THE EFFECTS OF A SIX WEEK ECCENTRIC EXERCISE PROGRAM ON KNEE PAIN, KNEE FUNCTION, QUADRICEPS FEMORIS AND HAMSTRING STRENGTH, AND ACTIVITY LEVELS IN PATIENTS WITH CHRONIC PATELLAR TENDINITIS

by

TYLER LEE DUMONT

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Department of School of Rehabilitation Sciences

The University of British Columbia
Vancouver, Canada

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Abstract

A non-concurrent multiple baseline design was used to evaluate the effects of a 6-week eccentric exercise program (EEP) on self-ratings of knee pain (intensity & unpleasantness), self-ratings of knee function, measures of isokinetic and isometric quadriceps femoris and hamstring muscle strength, and daily activity levels in four patients with chronic patellar tendinitis (CPT). Patients (3 female, 1 male, mean age 23.75 yrs) diagnosed with CPT provided informed consent to participate in this study. Repeated measures consisted of the: Descriptor Differential Scale, Knee Function Visual Analogue Scale, Kin Com isokinetic dynamometer for measures of isometric peak force of the quadriceps femoris and hamstrings of the affected and unaffected lower extremities, and Daily Activity Levels. Periodic isokinetic average torque measures of the quadriceps femoris and hamstrings of both lower extremities were also collected. The EEP consisted of a warm-up, stretching of the quadriceps femoris and hamstrings, progressive "drop squats", repeated stretching, and icing of the affected knee. Results were analyzed visually and statistically (C-statistic). Results of this study indicated reductions in ratings of intensity of knee pain (n=3) and ratings of unpleasantness of knee pain (n=2), improved ratings of knee function (n=3), increased isometric peak force of the quadriceps femoris of the affected side (n=4) and the unaffected side (n=3), minimal change in isometric peak force of the hamstrings of the affected and unaffected sides, and inconsistent changes in daily activity levels. Generally, isokinetic concentric and eccentric average torque increased for the quadriceps femoris of the affected and unaffected sides, however, minimal change occurred for the hamstrings of the affected and unaffected sides. The EEP appears to have clinical benefit for patients with CPT, however, due to the long-term nature of chronic tendinopathy, it appears the EEP may need to be applied for longer durations.

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LIST OF ABBREVIATIONS, SYMBOLS AND DEFINITIONS

AMSMC	Allan McGavin Sports Medicine Centre
CPT	chronic patellar tendinitis
DDS	Descriptor Differential Scale
EEP	eccentric exercise program
GTO	golgi tendon organ
L	left
MTU	muscle tendon unit
N	Newton
Nm	Newton-metre
p	probability
PFPS	patello-femoral pain syndrome
R	right
SRS	School of Rehabilitation Science
TLL	true leg length
UBC	University of British Columbia
VAS	visual analogue scale

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INTRODUCTION

Chronic overuse injury has been defined as a longstanding or recurring orthopaedic problem and pain in the musculoskeletal system, which results from repetitive tissue microtrauma during exertion.[1] Curwin & Stanish[2] suggested overuse tendon injury results from the application of repeated strains of 4-8% to the tendon. This may result in micro or macroscopic tendon disruption and manifest itself in a variety of tendinopathies (i.e. paratenonitis, tendinitis, tendinosis).[3]

Paratenonitis has been defined as inflammation of the paratenon alone and includes such terms as peritendinitis, tenosynovitis, and tenovaginitis.[4] Tendinitis is defined as tendon injury that results in an inflammatory repair response that may lead to secondary paratenonitis.[4] Tendinosis is defined as intra-tendinous degeneration without an inflammatory response.[4]

Chronic tendinopathy is considered to be primarily a degenerative process without the presence of significant inflammation.[3] The implication for treatment of patients with chronic tendinopathy is to adjust the focus away from relieving inflammatory symptoms to restoring function that is lost with degeneration.[5] The pathways that lead to tendinosis are not well understood.[6] Leadbetter[4] has proposed a theoretical model for the development of tendon degeneration that involves a failed cell matrix adaptation to excessive loading.

It has been suggested that exercise should be the cornerstone of treatment of patients with chronic tendinopathy.[7] The use of eccentric exercise in the rehabilitation of patients with chronic tendinopathy has been frequently advocated[7-15] since first being proposed by Curwin and Stanish.[2]

It has been reported there is little scientific evidence for the majority of treatments proposed and used for chronic tendinopathies.[3] Specifically, there is

a paucity of evidence for the use of the eccentric exercise program (EEP)[2] developed for the treatment of chronic tendinopathies.[16]

In their book, Curwin & Stanish[2] presented survey data of 200 patients with chronic tendinitis (chronic patellar tendinitis, Achilles tendinitis, and lateral epicondylitis). All patients were treated with the eccentric exercise program consisting of a warmup, stretching of the involved muscle-tendon unit (MTU), specific progressive eccentric exercises, repeated stretching, and application of ice to the involved MTU. They reported complete relief of symptoms in 44% of the subjects and marked reduction in pain and functional disability in an additional 43.5% of the subjects. These reports appear remarkable, however, these data were not published in a peer-reviewed journal.

Review of the literature indicates that only four groups of investigators have evaluated the efficacy of eccentric exercise in the treatment of chronic tendinopathy. Cannell's[17] unpublished master's thesis examined the effects of a 12 week eccentric squat program on patients with chronic patellar tendinitis (jumper's knee). Significant increases in hamstring strength and a significant reduction in pain levels were reported. The quadriceps strength measures did not improve significantly.

In 1989, Jensen and Di Fabio[18] examined the effects of eccentric quadriceps training on the Kin Com isokinetic dynamometer with patients having chronic patellar tendinitis. After the eight-week program, no significant increase in eccentric quadriceps work was detected in the affected lower extremities. The program was significantly different than that proposed by Curwin & Stanish[2] in terms of the number of repetitions and sets of the exercise performed. Jensen & Di Fabio[18] did not comment upon changes in function of the subjects during the intervention period. They[18] postulated that pain or a protective reflex may have prevented strength gains in the affected limb.

In 1992, Niesen-Vertommen et al.[19] compared eccentric and concentric plantarflexor strength training programs in patients with chronic Achilles tendinitis over a 12 week period. This program closely followed the recommendations of Curwin & Stanish [2] with the exception that the exercise was performed 6 days per week using 5 sets of 10 repetitions. The subjects reported significant reductions in pain with the eccentric training but did not demonstrate significant strength gains or faster return to activity compared to the concentrically-trained group.

Karlsson et al.[20] proposed and tested a protocol for the treatment of patients with jumper's knee based upon progressive strengthening and stretching of the quadriceps femoris muscles. Eccentric quadriceps strengthening was considered a key component of the treatment, however, guidelines for the progression of exercise were not provided. They[20] reported 70% of the involved knees demonstrated an "excellent" response (defined as no or minimal pain during heavy exertion, full return to sport, normal range of motion, none or mild inferior patellar pole tenderness) while the remainder of patients required surgery.

Three[17-19] of the above four studies indicate eccentric training may reduce pain in patients with chronic tendinopathy, however, improvement in strength of the involved MTU and function were not clearly demonstrated. These studies also employed eccentric exercise programs significantly different than that suggested by Curwin & Stanish[2]. The treatment program results of Karlsson et al.[20] suggested improved function, however, the program was not clearly defined. As a result, there remains a need to specifically test the EEP developed by Curwin & Stanish.[2]

The purpose of this study was to evaluate the effects of a 6 week EEP[2] on measures of quadriceps femoris and hamstring muscle strength, self-ratings

of knee pain, self-ratings of knee function, and self-ratings of activity levels in patients with chronic patellar tendinopathy. This was done using a single subject research design known as the non-concurrent multiple baseline design.[21] The following hypotheses were tested:

Null Hypotheses:

1. Following a 6-week EEP, there will be no change in ratings of the intensity of knee pain.
2. Following a 6-week EEP, there will no change in ratings of the unpleasantness of knee pain.
3. Following a 6-week EEP, there will no change in ratings of knee function.
4. Following a 6-week EEP, there will be no change in measures of average eccentric and average concentric torque of the quadriceps femoris and hamstring muscles of both lower extremities.
5. Following a 6-week EEP, there will be no change in measures of peak isometric force of the quadriceps femoris and hamstring muscles of both lower extremities.
6. Following a 6-week EEP, there will be no change in the amount of time engaged in physical activity.

Alternate Hypotheses:

1. Following a 6-week EEP, there will be a reduction in ratings of the intensity of knee pain.
2. Following a 6-week EEP, there will be a reduction in ratings of the unpleasantness of knee pain.
3. Following a 6-week EEP, there will be an increase in ratings of knee function (improved function).

4. Following a 6-week EEP, there will be an increase in measures of average eccentric and average concentric torque of the quadriceps femoris and hamstring muscles of both lower extremities.

5. Following a 6-week EEP, there will be an increase in measures of peak isometric force of the quadriceps femoris and hamstring muscles of both lower extremities.

6. Following a 6-week EEP, there will be an increase in the amount of time engaged in physical activity.

Preliminary Work Related to the Study

Several pilot studies were performed prior to beginning this study. The first pilot study involved examination of the intra-tester reliability of several measures used in the physical examination of subjects (Appendix 14). These measures included: Q-angle, true leg-length (TLL), and sub-talar joint neutral (STJN).

A second pilot study was completed to determine the test-retest reliability of measuring peak isometric torque of the hamstring muscles in a prone position on the Kin Com isokinetic dynamometer using normal subjects (Appendix 13).

METHODS

Subjects

Four subjects (3 female, 1 male) with chronic patellar tendinitis (non-traumatic origin) were recruited for this study. The sampling process was one of convenience whereby subjects with chronic patellar tendinitis were informed of the study, either by the physicians at the Allan McGavin Sports Medicine Centre (AMSMC) or one of the investigators (Tyler Dumont). Prior to beginning the study, informed consent of each subject was obtained.

Approval for this study was granted by the Clinical Screening Committee for Research Involving Human Subjects at the University of British Columbia.

The inclusion criteria were as follows:

a) Female or male aged 20-35 years.

b) Diagnosis of chronic patellar tendinitis (non-traumatic origin) (> 3 months) by either a physician at the AMSMC or the co-investigator (Tyler Dumont).

c) No other musculoskeletal or neurological condition (i.e. past history of knee trauma or surgery, Osgood-Schlatter's disease, Sinding-Larsen-Johansson disease).

d) No reported use of anti-inflammatory medication for 2 weeks prior to the study.

e) No other concurrent treatment to the affected area (i.e. electrical modalities, massage).

Table 1. Subject Characteristics

Subject	Gender	Age (yrs)	Height (cm)	Weight (kg)	Affected knee	Activities
#1	female	20	177	63	L CPT	varsity high jumper
#2	female	22	176	74	R CPT	recreational sports
#3	male	28	185	78	L CPT R PFPS	competitive volleyball
#4	female	25	170.7	65	R CPT R PFPS	national team rower

Setting

The study was conducted at the AMSMC, Vancouver, British Columbia in the exercise physiology lab and physical therapy clinic. The facility is staffed by sports medicine physicians, physical therapists, exercise physiologists, and

support staff. The study was conducted by the co-investigator, Tyler Dumont, and was supervised by Dr. Donna MacIntyre.

Instrumentation

Pre and Post Measures:

The Kin Com Isokinetic Dynamometer (model 125AP)(Chattanooga Group Inc., TN) was used to determine average eccentric and concentric torque of the quadriceps femoris muscle and hamstring muscles of both lower extremities prior to commencing the study and upon completion of the 6-week EEP. The Kin Com is self-calibrating during operation and provides an on-screen message if errors occur. The Kin Com contains a diagnostic program and calibration procedures should any errors occur. During static testing, Farrell & Richards[22] have reported the measurements of the Kin Com to be repeatable and accurate to known weights placed upon the dynamometer lever arm (Intraclass correlation coefficient or ICC=0.99). During dynamic testing of the knee extensors (quadriceps femoris) and flexors (hamstrings), Kramer[23] reported test-retest reliability (ICC) values for concentric and eccentric average and peak torques. The ICC values for male and female subject concentric average torque and eccentric average torque of the knee extensors at 45 degrees / second ranged from 0.82 to 0.84 and 0.81 to 0.83 respectively. The ICC values for male and female subject concentric average torque and eccentric average torque of the knee flexors at 45 degrees / second ranged from 0.75 to 0.80 and 0.77 to 0.83 respectively.

Repeated Measures:

The Descriptor Differential Scale (DDS)[24] (Appendix 3) was used to rate the intensity and unpleasantness of knee pain in the affected lower extremity following the performance of a provocative test (Appendix 2). Gracely & Kwilosz[24] have reported the DDS to be reliable in the assessment of pain

between hours one and two (Pearson product-moment correlations of 0.78 for unpleasantness and 0.82 for intensity). Additionally, MacIntyre[25] has reported test-retest reliability (Pearson-Product Moment Correlation Coefficients) of the DDS in a study examining delayed onset muscle soreness. The test-retest reliability of the intensity ranged from 0.72 to 0.99 over five test times (0-168 hr). The test-retest reliability of the unpleasantness ranged from 0.58 to 0.98 over five test times (0-168 hr).

Function ratings were collected using a subjective knee questionnaire employing modified visual analogue scales (knee function VAS[26]) (Appendix 4). This scale was developed to assess knee function in activities of daily living and sports for adult patients with various knee disorders. The reliability of the knee function VAS score for healthy individuals was r = 0.86.[27] The knee function VAS score has been correlated with two other knee function scales, the Lysholm score[28] and the Cincinnati score[29], with r values of 0.88 and 0.91 respectively.[27] Evaluation of the content validity of this questionnaire, translated into German, by 36 "experts" (trauma surgeons, orthopaedic surgeons) demonstrated 85% of the "experts" would use this questionnaire for their patients.[27]

A daily activity log was used to document the amount of time (minutes) engaged in physical activity throughout the duration of the study (Appendix 5).

The Kin Com was used to collect repeated measures of peak isometric force of the quadriceps femoris muscle and hamstring muscles of both lower extremities during the baseline and intervention phases. Reinking et al.[30] reported an ICC value of 0.81 (p=0.01) for male and female isometric quadriceps femoris muscle peak force measures using the Kin Com isokinetic dynamometer.

Test-retest reliability of hamstring peak isometric force in a prone position on the Kin Com demonstrated an r value of 0.986 for the left side and 0.927 for the right side (Appendix 13). According to the classification system of Richman & Makrides[31] these r values can be classified as "very reliable".

Procedures

Study Design

A non-concurrent multiple baseline design[21] was employed with four patients having chronic patellar tendinitis. The baseline phase involved the collection of the following measures: pre-measures of quadriceps femoris and hamstring average eccentric and concentric torque of both lower extremities (Kin Com), repeated measures (every 3 days) of self-ratings of knee pain (DDS), self-ratings of knee function (knee function VAS), time engaged in physical activity (daily log book), and peak isometric force of the quadriceps femoris and hamstring muscles of both lower extremities (Kin Com). The shortest baseline involved the collection of 5 sets of measures over 11 days. Each subsequent baseline was extended by gathering 2 additional sets of repeated measures. The longest baseline required 32 days to complete (11 sets of measures). This met the criterion set by Barlow & Hersen[32] in that at least three data points are needed in the baseline phase to determine a trend in the data. The subjects were randomly assigned to each baseline as they entered the study. The random assignment of subjects resulted in the following order: subject #1 - 5 repeated measures, subject #2 - 7 repeated measures, subject #3 - 9 repeated measures, and subject #4 - 11 repeated measures.

Each subject performed the EEP 7 days per week for 6 consecutive weeks. During the intervention phase, repeated measures of knee pain, knee function, time engaged in physical activity, and peak isometric torque were

collected every three days (providing approximately 14 sets of measures). Following the 6-week EEP, post-intervention measures of quadriceps femoris and hamstring average eccentric and concentric torque of both lower extremities were collected.

A follow-up test session was conducted approximately 5 weeks following completion of the intervention phase. Testing included the same repeated measures as used in the intervention phase. The only exception was subject #1, whose follow-up strength testing was done isometrically instead of isokinetically. Appendix 6 provides an outline of the study design. This design was chosen to evaluate the EEP on an individual basis and to assess the clinical relevance of any changes observed. This was achieved by systematic comparison of the baseline phase to the intervention phase. The benefit of this design was that as subjects became available, they were randomly assigned to a pre-determined baseline length and began the study.[21] The non-concurrent design also ruled out history as a threat to internal validity of the results.[21] However, this design is considered weaker than the concurrent multiple baseline because external factors related to the passage of time (i.e. seasons) may be different for each baseline.[33,34]

Test and Treatment Administration

The testing procedures for the pre and post measures (eccentric/concentric average torque of quadriceps and hamstrings of both lower extremities) and the repeated measures (knee pain, knee function, daily activity log, peak isometric torque of the quadriceps and hamstrings of both lower extremities) were identical for all subjects. The order of testing was as follows: pre-baseline measures (average torque - unaffected side followed by affected side), baseline phase and intervention phase repeated measures (pain ratings, function ratings, time engaged in physical activity, and peak isometric

force - unaffected side followed by affected side), and post-intervention measures (average torque - unaffected side followed by affected side). Prior to beginning the study, the subjects received an education session describing the study (Appendix 7). Prior to beginning the intervention phase, the subjects received a detailed education session on how to implement and progress the EEP (Appendix 8). The test procedures were as follows:

Diagnosis by Investigator:

The subjects underwent a physical examination to confirm the diagnosis of chronic patellar tendinitis by the co-investigator, Tyler Dumont (Appendix 9). This examination was performed prior to entry into the study. The examination included a subjective history and objective physical examination. Within the physical exam, two measures were documented (Q-angle and TLL). A pilot study determined the intra-rater reliability for these measures to be (Appendix 14).

Pre and Post measures:

Quadriceps Femoris and Hamstring Eccentric/Concentric Average Torque:

The Kin Com isokinetic dynamometer was used to collect data regarding average eccentric and concentric torque of the quadriceps femoris and hamstring muscles of the affected and unaffected lower extremities prior to beginning the baseline phase, and at the end of the intervention phase. Testing was done at an angular velocity of 45 degrees / second. This velocity was selected because it is readily achievable and it minimizes the effect of artifact of the leg accelerating to a selected velocity.[23]

The subjects were in a seated position with their backs against a support with the hips at 80 degrees of flexion. The pelvis was stabilized by a waist belt and two diagonal harnesses, crossing the shoulders, held the torso stable. The

rotational axis of the knee (lateral femoral epicondyle) was aligned with that of the dynamometer. The resistance cuff was positioned at a point on the lower leg that was 75% of the length of the fibula. All testing was conducted through a range of 90 degrees (90 degrees knee flexion to zero degrees knee flexion). The subjects were provided with a remote "kill" switch to the Kin Com that enabled them to terminate the testing at any time. Subject positioning is illustrated in Appendix 10.

As a warmup, the subjects performed three submaximal cycles and one maximal cycle for the muscle groups being tested (quadriceps femoris and hamstrings). The knee extensor cycle consisted of concentrically extending the knee from 90 degrees flexion to zero degrees flexion then immediately resisting the dynamometer as it pushed against the leg during the eccentric phase from zero degrees flexion to 90 degrees flexion. The knee flexor cycle consisted of concentrically flexing the knee from zero degrees flexion to 90 degrees flexion then immediately resisting the dynamometer as it pushed against the leg during the eccentric phase from 90 degrees flexion to zero degrees flexion. Following this practice session the subject received a two minute rest period.

Testing involved 4 cycles of maximal effort for the muscle group being tested. All torques were corrected for the effect of gravity on the lower leg segment and the resistance cuff of the dynamometer. During testing, all subjects received moderately loud verbal encouragement. Following the testing, ice was be applied to the affected knee for 15 minutes to minimize potential exacerbation of knee symptoms.

Repeated Measures:

Pain Rating: The DDS (Appendix 3) was used to rate the intensity and unpleasantness of knee pain immediately following the provocative test (Appendix 2) during each measurement interval of the baseline and

intervention periods. The DDS was completed on paper at the AMSMC. The 12 descriptor items for each of the two dimensions assessed were randomly ordered at each assessment to reduce the effects of subject recall. For each descriptor the subject indicated the magnitude of the intensity or unpleasantness as equal, greater, or lesser to that implied by the descriptor term by placing a mark upon a line of 21 dashes. The dashes represented a numerical range of zero to twenty. Thus, a score ranging from zero to 20 could be assigned to each descriptor term. A mean score was calculated for the 12 descriptors in each dimension of pain.

Function Rating: In the knee function VAS (Appendix 4), a modified visual analogue scale was provided for each of the 28 questions pertaining to knee function. The VAS for each question has a highest score of 10 representing "no handicap" and a lowest score of 1 representing "maximum handicap". The scores, having a range from 28 to 280, were converted to percentages (10 to 100). A relative score was calculated to compensate for any unanswered questions. For each self-rating of knee function, the subject completed a copy of the knee function VAS questionnaire.

Peak Isometric Force: The Kin Com isokinetic dynamometer was used to collect measures of peak isometric force of the quadriceps femoris and hamstring muscles of both lower extremities. The positioning for quadriceps femoris testing was identical to that used in the pre and post tests of average torque (Appendix 10). Peak isometric force of the quadriceps was measured at 60 degrees of knee flexion. The positioning for hamstring testing was identical to that used in the pilot study performed by the co-investigator (Appendix 11). Peak isometric force of the hamstrings was measured at 30 degrees of knee flexion. The angular velocity was set at zero degrees per second and the subject performed a 5 second isometric contraction. Isometric testing of the

quadriceps was performed in sitting with the knee at 60 degrees of flexion and the hip in 95 degrees of flexion.[30] Isometric testing of the hamstrings was performed in a prone position with 30 degrees of knee flexion and the hip in zero degrees of extension.[35] Four warm-up contractions, at approximately 50% effort, were performed by the knee extensors and knee flexors. Four maximal trials were performed for the knee extensors and knee flexors, with the peak torque recorded.

Time Engaged in Physical Activity: A daily activity log sheet (Appendix 5) was completed by each subject throughout the baseline and intervention phase. The daily activity log sheets were collected by the co-investigator at the beginning of each repeated measure test session.

Intervention Phase:

Eccentric Exercise Program: Prior to commencing the EEP, all subjects received a verbal description of the EEP and how to progress the program (Appendix 8). Compliance to the EEP was recorded in the bottom of the daily log sheets by each subject. Subjects were educated to perform the EEP 7 days per week. The components of the daily program were as follows:

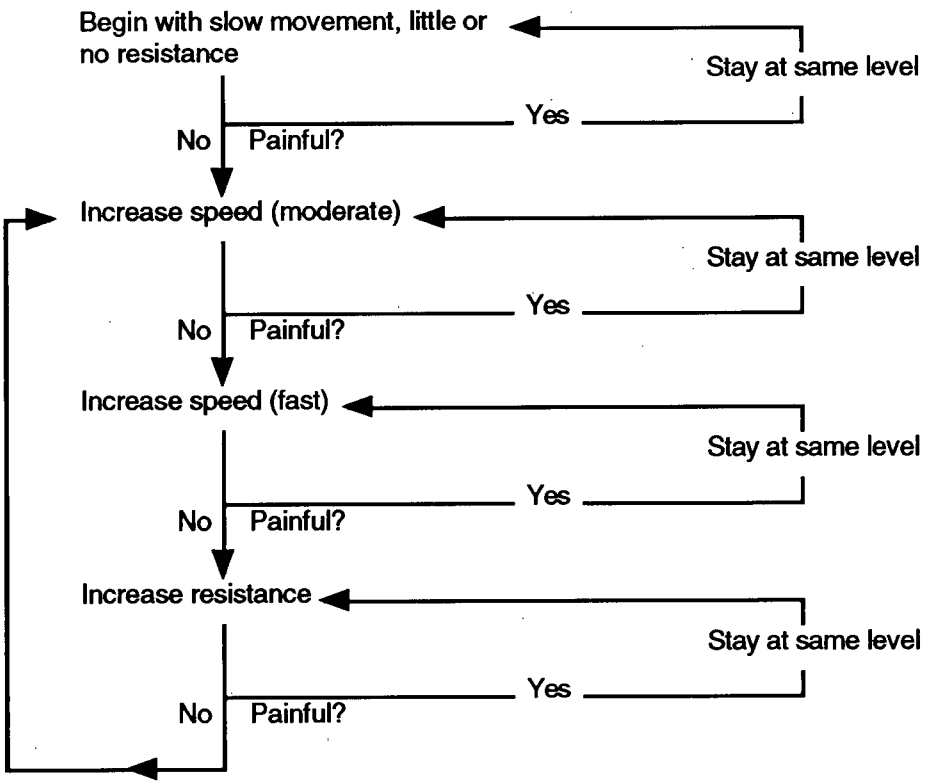
Warmup: A general body warmup was performed (pushups, situps) for 5 minutes.

Stretching: The subject performed three-30 second stretches to the quadriceps femoris and hamstring muscle groups of both lower extremities. The quadriceps femoris muscle was stretched in two different positions (Appendix 12). The first position was in standing and focused on the vastii muscles while the second position was in half-kneeling and focused on the rectus femoris muscle. The hamstring muscles were stretched in one position (Appendix 12).

Eccentric Exercise: The eccentric exercises ("drop" squats) were performed independently at home and exercise application and progression was reviewed

with each subject during the collection of repeated measures throughout the intervention phase. Three sets of ten repetitions of the "drop" squat exercises were performed daily, seven days per week, for 6 consecutive weeks. When the subjects reached the level requiring external loading, hand weights were provided from the SRS. The initial progression of external loading was 10% of body weight, with subsequent increases of 5% of body weight.[2] Progression of the EEP is illustrated in Figure 1.

Figure 1. Progression of the EEP (Curwin & Stanish, 1984)



Stretching: Following the "drop" squats, the subjects repeated the quadriceps femoris and hamstring muscle stretches as described earlier.

Ice: At the end of each session, cold (i.e. bag of crushed ice, frozen vegetables) was applied to the affected knee for 15 minutes.

Data Collection

Pre and Post Test Measures:

Average Torque: During average torque measures, all data were collected by the Kin Com on the four maximal eccentric and concentric contractions for each muscle group. The data were saved on the hard drive of the Kin Com computer system under a code name to preserve the confidentiality of the subjects. A backup copy of all data (floppy disk) was stored in a locked filing cabinet at the School of Rehabilitation Sciences (SRS), University of British Columbia.

Repeated Measures:

All completed DDS forms, knee function VAS forms, and daily activity log sheets were stored in a locked filing cabinet at the SRS.

Peak Isometric Force: During peak force measures, all data were saved on the Kin Com computer system under a code name to preserve the confidentiality of the subjects. A backup copy of all data (floppy disk) was stored in a locked filing cabinet at the SRS.

Data Management and Analysis

Pre and Post Test Measures:

The data collected during the quadriceps femoris and hamstring strength testing were analyzed by the Kin Com computer system as average eccentric torque and average concentric torque (Newton-metres) over repetitions 2 to 4. The data were plotted for each subject to provide a descriptive view of any changes in eccentric and concentric quadriceps and hamstring muscle strength. Statistical analysis of these data was not possible due to the small sample size (n=4).

Repeated Measures:

The DDS data were hand-scored and an average score out of 20 was calculated for each dimension of pain. The data collected by the VAS questionnaire were hand-scored and a percentage score ranging from 10 to 100 was calculated. The data collected from the daily log sheet provided a time value, in minutes, for the amount of physical activity performed each day. The data collected during peak isometric force testing were analyzed by the Kin Com computer system and an average peak force (Newtons) was calculated from the four maximal efforts of the quadriceps femoris and hamstring muscles.

Graphing of Data Series:

The results of this study have been displayed graphically as simple line graphs with vertical solid lines separating phases (Figures 2-17, 26-29). The phase labelled "baseline" represented the time frame where dependent variable measures were collected without the independent variable being applied (ranging from 2 to 5 weeks). The phase labelled "intervention" represented the time frame where dependent variable measures were collected during application of the independent variable (approximately 6 weeks). The phase labelled "follow-up" represented a final collection of dependent variable measures at approximately 5 weeks following the intervention phase. A series of composite graphs for all four subjects for each dependent variable measure are presented in Appendix 18. The dependent variable measures were also graphed individually in a larger scale for each subject to facilitate visual analysis (Figures 2-17, 26-29). This series of graphs also contains baseline linear regression lines (least squares method). The regression lines have been extended into the intervention and follow-up phases to aid in visual analysis of trend. The x-axes (horizontal) for all graphs represented the passage of time in

terms of days and the y-axes represented the respective dependent variable measure.

The graphs representing the isokinetic measures of average torque of the quadriceps and hamstring muscles have been graphed in a different format than described above (Figures 18-25). The x-axis has been labelled as "pre-test", "post-test", and "follow-up test". The y-axis represented average torque (Nm).

Visual Analysis of Data:

Visual analysis of single subject research data is considered by some authors to be an adjunct to statistical analysis.[36] However, single-subject researchers have typically relied upon visual analysis for the interpretation of experimental effects.[37] For the purposes of this study, both visual and statistical analytical techniques have been applied.

Visual analysis was used to describe changes in level, variability, trend within the data when apparent for all repeated measures for all subjects. Level is defined as the relative value of the data pattern on the dependent variable.[37] Changes in level indicate changes in the value or magnitude of the data series as measured on the outcome variable at the point of intervention.[38] Trend is defined as the direction in which the data are progressing.[37] An accelerating trend is one that consistently progresses in an upward direction. A decelerating trend is one that consistently progresses in a downward direction. Variability is defined as the amount of fluctuation of data points in a series.[38]

Statistical Analysis of Data:

The C-statistic[39] (Appendix 17) was applied to the baseline data points of all dependent variable measures with the exception of the isokinetic measures. The C-statistic was chosen because it can be applied to serially dependent data and small data sets (8 or more in series).[39] This statistic has

been applied to baseline data sets with fewer than 8 in a series (subjects #1 and #2) in this study and is acknowledged to be a limitation within the study. This statistic provided a numeric estimate of the stability (variability) of a data set and allowed the researcher to make quantitative statements about variability and the presence or absence of trends in the data.[38] The alpha level was set at $p < 0.05$.

A non-significant baseline C-statistic indicates no significant trend exists in the baseline data series. A C-statistic can then be computed using the entire data series. A significant C-statistic calculated from the entire data sets indicates there is a statistically significant trend across baseline and intervention phases.

A significant baseline C-statistic indicates a significant trend exists in the baseline data series and precludes the calculation of a C-statistic for the entire data set, unless a transformation of the data series is performed.[38] For the purposes of this study, when significant baseline C-statistics were obtained, no data transformations were performed and the statistical analysis was terminated.

RESULTS

Throughout the results, "affected side" will refer to the lower extremity having CPT, while "unaffected side" will refer to the lower extremity with PFPS (subjects #3 and #4) or no musculoskeletal disorder (subjects #1 and #2).

Descriptor Differential Scale

Subject #1 (Figure 2)

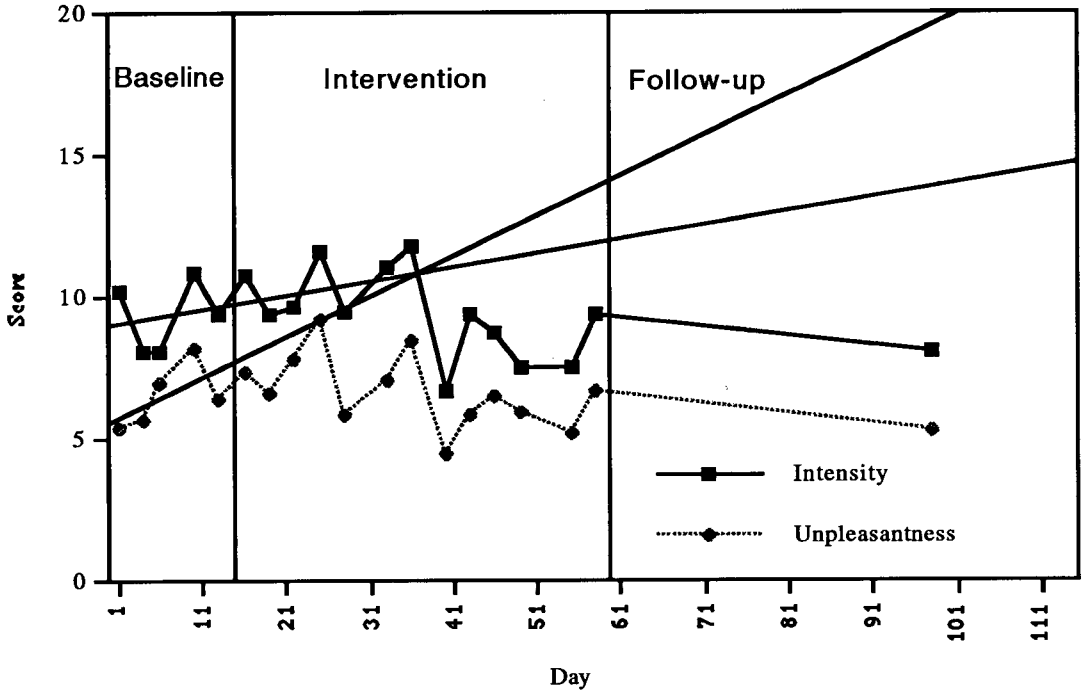
Intensity: The baseline linear regression line demonstrated an accelerating trend in intensity ratings. At approximately 3 weeks into the intervention phase, the trend appeared to show a deceleration or downward effect. An increase in variability of data points was apparent in the intervention phase. At approximately 3 weeks into the intervention phase, the level appeared to be lower than that of the data points in the baseline phase. At follow-up, the intensity level remained at the same level as the last few weeks of the intervention phase.

