1995

An Investigation of Differential Diagnosis of Patellofemoral Pain Syndromes

Shannon Marie Buckmier
University of North Dakota

AN INVESTIGATION OF DIFFERENTIAL DIAGNOSIS OF PATELLOFEMORAL PAIN SYNDROMES

by

Shannon Marie Buckmier
Bachelor of Science in Physical Therapy
University of North Dakota, 1994

An Independent Study

Submitted to the Graduate Faculty of the

Department of Physical Therapy

School of Medicine

University of North Dakota

in partial fulfillment of the requirements

for the degree of

Master of Physical Therapy

Grand Forks, North Dakota
May
1995

This Independent Study, submitted by Shannon Marie Buckmier in partial fulfillment of the requirements for the Degree of Master of Physical Therapy from the University of North Dakota, has been read by the Faculty Preceptor, Advisor, and Chairperson of Physical Therapy under whom the work has been done and is hereby approved.

Peggy M. Mohr
(Faculty Preceptor)

Thomas Mohr
(Graduate School Advisor)

Thomas Mohr
(Chairperson, Physical Therapy)

PERMISSION

Tilte	An Investigation of Differential Diagnosis of Patellofemoral Pain Syndromes
Department	Physical Therapy
Degree	Master of Physical Therapy

In presenting this Independent Study Report in partial fulfillment of the requirements for a graduate degree from the University of North Dakota, I agree that the Department of Physical Therapy shall make it freely available for inspection. I further agree that permission for extensive copying for scholarly purposes may be granted by the professor who supervised my work or, in her absence, by the Chairperson of the department. It is understood that any copying or publication or other use of this Independent Study Report or part thereof for financial gain shall not be allowed without my written permission. It is also understood that due recognition shall be given to me and the University of North Dakota in any scholarly use which may be made of any material in my Independent Study Report.

Signature Shannon Buckmier

Date 12/15/94

TABLE OF CONTENTS

TITLE PAGE......i

APPROVAL PAGE......ii

PERMISSION PAGE......iii

ACKNOWLEDGEMENTS......v

ABSTRACT......vi

CHAPTER ONE: INTRODUCTION......1

CHAPTER TWO: ANATOMY, FUNCTION, AND BIOMECHANICS......3

CHAPTER THREE: PLICA SYNDROME......10

CHAPTER FOUR: CHONDROMALACIA PATELLAE......16

CHAPTER FIVE: PATELLAR TRACKING......22

CHAPTER SIX: PATELLAR INSTABILITIES: SUBLUXATIONS & DISLOCATIONS......26

CHAPTER SEVEN: CONCLUSION......31

APPENDIX......38

REFERENCES......34

ACKNOWLEDGMENTS

I would like to sincerely thank Peg Mohr, PhD, for all her efforts with the preparation of this independent study. I would also like to express my gratitude to everyone in the Physical Therapy Department at the University of North Dakota for all their support throughout the past three years.

I would like to thank my friends, relatives, and especially my parents for their never ending love, encouragement, and support over the years. I hope that I will always be there for them as they were for me. I would also like to dedicate this manuscript to my Grandmother who passed away this last August.

ABSTRACT

Dysfunction of the patellofemoral joint has long been a topic of discussion and debate for caregivers of patients with this syndrome. Various diagnosis related to the patellofemoral joint of the knee have been reported. Patellofemoral pain refers to the clinical presentation of pain in the anterior region of the knee. It is one of the most common musculoskeletal complaints in children, adolescents, and adults. It is a major source of pain and dysfunction in both genders, but is more common in females.

The purpose of this literature review is to help distinguish between several differential diagnosis included as part of patellofemoral pain syndromes. These include plica syndrome, chondromalacia patellae, extensor mechanism or tracking problems, and instability problems of subluxation and dislocations. Aspects of each of the above conditions will be discussed in the manner of definition, presentation, clinical signs and symptoms, and special tests specific to each syndrome along with a brief diagnosis and treatment for each condition. I will conclude my findings by comparing and contrasting each condition.

CHAPTER ONE

INTRODUCTION

Dysfunction of the patellofemoral joint has long been a topic of discussion and debate for caregivers of patients with this syndrome. Various diagnosis related to the patellofemoral joint and the extensor mechanism of the knee have been reported.[1] Patellofemoral pain refers to the clinical presentation of pain in the anterior region of the knee.[2,3,4] Patellofemoral pain is the most common symptom complex in the knee joint[5] and is one of the most common musculoskeletal complaints in children, adolescents, and adults.[3] It is a major source of pain and dysfunction in both genders,[6,7] but is found to be more common in females.[8,9,10] It afflicts not only the athletic population but sedentary individuals as well.[6,7] Shellock[10] stated that approximately 30% of exertion-related injuries in athletes involve the patellofemoral joint; and in some active populations such as runners, the patellofemoral joint is the most frequently injured joint.[10,11]

These patients often present with nonspecific signs and symptoms of varying degrees. The exact source of patellofemoral pain cannot always be determined and it is diagnosed as "anterior knee pain." The knee joint is made up of articular cartilage which does not contain pain receptors. Therefore, the relationship between cartilage degeneration and pain is inconsistent.[6] These conditions are often frustrating for the patient[4] as well as the caregiver.[3] The precipitating events, the location and duration of the pain are all important factors in narrowing the differential diagnosis, as are a detailed history and physical examination.[3,7]

The purpose of this literature review is to help distinguish between several differential diagnosis included as part of patellofemoral pain syndromes. These include plica syndrome, chondromalacia patellae, extensor mechanism or tracking problems, and instability problems of subluxation and dislocations. Aspects of each of the above conditions will be discussed in the following manner: definition, presentation, clinical signs and symptoms, and special tests specific to each syndrome along with a brief diagnosis and treatment for each. I will conclude my findings by comparing and contrasting each condition.

CHAPTER TWO

ANATOMY, FUNCTION AND BIOMECHANICS

This chapter will focus on the anatomy, function, and biomechanics of the knee. An understanding of these factors is of primary importance when trying to distinguish between patellofemoral pain syndromes of plica, chondromalacia patellae, tracking, and instability problems.

The human knee is the largest[12] and most complex joint in the body.[13] It is formed during the fourth month of embryologic development.[14,15] The knee joint is comprised of the tibiofemoral and patellofemoral joints.[12]

The tibiofemoral joint is the largest hinged joint in the body,[16] formed by the two longest bones of the body, the femur and the tibia, therefore, an enormous amount of stress may be placed upon the knee joint. The long lever arms of the femur and tibia produce an increased mechanical advantage which places the knee joint at an increased risk of injury.[12,16] Stability of this joint depends on the strength of all the muscles and ligaments attached to the joint.[13,16]

Tibiofemoral motion occurs through rolling and gliding. During flexion, rolling is illustrated by the contact point of the tibia moving posteriorly. Gliding occurs because the contact point on the femur moves a greater distance than the contact point on the tibia. Along with rolling and gliding, the tibia rotates externally throughout the range of motion

(ROM) from flexion to extension. This is referred to as the screw-home mechanism; and it provides additional stability to the tibiofemoral joint.[12]

