAL TREATMENT

The method of surgical repair of a ligament will depend to a large extent on the particular ligament involved and the nature of the injury. The collateral ligaments of the stifle, for example, are more amenable to primary repair because of their location compared to the intra-articular cruciate ligaments which are usually replaced.

Interstitial Failure

Primary Repair

If the ligament injury is relatively fresh without fixed retraction of the ends, then primary end-to-end apposition is the repair of choice (O'Donoghue and others 1961). Primary repair of a mid substance ligament rupture, or imbrication of a stretched ligament, may be achieved by a number of suture patterns. The simple horizontal mattress suture pattern may be used alone or, more commonly, in conjunction with other suture patterns to appose the ligament ends. A Bunnell-Mayer pattern is often used in tendon repair because of its high mechanical strength (Ketchum and others 1977). Ligaments are often too short to allow the use of this pattern, however. In addition, it has lost favour because of reported interruption of intrinsic blood flow in tendons (Stein and others 1985). The locking-loop, or modified Kessler pattern, is especially suited for the repair of small or flat ligaments, as a minimum amount of suture material is required for ligament holding with this pattern (Aron 1981). When flexible, smooth suture material is used; this configuration allows the loops to tighten on the ligament fibres. If the ligament suture is placed so that the suture patterns in the proximal and distal stumps are mirror images of each other, the ligament ends will be well apposed with minimal buckling (Figure 21, page 39).

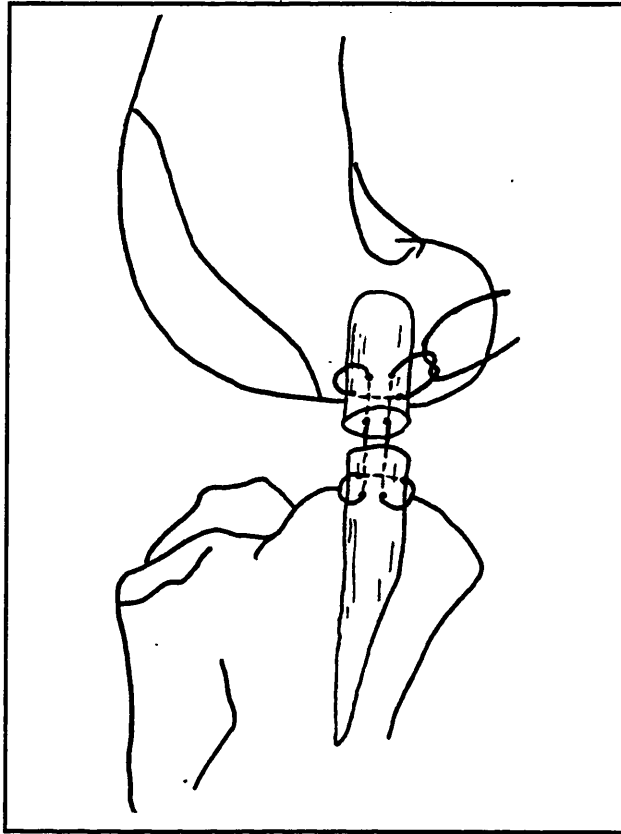


Figure 21 Diagram illustrating the locking-loop suture pattern.

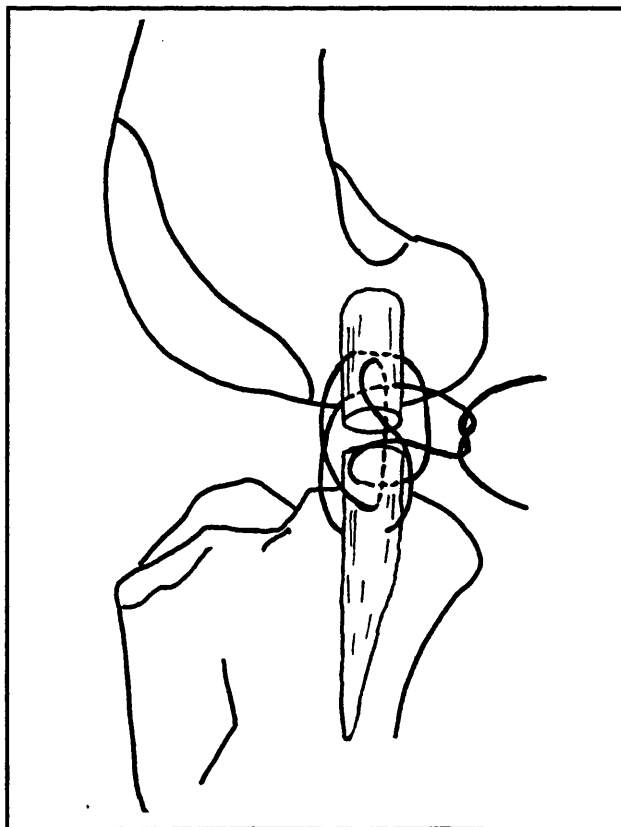


Figure 22 Diagram illustrating the three-loop pulley suture pattern.

The three-loop pulley pattern is composed of three continuous horizontal mattress sutures (loops) positioned in separate planes approximately 120 degrees apart (Figure 22, page 39). When a sutured ligament is placed under tension, the suture material glides within the ligament in a pulley fashion so that equal tensile load is supported by each of the three loops. The three loop pulley had greater tensile strength and less distraction between the suture ends when compared in vitro to the locking-loop pattern (Berg and Egger 1986).

Failure of both the three-loop pulley pattern and the locking-loop pattern depends on the size of the structure being sutured and the suture material (Berg and Egger 1986). With smaller structures, such as the collateral ligaments in the dog, both patterns fail most commonly because the suture pulls free from the tissue.

Regardless of the suture pattern used, a monofilament suture material is recommended because of its ability to glide within the tissues. The size and type of suture material must provide adequate tensile strength so that the sutured ends do not separate for the duration of the healing period. Non-absorbable suture materials such as: monofilament nylon, polypropylene, orthopaedic wire, and absorbable materials such as polydioxanone have been recommended (Rudy 1974, Crane 1983).

Repair Augmentation

Primary repair of the stifle collateral ligaments may be protected from excessive tension by placing screws at the origin and insertion points of the ligament and connecting the screws with a figure-8 suture (Rudy 1974). A heavy suture of orthopaedic wire, carbon fibre, or a non-absorbable synthetic suture of nylon, polypropylene, or polyester is recommended (Denny 1980, Hulse and Shires 1985, Farrow and Newton 1985, Robins 1990). The augmentation provides an internal splint while healing of the ligament occurs. This method may also be used to provide a replacement ligament in severely traumatised ligaments where primary repair is not possible. Fibrous tissue envelopes the suture and ligament remnants and can eventually provide a functional substitute for the original ligament as it reorganises in response to tension stress (Brinker and others 1990).

Ligament-Bone Failure

Where the ligament is torn from its origin or insertion it may be reattached to the bone by suturing or with a screw and spiked washer. In the suture technique, the torn ligament end is sutured with a Bunnell-Mayer, or modified Kessler suture pattern, and the suture is anchored to either a screw (Brinker and others 1990) or through tunnels drilled in the bone (Hulse and Shires 1985). The other technique requires a screw and a spiked washer to trap the ligament end against the bone (Parker and Schubert 1981).

Ligament-Bone Avulsion

With this type of failure, the ligament end has an associated piece of cancellous bone. Stability is achieved by implant fixation of the avulsed bone to the parent bone and fixation by multiple crossing K-wires, K-wires and a tension band wire, a lag screw, or a screw and spiked washer (Brinker and others 1990).

Ligament Replacement

A wide variety of tissues and materials have been used for ligamentous reconstruction; however, the ligament which has received the most attention in the veterinary literature is the cranial cruciate ligament. Various cranial cruciate ligament prostheses and collagenous tissues have been used in isolation and together, both intra-capsularly and extra-capsularly, in an attempt to provide joint stability and reduce functional disability. Although the clinical outcome of these different techniques is reported, objective clinical and biomechanical data is lacking, and no one technique or group of techniques has been proved to be superior to the others (Kirby 1993).

Autogenous Tissues

A pedicle graft of autogenous fascia lata has been recommended as a replacement for the cranial cruciate ligament (Paatsama 1952). Numerous modifications of the original Paatsama technique have been published for the treatment of the cranial cruciate ligament rupture in the dog (Dickinson and Nunamaker 1977, Rudy 1974, Arnoczky and others 1979², Kennedy and others 1980, Hulse and others 1980, Shires and others 1984, Bennett and May 1991²). In addition, other tissues such as: autogenous skin (Vaughan and Bowden 1964), the long digital extensor tendon (Roush and others 1970), the tendon of the peroneus longus muscle (Rathore 1959), and bone-ligament-bone grafts using patella ligament (Dueland 1966) and lateral collateral ligament (Smith and Torg 1985) have been used to replace the cranial cruciate ligament.

The caudal cruciate ligament has been reconstructed using the long digital extensor tendon (Egger 1983), the medial collateral ligament (Egger 1983), the popliteus tendon (Hulse and Shires 1985), and the medial meniscus (Robins 1990). Surgical repair of a ruptured caudal cruciate ligament is considered unnecessary by some authors (Pearson 1971, Harari and others 1987).

The collateral ligaments have been reconstructed using a pedicle graft of autogenous fascia lata (Rudy 1974) and the tendon of the peroneus longus muscle (Rathore and Rathore 1976). Fresh frozen allogenic tendons have also been used for extra-articular ligament reconstruction (Horibe and others 1990).

Autogenous tissues which are transferred to provide intra and extra-articular ligament reconstruction do not all function in a useful manner. Many grafts elongate or fail under low forces, perhaps remaining as weak collagenous tissues, but providing no ligamentous restraint (Kennedy and others 1980). Factors governing the success of a biological graft are: its initial mechanical strength,

weakening due to tissue necrosis after implantation, improper positioning, and postoperative protection of the graft (Noyes and others 1984).

Synthetic Materials

Strande (1967) has stated that: "The ideal synthetic material suitable for ligament replacement has yet to be found. Ideally it should possess great tensile strength, a little elasticity and should tolerate wear and tear in the joint for several months without causing irritation".

Braided or monofilament nylon (Vaughan 1963, Pond and Campbell 1972), Terylene (Singleton 1969, Stead and others 1991), Teflon (Butler 1964), Dacron (Hinko 1981, Arnoczky and others 1986), Gore-Tex (Petersen and Taylor 1995), and composites of Teflon, Dacron and silastic (Gupta and Brinker 1969, Leighton and Brightman 1976) have all been used to replace the cranial cruciate ligament. Varying degrees of success, biocompatibility, and similarity to the biomechanical properties of the normal cruciate ligament have been claimed. Unfortunately, many grafts fray and rupture after a few months of weight-bearing.

Carbon fibre has been used in cranial cruciate ligament (Denny and Goodship 1980) and collateral ligament reconstruction (Robins 1990). The carbon fibres have considerable inherent tensile strength. They also induce correctly aligned collagenous tissue formation by acting as a biocompatible scaffold for the growth of fibroblasts and subsequent deposition of collagen. In this way they provide a long term biological replacement ligament (Vaughan and Edwards 1978). Some workers have experienced disappointing results when using this material intra-articularly (Robins 1990). Also problems with sepsis and difficulty in removing the fragmented fibres have been encountered (Vaughan 1981).

Marlex mesh has been successfully used for the total replacement of the stifle collateral ligaments in the dog (Winston and others 1978).

SECTION 1: PART 5

MENISCAL HEALING AND TREATMENT OF MENISCAL INJURIES

MENISCAL HEALING AND BLOOD SUPPLY

Experimental studies in dogs have demonstrated that the peripheral meniscal blood supply is capable of producing a reparative response similar to that produced in other connective tissues (Cabaud and others 1981). Lesions within the vascular portion of the meniscus are completely healed by production of a fibrovascular scar in ten weeks (Arnoczky and Warren 1983). However, modulation of this scar tissue into normal appearing fibrocartilage takes several months and the strength of this repair tissue has not been delineated (DeHaven and Arnoczky 1994).

Vascular studies in the dog have shown that the body of the meniscus has a very limited blood supply. Vessels arise from the synovia and penetrate only the outer 25% of each meniscus (Arnoczky and Warren 1983). Injuries to the inner avascular part of the menisci are incapable of healing (King 1936).

In man, meniscal lesions are often classified by the location of the tear relative to the blood supply of the meniscus and by the vascular appearance of the peripheral and central surfaces of the tear (DeHaven and Arnoczky 1994). The so called red-red tears (peripheral capsular detachment) have a functional blood supply on the capsular and meniscal sides of the lesion and have the best prognosis for healing. The red-white tears (in the meniscal rim through the peripheral vascular zone) have an active peripheral blood supply while the inner surface of the lesion is devoid of functioning vessels. These lesions should have sufficient vascularity to heal by the aforementioned process. The white-white tear (completely in the avascular zone) is without a blood supply and theoretically cannot heal.

HEALING ENHANCEMENT TECHNIQUES

Healing enhancement techniques have developed in an effort to extend the level of repair into the avascular zone of the meniscus. These techniques include: vascular access channels (Arnoczky and Warren 1983), synovial abrasion (Henning and others 1987), and the use of exogenous fibrin clots (Arnoczky and others 1988).

Experimental studies have shown that connection of a lesion in the avascular portion of the meniscus to the peripheral blood supply via a vascular access channel can stimulate healing of the lesion and regeneration of the meniscus following partial meniscectomy (Hulse and others 1986). Similarly, abrading the synovial fringe on the tibial and femoral surfaces of the meniscus produces a vascular pannus that

migrates into the lesion and supports a reparative response (Henning and others 1987). Exogenous fibrin clots when placed in a stable lesion can support a reparative response by providing potent chemotactic and mitogenic stimuli, as well as a scaffold on which the cellular response is supported (Arnoczky and others 1988).

TREATMENT OF MENISCAL INJURIES

Primary repair

The ability of meniscal lesions to heal has led to the rationale for the primary repair of peripheral meniscal injuries. Several reports in man have demonstrated excellent results following primary repair of peripheral detachments and longitudinal tears in the vascular zone of the meniscus (Hamberg and others 1983, Warren 1990). Tears that are considered not suitable for repair include those involving moderate or severe damage to the meniscus, complete radial tears, and tears within the avascular zone (DeHaven and Arnoczky 1994).

In man almost all meniscal tears, except the very peripheral, are repaired closed using arthroscopic techniques. Three techniques have evolved: the inside-to-outside technique (Barber and Stone 1985), the outside-to-inside technique (Morgan and Casscells 1986), and the all-inside technique (Morgan 1991). Peripheral tears in man have been sutured by open technique, using a small (1.5 metric) absorbable suture such as polyglactan in a vertical pattern tied inside the capsule (DeHaven and Arnoczky 1994), or a heavier (3 metric) suture such as monofilament nylon or polydioxanone tied outside the capsule (Hamberg and others 1983).

Aftercare following meniscal repair in man consists of six-weeks of maximum protection in a cast, to allow initial healing, and a subsequent six-month interval of protection from vigorous stresses, to allow for maturation of the healing collagen tissue (DeHaven and Arnoczky 1994).

Total meniscectomy and partial meniscectomy

Many meniscal lesions are not amenable to primary surgical repair and removal of the damaged portion (partial meniscectomy), or the whole of the damaged meniscus (total meniscectomy), must be considered. Meniscectomy itself, whether partial or total, is not a benign procedure and results in increased stress acting across the joint which predisposes to osteoarthritis (Kraus and others 1976). However, it has been shown that conservatively treating meniscal lesions can result in more degenerative changes than total or partial meniscectomy (Cox and Cordell 1977, Shapiro and Gilncher 1980).

The choice of partial or total meniscectomy in the treatment of meniscal lesions remains controversial. Total meniscectomy relies on the regeneration of meniscus-like tissue to fill the incongruity between the femoral and tibial condyles. In the dog meniscal regeneration has occurred by seven months after meniscectomy; at this

time it has a similar histological appearance to normal meniscal tissue (DeYoung and others 1980). Meniscal regeneration has been found to be variable and unpredictable (Elmer and others 1977). Further long term biomechanical studies are required to determine its functional capacity (DeYoung and others 1980).

Partial meniscectomy preserves the peripheral rim of the meniscus which helps maintain joint stability and protects the underlying articular cartilage from normal physiological loads (Rodin and others 1984); it relies on inspection of the whole of the meniscus and removal of the injured portion. However, there are often multiple tears in the meniscus that remain undetected until the entire meniscus is removed (Flo 1993). After removal of the damaged portion of the meniscus, the cut edge frequently does not communicate with the vascular supply and, therefore, has no regenerative capacity. The articular surface area may be reduced, resulting in increased force per unit area in the central zone of articulation and wearing of the cartilage surface (Cox and others 1975). Hulse and others (1986) demonstrated that vascular access channelling stimulated regeneration of the axial border following partial meniscectomy.

SECTION 2

**MULTIPLE LIGAMENTOUS
INJURIES OF THE CANINE STIFLE
JOINT**

SECTION 2: PART 1

MULTIPLE LIGAMENTOUS INJURIES OF THE CANINE STIFLE JOINT

INCIDENCE AND PREVALENCE

Injury to the ligaments of the stifle joint is a common occurrence in the dog (Rudy 1974). The most frequent injury reported in the literature is an isolated interstitial tear of the cranial cruciate ligament, while isolated caudal cruciate ligament and isolated medial or lateral collateral ligament injuries are rarely reported (Arnoczky 1980, Denny and Minter 1973, Reinke 1982). Similarly, the lack of reports in the literature call attention to the rarity of the spontaneous rupture of two or more ligaments in the stifle joint of the dog (Denny and Minter 1973, Hulse and Shires 1986), the cat (Toombs and Wallace 1979), and in man (Kennedy 1963).

Aron (1988) reported 12 cases of stifle dislocation in the dog in a series of 108 stifles treated for ligament instability (11% prevalence) over a five-and-a-half-year period (annual incidence = 2.2 cases). Denny and Minter (1973) reported five cases of stifle dislocation in the dog in a series of 167 stifles (3% prevalence) treated for ligament rupture over a four-year period (annual incidence = 1.3 cases).

Other reports in the literature also consist of only small numbers of cases. Welches and Scavelli (1990) diagnosed five dogs with stifle dislocation over an eight-and-a-half-year period (annual incidence = 0.6 cases). Hulse and Shires (1986) reported four cases of multiple ligament injury in the dog diagnosed over a one-and-a-half year period (annual incidence = 2.7 cases). Phillips (1982) diagnosed one dog with stifle dislocation over a five-year period (annual incidence = 0.2).

SIGNALMENT

The mean age at surgery of dogs treated for multiple ligamentous injuries to the stifle joint was: 3 years (Parker and Schubert 1981), 4 years (Panco 1985), 4.8 years (Hulse and Shires 1986), 4.9 years (Aron 1988), 5 years (Denny and Minter 1973), and 8 years (Welches and Scavelli 1990). In all cases, it appears that only skeletally mature dogs are affected. This feature was also noted by Phillips (1982) in the cat. In the immature animal, trauma to the stifle joint often results in Salter type I and II fractures through the proximal tibial metaphyseal growth plates (Nunamaker 1985).

Where sex has been recorded in the literature, the ratio of the total number of males:females affected is: 7:4 (Aron 1988), 3:2 (Denny and Minter 1973), 2:2 (Hulse and Shires 1986), 2:1 (Welches and Scavelli 1990). There have been two single case reports affecting male dogs (Parker and Schubert 1981, Panco 1985). The high incidence of multiple ligamentous injuries to the stifle joint in male dogs can be explained by the work of Kolata and others (1974) who reported that injured males of either species (65.4% of dogs and 69% of cats) were significantly more frequent than were injured females. This was largely because males were more commonly admitted following road traffic accidents, animal interaction, and injury of unknown cause. Their findings supported the opinion that males are more aggressive and tend to wander more than females.

A wide variety of breeds are reported in the literature but the numbers are too small to make any meaningful conclusions. However, a general impression would be that mainly medium to large breed dogs are affected. The mean weight of the four cases reported by Hulse and Shires (1986) was 27 kg. It is interesting to note that six out of the 12 dogs (50%) reported by Aron (1988) were Labrador retrievers. Nevertheless, smaller breeds of dogs are also affected. The mean weight of the three dogs reported by Welches and Scavelli (1990) was 9.5 kg.

AETIOLOGY

Multiple ligamentous injuries of the stifle result from large extrinsic bending and torsional forces imparted directly to the stifle joint (Smith 1995). High-energy trauma such as road traffic accidents, or falls from heights are required to produce multiple ligament failure and dislocation of the joint (Hulse and Shires 1986). These types of injuries can also occur when the leg becomes caught while the animal is jumping a fence or gate (Robins 1990). Severe forces are required to overcome the multiple ligaments, menisci, tendons and muscles which interact to provide joint stability (Aron 1988). When a joint is directly or indirectly loaded to a lesser degree, then the cranial cruciate ligament only is predisposed to rupture (Arnoczky 1980).

In all of the cases reported by Denny and Minter (1973), Parker and Schubert (1981), and Panco (1985), the injury resulted from automobile trauma. Ten of the cases reported by Aron (1988) were involved in road traffic accidents. In three cases the aetiology was unknown; however, the owners thought the animals probably had been hit by a car. One of the dogs reported by Hulse and Shires (1986) had injured itself after a fall; the others were involved in road traffic accidents.

CLASSIFICATION

Multiple ligamentous injuries can occur in a variety of combinations and can result in varying degrees of gross joint instability with the articular surfaces becoming displaced from their normal positions. If there is some contact between articular surfaces, the joint is said to be subluxated or incompletely dislocated. If there is no contact between the articular surfaces, the joint is said to be luxated or completely dislocated (Pond 1971). The terms luxation and dislocation are, therefore, synonymous.

Stifle dislocations are described in terms of tibial displacement with respect to other structures about the stifle (Kennedy 1963). For example, a tibia lying medial to the femur is a medial dislocation. Using this terminology, there may be five main types of dislocation (cranial, caudal, lateral, medial, and rotatory) as well as combinations of these. Stifle dislocations may be further classified into acute or chronic, depending on the duration of the injury; or closed or open, depending on whether external contamination has occurred (Eaton-Wells and Whittick 1990).