The C-statistic calculated for the baseline phase and the entire data set (baseline and intervention) demonstrated no significant trend in the data ($p < 0.05$).

Unpleasantness: The baseline linear regression line demonstrated an accelerating trend in unpleasantness ratings. At approximately 3 weeks into the intervention phase, the trend appeared to show a downward effect. An increase in variability of data points was apparent in the intervention phase. No obvious change in level between baseline or intervention phase was apparent. All data points for unpleasantness were below the intensity data points. At follow-up, unpleasantness remained at the same level as the last few weeks of the intervention phase.

The C-statistic calculated for the baseline phase and the entire data set (baseline and intervention) demonstrated no significant trend in the data ($p < 0.05$).

Figure 2. Descriptor Differential Scale - Subject #1 (L CPT)



Subject #2 (Figure 3)

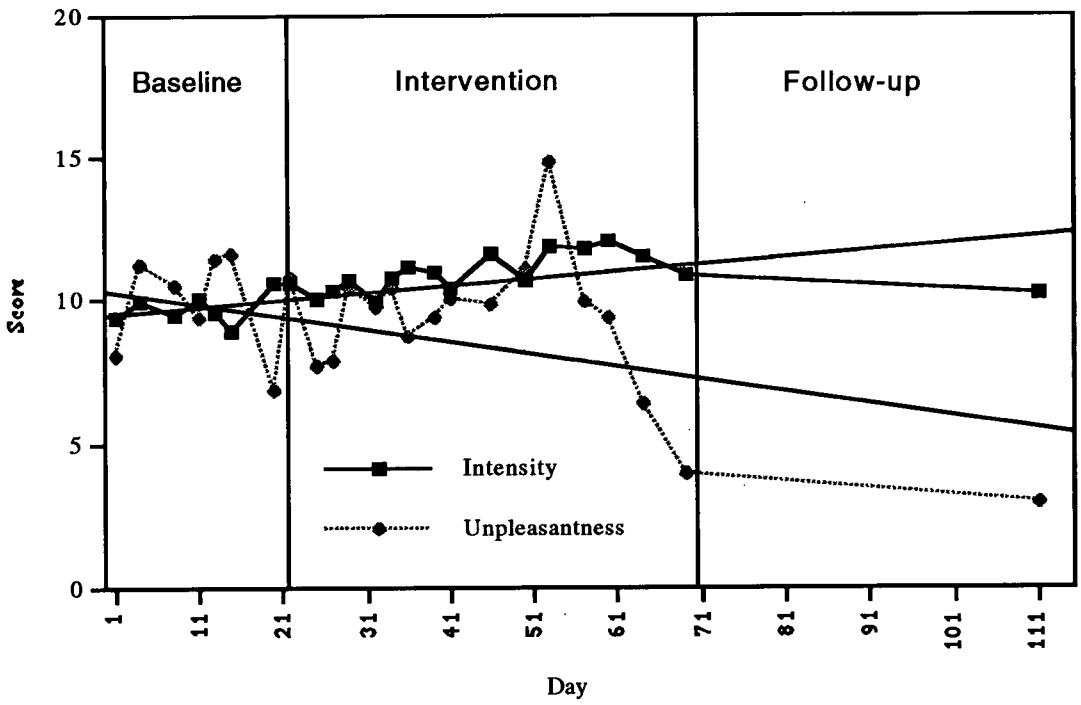
Intensity: The baseline linear regression line demonstrated an accelerating trend in intensity ratings. The intervention phase data points followed the regression line closely and no change in variability or trend of the data points between phases was noted.

The C-statistic calculated for the baseline phase demonstrated no significant trend in the data ($p < 0.05$). However, a significant trend ($p < 0.05$) was detected when the C-statistic was applied to the entire data set. This indicated a significant increase in intensity occurred during the intervention phase. At follow-up the intensity level returned to that of the baseline.

Unpleasantness: The baseline linear regression line demonstrated a decelerating trend in unpleasantness ratings. During the first 4 weeks of the intervention phase, unpleasantness showed an accelerating trend. This was followed by a sudden change to a decelerating trend that passed below the regression line. The intervention phase trend was similar to that shown in subject #1's DDS ratings of intensity and unpleasantness. At follow-up, unpleasantness reached its lowest point, and was markedly lower than intensity.

The C-statistic calculated for the baseline phase and the entire data set demonstrated no significant trend in the data ($p < 0.05$).

Figure 3. Descriptor Differential Scale - Subject #2 (R CPT)



Subject #3 (Figure 4)

Intensity: The baseline linear regression line demonstrated an accelerating trend in intensity ratings. Increased variability of the intervention data points was noted. All intervention data points fell below the baseline linear

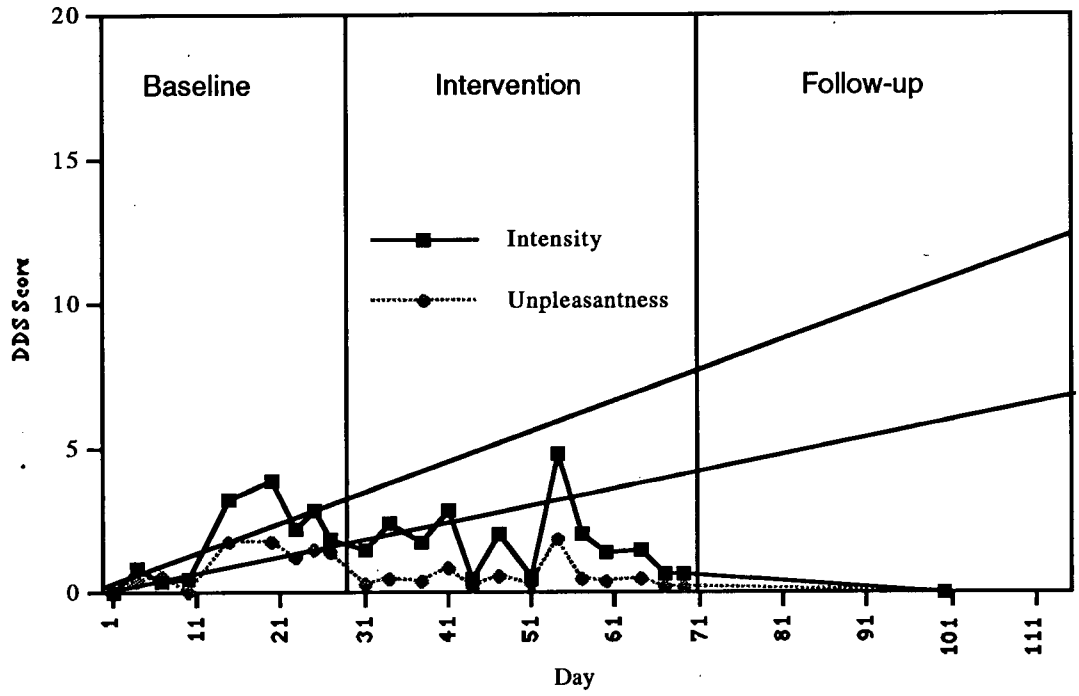
regression line. At follow-up, intensity was rated as zero. The scores were low throughout the study and suggested that the provocative test (Appendix 2) was ineffective for this subject.

The C-statistic calculated for the baseline demonstrated a significant trend ($p < 0.05$). Consequently the C-statistic was not calculated for the entire data set.

Unpleasantness: The baseline linear regression line demonstrated an accelerating trend in unpleasantness ratings. A reduction in variability of the intervention data points was noted. All intervention data points fell below the baseline linear regression line. At followup, unpleasantness was rated as zero. The scores were low throughout the study and suggested that the provocative test (Appendix 2) was ineffective for this subject.

The C-statistic calculated for the baseline demonstrated a significant trend ($p < 0.05$). Consequently the C-statistic was not calculated for the entire data set.

Figure 4. Descriptor Differential Scale - Subject #3 (L CPT, R PFPS)



Subject #4 (Figure 5)

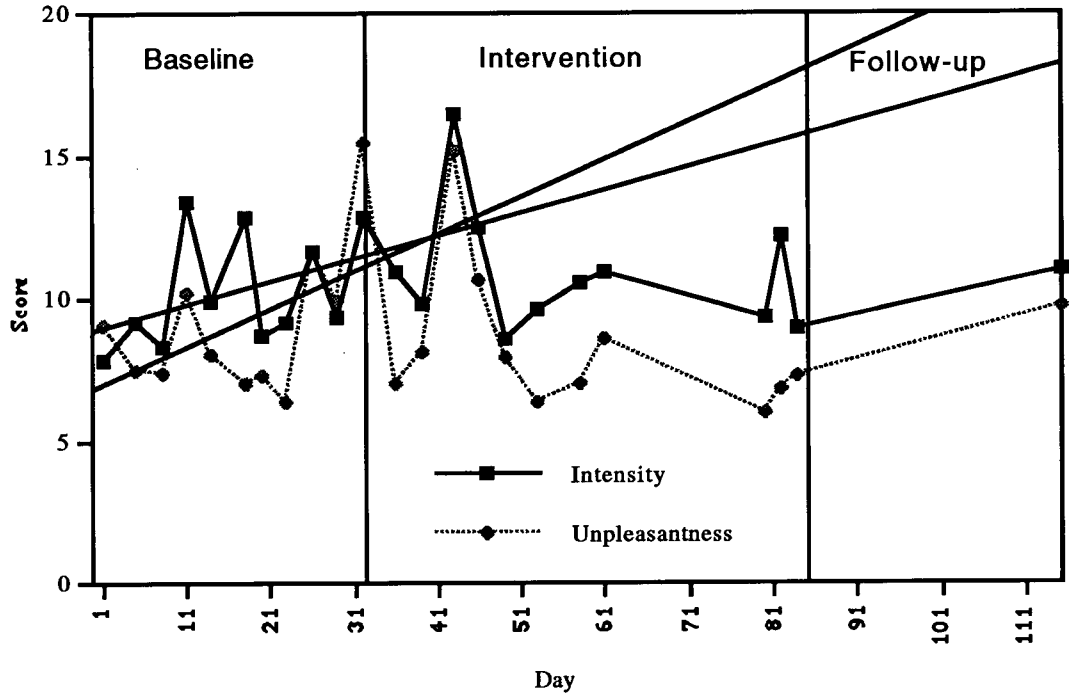
Intensity: The baseline linear regression line demonstrated an accelerating trend for intensity ratings. At approximately 3 weeks into the intervention phase, a reduction in intensity ratings was noted. The last 3 weeks of the intervention also demonstrated no trend. At follow-up, the intensity rating remained at the same level as during the last half of the intervention phase.

The C-statistic calculated for the baseline demonstrated a significant trend ($p < 0.05$). Consequently the C-statistic was not calculated from the entire data set.

Unpleasantness: The baseline linear regression line demonstrated a steeper accelerating trend in unpleasantness when compared to intensity. At approximately 3 weeks into the intervention phase, a reduction in unpleasantness ratings paralleled the reduction in intensity ratings. At follow-up, the unpleasantness value was elevated compared to the last half of the intervention phase.

The C-statistic calculated for the baseline demonstrated a significant trend ($p < 0.05$). Consequently the C-statistic was not calculated from the entire data set.

Figure 5. Descriptor Differential Scale - Subject #4 (R CPT, L PFPS)



Summary of DDS data: Throughout the study, the intensity ratings were generally greater than that of unpleasantness. A pattern of increased symptoms during the first 2-3 weeks of the EEP was shown for subject #1 (intensity, unpleasantness), subject #2 (unpleasantness), and subject #4 (intensity, unpleasantness).

During the end of the intervention phase, the majority of the intensity data points fell below the baseline linear regression line's for subjects #1, #3, and #4. This indicated a change toward a downward trend of intensity ratings. A similar pattern was demonstrated for the unpleasantness ratings for subjects #1, #3 and #4. This indicated a change toward a downward trend of unpleasantness ratings.

At follow-up, two subjects (#1,#2) demonstrated a maintenance of intensity and unpleasantness levels. Subject #4 demonstrated an increase in

unpleasantness ratings. Subject #3 reported no symptoms with the provocative test.

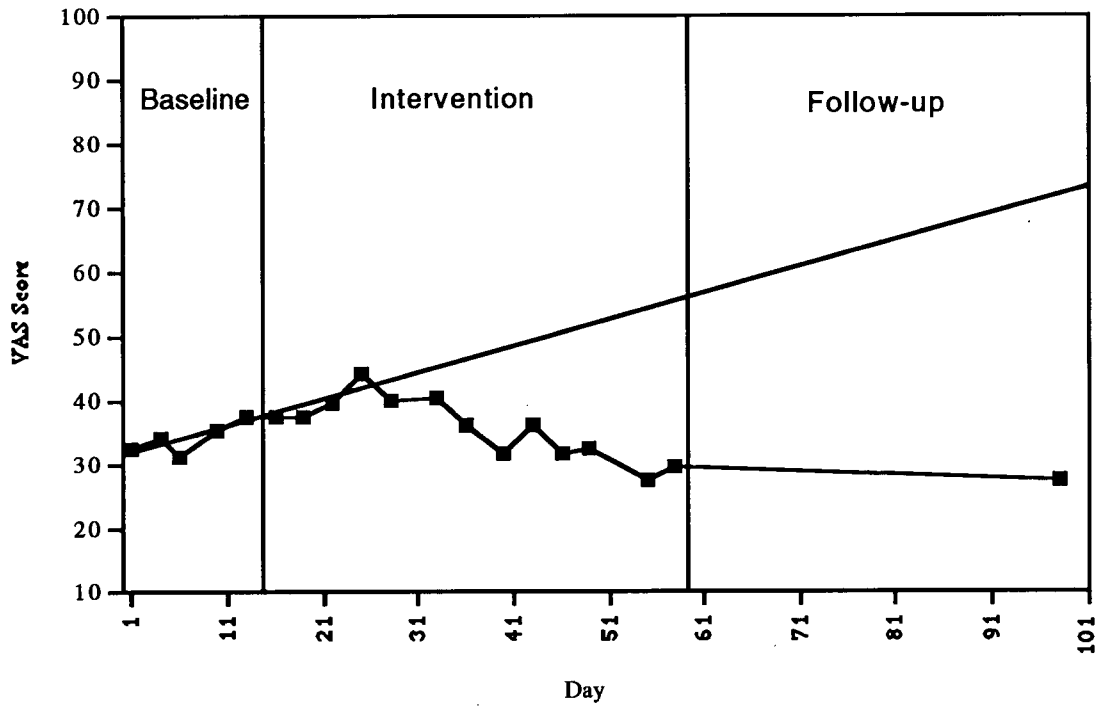
Knee Function VAS

Subject #1 (Figure 6)

The baseline linear regression line demonstrated an accelerating trend (worsening knee function). The majority of intervention data points fell below the linear regression line. At three weeks into the intervention, the VAS scores demonstrated a decelerating trend (improving knee function). No change in variability of data points was noted between baseline and intervention phases. At follow-up, the VAS score reached its lowest value and indicated continued improvement in knee function.

The C-statistic calculated for the baseline data set was not significant. The C-statistic calculated for the entire data set was significant ($p < 0.05$), and indicated a significant improvement in knee function.

Figure 6. Knee Function VAS - Subject #1 (L CPT)

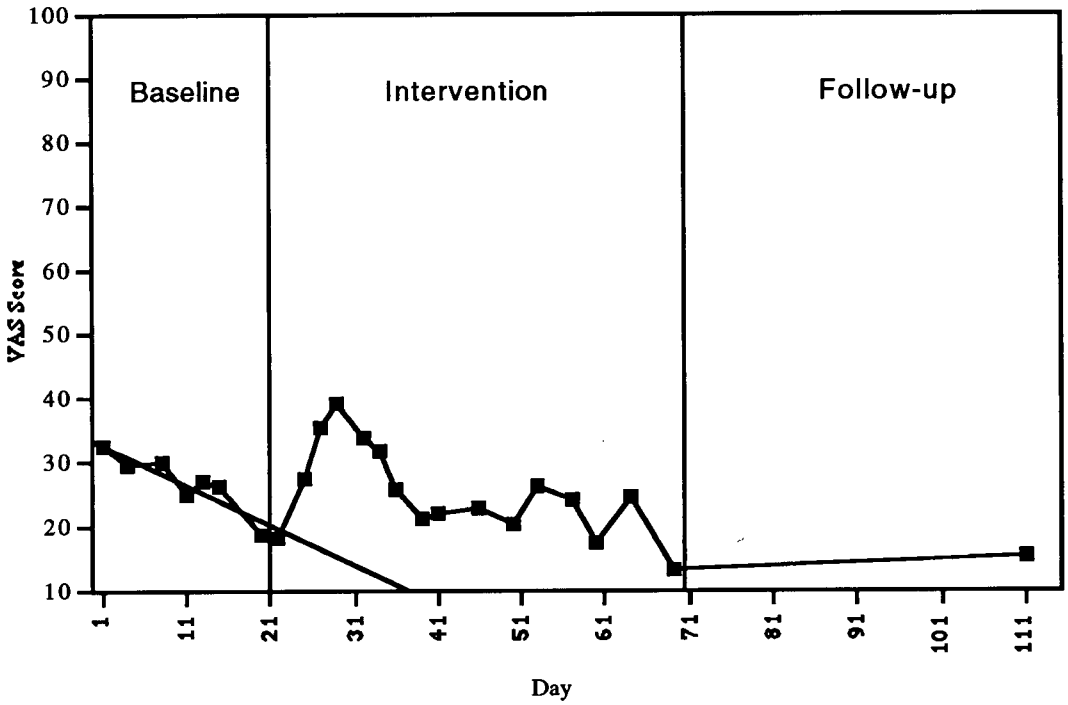


Subject #2 (Figure 7)

The baseline linear regression line demonstrated a steep decelerating trend (improving knee function). All intervention data points were above the regression line. An increase in VAS scores was noted in the first 2 weeks of the intervention phase. The following 4 weeks demonstrated a continuation of the decelerating trend (at a level above the baseline linear regression line). At follow-up, the VAS score remained at the same level as the latter part of the intervention phase.

The C-statistic calculated for the baseline demonstrated a significant trend ($p < 0.05$). Consequently the C-statistic was not calculated from the entire data set.

Figure 7. Knee Function VAS - Subject #2 (R CPT)

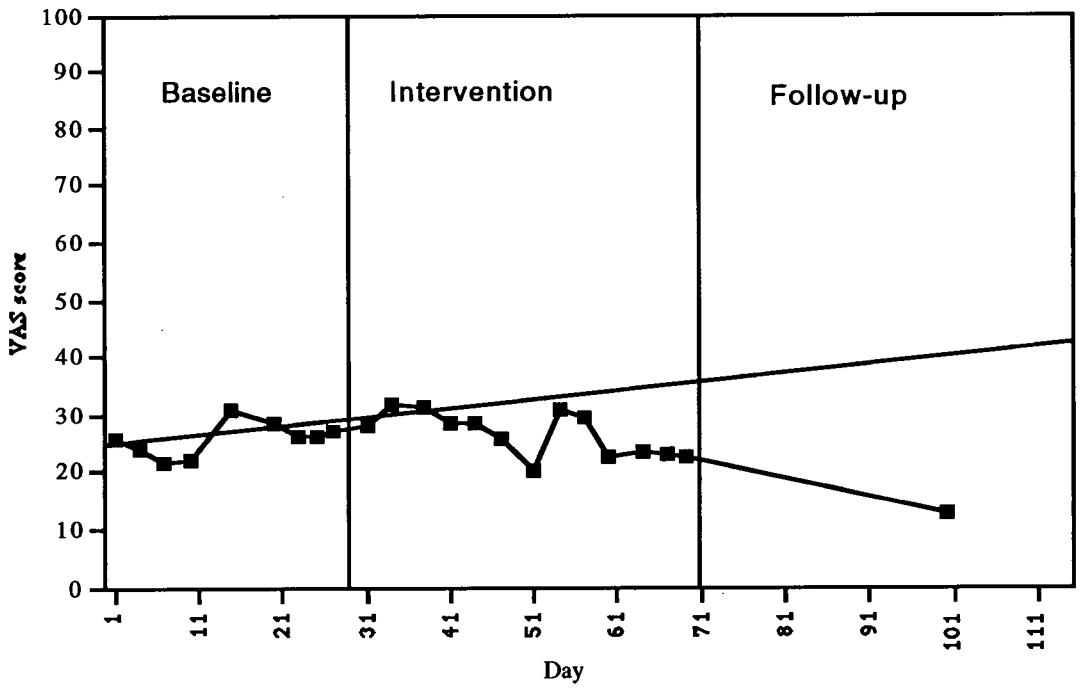


Subject #3 (Figure 8)

The baseline linear regression line demonstrated a slight accelerating trend. The majority of intervention data points fell below the baseline linear regression line and demonstrated a decelerating trend. At follow-up, the VAS score reached its lowest value and indicated continued improvement in knee function.

The C-statistic calculated for the baseline demonstrated a significant trend ($p < 0.05$). Consequently the C-statistic was not calculated from the entire data set.

Figure 8. Knee Function VAS - Subject #3 (L CPT, R PFPS)



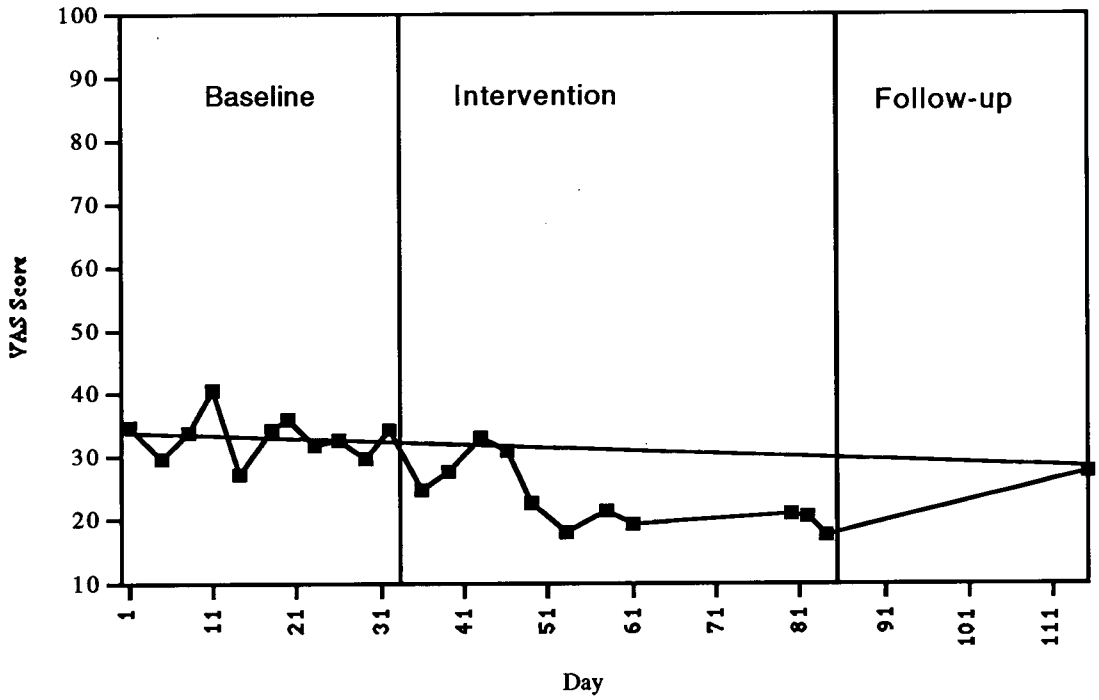
Subject #4 (Figure 9)

The baseline linear regression line demonstrated a slight decelerating trend. The majority of intervention data points fell below the baseline linear regression line and demonstrated a continued decelerating trend. The follow-up

VAS score rose to the level shown in the early part of the intervention phase and indicated a worsening of knee function.

The C-statistic calculated for the baseline was not significant. However, a significant value ($p < 0.05$) was present when the C-statistic was applied to the entire data set. This indicated a significant improvement in knee function in the intervention phase.

Figure 9. Knee Function VAS - Subject #4 (R CPT, L PFPS)



Summary of VAS scores: A decrease in knee function (increased VAS scores) was noted in the first 2-3 weeks of the intervention phase for three of the subjects (#1,#2,#4). This may be considered consistent with the expected aggravation of symptoms in the first 2-3 weeks of the EEP. A decelerating trend (improved function) was present in the second half of the intervention phase for all subjects. At follow-up, three subjects (#1-3) demonstrated a maintenance of

improved knee function obtained during the intervention phase. Subject #4 demonstrated a loss of knee function upon follow-up.

Isometric Quadriceps and Hamstrings Peak Force

Subject #1 (L CPT)

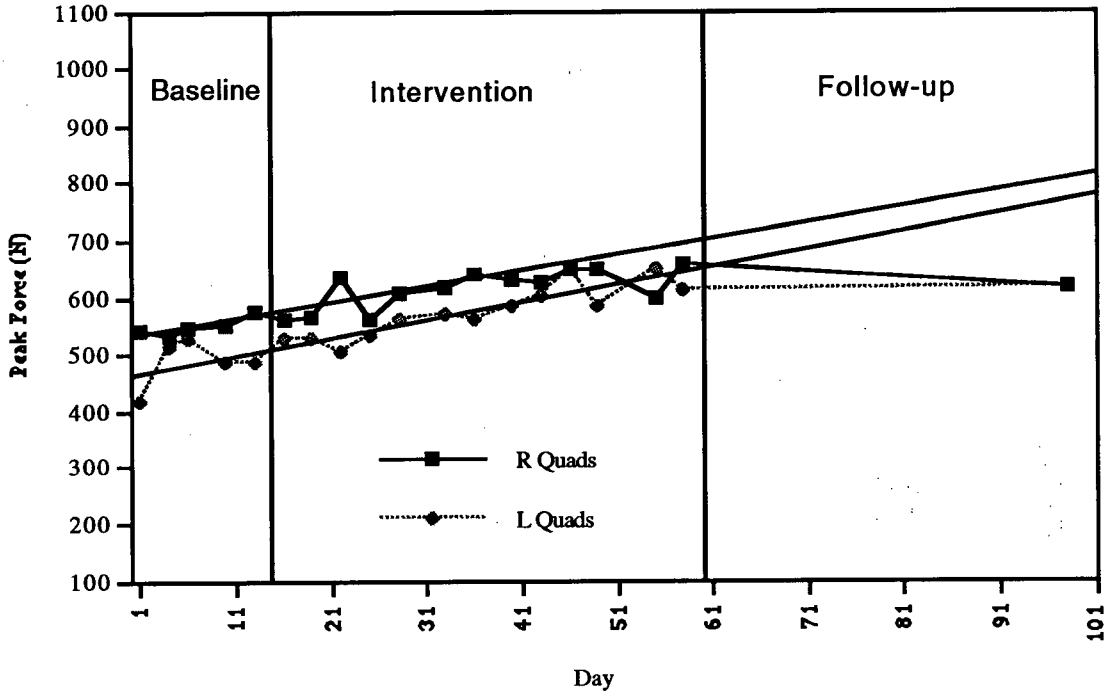
Quadriceps: (Figure 10)

During the baseline, both unaffected (R) and affected (L) sides demonstrated accelerating baseline linear regression line's with similar slopes. The unaffected side (R) was at a higher level than the affected side (L). The baseline C-statistics for both the unaffected (R) and affected (L) sides were not significant.

During the first 5 weeks of the intervention phase, the unaffected side (R) data points followed the baseline linear regression line (no change in trend). During the last week of this phase, the last 2 data points appear to show a reduction in the rate of strength gains. The majority of these data points remained above the data points for the affected side (L). During this phase, the affected side (L) data points closely followed the baseline linear regression line (no change in trend). During the last week of the intervention phase, the unaffected side (R) and affected side (L) data points appeared to be at the same level. A significant value ($p < 0.05$) was present when the C-statistic was applied to the entire data set (baseline and intervention data points) for both the unaffected (R) and affected (L) sides, and indicated that both sides demonstrated a significant improvement in strength.

At follow-up, both unaffected (R) and affected (L) sides demonstrated very similar values and remained elevated in comparison to baseline values. This demonstrated a maintenance of the strength gains achieved during the intervention phase. Isometric follow-up testing was only done for subject #1.

Figure 10. Isometric Quadriceps Peak Force - Subject #1 (L CPT)



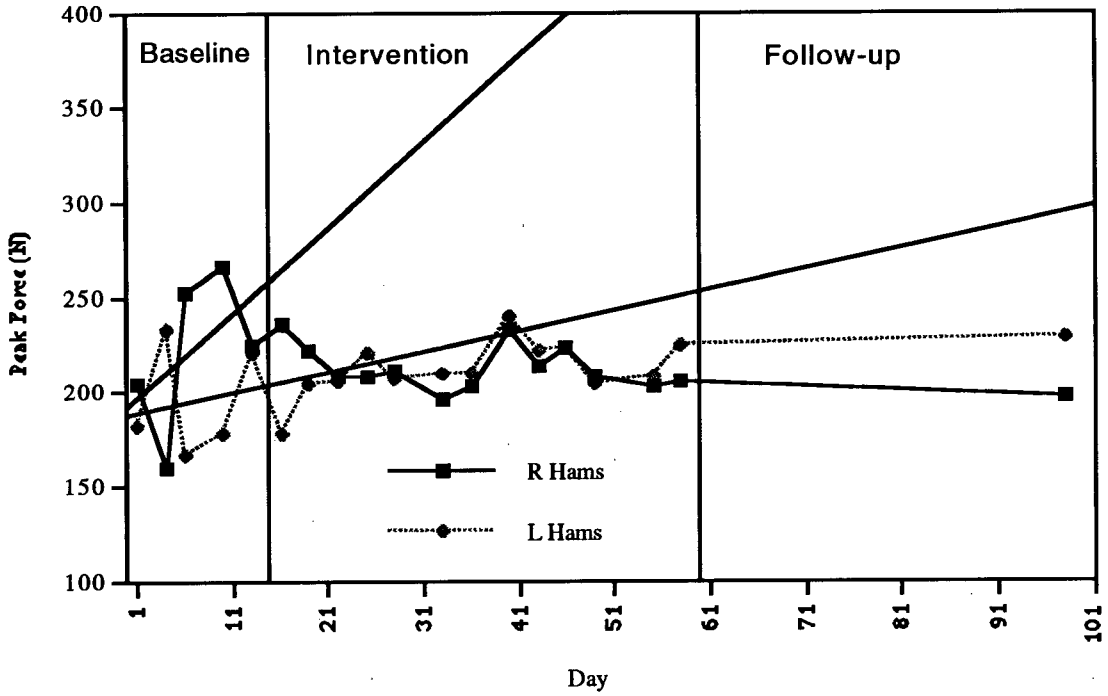
Hamstrings: (Figure 11)

During the baseline, the unaffected side (R) demonstrated a steep accelerating baseline linear regression line with considerable variability of the data points. The affected side (L) demonstrated an accelerating baseline linear regression line with considerable variability of the data points. The baseline C-statistics for both the unaffected (R) and affected (L) sides were not significant.

During the intervention phase, all unaffected side (R) data points fell below the baseline linear regression line and demonstrated reduced variability. These data points were level (no trend). The majority of affected side (L) data points fell below the baseline linear regression line and demonstrated reduced variability. These data points demonstrated a slight accelerating trend. The C-statistics, calculated for the entire data set, were not significant for both sides.

At follow-up, both unaffected (R) and affected (L) sides remained at the same level as their respective intervention data points. The affected side (L) was at a higher level than the unaffected side (R). Isometric follow-up testing was only done for subject #1.

Figure 11. Isometric Hamstring Peak Force - Subject #1 (L CPT)



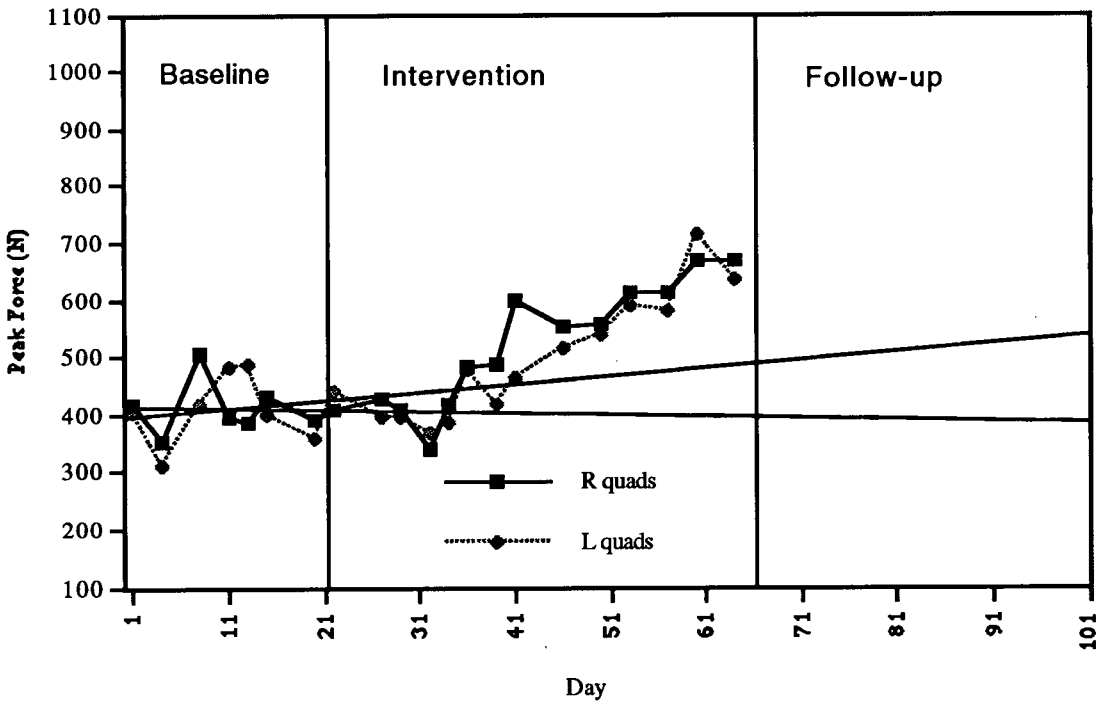
Subject #2 (R CPT)

Quadriceps: (Figure 12)

During the baseline phase, the unaffected side (L) baseline linear regression line demonstrated a slight decelerating trend. These data points demonstrated moderate variability. The affected side (R) baseline linear regression line demonstrated a slight accelerating trend. These data points demonstrated moderate variability. Both unaffected (L) and affected (R) sides appeared to be at the same level. The baseline C-statistics for both the unaffected (L) and affected (R) sides were not significant.