The patellofemoral joint, which is of primary importance in this case, is a synovial[13] modified plane joint.[16] Gliding motion occurs at this joint with the patella being displaced approximately seven centimeters along the femoral condyles providing the entire ROM is present.[12]

The patella is the largest sesamoid bone in the body.[2,8,13] It is embedded within the quadriceps tendon proximally at its base and within the patellar tendon distally at its apex.[8] It is triangular in shape with its apex being connected to the tibial tuberosity by the ligamentum patellae.[13] This attachment helps to prevent anterior displacement of the femur on the tibia.[4] The patella consists of the thickest layer of cartilage in the body,[16] measuring 4-5 millimeters at its thickest region of the patellar body.[2] The patellar surface consists of two primary facets: medial and lateral.[2] It may have up to seven facets with four on the medial side secondary to an additional odd facet found here, and three on the lateral side.[5] The odd facet serves as the most medial portion of the articular surface of the patella.[17]

The patella serves six main functions:

(1) It provides anterior displacement of the quadriceps tendon throughout the entire ROM. This mechanism facilitates extension and offers mechanical advantage which improves knee extension force by 25-30%.[2,4,5]

(2) The quadriceps and patellar tendons are protected from friction, thus, permitting the extensor apparatus to tolerate high compressive loads.[2,12]

(3) A cartilage-on-cartilage articulation provides a relatively low coefficient of friction, which increases the efficiency of the quadriceps.[2,8]

(4) The divergent forces of the four quadriceps heads become centralized and are transmitted to the patellar tendon.[2]

(5) The patella provides a bony shield for the knee joint, protecting the articular cartilage of the anterior femur and tibia from direct trauma.[2,4]

(6) The patella serves a cosmetic function.[2,16]

McConnell[7] identifies four ways to describe patellar position with respect to the femur. These include medial-lateral tilt, anterior-posterior tilt, patellar rotation, and lateral patellar glide.

The medial-lateral patellar tilt is represented by the level of each border of the patella. The two borders should be level in the frontal plane. Many patients with patellofemoral symptoms will show lateral tilting, with the medial patellar border being more prominent.[4,7]

Anterior-posterior tilt refers to the position of the inferior pole of the patella in regard to the superior pole. Upon contraction of the quadriceps muscle, if the inferior pole is tilted posteriorly compared to the superior pole, it can irritate the fat pad. This can lead to pain upon extension or hyperextension of the knee.[4,7]

The rotational component determines if there is any deviation of the long axis of the patella from the long axis of the femur by assessing the position of the inferior pole of the patella with the superior pole. The most common finding is external rotation of the patella, which is seen when the inferior pole of the patella is lateral to the superior pole.[4,7]

The glide component is measured by first observing the knee with the quadriceps relaxed and in an extended position. The patella should be positioned in midline between the two condyles. Statically the patella may appear normal, however, once the quadriceps muscle is contracted, the patella may move laterally.[4,7]

The patellofemoral joint stability is dependent upon tendons and ligaments.[3] Patellofemoral joint stability and normal function of the patella during flexion are maintained by the following: interaction between static stabilizers, dynamic stabilizers, congruency between bony structures, alignment of the femur and tibia, and the quadriceps or Q angle.[4,6,10]

Static stabilizers are structures which exert forces acting on a body in equilibrium.[12] These include ligaments around the knee such as the patellar tendon, medial and lateral patellofemoral ligaments, as well as the medial and lateral retinaculum.[4,10]

Dynamic stabilizers are variable forces acting on a body in motion such as those exerted by the quadriceps muscle group.[10] As an overall function, the quadriceps muscle group acts on the patella to extend the knee, but this is not true for individual muscles of the group. The rectus femoris works dynamically to extend the knee. The vastus medialis oblique (VMO) muscle individually cannot extend the knee. The VMO is the primary controller of medial movement of the patella to maintain proper alignment of the patella in the femoral sulcus.[3,4] The VMO also aids in the final movement into internal rotation of the femur on the tibia. According to Fox,[5] the VMO is phylogenetically the weakest of the quadriceps group and appears to be the first muscle to show atrophy and the last to respond to rehabilitation. The vastus lateralis along with a section of the iliotibial (IT) band provide the lateral dynamic force.[3] With the quadriceps group being unbalanced, the VMO generally is overwhelmed by the rest of the group, resulting in a greater lateral than medial force.[5]

Congruency between bony structures is dependent upon the shape of the patella and the femoral trochlear groove because of their articulation during flexion.[10] The shape of the facets on the patella's undersurface help determine the "fit" between the patella and the femoral groove. The femoral trochlear groove or sulcus is angled slightly medially, with the lateral femoral condyle higher than the medial; the normal sulcus angle is 137 degrees.[5,8] The sulcus is important in keeping the patella "seated" and tracking properly. The medial and lateral retinaculum keep the patella "centered" between the femoral condyles during motion.[8] The dynamic motion of the patella through its ROM is initiated with the patella positioned laterally in full extension and gradually moving medially and centrally with increased flexion.[5]

The alignment of the femur and tibia as well as the entire lower extremity are extremely important factors in the proper function of the patellofemoral joint.[5] Malalignment is the leading cause of patellofemoral pain syndromes.[10]

The Q angle is defined as the angle between the rectus femoris muscle and the patellar tendon. The normal Q angle for males is 8-14 degrees and for females is 11-20 degrees.[4] The normal Q angle has been defined as 14 degrees with anything above 20 degrees being abnormal.[5,8,18]

Patellofemoral biomechanics pertain to the articulation of the patellar facets with the femoral trochlea on the distal femur.[3,5] In full extension, the patella is in its most unstable position[6] and sits at the proximal trochlea, against the suprapatellar fat pad. Initial contact occurs at 20 degrees of flexion with the most distal aspect of the patellar cartilage coming into contact with the trochlea.[2,5] At 30 degrees of flexion, the patella is well seated in the trochlear groove.[3] At 90 degrees of flexion, the patella is positioned within the condylar fossa, contacting regions of both the medial and lateral facets of the patella with the femur. At 135 degrees of flexion, the odd facet articulates with the femoral condyle.[2,3,16]

As the knee flexes, the contact points of the patellofemoral joint move proximally on the patella and distally on the femoral intercondylar sulcus.[4] Biomechanical studies have shown that as knee flexion increases, so do tendofemoral and patellofemoral compression forces.[5,17] As the knee flexes and extends, the patellofemoral joint undergoes changes in contact area and joint reaction forces; however, because the force increases more rapidly than the surface area, the stress on the patella increases significantly with flexion.[5] At full extension of the knee, the patellofemoral joint reaction force approaches zero.[4] The force will vary, increasing with flexion and decreasing with extension.

The angle at which the patellofemoral joint reaction forces are greatest can also vary depending on the activity.[4] During level gait with the knee flexed to nine degrees, the force across the patellofemoral joint is 0.5 times body weight. This force increases to 3.3 times body weight with the knee flexed to 60 degrees during stair climbing.[2,4,16] Squatting with the knee flexed to 130 degrees produces patellofemoral joint reaction force of 7.8 times body weight. Abnormal tracking can generate even greater forces across the patellofemoral joint.[2,5,12,16] This information has been presented in the table below for easier viewing of patellofemoral joint reaction forces.