Subluxation/Dislocation

Whether a stifle is subluxated or completely dislocated following injury will depend on the extent of damage to the stabilising structures of the stifle joint. Following multiple ligament failure the degree of subluxation present on clinical examination is a measure of the status of the secondary restraints (Noyes and others 1984). Complete dislocation is only possible if there is disruption of both the primary and secondary joint restraints

Of the 12 dislocated stifles reported by Aron (1988), six were lateral, three were cranial, two were craniolateral, and two were cranial and rotated. Denny and Minter (1973) reported two cranial, two craniolateral, and one medial stifle dislocations. In both of these series of cases, predominantly cranial and lateral dislocations occurred. Aron (1988) proposed that the quadriceps, hamstring, gastrocnemius, and popliteus muscles and tendons, resist caudal and medial displacement of the tibia when multiple stifle joint ligaments are damaged. No open or caudal dislocations have been reported in the literature.

Combinations of Ligament Injuries

At the time of writing, there were a total of 26 cases of multiple ligamentous stifle injury in the dog reported in the veterinary literature. Eight different combinations of damaged ligaments were recorded. The most frequent combination, which occurred 14 times (53.9%), was both cruciate ligaments and the medial collateral ligament. Other combinations were both cruciate ligaments, three times (11.5%); both cruciates and the lateral collateral ligament, one time (3.8%); both cruciate ligaments and both collateral ligaments, three times (11.5%); both collateral ligaments, one time (3.8%); cranial cruciate and medial collateral ligaments, two times (7.7%); cranial cruciate and lateral collateral ligaments, one time (3.8%); and the caudal cruciate and medial collateral ligaments, one time (3.8%) (Aron 1988,

Denny and Minter 1973, Hulse and Shires 1986, Panco 1985, Parker and Schubert 1981, Welches and Scavelli 1990).

Hulse and Shires (1986) speculated that tissues on the medial side of the stifle joint are more commonly injured because the point of impact of trauma (especially automobile trauma) on the lateral aspect generates tensile forces medially, causing failure of the medial restraints.

OTHER ASSOCIATED INJURIES

Isolated ligament injury is rare because the large joint displacements required to produce ligament failure usually result in concomitant injury to other structures (Noyes and others 1984). With severe trauma it is common to damage the joint capsule, menisci and articular cartilage. Injury to the patellar tendon, long digital extensor tendon, and popliteal tendon has also been reported (Hulse and Shires 1986, Denny and Minter 1973, Robins 1990).

Although associated fractures involving the stifle joint are not common, other musculoskeletal injuries are often reported. Avulsion fractures at ligament origins or insertions were reported in 7.7% (2 of 26) of the published cases of multiple ligamentous stifle injury in the dog (Panco 1985, Parker and Schubert 1981). There have been no reports of articular fractures affecting the dislocated stifle. However, fractures and luxations away from the stifle were reported in 38.5% (10 of 26) of the published cases of multiple ligamentous stifle injury in the dog. These included one contralateral and three ipsilateral coxofemoral luxations (Aron 1988, Welches and Scavelli 1990), two pelvic fractures (Aron 1988, Hulse and Shires 1986), two ipsilateral femoral fractures (Aron 1988, Hulse and Shires 1986), one ipsilateral tibial fracture (Hulse and Shires 1986) and one spinal fracture (Parker and Schubert 1981).

Vascular and nerve injuries were reported as complications in 32% (7 of 22) of traumatic dislocations of the knee joint in man (Kennedy 1963). Hyperextension injuries, resulting in anterior dislocation of the knee, exert tremendous traction throughout a large segment of the popliteal artery, and the lumen may be occluded or the artery may rupture completely. Sudden obliteration of the popliteal artery at this level is not compatible with survival of the leg distal to the site of occlusion because of the poor collateral circulation in this region (Kennedy 1963). The mechanism of nerve injury accompanying dislocation of the knee in man remains obscure. In cadaver experiments, it was noted that in anterior dislocation the common peroneal nerve frequently was caught behind the lateral femoral condyle as the knee went into hyperextension. As a result the nerve was severely stretched over this bone prominence (Kennedy 1963). There have been no reports of vascular or nerve injuries following dislocation of the stifle joint in the dog.

SECTION 2: PART 2

THE CLINICAL AND DIAGNOSTIC FEATURES OF MULTIPLE LIGAMENTOUS INJURIES OF THE CANINE STIFLE JOINT

GENERAL CLINICAL FINDINGS

It has already been stated that dogs with multiple ligamentous stifle injuries have been severely traumatised, often as a result of road traffic accidents. These dogs are often admitted as an emergency; they may be in a state of shock and have signs referable to other soft tissue or orthopaedic injuries. It is important therefore, to conduct a full clinical examination so that all injuries including those distant to the stifle joint, are not overlooked (Laing 1993).

Multiple ligamentous injury to the stifle usually results in a non-weight bearing lameness. In the acute injury, clinical signs may vary from obvious stifle dislocation with malalignment of the joint and gross instability, to more subtle signs of stifle joint effusion, soft tissue swelling and increased joint laxity. Manipulation of the stifle is painful and there may be associated crepitation and reduced range of motion. Crepitation, or a "popping" sound during flexion and extension, indicates meniscal injury or femorotibial joint subluxation (Hulse and Shires 1985). In some cases the skin may be bruised with localised oedema over the damaged collateral ligament. Soft tissue swelling may be extensive and extend distally to the hock.

In chronic cases, pain, swelling and lameness may not be as obvious. Instead, a diffuse fibrous thickening of the joint capsule may be detected adjacent to the injured ligament, similar to that seen with chronic cruciate ligament instability (Laing 1993).

ASSESSMENT OF STIFLE JOINT STABILITY

Physical examination is the most important tool in the diagnosis of collateral and cruciate ligament injury (Arnoczky and others 1980).

Full assessment of stifle stability must be made under heavy sedation or general anaesthesia since the patient will be in pain and, therefore, reluctant to allow joint manipulation. Soft tissue swelling and oedema may also hinder joint manipulation and accurate evaluation of joint instability in the unsedated patient (Hulse and Shires 1986).

The stifle joint should be examined through its full range of motion and any changes noted in flexion, extension, angulation, or rotation. Palpation of the contralateral stifle joint is often useful for comparison.

Craniocaudal Stability

The standard test to determine cranial subluxation of the tibia is the cranial drawer test. Positive cranial displacement (cranial drawer sign), without a firm end point, is an indication of cranial cruciate ligament rupture (Rudy 1974). The amount of displacement varies according to the degree of muscle tension, position of the joint, the length of time since the injury, the degree of ligament rupture, and damage to concomitant ligaments and secondary joint restraints (Arnoczky and Marshall 1981). In isolated complete tears, cranial displacement of the tibia is greatest in flexion as the tight collateral ligaments and other secondary joint restraints limit cranial displacement in extension (Vasseur and Arnoczky 1981).

Injury to the caudal cruciate ligament results in caudal displacement of the tibia with respect to the femur. The action of the caudal thigh muscles maintain the tibia in this caudally subluxated position and stressing the joint by palpation, forces the joint cranially to its normal position. This movement resembles the "cranial drawer sign" and can lead to misdiagnosis of the condition (Egger 1983).

Egger (1983) stated that cranial and caudal cruciate ligament ruptures may be differentiated by the degree of tibial displacement in flexion and extension. The disrupted cranial cruciate ligament reveals a positive cranial drawer in both flexion and extension. The disrupted caudal cruciate ligament, on the other hand, shows drawer motion in flexion but not extension. However, studies involving the selective severance of the cruciate ligaments in cadavers did not demonstrate significant differences in craniocaudal translation between the two ligaments in flexion and extension (Arnoczky and Marshall 1977). These injuries may be differentiated by comparing both stifles with the patient in dorsal recumbency and the femurs positioned at right angles to the table with the tibias parallel to the table. The injured leg may reflect a "caudal sag", indicating caudal cruciate ligament rupture (Hulse and Shires 1985).

Rupture of both the cranial and caudal cruciate ligaments results in marked craniocaudal instability (Arnoczky and Marshall 1977). In the absence of concurrent collateral ligament damage, craniocaudal translation is greatest in flexion.

The specific preoperative identification of isolated cruciate ligament rupture from concurrent cranial and caudal cruciate ligament rupture is difficult (Hulse and Shires 1986). In a series of caudal cruciate ligament ruptures reported by Johnson and Olmstead (1987), 50% (7 of 14) were misdiagnosed as cranial cruciate ligament ruptures. These misdiagnoses were attributed to: lack of suspicion of the lesion, inability to determine the neutral position of the stifle, failure to compare instability in flexion and extension, and failure to observe the presence of a "caudal sag" of the tibial tuberosity (Egger 1983, Johnson and Olmstead 1987).

Angular Stability

When testing for a medial or lateral restraint injury, the examiner must extend the stifle joint and place the tibia in neutral position. With the limb held in this position, a valgus force is applied to the distal limb to test for a medial restraint injury, and conversely a varus force is applied to the distal limb to test for a lateral restraint injury. When marked varus or valgus angulation is present, damage to both the primary restraints (collateral ligaments), as well as one or more of the secondary restraints (joint capsule, meniscal attachments, cruciate ligaments) is also present. Vasseur and Arnoczky (1981) showed that severing either collateral ligament resulted in only slight varus or valgus angulation which increased significantly when the cruciate ligaments were severed.

Difficulties have been encountered in accurately determining damage to the collateral ligaments preoperatively (Aron 1988). Some of these clinical paradoxes can be explained by the work of Noyes and others (1980) who demonstrated that despite rupture of the primary restraints in the knees of man, the secondary restraints, although small, may block the clinical detection of laxity because it is performed with small manual forces.

Rotational Stability

An increase in internal rotation of the tibia is detected with cranial and caudal cruciate ligament rupture. This is most evident when the joint is in flexion as the lateral collateral ligament is loose in this position and has no limiting affect. Rupture of the cruciate ligaments has no effect on tibial external rotation (Arnoczky and Marshall 1977).

Rupture of the medial collateral ligament results in increased external rotation of the tibia in flexion and both collaterals limit external rotation in extension. Rupture of either collateral ligament also results in increased internal rotation of the tibia when the stifle joint is held in extension (Vasseur and Arnoczky 1981).

The changes in joint motion seen following injury to the femorotibial ligaments are summarised in Table1, page 54.

Table 1: INCREASE IN JOINT MOTION FOLLOWING SEVERENCE OF THE FEMOROTIBIAL LIGAMENTS IN DOGS

(Data from: Vasseur and Arnoczky 1981, Arnoczky and Marshall 1977)

	LCL Alone	LCL and CL	MCL Alone	MCL and CL	CL Alone
Varus Angulation					
flexion	+	++	0	0	0
extension	+	++	0	0	0
Valgus Angulation					
flexion	0	0	+	++	0
extension	0	0	+	++	0
Internal Rotation					
flexion	0	+++	0	+++	++
extension	++	++	++	++	0
External Rotation					
flexion	+	+	++	++	0
extension	++	++	+	+	0
Drawer Movement					
flexion	0	+++	0	+++	+++
extension	0	+++	0	+++	++

0 = no change; +/++/+++ = slight/moderate/marked increase; LCL = lateral collateral ligament; MCL = medial collateral ligament; CL = cruciate ligament.

Note. Severing the cranial or caudal cruciate ligament yields similar changes in joint angulation and rotation.

ANCILLARY DIAGNOSTIC TECHNIQUES

Radiography

Although radiographic evaluation of the stifle joint can provide valuable additional information to the findings of manipulation and palpation, it does not have a primary role in the diagnosis of joint instability (Park 1979). Several radiographs, including a craniocaudal view, a mediolateral view, and stressed views, are recommended to evaluate multiple ligamentous injury of the stifle joint prior to surgery (Laing 1993).

In the acute injury, radiographs can give an indication of abnormal relationship of bones, altered width of joint space, articular fractures, avulsion fractures, and distension of the joint capsule. Chronic cases may show additional changes such as: peri-articular osteophytes, peri-articular enthesiophytes, calcification of peri-articular or intra-articular soft tissues or cartilage, intra-articular joint mice and subchondral bone sclerosis.

Valgus and varus stressed views of the stifle can provide more information on lateral and medial restraint injuries (Park 1979). When a valgus force is applied to the distal limb, the joint space will widen medially if there is a medial restraint injury. Similarly a varus force will highlight a lateral restraint injury.

Surgery

Clinical and radiographic assessment of multiple ligamentous injuries of the stifle joint is sometimes confusing and requires surgical exploration for confirmation and identification of specific damage (Aron 1988).

SECTION 2: PART 3

MANAGEMENT OF MULTIPLE LIGAMENTOUS INJURIES OF THE CANINE STIFLE JOINT

NON-SURGICAL TREATMENT

The non-surgical treatment of multiple ligamentous injuries to the stifle joint of the dog generally yields poor functional results and is not recommended (Laing 1993, Smith 1995). Similarly, experimental studies in dogs have shown poor healing of the transected medial collateral ligament in stifle with a torn cranial cruciate ligament (Woo and others 1990). These findings conflict with the excellent results achieved in man by the non-operative treatment of an injury of the medial collateral ligament with an associated anterior cruciate ligament injury (Shelbourne and Patel 1995, Ballmer and others 1991). Conservative treatment with a cast for this type of injury has resulted in stable and predictable healing of the medial collateral ligament. Delayed surgical treatment of the anterior cruciate ligament is performed only in patients who have high demands with regard to activity (Shelbourne and Patel 1995).

Reduction of a dislocated stifle joint can usually be accomplished through closed manipulation and the joint may be supported prior to surgery using a lateral spica splint and/or a Schroeder-Thomas splint (Nunamaker 1985).

GENERAL PRINCIPLES FOR SURGICAL TREATMENT

Surgical treatment of multiple ligamentous injuries of the stifle joint is generally performed as soon as the patient is a safe anaesthetic risk, and excessive soft tissue oedema has subsided. Mean intervals from the time of injury to surgery reported in the literature were 6.5 days (Aron 1988) and 7.1 days (Welches and Scavelli 1990). The timing of surgical repair can influence the way in which ligaments heal and it is better to perform surgery within ten days of injury (Jack 1950). However, it has been demonstrated in man that acute operative repair of multiple ligamentous injuries of the knee results in an increased prevalence of arthrofibrosis (Shelbourne and Patel 1995). Delaying surgery for four to eight weeks after the injury, or until the inflammatory phase of healing has subsided and range of motion has returned, resulted in a marked decrease in the prevalence of postoperative stiffness of the knee.

Thorough visual inspection, and stress palpation of all ligaments around the stifle is recommended at the time of surgery to accurately identify all of the damaged ligaments (Aron 1988).

The menisci should be preserved whenever possible as they are important stabilisers in the ligament deficient stifle. Meniscectomy produces high stress levels during weight bearing at the area of cartilage contact and, in combination with ligamentous deficiencies, it necessitates more extensive surgical reconstruction to achieve adequate stifle stability (Smith 1995). Meniscal repair is easiest when the joint is most unstable and, therefore, should be performed prior to any ligament reconstruction.

Precise reduction and rigid fixation of articular fractures is required to minimise future degenerative joint disease (Roy and Dee 1994). Anatomical reattachment of avulsion fractures is essential to restore normal ligament function during all positional changes of the stifle (Reinke 1982).

Where possible, the torn ends of the medial and lateral collateral ligaments should be apposed with sutures before attempting a definitive intra, or extra-articular stifle stabilisation. Collateral ligaments should always be sutured with the stifle joint in extension to prevent iatrogenic shortening of the ligament, which either limits stifle extension or stresses the repair when the patient extends the joint (Brinker and others 1990).

The purpose of reconstruction is to re-establish a stable stifle with isometric motion throughout its range (Smith 1995). This does not imply that each torn ligament must be replaced individually but rather that sufficient fixation must be placed in or around the stifle joint to achieve stability and a functional range of motion. The fixation must provide stability for a period sufficient to allow ligamentous healing and peri-articular fibrosis which ultimately will maintain the stability of the joint over the long term.

The positioning of the definitive fixation is extremely important and requires a thorough understanding of the anatomy and function of the major structural components of the stifle joint. For example, in the case of screw and suture repair of the collateral ligaments, screw placement in the femur is critical and must always approximate the centre of rotation of the femoral condyles (Smith 1995). Malpositioning of the femoral screw will result in non-isometric (binding) motion of the joint causing limited range of motion and premature failure of fixation.

SURGICAL TECHNIQUES

Primary Repair and Ligament Reconstruction

Hulse and Shires (1986) suggest that the primary repair or reconstruction of all damaged joint restraints is necessary for the successful treatment of multiple ligamentous stifle injuries. They believe that the intra-capsular reconstruction of both cruciate ligaments together with the careful reconstruction of the joint capsule and any peripheral meniscal injuries, to be vitally important. The secondary proliferation of peri-articular soft tissues, while adding to long-term stability of the joint, was not considered to be the primary stabilising factor.

Reconstruction was accomplished with the fascia lata under-and-over technique for the cranial cruciate ligament (Shires and others 1984) and popliteal tendon transposition for the caudal cruciate ligament (Hulse and Shires 1985). Primary repair of the collateral ligament failures was achieved with a screw and spiked washer or by suturing. Satisfactory limb function was achieved using this method of fixation in a series of four dogs. A consistent finding was a reduction in stifle joint range of motion, particularly in flexion, and this was attributed to peri-articular joint capsule fibrosis (Hulse and Shires 1986).

Some authors recommend that when both cruciate ligaments are ruptured, only the cranial ligament needs to be replaced whilst the caudal joint capsule may be reefed or tightened to stabilise the caudal drawer (Nunamaker 1985, Denny and Minter 1973).

Extra-articular Suture Stabilisation

Aron (1988) reported a series of stifle joint dislocations repaired with extra-articular stabilisation only. It was suggested that the extensive soft tissue damage which occurs with multiple ligamentous stifle injuries causes pronounced inflammatory and collagen repair stages of healing. This results in fibrosis of the peri-articular soft tissue which is sufficiently strong enough to provide long-term joint stability (Aron 1988).

Primary repair of the collateral ligaments was augmented by placing suture prostheses around screws (Rudy 1974). Cranial cruciate ligament stabilisation was achieved by placing extra-articular sutures from the lateral and medial fabellae to the tibial tuberosity and mid substance of the patellar ligament (Flo 1975). For caudal cruciate ligament stabilisation, extra-articular sutures were placed from the head of the fibula and the caudomedial tibial plateau to the origin of the patellar ligament (DeAngelis and Betts 1973). All 12 dogs in this study achieved good to excellent functional results within three months of surgery. In all cases there was a reduction in stifle joint range of motion especially in flexion.

Smith (1995) describes a technique of extra-articular stabilisation of the damaged joint using screws joined by orthopaedic wire sutures. The aim of this technique is to stabilise the joint by the triangulation of fixation from the origins of the collateral ligaments to the proximal tibia. Fixing the centre of rotation at the origin of the collateral ligament results in a sliding-type motion rather than the normal combination of rolling and sliding motion. This modification of stifle kinematics is said to be an acceptable compromise particularly considering the extreme functional damage of multiple ligamentous injuries of the stifle joint.

Trans-articular Pinning

Trans-articular pinning provides temporary internal fixation of the stifle to allow sufficient peri-articular fibrosis formation to provide long-term stability once the pin is removed (Nunamaker 1985). The stifle is held at a functional angle, and one or two pins are placed from tibia to femur crossing the joint through the non-articular areas of the intercondylar eminence of the tibial plateau and intercondylar notch of the femur (Welches and Scavelli 1990). No attempt is made to reconstruct the damaged ligaments and the pins are removed after three to four weeks.

Trans-articular pinning is thought to be quick, inexpensive, easy to perform, and especially suited for small dogs (up to 15 kg) with damaged tissues unsuitable for primary reconstruction, or cases in which anaesthesia time should be limited (Welches and Scavelli 1990). In a series of three small dogs and seven cats, excellent long-term results were obtained in 70% of the cases using this technique (Welches and Scavelli 1990). Complications such as pin migration and bending of the pin were seen in 30% of these cases.

Stifle Arthrodesis

Stifle arthrodesis has been recommended in acute cases of multiple ligamentous stifle injuries where there is overwhelming bone and soft tissue injury rendering the joint non-functional; or, in chronic cases, with intractable pain secondary to degenerative joint disease (Newton 1985).

A number of methods for performing stifle arthrodesis have been described and these include: cranial placement of a plate, full- and half-pin external fixation and trans-articular pins and screws (Newton 1985). A review of eight dogs undergoing unilateral or bilateral stifle arthrodesis, showed that acceptable function was achieved in seven of the dogs treated (87.5%) (Cofone and others 1992). Factors affecting the return of limb function included the angle of arthrodesis and lesions in the ipsilateral coxofemoral joint.

Amputation

Amputation of the pelvic limb is indicated when its dysfunction is beyond repair and the limb is more a hindrance than a help. Some authors feel that the overall function of a limb with a stifle arthrodesis is not as good in most dogs as with amputation (Brinker and others 1990).

POSTOPERATIVE MANAGEMENT

Almost all cases of multiple stifle ligament reconstruction reported in the literature were given some form of external support for three to eight weeks postoperatively. The types of external support recorded were: soft padded bandaging (Hulse and Shires 1986), Schroeder-Thomas splints (Smith 1995), plaster casts (Denny and Minter 1973), resin impregnated fibre-glass casts (Parker and Schubert 1981, Panco 1985) and trans-articular external skeletal fixators (Aron 1988).

Welches and Scavelli (1990) recommended the use of the trans-articular external skeletal fixator to provide more rigid fixation when using the trans-articular pinning technique in larger dogs.

Exercise was restricted in all cases for an average of six to 12 weeks postoperatively.

SECTION 3

CLINICAL CASES

SECTION 3: PART 1

MATERIALS AND METHODS

CASE SELECTION

Eleven dogs were referred to the University of Glasgow Veterinary School (GUVS) for investigation and treatment of lameness, all of which had a severely unstable stifle joint secondary to multiple ligamentous injury as one of the clinical signs. The cases included were only those examined and treated by the author during the period October 1992 to August 1995. A diagnosis of multiple ligamentous injury of the stifle joint was established on the basis of clinical and radiographic findings, supplemented by the findings at surgery. In all cases clinical and radiographic examinations were performed with the aid of heavy sedation or general anaesthesia (Appendix 1, page 110). The diagnostic criteria for ligament injury in the stifles were: partial or complete interruption of the ligament, ligament-bone failure from its point of attachment or ligament-bone avulsion with an osseous fragment.