During the first 2 weeks of the intervention phase, the unaffected side (L) demonstrated minimal change in trend and a slight decrease in variability. During the remainder of the intervention phase, the unaffected side (L) demonstrated a steeper accelerating trend (all data points above the baseline linear regression line). A similar pattern was shown with the affected side (R). The majority of affected side (R) data points were at a higher level than the data points of the unaffected side (L). A significant value ($p < 0.05$) was present when the C-statistic was applied to the entire data set (baseline and intervention data points) for both the unaffected (L) and affected (R) sides, and indicated that both sides demonstrated a significant improvement in strength.

Figure 12. Isometric Quadriceps Peak Force - Subject #2 (R CPT)



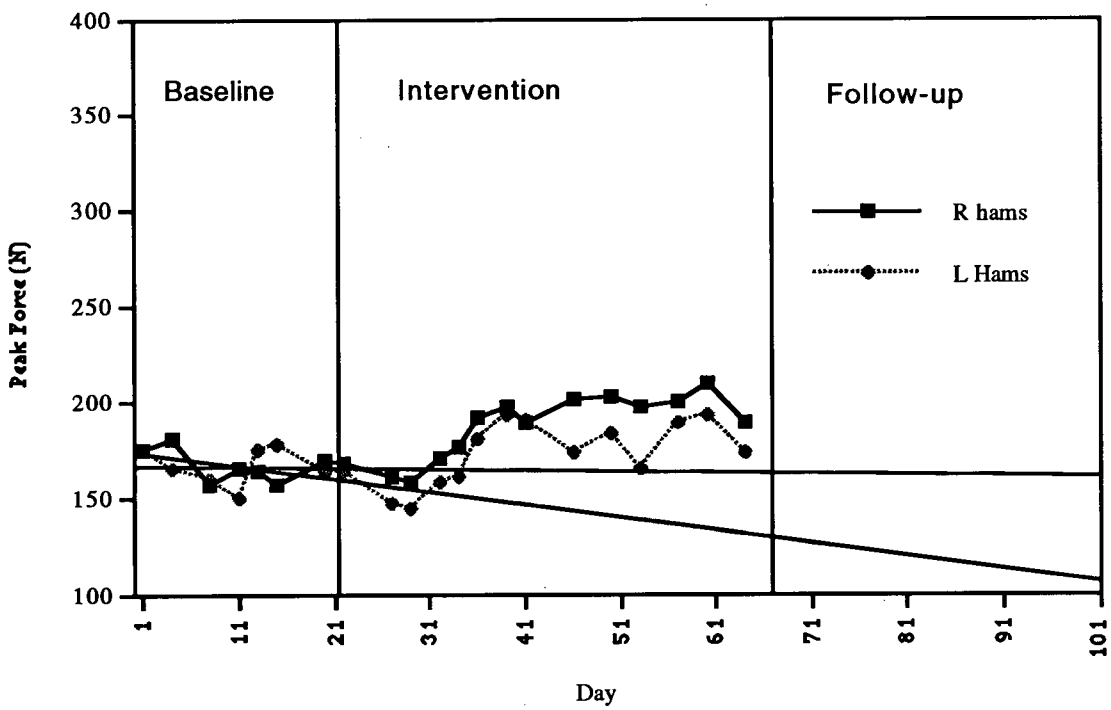
Hamstrings: (Figure 13)

During the baseline phase, the unaffected side (L) baseline linear regression line demonstrated a slight decelerating trend. The affected side (R) baseline linear regression line demonstrated a slightly steeper decelerating

trend. Both sides were at the same level. The baseline C-statistics for both the unaffected (L) and affected (R) sides were not significant.

Following the first week of intervention, the unaffected side (L) demonstrated a change to an accelerating trend (most data points above the baseline linear regression line). The affected side (R) demonstrated a similar pattern, with the exception that the majority of these data points were above the data points of the unaffected side (L). The affected side (R) also demonstrated lower variability than the unaffected side (L). A significant value ($p < 0.05$) was present when the C-statistic was applied to the entire data set (baseline and intervention data points) for both the unaffected (L) and affected (R) sides, and indicated that both sides demonstrated a significant improvement in strength.

Figure 13. Isometric Hamstring Peak Force - Subject #2 (R CPT)



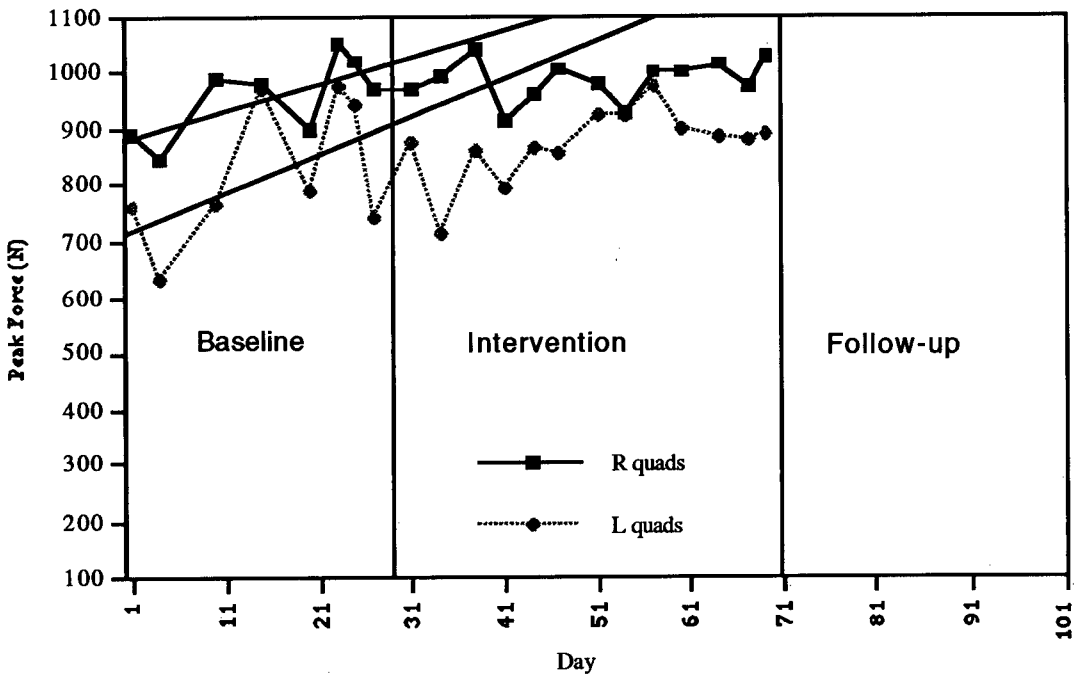
Subject #3 (L CPT, R PFPS)

Quadriceps: (Figure 14)

During the baseline, the unaffected side (R) baseline linear regression line demonstrated a steep accelerating trend with considerable variability in the data points. The affected side (L) demonstrated a similar pattern with the exception that the data points were at a lower level when compared to the unaffected side (R). The baseline C-statistics for both the unaffected (R) and affected (L) sides were not significant.

During the intervention phase, the unaffected side (R) demonstrated an accelerating trend, with all data points below the baseline linear regression line. The affected side (L) continued to demonstrate an accelerating trend, but all data points were below the baseline linear regression line and at a lower level than the unaffected side (R). The C-statistics calculated for the entire data set (baseline and intervention data series) were not significant for both the affected (L) and unaffected (R) sides.

Figure 14. Quadriceps Isometric Peak Force - Subject #3 (L CPT, R PFPS)

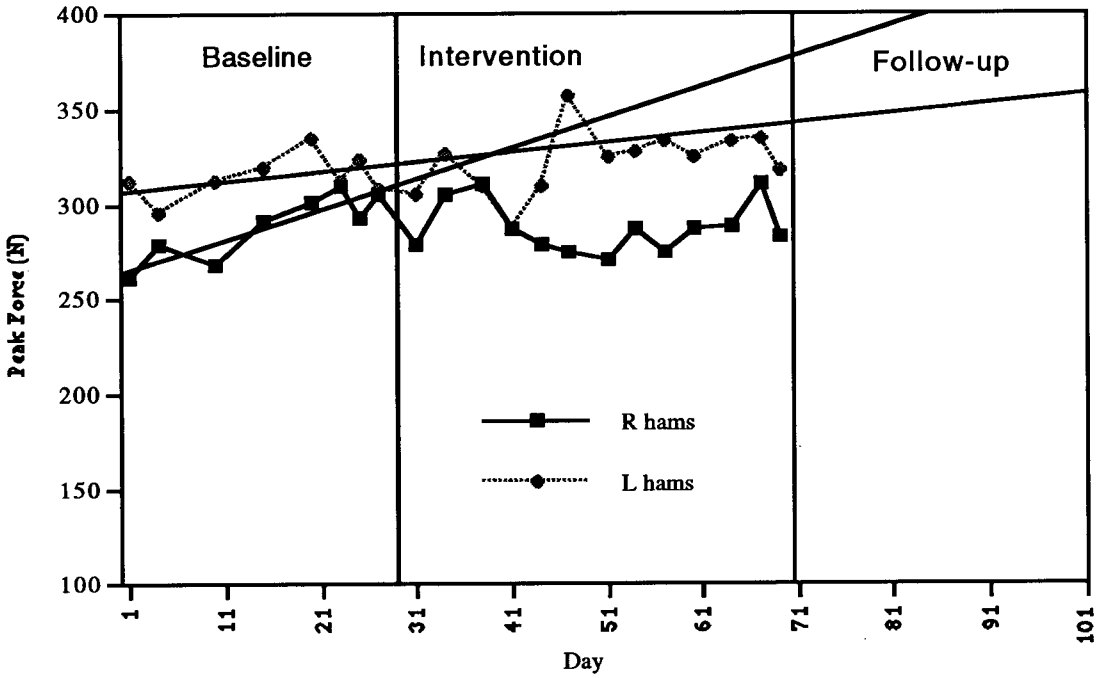


Hamstrings: (Figure 15)

During the baseline, the unaffected side (R) baseline linear regression line demonstrated an accelerating trend with limited variability of the data points. These data points were below the level of the affected side (L). The affected side (L) baseline linear regression line demonstrated a more gradual accelerating trend. A significant baseline C-statistic ($p < 0.05$) calculated for the unaffected (R) side indicated a significant upward trend. The baseline C-statistic for the affected side was not significant.

During the intervention phase, the unaffected side (R) demonstrated no trend with all data points below the baseline linear regression line. The majority of these data points remained lower than the affected side (L) data points. The affected side (L) data points followed the baseline linear regression line with some increased variability. A C-statistic was not calculated for the entire data set of the unaffected side (R) due to its significant baseline C-statistic. The C-statistic for the entire data set of the affected side (L) was not significant.

Figure 15. Isometric Hamstring Peak Force - Subject #3 (L CPT, R PFPS)



Subject #4 (R CPT, L PFPS)

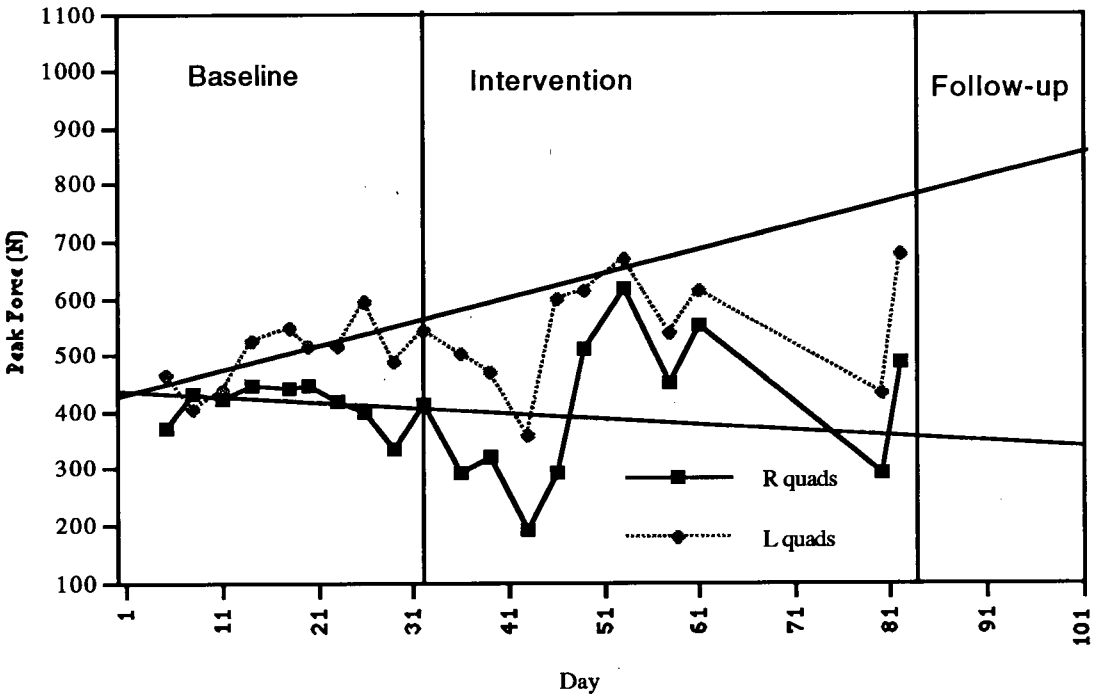
Quadriceps: (Figure 16)

During the baseline, the unaffected side (L) baseline linear regression line demonstrated an accelerating trend with most data points above those for the affected side (R). The affected side (R) baseline linear regression line demonstrated a slight decelerating trend. A significant baseline C-statistic ($p < 0.05$) for the unaffected side (L) indicated a significant increase in strength. The baseline C-statistic for the affected side (R) was not significant.

During the intervention phase, the unaffected side (L) data points showed increased variability. The majority of these data points fell below the baseline linear regression line but remained above the level of the affected side (R) data points. The affected side (R) data points showed increased variability with half the points above the baseline linear regression line and half below the baseline linear regression line. Both unaffected (L) and affected (R) sides

demonstrated a similar pattern of decreased values during the initial 2 weeks of the intervention, followed by sudden increase in values during the next 2 weeks. The last 2 data points for both sides demonstrated considerable variability. A C-statistic for the entire data set was not calculated for the unaffected side (L) due to its significant baseline C-statistic. The C-statistic for the entire data set of the affected side (R) was not significant.

Figure 16. Quadriceps Isometric Peak Force - Subject #4 (R CPT, L PFPS)



Hamstrings: (Figure 17)

During the baseline phase, the unaffected side (L) baseline linear regression line demonstrated a slight decelerating trend with considerable variability of the data points. The majority of these data points were higher than those for the affected side (R). The affected side (R) baseline linear regression line demonstrated a steep decelerating trend with lower variability of the data points as compared to the unaffected side (L). The baseline C-statistic for the unaffected side (L) was not significant. A significant baseline C-statistic ($p <$

0.05) was calculated for the affected (R) side and indicated a significant downward trend.

During the first 2 weeks of the intervention phase, the data points for the unaffected side (L) demonstrated a reduction in values. During the last 2 weeks of this phase, the data points followed the baseline linear regression line. During the intervention phase, the affected side (R) data points demonstrated a change towards an accelerating trend (similar pattern to unaffected side (L) graphically). Both unaffected (L) and affected (R) sides were at the same level during the last 4 weeks of the intervention phase. The C-statistic calculated for the entire data series was significant ($p < 0.05$) for the unaffected (L) side and indicated a significant change in trend.

Figure 17. Isometric Hamstring Peak Force - Subject #4 (R CPT, L PFPS)

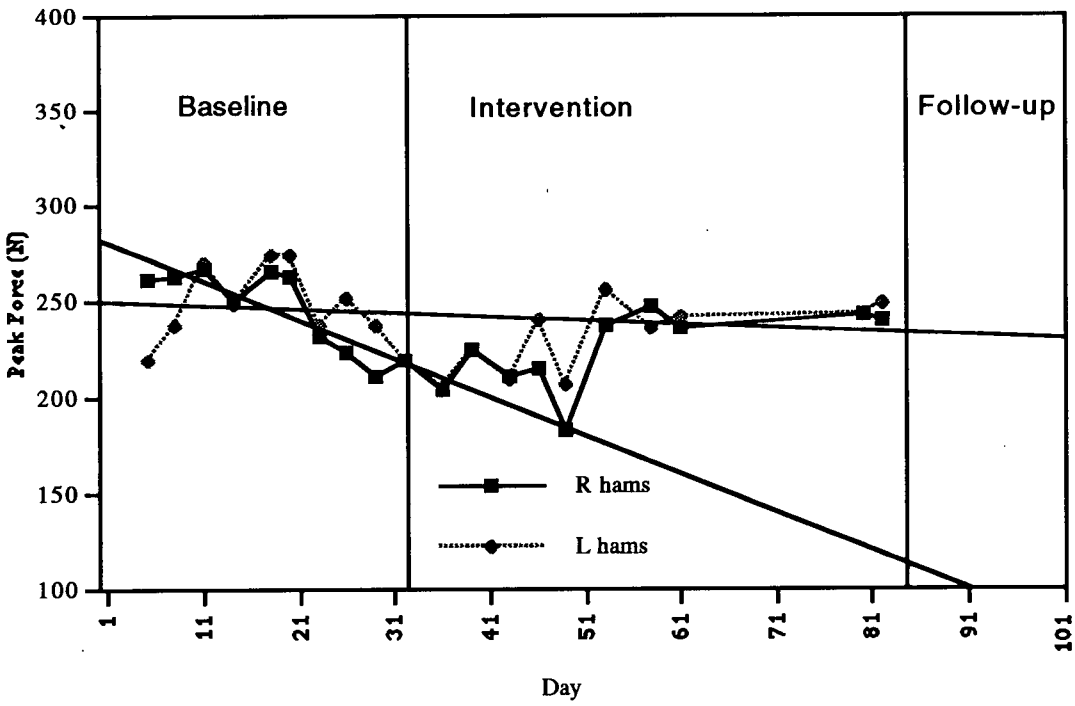


Table 2. Summary Table of Isometric Quadriceps Peak Force

SUBJECT	SIDE	BASELINE	INTERVENTION	FOLLOW-UP
#1	unaffected (R)	accelerating baseline linear regression line (higher level than affected side)	followed baseline linear regression line	maintenance of same level
	affected (L)	accelerating baseline linear regression line	followed baseline linear regression line (reached same level as unaffected side)	maintenance of same level
#2	unaffected (L)	decelerating baseline linear regression line	accelerating trend last 4 weeks of EEP	
	affected (R)	decelerating baseline linear regression line (similar level to unaffected side)	accelerating trend last 4 weeks of EEP (similar level to unaffected side)	
#3	unaffected (R)	accelerating baseline linear regression line	reduction in accelerating trend (levels off)	
	affected (L)	accelerating baseline linear regression line (higher level than unaffected side)	accelerating trend but below baseline linear regression line (lower level than unaffected side)	
#4	unaffected (L)	accelerating trend (higher level than affected side)	initial reduction in level, accelerating trend continued (below baseline linear regression line) (higher level than affected side)	
	affected (R)	decelerating trend	initial reduction in level, followed by an accelerating trend	

Table 3. Summary Table of Isometric Hamstring Peak Force

SUBJECT	SIDE	BASELINE	INTERVENTION	FOLLOW-UP
#1	unaffected (R)	accelerating baseline linear regression line	no trend	maintenance of same level
	affected (L)	accelerating baseline linear regression line (similar level to unaffected side)	accelerating trend (similar level to unaffected side)	higher level than unaffected side
#2	unaffected (L)	decelerating baseline linear regression line	accelerating trend	
	affected (R)	accelerating baseline linear regression line (similar level to unaffected side)	accelerating trend (higher level than unaffected side)	
#3	unaffected (R)	accelerating baseline linear regression line	no trend	
	affected (L)	accelerating baseline linear regression line (higher level than unaffected side)	accelerating trend (higher level than unaffected side)	
#4	unaffected (L)	decelerating baseline linear regression line	accelerating trend last 4 weeks of EEP	
	affected (R)	decelerating baseline linear regression line (slightly higher level than unaffected side)	accelerating trend last 4 weeks of EEP (similar level to unaffected side)	

Isokinetic Quadriceps and Hamstring Average Torque

These data points, collected prior to the baseline phase, immediately following the intervention phase, and at approximately one month following the intervention phase, have not been analyzed statistically. The isokinetic measures were collected to help provide further information regarding strength changes. However, they were not used as a repeated measure as this type of testing may exacerbate knee symptoms.

It should be noted that the eccentric values were higher than concentric values for each measure of quadriceps average torque and hamstring average torque for all the subjects, as predicted by the force-velocity relationship[40]. It should also be noted that subject #1 does not have follow-up data points for this measure. Follow-up data points were collected isometrically for subject #1.

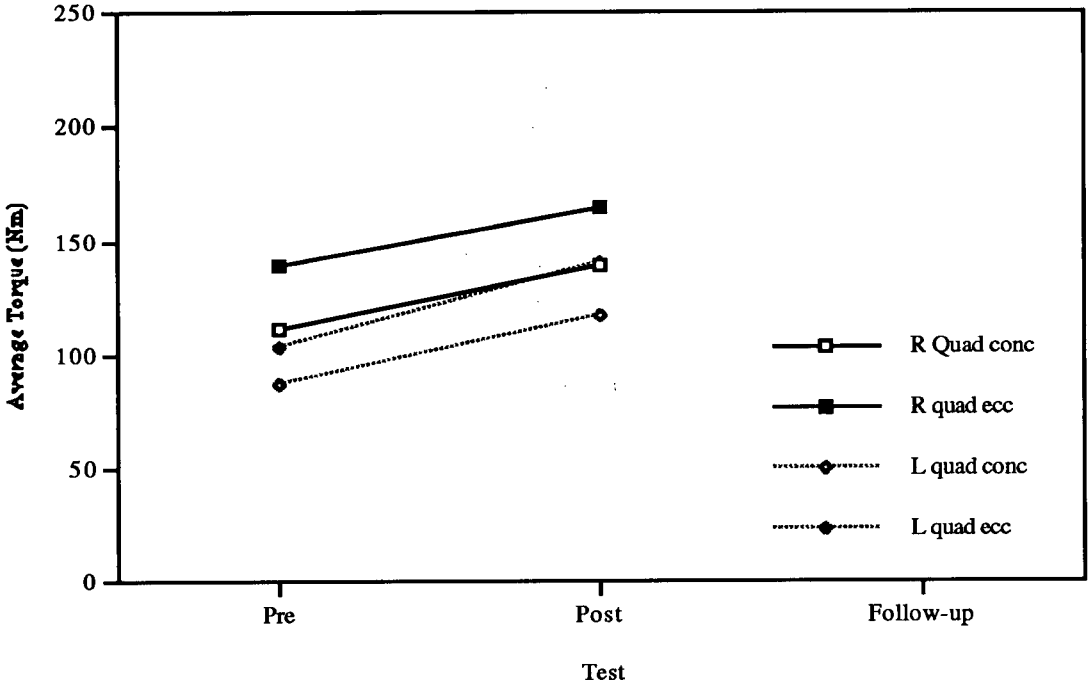
Subject #1 (L CPT)

Quadriceps: (Figure 18)

At pre-test, the unaffected (R) side demonstrated higher concentric (28%) and eccentric (35%) values compared to the affected (L) side.

At post-test, both affected (L) and unaffected (R) quadriceps measures were higher than pre-test values, however the affected (L) side remained lower than the unaffected (R) side. The unaffected side (R) concentric value and unaffected side (R) eccentric value improved by 25% and 19% respectively compared to the pre-test. The affected side (L) concentric value and affected side (L) eccentric value improved 34% and 36% respectively when compared to the pre-test.

Figure 18. Isokinetic Quadriceps Average Torque - Subject #1 (L CPT)

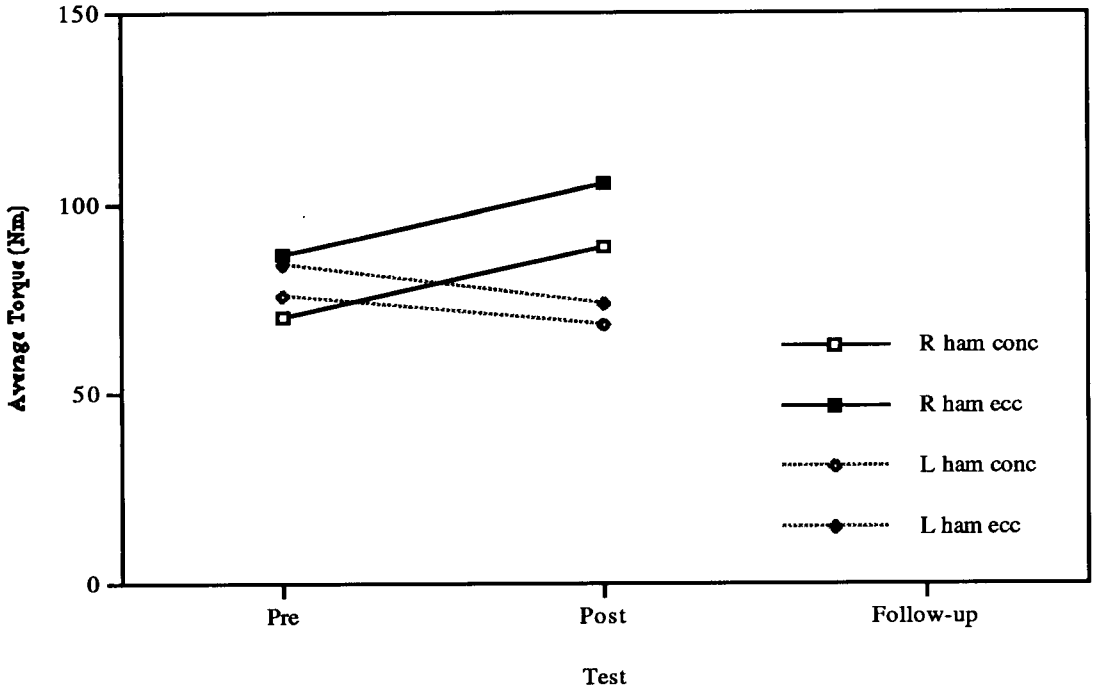


Hamstrings: (Figure 19)

At the pre-test, the affected side (L) and unaffected side (R) hamstrings demonstrated similar concentric and similar eccentric values.

At the post-test, the unaffected side (R) hamstring demonstrated improved concentric (27%) and eccentric (22%) values compared to the pre-test. The affected side (L) hamstring demonstrated a reduction in both concentric (11%) and eccentric (12%) values when compared to the pre-test.

Figure 19. Isokinetic Hamstring Average Torque - Subject #1 (L CPT)



Subject #2 (R CPT)

Quadriceps: (Figure 20)

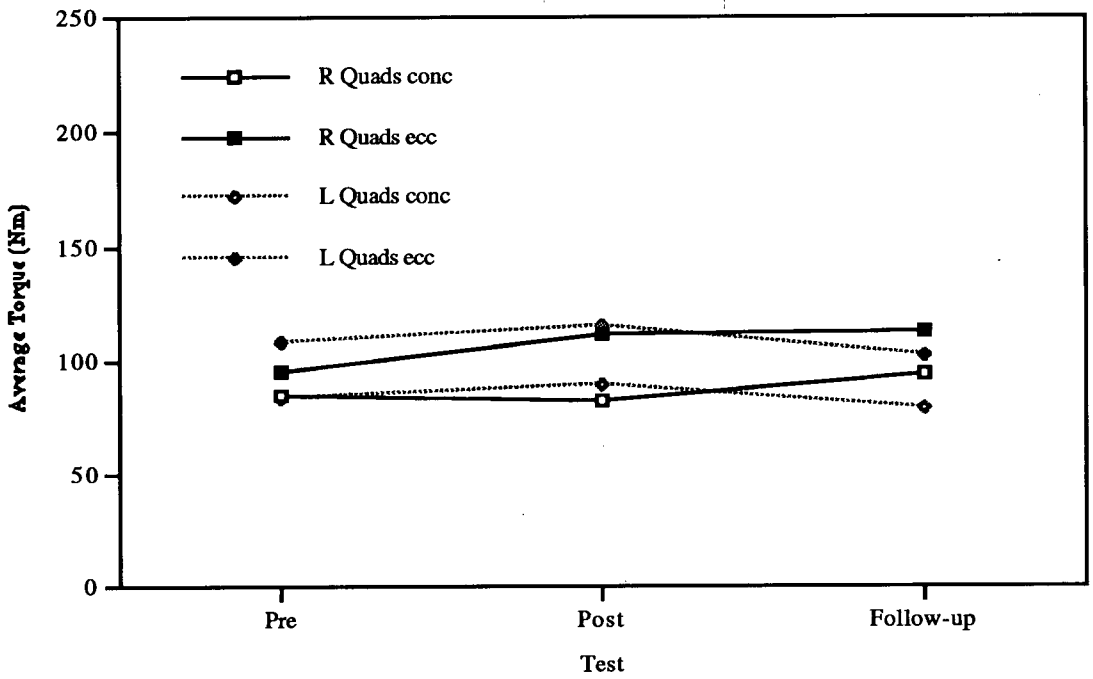
At the pre-test, the affected side (R) and unaffected side (L) quadriceps demonstrated similar concentric values. The eccentric value for the unaffected (L) side was 14% greater than the eccentric value for the affected (R) side.

At the post-test, the unaffected side (L) demonstrated a slight improvement in concentric (6%) and eccentric (6%) values compared to the pre-test. The affected side (R) demonstrated a slight reduction (4%) concentrically and a slight increase (17%) eccentrically when compared to the pre-test.

At follow-up, the unaffected side (L) demonstrated a slight reduction in concentric (11%) and eccentric (11%) values when compared to the post-test. The affected side (R) demonstrated a continued slight improvement in

concentric (15%) and eccentric (1%) values compared to the post-test, and exceeded the unaffected side (L) for both measures.

Figure 20. Isokinetic Quadriceps Average Torque - Subject #2 (R CPT)



Hamstrings: (Figure 21)

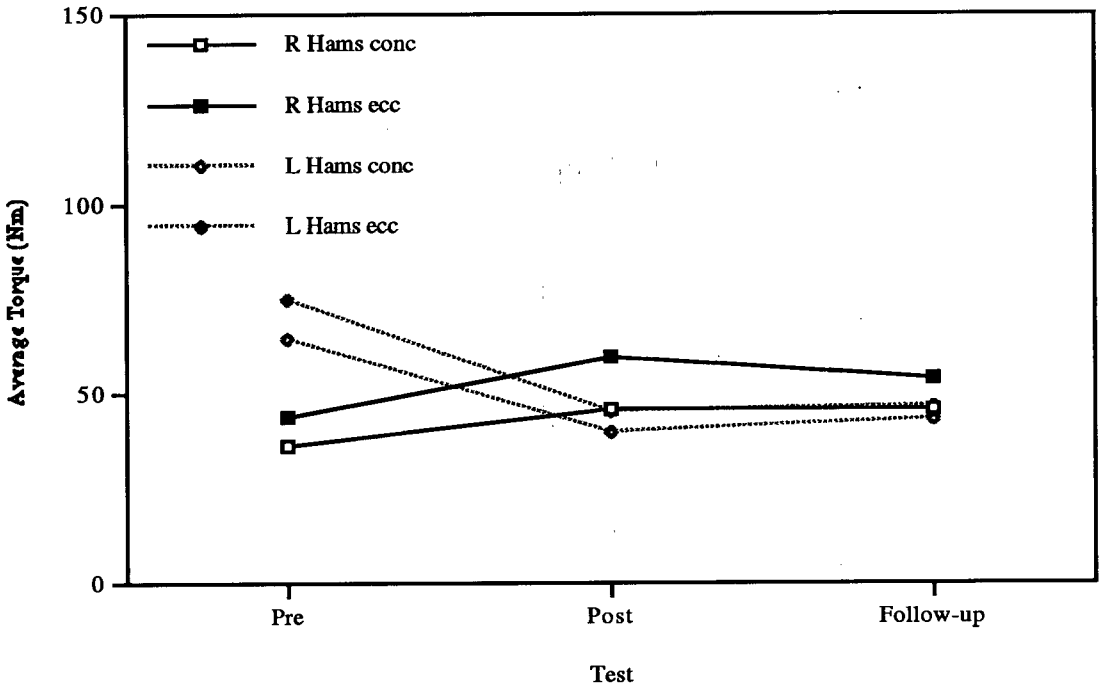
At pre-test, the unaffected side (L) had higher values than the affected side (R) both concentrically (80%) and eccentrically (70%).