Degrees Of Knee Flexion	Patellofemoral Joint Reaction Forces
Full Extension (0 degrees)	0.0 X Body Weight (BW)
Level Gait (9 degrees)	0.5 X BW
Stair Climbing (60 degrees)	3.3 X BW
Squatting (130 degrees)	7.8 X BW

*Abnormal tracking can generate even greater forces across the patellofemoral joint.

CHAPTER THREE

PLICA SYNDROME

The knee is embryonically formed by the fusion of three separate synovial compartments during the fourth month of development. Normally, the synovial membranes are reabsorbed[14] and the knee becomes a single cavity.[19] Absorption of the membranes may be incomplete allowing synovial folds to project into the knee joint.[15] Remnants that persist into adulthood are called synovial plica.[7,14,15,19,20,21,22,23] Synovial plica of the knee are present in approximately 20-60% of the population,[6,21,24] but rarely cause symptoms.[6,20,21] There are three major types of plica including the infrapatellar, suprapatellar, and mediopatellar plica.

Infrapatellar plica is the most common but least symptomatic of the three.[20,21,23] It originates from the ligamentum patellae and the intercondylar notch[25] and widens as it extends distally to insert on the infrapatellar fat pad. Posteriorly the infrapatellar plica borders the anterior cruciate ligament.[15,19]

The suprapatellar plica represents a remnant of the embryonic septum dividing the inferiorly placed medial and lateral compartments from the suprapatellar pouch. The suprapatellar plica rarely completely divides the suprapatellar pouch - leaving a small opening called a porta where loose bodies can hide.[19] Usually the suprapatellar plica is seen as a small cresent-shaped synovial fold origionating off the inferior surface of the quadriceps tendon and inserting into the medial wall of the suprapatellar pouch.[15] It has been stated that the suprapatellar plica may cause synovitis and exercise related edema.[15]

The primary debate is in reference to the mediopatellar plica.[23] It is the most commonly symptomatic plica and the one usually implicated in symptoms describing the plica syndrome.[15] Symptoms from a pathological mediopatellar plica are most commonly seen during adolescents, but can be seen at any age.[14] The mediopatellar plica originates medially to the undersurface of the quadriceps tendon[25] and may be continuous with the suprapatellar plica.[20] It extends distally around the medial edge of the patella, lying flat against the medial femoral condyle and inserting into the infrapatellar fat pad.[19]

The mediopatellar plica has been classified into four variations.[15,25] Type A is a cord-like elevation in the medial synovial wall. Type B is a shelf-like structure that does not entirely cover the anterior surface of the medial femoral condyle. Type C is a larger shelf-like structure that does cover the anterior surface of the medial femoral condyle. Type D is similar to type C; however, there is a separation between the shelf and the synovial wall, creating a bucket handle.

Normally, plica are soft, pliant, and highly elastic tissues.[24] Plica may become symptomatic by any mechanism that converts it into a bowstring.[20,21,24] The plica glides under the medial facet of the patella at a range of 40-80 degrees of flexion and may become impinged between the patella and the medial femoral condyle causing pain, inflammation, and snapping to occur.[15,25] Impingement of the mediopatellar plica causes wear of the underlying articular cartilage leading to chondromalacia[15] of either the medial facet of the patella or the medial femoral condyle.[14] If mechanical injury or inflammation from impingement have occurred, the plica may become thickened, tight, and inelastic bands that interfere with the normal mechanics of the knee.[15,20,21,23] Continued trauma will lead to tough, inelastic fibrotic bands.[20,22,23,24,25] It should be noted that plica need not contact the medial femoral condyle in order to be symptomatic. The tethering effect of thickened plica may interfere with normal quadriceps function and place excessive traction on the synovial insertions which are rich in nerve endings.[14,20]

Plica may become symptomatic secondary to direct trauma[19,22] such as a fall, dashboard injury, or striking a solid object with the anteromedial surface of the knee.[14] It may also occur indirectly such as in overuse syndrome secondary to repetitive microtrauma,[11] synovitis, or hemarthrosis.[20]

The diagnosis of plica syndrome must be one of exclusion.[14,21] The clinical diagnosis rests on the data obtained from a detailed history and physical examination.[19] In the following paragraphs, a description of common signs and assessment techniques will be reviewed.

The most common present complaint reported was anteromedial knee pain.[14,19,20,21,23,24] Characteristically, this pain is usually an intermittent,[19] dull, and aching type of pain.[8,24] This pain is generally increased with activities requiring flexion and extension of the knee particularly stairclimbing[19,24,25,26] or repetitive knee flexion activities such as running and jumping.[7,14] Some patients noted that the pain is greatest after long periods of sitting with the knee in a flexed position. This position draws the plica taut across the condyle leading to considerable discomfort. This has been termed a positive "Theatre Sign."[19,24]

Common symptoms associated with mediopatellar plica include snapping, popping, or clicking and instability or giving way sensation.[8,20,21,23] Other nonspecific symptoms supporting the diagnosis including intermittent edema,[15,23] weakness, stiffness,[15] and intermittent catching episodes.[7]

Upon physical examination, pain will be localized medial to the patella approximately one fingerwidth proximal to the inferior pole of the patella.[14] A symptomatic mediopatellar plica can often be palpated as a tender band like structure paralleling the medial border of the patella.[19] Applying pressure over this area should reproduce the patient's symptoms.[14] Some patients will have tenderness over the irritated femoral condyle,[15,19] others will reveal tenderness upon palpation along the medial facet of the patella.[11,25] Atrophy of the thigh, dynamic patellar crepitus, loss of motion, and effusion[15,19] are other possible physical findings which are important primarily when occurring in succession with previously mentioned signs and symptoms of plica syndrome.

Special tests that could be performed to aid in the diagnosis of plica syndrome include the following:

(1) Palpation may reveal tenderness along the medial facet of the patella.[11,14,23]

(2) Patellar Grind or Compression Sign[11,14,23,26,27] would be positive secondary to trapping of the medial patellar plica between the patella and the medial femoral condyle.

(3) Mediopatellar Plica or Apprehension Test would be positive if pain is indicated upon the examiner's displacement of the patella medially while supporting a flexed knee. This test again causes pinching of the mediopatellar plica.[16,21]

(4) The Plica "Stutter" Test is positive if upon active knee extension, the patella stutters or jumps during midrange through an otherwise smooth movement. The examiner palpates for this movement pattern during the active range of motion (AROM).[16]

(5) Hughston's Plica Test is positive if the examiner palpates a "popping" of the plica band under his fingers while passively flexing and extending the patient's leg which is held with internal rotation of the tibia and the patella positioned medially.[16]

These assessment techniques are helpful in diagnosing plica syndrome. However, many of the clinical signs and symptoms are frequently evident with conditions such as chondromalacia, torn menisci, and loose bodies.[15] Therefore arthroscopy or arthrotomy can be utilized along with a detailed history and physical examination to confirm this diagnosis.[6,15,19] Refer to Chapter VII for further comparison of each diagnosis discussed in this literature review.