SURGICAL TECHNIQUE

All of the dogs in this series were treated surgically using a lateral parapatellar approach to the stifle (Piermattei 1993). A strip of fascia was harvested from the lateral third of the patellar ligament and the proximal fascia lata (Hulse and others 1980) in those cases that had not had previous stifle surgery (Figure 23, page 63). Inspection of the joint was performed through a lateral arthrotomy and all remnants of the damaged cruciate ligament(s) were removed. Repair of the articular fracture in case 10 was performed at this stage. Where the menisci could not be preserved a partial or complete meniscectomy was performed, depending on the extent of meniscal damage. Meniscal peripheral avulsion injuries were repaired by suturing the abaxial boarder of the meniscus to the surrounding joint capsule using horizontal mattress sutures of 3 metric polydioxanone (PDS; Ethicon¹).

Next the damaged collateral ligaments were repaired. Adequate exposure of the lateral collateral ligament was gained through the original incision, and exposure of the medial collateral ligament was performed by medial retraction of the original incision or by a separate medial approach (Piermattei 1993). Where possible, primary repair of interstitial collateral ligament tears was achieved using 3 metric polydioxanone placed in a locking loop suture pattern. The edges of the ligament were further apposed with simple horizontal mattress sutures of 2 metric polydioxanone (Figure 24, page 63).

¹Ethicon Ltd., Veterinary Division, P.O. Box 408, Bankhead Ave., Edinburgh, EH11 4HE, UK.

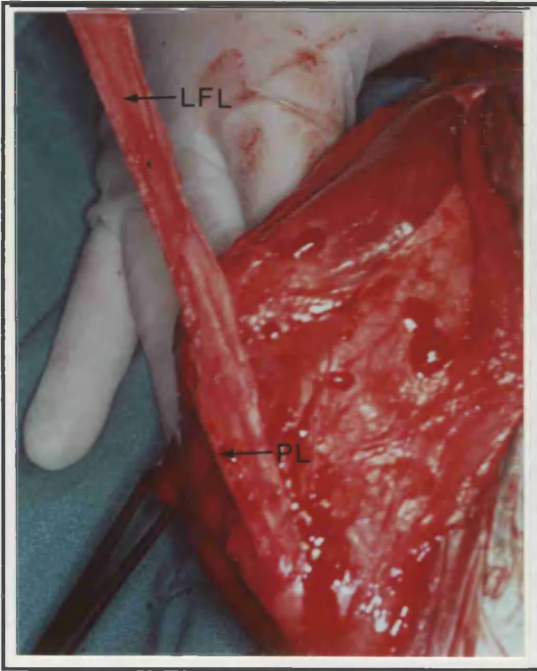


Figure 23 Fascial graft used in cruciate ligament reconstruction.

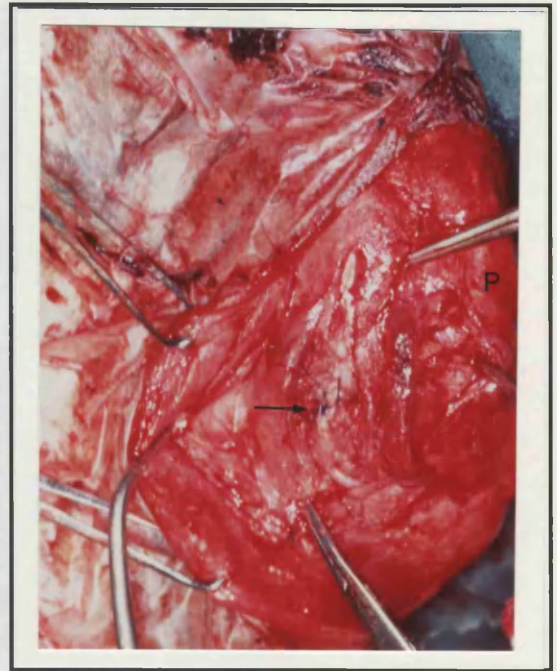


Figure 24 Primary repair of the medial collateral ligament.

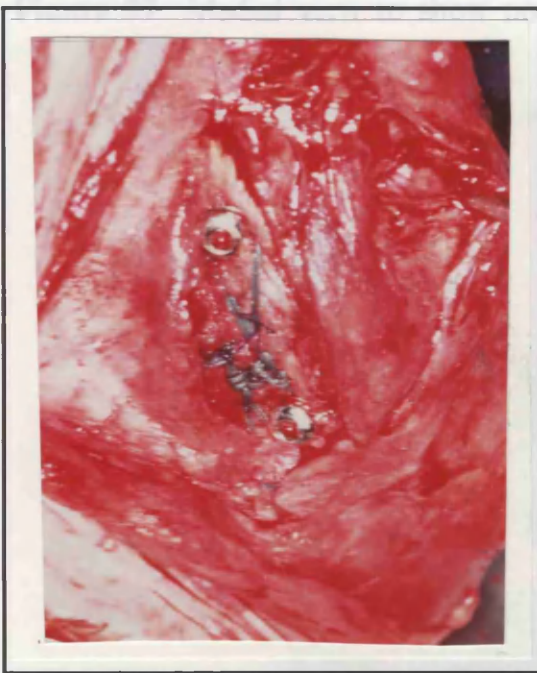


Figure 25 Augmented medial collateral ligament repair.



Figure 26 Fascial graft sutured to the lateral femoral condyle.

P = patella, PL = patellar ligament, LFL = lateral fascia lata, MCL = medial collateral ligament, LTR = lateral trochlear ridge. LF = lateral fabella, FG = fascial graft.

Figure 28 Lateral view of knee joint showing the graft and Figure 29 suture used in lateral collateral ligament repair.

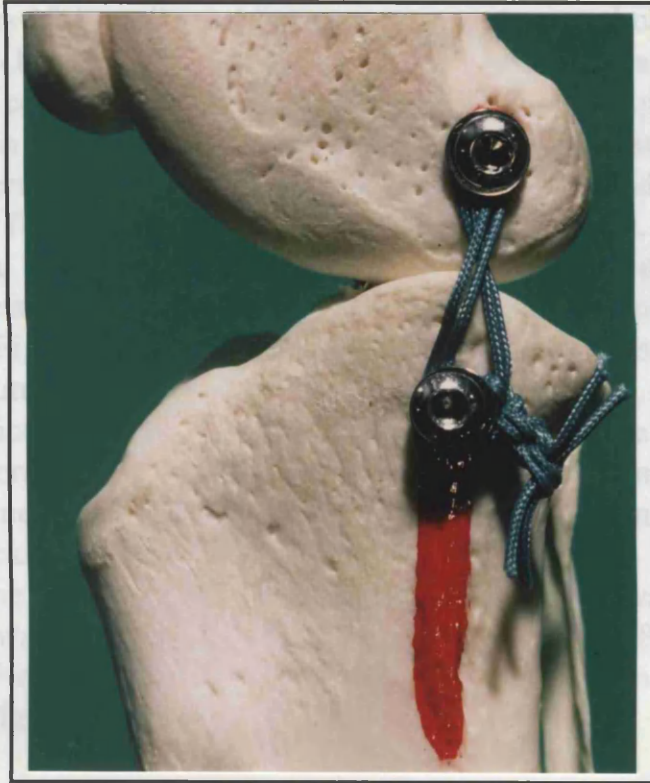


Figure 27 Medial view to show position of screw and figure-8 suture used in medial collateral ligament repair.

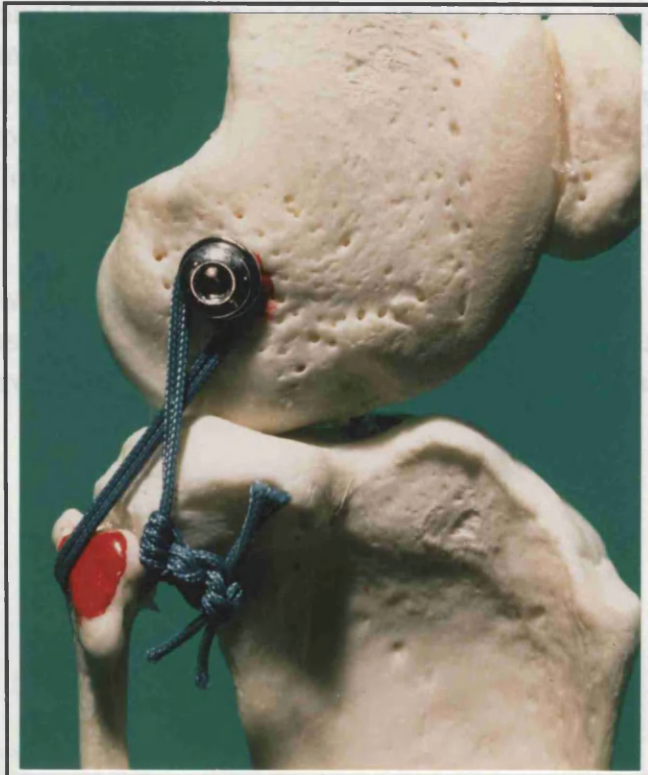


Figure 28 Lateral view to show position of screw and figure-8 suture used in lateral collateral ligament repair.

All primary repairs of the collateral ligaments were augmented using the screw and figure-8 suture technique (Rudy 1974). Either 3.5 mm cortical screws or 4 mm partially threaded cancellous screws were used as suture anchor points. These were placed at the origin and insertion of the injured collateral ligament. No screws were placed at the insertion of the lateral collateral ligament, instead, a 2mm diameter hole drilled in the fibular head was used as a suture anchor point. A figure-8 double suture of 5 metric braided polyester (Ethibond; Ethicon²) was placed between the anchor points; the stifle was fully extended and the suture tightened and tied using a five-throw knot (Figures 27 & 28, page 64). Once tied, the ends of the suture were left longer and sutured into the surrounding soft tissue with 3 metric polyglactan 910 (Vicryl; Ethicon³) (Figure 25, page 63). This technique was also used to reconstruct collateral ligaments which were not amenable to primary repair. Repair of collateral ligament-bone failures and ligament-bone avulsions was achieved by placing a half locking loop suture pattern in the ligament ends and tying this to a screw placed at the point of ligament detachment. The screw was tightened so that a plain or spiked washer⁴ trapped the ligament against the bone (Parker and Schubert 1981). In case 5 only a screw and spiked washer was used to repair the avulsed lateral collateral ligament insertion.

Finally, the cranial cruciate ligament was reconstructed by passing the fascial graft through the joint using a curved graft passer⁵ to emerge at the lateral fabella (Hulse and others 1980). The graft was tensioned and, either sutured to the lateral femorofabellar ligament and lateral periosteum of the femoral condyle using 3 metric polydioxanone (Figure 26, page 63) or, secured to the lateral femoral condyle using a titanium staple⁶ or screw and spiked washer. In cases 8 and 9, the cranial cruciate ligament was replaced with lateral and medial extra-capsular sutures of 4.5 metric polydioxanone passed between the fabellae and a hole drilled through the tibial tuberosity (Flo 1975). No attempt was made to reconstruct the caudal cruciate ligament in any of the cases.

The joint capsule was closed with 3 metric polyglactan 910 in a simple continuous pattern and the fascia lata with 3 metric polydioxanone in a simple interrupted pattern. The closure of the remaining soft tissues and skin was routine.

²Ethicon Ltd., Veterinary Division, P.O. Box 408, Bankhead Ave, Edinburgh, EH11 4HE, UK.

³Ethicon Ltd., Veterinary Division, P.O. Box 408, Bankhead Ave, Edinburgh, EH11 4HE, UK.

⁴3.5mm Polyacetal Resin Plastic Spiky Washer, Veterinary Instrumentation, 62 Cemetery Rd, Sheffield, S11 8FP, UK.

⁵Large Curved Graft Passer 4.5cm, Veterinary Instrumentation, 62 Cemetery Rd, Sheffield, S11 8FP, UK.

⁶ACL Spiky Staples, Veterinary Instrumentation, 62 Cemetery Rd, Sheffield, S11 8FP, UK.

POSTOPERATIVE MANAGEMENT

The perioperative and postoperative analgesic and antibiotic regimens were similar in all cases and these are detailed in Appendix 2, page 111.

Immobilisation and Exercise

The stifle joint was supported in a modified Robert Jones bandage for three to five days postoperatively.

In all cases, early ambulation was encouraged immediately postoperatively. However, exercise was restricted for the first six weeks and confined to short 10 minute walks on a leash. Owners were encouraged to perform passive range of motion exercises several times a day for three weeks postoperatively. The duration and frequency of controlled exercise was gradually increased after six weeks postoperatively, depending on the clinical and radiographic findings at follow-up. Dogs were allowed unlimited exercise from 16 weeks postoperatively.

POSTOPERATIVE ASSESSMENT

At least two postoperative examinations were performed in each case (except case 6). These were carried out between six and 12 weeks and at some time greater than five months postoperatively.

At the early postoperative examination, patient limb usage while standing, walking, and trotting was noted. Lameness was graded subjectively by the author on a graduated scale of 0 to 10. The lowest score (0/10) represented a completely sound individual, and the highest score (10/10) a continuously lame non-weight bearing individual. Stability (craniocaudal and varus/valgus), stifle joint thickening and presence of pain and crepitation on manipulation of the joint were assessed by palpation. A sample of synovial fluid was aspirated from the affected joint and analysed in two dogs (cases 8 and 9) at this time.

At long-term follow-up, subjective owner appraisal of the dog's limb function was noted. Functional performance was rated as excellent, good, fair, or poor, based on a system devised by Smith (1985).

1. Excellent = No lameness under any circumstance.
2. Good = Transient lameness after exertion.
3. Fair = Intermittent lameness associated with normal day-to-day activities.
4. Poor = Continuous lameness.

The physical examination described above was repeated by the author. In addition, muscle mass of the affected hind limb was compared to that of the unaffected hind limb by measuring the mid-thigh muscle mass circumference (Figure 29, page 68). Stifle joint range of motion of the affected and unaffected hind limbs was measured with a goniometer. For evaluation of range of motion, the zero point was the point at which the tibial axis formed an angle of 90 degrees with the femoral shaft. Flexion and extension were measured in degrees from the zero point (Newton and Nunamaker 1985) (Figures 30 to 32, page 68).

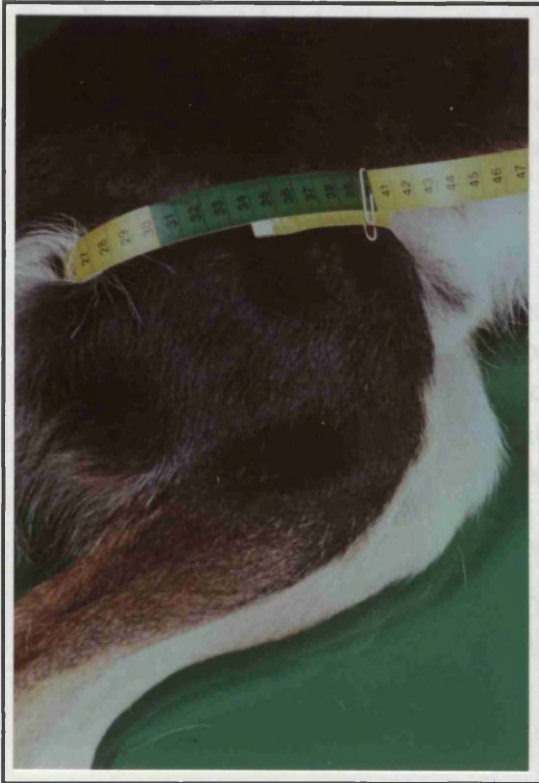


Figure 29 Measurement of mid-thigh muscle mass circumference.

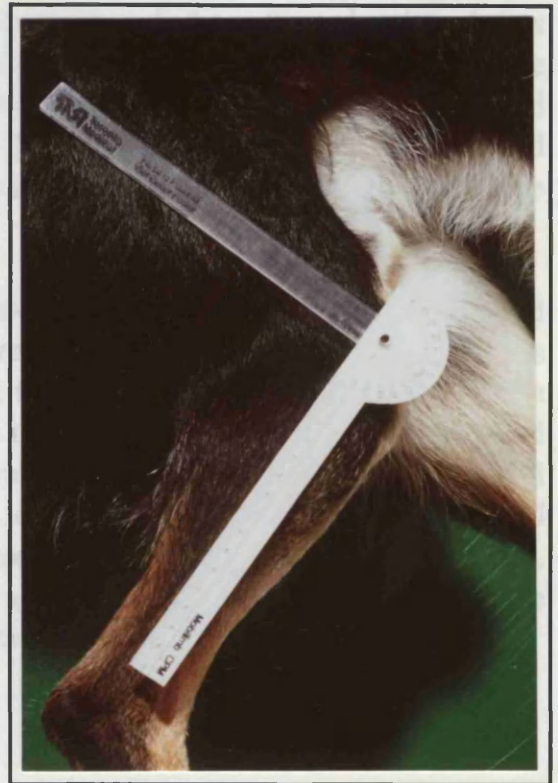


Figure 30 Measurement of range of motion, zero point.



Figure 31 Measurement of range of motion, maximum flexion.

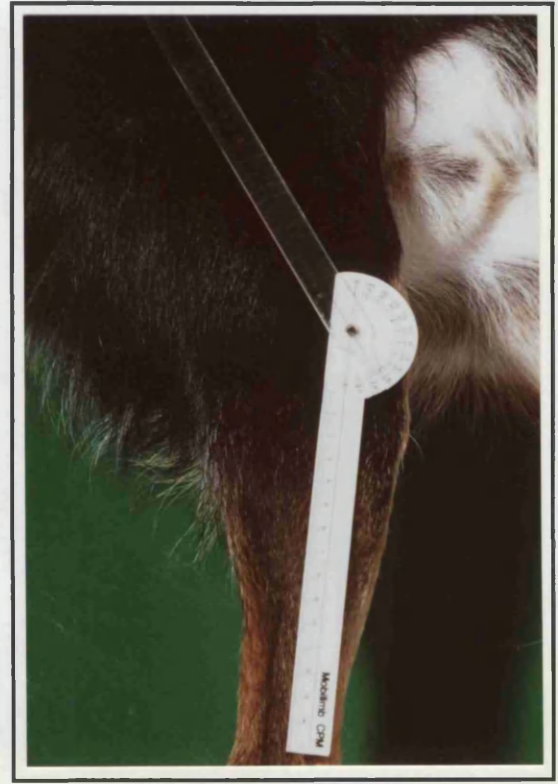


Figure 32 Measurement of range of motion, maximum extension.

Craniocaudal and lateral radiographs were taken of the affected stifle joints preoperatively and at long-term follow-up. These assessed for osteoarthritic change using a scoring system based on modifications of the system devised by Bennett and others (1988) (Figures 33 & 34, pages 70 & 71). All radiographs were viewed and scored by an experienced radiologist⁷ who was unaware of the individual case, time interval, or treatment given.

Comparison of the clinical parameters of mid-thigh muscle mass circumference and stifle joint flexion and extension, preoperatively and long-term postoperatively, was performed using the Wilcoxon Signed Rank test of significance on the paired data for each case. A one-tailed test was used as the hypothesised change was expected to be in one direction only. In the comparison of the paired joint lateral and medial osteophyte scores for preoperative and long-term postoperative injured and uninjured data, the same one-tailed Wilcoxon Signed Ranked test was also used. Significance was set at the 5% level for all analyses. All computations were performed using Statistix version 4 (Analytical Software).

⁷Dr Martin Sullivan BVMS, PhD, DVR, MRCVS. Department of Veterinary Clinical Studies, University of Glasgow Veterinary School, Bearsden, Glasgow, G161 1QH, UK.

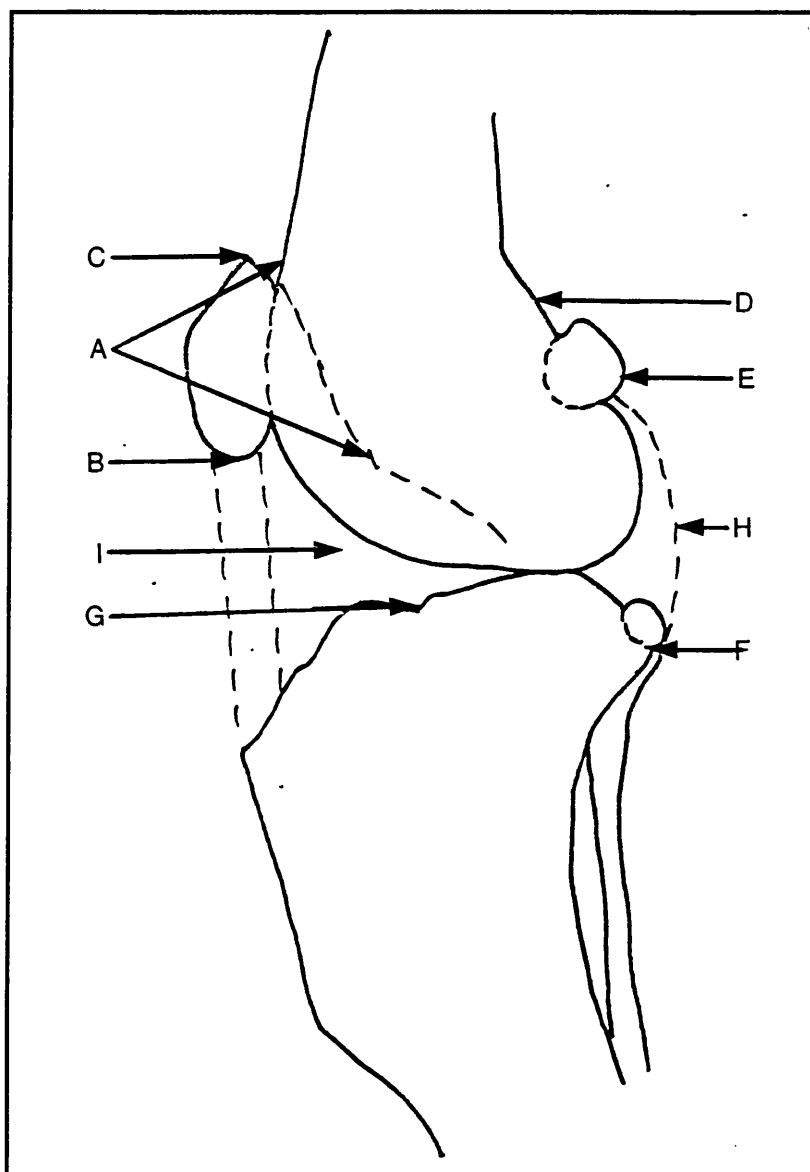


Figure 33 Lateral view of the stifle joint illustrating the scoring system used to identify pathological changes.