At post-test, when compared to the pre-test, the unaffected side (L) demonstrated a marked reduction in concentric (39%) and eccentric (40%) values, such that both were below the affected side (R) level. When compared to the pre-test, the affected side (R) demonstrated an increase in concentric (28%) and eccentric (36%) values, but did not reach the pre-test levels of the unaffected side (L).

At follow-up, the unaffected side (L) demonstrated a slight increase in concentric (8%) and eccentric (4%) values when compared to the post-test. The

affected side (R) demonstrated no change concentrically and a slight reduction (10%) eccentrically when compared to the post-test.

Figure 21. Isokinetic Hamstring Average Torque - Subject #2 (R CPT)



Subject #3 (L CPT, R PFPS)

Quadriceps: (Figure 22)

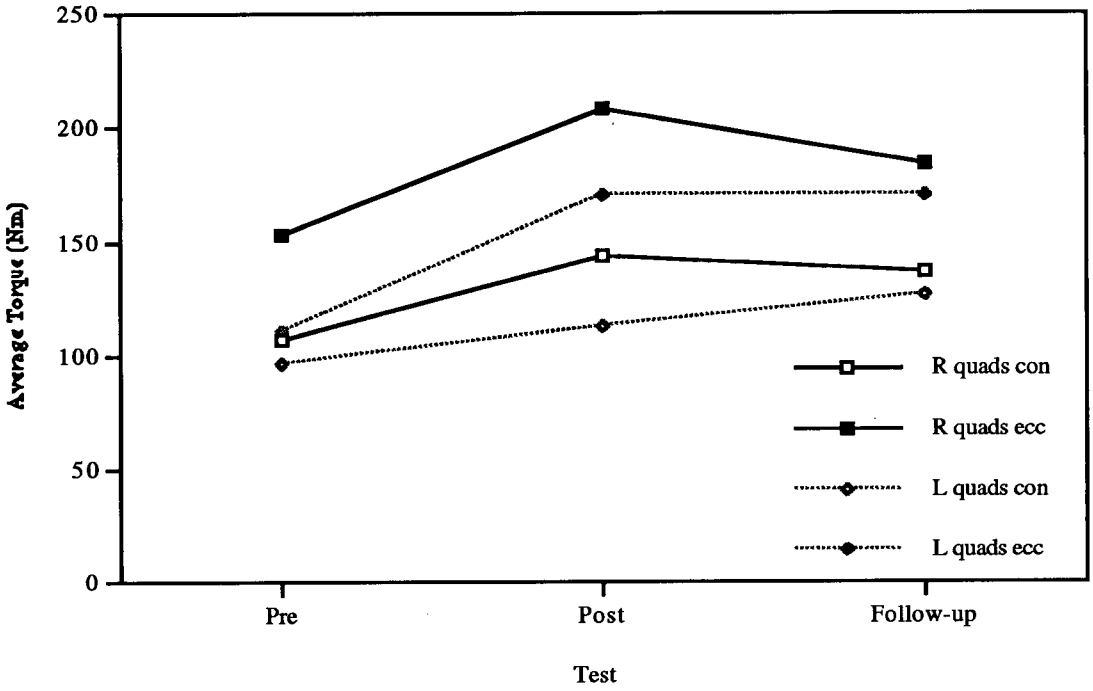
At pre-test, the unaffected side (R) had higher values than the affected side (L) both concentrically (11%) and eccentrically (39%).

At post-test, the unaffected side (R) demonstrated higher values both concentrically (35%) and eccentrically (37%) when compared to the pre-test. The affected side (L) also demonstrated increased values concentrically (17%) and eccentrically (55%) when compared to the pre-test, but did not reach the post-test levels of the unaffected side (R).

At follow-up, the unaffected side (R) decreased in both concentric (5%) and eccentric (12%) values when compared to the post-test, but remained

above pre-test levels. The affected side (L) demonstrated continued improvement beyond post-test levels concentrically (13%). The eccentric value remained the same as the post-test level. The affected side (L) measures decreased slightly below the unaffected side (R) at follow-up (concentric 8% lower, eccentric 7% lower).

Figure 22. Isokinetic Quadriceps Average Torque Subject #3 (L CPT, R PFPS)



Hamstrings: (Figure 23)

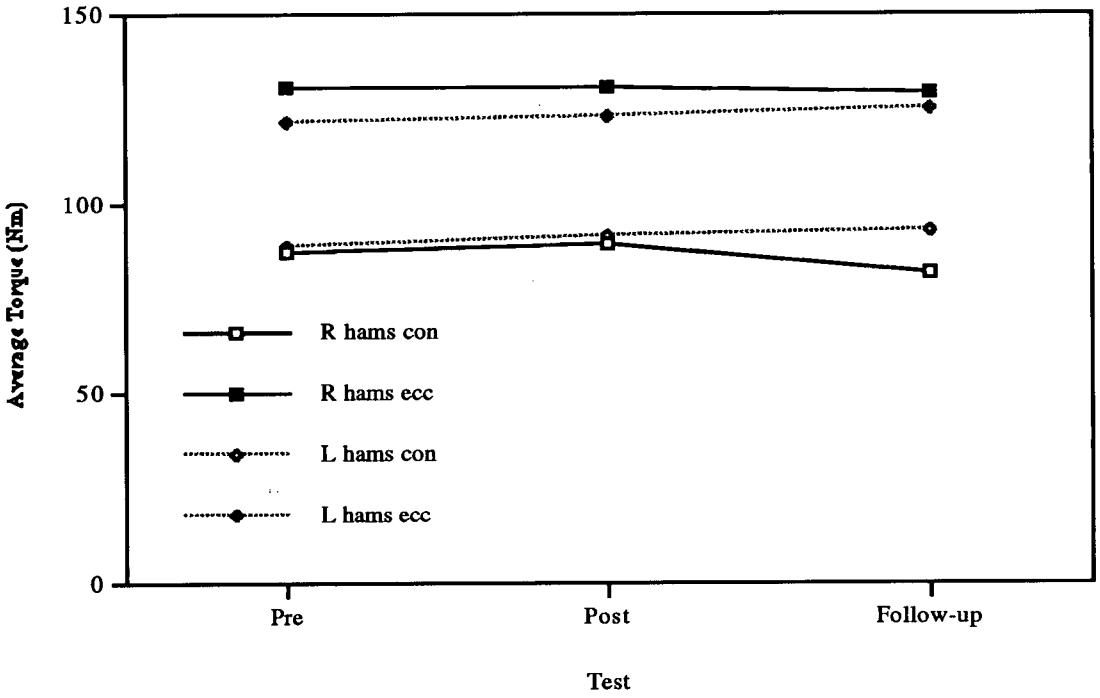
At pre-test, the unaffected side (R) and affected side (L) demonstrated similar values. The unaffected side (R) was 1% lower concentrically and 7% higher eccentrically when compared to the affected side (L).

At post-test, very little change was demonstrated for both sides.

At follow-up, the unaffected side (R) demonstrated a slight reduction concentrically (9%) and eccentrically (1%) when compared to post-test levels. The affected side (L) demonstrated slight increases both concentrically (1%)

and eccentrically (2%) when compared to post-test levels. Overall, both affected (L) and unaffected (R) sides demonstrated minimal change throughout the study period.

Figure 23. Isokinetic Hamstring Average Torque Subject #3 (L CPT, R PFPS)



Subject #4 (R CPT, L PFPS)

Quadriceps: (Figure 24)

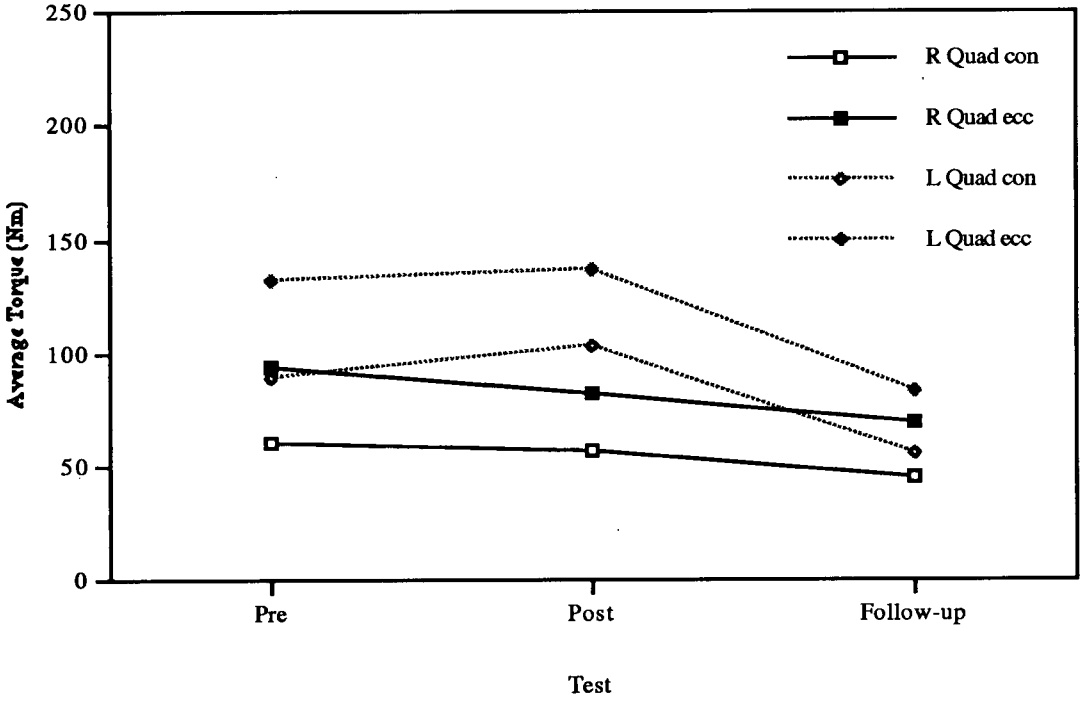
At pre-test, the unaffected side (L) was higher concentrically (46%) and eccentrically (40%) when compared to the affected side (R).

At post-test, the unaffected side (L) demonstrated an increase both concentrically (16%) and eccentrically (4%) when compared to pre-test levels. The affected side (R) demonstrated a reduction in both concentric (7%) and eccentric (13%) values when compared to pre-test levels.

At follow-up, the unaffected side (L) demonstrated a reduction in concentric (45%) and eccentric (39%) values when compared to post-test

levels. The affected side (R) also demonstrated a reduction of concentric (21%) and eccentric (15%) values when compared to post-test levels. The unaffected side (L) remained slightly higher (22% concentrically, 14% eccentrically) than the affected side (R) at follow-up.

Figure 24. Isokinetic Quadriceps Average Torque Subject #4 (R CPT, L PFPS)



Hamstrings: (Figure 25)

At pre-test, the unaffected side (L) was higher concentrically (19%) and eccentrically (33%) when compared to the affected side (R).

At post-test, the unaffected side (L) demonstrated a reduction of both concentric (26%) and eccentric (28%) values when compared to pre-test values. The affected side (R) demonstrated an increase in both concentric (31%) and eccentric (34%) values when compared to pre-test levels. The affected side (R) was higher concentrically (47%) and eccentrically (39%) when compared to the unaffected side (L) at the post-test.

At follow-up, the unaffected side (L) demonstrated an increase in concentric (38%) and eccentric (27%) values when compared to post-test levels. The concentric value exceeded the pre-test value slightly (2%) and the eccentric value remained below (8%) the pre-test value. The affected side (R) demonstrated a reduction both concentrically (22%) and eccentrically (26%) when compared to the post-test. The concentric value was slightly higher (3%) and the eccentric value was slightly lower (1%) when compared to pre-test levels.

Figure 25. Isokinetic Hamstring Average Torque Subject #4 (R CPT, L PFPS)

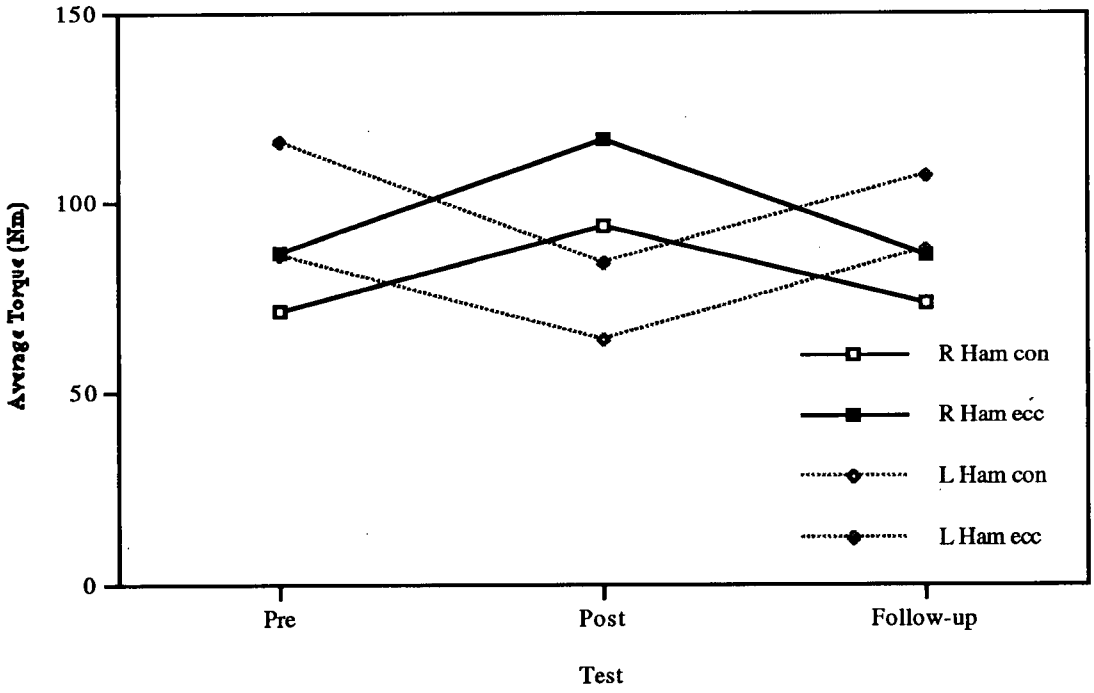


Table 4. Summary of Isokinetic Quadriceps Average Torque.

SUBJECT	SIDE	PRE-TEST	POST-TEST	FOLLOW-UP
#1	unaffected (R)	stronger: conc (28%) ecc (35%)	▲conc (25%) ▲ecc (19%)	
	affected (L)		▲conc (34%) ▲ecc (36%)	
#2	unaffected (L)	stronger: ecc (14%)	▲conc (6%) ▲ecc (6%)	▼conc (11%) ▼ecc (11%)
	affected (R)		▼conc (4%) ▲ecc (17%)	▲conc (15%) ▲ecc (1%)
#3	unaffected (R)	stronger: conc (11%) ecc (39%)	▲conc (35% ▲ecc (37%)	▼conc (5%) ▼ecc (12%)
	affected (L)		▲conc (17%) ▲ecc (55%)	▲conc(13%) no change ecc
#4	unaffected (L)	stronger: conc (46%) ecc (40%)	▲conc (16%) ▲ecc (4%)	▼conc (45%) ▼ecc (39%)
	affected (R)		▼conc (7%) ▼ecc (13%)	▼conc (21%) ▼ecc (15%)

note: pre-test % relative to affected side value.
post-test % relative to same side pre-test value.
follow-up % relative to same side post-test value.

Table 5. Summary of Isokinetic Hamstring Average Torque.

SUBJECT	SIDE	PRE-TEST	POST-TEST	FOLLOW-UP
#1	unaffected (R)	similar values R and L conc & ecc	▲conc (27%) ▲ecc (22%)	
	affected (L)		▼conc (11%) ▼ecc (12%)	
#2	unaffected (L)	stronger: conc (80%) ecc (70%)	▼conc (39%) ▼ecc (40%)	▲conc (8%) ▲ecc (4%)
	affected (R)		▲conc (28%) ▲ecc (36%)	no change conc ▼ecc (10%)
#3	unaffected (R)	stronger: ecc (7%)	minimal change	▼conc (9%) ▼ecc (1%)
	affected (L)		minimal change	▲conc (1%) ▲ecc (2%)
#4	unaffected (L)	stronger: conc (19%) ecc (33%)	▼conc (26%) ▼ecc (28%)	▲conc (38%) ▲ecc (27%)
	affected (R)		▲conc (31%) ▲ecc (34%)	▼conc (22%) ▼ecc (26%)

note: pre-test % relative to affected side value.
post-test % relative to same side pre-test value.
follow-up % relative to same side post-test value.

Summary of Isokinetic Testing

Dvir[41] has suggested that in cases of unilateral joint involvement, the contralateral side constitutes that basis for comparison in isokinetic strength testing. Dvir[41] reported this assumption is reasonable for symmetrical use of the body segments. Dvir[41] has also suggested that imbalance of strength of up to 10% can be considered "normal". Imbalance of strength between 10% and 20% is "possibly abnormal", and imbalance of strength greater than 20% is "probably abnormal"[41].

Thus, at pre-test of the quadriceps, subjects #1, #3, and #4 demonstrated an imbalance of concentric and eccentric strength between the affected and unaffected lower extremities the may be classified as "probably abnormal". Subject #2 demonstrated a "possibly abnormal" imbalance of concentric strength and a "probably abnormal" imbalance of eccentric strength .

At pre-test of the hamstrings, subjects #2 and #4 demonstrated a "probably abnormal" imbalance of concentric and eccentric strength between the affected and unaffected lower extremities. Subjects #1 and #3 did not demonstrate hamstring muscle imbalance.

Daily Activity Levels

Subject #1 (Figure 26)

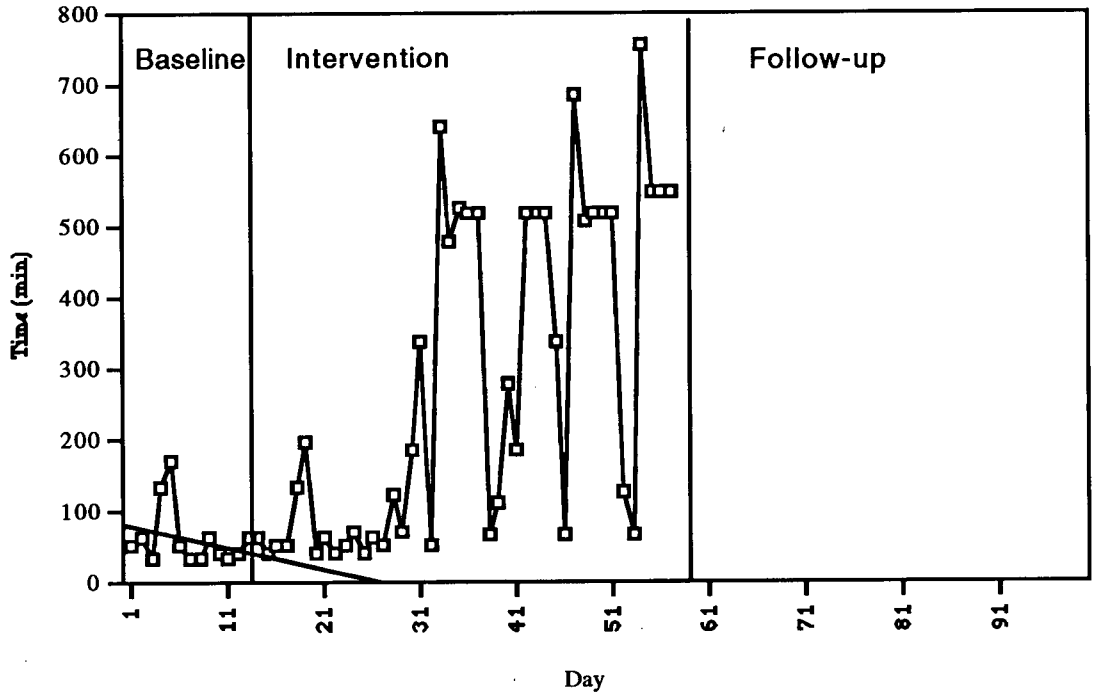
The baseline linear regression line demonstrated a decelerating trend. The baseline activity level was low and demonstrated little variability. All intervention data points were above the baseline linear regression line. The intervention phase demonstrated increased variability and a higher level of activity.

The C-statistic calculated for the baseline was not significant. However, a significant value ($p < 0.05$) was present when the C-statistic was applied to the

entire data set. This indicated a significant increase in daily activity in the intervention phase.

It should be noted that the baseline phase coincided with the final examination period at UBC and consequently subject #1 had low activity levels. The last 4 weeks of the intervention phase coincided with subject #1 starting a summer job involving manual labor.

Figure 26. Daily Activity Levels - Subject #1 (L CPT)

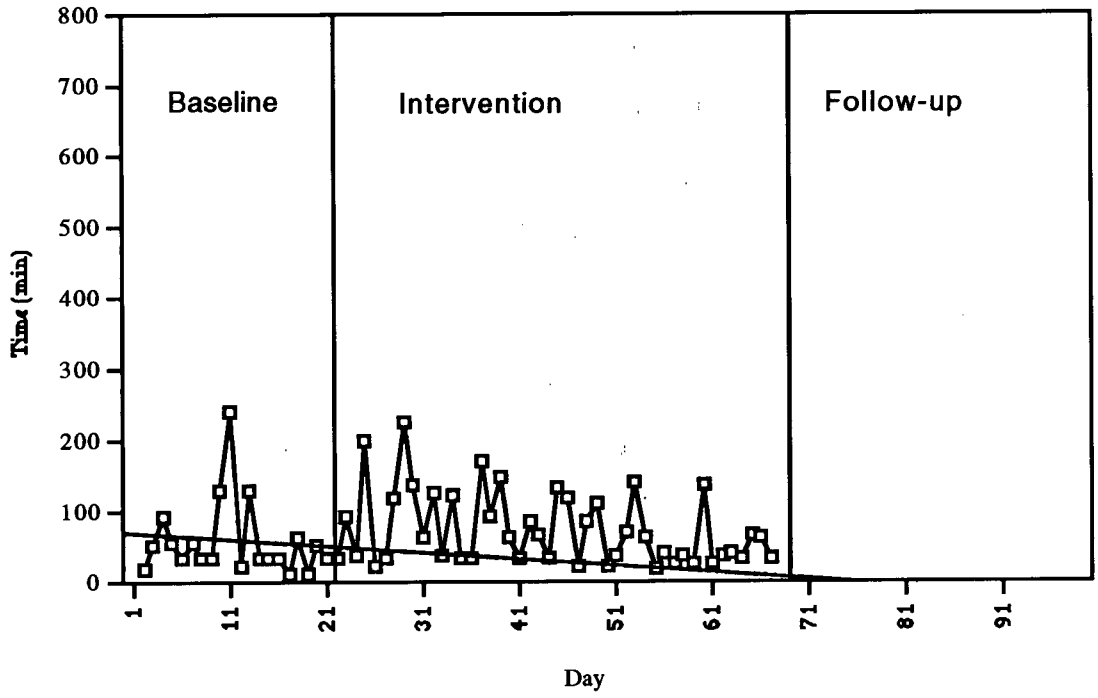


Subject #2 (Figure 27)

The baseline linear regression line demonstrated a gradual decelerating trend. The baseline activity level was low and demonstrated moderate variability. The majority of intervention data points were above the baseline linear regression line. The intervention phase activity level was higher in the first four weeks. An increase in variability of intervention data points was noted. No change in trend was noted between phases.

The C-statistics calculated for the baseline phase and the entire data series were not significant. This indicated there was no significant change in the trend of daily activity levels between the baseline phase and intervention phase.

Figure 27. Daily Activity Levels - Subject #2 (R CPT)

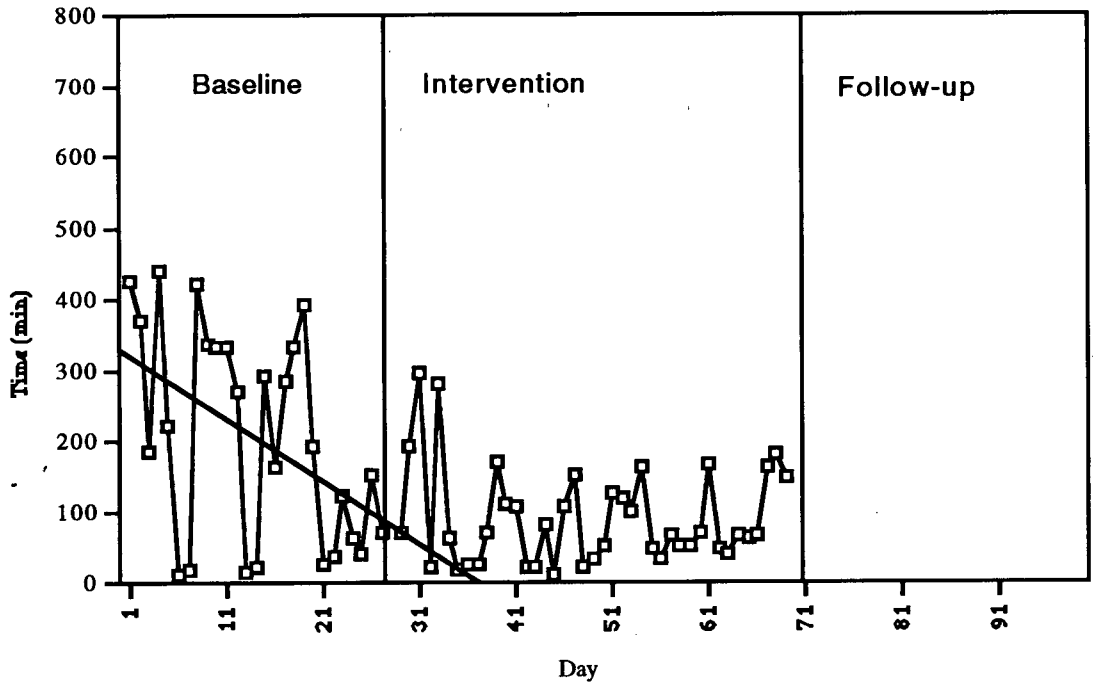


Subject #3 (Figure 28)

The baseline linear regression line demonstrated a steeply decelerating trend. The baseline data points demonstrated considerable variability and a higher level as compared to the intervention phase. The majority of intervention data points were above the baseline linear regression line. It should be noted that subject #3 discontinued a seasonal manual labor job in the first 2 weeks of the intervention phase. This resulted in lower activity levels during the intervention phase.

The C-statistic calculated for the baseline demonstrated a significant trend ($p < 0.05$). Consequently the C-statistic was not calculated from the entire data set.

Figure 28. Daily Activity Levels - Subject #3 (L CPT, R PFPS)



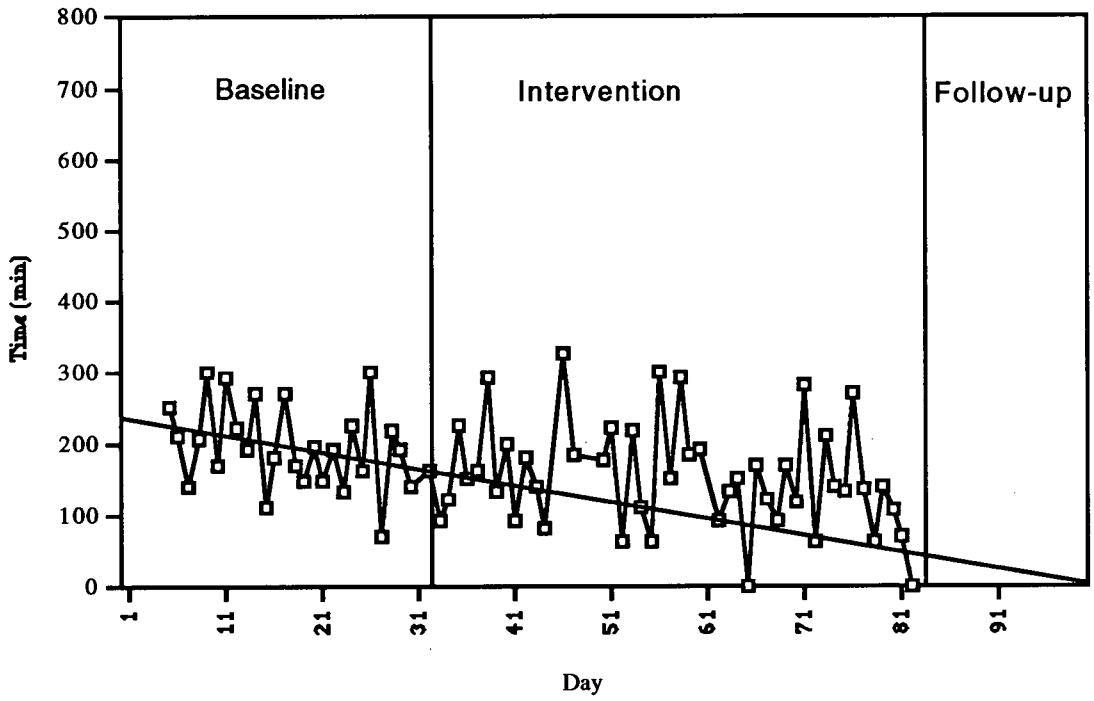
Subject #4 (Figure 29)

The baseline linear regression line demonstrated a decelerating trend. The majority of intervention data points were above the baseline linear regression line. The intervention data points demonstrated increased variability as compared to the baseline phase. However, no change in level of activity was noted between phases. It should be noted that subject #4, an international level rower, was performing strength and aerobic conditioning exercises at relatively high intensity levels throughout the study.

The C-statistics calculated for the baseline phase and the entire data series were not significant. This indicated there was no significant change in the

trend of daily activity levels between the baseline phase and intervention phase.

Figure 29. Daily Activity Levels - Subject #4 (R CPT, L PFPS)



Summary of Daily Activity Levels: During the baseline phase, all subjects demonstrated decelerating baseline linear regression line's. Subject #3 demonstrated considerable variability of baseline data points.

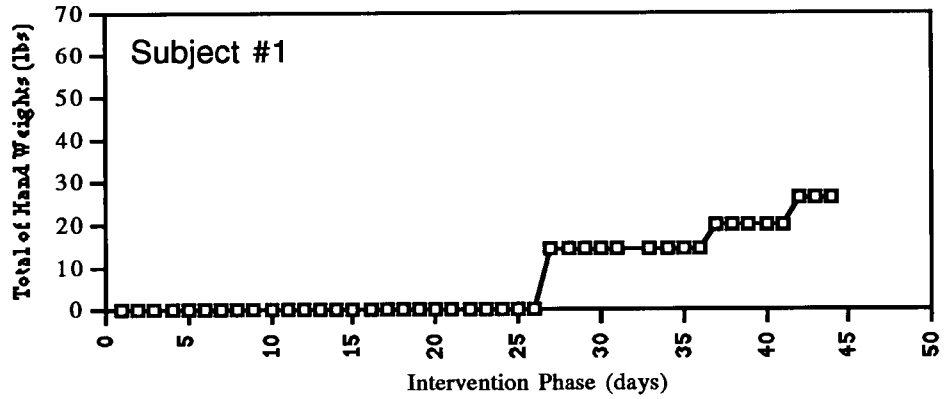
During the intervention, the majority of the data point were above the baseline linear regression line's for all subjects. Subject #1 demonstrated a marked increase in daily activity levels while subject #3 demonstated a marked decrease in daily activity levels. The daily activity levels of subjects #2 and #4 did not demonstrate a visually detectable change.