It is recommended that a conservative approach to treatment should always initially be attempted.[1,4,5,6,7,8,24,27,28,29,30] This would include the use of modalities such as ice massage, ultrasound,[15] phonophoresis,[7] local heat, rest and anti-inflammatory medication.[24] Quadriceps strengthening and hamstring stretching will help to decrease compressive forces on the anterior surface of the knee by increasing posterior structural flexibility.[14,15,19,24]

Only after the failure of conservative techniques, undertaken for an adequate duration of time, will surgery be an option. Shahriaree[14] proposes that the only surgically effective treatment of this syndrome is excision of the mediopatellar plica. Scarring after simple division can occur, allowing another symptomatic plica to reform, so complete excision is recommended.[14,15] Broom and Fulkerson[20] have reported excellent results in greater than 80% of patients following plica excision.

CHAPTER FOUR

CHONDROMALACIA PATELLAE

This chapter is a discussion on chondromalacia patellae which is a term that refers to pathological changes in the articular cartilage of the patella,[3,18,22,31] such as softening[5,26,32] or lesions[9,29] that lead to progressive degeneration.[8,24]

Four stages or grades have been used to describe the extent of involvement of the patellar cartilage. In Stage I or closed chondromalacia, there is swelling and softening of the articular cartilage. Stage II involves fissuring within the softened areas. Stage III involves the breakdown of the surface resulting in fasciculation or fibrillation presenting a "crab meat" appearance. Stage IV is characterized by destruction of articular cartilage and exposure of subchondral bone. Stage IV is indistinguishable from osteoarthritis.[2,29] Stages I, II, and III are usually confined to the patella, where as Stage IV commonly involves the surface of the femur.[29]

Goodfellow et al[33] distinguished between chondromalacia associated with surface degeneration, which is most common on the medial patellar facet and is part of aging, and basal degeneration, which is pathological and may account for symptomatic knee pain. Basal degeneration is found to occur at the junction of the odd and medial facet and at the inferior part of the patellar crest.[29] These regions are subjected to high compressive and shear forces in flexion. In the early stages, this disorder of the deep layers does not involve the surface area.[34] Initially the area will show softening to probing and may lead to further breakdown, resulting in blisterlike disruption with fasciculation. The blister is believed to produce pain by altering the distribution of pressure on the underlying subchondral bone.[29,35]

Insall et al[31,35] discussed the geographical distribution of cartilaginous lesions and found the midpoint of the patellar crest, extending equally both medial and laterally, to be the most frequently involved area. The upper and lower thirds of the patellar surface are usually spared. Chondromalacia of the femoral groove was not reported to be severe or uniform in distribution. Goodfellow et al[33] noted the most frequent site for surface fibrillation was the odd facet which has been demonstrated to be in contact with the condylar surface in extremes of flexion.[16,17]

Chondromalacia patellae is more common in adolescents and young adults, with a predominance in females.[2,24,31] Davidson,[8] Tria, Palumbo, and Alicea[5] argue that it occurs more often in older patients and presents with a longer history of discomfort. It has been found that chondromalacia is age dependent, very common and can be expected in most knees after the third year of life, and usually asymptomatic.[2,29,35]

Degeneration or softening of articular cartilage provokes a chemical synovitis and then effusion. The synovium, which has a rich nerve supply, may become irritated, which produces pain. However, many patients with patellofemoral pain have an intact articular surface with no evidence of local synovial irritation.[34] Several investigators[33,35] have noted the tendency for cartilage that is habitually out of contact with other articular contact to undergo surface fibrillations. Hypopressure and disuse of the medial patellar facet may cause malnutrition and early degeneration changes of the articular cartilage because of the lack of normal kneading and function.[34] These medial lesions appear to be age related and nonprogressive.[35] Chondromalacia occurring on the lateral facet may be secondary to overload such as in the case of patellar tilt or excessive lateral pressure syndrome. These lesions tend to be progressive, leading to cartilage erosion and arthrosis.[35]

The pathophysiology of chondromalacia patellae is unknown, although it is generally accepted as being secondary to patellar malalignment.[31,34,35] It does not occur spontaneously. It is a response of the articular cartilage over time to some pathological situation, usually abnormal patellar tracking.[7,31,35] Some possible etiological factors include biomechanical, biochemical, vascular and malalignment problems.[8,22,35] Degenerative changes can also occur following acute, severe, or repeated minor trauma.[8,24,35]

A natural history of the condition remains unclear and may vary with the cause and location of the lesion.[35] Several signs and symptoms have been described by patients with chondromalacia, but these criteria are not specific.[6] It has been concluded that symptoms were unreliable in predicting the presence of chondromalacia.[2,7] Signs and symptoms include: crepitation, tenderness, effusion, difficulty with descending stairs, instability or giving way sensation, stiffness, quadriceps atrophy, and pain.[2,6,8,24] Patients often have a history of an old knee injury followed by later onset of locking, catching, and weakness of the knee.[24] Risk factors associated with the development of chondromalacia include a poorly developed VMO, malalignment,[2,8,22,35] patella alta, trauma, and tight hamstrings.[2,8]

Physical examination of these patients will reveal the most valuable signs of quadriceps wasting greater than two centimeters, effusion, and retropatellar crepitus.[2,24] Palpation will frequently reveal tenderness on the lateral facet and is associated with genu valgus and pronation of the foot. This results in the patella rubbing laterally at the knee. It has been found that a large percentage of these patients displayed a leg length difference with the shorter leg on the involved side.[24] These patients tended to hyperextend the involved knee during stance and push-off phases of gait. With the use of a heel lift and proper gait instruction without knee hyperextension, the symptoms of chondromalacia were relieved.[24]

Chondromalacia patellae has been thought to be underestimated as an etiology of patellofemoral pain, but there are a number of reports that indicated the correlation of physical signs and symptoms with the appearance of the patellar articular cartilage is poor.[31,35] The identical pain syndrome can exist with normal appearing articular cartilage;[35] approaching 40% even in young patients.[36] It was reported that only 50% of those with symptoms commonly associated with chondromalacia patellae actually showed objective evidence of chondromalacia at arthroscopy.[36] It has also been observed and found that low grade chondromalacia (Stage I and II) may be found in normal individuals at arthroscopy.[31] Patients with more advanced surface changes have reported less pain than those with minor changes.[5] Therefore, it is difficult to predict the extent of changes in the patellar articular cartilage merely from the clinical features.[31]

There are several special tests designed to aid in the diagnosis of chondromalacia patellae:

(1) Clarke's Sign is considered positive if the test causes retropatellar pain and the patient cannot hold or maintain a quadriceps contraction while the examiner is pressing down just proximal to the superior pole of the patella. This test can be performed at various degrees of flexion as well as full extension.[3,16]

(2) Waldron's Test is positive if pain and crepitus occur together while the patient performs several deep knee bends slowly. The examiner palpates for crepitus (significant only if accompanied by pain), noting where it occurs in the range of motion, the amount of pain, and whether there is "catching" or poor tracking of the patella throughout the movement.[30]

(3) Passive Patellar Tilt Test evaluates the tension of the lateral restraints on the patella and is performed by lifting the lateral edge of the patella while depressing the medial edge. The normal angle is 15 degrees with anything less indicating tight lateral restraints, which predisposes patients to chondromalacia patellae[7,30] and chronic tilt of the patella.[2]