- A Osteophytes at proximal aspect of trochlear groove and along margin of trochlear ridge.
- B Osteophytes at distal pole of patella.
- C Osteophytes at proximal pole of patella.
- D Osteophytes on the caudal femur associated with joint capsule attachment.
- E Osteophytes on fabellae.
- F Osteophytes on caudal edge of tibia.
- G Osteophytes on tibial plateau.
- H Distension of caudal joint capsule with displacement of fascial plane.
- I Loss of the infra-patellar fat pad.

Each pathological change was scored 0 (absent), 1 (equivocal change), 2, 3, 4, or 5 (very advanced change).

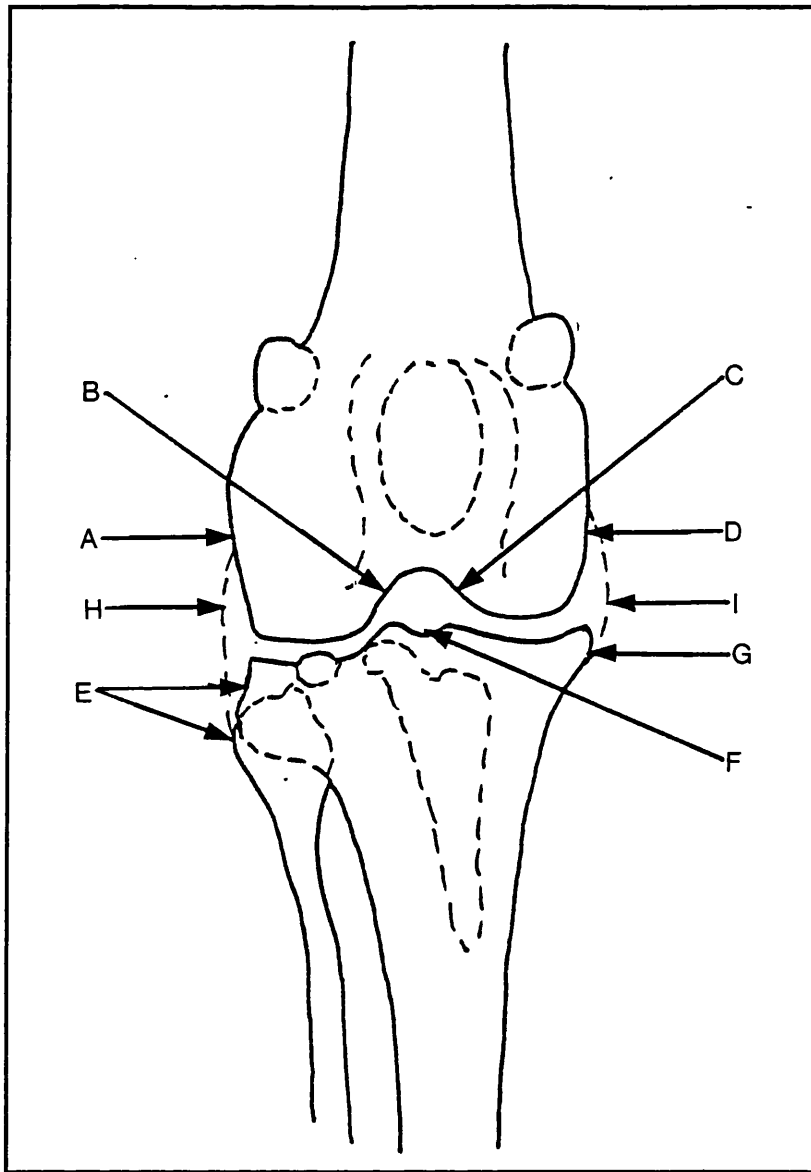


Figure 34 Craniocaudal view of the stifle joint illustrating the scoring system used to identify pathological changes.

- A Osteophytes on abaxial margin of lateral femoral condyle.
- B Osteophytes on axial margin of lateral femoral condyle.
- C Osteophytes on axial margin of medial femoral condyle.
- D Osteophytes on abaxial margin of medial femoral condyle.
- E Osteophytes on lateral margin of proximal tibia and fibular head.
- F Osteophytes on central tibial plateau.
- G Osteophytes on medial margin of proximal tibia.
- H Lateral soft tissue thickening.
- I Medial soft tissue thickening.

Each pathological change was scored 0 (absent), 1 (equivocal change), 2, 3, 4, or 5 (very advanced change).

SECTION 3: PART 2

CASE SUMMARIES

CASE 1: No. 124018

Signalment: "Kate" a two-year-old entire female working Border collie. Weight = 15.4 kg.

History: This dog was presented with a left hind limb lameness of two days duration which occurred whilst working sheep. The dog was seen to step in a hole and tumble becoming immediately lame. The limb was held in a non-weight bearing position for a day, after which, the dog began to touch the ground with its toes, when standing.

Clinical Findings: A routine clinical examination revealed no abnormalities except those relating to the affected limb. Manipulation of the stifle joint was intensely painful and a stifle joint effusion was palpable. There was marked craniocaudal instability, increased tibial internal and external rotation, and valgus instability.

Radiographic Findings: Radiographs revealed increased soft tissue swelling within and around the joint and reduction of the infra-patellar fat pad. A valgus stressed craniocaudal view showed medial widening of the joint space (Figure 35, page 73).

Diagnosis: Left stifle joint subluxation.

Stifle Joint Injuries:

Complete interstitial rupture of the cranial cruciate ligament

Complete interstitial rupture of the caudal cruciate ligament

Ligament-bone failure at the origin of the medial collateral ligament

Avulsion and crushing injuries to the caudal horn of the medial meniscus

Management: The cranial cruciate ligament was reconstructed, as described, and the caudal horn of the medial meniscus was removed. The origin of the medial collateral ligament was sutured to a screw and the repair was augmented, as described above (Figure 36, page 73).

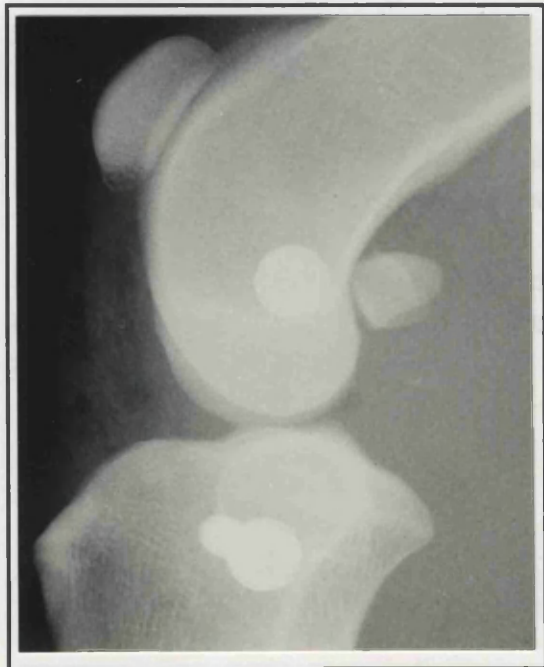


a.

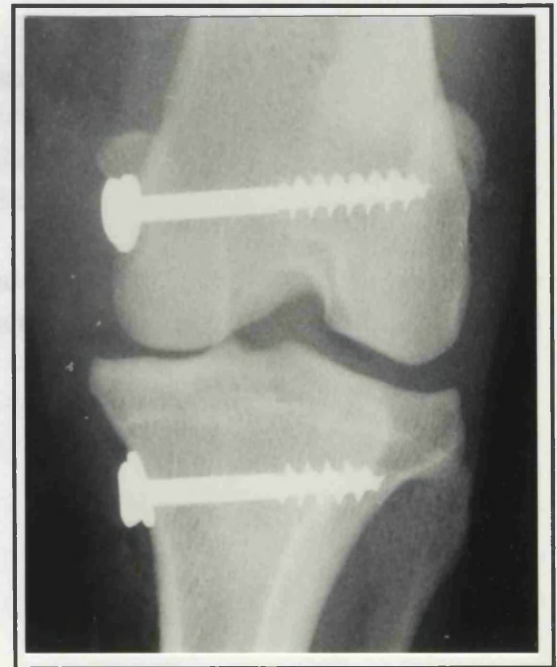


b.

Figures 35a & b Case 1. Preoperative lateral and craniocaudal radiographs of the left stifle joint.



a.



b.

Figures 36a & b Case 1. Immediate postoperative lateral and craniocaudal radiographs of the left stifle joint.

CASE 2: No. 122315

Signalment: "Jim", a six-year-old entire male working Border collie. Weight = 21.7 kg.

History: This dog was presented with a left non-weight bearing hind limb lameness of seven days duration following an incident in which it tumbled down a banking whilst working sheep.

Clinical Findings: A routine clinical examination revealed no abnormalities except those relating to the affected stifle joint. Manipulation of the joint was intensely painful and normal range of motion was reduced, particularly in extension. A soft fluctuant swelling was present around the patellar ligament and medial to the stifle joint. There was craniocaudal and valgus instability, in addition to increased tibial internal rotation in extension, and increased external rotation in flexion.

Radiographic Findings: Radiography revealed increased soft tissue swelling within and around the stifle joint with reduction of the infra-patellar fat pad. A valgus stressed craniocaudal view showed medial widening of the joint space.

Diagnosis: Left stifle joint subluxation.

Stifle Joint Injuries:

Complete interstitial rupture of the cranial cruciate

Complete interstitial rupture of the medial collateral ligament

Peripheral avulsion of the medial meniscus

Peripheral avulsion, crushing and tearing injuries to the lateral meniscus

Management: The cranial cruciate and medial collateral ligaments were reconstructed as described. The lateral meniscus was removed and the medial meniscus was sutured in position.

CASE 3: No. 126109

Signalment: "Dell" an eight-year-old entire female working Border collie. Weight = 15.5 kg.

History: This dog was presented with a right hind limb lameness of 19 days duration which occurred after jumping a fence. The dog became momentarily suspended by the limb after it was caught between the top wires of the fence. The limb was held in a non-weight bearing position for a few days. However, after a course of analgesics the dog was able to take some weight on the limb when standing.

Clinical Findings: A routine clinical examination revealed no abnormalities except those relating to the affected limb. There was atrophy of the quadriceps muscle, stifle joint effusion, and medial and lateral thickening over the joint. Manipulation of the stifle joint, particularly forced extension, was intensely painful. There was marked craniocaudal instability, increased tibial internal rotation and varus instability.

Radiographic Findings: Radiography revealed increased soft tissue swelling within the joint with reduction of the infra-patellar fat pad. A varus stressed craniocaudal view showed medial widening of the joint space with avulsion fragments lateral to the femoral condyle and within the intercondylar area (Figures 37a & b, page 76).

Diagnosis: Right stifle joint subluxation.

Stifle Joint Injuries:

Complete interstitial rupture of the cranial cruciate ligament

Partial rupture of the caudal cruciate ligament

Ligament-bone avulsion of the origin of the lateral collateral ligament

Ligament-bone avulsion of the origin of the popliteus muscle tendon

Peripheral avulsion and crushing injuries to the caudal horn of the medial meniscus

Management: The cranial cruciate was reconstructed as described. The caudal horn of the medial meniscus was removed. The origin of the lateral collateral ligament and the popliteus muscle tendon were sutured to a screw and washer placed in the lateral femoral condyle and the repair augmented as described (Figures 38a & b, page 76).



a.



b.

Figures 37a & b Case 3. Preoperative lateral and craniocaudal radiographs of right stifle joint. Note the soft tissue swelling and the small avulsion fragments at the lateral femoral condyle.



a.



b.

Figures 38a & b Case 3. Three month postoperative lateral and craniocaudal radiographs of right stifle joint.

CASE 4: No. 125193

Signalment: "Jet" a seven-year-old entire male Labrador cross. Weight = 30 kg.

History: This dog was presented with a right hind limb lameness of three days duration which occurred after jumping a gate. The dog caught the limb between the top boards of the gate and became suspended by the limb for several seconds. After the incident, the limb was held up under the body although, by the time of presentation, the dog had started to touch the toes to the ground.

Clinical Findings: A routine clinical examination revealed no abnormalities except those relating to the affected limb. Stifle joint effusion was palpable and there was soft tissue swelling over the lateral collateral ligament. Manipulation of the stifle joint was intensely painful and there was marked craniocaudal instability, increased tibial internal rotation, and varus instability.

Radiographic Findings: Radiography revealed increased soft tissue swelling within and around the joint with reduction of the infra-patellar fat pad. A varus stressed craniocaudal view showed lateral widening of the joint space.

Diagnosis: Right stifle joint subluxation.

Stifle Joint Injuries:

Complete interstitial rupture of the cranial cruciate ligament

Complete interstitial rupture of the caudal cruciate ligament

Ligament-bone failure at the origin of the lateral collateral ligament

Avulsion and crushing injuries to the caudal horn of the medial meniscus

Management: The cranial cruciate was reconstructed as described and the caudal horn of the medial meniscus was removed. The origin of the lateral collateral ligament was sutured to a screw and the repair was augmented as described.

CASE 5: No. 126408

Signalment: "Ted", a one-and-a-half year-old entire male cross breed. Weight = 15.5 kg.

History: This dog was presented with a non-weight bearing right hind limb lameness after being involved in a road traffic accident.

Clinical Findings: A routine clinical examination revealed multiple minor superficial wounds over the limbs and face but no other serious abnormalities, except those relating to the affected limb. Manipulation of the hip and stifle joint was intensely painful, normal range of motion was reduced and a soft fluctuant swelling was present around the patellar ligament and lateral to the stifle joint. Palpation of the hip joint revealed craniodorsal displacement of the greater trochanter. Marked craniocaudal and varus instability, as well as increased tibial internal rotation, was present in the stifle joint.

Radiographic Findings: Radiographs of the stifle joint revealed increased soft tissue swelling within and around the joint with reduction of the infra-patellar fat pad. A varus stressed craniocaudal view showed lateral widening of the joint space with an avulsed fragment lateral to the fibular head (Figure 39, page 79). Radiographs of the right coxofemoral joint revealed a craniodorsal dislocation.

Diagnosis: Right stifle subluxation. Right craniodorsal coxofemoral dislocation.

Stifle Joint Injuries:

Complete interstitial failure of the cranial cruciate ligament

Stretched and partially torn caudal cruciate ligament

Ligament-bone avulsion of the insertion of the lateral collateral ligament

Management: The cranial cruciate ligament was reconstructed using a fascial graft; the lateral collateral ligament was reattached to the fibular head using a screw and spiked washer (Figure 40, page 79). The luxated hip was reduced and stabilised using toggle-pin fixation (Brinker and others 1990).

CASE 5: No. 123

Signalment: "Solo"
kg.

History: This dog was
duration which occurred
limb after it was caught
release him. The limb
then the dog began to

Clinical Findings: A
those relating to the
tissue swelling over
painful and there was
rotation, and varus



Figure 39 Case 5. Preoperative craniocaudal radiograph of the right stifle joint. The lateral joint space is widened laterally and there is an avulsed fragment of bone lateral to the fibular head.

Diagnosis: Right stifle joint subluxation.

Stifle Joint Injuries:

- Complete inter
- Complete infer
- Complete later
- Rupture of the

Management: The
reconstructed and the



Figure 40 Case 5. Immediate postoperative craniocaudal radiograph of the right stifle joint. The avulsed fragment has been removed and the lateral collateral ligament insertion secured to the fibular head with a screw and spiked washer.

CASE 6: No. 123011

Signalment: "Solo" a nine-year-old entire male Labrador retriever. Weight = 29 kg.

History: This dog was presented with a right hind limb lameness of three days duration which occurred after jumping a gate. The dog became suspended by the limb after it was caught between the top bars of the gate and required the owner to release him. The limb was held in a non-weight bearing position for two days and then the dog began to touch the ground with its toes when standing.

Clinical Findings: A routine clinical examination revealed no abnormalities except those relating to the affected limb. A joint effusion was palpable and there was soft tissue swelling over the stifle area. Manipulation of the stifle joint was intensely painful and there was marked craniocaudal instability, increased tibial internal rotation, and varus instability.

Radiographic Findings: Lateral views revealed increased soft tissue swelling within and around the joint and reduction of the infra-patellar fat pad. A varus stressed craniocaudal view showed lateral widening of the joint space.

Diagnosis: Right stifle joint subluxation.

Stifle Joint Injuries:

- Complete interstitial rupture of the cranial cruciate ligament
- Complete interstitial rupture of the caudal cruciate ligament
- Complete interstitial rupture of the lateral collateral ligament
- Rupture of the lateral joint capsule

Management: The cranial cruciate ligament and lateral collateral ligament were reconstructed and the joint capsule sutured, as described.

CASE 7: No. 122697

Signalment: "Dusty" a four-and-a-half-year-old entire male working Labrador retriever. Weight = 25 kg.

History: This dog was presented with a right hind limb lameness of 15 days duration which occurred after jumping a fence. The dog had become suspended by the limb after it was caught between the top boards of the fence. The limb was placed in a modified Robert-Jones bandage until referral and some weight was taken on the supported limb.

Clinical Findings: A routine clinical examination revealed no abnormalities except those relating to the affected limb. A joint effusion was palpable; there was quadriceps atrophy and thickening around the stifle joint. Manipulation of the stifle joint was intensely painful with marked cranio-caudal instability, increased tibial internal rotation, and varus instability.

Radiographic Findings: Lateral views revealed increased soft tissue swelling within the joint with reduction of the infra-patellar fat pad. A varus stressed cranio-caudal view showed lateral widening of the joint space.

Diagnosis: Right stifle joint subluxation.

Stifle Joint Injuries:

- Partial interstitial rupture of the cranial cruciate ligament
- Complete interstitial rupture of the caudal cruciate ligament
- Complete interstitial rupture of the lateral collateral ligament
- Peripheral avulsion of the lateral meniscus
- Rupture of the lateral joint capsule

Management: The cranial cruciate ligament and lateral collateral ligament were reconstructed, as described. The lateral meniscus was sutured in position and the lateral joint capsule repaired.

CASE 8: No. 123611

Signalment: "Toby" a three-year-old entire male golden retriever. Weight = 36 kg.

History: This dog had a history of partial rupture of the left cranial cruciate ligament which was surgically repaired by the method described by Hulse and others (1980). Two months after the surgery the owner considered the dog to be fully recovered. One month later, the dog developed an acute onset non-weight bearing left hind limb lameness. To the owners knowledge, there was no history of trauma at the time of onset.

Clinical Findings: A routine clinical examination revealed no abnormalities except those relating to the affected limb. Quadriceps muscle atrophy, stifle joint effusion, and medial thickening were present. There was evidence of pain, crepitation and marked craniocaudal and varus instability on manipulation of the stifle joint.

Radiographic Findings: The radiographs revealed signs of osteoarthritis, increased soft tissue swelling within the joint, and reduction of the infra-patellar fat pad. A varus stressed craniocaudal view showed lateral widening of the joint space (Figures 41a & b, page 83).

Synovial Fluid Analysis: The synovial fluid analysis revealed a high white cell count consisting predominantly of polymorph neutrophils (Cell count = $83.8 \times 10^9 l^{-1}$, 92% PMN). No bacteria were cultured from the synovium, fibrin clots within the joint, or the synovial fluid.

Diagnosis: Left stifle joint subluxation and joint sepsis.

Stifle Joint Injuries:

- Absence of the fascial graft used to reconstruct the cranial cruciate ligament
- Complete interstitial rupture of the caudal cruciate ligament
- Stretched medial and lateral collateral ligaments
- Partial rupture of the lateral collateral ligament
- Peripheral avulsion of the lateral meniscus

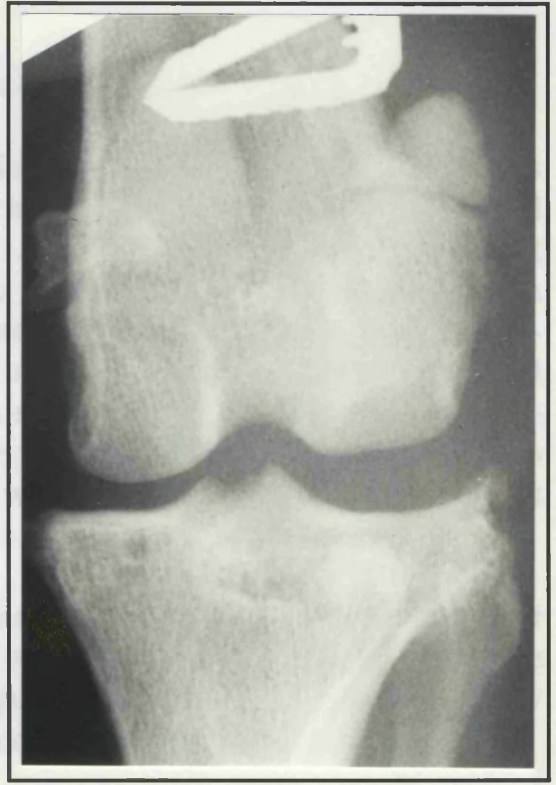
A biopsy of the joint capsule revealed changes consistent with a sub-acute synovitis.

Management: The craniocaudal instability was stabilised by passing an extra-articular suture from the lateral fabella to the tibial tuberosity. The lateral collateral ligament was shortened and repaired, as described above (Figures 42a & b, page 83). The medial joint capsule and collateral ligament were imbricated using horizontal mattress sutures of 3 metric polydioxanone. A six week course of cephalexin tablets (Ceporex; Pitman-Moore Ltd⁸) given at a dose of 10 mg/kg bid was prescribed.