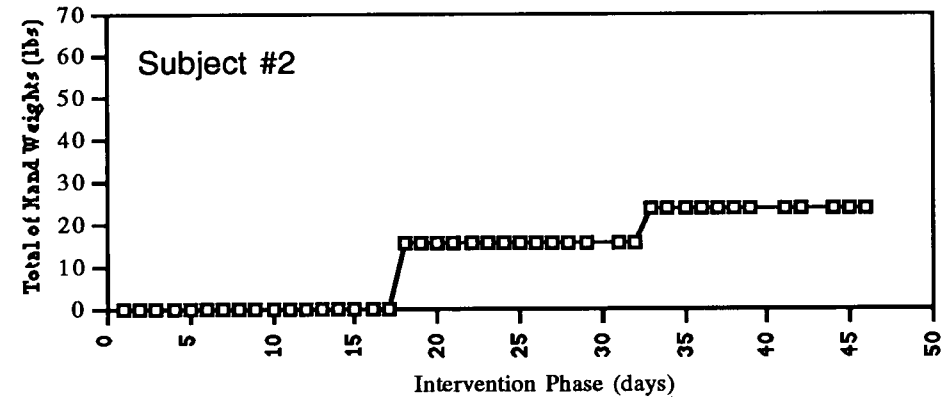
Progression of EEP

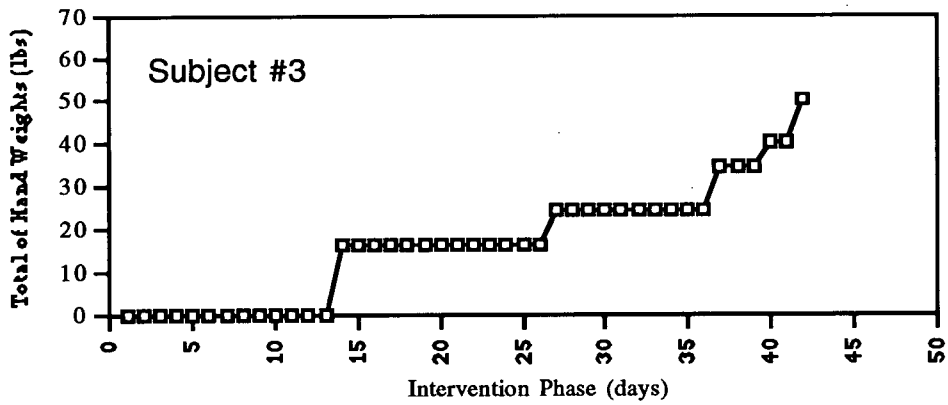
The progression of hand weights used by each subject during the EEP are illustrated in Figure 30. Subject #1 started using hand weights after 26 days of the EEP. She started with 7 lbs per hand (10% of body weight) and

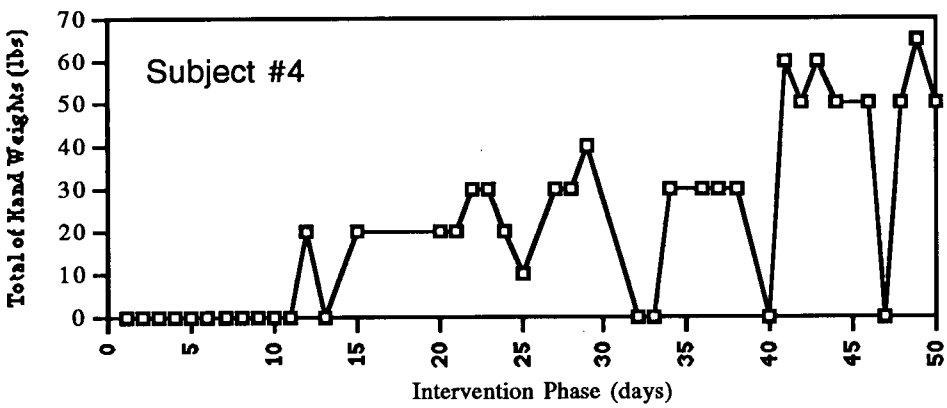
progressed to 12 lbs per hand (17% of body weight) by the end of the EEP. Subject #2 started using hand weights after 17 days of the EEP. She started with 8 lbs per hand (10% of body weight) and progressed to 13 lbs per hand (16% of body weight) by the end of the EEP. Subject #3 started using hand weights after 13 days of the EEP. He started with 8 lbs per hand (10% of body weight) and progressed to 25 lbs per hand (29% of body weight) by the end of the EEP. Subject #4 started using hand weights after 11 days of the EEP. She started with 10 lbs per hand (14% of body weight) and progressed to 30 lbs per hand (42% of body weight) by the end of the EEP.

Figure 30. Graphs of Hand Weight Progression









Treatment Integrity

Treatment integrity refers to the degree to which treatments are implemented as intended[42]. Treatment integrity has been evaluated indirectly in this study via completion of self-reports by all subjects at the end of each exercise session (Appendix 5). These self-reports were used to gain an indication of subject compliance in performing the home exercise program. The results are represented in Table 6.

Table 6. Subject Compliance to EEP.

Subject	Number of intervention days	Number of days intervention not performed	Compliance (%)
#1	44	4	91
#2	44	5	89
#3	39	1	97.5
#4	51	16	69

DISCUSSION

Following the discussion of the major findings of this study, the limitations pertaining to the measures and study design employed in this study will be presented. The clinical relevance of the study will be discussed followed by a summary of the hypotheses and recommendations for future research. Throughout the discussion "affected side" will refer to the lower extremity having CPT, while "unaffected side" will refer to the lower extremity with PFPS (subjects #3 and #4) or no musculoskeletal disorder (subjects #1 and #2).

The purpose of this study was to determine the effects of the EEP developed by Curwin & Stanish[2] in terms of knee pain, knee function, quadriceps strength, hamstring strength, and daily activity levels in four patients with CPT. The most prominent finding of this study was the apparent quadriceps strength deficit of the affected side. Isokinetic testing detected a quadriceps strength deficit of the affected side concentrically in three subjects (#1, #3, and #4) and eccentrically in all four subjects. Isometric testing also detected a quadriceps strength deficit of the affected side in two of the subjects (#1 and #4).

No other studies examining isokinetic strength of the quadriceps or hamstrings in patients with CPT have been located. Only one study has been found that assessed isometric strength of the quadriceps and hamstrings in patients with CPT.[43] Kujala et al.[43] demonstrated that patients with CPT had lower isometric knee extension and knee flexion torques compared to normal subjects, however, these differences were not significant.

The results of this study indicate that quadriceps muscle strength is adversely affected by CPT. The symptom of knee pain resulting from CPT may be the cause of the quadriceps deficit. Knee pain may result in disuse atrophy (decreased strength) of the affected side or inhibit quadriceps function of the

affected side during isometric or isokinetic testing. In addition, isometric or isokinetic maximal testing may induce further knee pain and result in further limitation of the quadriceps to generate tension. It has been shown that knee pain leads to reflex inhibition of the quadriceps while sparing the hamstrings.[44] The relationship of knee pain and isometric quadriceps strength is best illustrated in the composite graphs of repeated measures for each subject (Appendix 18). Visual examination of the intervention phase of these graphs (Appendix 18) demonstrated that as pain (intensity) ratings declined, isometric quadriceps peak force increased. However, it was not possible to discern whether the increases in isometric quadriceps peak force were due to decreases in knee pain or from training effects of the EEP on the quadriceps femoris muscles.

Another prominent finding of this study was that patients with CPT do not appear to have an isometric or isokinetic hamstring strength deficit of the affected side. Isokinetic hamstring pre-test results (Figures 19, 21, 23, & 25) demonstrated no clear pattern of hamstring strength imbalance. As mentioned earlier, no other studies have been found that document isokinetic hamstring strength in patients with CPT. Baseline isometric hamstring peak force testing (Figures 11, 13, 15, 17) did not demonstrate a marked strength imbalance between the affected and unaffected sides, with the exception of subject #3, who demonstrated higher levels of affected side hamstring peak force. As reported earlier, Kujala et al.[43] have examined isometric torque of the hamstrings in patients with CPT and indicated no significant difference compared to normal subjects. However, they[43] did not report a comparison of hamstring torques between the affected side and unaffected side for the patients with CPT.

An additional finding was that the EEP did not result in increased isometric or isokinetic hamstring strength of the affected side or unaffected side, although the quadriceps muscle group demonstrated increased isometric and isokinetic strength of the affected and unaffected sides. It has been reported that co-activation of the quadriceps and hamstring muscles occurs during open kinetic chain exercise (i.e. seated knee extension)[45,46] and closed kinetic chain exercises (i.e. squatting).[47] The proposed role of the hamstring muscles during open and closed kinetic chain exercise is to maintain joint stability, equalize articular surface pressure distribution, and minimize anterior tibial displacement.[45,47] During the "drop squat" exercise it may be assumed that the role of quadriceps muscle is to decelerate the downward moving body via eccentric muscle action. Because the hamstring muscles are not the primary muscle involved in performance of the "drop squat" they may not be loaded to an extent that stimulates strength gains. The lack of change in isometric and isokinetic hamstring strength during this study appears to support this concept.

Another finding of this study was that although pain (intensity and unpleasantness) demonstrated a reduction during the 6 week intervention phase, it did not completely resolve. Curwin & Stanish[2] suggested that most subjects should be asymptomatic following performance of the EEP for a period of 6-8 weeks. They[2] have proposed that patients with "severe" tendinitis require a gradual return to full athletic involvement and the entire treatment period may take as long as 10-12 weeks. They[2] also reported most patients had minimal or no symptoms after performing the EEP for 6 weeks in their study treating 200 patients with various forms of chronic tendinitis. However, this study was not published in a peer-reviewed journal.[2]

Recently, it has been suggested that conservative management of chronic tendinopathy is a long-term process that may take 6-12 months or

longer to reach an acceptable outcome.[16] With regard to patients with CPT, Cook et al.[48] reported in a retrospective study of 100 subjects with CPT, that 33% of subjects were kept out of sport for over six months due to knee symptoms. Thus, the treatment duration suggested by Curwin & Stanish[2] may have been under-estimated and the results of the current study suggest physical therapists should not expect complete resolution of symptoms within 6-8 weeks.

With regard to the measures of intensity and unpleasantness of knee pain, it was apparent throughout this study that unpleasantness ratings were lower than intensity ratings. A possible explanation may be that because these subjects were able to function at relatively high levels (i.e. no impairment of functions such as walking), the symptoms of CPT were not as disturbing emotionally. The finding of intensity ratings being greater than unpleasantness ratings has also been documented in a study examining subjects with delayed onset muscle soreness.[25] Intensity and unpleasantness are considered two different domains of pain and the absolute scores may be different[25] because the emotional aspect of pain may not be as important to the subjects as intensity.

Another important finding pertaining to knee pain was that visual analysis demonstrated a pattern of increased intensity ratings (subjects #1 and #4) and increased unpleasantness ratings (subjects #1, #2, and #4) during the first 2-3 weeks of the intervention phase (Figures 2-5). This was consistent with the suggestion by Curwin[7] that subjects may experience an increase in symptoms during the first 2-3 weeks of the EEP.

An interesting pattern observed in this study was that the baseline linear regression line of all four subjects demonstrated an accelerating trend of intensity ratings. The baseline phase C-statistic for the intensity ratings of

subjects #3 and #4 demonstrated a significant upward trend. Additionally, three of the subjects (#1, #3, #4) had baseline linear regression line's demonstrating an accelerating trend in unpleasantness. The baseline phase C-statistic for subjects #3 and #4 demonstrated a significant upward trend in unpleasantness ratings. These findings may be an indication that the natural history of CPT involves progressive worsening of symptoms. In a retrospective study of 100 athletes with jumper's knee, Cook et al.[48] concluded that the natural history of jumper's knee is not self limiting. Additionally, other authors[12,49-52] have suggested that jumper's knee is a progressive condition.

Patients with CPT often report symptoms of stiffness or aching after prolonged sitting.[2,12,53,54] Additionally, pain may result from loading of the patellar tendon (i.e. sudden manual eccentric loading of the quadriceps or hopping on the affected side).[2,12,43,55] The provocative test (Appendix 2) was developed for this study in order to provide a consistent and reproducible stressor for the subject's self-rating of knee pain. The provocative test required that the subject hop down from a box (12 inches high) and land on the affected side foot. The provocative test was also designed to induce a sudden eccentric muscle action of the quadriceps femoris muscle of the affected side.

Other studies[18,20,52,54,56-60] examining the diagnosis or treatment of patients with CPT have not used a consistent stressor to provide a reference for measures of knee pain. These studies[18,20,52,54,56-60] used subjective scales or questionnaires, based upon recall of knee symptoms during various activities, to measure knee pain. Melzack[61] has suggested that when examining pain, both the intensity of pain and the affective component of pain should be assessed. The DDS[24] was implemented in this study to measure both the intensity of knee pain and the affective component (unpleasantness) of knee pain. Thus, a strength of this study was the use of a consistent stressor

(provocative test) as a reference for the measurement of knee pain, and the use of a scale (DDS) that examined both the intensity and affective component of knee pain in patients with CPT.

In 1978, Roels and colleagues[59] modified the scale developed by Blazina et al.[53] that classifies patients with CPT into 4 different phases according to pain and functional limitations. These two classification systems have been used in a variety of studies examining the diagnosis and treatment of patients with CPT.[20,52,56,59,62] However, a more detailed functional scale was desired for this study. The Knee Function VAS[26] was selected for this study because it contained 28 specific functional questions with a corresponding modified VAS (row of 10 boxes, not a single straight line). Specifically, the knee function VAS had questions pertaining to knee function on stairs, running, decelerating, cutting, jumping and squatting. These activities have been shown to be aggravating factors for patients with CPT.[13,18,20,53,55,63] In addition, the knee function VAS reliability and validity have been tested on a variety of patient populations, including patients with CPT.[27]

The results of the knee function VAS measures (Figures 6-9) indicated that three out of four subjects (#1, #2, and #4) had lower knee function VAS scores (improved knee function) at the end of the EEP. Improved knee function was also demonstrated by the fact that all subjects tolerated increasing amounts of hand-weight during the EEP (Figure 30).

However, at the end of the intervention phase and on follow-up testing, all subjects continued to report some degree of knee dysfunction. This finding further supports the contention that complete rehabilitation of patients with CPT may require a period greater than the 6-8 weeks suggested by Curwin & Stanish.[2]

A pattern of reduced knee function VAS ratings during the initial 2-3 weeks of the intervention phase was present for three of the subjects (#1, #2, #4). This pattern may be considered consistent with the observations of Curwin[7] that subjects may experience an increase in knee symptoms during the first 2-3 weeks of the EEP. Increased knee symptoms would be expected to adversely affect self-ratings of knee function. It has been shown that knee pain leads to reflex inhibition of the quadriceps while sparing the hamstrings.[44] Thus, increased knee pain would result in increased inhibition of the quadriceps and subsequently a reduction in knee function.

It should be noted that following the intervention phase, all subjects were advised to continue the EEP for at least 2 weeks. This followed the guideline of Curwin[7] that the EEP should be performed for 6-8 weeks. Compliance during the follow-up phase was not monitored. This may account for the continued improvement or maintenance of intervention phase improvements in knee function (decreased knee function VAS scores) for subjects #1, #2, and #3.

Daily activity levels (Figures 26-29) were monitored throughout this study to detect any changes in activity level that might occur during the intervention phase, and to assist in the interpretation of the other dependent variables (i.e. increased activity level resulting in increased DDS intensity score on a particular day). At the outset of this study, it was anticipated that if the EEP reduced knee symptoms and improved knee function, there would be an increase in the amount of daily activity during the intervention phase.

However, the daily activity levels did not consistently increase during the EEP. This may be due to a limitation of the daily activity log (Appendix 5) which will be discussed in the limitations section. All subjects demonstrated decelerating baseline linear regression line's for daily activity level (Figures 26-

29) which may provide evidence for a natural history of progressive deterioration for patients with CPT.

During the intervention phase, all subjects demonstrated markedly different patterns of activity. Subject #1 demonstrated a marked increase in intervention phase activity levels. However, as mentioned in the results section, this period coincided with the start of a manual labor job. Subject #3 demonstrated a marked reduction in intervention phase activity levels. However, as mentioned in the results section, this period coincided with a reduction in manual labor due to seasonal change. Subjects #2 and #4 had minimal change in daily activity levels.

Limitations

Limitations of the provocative test and DDS

A potential limitation in the collection of pain measures was that the provocative test may not have been demanding enough for subject #3. Subject #3 demonstrated very low intensity and unpleasantness ratings throughout the study, indicating the provocative test may not have effectively elicited knee symptoms. It may be the case that subject #3 had a milder case of CPT compared to the other subjects. Therefore, it may be necessary to develop a test that is relative to the patient's height and / or weight in order to effectively stress the extensor mechanism of the knee.

Limitations of the Knee Function VAS

All subjects within the study had knee function VAS scores below 50 out of a possible 100 (worst knee function rating possible). All subjects demonstrated limited variability of baseline and intervention data points, as compared to the other measures such as the DDS or isometric peak force tests. These findings may indicate a lack of sensitivity of the knee function VAS, or

that patients with CPT have impaired function only in higher level activities (i.e. running, jumping).

Limitations of Isometric Testing

The isometric strength testing of the quadriceps and hamstring muscles was performed in only one position throughout this study (Appendices 10 & 11). Isometric strength impairments may have been present at other joint angles not assessed within this study. However, in order to minimize the number of repeated measures, and potential aggravation of the involved knee, quadriceps and hamstring testing was performed at one selected position.

A potential limitation for both the isokinetic and isometric strength testing employed in this study is a violation of the specificity principle, which suggests that testing methods should be as similar as possible to the training method utilized.[64] The EEP examined in this study used a closed kinetic chain exercise ("drop squat") while strength tests (isometric and isokinetic) were performed in open kinetic chain positions (Appendices 10 & 11). Fry et al.[65] also suggested that training progress is most appropriately assessed when using the same modality for testing as for training. However, the transference between closed kinetic chain exercise training (i.e. leg press, squatting) and open kinetic chain testing (i.e. isokinetic dynamometer positions as used in this study) have been supported.[66]

Three subjects (#1, #3, #4) had accelerating baseline linear regression line's for the measures of isometric quadriceps peak force. The accelerating trends may be due to a learning effect on the Kin Com isokinetic dynamometer. However, if this were the case then all of the baseline linear regression line's should have demonstrated an accelerating trend. Additionally, accelerating baseline linear regression line's should have been present in all the isometric hamstring peak force measures.

Limitations of Isokinetic Testing

A potential concern is that the repeated isokinetic testing may be confounded by a learning effect. However, it has been reported that following initial familiarization with isokinetic testing, day to day variability in performance of isokinetic tests may be attributed to biological, technological, and associated random error in measurement rather than systematic learning effects.[67] Technological error in measurement was controlled in this study via self-calibration of the Kin Com isokinetic dynamometer. Standardization of testing procedures in this study controlled for other sources of measurement error.

Lower extremity dominance may be factor to consider in both isometric and isokinetic strength testing. It has been suggested that during isokinetic testing of non-athletic males and females, the nondominant knee produces less torque that the dominant knee.[68] However, Di Stefano et al.[69] reported that during isokinetic testing of athletic subjects, there was no difference between dominant and non-dominant legs. All subjects included within this study were considered to be athletic (Table 1). Lower extremity dominance was not determined in this study, however, knowledge of lower extremity dominance may have assisted in the data analysis.

Lysholm[70] has reported a significant inverse relationship between pain and torque during isokinetic quadriceps strength testing. It was reported that higher velocities (180 degrees per second versus 30 degrees pers second) produced more pain and less torque. Thus, knee pain must be acknowledged as a limiting factor in torque production throughout the study.

Limitations of the measure of Daily Activity Levels

A potential limitation of this measure pertains to the fact that only the duration of activity was monitored. Monitoring the "intensity" of activities according to the amount of stress or load that is placed upon the knee extensor

mechanism may have assisted in the interpretation of the data. For example, even though duration of activity may not have changed, the subject may have participated in activities of greater intensity (i.e. jumping sports) that were previously poorly tolerated.

Limitations of study design

The goal of using a multiple baseline design was to detect whether or not each time the intervention was introduced to each subject , consistent changes occurred in the dependent variables (knee pain, knee function, isometric and isokinetic quadriceps femoris and hamstring strength, and daily activity levels). Thus a multiple baseline design may demonstrate the effect of an intervention by showing the dependent variable changes when and only when the intervention is applied.[71] A multiple baseline design may be conducted concurrently (all subjects start the baseline at the same time) or non-concurrently (subjects enter the study as they become available and are randomly assigned baselines of different duration). This study employed a non-concurrent multiple baseline design across four individuals.

A potential limitation with multiple baseline designs is that some subjects are required to sustain a prolonged baseline phase.[71] The ethics of temporarily withholding treatment may be debated. However, all subjects provided informed consent to participate in the study and were informed they could withdraw from the study at any time without consequence. As it has been suggested that CPT is a progressive condition[48], the subjects exposed to prolonged baseline phases may have experienced continued deterioration of knee function and increased knee symptoms.

A limitation specific to non-concurrent multiple baseline designs is that external factors related to the passage of time may be different for each baseline.[34] For example, seasonal changes directly influenced the activity levels of subjects #1 and #3.

Clinical Relevance

This study has suggested that when employing the EEP[2] to rehabilitate patients with CPT, complete recovery may not occur within the recommended 6-8 weeks. Recent findings have supported this concept by showing that one third of athletes with CPT were unable to return to sport for more than 6 months.[48] Therefore, it is important that both the physical therapist and patient be aware of the potentially long conservative treatment period.

It has been reported that during the initial 2-3 weeks of the EEP patients may experience an increase in their symptoms.[2] The DDS and knee function VAS results of this study have supported this observation. It is important that the physical therapist and patient be aware of this initial exacerbation and avoid terminating the use of the EEP prematurely.

Summary of Hypotheses

The null and alternate hypotheses of this study were presented in the introduction. Alternate hypothesis #1 stated that following the 6-week EEP, there will be a reduction in ratings of the intensity of knee pain. This hypothesis was supported by three (#1,#3,#4) of the four subjects. Subject #2 demonstrated an increase, both visually and statistically, in intensity of knee pain during the intervention phase.

Alternate hypothesis #2 stated that following the 6-week EEP, there will be a reduction in ratings of the unpleasantness of knee pain. This hypothesis was supported by half of the subjects (#2,#4). The unpleasantness ratings remained at the same level between phases for subjects #1 and #3.

Alternate hypothesis #3 stated that following the 6-week EEP, there will be an increase in ratings of knee function (improved function). This hypothesis was supported by three (#1,#2,#4) of the four subjects. Ratings of knee function remained at the same level between phases for subject #3.

Alternate hypothesis #4 stated that following the 6-week EEP, there will be an increase in measures of average eccentric and average concentric torque of the quadriceps femoris and hamstring muscles of both lower extremities. The results related to this hypothesis are presented in Table 7.

Table 7. Summary of Alternate Hypothesis #4.

Alternate Hypothesis	Sides	Result
Quadriceps: Increased conc. average torque	affected side	supported by subjects #1,#3 (slight decrease subjects #2,#4)
	unaffected side	supported by all subjects
Quadriceps: Increased ecc. average torque	affected side	supported by subjects #1,#2,#3 (slight decrease subject #4)
	unaffected side	supported by all subjects
Hamstrings: Increased conc. average torque	affected side	supported by subjects #2,#4 (slight decrease subject #1, no change subject #2)
	unaffected side	supported by subject #1 (no change subject #3, decrease for subjects #2, #4)
Hamstrings: Increased ecc. average torque	affected side	supported by subjects #2,#4 (no change subject #3, decrease subject #1)
	unaffected side	supported by subject #1 (no change subject #3, decrease for subjects #2, #4)

Alternate hypothesis #5 stated that following the 6-week EEP, there will be an increase in measures of peak isometric force of the quadriceps femoris and hamstring muscles of both lower extremities. The results related to this hypothesis are presented in Table 8.

Table 8. Summary of Alternate Hypothesis #5.

Alternate Hypothesis	Sides	Result
Quadriceps: Increased peak force	affected side	supported by subjects #1,#2,#4 (no change for subject #3)
	unaffected side	supported by all subjects
Hamstrings: Increased peak force	affected side	supported by subject #2 (no change subjects #1,#3,#4)
	unaffected side	supported by subject #2 (no change subjects #1,#3,#4)

Alternate hypothesis #6 stated that following the 6-week EEP, there will be an increase in the amount of time engaged in physical activity. However, this hypothesis was supported only by subject #1. Subjects #2 and #4 had no change in activity levels between phases and subject #3 had a decrease in activity levels during the intervention phase.

Recommendations:

Recommendations for future research include testing the EEP^2 on patients with other forms of chronic tendinopathy. Further examination of the EEP^2 and other conservative treatment protocols is necessary to determine the most effective treatment for patients with chronic tendinopathies. Future studies examining the conservative treatment of patients with chronic tendinopathy may consider monitoring patients for periods up to 1.5 - 2 years. Further testing and development of a specific provocative test for the collection of pain measures in patients with CPT should be performed. Additionally, the development and use of measures that document direct change within the affected tendon tissue will assist in the evaluation of conservative treatment approaches.

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Appendix 1

Review of Literature

OVERUSE TENDON INJURY

INTRODUCTION

Overuse injuries occur when the body can not adapt to the cumulative stress of a repetitively applied force.[1] It has been estimated that 50% of all sports injuries are due to overuse and that the tissue most often affected is the musculotendinous unit.[2,3,4] Overuse injury tendon often involves an interaction between intrinsic factors (i.e. structural alignment) and extrinsic factors (i.e. over-training).[5] An understanding of these predisposing risk factors is necessary in the treatment and prevention of tendon overuse injuries.[5]

Leadbetter[6] suggested the pathology of tendon overuse injury may form a spectrum, with tendinitis at one end and tendinosis at the other. Leadbetter[7] proposed that most overuse tendon injuries may be classified as tendinosis, with degeneration and cell damage as the major components of the pathological picture. It has been reported that the goal of rehabilitation of overuse tendon injuries is not the relief of the symptoms associated with the *itis* , but to focus on the restoration of function that is lost with the *osis*.[8]

The purpose of this review is to discuss overuse tendon injury, with particular attention on patellar tendinitis (jumper's knee), with regard to the epidemiology, etiology, tendon structure and function, pathophysiology of injury, diagnosis, and treatment.

EPIDEMIOLOGY

Over the past few decades the role of sports and physical activity has become more valued in modern society.[3] This has been followed by an increased risk of sports injuries, especially overuse injuries, because athletes and fitness enthusiasts are training with greater durations and intensities.[3] The

number of overuse injuries is not known, however, in the United States it is estimated to be 30-50% of all sports related injuries.[9] Researchers[10] estimate that 50% of all sports injuries are due to overuse and the tissue most commonly affected is the musculotendinous unit (MTU).[2,3,4]

Two general variables of interest in the epidemiology of overuse tendinitis are age and gender. Adolescent athletes tend to have injuries more frequently at the boney insertions (i.e. Osgood Schlatter's disease, calcaneal apophysitis) of tendons rather than the tendon itself.[3]

Jarvinen[3] reported that the majority of patients with tendon injury are men. However, the proportion of female participants in sports injury surveys has increased during the past few decades from 14-18% to 20-30% in different sports medicine clinics.[11] The increase in proportion of females may be due to increased participation in sports and physical activity and increased interest in sports that have a high risk for acute tendon injury and overuse tendon injury.[3]

Approximately 30% of sports injuries treated on an outpatient basis in sports clinics concern the knee.[12,13] Kujala and colleagues[13] reported that in adults, patellar tendinitis and patellar chondropathy were the most common knee disorders. Patellar tendinitis at the inferior pole accounted for 20% of knee disorders while patellar peritendinitis / insertiotendinitis inferior accounted for an additional 6% of knee disorders. Kannus et al.[12] reported the most common knee disorders in adults were chronic Achilles tendinitis, iliotibial band friction syndrome, and jumper's knee. The incidence of patellar tendinitis is reported to have increased since its initial description due to increased participation of people in sports and fitness activities.[14,15]

ETIOLOGY

Overuse tendon injury may be caused by intrinsic or extrinsic factors, either alone or in combination.[5] Extrinsic factors usually dominate acute

injuries, while overuse injuries are usually multifactorial.[2,5] In order to correctly treat and/or prevent overuse tendon injuries it is necessary to address these factors.

Intrinsic Risk Factors

The most common intrinsic factors include malalignments, leg length discrepancy, muscle weakness and imbalance, decreased flexibility, joint laxity, female gender, age, obesity, and predisposing diseases.[5]

With regard to malalignment, several chronic tendon disorders have been associated with hyperpronation, including tibialis posterior tendinitis, Achilles tendinitis, patellar tendinitis, and iliotibial band friction syndrome.[16]

Leg length discrepancy has been suggested to be an etiological factor in the development of hip osteoarthritis, low back pain, trochanteric bursitis, patellar tendinitis, stress fractures, and iliotibial band friction syndrome.[17]

Muscle weakness and imbalance may predispose injury to the MTU by reducing the energy absorbing capacity, however, its significance in injury prevention is a matter of debate.[5] Kannus[5] stated the role of decreased flexibility is unclear as it is difficult to conclude whether it is a cause or consequence of injury.[5]

Post-traumatic joint laxity resulting in excessive motion in normal and/or abnormal directions may be a factor in tendon overuse injury.[5]

Kannus et al.[11] have reported a higher incidence of tendon and other overuse injuries among women. Differences in body anatomy and biomechanics may predispose women to overuse injury.[11]

Due to the risk of overuse injuries such as Osgood-Schlatter's disease or little leaguer's elbow, repetitive hard training should be avoided during rapid growth in children.[5] In elderly athletes, the sports injuries are more frequently due to overuse and commonly have a degenerative basis.[18] The effects of

aging on tendon structure and function will be discussed in further detail later in this review.

Extrinsic Risk Factors

Extrinsic risk factors related to chronic tendon disorders include excessive loading, training errors, environmental conditions, equipment, and rules governing sport.[5] A primary etiological factor in overuse tendon injury is that the tendon is subjected to potentially damaging forces.[1,19] The nature of tendon damage from excessive loading will likely vary with the type of force (compressive versus tensile), the magnitude of loading, and the pattern of application.[1] Excessive loading may be in the form of tension or compression.[20,21]

The application of tension to a MTU, generated either from its own structure or applied by external reaction forces, is a common etiological factor.[20] Discussion of tension will include the pattern of loading, the association with eccentric muscle action, and contributing factors to tensile loading.

With regard to the pattern of tensile loading, there may be a sudden application or removal of tension, or repetitive overuse.[20] Sudden application or removal of tension is reported to be a common mechanism of acute MTU injury.[1,22,23] This usually results in macrotraumatic injury to the tendon structure.[10] This injury may result in structural weakening (i.e. partial rupture) that predisposes the tendon to greater injury during subsequent loading.[21]

With repetitive overuse, the tendon is chronically subjected to relatively large loads which causes microscopic failure of some of the fibrils within the tendon, or slippage between fibrils, which leads to tendon injury.[24] Thus, physiological loads that are repeated too often to allow recovery of the tendon result in structural fatigue of the tendon.[20] This microtraumatic injury often

develops gradually and is often related to high training levels.[1,19] Leadbetter[6] has developed a theoretical model that illustrates the pathway of microtraumatic and macrotraumatic tissue injury (Figure 1).

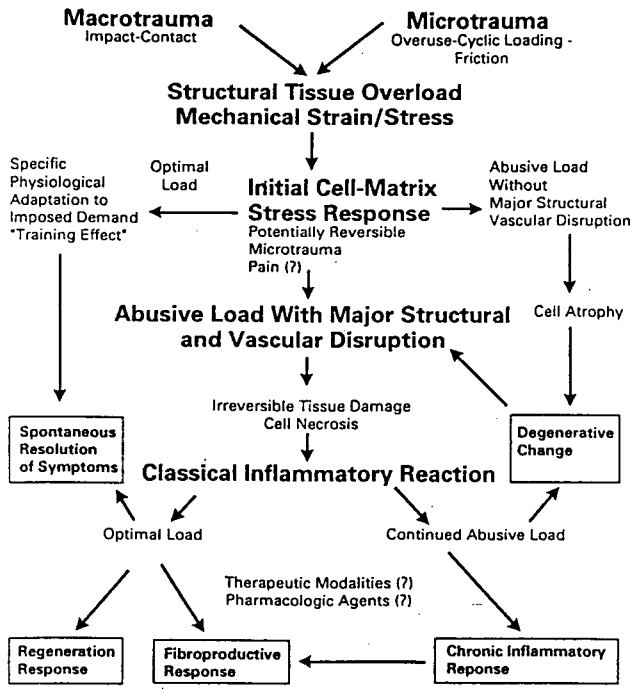


Figure 1. Microtraumatic and macrotraumatic pathways of tissue overload (adapted from Leadbetter[25]).

Eccentric muscle action is considered a significant factor in tendon injury.[2] The connection of tendon injury to eccentric muscle action is related to the fact the under eccentric conditions the tendon is exposed to larger loads than under concentric or isometric conditions.[1,23,26] Almost all shortening activations of the MTU are preceded by lengthening while the MTU is active (stretch-shortening cycle).[2,20] This pattern stretches the MTU, creating a passive force in the muscle due to elongation of elastic elements, and provides elastic energy. This elastic energy storage allows the muscle to produce more force at less metabolic cost. However, at this point there is a simultaneous

application of maximum force and maximum elongation to the tendon, which may cause damage.[20]

Compression is also a loading factor in overuse tendon injury.[20,21] The tensile strength of a tendon may decrease over time if it is subjected to chronic compressive loads.[20] The reduction in tensile strength is due to increased collagen turnover that results in fewer mature crosslinks.[20] Additionally, compressive loading may decrease tendon blood flow as demonstrated in supraspinatus tendinitis.[20] The intrinsic and extrinsic factors previously described may also apply to excessive compression.

Excessive loading of tendon, either by tension or compression, requires examination of the type of movements performed, the speed of movements, the number of repetitions, footwear (if applicable), and the surface on which the activity is performed.[5]

Training errors may include rapid progression (i.e. over-distance, intensity, hills), poor technique, and fatigue.[5]

A variety of environmental malconditions may predispose chronic tendon disorders, including dark, heat or cold, humidity, altitude and wind.[5]

Poor equipment may also predispose chronic tendon disorders, such as incorrect footwear while running or a tennis racquet that is strung too tightly.[5]

Finally, some rules governing sport are ineffective in preventing injury, and should be modified to aid in preventing overuse tendon injuries.[5] An example is restricting the number of pitches by a pitcher per season in junior baseball.[17]

ETIOLOGY OF CHRONIC PATELLAR TENDINITIS

The factors thought to contribute to the development of jumper's knee may also be classified as intrinsic or extrinsic.[27] Intrinsic factors may include age, alignment of the lower extremity, leg-length discrepancy, and height.

Extrinsic factors may include direct trauma, excessive tensile forces, and physical training parameters (frequency, type of training / technique, training surface, type of sport).