(4) Lateral Pull Test is positive if, upon contraction of the quadriceps, there is excessive lateral patellar movement resulting in pain. Normal movement of the patella would be superior or superiolaterally in equal proportions.[5]

(5) Zohler's Sign is positive in a large proportion of the normal population and is performed by pulling the patella distally and holding it while the patient performs a quadriceps contraction. Pain indicates a positive test.[16]

(6) Frund's Sign is positive if compression of the patella onto the femoral condyles produces pain and similar results are evident at various angles.[8]

(7) McConnell Test is completed by having the patient perform an isometric quadriceps contraction at various angles, with the patient's femur laterally rotated. At a painful angle, the extremity is passively returned to neutral position, the examiner presses the patella medially, and the extremity is repositioned at the previously painful angle. If pain is decreased, the pain is considered to be patellofemoral in origin.[16]

Diagnosis of chondromalacia patellae is most often an incidental finding being either a normal part of aging or a reaction of the articular cartilage to abnormal stresses.[29,30,35] It has been reported that chondromalacia patellae is an extremely difficult diagnosis to make with purely clinical or radiographic means.[7] An accurate and detailed history and physical examination are extremely important. The role of arthroscopy allows for precise definition of chondral defects with respect to size, location, and character.[22]

Conservative treatment of chondromalacia patellae is focused on: (a) towards eliminating the cause, (b) adequate dosage of nonsteroidal anti-inflammatory medication to remove the potential for flare-ups or infection, and (c) a quadriceps strengthening program. Arthroscopic treatment is directed at the specific cause of the condition. Malalignment deformities as well as metabolic pathology are corrected. Chondral defects are treated by arthroscopic debridement and stabilization of the lesions.[22] The recommended treatment for basal degeneration is local excision of the blister and drilling of the subchondral bone.[29]

CHAPTER FIVE

PATELLAR TRACKING

Normal patellar alignment and tracking is displayed with the ridge of the patella positioned in the femoral trochlear groove as it travels in a vertical plane during knee flexion, without transverse displacement of the medial or lateral patellar facets. This orientation causes the patella to appear "centered" in the femoral trochlear groove.[10]

For normal function and tracking to occur there must be an equal balance or pull from both soft tissues and bony structures. Soft tissue tightness has a great impact on patellar alignment. If the rectus femoris is tight, full excursion of the patella may be inhibited. If the IT band is tight, the patella will track laterally. Tightness of the hamstrings or gastrocnemius will restrict talocrural dorsiflexion, producing compensatory pronation in the subtalar joint. This creates internal femoral rotation causing an increased dynamic Q angle.[1,4] Hyperextension will also produce an increase in Q angle due to the screw home mechanism that causes external tibial rotation.[5] Insall et al[35] proposed that lateral tracking was due to an increased Q angle causing abnormal shearing stresses on the articular cartilage which could eventually lead to chondromalacia.

At times lateral tracking can be visible clinically by a lateral deviation of the patella as the knee is actively extended. Habitual lateral tracking may produce adaptive changes and, with time, the quadriceps tendon may come to lie more to the lateral side of the knee. The vastus medialis, which is mostly involved in tracking the patella rather than extending the knee,[26] becomes stretched while the vastus lateralis becomes contracted. These alterations can be accelerated by an episode of patellar dislocation. In its most advanced form, habitual dislocation results in an irreducible patella that is positioned laterally.[29]

The pathophysiology of patellar tracking dysfunction include deficiencies of supporting muscles and guiding mechanisms, bony abnormalities, malalignment of the lower extremity, and trauma.[5,34] Deficiencies of patellar stabilizers may be due to quadriceps femoris abnormality and its expansions including: VMO atrophy;[4] laxity of the medial retinaculum; tightness of the lateral retinaculum, hamstrings, IT band, or gastrocnemius; and patella alta which is an abnormally high riding patella.[1,34] Bony abnormalities include irregular size and shape of the patella. Also, abnormality in the shape of the femoral trochlear groove may occur such as a shallow lateral femoral condyle.[8,10] Lower extremity malalignment includes factors such as: an increased Q angle, genu valgum, genu recurvatum, femoral anteversion, lateral displacement of the tibial tubercle or excessive tibial torsion, and excessive foot pronation.[29,30,34] Trauma may also play a role in the pathophysiology of maltracking. Trauma may be due to a direct acute injury, indirect effects of an effusion, or by the chronic repetitive stress of running.[34]

Clinical signs and symptoms associated with maltracking are nonspecific[7] and include the following: (a) vague peripatellar or medial pain that worsens with activity such as stairs, squatting and prolonged sitting, (b) instability or giving way sensations and

VMO atrophy, (c) snapping and popping which may occur with activity.

The following special tests are some of the more general tests utilized to detect maltracking dysfunctions.

(1) The Patellar Glide Test assess medial and lateral retinacular tightness. The patella is divided into quadrants and displaced medially or laterally while the knee is supported in 20-30 degrees of flexion. A lateral patellar glide of three or greater indicates an incompetent medial restraint. A medial glide of one quadrant indicates a tight lateral retinaculum with a medial glide of three or more indicating patellar hypermobility and possible subluxation.[4,30]

(2) Passive Patellar Tilt Test has been described in the previous chapter and is used to identify tight lateral structures.

(3) Q angle measured in full extension describes the degree of lateral pull exerted on the patella by the patellar tendon and the action of the quadriceps muscle. An exaggerated Q angle predisposes the patella to excessive lateral tracking and potential subluxation or dislocation.[3,30]

(4) Muscle Bulk Measurements of the thigh assess atrophy of the quadriceps, specifically the VMO. Atrophy of the VMO can develop into a long term painful condition.[3] Measurements are taken at points that can be reliably replicated.

(5) Ober's Test indicates tightness of the IT band which can lead to lateral tracking.

(6) Range of Motion Evaluation determines hamstring and gastrocnemius tightness, which will restrict dorsiflexion, produce compensatory pronation and internal

rotation, causing an increased dynamic Q angle.[4]

Arthroscopy and axial radiographs can provide additional information on factors that are important in patellar tracking.[2,8] Studies have shown instability abnormalities of this joint are most clearly identified by evaluating early range of motion because pathological patellar tracking starts at the initial portion of the range. Imaging at 25 degrees of flexion or greater frequently misses clinically important information,[10,37] but one must be sure to consider the entire range of motion when evaluating tracking dysfunctions.[2,5] It has been demonstrated that knee flexion beyond 20 degrees will bring some abnormally aligned patella back into the trochlear sulcus.[34]

Current trends in the treatment of patellar tracking problems are conservative with orientation less toward strengthening of the quadriceps and VMO and more toward improving VMO control of the patella.[1] Improving lateral retinacular mobility[34] and muscular flexibility will enhance tracking of the patella and facilitate VMO function. McConnell[4,30] has been successful using taping techniques to hold the patella in proper position during activity. These McConnell taping techniques assist patellar tracking by providing improved stability and reducing mechanical stress.[1,7] McConnell[4,30] has reported a success rate of 90-96% in various studies.