⁸Pitman-Moore Ltd., Harefield, Uxbridge, Middlesex, UB9 6LS, UK.



a.



b.

Figures 41a & b Case 8. Preoperative lateral and craniocaudal radiographs of the left stifle joint. Note the radiographic signs of osteoarthritis and the radiolucent halo present around the staple.



a.



b.

Figures 42a & b Case 8. Immediate postoperative lateral and craniocaudal radiographs of the left stifle joint.

CASE 9: No. 115198

Signalment: "Brando" a four-year-old entire male Rottweiler. Weight = 44 kg.

History: This dog had a history of left cranial cruciate ligament rupture which was surgically repaired by the method described by Hulse and others (1980). A partial medial meniscectomy was also performed. As a result of this surgery the joint became infected and *Staphylococcus hominus* was isolated from the synovial fluid a few days postoperatively. The dog was treated with cephalexin (Ceporex; Pitman-Moore Ltd) at 20mg/kg bid for six weeks and appeared to make an uneventful recovery. Seven months later the dog was represented with an acute onset non-weight bearing left hind limb lameness. To the owners knowledge, there was no history of trauma as the injury had occurred whilst the dog had been confined to its kennel.

Clinical Findings: A routine clinical examination revealed no abnormalities except those relating to the affected limb. There was quadriceps muscle atrophy, stifle joint effusion, and medial thickening. Manipulation of the stifle joint was intensely painful and there was marked craniocaudal and valgus instability.

Radiographic Findings: The radiographs revealed signs of osteoarthritis, increased soft tissue swelling within the joint and reduction of the infra-patellar fat pad. A valgus stressed craniocaudal view showed medial widening of the joint space.

Synovial Fluid Analysis: The synovial fluid analysis revealed a high white cell count consisting predominantly of polymorph neutrophils (Cell count = $84.9 \times 10^9 l^{-1}$, 94% PMN). No bacteria were cultured from the synovium or synovial fluid.

Diagnosis: Left stifle joint subluxation and joint sepsis.

Stifle Joint Injuries:

- Absence of the fascial graft used to reconstruct the cranial cruciate ligament
- Complete interstitial rupture of the caudal cruciate ligament
- Partial interstitial rupture and stretching of the medial collateral ligament

Management: The craniocaudal instability was stabilised by passing two extra-articular sutures from each fabella to the tibial tuberosity. The medial collateral ligament was shortened and repaired as described above. A 12 week course of clindamycin capsules (Antirobe, Upjohn Ltd.⁹) given at a dose of 5.5 mg/kg bid was prescribed.

⁹Upjohn Ltd., Animal Health Division, Crawley, RH10 2LZ, UK.

CASE 10: No. 124690

Signalment: "Joe" a three-year-old entire male working Border Collie. Weight = 20.5 kg.

History: This dog was presented on the day of injury with an acute onset non-weight bearing left hind limb lameness. The injury occurred whilst the dog was gathering sheep over open hills at speed.

Clinical Findings: A routine clinical examination revealed no abnormalities except those relating to the affected limb. There was soft tissue swelling over the affected stifle joint. Manipulation of the joint was intensely painful, normal range of motion was reduced, and crepitation was present. There was marked craniocaudal instability, increased internal and external tibial rotation, and valgus instability.

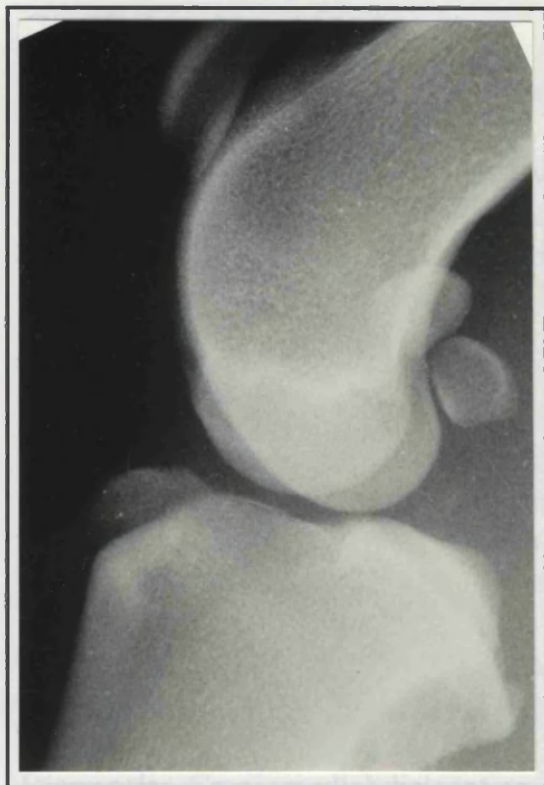
Radiographic Findings: Radiography revealed caudal and medial dislocation of the tibia. A small displaced fragment of bone was evident at the cranial aspect of the proximal tibia. A valgus stressed craniocaudal view showed medial widening of the joint space (Figures 43a & b, page 86).

Diagnosis: Articular fracture and caudomedial dislocation of the left stifle joint.

Stifle Joint Injuries:

- Complete interstitial rupture of the cranial cruciate ligament
- Complete interstitial rupture of the caudal cruciate ligament
- Ligament-bone failure at the insertion of the medial collateral ligament
- Rupture of the cranial meniscotibial and intermeniscal ligaments
- Complete peripheral avulsion and caudal displacement of the menisci
- Rupture of the caudal joint capsule
- Fracture and displacement of the craniolateral tibial plateau

Management: The craniolateral tibial plateau fracture was reduced and stabilised with a lag screw and K wire. The cranial cruciate and medial collateral ligaments were reconstructed, as described (Figures 44a & b, page 86). The menisci were sutured in position and the intermeniscal ligament was sutured with a locking loop suture of 2 metric polydioxanone.



a.

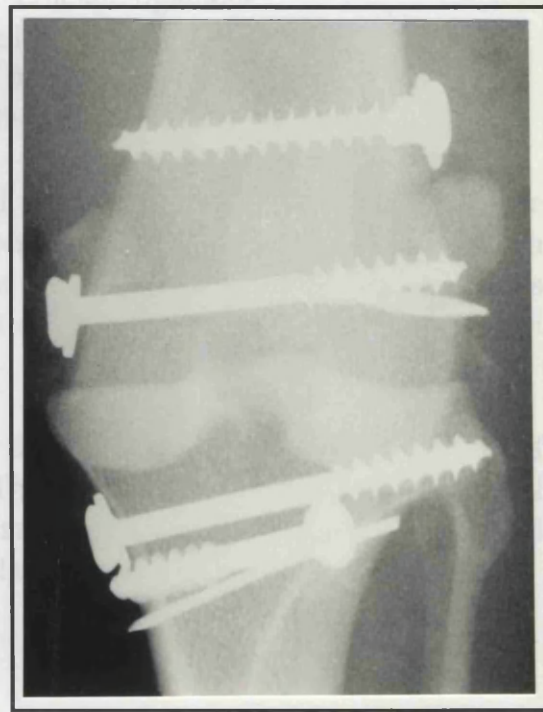


b.

Figures 43a & b Case 10. Preoperative lateral and craniocaudal radiographs of the left stifle joint. Note the articular fracture and the caudomedial dislocation of the joint.



a.



b.

Figures 44a & b Case 10. Immediate postoperative lateral and craniocaudal radiographs of the left stifle joint. A broken piece of drill bit was left *in situ* near the distal femoral screw.

CASE 11: No. 123050

Signalment: "Sam" a ten-year-old entire male Lurcher cross. Weight = 14.5 kg.

History: This dog was presented with a right hind limb lameness of ten days duration having caught it in a fence whilst chasing a rabbit. From the time immediately after the incident until presentation the dog did not bear any weight on the affected limb.

Clinical Findings: A routine clinical examination revealed no abnormalities except those relating to the affected limb. There was obvious mal-alignment of the stifle joint with craniomedial displacement of the tibia, medial displacement of the patella, and internal rotation of the crus. Extensive bruising and soft tissue swelling were present around and distal to the stifle joint. Manipulation of the joint was painful and there was minimal range of motion.

Radiographic Findings: Radiographs showed cranial and medial dislocation of the tibia (Figures 45a & b, page 88). The patella was luxated medially.

Diagnosis: Craniomedial dislocation of the right stifle joint.

Stifle Joint Injuries:

- Complete interstitial rupture of the cranial cruciate ligament
- Partial interstitial rupture of the caudal cruciate ligament
- Complete interstitial rupture of the medial collateral ligament
- Ligament-bone failure at the origin of the lateral collateral ligament
- Full thickness longitudinal tearing of the medial and lateral meniscus

Management: The cranial cruciate, medial, and lateral collateral ligaments were reconstructed as described. A single transcondylar screw and nut¹⁰ was used as an anchor point for the suture at the origin of the medial and lateral collateral ligaments (Figures 46a & b, page 88). The axial portions of the medial and lateral menisci were removed.

Complications: Three days postoperatively the distal femur fractured at the site of the staple used to secure the fascial graft. The staple was removed and the fascial graft was sutured to the lateral femorofabellar ligament. The fracture was reduced and stabilised with a dynamic compression plate.

¹⁰3.5 mm lockable nut, Veterinary Instrumentation, 62 Cemetery Road, Sheffield, S11 8FP, UK.



a.

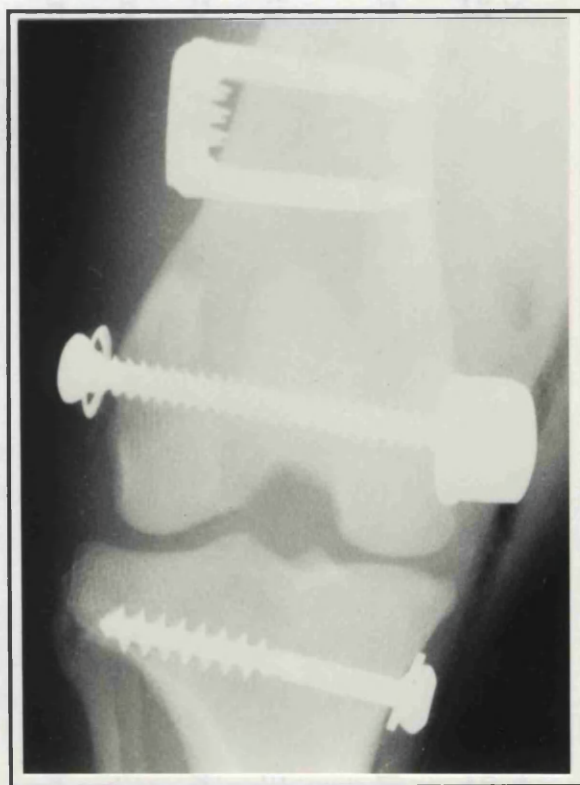


b.

Figures 46a & b Case 11. Preoperative lateral and craniocaudal radiographs of the right stifle joint.



a.



b.

Figures 47a & b Case 11. Immediate postoperative lateral and craniocaudal radiographs of the right stifle joint. Note the hair-line fracture present at the proximal leg of the staple.

Table 2: SUMMARY OF PATIENT DATA

Case No.	Age (years)	Sex	Breed	Weight (kg)	Source of Trauma	Structures of Stifle Damaged	Injury-Surgery interval (days)
1	2	F	Border collie	15.4	Stepped in a hole	CrCL, CaCL, MCL, MM.	2
2	6	M	Border collie	21.7	Tumbled down a bank	CrCL, MCL, MM, LM.	7
3	8	F	Border collie	15.5	Caught in a fence	CrCL, CaCL, LCL, PT, MM.	19
4	7	M	Labrador cross	30	Caught in a gate	CrCL, CaCL, LCL, MM.	3
5	1.5	M	Cross breed	15.5	Road traffic accident	CrCL, CaCL, LCL.	1
6	9	M	Labrador retriever	29	Caught in a gate	CrCL, CaCL, LCL, LJC.	3
7	4.5	M	Labrador retriever	25	Caught in a fence	CrCL, CaCL, LCL, LM, LJC.	15
8	3	M	Golden retriever	36	Unknown	CrCL, CaCL, MCL, LCL, LM.	1
9	4	M	Rottweiler	44	Unknown	CrCL, CaCL, MCL.	6
10	3	M	Border collie	20.5	Gathering sheep at speed	CrCL, CaCL, MCL, MM, LM, CaJC, AF.	1
11	10	M	Lurcher	14.5	Caught in a fence	CrCL, CaCL, MCL, LCL, MM, LM.	10

CrCL = Cranial cruciate ligament; CaCL = Caudal cruciate ligament; MCL = Medial collateral ligament; LCL = Lateral collateral ligament; MM = Medial meniscus; LM = Lateral meniscus; AF = Articular fracture; LJC = Lateral joint capsule; CaJC = Caudal joint capsule; PT = Popliteal tendon.

SECTION 3: PART 3

RESULTS

PREOPERATIVE AND OPERATIVE DATA

Incidence and Prevalence

A total of 224 dogs were referred to the University of Glasgow Veterinary School from October 1992 to August 1995 for the treatment of stifle instability, secondary to ligament injury. Of these cases, 212 were restricted to isolated injury of the cranial cruciate ligament. Twelve cases (prevalence = 5.4%) involved injury to two or more ligaments of the stifle joint (annual incidence = 4.2 cases). The author was solely responsible for the management of 11 cases which form the basis for this study.

Patient Data

The mean age of the patients was 5.3 years (range 1.5 to 10 years), body weight was 24.3 kg (range 14.5 to 44 kg) and the male:female ratio was 9:2. There were four Border collies, two Labrador retrievers, one Golden retriever, one Rottweiler, one Lurcher, and one cross breed affected. Five dogs caught the limb when jumping over a fence or gate, three dogs injured the limb while working with sheep and one dog was involved in a road traffic accident. In two dogs, there was no known traumatic incident, and injuries to the ligaments was considered to be secondary to stifle joint infections. The mean injury to surgery interval was 6.2 days (range 1 to 19 days). The left hind limb was affected in six dogs and right hind limb in five.

Clinical Findings

All dogs were lame at presentation, bearing little or no weight on the affected limb. In acute (< 11 days old) traumatic injuries there was soft tissue swelling surrounding the stifle joint and sometimes bruising was apparent. Manipulation of the stifle joint was intensely painful, particularly at the extremes of joint motion, and normal stifle range of motion was always reduced. Joint instability was obvious and present in more than one plane, depending on the ligamentous injury.

Similar findings were present with older injuries; however, soft tissue swelling had subsided and the peri-articular tissues appeared to be thickened. Stifle joint effusion was detectable as well as atrophy of the ipsilateral quadriceps muscle.

Only one dog, which was involved in the road traffic accident, had other orthopaedic injuries (coxofemoral dislocation).

Radiographic Findings

Preoperative lateral and craniocaudal radiographic scores are listed in Tables 6 & 7, pages 98 & 99. In the traumatic cases preoperative radiographic scores were low; any significant radiographic findings were confined to the soft tissues. The two infected stifle joints showed moderate radiographic signs of osteoarthritis.

Stressed craniocaudal radiographs always demonstrated valgus and/or varus subluxation, depending on the collateral ligament injured. There was radiographic evidence of ligament-bone avulsion in two cases (case 3 and 5), and an articular fracture was present in one dog (case 10).

Surgical Findings

Combinations of ligament injuries

In total, there were four different combinations of ligament injuries. All cases involved the cranial cruciate ligament and in ten cases both the cranial and caudal cruciate ligaments were involved. The most common combination injury, which occurred in five cases, was that involving both cruciate ligaments and the lateral collateral ligament. Other combinations of ligament injuries were: two cases of both cruciate and collateral ligaments; three cases of both cruciate ligaments and the medial collateral ligament; and in a single case, the cranial cruciate and medial collateral ligaments.

Modes of ligament failure

Cranial Cruciate Ligament

There were eight complete interstitial ruptures of the cranial cruciate ligament and two partial ruptures. In two cases, the fascial graft that had been used for prior reconstruction of the cruciate ligament was no longer present.

Caudal Cruciate Ligament

There were seven complete interstitial ruptures of the caudal cruciate ligament and three partial ruptures.

Collateral ligaments

There were four complete interstitial ruptures of the collateral ligaments and three partial ruptures. Ligament-bone failure was seen in four cases and ligament-bone avulsion in two.

Meniscal injuries

In eight cases there was injury to the menisci. The medial meniscus was injured in three cases, the lateral meniscus in two, and both menisci were injured in three cases. The most common meniscal injury was the peripheral avulsion injury which occurred alone five times and together with crushing injuries four times. Longitudinal tears occurred in two menisci. In one case there was rupture of the cranial meniscotibial and intermeniscal ligaments.

Joint capsule rupture

Full thickness tears in the joint capsule were observed in three cases.

POSTOPERATIVE DATA

EARLY POSTOPERATIVE EXAMINATION

All dogs, except case 6, were available for reassessment from six to 12 weeks postoperatively (mean interval = 8.4 weeks). All were lame at this examination; however, in some cases this was only just detectable. Any instability present was detectable in the craniocaudal plane and was minimal except in case 10 where the joint was found to be very unstable. Each case showed varying degrees of stifle joint thickening and mild joint effusion. Manipulation of the joint was painless in all but one case (case 10) and crepitation was evident in six out of the ten cases. A summary of these findings is presented in Table 3, page 93.

Synovial fluid was aspirated from the stifle joints of cases 8 and 9. No bacteria were isolated from these samples and the cells present were of low numbers and predominantly mononuclear in nature. Cell counts were $3.4 \times 10^9 \text{ l}^{-1}$ and $11.4 \times 10^9 \text{ l}^{-1}$ respectively. Synovial fluid analysis was repeated in case 9 six weeks later and the cell count had fallen to $2.4 \times 10^9 \text{ l}^{-1}$. Radiographs of the stifle joint of case 9 taken at this time revealed areas of lucency in the bone surrounding the implants (Figure 48, page 94). The implants and sutures were removed and bacterial culture isolated gram positive branching rods. As a result of sensitivity testing, the dog was placed on a six week course of ampicillin tablets (Amfipen, Mycofarm UK Ltd.¹¹) at a dose of 10 mg/kg bid. The implants in case 8 were removed 13 months postoperatively after the development of an intermittent lameness of three months duration. *Staphylococcus* organisms were isolated from the implants and these were sensitive *in vitro* to ampicillin. A four week course of ampicillin tablets at a dose of 10 mg/kg bid was prescribed. In both cases the polyester suture had broken at the time of removal and was imbedded in fibrous tissue. There was no palpable decrease in joint stability after the implants and sutures were removed. Both dogs began to use the limb in a normal manner four weeks later.

The poor results achieved in case 10 necessitated further surgery and the owner elected for amputation of the limb. Examination of the stifle joint revealed caudomedial dislocation of the tibia with extensive damage to the articular cartilage on the lateral femoral condyle as a result of abrasion on the screw and K wire. The intermeniscal ligament repair had failed and the menisci were crushed and displaced caudally in the joint. The dog made an uneventful recovery following amputation of the limb and was not included for long-term evaluation.

¹¹Mycofarm Uk Ltd., Science Park, Milton Road, Cambridge, CB4 4FP, UK.

Table 3: SUMMARY OF FINDINGS AT EARLY POSTOPERATIVE EXAMINATION

Case No	Postoperative Period (weeks)	Lameness Assessment	Cr/Ca	Instability Valgus/Varus	Thickening	Effusion	Pain	Crepitation
1	7	2/10	0	0	Mild	Mild	Absent	Absent
2	6	5/10	0	0	Moderate	Mild	Absent	Absent
3	7	3/10	0	0	Moderate	Mild	Absent	Present
4	11	2/10	1	0	Mild	Mild	Absent	Absent
5	12	1/10	1	0	Mild	Mild	Absent	Present
6	Not Available							
7	8	3/10	1	0	Moderate	Mild	Absent	Absent
8	7	3/10	1	0	Moderate	Mild	Absent	Present
9	6	4/10	1	0	Moderate	Moderate	Absent	Present
10	8	8/10	3	2	Severe	Moderate	Present	Present
11	12	1/10	0	0	Moderate	Mild	Absent	Present

Lameness: Graduated scale where; 0/10 = completely sound at the walk and trot, 10/10 = continuous non-weight bearing lameness.

Instability: 0 = stable, 1 = 1-3mm movement, 2 = 4-6mm movement, 3 = 7-9mm movement.

Mild = only just detectable, Moderate = easily detectable, Severe = obvious over the whole of the joint.

LONG-TERM POSTOPERATIVE EXAMINATION

Long-term postoperative examinations were carried out from 23 to 91 weeks postoperatively (mean 47.4 weeks) and all the dogs achieved a functional rating by the owner of good to excellent at this time (Table 4, page 96).

Case 11 was presented lame at long-term examination after fracturing its femur. This fracture occurred at the proximal end of the shaft, area previously repaired. The fracture occurred 10 weeks postoperatively, to repair the fracture was done. The fracture was comminuted, unilateral, extensional external rotation, and displaced. The femoral head and neck were intact. Further information on this case is given in Table 4.

Stifle joint examination was carried out on 11 dogs. The mean age was 10.5 years. The mean weight was 20.5 kg. The mean time postoperatively was 47.4 weeks. The mean time postoperatively was 47.4 weeks.

There was no significant difference in thigh muscle mass between the injured and uninjured sides (orthopedic and orthopedic, respectively) (Table 5, page 98).

No significant difference in joint flexion and joint extension was observed between the injured and uninjured sides (Table 5, page 98). There was a mean loss of 25 degrees of joint flexion and 14 degrees of joint extension in the injured side.



Radiographic scores for the stifle joints at long-term follow-up was significantly greater than preoperative radiographic scores ($P=0.004$) (Tables 6 & 7, pages 98 & 99).

Figure 48 Case 9. Twelve week postoperative craniocaudal radiograph of the left stifle joint. Note the areas of bone lucency around the implants.

There was no significant difference in the amount of bone lysis involving the apex and base of the patella, margins of the femoral trochlear groove, femoral and tibial condyles, and fabellae of the gastrocnemius muscle. Varying amounts of bone lysis was present beneath nearly all screws and washers used in collateral ligament repair. Radiographic evidence of implant loosening occurred in three cases (Figures 49 to 51, pages 100 and 101). Osteophyte formation at sites d and g, or a and v, was significantly greater ($P=0.02$) on the sides of the injured collateral ligaments when compared with the uninjured sides.