Ferretti[27] has reported that intrinsic factors are not the primary cause of jumper's knee but should be considered during treatment. It has been reported that in patients over 15 years age, jumper's knee may appear at any age with no significant peak, and there is no significant difference between the incidence of jumper's knee in males and females.[17]

Ferretti[17] has examined lower extremity alignment (knee alignment, Q-angle, patella position, femoral rotation, tibial rotation, foot structure, and height) in a group of patients with patellar tendinitis and reported no significant causal relationship. Cannell[28] reported that the average structural malalignment of patients with patellar tendinitis was no different from patients presenting with other overuse injuries. Kujala et al.[13] compared patellar tendon length radiographically between a group of normal subjects and a group of patients with patellar apicitis and reported significantly ($p<0.01$) longer patellar tendons in the patient group. They also reported a significant leg-length inequality (affected leg longer) ($p<0.001$) in the patient group. However, Duri & Aichroth[29] reported that leg-length discrepancy was not a feature in their patients with patellar tendinitis. Duri & Aichroth[29] also reported that 24% of the patients they examined demonstrated femoral anteversion. Martens et al.[30] have reported no significant correlation of height to the development of patellar tendinitis. However, Blazina et al.[14] reported that these patients are usually, but not always, tall. Duri & Aichroth[29] reported that 71% of the male patients in their sample were greater than 6 feet in height. Intrinsic factors may play a role in the development of patellar tendinitis, however, a clear pattern of abnormality does not appear to be present.

Direct trauma to the patellar tendon or its attachments has been reported to induce patellar tendinitis.[29,30] However, excessive tensile loading of the patellar tendon (i.e. repetitive or excessive eccentric loading as with jumping, running, and kicking) is thought to be the most important factor in the development of patellar tendinitis.[15,27,31] Richards et al.[32] reported that in elite volleyball players, large vertical ground-reaction force during the take-off phase of both spike and block jumps was a significant predictor of patellar tendinitis.

Ferretti[27] reported that training more than 4 times per week was a risk factor for the development of patellar tendinitis in a study on volleyball players. Duri & Aichroth[29] reported an increase in frequency of training sessions was responsible for 44% of the patients they examined with patellar tendinitis. However, they[29] did not quantify the increase in frequency of training sessions.

In Ferretti's[27] study of volleyball players, comparison of the training mode (either conventional weight lifting or plyometric exercises) showed no significant difference in the incidence of patellar tendinitis. Richards et al.[32] examined jumping biomechanics in elite volleyball players and reported that increased knee flexion angle on spike jump and block jump landings was a strong predictor of patellar tendinitis. Additionally, large tibial external rotation moments during spike jump and block jump take-off predicted patellar tendinitis.[32] It has been suggested that the knee valgus (tibial external rotation moment) moment may cause the patella to move into an unfavorable lever position and increase patellofemoral joint loads.[33]

In terms of the training surface, it has been reported that hard surfaces (i.e. concrete) are an important causative factor for jumper's knee in volleyball players.[27] Duri & Aichroth[29] reported that a hard training surface was an influencing factor in 63% of their patients with jumper's knee.

In terms of the type of sport engaged, Ferretti[27] interviewed 407 volleyball players and found 23% were suffering or had suffered typical symptoms of jumper's knee. In a non-random sample of patients with jumper's knee, Martens et al.[30] reported that the most common sports that patients engaged in were volleyball and soccer. Curwin & Stanish[1] reported in their survey that volleyball and basketball players represented 75% of the patients with patellar tendinitis.

STRUCTURE AND FUNCTION OF TENDON

Tendon Structure

In the most general sense, tendons are rope-like structures that attach muscle to bone at each end of the muscle. Tendon was once thought to be an inert biological structure, but is now regarded as a heterogeneic group of structures with variations in collagen orientation, cell character, collagen cross-linking, configuration, vascularity, load pattern, biomechanical profile, shape, and the presence or absence of synovial lining.[25] The mechanical properties of tendons are related to the constituents of the tissue, the relative amount of each constituent, and the structural organization of the constituents.[21]

Composition

Water, cells, fibers, and ground substance are the primary components of all collagenous tissues.[21] The fibers and ground substance are collectively referred to as the extracellular matrix.[21] The extracellular matrix is approximately 70% water.[21] The fibroblast is considered the cellular component of tendon and is responsible for the production of ground substance and fibers.[21] The fibroblasts comprise 20% of the tissue volume while the extracellular matrix comprises 70-80% of the tissue volume.[21] The fibroblasts are located within the fascicle between fibril bundles.[21] These cells are spindle shaped with thin cytoplasmic projections extending around the fibrils.[21]

Collagen is the primary fiber component of tendon and is reported to comprise 70-80% of the dry weight of tissue.[2,20] There are several forms of collagen but Type I collagen comprises 95% of the collagen present in tendon tissue.[20,21] Collagen is the major load bearing component in dense connective tissues such as tendon or ligament.[34] Collagen is relatively stiff and may undergo only a 10% increase in length prior to failure.[35]

Elastin fibers are present in very small amounts in tendon tissue.[21] These fibers may elongate up to 150-200% prior to failure[36,37] and contribute to some flexibility of tendon.[38]

The ground substance of tendon is composed of glycoproteins, proteoglycans, plasma proteins, and other small molecules.[21] The ground substance is a gel-like material that provides structural support and a medium for the diffusion of nutrients and gases.[38] The ground substance also produces friction that helps the collagen fibers to adhere to one another, yet at the same time provides lubrication and spacing that allows the fibers to slide back and forth and not become excessively crosslinked.[39,40] Glycosaminoglycans (GAG), which comprise only 1% of the tissue dry weight, are important because of their ability to bind to water which forms 65-75% of the total weight of tendon.[20] GAGs may combine with other GAGs or with a core protein to form proteoglycans.[20] Different types and amounts of proteoglycans are found in areas of tendons subject to compressive rather than tensile forces, and these differences are accompanied by variations in the amount, type, and organization of collagen in those areas.[41,42]

Structural Organization

The structural organization of tendon helps determine the tissue's ability to withstand stress and also the location and characteristics of tissue failure.[21] The structural unit of collagen is tropocollagen which is formed in the fibroblast

as procollagen.[38] Procollagen consists of 3 polypeptide chains that are woven to form a triple helix.[43] When procollagen leaves the fibroblast the non-helical peptides at both ends of the helix are cleaved enzymatically to form tropocollagen.[43] The tropocollagen molecules are staggered relative to adjacent molecules overlapping by 25% of the molecule length (Figure 2).[20] The formation of crosslinks between collagen molecules accounts for the tensile strength of collagen, prevents enzymatic, mechanical, or chemical breakdown, and helps direct the organization of collagen molecules into fibrillar structures.[44,45] Both intermolecular and intramolecular crosslinks can be formed. The intermolecular, or mature, crosslinks are thought to be an important indicator of a tendon's tensile strength.[44]

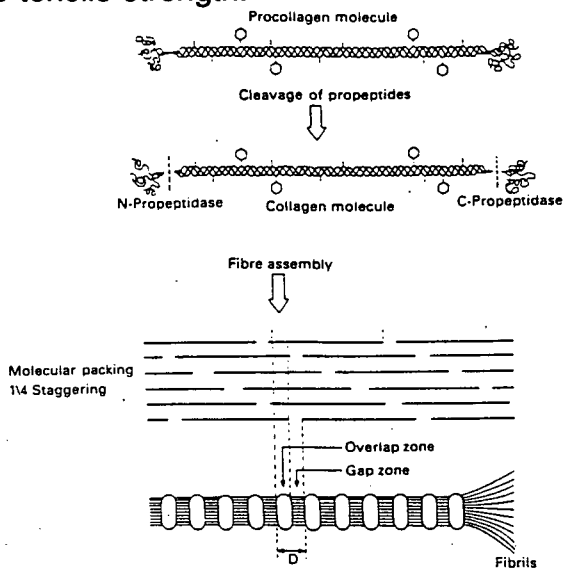


Figure 2. The conversion of procollagen molecules to collagen molecules and the molecular staggering (adapted from Curwin[20]).

In cross-section the following structure of tendon is present (Figure 3).[21] Five tropocollagen molecules form a microfibril. The microfibrils aggregate to form a sub-fibril and sub-fibrils group to form a fibril. A striated appearance at the fibril level during electron microscopy is due to the staggering of tropocollagen molecules.[43] A group of fibrils form a fascicle that is separated

from adjacent fascicles by a sheath called the endotenon. The endotenon also encloses the nerves, lymphatic system, and blood vessels supplying the tendon.[20] It has been reported that individual fascicles are associated with discrete groups of muscle fibers or motorunits and thus may be stressed independently of other fascicles if only those muscle fibers are activated.[20] The fascicles then group to form the gross tendon structure that is surrounded by a sheath called the epitenon.[20,21] Additionally the epitenon is covered by a double layered sheath of areolar tissue called the paratenon.[20] A small amount of fluid lies between the epitenon and paratenon and acts to decrease friction.[21] The paratenon is loosely attached to the outer surface of the epitenon.[20] The paratenon is more developed as a synovial sheath at sites where the tendon passes around boney or retinacular pulleys.[21]

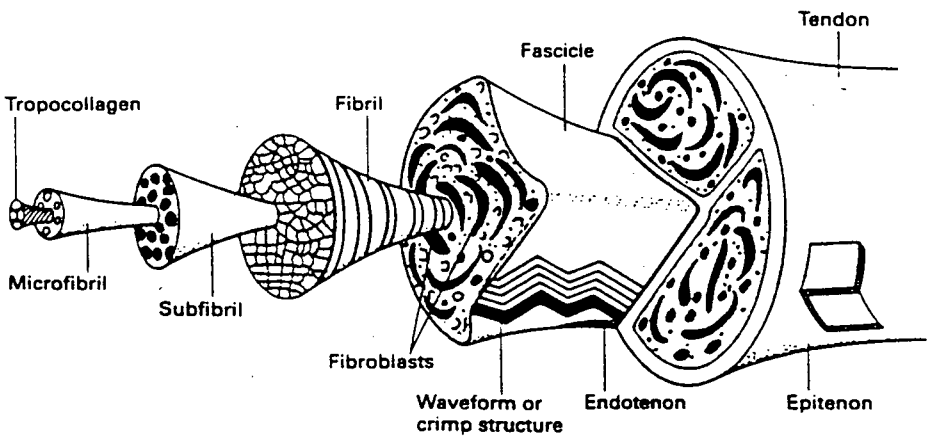


Figure 3. The hierarchical organization of tendon from molecule to tendon (adapted from Curwin & Stanish[1]).

Blood Supply of Tendon

The blood supply of tendon is highly variable and is usually divided into three regions (muscle-tendon junction, tendon, tendon-bone junction):[38]

Musculotendinous junction

At this interface, the blood supply is from the superficial vessels in the surrounding tissues.[38] Capillaries supply both the muscle and tendon but there is no anastamosis between the capillaries.[38] However, Clancy & Hagan[46] reported that larger vessels in the perimysium continue on with the endotenon.

Length of the Tendon

Those tendons with a paratenon have been described as vascular tendons, while those with a tendon sheath are referred to as avascular.[46] In vascular tendons the main blood supply to the middle section of tendon is via the paratenon.[38,46] Small vessels in the paratenon travel transversely toward the tendon and branch several times before running parallel to the long axis of the tendon.[38] The vessels enter the tendon along the endotenon and are flanked by two venules.[38] The capillaries and venules form a loop but do not penetrate the collagen bundles.[38] In tendons that are enclosed in a synovial sheath the main blood supply is via the mesotendon.[38] The mesotendon, also known as vincula tendinum, are folds of synovial membrane, strengthened by fibrous tissue, which conduct blood vessels to the tendon.[47] Sheathed tendons also receive nutrition from synovial fluid via diffusion.[46]

Tendon-Bone junction

At this interface the fibrocartilaginous layer between the tendon and bone block any direct anastamosis with vessels originating from the bone and vessels originating from the tendon.[38] It has been reported that indirect connections occur between vessels in the periosteum and endotenon.[1,19] Viidik[48] suggests the relatively poor vascularization at the bone-tendon junction may be one of the reasons why injuries at this site often become chronic.

The blood supply to tendon can become compromised at sites of friction, compression, or torsion.[38,46] Areas of hypovascularity in tendons have been

identified but their significance in the development of tendon overuse injuries is unclear.[49] Gross[21] reported that, in general, the vascular supply of tendon is sparse, and in some areas the tendon is relatively avascular. However, Hess et al.[2] suggested tendon is well vascularized tissue, although less so than muscle.

Nerve Supply of Tendon

The nervous supply to tendon is sensory in nature and is derived from the appropriate overlying nerve.[1,38] Proprioceptive information supplied by mechanoreceptors is sent to the central nervous system.[2] The afferent receptors are the Golgi tendon organs (GTO) and lamellated corpuscles which are located within 1 cm of the musculotendinous junction.[1,50] The GTOs lie in series with the extrafusal fibers and monitor increases in muscle tension.[38] The GTOs act to reduce muscle tension during excessive muscular contraction and prevent tendon injury.[38] The lamellated corpuscles respond to stimuli transmitted by the surrounding tissues (i.e. pressure produced by muscle contraction) and provide finely tuned feedback.[1]

Musculotendinous Junction

The musculotendinous junction is subjected to great mechanical stress during the transmission of contractile force from the muscle to the tendon.[38] The interface between two different physiologic tissues can be the site for stress concentration and potential failure under loading conditions.[21] As a result, this junction has been configured to minimized this concentration of stress.

The musculotendinous junction is a specialized area joining extracellular collagen fibrils to intracellular myofibrils.[51] It is a four layered region of longitudinal infoldings connecting the actin filaments of terminal sarcomeres to tendon collagen fibers.[52]

The folding of junctional membrane has three functional implications.[53] First, the folding acts to increase the surface area. It has been suggested the

surface area is increased 22.2 times greater than the junctional area where cell ends meet as right-circular cylinders.[54] Because stress is a function of force divided by the area, the increased area at the junction reduces the stress. Second, foldings align the membrane at low angles relative to applied force vectors, thus subjecting the membrane primarily to shear forces (Figure 4). Junctions loaded in shear where the force is parallel to the membrane surface are stronger than junctions where a large tensile component is perpendicular to the membrane.[54] Third, the folding creates an adhesive interface between the cell and extracellular matrix at low angles relative to applied force vectors.

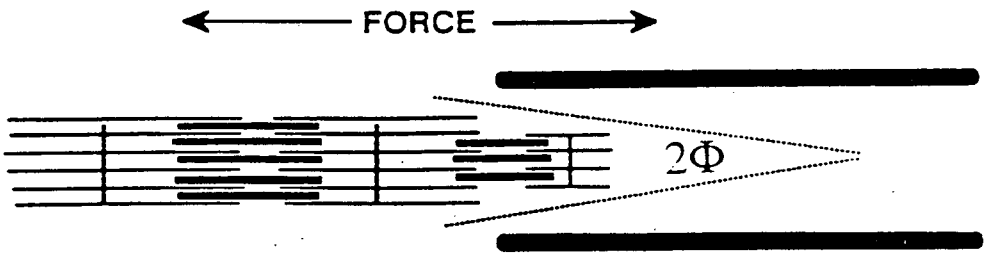


Figure 4. Diagram of the musculotendinous junction. The junctional membrane lies at an angle relative to the myofilaments (adapted from Zernicke & Liotz[51]).

Additionally, the stress concentration may be minimized by the transition provided by the terminal sarcomere units which are shorter than those within the rest of the muscle, generate less tension, and possess greater stiffness.[55] Gross[21] suggested that a weaker zone may lie between the middle and terminal sarcomere units and may be the location of failure at the musculotendinous junction, as it has been reported that avulsed tendons often have terminal sarcomere units still attached.[21] Indirect MTU injuries (i.e. delayed onset muscle soreness, muscle strains) are often located at the musculotendinous junction.[56]

Tendon-Bone Junction

The insertion of tendon into bone may be classified as direct or indirect.[57] A direct insertion is characterized by a small amount of superficial fibers blending with the periosteum and a large amount of deep fibers that approach the bone at right angles. As these deep fibers insert, they progress through four zones: tendon proper, fibrocartilage, mineralized fibrocartilage, and bone. A tidemark zone (blue line or cement line) separates the non-mineralized fibers from the mineralized fibers and marks the outer limit of calcification. The transition from tendon to bone occurs abruptly (approximately 1 mm).

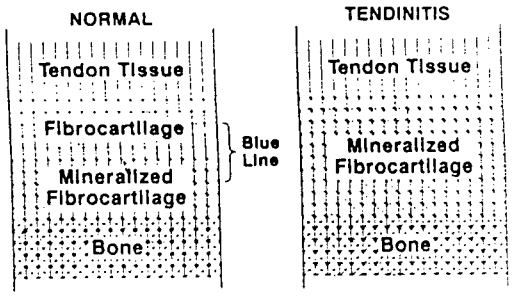


Figure 5. Zones of the osteo-tendinous junction. Loss of the blue line occurs in tendinitis as normal fibrocartilage undergoes mineralization and ossification (adapted from Molnar & Fox[58]).

An indirect insertion is characterized by a large amount of superficial fibers blending into the periosteum. The deep fibers approach the bone at an acute angle and do not progress through a fibrocartilage zone. A tidemark zone separates non-mineralized fibers from bone.

Patellar Tendon Structure

The extensor mechanism includes the following anatomical structures: the quadriceps femoris musculature and its tendinous attachment into the patella via the quadriceps tendon, the patellofemoral articulation, the patellar

tendon and its attachment to the patella proximally and the tibial tuberosity distally, and all supporting soft tissue.[59] The trochlear groove, between the femoral condyles of the femur, articulates with the patella to form the patellofemoral joint. The patella is the largest sesamoid bone and has a thick layer of articular cartilage on its posterior surface.[31] The patella is a rounded triangular bone with a superior base and inferior apex. The inferior surface of the patella is roughened for the attachment of the patellar tendon.[60]

The quadriceps femoris muscle is the primary extensor of the knee and is divided into four parts including the rectus femoris, vastus intermedius, vastus lateralis, and vastus medialis.[61] These parts unite into a single broad, flat tendon (quadriceps tendon) that inserts into the superior base of the patella.[62] Some of the more medial and lateral fibers of the quadriceps tendon pass alongside the patella and merge into the knee capsule, forming the medial and lateral retinacula.[31] Superficial fibers of the quadriceps tendon pass over the anterior surface of the patella and merge at the patellar apex to form the patellar tendon.[31] Thus, the patella is enclosed in the quadriceps tendon with a posterior opening for articulation with the trochlear groove of the femur.[63]

The patellar tendon originates at the inferior pole of the patella and inserts into the tibial tuberosity. It is attached to the proximal margins of the patellar apex and a depression in the inferior posterior surface of the patella.[63] The posterior aspect of the patellar tendon is separated from the synovial membrane of the knee joint by the infra-patellar fat pad, and from the tibia by the deep infra-patellar bursa.[63]

Blood supply to the quadriceps tendon is via branches of the superior genicular arteries while the patellar tendon is supplied via the inferior lateral genicular and recurrent anterior tibial arteries laterally, and from the inferior medial genicular artery medially.[64] Blood supply to the quadriceps tendon and

patellar tendon also arises from the musculotendinous junction, paratenon, or at the bone-tendon junction.[1]

Sensory innervation of the patellar tendon is derived from the infra-patellar nerve, a branch of the saphenous nerve.[65] Many GTOs (type III mechanoreceptors designed to prevent excessive tension) are located at the ends of these sensory nerves.[66] Additionally, Ruffinian corpuscles (type I mechanoreceptors that control postural muscle tone), Pacinian corpuscles (type II mechanoreceptors that control dynamic muscle tone), and free nerve endings (Type IV nociceptors responsible for sensitivity to pain) are located at the ends of these sensory nerves.[66]

Tendon Function

Tendons function to transmit the force produced by contractile and non-contractile elements within the MTU.[21] Force is transmitted through tendon to act on a boney segment and produce movement about a joint axis.[21] This may be accomplished in three ways. A tendon may travel a direct course along a line from proximal to distal insertions (i.e. Achilles tendon). A tendon may course around a boney pulley prior to insertion to a target bone (i.e. extensor pollicis longus around Lister's tubercle). A third route is having the tendon course around a retinacular structure prior to insertion to the target bone (i.e. extensor digitorum communis under the extensor retinaculum of the wrist).

Stress-Strain Relationship

Tendons are viscoelastic structures and as a result they are rate-dependent as well as size-dependent with regard to the stress-strain curve.[38] The presence of ground substance causes tendon at rest to appear crimped or have a wavy configuration that disappears once the tendon undergoes 2% strain (Figure 6).[38] This straightening of fibers in response to tension produces a toe region of the stress-strain curve of tendon.[38] Application of strain beyond

the toe region (2-4% strain) results in progressive deformation of tendon structures.[20] A linear relationship develops between the stress and strain following the toe region.[20] At 4-8% strain, the collagen fibers slide past one another as crosslinks start to fail.[38] The severity of tendon injury increases with the progressive collapse of lateral adhesions between components, beginning at the fibrillar level.[20] As fibrils begin to rupture, an irregular plateau region is rapidly followed by complete rupture of the tendon, usually at 8-10% strain.[67] O'Brien[38] suggested there is a large gap between stresses that are experienced at physiologic loading (<4% strain[68]) and those that cause tendon failure (at 8-10% strain). Curwin[20] reported that most tendon injuries classified as tendinitis probably involve loading in the linear region of the stress-strain curve. Curwin[20] reported tendon damage can occur during tissue loading or unloading. This may explain why both the application and release of unexpected or sudden force is often associated with tendinitis.[20]

Curwin[20] suggested that there are three factors affecting the stress-strain curve. First, the greater the size of a tendon (cross-sectional area) the greater the stress that can be tolerated. Second, the longer a tendon the greater the absolute change in length (but not percentage strain) before fibril disruption. Third, the variability of tendon composition results in variable stress-strain curves between different tendons.

Tendon also demonstrates stress relaxation and creep as do other viscoelastic connective tissues.[69] Stress relaxation refers to a gradual decrease in tension (stress) while a constant length (strain) is maintained. Creep refers to a gradual increase in length (strain) while a constant level of tension (stress) is applied.

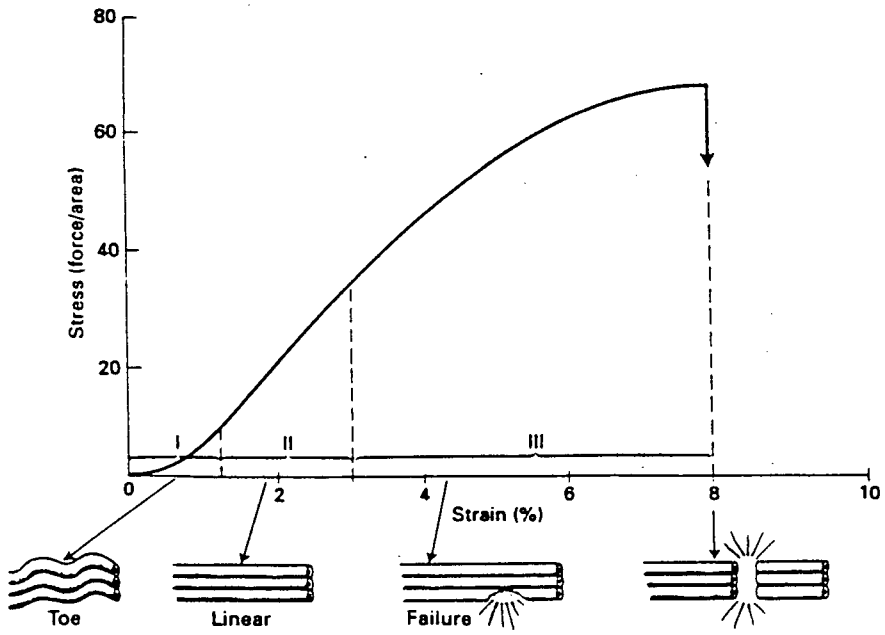


Figure 6. The stress-strain relationship for tendon. A toe region demonstrates straightening of crimped fibrils that is followed by a linear region up to 8% strain (adapted from Curwin[20]).

It must be recognized that tendon is not only subject to tensile stress, but to friction and compression against associated pulley mechanisms or external sources (i.e. tight shoe laces).[21]

Factors Affecting the Mechanical Behavior of Tendon

Tendon is not an inert structure, it is metabolically active and undergoes a continual process of resorption and repair.[21] A variety of factors can alter the balance of tendon resorption and repair including exercise, disuse, aging, vascularity, hormonal influences, genetic influences, and nutritional influences.

Healing and normal tendons can adapt to increased load (exercise) either by hypertrophy or by changing their material properties to become stronger per unit area.[70,71,72] Tendons have been shown to increase their connective tissue content in response to increased load conditions.[72,73] Exercise increases collagen synthesis, the number and size of fibrils, and the concentration of metabolic enzymes.[74] Exercise also changes collagen crosslinking, alters water and electrolyte content, and changes collagen fibril

arrangement.[2] These changes act to increase the maximum static strength and tensile strength of tendons.[38,74]

All musculoskeletal tissues atrophy under conditions of decreased load.[20] Both collagen content and crosslinking decrease and the tissue becomes weaker.[75] Inactivity results in increased collagen degradation, decreased tensile strength, and decreased concentration of metabolic enzymes.[76]

Aging has a detrimental impact on tendon and increases the likelihood of overuse injury.[2] Aging is characterized by a failure to maintain homeostasis under conditions of physiologic stress.[25] The highest frequency of tendon failure in humans occur at ages greater than 30 years and may reflect progressive deterioration of collagen.[4] Yamada[77] reported a fairly constant ultimate strength of human Achilles tendon from the 3rd through 6th decades, with a rapid decline beginning in the 7th decade.

The effects of aging include a decline in collagen content, a reduction in the quality of labile collagen, a reduction in the tensile strength of collagen, an increase in the density and aggregation of the extracellular matrix that alters permeability and affects tendon nutrition, a decrease in elastin and proteoglycan matrix resulting in less elasticity, and a reduction in water content from 80% at birth to approximately 30% in old age.[2,21,38,78] These changes result in increased tendon stiffness and increased risk of shear stress injury.[25,38]

It has been reported that tendon injuries often occur in areas of reduced blood supply.[21,49] This may provide evidence toward the theory of hypoxic intratendinous degeneration and the etiology of tendinosis (see pathophysiology section).[25]

It has been reported that decreased estrogen levels, premenopausal hysterectomy, and premature menopause may be associated with the incidence of tendinosis in women.[79] Additionally, people with diabetes are known to have difficulty with soft tissue healing which may be related to insulin levels.[25] Endocrine responses to stress, such as increased glucocorticoid and catecholamine release, may have a negative effect on connective tissue[80], increasing turnover and thus resulting in decreased crosslinking.[81]

Mesenchymal syndrome may be a potential genetic influence in failed tendon healing.[79] This condition results in tendinosis at multiple sites in approximately 15% of patients and in sites not subjected to obvious overuse. Also, people having blood type O have been statistically related to tendon rupture.[82]

It is known that a normal healthy diet provides the appropriate amounts of amino acids for protein synthesis and the necessary co-factors, such as vitamin A, vitamin C, and copper, important for collagen synthesis and crosslinking.[20] However, the nutritional influences on chronic tendinitis remain largely unexplored.[20]

Patellar Tendon Function

The patella functions as a critical component of the knee extensor mechanism by acting as a fulcrum to increase the extensor moment arm.[83,84] The patella has been estimated to increase knee extension force by 50%.[85] The force transmitted in the quadriceps tendon is not equal to the force in the patellar tendon during muscle contraction.[31] It has been shown in-vitro that as the knee flexes in an open kinetic chain, the tension in the patellar tendon becomes less than the tension in the quadriceps tendon.[86] Ellis and colleagues[87] corroborated these results by reporting that tension in the patellar tendon in-vitro reduced to approximately 50% of that in the quadriceps tendon

when the knee was flexed to 80-100 degrees in an open kinetic chain. This was thought to be a function of the geometry of the patellofemoral joint, not due to frictional forces. However, in a closed kinetic chain, quadriceps and patellar tendon tension increases with increasing knee flexion.[85]

The quadriceps tendon and patellar tendon are able to withstand high tensile loads but are unable to withstand high compressive loads or friction.[88] Analysis of patellar tendon tensile forces during descending and rising from a deep squat demonstrate that maximum forces occur when descending (eccentric quadriceps muscle action).[89] This phenomenon was attributed to a larger momentum achieved while descending, requiring a larger "kick" to decelerate the body.[89] Patellar tendon forces have been estimated to be 16 X body weight when landing from a height of 42 inches.[90] Patellar tendon failure has been observed during weight lifting at loads estimated to be 17.5 X body weight.[91]

PATHOPHYSIOLOGY OF OVERUSE TENDON INJURY

Curwin & Stanish[1] have suggested that overuse tendon injury results from repetitive tendon strain of 4-8% until injury occurs. The tendinous tissue may become fatigued until the repair processes are overcome by microtraumatic destructive processes.[5] If the associated muscle becomes fatigued or weak, the ability of the MTU to absorb tension is reduced, and the tendon may be subjected to greater tensions.[5] The amount, rate, frequency and duration of stress applied to a tendon may have implications for the pathogenesis of chronic tendon injury.[5] Safe amounts of exercise that allow tissue adaptation are probably separated by a fine line from exercise that overcomes the normal cellular maintenance and repair.[49]

A theoretical classification system for chronic tendon injury has been developed by Leadbetter.[25] This system consists of four separate pathological conditions: paratenonitis, tendinitis, tendonosis, and paratenonitis with tendonosis. *Paratenonitis* is inflammation of the paratenon alone and includes such terms as peritendinitis, tenosynovitis, and tenovaginitis. *Tendinitis* is an injury that results in an inflammatory repair response that may lead to secondary paratenonitis. *Tendinosis* is considered intra-tendinous degeneration without an inflammatory response. *Paratenonitis with tendinosis* consists of paratenon inflammation with intra-tendinous degeneration. It has been reported that patients with chronic Achilles tendon symptoms often reveal the pattern of paratenonitis with tendinosis.[92] Backman et al.[93] have induced chronic tendinitis in rabbit Achilles tendons and have demonstrated that most inflammatory changes take place in the paratenon and are accompanied by areas of focal degeneration within the tendon (tendinosis).

The pathways that lead to tendinosis are not well understood.[94] Tendinosis is often found in conjunction with chronic forms of peritendinitis and tendinitis, however, it must be noted that in a healthy population aged 35 years or more, asymptomatic degenerative tendon changes can be found in every third person.[94] Several theories exist that attempt to explain the degenerative process. Leadbetter[25] suggested that a failed cell matrix adaptation to excessive loading results in tendinosis. Continued abusive loading may stimulate the release of cytokines, resulting in both autocrine (cell self-stimulation) and paracrine (stimulation of adjacent cells) modulation of further cell activity. It is suggested that the processes of inflammation, degeneration, and regeneration form a spectrum of cell matrix responses depending upon the mechanism of injury and the homeostatic balance of the tendon tissue.

Leadbetter[25] has developed a model of how the tendon cell matrix responds to changes in load use (Figure 7).

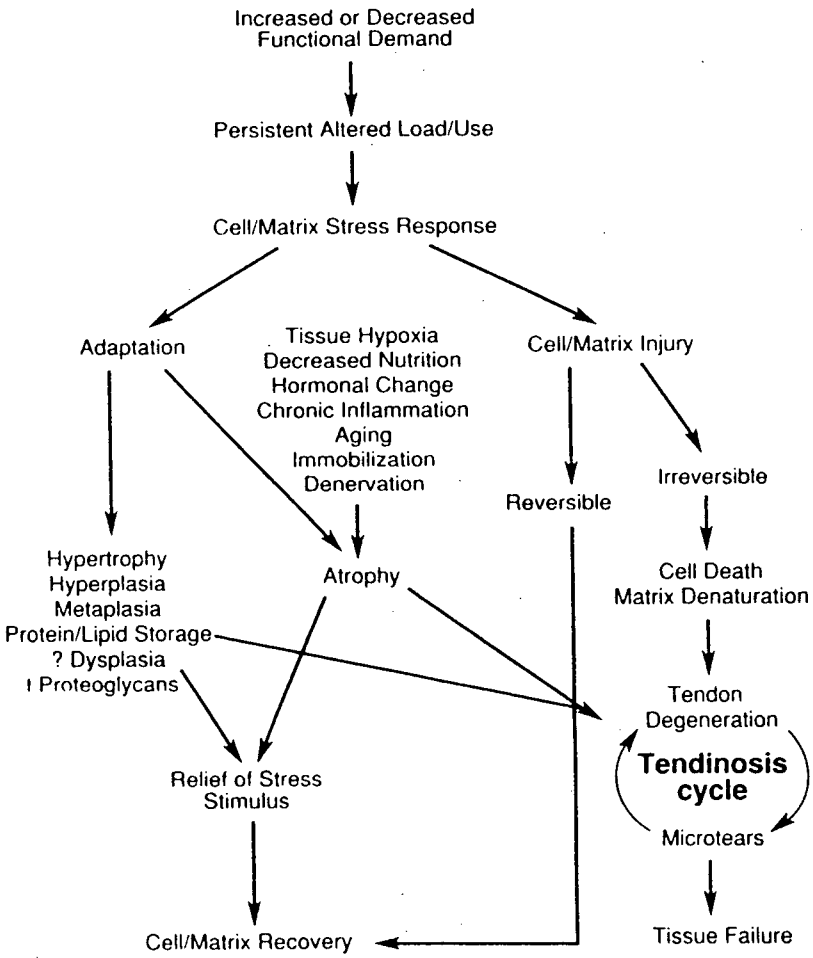


Figure 7. Cell matrix response to change in functional level. Tendinosis results from failed cell matrix adaptation to excessive load use (adapted from Leadbetter[25]).