CHAPTER SIX

PATELLAR INSTABILITIES: SUBLUXATION AND DISLOCATIONS

Lateral subluxation of the patella is displayed by the malalignment of the patella on the femoral trochlear groove. With this condition, the ridge of the patella is displaced laterally relative to the femoral trochlear groove. This is the most common form of abnormal patellar malalignment and it occurs in varying degrees. Lateral subluxation of the patella is commonly found in athletes that undergo torsional movements of the lower extremity during their activities.[10] Pathophysiology of lateral subluxation of the patella can occur secondary to large peripatellar effusions, and tight lateral retinacular forces combined with insufficient medial stabilizing forces.[10]

The majority of individuals with patellofemoral joint pain have a form of malalignment that causes abnormal stresses on the articular cartilage. A cartilage defect can frequently be found at the impact site between the patella and femoral trochlear groove. This defect is a result of the chronic hyperpressure that develops from contact stress due to lateral subluxation of the patella. Underloading of the articular cartilage may also lead to a degenerative process as a result of lack of a nutritive stimulus.[10,30] Underloading, overloading, and/or trauma may lead to a degenerative process in the articular cartilage.[6,38]

Medial subluxation of the patella occurs because of medial displacement of the patellar ridge or the center of the femoral trochlea relative to the femoral trochlear groove. This syndrome is most common in individuals who have had surgical realignment procedures such as a lateral retinacular release, if there is overcompensation of the lateral subluxation.[10] Medial subluxation may cause localized hyperpressure and be associated with cartilage defect of the medial patellar facet. Etiological mechanisms can exist individually or in combination including an insufficient lateral retinaculum, overtaut medial retinaculum, abnormal patellofemoral anatomy, unbalanced quadriceps musculature, and excessive internal rotation of the lower extremity.[10]

Clinical signs and symptoms of patellar subluxation include a sense of instability and recall of an initial episode in which the knee "gave way" due to extreme subluxation or dislocation.[2,7,8,30] Common complaints include locking, popping, and apprehension particularly during activities of running, cutting, and pivoting.[6,7,18]

Clinical findings during the physical examination might include muscle atrophy,[7] medial or lateral retinacular tenderness, high and laterally displaced patella,[8] an increased Q angle,[2] hypermobility,[8] crepitus, and a positive apprehension sign.[2,7,8] The patella may display an abnormal course, especially at 10-20 degrees of flexion as it makes its entrance and exit into the trochlea.[2] Patellar subluxation can lead to problems of extensor mechanism instability, increased risk of dislocation, apprehension, patellar hypermobility, and some risk of articular damage.[30]

It has been observed that many patients with simple subluxation or dislocation have little evidence of changes in articular cartilage. It has been reported that chondromalacia was found to occur more frequently in patellar subluxation (93%) than in patellar dislocations (62%).[35] Recurrent patellar subluxation and dislocations are well known to cause articular damage leading to chondromalacia patellae.[5,29,31]

The following are three special tests that will aid in diagnosing patellar subluxation:

(1) The Sulcus Angle is used to evaluate the depth of the trochlea. It is the angle formed by the highest points of the medial and lateral femoral condyles and the lowest point of the intercondylar sulcus. The normal sulcus angle is 138 degrees. The greater the angle is, the shallower the sulcus is, with enhanced potential for patellar instability. A shallow sulcus angle has been associated with recurrent subluxation.[2,5,8]

(2) The Congruence or Lateral Patellofemoral Angle is used to assess the likelihood of subluxation. An abnormal angle has been associated with recurrent subluxation and chondromalacia patellae. This angle helps to demonstrate the relationship of the patella to the femoral sulcus. The sulcus angle is bisected to establish a zero reference line. A second line is drawn from the lowest point on the articular cartilage of the patella to the apex of the sulcus angle. This is the congruence angle with a normal value of a minus six degrees. If the apex is lateral to the reference line, the angle is positive and vise versa.[2]

(3) The Q angle measured at 90 degrees of knee flexion can identify lateral subluxation of the patella which may have been falsely reduced or missed with the Q angle measured in full extension. To avoid underestimation of the Q angle, the patella must be seated within the trochlear notch accomplished by knee flexion of 90 degrees. Reference points include the AIIS, center of the patella, and the mid-tibial tubercle. The

patient is positioned supine with both hip and knee flexed to 90 degrees. A normal range of minus four to a positive six degrees exists with any angle greater than eight degrees strongly indicating an abnormally lateralized distal patellar vector.[30]

The most sensitive measure of diagnosing patellar subluxation, readily available to the clinician, is the axial radiograph.[18] Taken at 45 degrees of flexion, it may display an apparent subluxation, as well as, patella alta.[2,18]

Specific VMO training and exercises are recommended in the treatment of patellar subluxation. Stabilizing braces and McConnell taping techniques are used to give subjective relief and can decrease lateral patellar displacement during initiation of knee flexion, but are ineffective in the correction of patellar tilt.[6,10] Rest, activity modification, anti-inflammatory medications, ice, local anaesthetics, and corticosteroid injections can aid in treatment.[2,5,6,8]

Dislocation of the patella occurs when the patella is displaced, usually laterally, from its normal joint position, completely separating the two joint surfaces. Dislocation is accompanied by tremendous pain and may be complete or incomplete.[32]

Pathophysiology of patellar dislocations can include direct trauma, a shallow trochlea, abnormal patellar structure or form, pre-existing malalignment, patella alta, weak medial stabilizers, or a tight lateral retinaculum. There may also be a positive family history.[2] Patella alta is associated with patellofemoral instability, patellar dislocations, and chondromalacia patellae.[2,6,8,10,18,29] Patellar position during patella alta increases the likelihood of medial or lateral movements of the patella during flexion. Patella alta is present when the inferior pole of the patella is positioned above the superior aspect of the femoral trochlear groove with the knee extended.[10]

Clinical features present after a dislocation episode include tenderness along the medial border of the patella,[3] patellar hypermobility, a large Q angle, VMO dysplasia, swelling, and a positive apprehension sign.[2,7] Patients with recurrent dislocations often display patella positioned laterally and tilted.[6,7] Patients are often free of pain during the time period around the dislocation.[5]

The primary special test associated with patellar dislocations is Fairbank's Apprehension Test. The patient will feel like the patella is going to dislocate as the examiner presses the patella laterally. The patient will be apprehensive and perform a quadriceps contraction to pull the patella back "into line" indicating a positive test.[2,3,6,16,31]

Diagnosis of patellar dislocations can be made through a complete history, physical examination, and axial radiographs to show patellar malalignment.[2] Treatment of recurrent dislocations requires surgery and a rehabilitation program.[39] Surgery is utilized only in the case of malalignment.[29] Lateral retinacular release or excision of a portion of the lateral retinaculum has been shown to have an overall 80% good to excellent results in patients with recurrent dislocations of the patella.[2,30,38,39,40]

CHAPTER SEVEN

DISCUSSION AND CONCLUSION

This chapter will contrast the similarities and differences between the patellofemoral pain syndromes previously discussed. Conclusions and recommendations will be based on my discovery throughout this literature review.