LONG-TERM POSTOPERATIVE EXAMINATION

Long-term postoperative examinations were carried out from 22 to 91 weeks postoperatively (mean 47.4 weeks) and all the dogs achieved a functional rating by the owner of good to excellent at this time (Table 4, page 96).

Case 11 was presented lame at long-term examination after fracturing its femur. This fracture occurred at the proximal end of the plate, used previously, to repair the fracture occurring at the staple. The plate was removed and the fracture was reduced and stabilised with an intramedullary pin and a two pin, unilateral, uniplaner, external fixator. Twelve weeks later the dog broke the proximal external fixator pin and bent the bar. Radiography at this time confirmed that the femoral fracture had healed and the remains of the fixator were removed. Further information on this dog was unobtainable.

Stifle joint instability was an unusual finding at long-term examination. A small amount (1 to 3 mm) of craniocaudal movement was present in three cases and this was only detectable after heavy sedation. Stifle joint thickening was present in all cases and crepitation was a feature of six cases .

There was no statistical difference found between affected and unaffected limb mid-thigh muscle mass circumferences ($P=0.13$). Cases 5 and 11 had the largest muscle mass differences; however, in each case, there was history of a concurrent orthopaedic condition affecting the limb (coxofemoral luxation and fractured femur respectively) (Table 5, page 97).

No significant reduction in stifle joint extension was observed between the affected and unaffected limbs ($P=0.09$); however, there was a significant reduction in stifle joint flexion ($P=0.03$). Nine cases maintained joint extension within five degrees, and joint flexion within ten degrees of the contralateral joint. In case 11 there was a loss of 25 degrees of motion in flexion, and 30 degrees of motion in extension (Table 5, page 97).

Radiographic scores for the stifle joints at long-term follow-up was significantly greater than preoperative radiographic scores ($P=0.004$) (Tables 6 & 7, pages 98 & 99). Abnormalities frequently observed on radiographs at long-term follow-up included: joint effusion, soft tissue thickening and osteophyte formation involving the apex and base of the patella, margins of the femoral trochlear groove, femoral and tibial condyles, and fabellae of the gastrocnemius muscle. Varying amounts of bone lysis was present beneath nearly all screws and washers used in collateral ligament repair. Radiographic evidence of implant loosening occurred in three cases (Figures 49 to 51, pages 100 and 101). Osteophyte formation at sites d and g, or a and e, was significantly greater ($P=0.02$) on the sides of the injured collateral ligaments when compared with the uninjured sides.

Table 4: SUMMARY OF FINDINGS AT LONG-TERM POSTOPERATIVE EXAMINATION

Case No	Postoperative Period (weeks)	Owner's Assessment	Lameness Assessment	Author's Cr/Ca	Instability Valgus/Varus	Thickening	Effusion	Pain	Crepitation
1	67.5	Excellent	0/10	0	0	Mild	Absent	Absent	Present
2	28	Excellent	0/10	0	0	Mild	Absent	Absent	Present
3	27	Excellent	0/10	0	0	Moderate	Absent	Absent	Absent
4	32	Excellent	0/10	0	0	Mild	Absent	Absent	Absent
5	22	Excellent	1/10	1	0	Mild	Absent	Absent	Absent
6	91	Good	0/10	0	0	Moderate	Absent	Absent	Present
7	38	Excellent	0/10	1	0	Moderate	Absent	Absent	Present
8	57	Excellent	0/10	1	0	Mild	Absent	Absent	Present
9	78	Excellent	0/10	0	0	Moderate	Absent	Absent	Absent
10	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
11	33	Good	10/10	0	0	Moderate	Absent	Absent	Present

Lameness: Owner's Assessment; Excellent = no lameness under any circumstance, Good = transient lameness after exertion.

Author's Assessment; Graduated scale 0/10 = completely sound at the walk and trot, 10/10 = continuously non-weight bearing

Instability: 0 = stable, 1 = 1-3mm movement, 2 = 4-6mm movement, 3 = 7-9mm movement.

Mild = only just detectable, Moderate = easily detectable, Severe = obvious over all of the joint.

Table 5: SUMMARY OF FINDINGS AT LONG-TERM POSTOPERATIVE EXAMINATION contd.

Case No	Affected Limb	Mid-Thigh Muscle Mass Circumference (cm)			Range of Motion					
		Left	Right	Difference	Left	Difference	Left	Right	Difference	
1	Left	32	32	0	60	60	0	70	70	0
2	Left	35	35	0	50	50	0	65	65	0
3	Left	31	31	0	55	55	0	55	50	-5
4	Right	38	38	0	50	45	-5	55	55	0
5	Right	34	32	-2	60	55	-5	60	55	-5
6	Right	36	36	0	50	50	0	60	60	0
7	Right	33	32.5	-0.5	60	50	-10	50	45	-5
8	Left	41	41	0	50	55	-5	55	60	-5
9	Left	45	45	0	40	40	0	65	65	0
10	Left	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
11	Right	25*	23.5*	-1.5	60*	35*	-25	65*	35*	-30

*These measurements were made twelve weeks after femoral fracture fixation.

Table 6: PREOPERATIVE AND LONG-TERM POSTOPERATIVE LATERAL RADIOGRAPH SCORES

Case No	Time* (weeks)	Lateral Radiographic Site Scores									Total
		A	B	C	D	E	F	G	H	I	
1	0	0	1	0	0	0	0	0	2	2	5
	67.5	2	3	0	0	3	1	4	1	2	16
2	0	1	0	0	0	0	0	0	5	4	10
	28	3	3	1	1	2	3	3	2	4	22
3	0	0	1	0	0	2	1	0	3	4	11
	27	2	4	0	0	3	1	2	1	5	18
4	0	0	0	0	0	0	0	0	4	3	7
	32	2	3	0	1	3	4	5	3	5	26
5	0	0	0	0	2	0	0	0	4	3	9
	22	2	2	0	0	1	2	0	0	4	11
6	0	0	0	0	0	0	0	0	1	5	6
	91	2	2	0	0	2	2	2	2	5	17
7	0	0	0	0	0	0	0	0	4	5	9
	38	2	2	0	0	0	3	3	4	4	18
8	0	3	3	0	3	4	4	3	4	4	28
	57	3	3	1	3	3	3	4	2	5	27
9	0	1	2	0	2	2	2	2	5	5	21
	78	2	3	0	1	1	2	2	3	4	18
11	0	0	0	0	2	0	0	0	5	5	12
	33	0	3	0	2	1	0	2	2	4	14

*Time (in weeks) at which the radiograph was taken postoperatively, where time zero is immediately preoperatively.

Scoring key: 0=absent, 1=equivocal change, 2, 3, 4, 5=very advanced change.

See Figure 33, page 70 for illustration of radiographic scoring site position.

Table 7: PREOPERATIVE AND LONG-TERM POSTOPERATIVE CRANIOCAUDAL RADIOGRAPH SCORES

Case No	Time* (weeks)	Cranio-caudal Radiographic Site Scores									Total
		A	B	C	D	E	F	G	H	I	
1	0	1	0	0	0	0	0	0	2	4	7
	67.5	3	2	0	0	0	1	4	1	3	14
2	0	2	0	0	0	0	0	0	4	4	10
	28	2	0	0	4	0	1	3	4	2	16
3	0	2	2	0	1	0	0	1	4	3	13
	27	4	0	0	2	4	0	3	2	4	19
4	0	1	0	0	0	0	0	0	5	5	11
	32	0	0	0	0	3	0	4	5	2	14
5	0	0	0	0	0	0	0	0	3	1	4
	22	0	0	0	1	2	0	3	3	3	12
6	0	0	0	0	0	0	0	0	5	1	6
	91	3	0	0	2	3	2	2	3	1	16
7	0	0	0	0	0	0	0	0	3	4	7
	38	4	0	0	2	4	3	2	3	0	18
8	0	3	1	1	2	2	2	2	3	4	20
	57	5	0	1	2	5	2	3	3	3	24
9	0	2	0	0	1	0	0	0	4	5	12
	78	3	0	0	2	2	0	3	3	1	14
11	0	0	0	2	0	0	0	2	4	0	8
	33	3	0	1	2	2	0	3	3	1	15

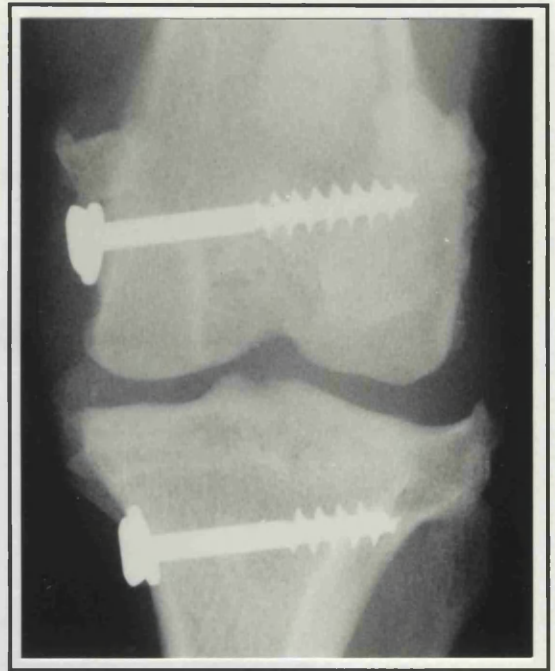
*Time (in weeks) at which the radiograph was taken postoperatively, where time zero is immediately preoperatively.

Scoring key: 0=absent, 1=equivocal change, 2, 3, 4, 5=very advanced change.

See Figure 34, page 71 for illustration of radiographic scoring site position.



a.



b.

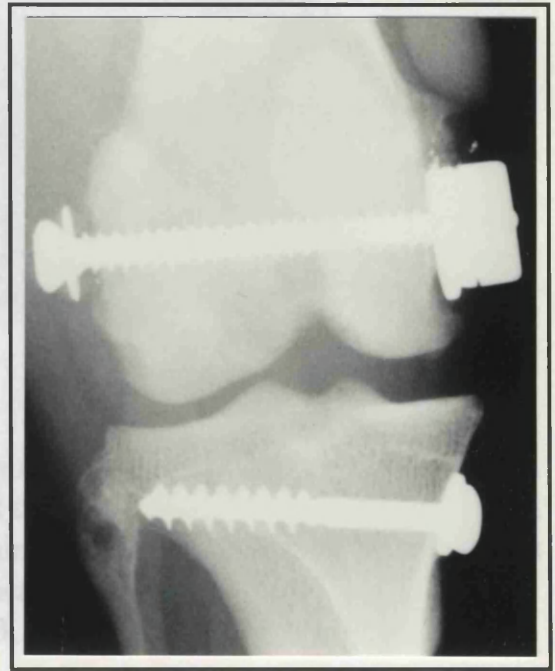
Figures 49a & b Case 1. Sixty-seven-and-a-half week postoperative lateral and craniocaudal radiographs of the left stifle joint. Note the focal bone lysis beneath the washer and the calcification of the medial meniscus.

Lateral scores: a=2, b=3, c=0, d=0, e=3, f=1, g=4, h=1, i=2, total=16.

Craniocaudal scores: a=3, b=2, c=0, d=0, e=0, f=1, g=4, h=1, i=3, total=14.



a.



b.

Figures 50a & b Case 11. Thirty-three week postoperative lateral and craniocaudal radiographs of the right stifle joint. Note the focal bone lysis at the fibular head and beneath the lateral washer.

Lateral scores: a=0, b=3, c=0, d=2, e=1, f=0, g=2, h=2, i=4, total=13.

Craniocaudal scores: a=3, b=0, c=1, d=2, e=2, f=0, g=3, h=3, i=1, total=13.

SECTION 3: PART 4

DISCUSSION

The information available in the present study corroborates earlier in the relatively scarce reports of multiple ligamentous injury of the canine knee joint. From 11 cases reported by Phillips (1982) and 11 cases reported by Phillips and others (1983) all having acute onset in the early or middle of life. The present study reports multiple ligamentous injury of the right stifle joint in a 3-year-old dog (Henry) in a traumatic accident on 10th October 1973. This low power injury was particularly severe (Phillips 1982).

The trauma was severe in this case sheep at speed accident. In sheep working on roads and the risk of injury is high. This may explain the delay in diagnosis.

All cases of multiple ligamentous injury of the stifle joint in the present series of acute traumatic multiple ligamentous injury were associated with one dog that was involved in a severe traffic accident.



Figure 51 Case 4. Thirty-two week craniocaudal radiograph of the right stifle joint. Note the excessive osteophyte formation and the mineralisation of peri-articular soft tissues. There is evidence of implant loosening as it is surrounded by a radiolucent halo and there is marked focal bone lysis beneath the washer.

Craniocaudal scores: a=0, b=0, c=0, d=0, e=3, f=0, g=4, h=5, i=2, total=14.

Two cases in this series developed a severe, acute onset, lameness without a history of trauma. Analysis of the synovial fluid, as well as the radiographic, gross and pathological findings, were suggestive of a chronic inflammatory process. However, no bacteria were cultured from either of the joints at the time of surgery, but organisms were isolated from the implants after their removal. Negative results following culture of synovial fluid or synovial membrane are not uncommon as the isolation of organisms from infected joints can be difficult (Montgomery and others

SECTION 3: PART 4

DISCUSSION

The information detailed in the present study contributes further to the relatively sparse reports of multiple ligamentous injury of the canine stifle joint. These 11 cases represent 30% of the total number of dogs in the reviewed literature recorded as having multiple ligamentous stifle joint injuries. The lack of reports call attention to the rarity of spontaneous rupture of two or more ligaments in the stifle joint of the dog (Denny and Minter 1973, Hulse and Shires 1986). The prevalence of multiple ligamentous injury of the stifle joint at the University of Glasgow Veterinary School from October 1992 to August 1995 was 5.4% with an annual incidence of 4.2 cases. This low prevalence is in keeping with other reports of 3% (Denny and Minter 1973) and 11% (Aron 1988). The annual incidence over the study period was particularly high compared with previous reports which ranged from 0.2 cases (Phillips 1982) to 2.7 cases (Hulse and Shires 1986).

The trauma necessary to produce multiple ligamentous injury of the stifle joint was severe in nine of the dogs in this series. These injuries occurred when working sheep at speed, after catching the limb in a fence or gate, or following a road traffic accident. In six cases the injury was associated with work or sport related activities. Working or sporting dogs are asked to perform at higher levels of physical activity and the risk of becoming entangled in a fence is greater than that of a household pet. This may explain the high prevalence of this condition affecting the Border collie in this series as it is the most common sheep dog breed in Scotland.

All cases of multiple ligamentous stifle injury reported in the literature occurred secondary to severe trauma, such as road traffic accidents or falls from heights. Associated with this trauma was a high prevalence (38.5%) of concurrent musculoskeletal injuries distant to the affected stifle (Denny and Minter 1973, Hulse and Shires 1986, Aron 1988). In the present series of cases, concurrent musculoskeletal injury occurred only in one dog that was involved in a road traffic accident. It can be speculated that the reason for the low prevalence of concurrent injuries in this series of cases was due to the nature of the trauma. Presumably, catching the limb in a fence or gate, or stepping in a hole at speed, resulted in large extrinsic forces imparted directly to the stifle joint and confined the injury to this area alone.

Two cases in this series developed a severe, acute onset, lameness without a history of trauma. Analysis of the synovial fluid, as well as the radiographic, gross, and pathological findings, were suggestive of a chronic inflammatory process. However, no bacteria were cultured from either of the joints at the time of surgery, but organisms were isolated from the implants after their removal. Negative results following culture of synovial fluid or synovial membrane are not uncommon as the isolation of organisms from infected joints can be difficult (Montgomery and others

1989). The owners of the dogs were not aware of any ongoing problem with the stifle joints and considered that each dog had made a full recovery from the previous stifle surgery. The lack of any detectable clinical symptoms in these cases is somewhat surprising; however, it has been reported that some joint infections can produce a low-grade pathology that causes very mild symptoms (Bennett and Taylor 1988). It is proposed that the injury in these two cases resulted from the enzymatic action of chronic low-grade infections, which digested the fascial grafts and weakened other supporting structures, so that relatively mild trauma was sufficient to cause multiple ligamentous injury.

Dislocation of the stifle joint in the dog is reported to be a very infrequent injury (Nunamaker 1985). Two dislocations occurred in this series: a caudomedial and a craniomedial dislocation. The remaining cases presented as grossly unstable subluxated stifle joints. However, many of these could be dislocated under general anaesthesia when the patella was manually luxated away from the side of the injured collateral ligament. The author concluded that in the conscious patient where there is severe disruption to the primary and secondary joint restraints, stifle joint dislocation is prevented by muscle tone and the presence of the patella within the trochlear groove. Cranial and lateral dislocations are most frequently reported as the quadriceps, hamstring, gastrocnemius, and popliteus muscles and tendons resist caudal and medial displacement of the tibia (Aron 1988). There have been no other reports of caudal dislocation of the stifle joint in the veterinary literature. Experimental studies in man have demonstrated that caudal knee dislocations are difficult to reproduce and require greater force than other knee dislocations (Kennedy 1963). In this case, the trauma required to caudally dislocate the stifle resulted in an articular fracture of the craniolateral tibial plateau as well as extensive damage to many of the femorotibial and meniscal ligaments and the caudal joint capsule.

Vascular and nerve injuries are commonly reported following dislocation of the knee in man (Kennedy 1963). These complications have not been recorded in the dog and were not observed in the two cases of stifle dislocation in this series. Aron (1988) attributed the lack of vascular injury following dislocation of the stifle joint in dogs and cats to a well developed collateral circulation around the stifle joint.

Clinical and radiographic assessment of ligament damage was sometimes confusing. This was especially true of the two cases with concurrent joint infections as there was a generalised increase in joint laxity due to stretching of the primary and secondary joint restraints. As a result, surgical exploration was necessary for confirmation of the specific ligament injury. In other cases, it was sometimes difficult, preoperatively, to distinguish partial cruciate or collateral ligament rupture from complete rupture. Identification of complete collateral ligament rupture was easiest when there was complete rupture of the secondary joint restraints or where there was radiographic evidence of ligament-bone avulsion. Others have reported difficulties in identifying isolated and combined ruptures of the cranial and caudal cruciate ligaments (Hulse and Shires 1986, Aron 1988) as well as the accurate

preoperative assessment of collateral ligaments (Aron 1988). Interpretation of the findings at surgery can also be difficult, and Noyes and others (1984) demonstrated that a ligament can appear grossly intact while having lost load carrying ability. These findings have lead one author to adopt the surgical philosophy that all ligaments should be inspected directly whilst undergoing stress palpation (Aron 1988).

In the present series of cases, the most common combination of ligament injuries was rupture of the cranial and caudal cruciate ligaments, and the lateral collateral ligament. There has been only one other report of this combination injury affecting a dog (Aron 1988) and the most frequent occurrence reported in the literature is damage to the cranial and caudal cruciate ligament, and the medial collateral ligament. It has been speculated that medial restraint injuries are more common because the point of impact of trauma is usually on the lateral aspect of the stifle joint; this generates large tensile forces medially (Hulse and Shires 1986). A lateral restraint injury occurred in all cases of the present series where the limb had become caught in a fence or gate. One can speculate that these lateral restraint injuries may have been due to the distal limb having become caught whilst the dog still had sufficient momentum to clear the obstacle and begin falling down the other side. The body weight of the dog falling exerted severe torsional and compressive forces medially, which generated large tensile and shear forces on the lateral aspect of the stifle joint causing failure of the primary and secondary lateral restraints.

The failure mode of the cruciate ligaments was always interstitial. The majority of the collateral ligament failures were also interstitial; however, there were four instances of ligament-bone failure and two ligament-bone avulsions. The differences in the modes of failure is related to the viscoelastic properties of the ligaments and bone insertion sites. Experimental tests have shown that ligaments are not strain-rate sensitive and there is a predominance of interstitial failures when high loading rates, typical of *in vivo* trauma, are applied. Bone, however, is more strain-rate sensitive and fails at a lower load when the strain-rate is slow (Noyes and others 1974). As a result, there is a higher incidence of ligament-bone avulsion failures at slower strain-rates. True ligament-bone failures are rare and are frequently accompanied by the two other modes of failure (Noyes and others 1974). It was not possible to correlate the type of trauma and rate of loading with the ligament-bone avulsions and failure modes observed in this series of cases.

Two methods of cranial cruciate ligament reconstruction were used in this series of cases. Similar clinical results have been reported for each technique when used to repair isolated cruciate ligament ruptures (Hulse and others 1980, Flo 1975) and in combination ligament injuries (Hulse and Shires 1986, Aron 1988). Using the parameters evaluated in this study there was no difference in outcome observed between the two methods. The intra-capsular method was not used in two cases that had had previous cruciate ligament reconstruction as in such cases the author finds it difficult to harvest the fascial graft due to scar tissue formation and

adhesions. In addition, an extra-capsular method was chosen so that autogenous grafts or implants were not placed within the infected environment of the joint.

The fascial graft used to reconstruct the cruciate ligament was sutured to the lateral femorofabellar ligament and lateral periosteum of the femoral condyle or, alternatively, secured to the lateral femoral condyle using a staple or screw and spiked washer. It has been shown, experimentally, that a screw and spiked washer placed away from the joint into cortical bone has significantly greater holding strength than other fixation methods (Amis 1988). Using the parameters evaluated in this study there was no difference in the outcome observed between the different methods of graft fixation. However, a femoral fracture occurred in case 11 which was a direct complication of staple fixation. Small fracture lines at the staple were seen on radiographs taken immediately postoperatively (Figures 47a & b, page 88) and it is proposed that these acted as stress risers that resulted in complete fracture a few days later. This case highlights a potential complication of driving a staple into cortical bone, stressing the importance of taking postoperative radiographs. The fracture may have been prevented by pre-drilling holes for the legs of the staple using a smaller diameter drill bit.