Histologically, tendinosis is a collection of entities (i.e. hypoxic degeneration, mucoid or myxoid degeneration, hyaline degeneration, fatty degeneration, fibrinoid degeneration, fibrocartilaginous metaplasia, boney metaplasia, fiber calcification, or some combination of these) that affects tendon cells (tenocytes), collagen fibers, and the cell matrix.[5] The etiology and pathogenesis of tendinosis in different individuals and different tendons is likely variable.[5]

Tissue hypoxia is thought to be a possible mechanism in tendon degeneration.[49] Mature tenocytes are capable of oxidative metabolism and thus hypoxia may decrease the viability of these cells and lead to degeneration.[5,49] Overuse may cause injury to the microvasculature and macrovasculature, impairing oxygen transport and metabolic activity for tissue repair.[5]

Another theory of tendon degeneration involves oxygen-derived free radicals.[49] Because certain areas of tendon may be subjected to hypoxia during exercise due to regional hypovascularity, ischemia-reperfusion injury involving free radicals may occur.[5]

Another theory suggests that degeneration, usually located in the core of tendons, may be due to exercise induced hyperthermia.[95] Analysis of cyclically loaded equine tendon resulted in peak intratendinous temperatures of 43-45 degrees Celsius. Since temperatures greater than 42 degrees Celsius result in fibroblast death and collagen damage in-vitro, Wilson & Goodship[95] suggest hyperthermia may be a factor in tendon degeneration. Relatively avascular regions of tendon may lack the ability to dissipate heat and suffer hyperthermic damage during exercise.[95]

PATHOPHYSIOLOGY OF CHRONIC PATELLAR TENDINITIS

With regard to patients with patellar tendinitis, most abnormalities have been located at the enthesial region of the posterior, proximal patellar tendon insertion.[96] Histological examination has revealed tenocyte hyperplasia, disorganized small vessel ingrowth, endothelial hyperplasia, and collagen fiber disorganization.[97] Yu and colleagues[97] reported that no polymorphonuclear cells, lymphocytes, or macrophages were present in any of the specimens. Ferretti et al.[98] reported evidence of collagen degeneration without the classic signs of an inflammatory response at the proximal patellar tendon. Ferretti et al.[98] also reported pseudo-cystic cavities between the border of the mineralized fibrocartilage and bone. Molnar and Fox[58] have reported the loss of the "blue line" between the fibrocartilage and mineralized fibrocartilage at the osteo-tendinous junction. It must be recognized that these histological studies were performed on patients who underwent surgical treatment of chronic patellar tendinitis because of unsuccessful conservative treatment. These findings may represent the late, fibrotic stage of patellar tendinitis and do not preclude the potential for an earlier presence of inflammation.[99]

CLASSIFICATION OF TENDON INJURY

A unifying classification system for overuse tendinitis does not exist and classification may be done according to anatomical site[2], pathophysiological changes[25], or function.[1]

Table 1 summarizes the anatomical locations of common tendinous injuries.

Table 1.
Common sites of overuse tendinitis

Tendon Involved	Common Name
Achilles	Achilles tendinitis
Patellar	'Jumper's knee'
Common wrist extensor tendon	Lateral epicondylitis, 'tennis elbow'
Common wrist flexor tendon and pronator teres	Medial epicondylitis, 'golfer's elbow'
Supraspinatus	'swimmer's shoulder', 'impingement'
Other rotator cuff tendons	rotator cuff tendinitis and / or tears
Biceps (long head)	bicipital tendinitis
Extensor pollicis brevis and abductor pollicis longus	de Quervain's syndrome
Posterior tibial	'shin splints'
Iliopsoas, rectus femoris, sartorius, pectineus, adductors	'groin pull'
Biceps femoris, semitendinosus, semimembranosus	'hamstring pull'

(adapted from Hess et al.[2])

The pathophysiological classification has been best detailed by Leadbetter[25] in Table 2. Gross[21] has suggested that the differentiation between these various pathophysiological classes may not be possible based upon clinical examination and may be evident only on surgical examination. Additionally, Gross[21] reported that the conservative treatment may not vary for the involvement of the tendon or a tendon sheath.

Table 2.
Pathophysiological Classification of Tendon Injury

Terminology	Pathology	Clinical Signs and Symptoms
paratenonitis	inflammation of paratenon only	Cardinal inflammatory signs: swelling, pain, crepitation, local tenderness, warmth, dysfunction.
Paratenonitis with tendnosis	paratenon inflammation associated with intratendinous degeneration	Same as above, with often palpable tendon nodule, swelling, and inflammatory signs.
Tendinosis	intratendinous degeneration due to atrophy (aging,micro-trauma, etc) of tendon sheath.	Often palpable tendon nodule that can be point tender. No swelling of tendon or
Tendinitis Acute (< 2 weeks) Subacute (4-6 weeks) Chronic (> 6 weeks)	symptomatic degeneration of tendon with vascular disrupt-ion and inflammatory repair response	Symptoms are inflammatory and proportional to vascular disruption, hematoma, or atrophy-related cell necrosis. Symptom duration defines each subgroup.

(adapted from Leadbetter[25])

Curwin and Stanish[1] provide a functional classification system for overuse tendinitis in Table 3. Curwin[20] has also classified tendinitis as either extrinsic or intrinsic. The extrinsic form implies that the condition results from forces outside the tendon, such as compression. An example would be a large acromion process impinging upon the supraspinatus tendon. The intrinsic form results from changes or inadequacies within the tendon. In this case the tendon is simply not strong enough to tolerate the applied tensile forces.

Table 3.
Functional Classification of Tendon Disorders

Intensity	Level	Pain	Performance
Mild	1	none	does not affect performance
	2	with extreme exertion only not intense disappears when activity stops	does not affect performance
Moderate	3	starts with activity lasts 1-2 h after activity	performance might be affected
	4	with any athletic activity increases during activity lasts 4-6 h after activity	performance level slightly decreased
Severe	5	immediately upon any activity involving tendon sudden increase in pain if activity is continued lasts 12-24 h after activity	performance markedly curtailed or prevented
	6	during daily activities	unable to participate

(adapted from Curwin & Stanish[1])

TENDON HEALING

Tendon injury may be divided into acute or chronic according to the rate of onset and mechanism.[25] Acute injuries involve a sudden trauma followed by a predictable resolution. Acute tendon injury often consists of mid-substance ruptures occurring either as a result of high strain rates or aberrant tissue.[71] Chronic injuries are characterized by a slow, insidious onset, indicating that a prior sub-threshold spectrum of structural damage results in a crisis episode involving pain and / or signs of inflammation.[25] Chronic injury may last months

or years and is distinguished by a persistence of symptoms.[25] An overlap between acute and chronic injury may exist at 4-6 weeks and is often termed the subacute stage of injury.

The mechanism that results in the progression from an acute inflammatory process to a chronic inflammatory process is not known. However, continued abusive load and irritation may stimulate the local release of cytokines, resulting in both autocrine and paracrine modulation of further cell activity.[100]

Leadbetter[25] described two categories of connective tissue injury: macrotraumatic, involving acute tissue destruction, and microtraumatic, involving chronic abusive load or use.

Macrotraumatic Tendon Injury Response

An acute injury results in regeneration, fiber-productive response and repair by scar tissue, or some combination of both.[25] Acute connective tissue injury may be classified into three phases that occur along a continuum.[7]

Acute Inflammatory Response

Leadbetter[25] reported three major consequences of this phase: (a) some initial wound strength is provided by crosslinking of fibronectin in collagen, (b) removal of damaged tissue from the initial trauma, and (c) endothelial cells and fibroblasts are recruited and stimulated to divide. The fibroblasts from the tendons epitenon and endotenon, granulation tissue, and phagocytes migrate to the site of injury.[101] This inflammatory phase lasts 3-5 days.[21,25] By day 5, there has been an increase in monocytes, eosinophils, neutrophils, lymphocytes, platelets, and macrophages.[102] It has been reported that immediately following injury the tendon is unable to synthesize collagen.[103] However, sparse and scattered collagen fibrils may be present at varying levels of maturity.[102]

Repair-Regeneration Phase

This phase begins at 2 days and last up to 6-8 weeks.[25] This phase is characterized by the presence of macrophages which are mobile and capable of releasing a wide variety of growth factors, chemotractants, and proteolytic enzymes when necessary for the activation of fibroblasts and tendon repair.[25] The reparative cell is the tenocyte, which when activated performs like a fibroblast or fibrocyte.[25] The tenocytes increase in size and number at the injury site.[101,103] The tenocyte is the source of collagen production, protein mediators of repair, and matrix proteoglycans.[25] Initially type III collagen in a woven pattern is rapidly deposited and later shifts toward the deposition of type I collagen which continues into the final maturation phase.[25] This shift from type III to type I production occurs as crosslinks form in the microfibrils, and as the hierarchical organization of fibrils into fibers occurs.[101] Driving this stage is a relative hypoxia in the wound micro-environment and rising lactate released by macrophages.[104] During this phase there is increasing vascularity and a relative paucity of inflammatory cells.[103]

Remodelling / Maturation Phase

This phase is characterized by a trend toward decreasing cellularity, a decrease in synthetic activity, increased organization of the extracellular matrix, and a normal biochemical profile.[25] If physiological loads have been applied, collagen maturation and functional linear realignment are usually seen by two months post-injury.[21,25] Biochemical differences in collagen type and arrangement, water content, DNA content, and GAG content persist indefinitely and the material properties of the scar never equal those of the intact tendon.[105] This remodelling process essentially goes on throughout life, but is most pronounced at 17-28 days post injury.[103]

Chronic Microtraumatic Tendon Injury Response

This form of injury is characterized by histological evidence of degeneration, especially in cases of spontaneous tendon rupture.[14] This degenerative process is thought to be due to a hypoxic degenerative process involving both tenocyte and matrix components.[94] Inflammatory cell infiltration and orderly phased wound repair as seen in macrotraumatic injury are absent or aborted.[25] This injury may be termed tendinosis consisting of a focal area of intratendinous degeneration that is initially asymptomatic.[25] The pathohistology of overuse tendon injuries in human adults display varying degrees of the following: tenocyte hyperplasia, a blast-like change in morphology from normal tenocyte appearance, prominent small vessel ingrowth with accompanying mesenchymal cells, paravascular collections of histiocytic or macrophage like cells, endothelial hyperplasia and microvascular thrombosis, collagen fiber disorganization with mixed reparation and degenerative change, and collagen fiber microtears and separations.[25] The collagen fibers may also demonstrate longitudinal splitting, disintegration, angulation with unique bent-fiber appearance (knicking), and abnormal variation in fiber diameter.[94] The synovial sheath and paratenon may be involved in microtraumatic injury, especially as a result of friction with excitation of the synovial cells.[25] Inflammatory cell populations may be prominent in the synovium and peritendinous structures, as well as the surrounding areas of intratendinous calcification, and at sites of previous intratendinous steroid injection.[25] Electron microscopy analysis of microtraumatic tendon degeneration may show alterations in size and shape of mitochondria in the tenocyte nuclei[25], and intracytoplasmic or mitochondrial calcification.[106] Mitochondrial injury during tendon degeneration may result in

the deposition of chalky appearing hydroxyapatite crystals in the collagen matrix.[25]

In discussing acute and chronic tendon injury, Leadbetter[25] has developed injury profiles to visualize the events that occur. The acute injury is characterized by a defined time of onset with the trauma episode being a sudden event (Figure 8).

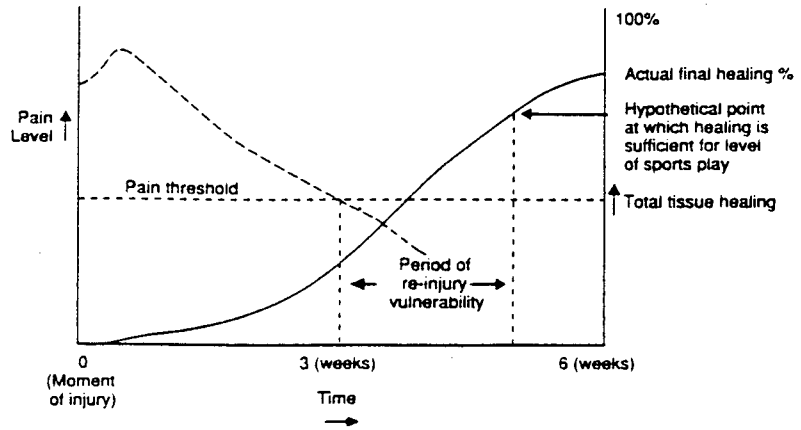


Figure 8. Hypothetical profile of acute macrotraumatic tissue injury. Curved dashed line = pain; curved solid line = tissue healing (adapted from Leadbetter[25]).

At the moment of injury, pain is likely to be severe and will gradually decrease with treatment. There is a period of vulnerability that is proportional to the severity of the injury, the rate of healing of the individual, the nature of the tissue injured, and the expected load demand upon return to activity. The pain that occurs in an acute injury evolves from the onset of the inflammatory cascade.[25]

In chronic injury, there is a moment of noxious injury that often occurs after overexertion that may be mildly inhibiting or very disabling (Figure 9). Inquiry into the pre-injury training patterns and cumulative load exposures is critical to determine why the tissue response has occurred. A subclinical injury (microtrauma) likely precedes the moment of conscious injury . The accumulation of repetitive scar adhesions, degenerative change, and adverse

effects in chronic microtrauma imply a slower recovery than for an acute injury. The pain associated with chronic injury is related to loss of tissue strength that leads to increased deformation with loading and the stimulation of mechanoreceptors.[25]

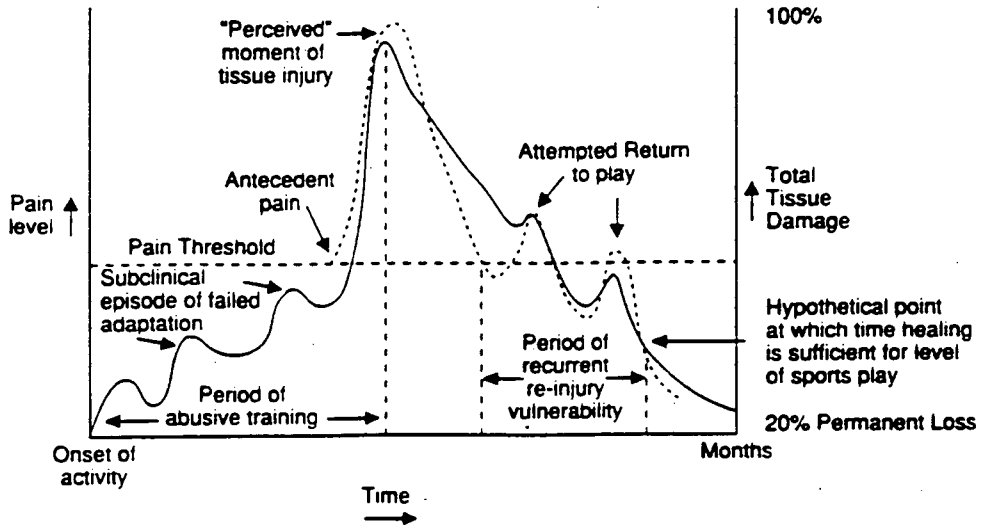


Figure 9. Hypothetical profile of chronic microtraumatic tissue injury. Curved dashed line = pain; curved solid line = percentage of tissue damage (adapted from Leadbetter[25]).

Torstensen et al.[15] reported there is no information about the natural healing process in chronic patellar tendinitis. Much of what is known about tendon healing is from studies on severed tendons[75,107] and it is unknown if the same process occurs in chronic tendinitis.[20] Histological studies[96,97] suggest that chronic patellar tendinitis may be in either the fibroblastic or remodelling stages. Only recently has a model been developed to induce chronic tendinitis in animals that may allow for the study of the healing process.[93]

DIAGNOSIS

Generally the history enables identification of a mechanism of injury that may be consistent with the etiological factors of overuse tendinitis presented earlier. It is critical to obtain an accurate history as the complaints may be

vague and nonspecific and performance unaffected in mild cases.[10] Typically the affected individual will continue with the athletic activities because the associated pain will disappear at times, such as with warmup.[2] An accurate history will also allow appropriate functional classification of the injury (see Table 3).

An anatomically based systematic physical examination is necessary as the classic signs of inflammation (redness, heat, swelling, pain) are not always present or identifiable.[25] An affected tendon may appear enlarged if it lies superficially.[21] The suspected MTU should be isolated, palpated, and stressed appropriately.[10] Palpation may detect local enlargement of the tendon and may elicit pain.[21] Palpation may also detect rupture or partial rupture of the tendon.[2] Pain may be elicited by passively stretching the involved MTU or having the patient actively contract the suspected MTU.[21] Functional tests are also useful in eliciting pain (i.e. hopping with suspected patellar tendinitis).[2] Because alterations in strength or biomechanics of other parts of the kinetic chain might occur in an attempt to compensate for decreases in function, the examination must be thorough and include all the potentially affected tissues.[10] Inflexibility of the involved MTU, weakness of the involved muscle or surrounding muscle, and / or muscle strength imbalance in the force couple are clinical findings that may be associated with overuse tendinitis.[8,20]

The diagnosis of patellar tendinitis is primarily determined through the patient's history and physical examination. The patient will usually report an insidious onset of pain.[14,15,31] However, on rare occasions, direct trauma to the patellar tendon and its attachments (i.e. kicking, fall onto knee) may be reported.[14,31] The pain is usually very localized to the patellar tendon or its attachments and is exacerbated by eccentric forces sustained just before the takeoff phase of jumping or during the landing phase of jumping.[1,61] The pain

may also occur with various sporting activities, squatting, and while using stairs.[1,29] Blazina et al.[14] developed an outline of the progression of pain and loss of function in patients with patellar tendinitis which was later modified by Roels et al.[108]

Table. 4
Phases of Patellar Tendinitis
Phase 1: Pain after practice or a game.
Phase 2: Pain at the beginning of the activity, disappearing after warming up and reappearing after completion of the activity.
Phase 3: Pain remains during and after activity and the patient is unable to participate in sports.
Phase 4: Complete rupture of the tendon.
(adapted from Roels et al.[108])

These patients will often report stiffness of the knee after prolonged sitting ("movie sign").[1,29,31,61]

Tenderness upon palpation of the inferior patellar pole (proximal attachment of the tendon), the patellar tendon itself, or the tibial tuberosity (distal attachment of the tendon) generally confirms the diagnosis of patellar tendinitis.[1] The most common site of tenderness is the inferior patellar pole.[29,109] Visible quadriceps wasting is sometimes present in these patients.[29,61] In rare cases, increased temperature of the skin overlying the tendon may be detected and peri-patellar tendon swelling may be noted.[1,15,30,110] Pezzullo et al.[31] reported that pain may be induced by passive terminal flexion of the knee. Pain is often elicited by application of a sudden eccentric load to the knee extensor mechanism or when the knee is extended against resistance.[1,13,31]

Other diagnostic techniques such as radiography, computerized tomography, magnetic resonance imaging, ultrasonography, and bone scans have been applied to these patients.[30,60,66,96,110-120] Radiography has been reported to demonstrate elongation of the inferior patellar pole or areas of ossification within the tendon substance in long standing cases, however,

radiographs of the patella are thought to be rarely helpful in the routine diagnosis of patellar tendinitis.[30] Radiographs may assist in ruling out other conditions such as patellar fractures or congenital anomalies of the knee.[61] The use of bone scans, capable of showing increased radioisotope uptake at the patellar pole, has been supported for pre-operative assessment of patients with chronic patellar tendinitis.[112] However, bone scans have been deemed unhelpful in routine diagnosis and management of patients with chronic patellar tendinitis.[112] Magnetic resonance imaging (MRI) has shown value in detecting and classifying the degree of tendon injury in several studies.[96,113] MRI is considered the gold standard for visualization of tendon pathology, however its considerable expense is a disadvantage.[121]

Ultrasonography is considered the most sensitive and specific instrument for imaging the patellar tendon and is recommended over radiographs, computed tomography, and magnetic resonance imaging.[60,66,110,111,113,114,118-120] Ultrasonographic changes have been reported to be consistent with histopathological findings of focal tendon degeneration.[113,114,119] Additionally, several authors have developed ultrasonographic classification schemes for patellar tendinitis.[116,118] Karlsson and colleagues[118] have classified patellar tendinitis (defined as partial patellar tendon ruptures) as grade 1 (<10mm), grade 2 (10-20mm), and grade 3 (>20mm). Wiley and colleagues[116] categorized patellar tendinitis as grade 1 (normal size), grade 2 (enlarged tendon), and grade 3 (enlarged tendon and hypoechoic region).

However, Lian et al.[117] demonstrated that specific ultrasound findings (i.e. paratenon changes, hypoechoic zones, or pathological proximal tendon thickening) in male elite volleyball players did not correlate significantly with the degree or duration of patellar tendinitis symptoms. Myllymaki and

colleagues[120] have suggested that patellar tendon lesions detected by ultrasonography are indicative that surgery is necessary. However, Khan et al.[115] reported that patellar tendon hypoechoic zones can resolve, remain unchanged, or expand in female athletes without predicting the symptoms of jumper's knee. As a result, Khan and colleagues[115] suggested sonographic hypoechoic regions should not constitute an indication for surgery. Several authors[66,121] have acknowledged that the execution, interpretation, and reproducibility of ultrasonography are strictly related to the examiner.

The differential diagnosis of patellar tendinitis includes peri-patellar disorders, intra-articular disorders of the knee, and patellofemoral disorders.[15] Peri-patellar disorders include fat pad inflammation (Hoffa's disease), bursitis, and apophysitis. Fat pad inflammation may be differentiated in that there is often swelling of the pad, and pain is elicited when the pad is squeezed manually or when the knee is hyperextended with over-pressure.[1,66,122] Bursitis at the knee (suprapatellar, subcutaneous infra-patellar, subcutaneous prepatellar, deep infra-patellar), especially the deep infra-patellar form, may mimic patellar tendinitis.[66] However, it is uncommon to have pain localized at the tibial tubercle (deep infra-patellar bursitis) beyond adolescence. Apophysitis proximally (Sinding-Larsen-Johansson disease) or distally (Osgood-Schlatter's disease) may occur in adolescence and will demonstrate localized tenderness and positive radiographic findings.[66]

Intra-articular disorders of the tibio-femoral joint include meniscopathies and degenerative joint conditions. Meniscal injuries often result in knee locking, giving-way of the knee, joint effusion, and sharp pain localized at the joint line.[1,66] Degenerative joint conditions may present with tenderness and pain localized at the joint line, effusion, crepitus, increased temperature or redness of the skin overlying the joint, and loss of joint range of motion.

Patello-femoral disorders include patello-femoral arthrosis, chondromalacia, and synovial infra-patellar plica. Patello-femoral arthrosis and chondromalacia may be confused with patellar tendinitis because these conditions can produce pain on squatting and stiffness after sitting, however, there is no tenderness at the usual sites of patellar tendinitis.[1,66] Additionally, the patellar grind test is often positive with these conditions.[66] Synovial infra-patellar plica can provoke locking sensations in knee extension and the pain is located at the superomedial or superolateral aspect of the patella.[66]

TREATMENT OF OVERUSE TENDON INJURY

Treatment of overuse tendinitis will be discussed in terms of conservative approaches, surgical management, and prevention.

Conservative Treatment

The goals of rehabilitation of patients with overuse tendinitis include: the relief of symptoms (primarily pain), to decrease inflammation when present, to promote healing and restoration of tissue integrity, and the return of the patient to sports and functional activities as soon as possible.[2,10] Conservative treatment is a long-term process that may take 6-12 months or longer to reach an acceptable result.[121] Rehabilitation may be divided into three phases that parallel the phases of tissue repair, including the acute phase, the recovery phase, and the maintenance phase.[2,8,10]

Acute Phase

The aim of treatment in this phase is to avoid further tissue disruption, prevent prolonged inflammation, promote ground substance synthesis, and prevent disruption of new blood vessels and collagen.[2,20] Treatment may consist of active rest with protection as needed (i.e. brace or cast), ice, anti-inflammatory modalities, and anti-inflammatory medication.[8,20] It has been suggested that once tissue healing allows, that protected range of motion

exercises be progressed to isometric exercises followed by light short-arc isotonic contractions.[8]

Recovery Phase

The goals of this phase are to increase collagen crosslinking, increase fibril size and alignment, and prevent the negative effects of reduced activity on the muscle and joint.[20] Appropriate loading is the stimulus for effective healing and it has been suggested that once full active range of motion is achieved that resistance exercises progress from isometric to concentric to eccentric.[10] At this stage it is suggested that patients begin task specific exercises and that emphasis be placed on regaining eccentric strength.[10] Modalities may be used to stimulate collagen synthesis, such as ultrasound.[20] Muscle re-education is important to ensure smooth kinetic chain function.[8]

Maintenance Phase

This phase is said to begin with return to play and continues to ensure overload does not recur.[8] Both strength and flexibility need to be restored using functional progressions of sport-specific drills.[8] Criteria for return to play include full range of motion, normal strength, normal balance, no alteration in biomechanics, and completion of a sport-specific activity progression.[8] It is advised that the maintenance program be continued indefinitely.[121]

Curwin[20] has suggested several principles for treating chronic tendinitis. First, it is important to identify and remove all negative external factors such as external compression or faulty biomechanics. Curwin[20] has suggested that inflexibility of the MTU is the most common contributing factor. Other intrinsic and extrinsic factors predisposing overuse tendon injury have been previously discussed in the etiology section. Second, estimate the phase of healing based upon clinical judgement. Curwin[20] acknowledged that this is based upon clinical experience and is very imprecise. Third, determine the appropriate

focus of the initial treatment to match the estimated stage of healing. Curwin[20] suggested most forms of chronic tendinitis are at the stage of remodelling and therefore force application is the most effective treatment. However, review of the pathophysiology of chronic microtraumatic tendon injury indicates that the classic healing sequence is not applicable and that degeneration may be the key process.[25] It is unknown whether degenerative tendon responds to exercise in the same manner as remodelling tendon. Fourth, institute the appropriate tensile loading program. This is based upon the concept that healing tendon must be loaded if collagen synthesis, alignment, and maturation via crosslinking are to be ideal.[93] Fifth, control any pain and inflammation through the use of ice, medication, and modalities.

The Role of Eccentric Exercise

Curwin[20] has suggested that a modality based approach is incorrect in the treatment of chronic tendinitis and that exercise should be the cornerstone of treatment. This is based upon the concept that tendon is capable of adapting to increased tensile loads and that chronic tendon injuries are a result of tensile loads exceeding the tendon's mechanical strength. Gross[21] outlined two justifications for the use of eccentric exercise. First, eccentric exercise creates the highest magnitude of forces on the MTU[26,123] and that this form of loading is appropriate to facilitate maturation of strong tendon. Second, functional eccentric loading trains individuals to activate muscles at the appropriate time to decelerate body segments. Improved deceleration may decrease the average forces transmitted through the tendon during repetitive functional activities, thereby reducing the risk of injury recurrence. Giffin & Stanish[10] reported that unless the MTU is trained to withstand maximum stresses, it will break down.

The Eccentric Exercise Program

The eccentric exercise program follows the principles discussed earlier and was originally developed by Curwin & Stanish.[1] The overall program has five steps (Table 5). The EEP consists of a warm-up, stretching of the involved MTU, the eccentric exercise, repeat stretching of the involved MTU, and then the application of ice to the affected MTU.[1]

The reasoning behind the warmup is to prepare the musculoskeletal system for action and its effects have been previously described.[124]

The rationale for the stretching phase, as alluded to earlier, is that inflexibility has been suggested to be a predisposing factor in chronic tendinitis.[1,20] It is theorized that stretching restores normal joint mobility and reduces the strain on the tendon during normal motion.[103] It is not known whether a tight MTU is a predisposing factor or a consequence of reduced use to protect the injured area.[103]

Curwin & Stanish[1] outlined several principles regarding the eccentric exercise. They suggested that the specificity principle[125] must be applied such that loading is progressed toward the type, speed and magnitude of loading required for the activity the patient wishes to resume. It is important to simulate the movement pattern associated with maximal tendon forces where lengthening is followed by a shortening contraction. In the case of chronic patellar tendinitis, Curwin and Stanish[1] recommended the use of eccentric squats as this is the context in which maximal loading occurs (i.e. landing or propulsive phase of jumping).

Next, the principle of progressive overload[126] is applied as it has been reported that increased loading is essential to induce adaptation in musculoskeletal tissues.[74] Curwin and Stanish[1] advocated the use of eccentric exercises due to the force-velocity relationship[26] in which eccentric muscle

action at high velocities results in the greatest tension measurements. Curwin and Stanish[1] used the symptom of pain as an indication of overload and stated that appropriate overload is achieved when the patient reports pain between the 20th and 30th repetitions of the eccentric exercise. However, it has been reported that a person's response to pain is a very inconsistent component of injury.[127]

Once the exercise is pain-free at 30 repetitions, it is then progressed by increasing the load or the velocity of eccentric exercise. It has been documented that, as the velocity of eccentric muscle action increases, the tension increases.[26] Progression of loading is also necessary whereby either the speed of the eccentric exercise or the external resistance are increased based upon the patient's symptoms.

The physiological responses reported to occur with cold application have been summarized by Knight[128] and include: decreases in temperature, inflammation, metabolic rate, circulation, and muscle spasm; an increase in tissue stiffness; and a transient increase, followed by a decrease, in pain. The theory behind the application of ice in chronic injuries is that the major problem is a chronic inflammatory response and thus ice is used to decrease this inflammatory process.[128]

The exercises are performed daily, with continuous progression, until the symptoms are no longer apparent with activity. The method of exercise progression is demonstrated in Figure 10. Curwin[20] reported that patients should be asymptomatic after 6 to 8 weeks of the program. Curwin[20] indicated there are no rules to guide reintroduction to activity but suggests that the patient be asymptomatic during activities of daily living and be performing the eccentric exercise rapidly. It is suggested that athletic involvement should start at 25% of the pre-injury level on alternate days and that progression be made in 10%

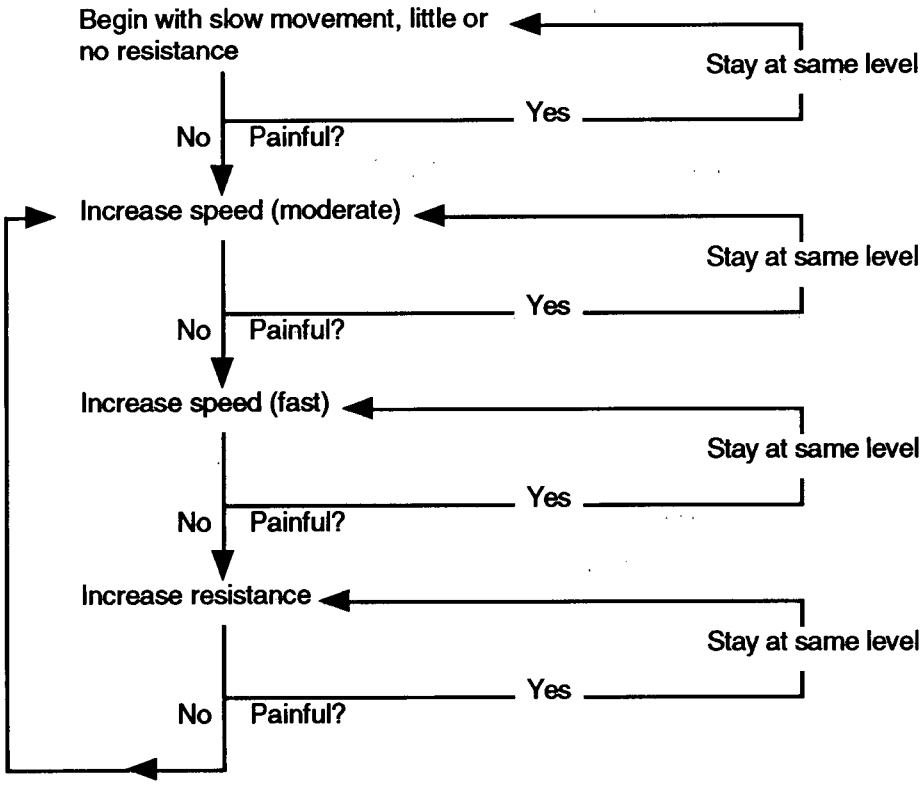
increments until full training has been resumed (taking approximately 8 weeks).[20]

Table 5.
Daily eccentric exercise protocol

1. Warm-up
    a. generalized exercise to increase body temperature and increase circulation
    b. should not load involved tendon
2. Stretch
    a. static stretch
    b. hold 30 seconds
    c. repeat 3 to 5 times
3. Eccentric exercise
    a. three sets of 10 repetitions; should feel reproduction of symptoms after 20 repetitions
    b. progression:
    Days 1 and 2: slow
    Days 3 to 5: moderate
    Days 6 and 7: fast
    c. increase external resistance; after day 7 repeat cycle
4. Stretch, as prior to exercise.
5. Ice: applied to tender or painful area for 10 to 15 minutes.

(adapted from Curwin[20])

Figure 10. General outline of eccentric exercise program (adapted from Curwin & Stanish[1])



Curwin[20] reported the most common reason for failure of the program is incorrect progression where either the patient is not progressed to the next level of intensity or the patient starts at too high a level. Curwin[20] indicated that some patients may experience a slight increase in symptoms during the activity in the first 2 to 3 weeks of the program.