It was concluded that patellofemoral pain syndromes generally have an insidious onset, often affecting both knees (one greater than the other).[2,3,5] Depending upon the syndrome, patellofemoral pain presents at various ages. Plica syndrome is most often present during adolescents,[14] chondromalacia patellae can occur at any age and is age dependent,[2,5,8,29,31,35] with maltracking and instability problems most common in the younger athletic population.[6,34] Patellofemoral pain syndromes were found to be more common in females than in males.[2,5,6,8,9,10,24,31] However, these syndromes may be found in individuals of all ages, both genders, and various activity levels.[2,6,7]

The pathophysiology of each patellofemoral pain syndrome has been previously discussed. The pathophysiology was concluded to be associated with a variety of causes[2] secondary to direct or indirect trauma. The majority of individuals with patellofemoral joint pain have some form of malalignment that cause abnormal stresses (hyperpressure or hypopressure) on the articular cartilage.[5,10,34] There is controversy in the literature surrounding the etiology of patellofemoral pain especially chondromalacia patellae,[31,34,35] but these syndromes can all lead to eventual articular cartilage breakdown.[38] It is recommended that careful explaination of the condition and its causative factors will help the patient understand the cause of the dysfunction and the planned recovery.[4]

Conclusive signs and symptoms present with the patellofemoral pain syndromes discussed include: vague peripatellar pain that increases with activity or prolonged knee flexion, instability or "giving way" sensation, possible VMO atrophy and edema.[2,3,14,15,19,23,34] Patients with plica syndrome complain more often of snapping, popping and clicking along with catching episodes during activity.[8,14,19,20,23,25,40] Patients with chondromalacia patellae displayed retropatellar crepitation and have greater difficulty descending stairs.[6,8,24] Patients with patellar instability problems of subluxation and dislocation will complain of locking or popping, apprehension with running, cutting, and pivoting drills,[6,7,18] and may have hypermobile patella displaced superiolaterally.[8] Patellar maltracking signs and symptoms mimic other patellofemoral pain syndromes. A detailed history along with a thorough physical examination are necessary in the evaluation of the patellofemoral joint and should include inspection of the patella, the entire lower extremity, and patellar tracking throughout its range of motion.[1,34]

Clinical signs and symptoms of patellofemoral pain syndromes are often similar and difficult to distinguish. Arthroscopy or axial radiographs can be utilized as an aid in confirming the diagnosis. Plica syndrome and chondromalacia patellae utilize arthroscopy, while instability and maltracking syndromes utilize axial radiographs to aid in the diagnosis.[2,3,8,22]

In conclusion, it is recommended that the minimum evaluation of the patellofemoral joint should include a detailed history, a thorough physical examination, and routine radiographs for a precise diagnosis to be made.[2,5,18,22] Only then can the most appropriate rehabilitation program be created.

Initial treatment of all patellofemoral pain syndromes even subluxation and initial dislocations are through conservative management of rest, activity modification, modalities, exercises, and external support; which has been reported to be successful in many studies (70-90%).[1,2,3,4,5,6,7,8,14,15,19,24,26,27,28,29,30,34,35] The basis of conservative treatment was to strengthen the VMO, whose fibers provide the main counteraction to lateral patellar tracking.[34] An isometric progressive resistance exercise program for the quadriceps with IT band and hamstring stretching is the preferred initial treatment.[2,4] Isometric quadriceps exercise with the knee extended results in the least amount of patellofemoral contact force while maximally stressing the quadriceps muscle. It is unclear why patellofemoral pain decreases with exercise but it has been suggested that exercise improves patellar tracking, decreases patellofemoral contact forces, improves the nutrition to the joint and decreases edema.[34]

In conclusion, most authorities agree that surgery should not be considered unless no improvement has occurred after three to six months of conservative treatment. The purpose of surgery is to correct malalignment.[2,3,8]

REFERENCES

1. Shelton GL. Conservative management of patellofemoral dysfunction. *Prim Care* 1992;19(2):331-347.

2. Greenfield MA, Scott WN. Arthroscopic evaluation and treatment of the patellofemoral joint. *Orthop Clin North Am.* 1992;23(4):587-600.

3. Ruffin MT, Kiningham RB. Anterior knee pain: The challenge of patellofemoral syndrome. *Am Fam Physician.* 1993;47(1):185-194.

4. McConnell JS. Cited by: Zappala FG, Taffel CB, Scuderi GR. Rehabilitation of patellofemoral joint disorders. *Orthop Clin North Am.* 1992;23(4):555-565.

5. Tria AJ, Palumbo RC, Alicea JA. Conservative care for patellofemoral pain. *Orthop Clin North Am.* 1992;23(4):545-554.

6. Kujala UM. Patellofemoral problems in sports medicine. *Ann Chir Gynaecol.* 1991;80(2):219-223.

7. McConnell JS. Cited by: Walsh WM, Helzer-Julin M. Patellar tracking problems in athletes. *Prim Care.* 1992;19(2):303-330.

8. Davidson K. Patellofemoral pain syndrome. *Am Fam Physician* 1993;48(7):1254-1262.

9. Molnar TJ, Fox JM. Overuse injuries of the knee in basketball. *Clin Sports Med.* 1993;12(2):349-362.

10. Shellock FG. Patellofemoral joint abnormalities in athletes: Evaluation by kinematic magnetic resonance imaging. *Top Magn Reson Imaging.* 1991;3(4):71-95.

11. Messier SP, Davis SE, Curl WW, Lowery RB, Pack RJ. Etiologic factors associated with patellofemoral pain in runners. *Med Sci Sports Exerc.* 1991;23(9):1008-1015.

12. Nordin M, Frankel VH. *Basic Biomechanics Of The Muskuloskeletal System.* 2nd. edition. Malvern, PA: Lea & Febiger; 1989:115-117,122,128-131.

13. Snell RS. *Clinical Anatomy For Medical Students.* 4th edition. Boston, MA: Little, Brown and Company; 1992:19,623,669-670,705.

14. Nottage WM, Sprague NF, Auerbach BJ, Shahriaree H. The medial patellar plica syndrome. *Am J Sports Med.* 1983;11(4):211-214.

15. Tindel NL, Nisonson B. The plica syndrome. *Orthop Clin North Am* 1992;23(4):613-618.

16. Magee DJ. *Orthopedic Physical Assessment.* 2nd edition. Philadelphia, PA: W.B. Saunders Co; 1992:416-425.

17. Mann RW, Roberts CA, Thomas MD, Davy DT. Pressure erosion of the femoral trochlea, patella baja, and altered patellar surfaces. *Am J Phys Anthropol.* 1991;85:321-327.

18. Merchant AC. Classification of patellofemoral disorders. *Arthroscopy; J Arthroscopic and Related Surg.* 1988;4(4):235-240.

19. Hardaker WT, Whipple TL, Bassett FH. Diagnosis and treatment of the plica syndrome of the knee. *J Bone Joint Surg.* 1980;62A(2):221-225.

20. Broom MJ, Fulkerson JP. The plica syndrome: A new perspective. *Orthop Clin of North Am.* 1986;17(2):279-281.

21. Kinnard P, Levesque RY. The plica syndrome a syndrome of controversy. *Clin Orthop and Related Research.* 1984;183:141-143.