The importance of a thorough inspection of the joint and identification of meniscal and joint capsule injuries cannot be overemphasised. Wherever possible, the menisci should be preserved and peripheral meniscal injuries and joint capsule meticulously reconstructed. The author believes that this is one of the most important factors in re-establishing stifle joint stability and contributes significantly to the eventual outcome. Other reports have also stressed the importance of repairing meniscal injuries (Hulse and Shires 1986, Smith 1995). During reconstruction of the collateral ligaments, the menisci were used to centre the convex femoral condyles on the convex tibial plateau. It is important that this part of the procedure is performed prior to securing the cruciate ligament fascial graft, thereby avoiding over tightening of the graft and caudal subluxation of the joint.

Damage to the menisci occurred frequently in this series of cases; peripheral avulsion injuries being the most common. This type of injury is considered to be rare and usually only occurs following multiple ligamentous injury to the stifle joint (Flo and others 1983). Total meniscectomy was performed in only one case; all other injuries were amenable to primary repair or partial meniscectomy. This is somewhat surprising considering the severe nature of the stifle joint derangement, although it is possible that peripheral avulsion of the meniscus allows it to move and avoid further injury. Similar observations have been made with stifle subluxation secondary to cranial cruciate ligament rupture, where the more mobile lateral meniscus has a much lower incidence of injury when compared to the relatively fixed medial meniscus (Flo and DeYoung 1978).

Many of the complications that occurred in case 10 were attributed to the inadequate management of the meniscal and joint capsule injuries. Radiographs taken immediately after the repair (Figures 44a & b, page 86) showed that the tibia was in

a caudally subluxated position. It can be speculated that this was due to inadequate imbrication of the caudal joint capsule and over tightening of the fascial graft. With the stifle subluxated caudally, excessive stress was placed on the tenuous meniscal ligament and peripheral meniscal avulsion repairs; their failure resulted in complete caudal dislocation of the joint. For severe meniscal injuries, such as those seen in this case, the author would recommend immobilisation of the stifle joint in an anatomically aligned weight-bearing position for a period of four to six weeks postoperatively using a trans-articular external fixator.

Difficulty was sometimes encountered in identifying the correct position for femoral screw placement at the origin of the damaged collateral ligament. There was a tendency to place the screw proximal to the centre of rotation of the condyle where the ligament was observed to insert on the bone. Accurate placement of the femoral screw is essential to prevent non-isometric (binding) motion of the joint resulting in limited range of motion and premature failure of fixation (Smith 1995). The author found it easiest to make a small incision in the joint capsule caudal to the collateral ligament so that the curvature of the femoral condyle could be visualised, and the position of the centre of rotation estimated.

Partially threaded cancellous screws were preferred to cortical screws as they have superior security in the cancellous bone; their smooth shaft reduced the risk of damaging the suture as the screw was tightened. Screw length varied from penetration of the width of the bone, to penetrating the width of a single condyle. There was no evidence of implant failure when short unicondylar screws were used. Accurate placement of a single trans-condylar screw, as used in case 11, to repair medial and lateral collateral ligament ruptures is difficult. An easier and more accurate alternative would be to place a unicondylar screw from the lateral and medial surfaces as described.

The polyester suture material used to augment or reconstruct the damaged collateral ligaments in this series was well tolerated by the tissues. Polyester is one of the strongest non-metallic suture materials available (Holt and Holt 1981) and loses little or no tensile strength after implantation in tissues (Stashak and Yurraspe 1978). It can therefore offer prolonged support for slow healing tissues such as ligaments. Polyester suture material has poor knot security (Stashak and Yurraspe 1978) and a five-throw knot with the ends sutured to the surrounding tissues was utilised to improve security. Soft tissue irritation, as a result of movement of the knot during stifle motion, was minimised by placing it as near to the distal screw as possible. In addition, removal of the suture was easy with the knot in this position and required minimal disruption to the healing ligament.

The use of polyester sutures in contaminated or infected wounds has been associated with persistent local infection and exaggerated tissue reaction (Varma and others 1981). Bacteria hide in the interstices of the suture and multiply beyond the reach of the body's defences (James and MacLeod 1961) and the accompanying inflammatory reaction can delay healing, lead to disruption of collagen fibres, and

weaken the surrounding tissues (Everett 1970). Persistent local infection necessitated the removal of the metal implants and sutures in cases 8 and 9. In case 9, there was radiographic evidence of areas of bone lucency surrounding the screw threads and case 8 developed an intermittent lameness from ten months postoperatively. Removal of the implants and appropriate antibiotic therapy resulted in an uneventful recovery in both cases. Monofilament stainless steel wire may have been a better choice of suture material to use in the infected wounds as it has high tensile strength and does not support infection (Swaim 1980).

Varying amounts of radiographic bony lysis was seen locally beneath all screws and washers at long-term follow-up, and was attributed to local tissue reaction due to mechanical irritation from the suture material. Similar findings of bony lysis have been reported to occur beneath screws and spiked washers used to secure fascial grafts in cruciate ligament reconstruction (Elkins and others 1991). In these cases implant removal was recommended as a preventative measure from four to six months postoperatively. There were no clinical indications in the present series to warrant implant removal other than in the infected joints. However, the author would recommend implant removal should a problem arise. The implants in case 9 were removed as early as three months postoperatively without loss of stifle stability.

The surgical method employed in the ten cases of this series that were followed long-term produced good to excellent results for limb function. These results compare favourably with those reported in the literature for other intra-articular (Hulse and Shires 1986), trans-articular (Welches and Scavelli 1990) and extra-articular (Aron 1988) techniques. It has been proposed that the good results achieved by a variety of methods supports the notion that any technique which adequately maintains reduction and stability during the collagen repair stages of healing can be clinically successful (Aron 1988). However, Hulse and Shires (1986) suggest that the proliferation of the peri-articular soft tissues is not the primary stabilising factor and that careful reconstruction of both the primary and secondary joint restraints is necessary for successful treatment of stifle joint derangement. Aron (1988) proposed that the pronounced collagen repair which occurs secondary to the extensive soft tissue damage is strong enough to provide long-term joint stability.

The success of the surgical method employed in the present study suggests that adequate stifle stability is achieved long-term without reconstruction of the caudal cruciate ligament. It has been reported that surgical repair of isolated caudal cruciate ligament ruptures is unnecessary (Pearson 1971, Harari and others 1987). Others have advocated the repair of only the cranial cruciate ligament when both cruciate ligaments have been injured (Nunamaker 1985, Denny and Minter 1973). When the caudal cruciate ligament is not reconstructed, it is the secondary joint restraints and the action of muscles and tendons which prevent caudal translation of the tibia prior to the formation of peri-articular fibrous tissue. On the basis of the outcome of case 10, the author proposes that where there is severe disruption of the

secondary joint restraints, particularly the caudal joint capsule and the menisci, additional support is required to prevent caudal translation of the tibia. This could be achieved by using extra-articular sutures or a trans-articular external fixator for four to six weeks, as described by Aron (1988). Similarly, it can be speculated that poor results may be achieved if this technique is used alone in cases where medial and lateral meniscectomy is performed. Meniscectomy produces high stress levels during weight bearing at the area of cartilage contact and, in combination with ligamentous deficiencies, it necessitates more extensive surgical reconstruction to achieve adequate stability (Smith 1995).

Loss of stifle joint range of motion, particularly flexion, has been a consistent finding in three different series of dogs with multiple ligamentous stifle injuries at long-term follow-up (Aron 1988, Hulse and Shires 1986, Welches and Scavelli 1990). The methods employed for joint stabilisation were different in each series and the cases consisted of a mixture of subluxated and dislocated stifle joints. Reduction in range of motion was considered to be secondary to peri-articular joint capsule fibrosis (Hulse and Shires 1986). Aron (1988) proposed that loss of flexion was more common because the animals used the limb more in full extension than in extreme flexion in the early collagen remodelling phase of healing. The results of the present study show minimal reduction, if any, in range of motion in nine out of ten cases. The author proposes that the comparatively favourable results with this technique were due to early postoperative mobilisation and physiotherapy. In all previous reports, stifle joints were immobilised immediately postoperatively for periods of three to eight weeks. Experimental studies in dogs have shown that early mobilisation after surgical repair of multiple stifle ligaments does not compromise ligament healing or result in undue ligament laxity. In fact, the mobilised stifles were found to be more stable and the medial collateral ligaments stronger (Piper and Whiteside 1980). Other studies have shown that early mobilisation after surgical repair of multiple stifle ligaments in dogs results in significantly less osteoarthritic change than with limited mobilisation and immobilisation (Ogata and others 1980).

The development of mild to moderate radiographic changes at long-term follow-up was noted in the present series of dogs, as well as those reported previously (Aron 1988, Hulse and Shires 1986, Welches and Scavelli 1990). Some authors feel that the peri-articular bone formation is due to trauma to the joint capsule (Hulse and Shires 1986), whilst others consider that trauma and abnormal joint biomechanics contribute to the changes seen on radiographs (Aron 1988). It is interesting to note that in the present series of cases there was a significant correlation between injury to the collateral ligament and increased local peri-articular new bone formation. The reason for the new bone formation in this situation, in the absence of instability, is likely to be due to periosteal irritation from trauma to the joint capsule at the time of injury, or persistent irritation from the polyester suture.

SECTION 3: PART 5

CONCLUSION

Multiple ligamentous injuries of the canine stifle joint is an uncommon condition. In this study, it affected mainly adult, male, working or sporting dogs and occurred secondary to severe trauma, or where chronic joint infection had weakened the supporting structures.

The surgical technique employed in the present series of cases is an effective method of reconstructing the canine stifle following multiple ligamentous injuries. It was found to be expeditious, technically easy to perform, and was adaptable for use in both small and large-size dogs. To achieve good results, precise reconstruction of collateral and cranial cruciate ligaments as well as careful evaluation and repair of meniscal and joint capsule injuries was necessary. Primary reconstruction of the caudal cruciate ligament was not found to be essential.

Intra-capsular or extra-capsular methods may be used in cranial cruciate ligament reconstruction. Augmentation of primary collateral ligament repair is recommended using the screw and figure-8 suture technique. Partially threaded cancellous screws that penetrate the width of a single condyle are recommended and care must be taken to position the femoral screw as closely as possible to the centre of rotation. Polyester suture material provides excellent stability, however, it is important to ensure good knot security. These sutures, when placed in an infected wound, may harbour infection and therefore require later removal. Under these circumstances, stainless steel wire may be a more suitable suture material. Implant removal after three months did not appear to affect stability.

Postoperative immobilisation of the stifle joint was found to be unnecessary. Early controlled exercise and physiotherapy prevented joint stiffness and maintained near normal range of motion in many of the cases postoperatively. Additional postoperative support may be required in some cases to prevent caudal tibial translation (where there is extensive disruption to the caudal joint capsule), or to protect tenuous meniscal repairs.

Radiographic signs of osteoarthritis developed postoperatively in all cases. Lateral or medial osteophyte formation was greatest where there was injury to the respective collateral ligaments and may represent periosteal irritation due to trauma to the joint capsule at the time of injury, or persistent irritation from the implants.

APPENDIX 1

SEDATION

All dogs were sedated with a mixture of intramuscular pethidine (Pethidine; Martindale Pharmaceuticals Ltd.¹²) at a dose of 2mg/kg and medetomidine (Domitor; Smithkline Beecham Animal Health¹³) at a dose of 10µg/kg. The effects of the medetomidine were later reversed with an intramuscular injection of atipamezole (Antisedan; Smithkline Beecham Animal Health¹⁴) at a dose of 50µg/kg.

GENERAL ANAESTHESIA

Premedication

All dogs were premedicated with a mixture of pethidine at a dose rate of 2mg/kg and acepromazine (ACP; C-Vet Limited¹⁵) at a dose rate of 0.03mg/kg given intramuscularly.

Induction

All dogs were induced with either propofol (Rapinovet; Coopers Animal Health Ltd¹⁶) or thiopentone (Intraval Sodium; Rhône Mérièux Ltd.¹⁷), depending on the anaesthetist's choice. The dose of intravenous propofol and thiopentone used was 4mg/kg and 10mg/kg respectively.

Maintenance

All cases were intubated and maintained on a gaseous mixture of halothane, nitrous oxide and oxygen. A Bain or Magill circuit was used with a fresh gas flow rate equal to the minute volume of respiration (approx. 200 ml/kg/min).

Fluids

Intravenous fluids were administered during all surgical procedures. Hartmans, or glucose saline solutions (Aqupharm; Animal Care Ltd.¹⁸) were delivered at rates of 5 to 10 ml/kg/hr. In cases where there was significant blood loss or a decrease in blood pressure then a colloid (Gelofusine Veterinary; B.Braun Medicals Ltd.¹⁹) was given to effect.

¹²Martindale Pharmaceuticals Ltd., Bampton Rd, Harold Hill, Romford, Essex, RM3 8UG, UK.

¹³Smithkline Beecham Animal Health, Walton Oats, Dorking Rd, Tadworth, Surrey, KT20 7NT, UK.

¹⁴Smithkline Beecham Animal Health, Walton Oats, Dorking Rd., Tadworth, Surrey, KT20 7NT, UK.

¹⁵C-Vet Ltd., Minster House, Western Way, Bury St, Edmunds, Suffolk, IP33 3SU, UK.

¹⁶Coopers Animal Health Ltd., Creive Hall, Crieve, Cheshire, CW1 1UB, UK.

¹⁷Rhône Mérièux Ltd., Harlow, Essex, CM19 5TS, UK.

¹⁸Animal Care Ltd., Lommon Rd., Dunnington, York, YO1 5RU, UK.

¹⁹B. Braun Medicals Ltd., Aylesbury, Bucks, HP20 1DQ, UK.

APPENDIX 2

ANALGESIA

All patients received analgesics from the time of premedication and in some cases analgesic treatment had already been given by the referring veterinary surgeon. Opioid analgesics were given intraoperatively approximately one hour after pethidine premedication. Papaveretum (Omnopon; Roche Products Ltd²⁰) was given intramuscularly at a dose of 0.3 mg/kg and this was repeated every 4 to 6 hours as required for 24hrs. An alternative to this regime was a single intravenous injection of carprofen (Zenecarp; C-Vet Ltd.²¹) given at a dose of 3 mg/kg. Twenty-four hours after surgery, all dogs were given carprofen tablets at a dose of 3 to 4 mg/kg given in 2 equally divided doses for seven days, and then half this dose once daily thereafter. The duration of treatment ranged from seven to fourteen days.

ANTIBIOTIC THERAPY

Unless otherwise stated antibiotics were administered perioperatively only. High doses (20 mg/kg) of either cephalexin (Ceporex; Pitman-Moore Ltd²²), or amoxicillin/clavulanic acid mixture (Synulox; Smithkline Beecham Animal Health²³) were given by intramuscular injection at the time of induction. A further two doses were given at 6 to 8 hour intervals.

²⁰Roche Products Ltd., Welwyn Garden City, Hertfordshire, AL7 3AY, UK.

²¹C-Vet Ltd., Minster House, Western Way, Bury St, Edmunds, Suffolk, IP33 3SU, UK.

²²Pitman-Moore Ltd., Harefield, Uxbridge, Middlesex, UB9 6LS, UK.

²³Smithkline Beecham Animal Health, Walton Oats, Dorking Rd, Tadworth, Surrey, KT20 7NT, UK.

REFERENCES

- AMIS, A.A. (1988) The strength of artificial ligament anchorages. A comparative experimental study. *Journal of Bone and Joint Surgery* **70-B**, 3, 397-403.
- ARNOCZKY, S.P., MARSHALL, J.L. (1977) The cruciate ligaments of the canine stifle: An anatomical and functional analysis. *American Journal of Veterinary Research* **38**, 11, 1807-1814.
- ARNOCZKY, S.P., TORZILLI, P.A., MARSHALL, J.L. (1977) Biomechanical evaluation of anterior cruciate ligament repair in the dog: An analysis of the instant centre of motion. *Journal of the American Animal Hospital Association* **13**, 553-558.
- ARNOCZKY, S.P., LANE, J.M., MARSHALL, J.L., and others. (1979)¹ Meniscal degeneration due to knee instability: An experimental study in the dog. *Transactions of the Orthopaedic Research Society* **4**, 79-84.
- ARNOCZKY, S.P., TARVIN, G.B., MARSHALL, J.L. and others (1979)² The over-the-top procedure: A technique for anterior cruciate substitution in the dog. *Journal of the American Animal Hospital Association* **15**, 283-290.
- ARNOCZKY, S.P., RUBIN, R.M., MARSHALL, J.L. (1979)³ Microvasculature of the cruciate ligaments and its response to injury. *Journal of Bone and Joint Surgery* **61-A**, 8, 1221-1229.
- ARNOCZKY, S.P. (1980) Surgery of the stifle - The cruciate ligaments (Part 1). *The Compendium on Continuing Education* **II**, 2, 106-115.
- ARNOCZKY, S.P., TARVIN, G.B., VASSEUR, P. (1980) Surgery of the stifle - The menisci and collateral ligaments (Part III). *The Compendium on Continuing Education* **II**, 2, 394-399.
- ARNOCZKY, S.P., MARSHALL, J.L. (1981) Pathomechanics of Cruciate and Meniscal Injuries. In: Bojrab, M.J. (Ed) *Pathophysiology in Small Animal Surgery*. Lea and Febiger, Philadelphia. pp 509-603.
- ARNOCZKY, S.P., WARREN, R.F. (1983) The microvasculature of the meniscus and its response to injury. An experimental study in the dog. *American Journal of Sports Medicine* **11**, 131-141.
- ARNOCZKY, S.P. (1985) Cruciate ligament rupture and associated injuries. In: Newton, C.D., Nunamaker, D.M. (Eds) *Textbook Of Small Animal Orthopaedics*. J.B. Lippincott Co., Philadelphia. pp 923-947.

- ARNOCZKY, S.P., WARREN, R.F., MINEI, J.P. (1986) Replacement of the anterior cruciate ligament using a synthetic prosthesis. *American Journal of Sports Medicine* **14**, 1, 1-6.
- ARNOCZKY, S.P., WARREN, R.F. SPIVAK, J.M. (1988) Meniscal repair using an exogenous fibrin clot. An experimental study in dogs. *Journal of Bone and Joint Surgery* **70-A**, 8, 1209-1217.
- ARON, D.N. (1981) A new tendon stitch. *Journal of the American Animal Hospital Association* **17**, 587-591.
- ARON, D.N. (1988) Traumatic dislocation of the stifle joint: Treatment of 12 dogs and one cat. *Journal of the American Animal Hospital Association* **24**, 333-340.
- BALLMER, P.M., BALLMER, F.T., JAKOB, R.P. (1991) Reconstruction of the anterior cruciate ligament alone in the treatment of a combined instability with complete rupture of the medial collateral ligament. A prospective study. *Archives of Orthopaedic and Trauma Surgery* **110**, 139-141.
- BARBER, F.A., STONE, R.G. (1985) Meniscal repair. An arthroscopic technique. *Journal of Bone and Joint Surgery* **67-B**, 1, 39-41.
- BENNETT, D.B., TAYLOR, D.J. (1988) Bacterial infective arthritis in the dog. *Journal of Small Animal Practice* **29**, 207-230.
- BENNETT, D.B., TENNANT, B., LEWIS, and others (1988) A reappraisal of anterior cruciate ligament disease in the dog. *Journal of Small Animal Practice* **29**, 275-279.
- BENNETT, D.B. (1990) Joints and Joint Diseases. In: Whittick, W.G. (Ed) *Canine Orthopaedics* 2nd edn. Lea and Febiger, Philadelphia. pp **761-853**.
- BENNETT, D.B., MAY, C. (1991)¹ Meniscal damage associated with cruciate disease in the dog. *Journal of Small Animal Practice* **32**, 111-117.
- BENNETT, D.B., MAY, C. (1991)² An over-the-top with tunnel technique for repair of cranial cruciate ligament rupture. *Journal of Small Animal Practice* **32**, 103-110.
- BERG, R.J., EGGER, E.L. (1986) In vitro comparison of the three loop pulley and the locking loop suture patterns for repair of canine weight bearing tendons and collateral ligaments. *Veterinary Surgery* **15**, 1, 107-110.
- BRINKER, W.O., PIERMATTEI, D.L., FLO, G.L. (1990) Principles of Joint Surgery, Diagnosis and Treatment of Orthopaedic Conditions of the Hindlimb. In: *Handbook of Small Animal Orthopaedics and Fracture Treatment* 2nd edn. W.B.Saunders Company, Philadelphia. pp **312-470**.

- BUTLER, H.C. (1964) Teflon as a prosthetic ligament in repair of ruptured anterior cruciate ligaments. *American Journal of Veterinary Research* **25**, 104, 55-59.
- BUTLER, D.L., NOYES, F.R., GROOD, E.S. (1980) Ligamentous restraints to anterior-posterior drawer in the human knee. *Journal of Bone and Joint Surgery* **62-A**, 2, 259-269.
- CABAUD, H.E., RODKEY, W.G., FITZWATER, J.E. (1981) Medical meniscus repairs. An experimental and morphological study. *American Journal of Sports Medicine* **9**, 129-134.
- CLAYTON, M.L., WEIR, G.J. (1959) Experimental investigations of ligamentous healing. *American Journal of Surgery* **98**, 373-378.
- COFONE, M.A., SMITH, G.K., LENEHAN, T.M., NEWTON, C.D. (1992) Unilateral and bilateral stifle arthrodesis in eight dogs. *Veterinary Surgery* **21**, 4, 299-303.
- COOPER, R.R., MISOL, S. (1970) Tendon and ligament insertion. A light microscopic study. *Journal of Bone and Joint Surgery* **56-A**, 2, 236-253.
- COX, J.S. NYE, C.E., SHAEFER, W., and others. (1975) The degenerative effects of partial and total resection of the medial meniscus in dogs knees. *Clinical Orthopaedics and Related Research* **109**, 414-419.
- COX, J.S., CORDELL, L.D. (1977) The degenerative effects of medial meniscus tears in dogs' knees. *Clinical Orthopaedics and Related Research* **125**, 236-242.
- CRANE, S.W. (1983) Suture Materials. In: Bojrab, J.M. (Ed), *Current Techniques in Small Animal Surgery* 2nd edn. Lea and Febiger, Philadelphia. pp 3-6.
- DEANGELIS, M.P., BETTS, C.W. (1973) Posterior cruciate ligament rupture. *Journal of the American Animal Hospital Association* **9**, 447-451.
- DENNY, H.R., MINTER, H.M. (1973) Long term results of surgery of canine stifle disorders. *Journal of Small Animal Practice* **14**, 695-713.
- DENNY, H.R. (1980) *A Guide to Canine Orthopaedic Surgery*. Blackwell Scientific Publications, Oxford, UK. pp 163-164.
- DENNY, H.R., GOODSHIP, A.E (1980) Replacement of the anterior cruciate ligament with carbon fibre in the dog. *Journal of Small Animal Practice* **21**, 279-286.
- DEHAVEN, K.E., ARNOCKY, S.P. (1994) Meniscal repair. *Journal of Bone and Joint Surgery* **76-A**, 1, 140-151.

- DEYOUNG, D.J., FLO, G.L., TVEDTEN, H. (1980) Experimental medial meniscectomy in dogs undergoing cranial cruciate ligament repair. *Journal of the American Animal Hospital Association* **16**, 639-645.
- DICKINSON, C.R., NUNAMAKER, D.M. (1977) Repair of ruptured anterior cruciate ligament in the dog: Experience of 101 cases using a modified fascia strip technique. *Journal of the American Veterinary Medical Association* **170**, 8, 827-830.
- DUELAND, R. (1966) Recent techniques for reconstruction of the anterior cruciate ligament. *Journal of the American Animal Hospital Association* **2**, 2-8.
- DYCE, K.M., MERLIN, R.H.A., WADSWORTH, F.J. (1952) The clinical anatomy of the stifle joint. *The British Veterinary Journal* **108**, 346-353.
- EATON-WELLS, R. WHITTICK, W.G. (1990) Luxations. In: Wittick, W.G. (Ed) *Canine Orthopaedics* 2nd edn. Lea and Febiger, Philadelphia. pp **491-518**.
- EGGER, E.L. (1983) Collateral ligament injuries. In: Bojrab, J.M. (Ed), *Current Techniques in Small Animal Surgery* 2nd edn. Lea and Febiger, Philadelphia. pp **628-630**.
- ELKINS, A.D., PECHMAN, R., KEARNEY, M.T., HERRON, M. (1991) A retrospective study evaluating the degree of degenerative joint disease in the stifle joint of dogs following surgical repair of anterior cruciate ligament rupture. *Journal of the American Animal Hospital Association* **27**, 533-540.
- ELMER, R., MOSKOWITZ, R., FRANKEL, V. (1977) Meniscal regeneration and postmeniscectomy degenerative joint disease. *Clinical Orthopaedics and Related Research* **124**, 304-310.
- EVANS, H.E., CHRISTENSEN, G.C. (Eds), (1979) *Miller's Anatomy of the Dog* 2nd edn. W.B. Saunders and Co., Philadelphia.
- EVERETT, W.G. (1970) Suture materials in general surgery. *Progress in Surgery* **8**, 14-37.
- FARROW, C.S., NEWTON, C.D. (1985) Ligamentous injury In: Newton, C.D., Nunamaker, D.M. (Eds), *Textbook Of Small Animal Orthopaedics*. J.B. Lippincott Co., Philadelphia. pp **843-854**.
- FLO, G.L. (1975) Modification of the lateral retinacular imbrication technique for stabilising cruciate ligament injuries. *Journal of the American Animal Hospital Association* **11**, 570-576.

- FLO, G.L., DEYOUNG, D. (1978) Meniscal injuries and medial meniscectomy in the canine stifle. *Journal of the American Animal Hospital Association* **14**, 683-689.
- FLO, G.L. (1983) Classification of meniscal lesions in twenty-six consecutive canine meniscectomies. *Journal of the American Animal Hospital Association* **19**, 335-340.
- FLO, G.L., DEYOUNG, D., TVEDTEN, H.W., and others. (1983) Classification of meniscal injuries in the canine stifle based upon gross and pathologic appearance. *Journal of the American Animal Hospital Association* **19**, 325-334.
- FLO, G.L. (1993) Meniscal injuries. *Veterinary Clinics of North America: Small Animal Practice* **23**, 4, 831-843.
- GUPTA, B.N., BRINKER, W.O. (1969) Anterior cruciate ligament prosthesis in the dog. *Journal of the American Veterinary Medical Association* **154**, 1057-1061.
- HAMBERG, P., GILLQUIST, J., LYSHOLM, J. (1983) Suture of new and old peripheral meniscus tears. *Journal of Bone and Joint Surgery* **65-A**, 2, 193-197.
- HARARI, J., JOHNSON, A.L., STEIN, L.E., and others. (1987) Evaluation of experimental transection and partial excision of the caudal cruciate ligament in dogs. *Veterinary Surgery* **16**, 2, 151-154.
- HENDERSON, R.A., MILTON, J.L. (1978) The tibial compression mechanism: A diagnostic aid in stifle injuries. *Journal of the American Animal Hospital Association* **14**, 474-479.
- HENNING, C.E., LYNCH, M.A., CLARK, J.R. (1987) Vascularity for healing of meniscus repairs. *Arthroscopy* **3**, 13-18.
- HINKO, P.J. (1981) The use of a prosthetic ligament in repair of the torn anterior cruciate ligament in the dog. *Journal of the American Animal Hospital Association* **17**, 563-567.
- HOLT, G.R., HOLT, J.E. (1981) Suture materials and techniques. *Ear Nose and Throat Journal* **60**, 23-28.
- HORIBE, S., SHINO, K., NAGANO, J. and others. (1990) Replacing the medial collateral ligament with an allogeneic tendon graft. *Journal of Bone and Joint Surgery* **72-B**, 6, 1044-1049.
- HULSE, D.A., MICHAELSON, F., JOHNSON, C., ABDELBAKI, Y.Z. (1980) A technique for reconstruction of the anterior cruciate ligament in the dog: A preliminary report. *Veterinary Surgery* **9**, 4, 135-140.

- HULSE, D.A., SHIRES, P.K. (1985) The Stifle Joint. In: Slatter, D.H. (Ed) Textbook of Small Animal Surgery, Vol. 2. W.B.Saunders Company, Philadelphia. pp 2193-2234.
- HULSE, D.A., SHIRES, P.K. (1986) Multiple ligament injury of the stifle joint in the dog. *Journal of the American Animal Hospital Association* **22**, 105-110.
- HULSE, D.A., SHIRES, P.K., ABDELBAKI, Y.Z., HUGH-JONES, M., KEARNEY, M.T. (1986) Vascular access channeling to increase meniscal regeneration in the dog. *Veterinary Surgery* **15**, 6, 414-419.
- JACK, E.A. (1950) Experimental rupture of the medial collateral ligament of the knee. *Journal of Bone and Joint Surgery* **32-B**, 3, 396-402.
- JAMES, R.C., MACLEOD, C.J. (1961) Induction of staphylococcal infections in mice with small inocula introduced on sutures. *British Journal of Experimental Pathology* **42**, 266-277.
- JAMES, S.L. (1980) Biomechanics of knee ligament reconstruction. *Clinical Orthopaedics and Related Research* **146**, 90-101.
- JOHNSON, D.E. (1985) Tendons, skeletal muscles, and ligaments in health and disease. In: Newton, C.D., Nunamaker, D.M. (Eds), Textbook Of Small Animal Orthopaedics. J.B. Lippincott Co., Philadelphia. pp 65-76.
- JOHNSON, A.L., OLMSTEAD, M.L. (1987) Caudal cruciate ligament rupture. A retrospective analysis of 14 dogs. *Veterinary Surgery* **16**, 3, 202-206.
- KENNEDY, J.C. (1963) Complete dislocation of the knee joint. *Journal of Bone and Joint Surgery* **45-A**, 5, 889-903.
- KENNEDY, L.C., ROTH, J.H., MENDENHALL, H.V., and others (1980) Intra-articular replacement in the anterior cruciate ligament deficient knee. *American Journal of Sports Medicine* **8**, 1-8.
- KETCHUM, L.D., MARTIN, N.L., KNAPPEL, O.A. (1977) Experimental evaluation of factors affecting the strength of tendon repairs. *Plastic Reconstructive Surgery* **59**, 708-719.
- KING, D. (1936) The healing of the semilunar cartilages. *Journal of Bone and Joint Surgery* **18**, 4, 333-342.
- KIRBY, B.M. (1993) Decision-making in cranial cruciate ligament ruptures. *Veterinary Clinics of North America: Small Animal Practice* **23**, 4, 797-819.

- KOLATA, R.J., KRAUT, N.H., JOHNSTON, D.E. (1974) Patterns of trauma in urban dogs and cats: A study of 1000 cases. *Journal of the American Veterinary Medical Association* **164**, 499-502.
- KRAUSE, W.R., POPE, M.H., JOHNSON, R.J., WILDER, D.G. (1976) Mechanical changes in the knee after meniscectomy. *Journal of Bone and Joint Surgery* **58-A**, 7, 599-604.
- LAROS, G.S., TIPTON, C.M., COOPER, R.R. (1971) Influence of physical activity on ligament insertions in the knees of dogs. *Journal of Bone and Joint Surgery* **53-A**, 2, 275-286.
- LAING, E.J. (1993) Collateral ligament injury and stifle luxation. *Veterinary Clinics of North America: Small Animal Practice* **23**, 4, 845-853.
- LEACH, D., JACOBS, J. (1990) Normal Arthrology. In: Whittick, W.G. (Ed) *Canine Orthopaedics* 2nd edn. Lea and Febiger, Philadelphia. pp 42-58.
- LEIGHTON, R.L., BRIGHTMAN, A.H. (1976) Experimental and clinical evaluation of a new prosthetic anterior cruciate ligament in the dog. *Journal of the American Animal Hospital Association* **12**, 735-740.
- MASON, M.L., ALLEN, H.S. (1941) The rate of healing of tendons. *Annals of Surgery* **113**, 3, 424-455.
- MILTNER, L.J., HU, C.H., FANG, H.C. (1937) Experimental joint sprain. *Archives of Surgery* **35**, 234-242.
- MONTGOMERY, R.D., LONG, I.R., MILTON, J.L., and others. (1989) Comparison of aerobic culturette, synovial membrane biopsy and blood culture medium in detection of canine bacterial arthritis. *Veterinary Surgery* **18**, 300-303.
- MORGAN, C.D., CASSCELS, S.W. (1986) Arthroscopic meniscal repair: A safe approach to the posterior horns. *Arthroscopy* **2**, 3-12.
- MORGAN, C.D. (1991) The "all-inside" meniscus repair. *Arthroscopy* **7**, 120-125.
- NEWTON, C.D. (1985) Arthrodesis of the Stifle, Tarsus, and Interphalangeal Joints. In: Newton C.D. and Nunamaker D.M. (Eds), *Textbook Of Small Animal Orthopaedics*. J.B. Lippincott Co., Philadelphia. pp 565-570.
- NEWTON, C.D., NUNAMAKER, D.M. (1985) *Textbook Of Small Animal Orthopaedics*. J.B. Lippincott Co., Philadelphia. pp 1104-1106.

- NOYES, F.R., DELUCAS, J.L., TORVIK, P.J. (1974) Biomechanics of anterior cruciate ligament failure: An analysis of strain-rate sensitivity and mechanisms of failure in primates. *Journal of Bone and Joint Surgery* **56-A**, 2, 236-253.
- NOYES, F.R. (1977) Functional properties of knee ligaments and alterations induced by immobilisation. A correlative biomechanical and histological study in primates. *Clinical Orthopaedics and Related Research* **123**, 210-242.
- NOYES, F.R., GROOD, E.S. BUTLER, D.L., MALEK, M. (1980) Clinical laxity tests and functional stability of the knee: Biomechanical concepts. *Clinical Orthopaedics and Related Research* **146**, 84-89.
- NOYES, F.R., KELLER, C.S., GROOD, E.S. BUTLER, D.L. (1984) Advances in the understanding of knee ligament injury, repair, and rehabilitation. *Medicine and Science in Sports and Exercise* **16**, 5, 427-443.
- NUNAMAKER, D.M. (1985) Fractures and Dislocations of the Stifle. In: Newton, C.D., Nunamaker, D.M. (Eds), *Textbook Of Small Animal Orthopaedics*. J.B. Lippincott Co., Philadelphia. pp 433-438.
- O'CONNOR, B.L., McCONNAUGHEY, S.J. (1978) The structure and innervation of cat knee menisci, and their relation to a "sensory hypothesis" of meniscal function. *American Journal of Anatomy* **153**, 431-442.
- O'DONOGHUE, D.H., ROCKWOOD, C.A., ZARICZNYI, B., KENYON, R. (1961) Repair of knee ligaments in dogs. *Journal of Bone and Joint Surgery* **43-A**, 8, 1167-1178.
- O'DONOGHUE, D.H., ROCKWOOD, C.A., FRANK, G.R., JACK, S.C., KENYON, M.D. (1966) Repair of the anterior cruciate ligament in dogs. *Journal of Bone and Joint Surgery* **48-A**, 3, 503-519.
- OGATA, K., WHITESIDE, L.A., ANDERSON, D.A. (1980) The intra-articular effects of various postoperative managements following knee ligament repair: an experimental study in dogs. *Clinical Orthopaedics and Related Research* **150**, 271-276.
- PAATSAMA, S. (1952) Ligament injuries of the canine stifle joint. A clinical and experimental study. Thesis, Helsinki.
- PANCO, P.J. (1985) Functional repair of a femorotibial luxation: An alternative to arthrodesis. *Veterinary Medicine* **80**, 35-42.
- PARK, R.D. (1979) Radiographic evaluation of the canine stifle joint. *The Compendium on Continuing Education* **1**, 11, 833-841.

- PARKER, R.B., SCHUBERT, T.A. (1981) Repair of ligamentous joint injuries in three dogs using spiked washers. *Journal of the American Animal Hospital Association* **17**, 45-50.
- PEARSON, P.T. (1971) Ligamentous and meniscal injuries of the stifle joint. *Veterinary Clinics of North America* **1**, 3, 489-501.
- PETERSEN, S.W., TAYLOR, R.A. (1995) Gore-Tex ligament prosthesis for the cranial cruciate deficient canine stifle: A preliminary study. *Scientific Abstract from the Veterinary Orthopaedic Society 22nd Annual Conference*.
- PHILLIPS, I.R. (1982) Dislocation of the stifle joint in the cat. *Journal of Small Animal Practice* **23**, 217-221.
- PIPER, T.L., WHITESIDE, L.A. (1980) Early immobilisation after knee ligament repair in dogs. An experimental study. *Clinical Orthopaedics and Related Research* **150**, 277-282.
- PIERMATTEI, D.L. (1993) Approach to the stifle through a lateral and medial incision. In: Piermattei, D.L. (Ed) *An Atlas of Surgical Approaches to the Bones and Joints of the Dog and Cat*, 3rd edn. WB Saunders Co., Philadelphia. pp 276-281.
- POND, M.J. (1971) Normal tissues and their reaction to injury. *Veterinary Clinics of North America* **1**, 3, 523-535.
- POND, M.J., CAMPBELL, J.R. (1972) The canine stifle joint. 1. Rupture of the anterior cruciate ligament. *Journal of Small Animal Practice* **13**, 1-10.
- RATHORE, S.S. (1959) Tissue transplants for repair of canine anterior cruciate ligament. Thesis, Michigan State University.
- RATHORE, M.S., RATHORE, S.S. (1976) A study on autogenous tissues transplants for the repair of canine collateral ligaments of stifle joint. *Indian Veterinary Journal* **53**, 620-626.
- REINKE, J.D. (1982) Cruciate ligament avulsion injuries in the dog. *Journal of the American Animal Hospital Association* **18**, 257-264.
- ROBINS, G.M. (1990) The Canine Stifle Joint. In: Wittick, W.G. (Ed) *Canine Orthopaedics* 2nd edn. Lea and Febiger, Philadelphia. pp 693-760.
- RODIN, E.L., DELAMOTTE, F., MAQUET, P. (1984) The role of the menisci in the distribution of stress in the knee. *Clinical Orthopaedics and Related Research* **185**, 290-293.

- ROUSH, J.C., HOHN, R.B., DEANGELIS, M. (1970) Evaluation of transplantation of the long digital extensor tendons for correction of anterior cruciate rupture in dogs. *Journal of the American Veterinary Medical Association* **156**, 309-312.
- ROY, R., DEE, J. (1994) Management of Articular Fractures, Sprains and Strains. In: Houlton, J.E.F and Collinson, R.W. (Eds) *Manual of Small Animal Arthrology*. British Small Animal Veterinary Association, Cheltenham, Gloucestershire. pp 55-61.
- RUDY, R.L. (1974) Stifle Joint. In: Archibald, J. (Ed) *Canine Surgery* 2nd edn. American Veterinary Publications Inc., Santa Barbara, California. pp 1104-1155.
- SEALE, K.S., HAYNES, D.W. (1981) The effect of meniscectomy on knee stability. *Transactions of the Orthopaedic Research Society* **6**, 236-240.
- SHAH, J.S., JATSON, M.I.V., HAMPSON, W.G.J. (1977) Low tension studies of collagen fibres from ligaments of the human spine. *Annals of the Rheumatic Diseases* **36**, 139-145.
- SHAPIRO, F., GILNCHER, M.J. (1980) Induction of osteoarthritis in the rabbit knee. *Clinical Orthopaedics and Related Research* **147**, 287-295.
- SHELBOURNE, K.D., PATEL, D.V. (1995) Management of combined injuries of the anterior cruciate and medial collateral ligaments. *Journal of Bone and Joint Surgery* **77-A**, 5, 800-806.
- SHIRES, P.K., HULSE, D.A., LIU, W. (1984) The under-and-over fascial replacement technique for anterior cruciate ligament rupture in dogs: A retrospective study. *Journal of the American Animal Hospital Association* **20**, 69-77.
- SINGLETON, W.B. (1969) Observations based upon the surgical repair of 106 cases of anterior cruciate ligament rupture. *Journal of Small Animal Practice* **10**, 269-278.
- SMITH, G.K., TORG, J.S. (1985) Fibular head transposition for repair of cruciate deficient stifle in the dog. *Journal of the American Veterinary Medical Association* **187**, 4, 375-383.
- SMITH, G.K. (1995) The principles for repair of multiple ligamentous injuries of the stifle. *Scientific Abstract from the Veterinary Orthopaedic Society 22nd Annual Conference*.
- STASHAK, T.S., YTURRASPE, D.J. (1978) Considerations for selection of suture materials. *Journal of Veterinary Surgery* **7**, 48-52.

- STEAD, A.C., AMIS, A.A., CAMPBELL, J.R. (1991) Use of polyester fibre as a prosthetic cranial cruciate ligament in small animals. *Journal of Small Animal Practice* **32**, 448-454.
- STEIN, L.E., PIJANOWSKI, G.L., JOHNSON, A.L. (1985) A histological evaluation of rabbit tendons sutured using the Bunnell pattern. *Veterinary Surgery* **14**, 145-148.
- STRANDE, A. (1967) Repair of the ruptured cranial cruciate ligament in the dog. Williams and Wilkins, Baltimore.
- SWAIM, S.F. (1980) Surgery of Traumatized Skin: Management and Reconstruction in the Dog and Cat. W.B. Saunders Co., Philadelphia. pp 247-261.
- TIPTON, C.M., JAMES, S.L., MERGNER, W., TCHENG, T. (1970) Influence of exercise on strength of medial collateral knee ligaments of dogs. *American Journal of Physiology* **218**, 3, 894-902.
- TIRGARI, M. (1978) The surgical significance of the blood supply of the canine stifle joint. *Journal of Small Animal Practice* **19**, 451-462.
- TOOMBS, J.P., WALLACE, L.J. (1979) Surgical management of multiple ligamentous injuries of the feline stifle joint: case report. *Veterinary Surgery* **8**, 34-37.
- VASSEUR, P.B., ARNOCKY, S.P. (1981) Collateral ligaments of the canine stifle joint: Anatomic and functional analysis. *American Journal of Veterinary Research* **42**, 1133-1137.
- VASSEUR, P.B. (1984) Clinical results following nonoperative management for rupture of the cranial cruciate ligament in dogs. *Veterinary Surgery* **13**, 243-246.
- VARMA, S., JOHNSON, L.W., FERGUSON, H.L., LUMB, W.V. (1981) Tissue reaction to suture materials in infected surgical wounds. A histopathological evaluation. *American Journal of Veterinary Research* **42**, 563-574.
- VAUGHAN, L.D. (1963) A study of the replacement of the anterior cruciate ligament in the dog by fascia, skin and nylon. *The Veterinary Record* **75**, 21, 537-541.
- VAUGHAN, L.D., BOWDEN, N.L.R. (1964) The use of skin for the replacement of the anterior cruciate ligament in the dog. A review of 30 cases. *Journal of Small Animal Practice* **5**, 167-171.
- VAUGHAN, L.D., EDWARDS, G.B. (1978) The use of carbon fibre (Grafil) for tendon repair in animals. *Veterinary Record* **102**, 13, 287-288.
- VAUGHAN, L.D. (1981) The use of carbon fibre implants for the repair of Achilles tendon rupture in dogs. *Journal of Small Animal Practice* **22**, 629-634.

- WARREN, R.F. (1990) Meniscectomy and repair on the anterior cruciate ligament deficient patient. *Clinical Orthopaedics and Related Research* **252**, 55-63.
- WELCHES, C.D., SCAVELLI, T.D. (1990) Transarticular pinning to repair luxation of the stifle joint in dogs and cats: A retrospective study of 10 cases. *Journal of the American Animal Hospital Association* **26**, 207-214.
- WINSTON, L.A., TERRY, A.F., JARDINE, J.H., and others (1978) The result of replacement of partial or total collateral ligaments with Marlex mesh in the knees of dogs. *Clinical Orthopaedics and Related Research* **137**, 287-290.
- WOO, S.L.Y., YOUNG, E.P., OHLAND, K.J., and others (1990) The effects of transection of the anterior cruciate ligament on healing of the medial collateral ligament. A biomechanical study of the knee in dogs. *Journal of Bone and Joint Surgery* **72-A**, 3, 382-392.
- YAHIA, L.H., NEWMAN, N.M., ST-GEORGES, M. (1992) Innervation of the canine cruciate ligaments. *Anatomical and Histological Embryology* **21**, 1, 1-8.