22. Malek MM, Fanelli GC. Patellofemoral pain an arthroscopic perspective. *Clin Sports Med.* 1991;10(3):549-567.

23. O'Dwyer KJ, Peace PK. The plica syndrome. *Injury.* 1988;19(5):350-352.

24. Saunders HD. *Evaluation, Treatment, and Prevention of Musculoskeletal Disorders.* Minneapolis, MN: Viking Press Inc.; 1985:165-167.

25. Munzinger U, Ruckstuhl J, Scherrer H, Gschwend N. Internal derangement of the knee joint due to pathologic synovial folds: The mediopatellar plica syndrome. *Clin Orthop and Related Research.* 1985;155:59-64.

26. Boucher JP, King MA, Lefebvre R, Pepin A. Quadriceps femoris muscle activity in patellofemoral pain syndrome. *Am J Sports Med.* 1992;20(5):527-532.

27. O'Neill DB, Micheli LJ, Warner JPJ. Patellofemoral stress a prospective analysis of exercise treatment in adolescents and adults. *Am J Sports Med.* 1992;20(2):151-156.

28. Finestone A, Radin E, Lev B, Sclamkovitch N, Wiener M, Milgrom C. Treatment of overuse patellofemoral pain. *Clin Orthop and Related Research.* 1993;293:208-210.

29. Insall J. Current concepts review. *J Bone Joint Surg.* 1982;64A(1):147-151.

30. McConnell JS. Cited by: Fulkerson JP, Kalenak A, Rosenberg TD, Cox JS. Patellofemoral pain. *Instr Course Lect.* 1992;41:57-71.

31. Mori Y, Kuroki Y, Yomamoto R, Fujimoto A, Okumo H, Kubo M. Clinical and histological study of patellar chondropathy in adolescents. *Arthroscopy.* 1991;7(2):182-197.

32. *Taber's Cyclopedic Medical Dictionary.* 16th edition. Philadelphia, PA: F.A. Davis Co; 1988:348,518.

33. Goodfellow J, Hungerford D, Woods C. Patello-femoral joint mechanics and pathology. 2. Chondromalacia patellae. *J Bone Joint Surg.* 1976;58B(3):291-299.

34. Doucette SA, Goble EM. The effect of exercise on patellar tracking in lateral patellar compression syndrome. *Am J Sports Med.* 1992;29(4):434-440.

35. Kelly MA, Insall JN. Historical perspectives of chondromalacia patellae. *Orthop Clin North Am.* 1992;23(4):517-521.

36. Royle SG, Noble J, Davies DRA, Kay PR. The significance of chondromalacic changes on the patella. *Arthroscopy.* 1991;7(2):158-160.

37. Brossman J, Muhle C, Schroder C, et al. Patellar tracking patterns during active and passive knee extension: Evaluation with motion-triggered cine MR imaging. *Radiology.* 1993;187:205-212.

38. Jackson RW, Kunkel SS, Taylor GJ. Lateral retinacular release for patellofemoral pain in the older patient. *Arthroscopy.* 1991;7(3):283-286.

39. Williams RM, Dymond JB. New outpatient treatment of recurrent patellar dislocations. *Orthop Rev.* 1992;21(11):1329-1332.

40. Koh TJ, Grabiner MD, De Swart RJ. *In vivo* tracking of the human patella. *J Biomech.* 1992;25(6):637-643.

APPENDIX

PLICA SYNDROME

PRESENTATION:	Most Common During Adolescents May Occur At Any Age Rare Occurrance
PATHOPHYSIOLOGY:	Spontaneously With Direct Trauma Insidious Onset From Indirect Trauma i.e. overuse, synovitis
SIGNS & SYMPTOMS:	Nonspecific Or Anteriomedial Intermittent Dull Aching Type Of Pain Pain Increases With Repetition Snapping, Popping, Or Clicking Weakness/Giving Way Sensation Catching Episodes Intermittent Edema Tight Sensation/Stiffness Theatre Sign
SPECIAL TESTS:	Palpation Will Reveal Joint Line And Medial Plica Tenderness + Patellar Grind/Apleys Compression Test + Mediopatellar Plica/Apprehension Test + Plica "Stutter" Test + Hughston's Plica Test/McMurrays Test
DIAGNOSIS:	One of Exclusion Detailed History Thorough Physical Exam Arthroscopy
TREATMENT:	Conservative Initially Excision

CHONDROMALACIA PATELLAE

PRESENTATION:	Age Dependent May Occur At Any Age Very common
PATHOPHYSIOLOGY:	Insidious Onset Unknown Etiology But Generally Accepted As Being Secondary To Patellar Malalignment
SIGNS & SYMPTOMS:	Retropatellar Crepitation Difficulty Descending Stairs Instability/Giving Way Sensation Quadriceps Atrophy Effusion Stiffness
SPECIAL TESTS:	Palpation Will Reveal Pain And Tenderness On The Lateral Facet Of The Patella + Clark's Sign + Waldron Test + McConnell Test + Passive Patellar Tilt Test + Lateral Pull Test + Zohler's Sign + Frund's Sign
DIAGNOSIS:	Detailed History Thorough Physical Examination Arthroscopy
TREATMENT:	Conservative Initially Arthroscopic Debridement

MALTRACKING

PRESENTATION:	Most Common In Young Females Often Bilateral Affecting One Knee > The Other Often a family history
PATHOPHYSIOLOGY:	Usually An Insidious Onset Deficiencies Of Supporting Muscles And Guiding Mechanisms Bony Abnormalities Lower Extremity Malalignment Trauma
SIGNS & SYMPTOMS:	Vague Peripatellar Or Medial Knee Pain Pain Increases After Activity i.e. hills, stairs, squatting, and prolonged sitting (+ Theatre Sign) Instability/Giving Way Snapping/Popping VMO Atrophy
SPECIAL TESTS:	Exaggerated Q angle Hamstring/Gastrocnemius Tightness Muscle Bulk Measurements + Patellar Glide + Passive Patellar Tilt Test + Obers Test
DIAGNOSIS:	Detailed History Thorough Physical Examination Axial Radiographs
TREATMENT:	Conservative Initially - Primarily Concerned With VMO Strengthening, Improving Hamstring Flexibility, And Patellar Mobility Surgery To Correct Malalignments

PATELLAR INSTABILITIES: LATERAL SUBLUXATION & DISLOCATIONS

PRESENTATION:	Common In Athletes Associated With Tremendous Joint Laxity
PATHOPHYSIOLOGY:	Large Peripatellar Effusions Tight Lateral Retinacular Structures & Weak Medial Stabilizers Direct Trauma Shallow Trochlear Groove Patella Alta Abnormal Patellar Structure Or Form Pre-existing Malalignment
SIGNS & SYMPTOMS:	Instability/Giving Way Sensation Patella Alta Medial & Lateral Retinacular Tenderness With Subluxations Pain During Dislocation Period Popping, Locking, & Apprehension With Running, Cutting, & Pivoting Drills Muscle Atrophy Swelling/Effusion Crepitus
SPECIAL TESTS:	Exaggerated Q Angle Hypermobile Patella Patella Alta Increased Sulcus Angle Edema VMO Dysplasia + Apprehension Test + Congruence/Lateral Patellofemoral Angle
DIAGNOSIS:	Detailed History Thorough Physical Examination Axial Radiographs
TREATMENT:	Conservative Initially Surgery To Correct Malalignment