Evidence for the Eccentric Exercise Program

Since the first presentation of a systematic eccentric exercise program by Curwin & Stanish[1] the use of eccentric exercise in the rehabilitation of chronic tendon injury has been frequently advocated.[4,10,20,22,103,129]

However, there is a paucity of evidence for the use of the eccentric exercise program.[121] In their book, Curwin and Stanish[1] presented survey data of 200 chronic tendinitis patients (chronic patellar tendinitis, Achilles tendinitis, and lateral epicondylitis). All patients were treated with the eccentric exercise program consisting of a warmup, stretching of the involved muscle-tendon unit, specific progressive eccentric exercises, repeated stretching, and application of ice to the involved MTU. They reported complete relief of symptoms in 44% of the subjects and marked reduction in pain and functional disability in 43.5% of the subjects. These reports appear remarkable, however, it should be noted these results were not published in a peer-reviewed journal.

Review of the literature indicates that only two groups have evaluated the efficacy of eccentric exercise in the treatment of chronic tendon injury. Jensen & Di Fabio[130] examined the effects of eccentric quadriceps training on the Kin Com with chronic patellar tendinitis patients. After the eight-week program no significant increase in eccentric quadriceps work was detected in the affected extremity. They did not comment upon changes in function of the subjects

during the intervention period. They postulated that pain or a protective reflex may have prevented strength gains in the affected limb.

Niesen-Vertommen and colleagues[131] compared eccentric and concentric plantarflexor strength training programs in chronic Achilles tendinitis patients over a 12-week period. The subjects reported significant reductions in pain with the eccentric training but did not demonstrate significant plantarflexor strength gains or faster return to activity compared to the concentrically trained group.

These studies[130,131] indicated eccentric training may reduce pain in chronic tendinitis patients, however, improvement in strength and function were not clearly demonstrated. These studies also employed eccentric exercise programs significantly different than that suggested by Curwin and Stanish.[1] Future studies need to specifically test the eccentric exercise program and evaluate it in terms of its effects on pain, strength and function in chronic tendinitis patients.

Review of Conservative Treatment Approaches for Patients with CPT

Various authors have proposed treatment protocols for patients with patellar tendinitis.[1,14,88,118,132] Blazina et al.[14] developed a protocol according to their classification system of patellar tendinitis (Table 6). This protocol has been tested experimentally and will be discussed in the following section.

Table 6
Treatment of patients with patellar tendinitis according to phase
Phase 1: ice pack or ice massage post-activity, anti-inflammatory medication, elastic knee brace or patellar brace.
Phase 2: same as in phase 1 along with heat application before activity. Steroid injection may be considered if symptoms and functional status worsening.
Phase 3: same as in phase 2 along with a prolonged period of rest. Patient advised to give up the offending sport or to consider surgery if these steps fail.
(adapted from Blazina et al.[14])

Curwin & Stanish[1] developed a specific protocol for patients with chronic patellar tendinitis, based upon the use of eccentric exercise to stimulate adaptation of tendon (Table 7). As noted in the previous section, Curwin & Stanish[1] have reported survey results of this protocol.

Table 7
Eccentric Exercise Program for Jumper's Knee

1. Warm-up
   a) General whole-body warm-up.
   b) Exercises not involving knee extension.
   c) Sufficient when sweating is elicited.
2. Stretching
   a) static stretch of quadriceps and hamstrings.
   b) Hold at least 30 seconds.
   c) Repeat 3 times.
3. Main Program
   a) Squatting movments.
   b) Focussing primarily on the rapid deceleration phase between the downward and upward movement phase.
      Week 1: No added resistance on days 1 and 2 (slow); days 3 to 7 (progressively faster).
      Week 2: Add resistance (10% body weight).
      Week 3 to 6: Add 10 to 30 lb progressively.
   c) Do three sets of 10 repetitions once daily.
   d) After 6 weeks, three sets of 10 three times weekly.
4. Warm-down
   a) static stretch as in item 2
5. Ice
   a) Ice on patellar tendon for 5 minutes after program.
6. Optional support
   a) Apply tensor bandage support if desired.

(adapted from Curwin & Stanish[1])

Black & Alten[132] suggested a protocol involving eccentric exercise of the ankle dorsiflexor muscles and hypothesized such treatment may : 1) stretch the patellar tendon, 2) change the quadriceps:foreleg strength ratio, 3) allow increased absorption of forces by the anterior compartment during activity, 4) improve the biomechanics of the foot and ankle, and 5) change the dynamics of the distal extensor mechanism. There is no known experimental evidence for this protocol.

Karlsson et al.[118] proposed and tested a protocol divided into three phases and based upon progressive strengthening and stretching of the quadriceps femoris (Table 8). Eccentric quadriceps femoris strengthening was considered a key component of the treatment. Results of this treatment protocol will be discussed in the following section.

Table 8

Acute Phase (daily training, 2-4 times a day):
1. Submaximal isometric contractions of the quadriceps for 3-5 seconds at different knee angles with 10 seconds of quadriceps stretching between each set of isometric contractions (4-6 sets, 30 repetitions).
2. Straight leg raising with 0-4 kg weights in a prone position and while lying prone on the uninjured side (2-4 sets, 20 repetitions).
3. Standing straight leg pulls with 0-5 kg weights on both legs in flexion, extension, abduction, and adduction (2 sets, 15-25 repetitions).
4. Walking on a soft mattress (5 minutes).
5. Stretching of the quadriceps muscle (3 sets, 10 seconds).
6. General strengthening exercises for the ankle, hamstrings, and hip muscles.

Rehabilitation Phase (daily training, 1-2 times a day):
1. Concentric and eccentric full-range knee extension exercises with 0-4 kg weights in a sitting position (4 sets, 20-40 repetitions).
2. Bicycling (5-30 minutes), 50-300 Watts.
3. Concentric and eccentric leg presses with 5-100 kg weights while standing, sitting, or lying with the knee at 90 degrees of flexion (2-4 sets, 20-40 repetitions).
4. Walking and jogging exercises on a soft mattress (5-30 minutes).
5. Stair and obstacle walking and jogging (5-30 minutes).
6. Eccentric full-range knee extension with 4-30 kg weights (2-4 sets, 20-40 repetitions). (performed when item1 is painfree).
7. Eccentric one leg presses with 50-100 kg or body weight while standing, sitting, or lying (2-4 sets, 20-40 repetitions).

Return to Activity Phase (3-4 times a week):
1. Exercises 6 and 7 (rehabilitation phase) continued.
2. The speed and load of exercises 4 and 5 (rehabilitation phase) are increased.
3. Bouncing and jumping activities are gradually added to further stimulate the elastic properties of the muscle and tendon and improve coordination and endurance.
(adapted from Karlsson et al.[118])

Eifert-Mangine[88] outlined a treatment protocol divided into three phases for recreational athletes with patellar tendinitis. (Table 9). As with the other protocols, eccentric quadriceps femoris strengthening was a prominent feature.

Table 9
Rehabilitation managment of recreational athletes with patellar tendinitis:

1. Initial Phase (weeks 1-4):
   a) Relative rest.
   b) NSAID's.
   c) Therapeutic modalities (ice, ultrasound).
   d) Flexibility exercises (3-5 reps, 20-30 seconds, stretch hams, calf, ITB, quads, TFL).
   e) Lower extremity strengthening in pain free range (especially VMO).
   f) General conditioning: strengthening of hip flexors, abductors, and adductors.
2. Intermediate Phase Pre-Activity (weeks 5-8):
   a) Therapeutic modalities.
   b) Continue flexibility exercises.
   c) Open kinetic chain eccentric quadriceps control.
   d) Closed kinetic chain eccentric quadriceps.
   e) Four position hip strengthening.
   f) Start endurance training (pool, bike, x-country ski machine).
   g) Balance training.
   h) Sport specific progressive training.
3. Return to Activity Phase (weeks 9-12):
   a) Continue flexibility exercises.
   b) Continue strengthening.
   c) Running program and skill specific activities.
   d) Aerobic conditioning.
   e) Patient education (return to activity, prevention).

(adapted from Eifert-Mangine et al.[88]).

The results of several different treatment protocols have been published. Several groups[30,108] have tested the protocol suggested by Blazina et al.[14] Roels et al.[108] reported the subjective treatment outcomes of 23 patients with CPT (graded as Phase 1, 2, or 3 according to Blazina et al.[14]) as "very good" (return to sport at same level, no symptoms), "good" (return to sport at same level, slight discomfort), or "poor" (no substantial improvement). Those patients with phase 1 and 2 CPT demonstrated a "good" response to treatment while those with phase 3 CPT had a "poor" response. Martens et al.[30] reported the subjective treatment outcomes of 90 patients with CPT (graded according to Blazina et al.[14]) as "excellent" (no symptoms), "good" (reduced symptoms, able to continue sport, some ache and/or stiffness following activity), or "poor" (residual complaints interfering with sports activity). They reported a minority of patients became symptom-free with conservative treatment and that a majority

of patients were rated as "good". The phase 3 patients frequently failed conservative treatment.

Ferretti et al.[109] reported the subjective treatment outcomes in 110 patients diagnosed with CPT (classified according to Blazina et al.[14]). The treatment involved adequate warm-up before activity, ice packs, stretching and strengthening of the quadriceps, physical therapy, and injection of hydrocortisone in 11 cases. A detailed description of the treatment application and exercise progression was not provided by the authors[109]. At follow-up patients were asked to rate their knee(s) with regard to pain and restriction of sporting activity and the results were reported as "very good", "good", and "poor". Following conservative treatment, where prolonged rest or a reduction in sports participation was not required, 62% of the patients rated as 1st and 2nd stage CPT were reported to be "very good" or "good". Third stage CPT patients did not recover as well, with 69% rated as "poor" following conservative treatment. Surgery was required for 38% of the stage 3 CPT patients.

Jensen & Di Fabio[130] reported the results of an 8-week isokinetic eccentric quadriceps femoris strengthening protocol on eight patients with CPT. The velocity of isokinetic exercise was progressed throughout the 8-week period. No significant increase in eccentric quadriceps work was detected in the affected extremity following the program. They postulated that pain or a protective reflex may have prevented strength gains in the affected limb.

Fritschy & de Gautard[60] also reported follow-up results of conservative treatment in 19 patients with CPT. They reported the treatment involved "classic" conservative techniques, physical therapy, electrotherapy, deep transverse frictions, and ice. However, no further detail was provided on how the treatment was implemented. On follow-up, all patients considered themselves healed.

Karlsson et al.[118] reported the results of their conservative treatment (outlined above) in 81 patients (91 symptomatic knees) with CPT. Patients were evaluated as "excellent" (none or minimal pain during heavy exertion, full return to sport, normal range of motion, none or mild inferior patellar pole tenderness) or "poor" (moderate to severe pain on exertion, significant impairment or no return to sport, moderate to severe inferior patellar pole tenderness). Seventy percent of the involved knees demonstrated an "excellent" response to treatment while the remainder went on to surgery. Patients graded as 2 or 3 (according to size of ultrasonographic hypoechoic zones) failed conservative treatment more often and required surgery.

Duri & Aichroth[29] presented preliminary results following a 6-week conservative treatment program on 21 patients with CPT. The physiotherapy treatment involved stretching of the hip and knee musculature, closed chain exercises, vastus medialis strengthening, stretching of the lateral patellar retinaculum when tight, reduction of knee swelling, and advice regarding the necessity of warm-up exercises before games, crosstraining, and gradual resumption of the definitive sports. Patient outcomes following subjective, objective, and functional testing were graded as "normal", "nearly normal", "abnormal", and "very abnormal". Six patients completed the treatment without steroid injections and were classified as follows: "normal" (n=1), "nearly normal" (n=4), and "abnormal" (n=1). In those patients having steroid injection as well as the above described treatment, 11% demonstrated "excellent" results and 41% demonstrated "good" results. Twenty-five percent of the patients required surgery.

Wiley et al.[116] prospectively monitored 23 patients with patellar tendinitis based on an ultrasound imaging classification (see above section). Conservative treatment utilizing physical therapy and eccentric squat exercises

were applied (34 symptomatic knees). Six subjects became painfree during the study , taking 16-18 months to recover. At the latest assessment, individuals still presenting with patellar tendinitis tendons exhibited an average total time with symptoms of 21 (grade 1), 30 (grade 2), and 28 (grade 3) months.

Several of the studies[30,108,118] indicated that CPT patients graded as 1st or 2nd phase (according to Blazina et al.[14]) tended to recover with conservative treatment whereas those graded as 3rd phase tend to require surgical treatment. The recovery period during conservative treatment of CPT patients may be longer than traditionally expected, according to the results of Wiley et al..[116] Several studies have not clearly outlined the conservative treatment implemented.[29,60,109,118] In order to determine the effectiveness of conservative treatment, future studies need to clearly define the implementation and progression of treatment.

Modalities for Treating Chronic Tendinitis

A variety of modalities have been proposed for treating tendon injuries including deep transverse frictions, ultrasound, laser, high voltage pulsed galvanic stimulation, ice, medications, bracing, and immobilization.

Cyriax[133] recommended the use of transverse frictions to release collagenous adhesions to adjacent tissues and to facilitate an increase in local blood flow. These adhesions may be a source of continued irritation and inflammation.[21]

The role of electrical modalities, although widespread, remains largely speculative in the treatment of chronic soft tissue injuries.[20] Hess et al.[2] advocated the use of ultrasound because the deep-heating effect may increase blood flow and reduce inflammation. However, it has been reported that ultrasound has no effect on inflammation.[134] The benefits of ultrasound may be that it increases collagen synthesis by fibroblasts, speeds wound healing, and

results in increased tensile strength in healing tendons.[135] Curwin[20] has suggested that there is little indication for prolonged use of ultrasound. Ultrasound may be combined with steroidal anti-inflammatory medications (phonophoresis) and has been reported to be clinically effective.[21]

The application of laser has been shown to increase fibroblast synthesis of GAGs and collagen, speed superficial wound healing, and decrease inflammation.[136] However, Curwin[20] reported there is no clinical or scientific evidence to support its use for treating deeper tissues like tendons.

High voltage pulsed galvanic stimulation has been reported to be effective in producing heat within tissues.[4,137] The effects of heat include increased extensibility of collagen in connective tissue, reduced joint stiffness, and reduction in muscle spasm and pain.[4] Electrical stimulation has shown a positive influence in acute tendon healing[138], however, its application in terms of dosage remain unclear.[20]

Ice has been shown to decrease the activity of inflammatory mediators and overall metabolic rate of injured tissue.[128] Curwin and Stanish[1] reported that ice provides analgesia and may offset inflammation induced during the exercise component of rehabilitation of chronic tendinitis.

Corticosteriods are considered the most potent anti-inflammatory drugs and are suggested to be appropriate for treating peritendinous structures (i.e. paratenon) if inflammation is marked or prolonged.[4,21,46] Corticosteroid injection into the tendon substance itself has been advised against because it predisposes the tendon to rupture.[20,129,139,] The adverse effects of corticosteroid injection include reduced collagen synthesis, mechanical disruption by the needle, an irritant effect from the solvent carrying the drug, and inhibition of fibroblast and macrophage activity.[20]

Non-steroidal anti-inflammatory drugs (NSAIDs) inhibit cycloxygenase and block the conversion of arachadonic acid to prostaglandins.[46] It is thought NSAIDs limit inflammation and allow faster return to activity.[46] It has been reported that there is an increase in total collagen content and ultimate strength of tendon following administration of NSAIDs.[140] Side-effects of NSAID use include gastrointestinal tract upset and potential kidney injury.[46] NSAIDs do not inhibit fibroblast or macrophage activity as seen with corticosteroid use.[20]

Although the mechanism is unclear, anabolic steroid use may be a predisposing factor to tendon injury and it is advised to be alert to potential users when treating tendon injury patients.[20]

Temporary immobilization may be of value in some situations, however, the negative effects of prolonged immobilization should be avoided.[121] Bracing has been used for various forms of overuse tendon injury (i.e. patellar tendon strap).[121]

Surgery

It has been suggested there is little place for surgery in the treatment of chronic tendinitis unless the tendon ruptures and the ends need to be approximated.[20] Conversely, several authors[46,121] have suggested surgery is indicated if prolonged conservative treatment is ineffective. The proposed rationale for surgery is the removal of scar tissue and to provoke the inflammation-repair response and revascularization.[46,129] Surgical intervention may consist of an incision into the tendon sheath, followed by several longitudinal incisions that split the tendon at the site of symptoms.[46] Following surgical intervention there is a period of prolonged immobilization and rehabilitation and it has been suggested that it is difficult to ascertain if the benefits are from the surgery or the prolonged recovery period after surgery.[20,129] In patients with CPT, if symptoms are not improving or relieved

after a 3-6 month course of conservative treatment and predisposing factors have been eliminated, surgical treatment may be considered.[121]

Prevention

Prevention is the key to all overuse injuries as almost all contributing factors can be identified and prevented.[4,121] When symptoms are noted it is crucial to evaluate all potential contributing factors and intervene as soon as possible.[4] These intrinsic and extrinsic factors have been previously addressed in the section on the etiology of overuse tendinitis.

CONCLUSION

Chronic tendinitis presents a clinical dilemma to many physical therapists, and the best treatment approach remains to be clearly elucidated. It is apparent that significantly different pathological processes occur in acute macrotraumatic tendon injuries versus chronic microtraumatic tendon injuries.[25] Additionally the pathophysiological mechanisms underlying overuse tendon injury remain to be interpreted. As a result, the treatment of chronic tendon injury may require a significantly different treatment approach than that for acute tendon injury. Optimal treatment should result in the resolution and prevention of symptoms. This requires a comprehensive treatment approach based upon a thorough history, complete physical examination, knowledge of tendon healing, and identification of the etiological factors of chronic tendon injury. Future studies should be directed at determining the pathophysiology of overuse tendon injury, optimal diagnostic procedures, and effective conservative treatment approaches.

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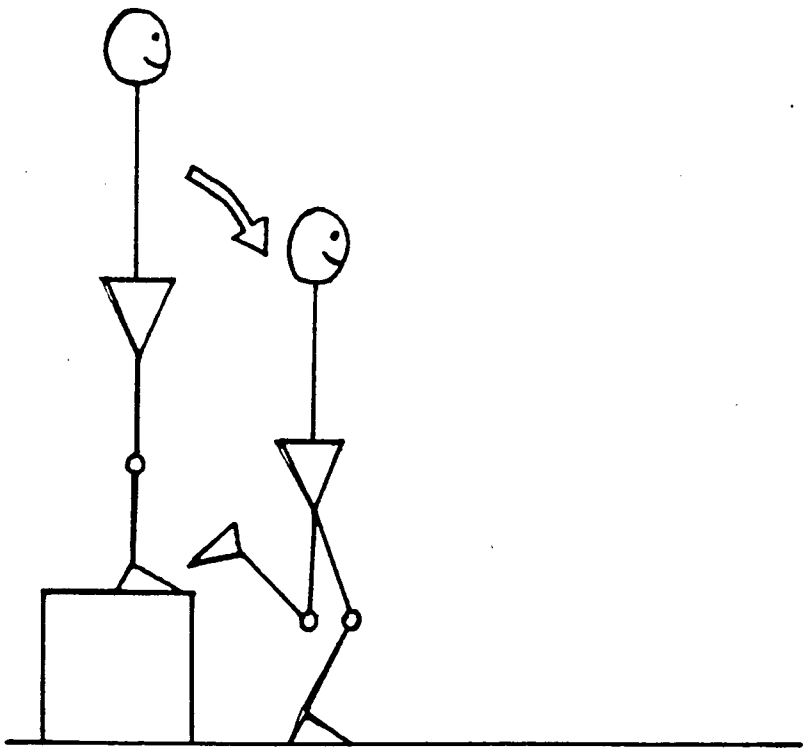
Appendix 2
Provocative Test



Step 1: Stand on top of box (12 inch height) on affected side foot.

Step 2: Hop from box to floor and land on affected side foot only.

Repeat steps 1 & 2 twice more, then perform DDS.

Appendix 3
Descriptor Differential Scale

PART A - Amount of Sensation

Rate your sensation in relation to each word with a check mark.

Strong

Extremely Intense

Very Weak

Barely Strong

Very Intense

Moderate

Slightly Intense

Weak

Faint

Intense

Very Mild

Mild

PART B - Amount of Unpleasantness

Rate your unpleasantness in relation to each word with a check mark.

Very Annoying

Slightly Distressing

Annoying

Very Unpleasant

Slightly Annoying

Very Distressing

Intolerable

Slightly Unpleasant

Very Intolerable

Distressing

Slightly Intolerable

Unpleasant

Appendix 4
Knee Function VAS

KNEE DISORDERS SUBJECTIVE HISTORY

NAME
CHART _____ DATE _____ SIDE L ☐ R ☐

For each question, place a mark in the box which you think describes your knee relative to the two extremes. Please complete both sides of this form

1. How often does your knee hurt?
   never ☐☐☐☐☐☐☐☐☐☐ daily, even at rest
2. How bad is the pain at its worst?
   none ☐☐☐☐☐☐☐☐☐☐ severe, requiring pain pills every few hours
3. Do you have swelling in your knee?
   never ☐☐☐☐☐☐☐☐☐☐ daily, even at rest
4. Does your knee give way or buckle?
   never ☐☐☐☐☐☐☐☐☐☐ I must guard my knee to prevent giving way even with normal everyday activity
5. Does your knee lock up so you are unable to straighten it?
   never ☐☐☐☐☐☐☐☐☐☐ I must guard my knee to prevent locking even with normal everyday activity
6. Does your knee catch or hang up when moving?
   never ☐☐☐☐☐☐☐☐☐☐ I must guard my knee to prevent catching even with normal everyday activity
7. Is your knee stiff?
   never ☐☐☐☐☐☐☐☐☐☐ I can barely move my knee because of stiffness
8. Are you able to walk on level ground?
   no problem ☐☐☐☐☐☐☐☐☐☐ unable
9. Are you able to walk on rough ground?
   no problem ☐☐☐☐☐☐☐☐☐☐ unable
10. Do you need crutches, a cane, or a walker to walk?
    never ☐☐☐☐☐☐☐☐☐☐ always
11. Do you feel grinding when your knee moves?
    never ☐☐☐☐☐☐☐☐☐☐ always
12. Do you have problems twisting or pivoting on your injured knee?
    none ☐☐☐☐☐☐☐☐☐☐ unable
13. Do you have problems carrying heavy objects because of your knee?
    none ☐☐☐☐☐☐☐☐☐☐ unable ☐ not attempted
14. Do you have problems climbing stairs?
    none ☐☐☐☐☐☐☐☐☐☐ unable ☐ not attempted
15. Do you have problems going down stairs?
    none ☐☐☐☐☐☐☐☐☐☐ unable ☐ not attempted
16. Do you have problems running?
    none ☐☐☐☐☐☐☐☐☐☐ unable ☐ not attempted
17. Do you have problems decelerating (slowing down) after running or jogging?
    none ☐☐☐☐☐☐☐☐☐☐ unable ☐ not attempted
18. Do you have problems cutting (changing directions while running by pivoting on affected knee)?
    none ☐☐☐☐☐☐☐☐☐☐ unable ☐ not attempted
19. Do you have problems jumping?
    none ☐☐☐☐☐☐☐☐☐☐ unable ☐ not attempted
20. Do you have problems in taking part in competitive sports?
    none ☐☐☐☐☐☐☐☐☐☐ unable ☐ not attempted
21. Do you have night pain?
    none ☐☐☐☐☐☐☐☐☐☐ strong
22. Do you have problems kneeling?
    no problem ☐☐☐☐☐☐☐☐☐☐ unable ☐ not attempted
23. Do you have problems squatting?
    no problem ☐☐☐☐☐☐☐☐☐☐ unable ☐ not attempted
24. Do you have problems getting in and out of a car?
    no problem ☐☐☐☐☐☐☐☐☐☐ unable
25. Does your knee ache while you are sitting?
    never ☐☐☐☐☐☐☐☐☐☐ always
26. Do you have problems getting in and out of a chair?
    no problem ☐☐☐☐☐☐☐☐☐☐ unable
27. Do you have stiffness or discomfort when you first start to walk?
    no problem ☐☐☐☐☐☐☐☐☐☐ always
28. Do you have problems turning over in bed?
    no problem ☐☐☐☐☐☐☐☐☐☐ unable

Appendix 5

Daily Acitivity Log

Name:_____
Date:_____

Instructions: Please estimate the time you spent (in minutes) performing any of the following activities during the day.

Activities: Duration (in minutes)

Walking.....
Jogging/Running.....
Bicycling.....
Stairs.....
Strength training (involving the knee).....
Swimming.....
Home physical activities (i.e. gardening).....
Work physical activities (i.e. labourer).....
Other (i.e. volleyball, basketball, squash, skating, etc.)
.....
.....
.....
.....

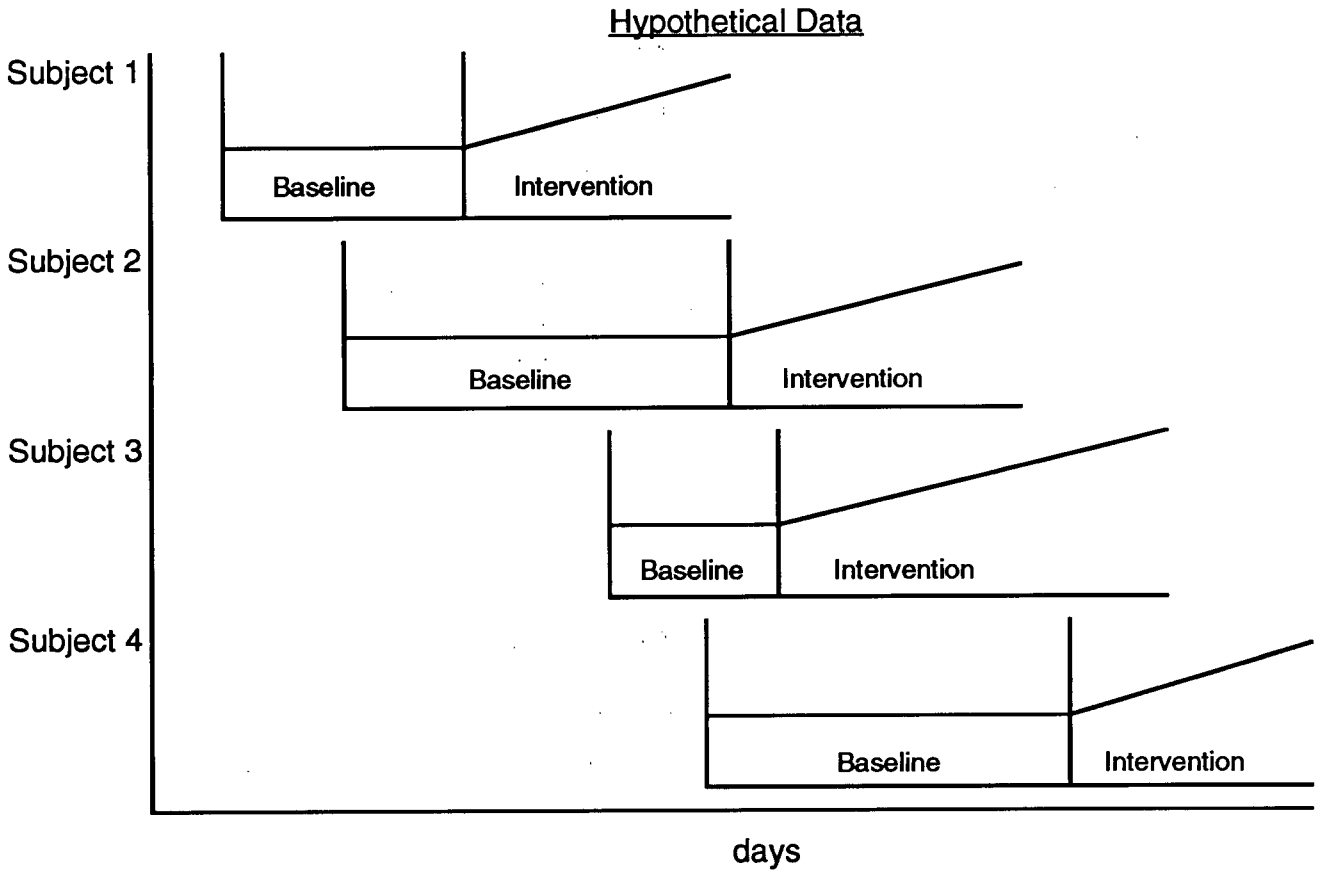
TOTAL=_____

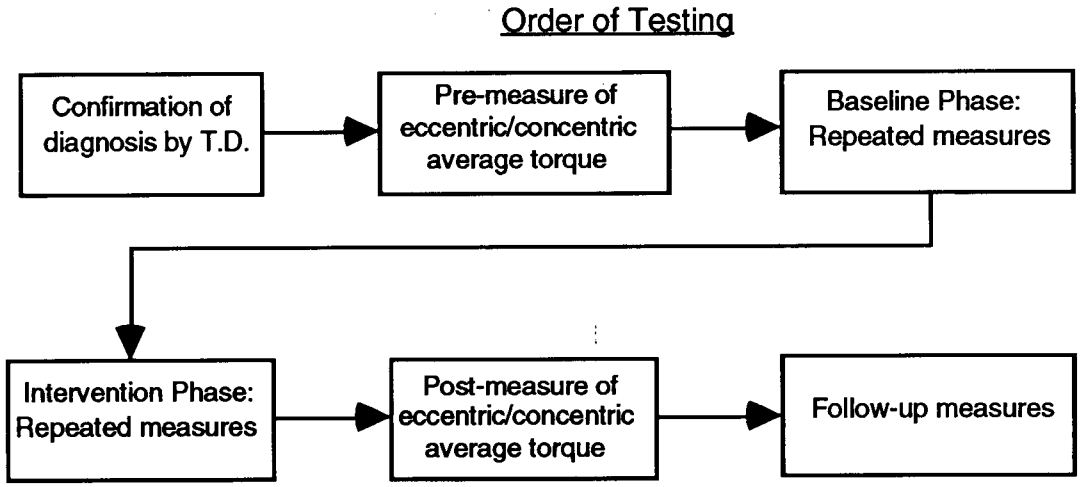
Eccentric Exercise Program

Instructions: Please fill in the blanks or circle the correct answer for the following questions.

Time of day you performed the program:_____
Speed of movement (slow / medium / fast)
Resistance (hand weights) (yes / no). If yes, how much weight? _____
Performed 3 sets of 10 (yes / no)
Performed stretching before and after squats (yes / no)
Iced knee following exercise (yes / no). If yes, how long?_____
Comments:_____
_____
_____

Appendix 6
Oultine of Study Design





Appendix 7
Education Session - Description of Study

Topics:
1) Provide definitions and examples of concentric, eccentric, and isometric muscle actions.

2) Review model of the knee in terms of structure and function. Explain pathology of patellar tendinitis.

3) Explain study design:
- Baseline phase compared to an Intervention (treatment) phase.
- Subject randomly assigned to a pre-determined baseline length (11-32 days).
- Measures to be performed:
    a) Pre-test of eccentric / concentric average torque of quadriceps femoris and hamstrings. Show Kin Com to subject and explain procedure. Inform patient that affected knee will be iced after testing to reduce potential aggravation of the tendinitis.
    b) Repeated Measures (performed throughout baseline and intervention phases):
        i) Self-rating of knee pain using computerized scale. Subject shown a copy of the DDS. Explain knee pain is relative to a provocative test. Demonstrate provocative test to subject. Subject may try test if desired.
        ii) Self-rating of knee function using VAS. Subject shown a copy of the knee function VAS.
        iii) Daily activity log format shown to subject. Provided an example.
        iv) Peak isometric torque test of quadriceps and hamstrings using the Kin Com. Explain procedure. Inform subject that the affected knee will be iced after testing to reduce potential aggravation of the tendinitis.
    c) Intervention: Provide brief description of EEP and its 5 components. Performed independently on a daily basis for 6 consecutive weeks. Inform subject they will receive a detailed explanation prior to the intervention phase.
    d) Post-test of eccentric / concentric average torque of quadriceps femoris and hamstrings (see section a)

4) Continuation of Physical Activity: Inform patients that it is not necessary to cease participation in activity unless they are unable to perform the activity satisfactorily or their symptoms are becoming worse.

5) Provide opportunity for review of material and time for questions.

(Estimated time: 30 minutes)

Appendix 8
Education Session - EEP

Eccentric Exercise Program Guidelines

The eccentric exercise program is to be performed independently on a daily basis for six consecutive weeks. Try to perform the program at a regular time each day. Avoid participation in sports or other potentially aggravating physical activities immediately prior or after the program. Please note you may experience a temporary increase in our knee symptoms during the first two weeks of the program.

Components of the Eccentric Exercise Program:

Warmup:

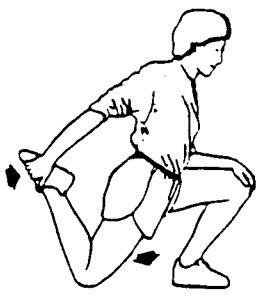
Perform approximately 5 minutes of an activity that does not stress the patellar tendon. Suggested activities include push-ups or sit-ups. The warm-up should elicit mild sweating.

Stretching:

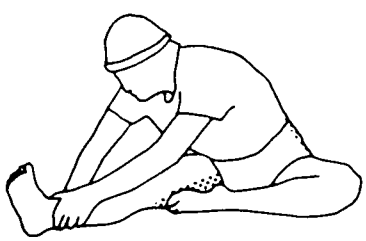
Perform each of the following stretches three times. Hold each stretch thirty (30) seconds.

1) Quadriceps Stretch - maintain tight abdominals so that your lower back does not arch.

2) Rectus Femoris Stretch - maintain tight abdominals so that your lower back does not arch. Do not lean forward with your trunk.

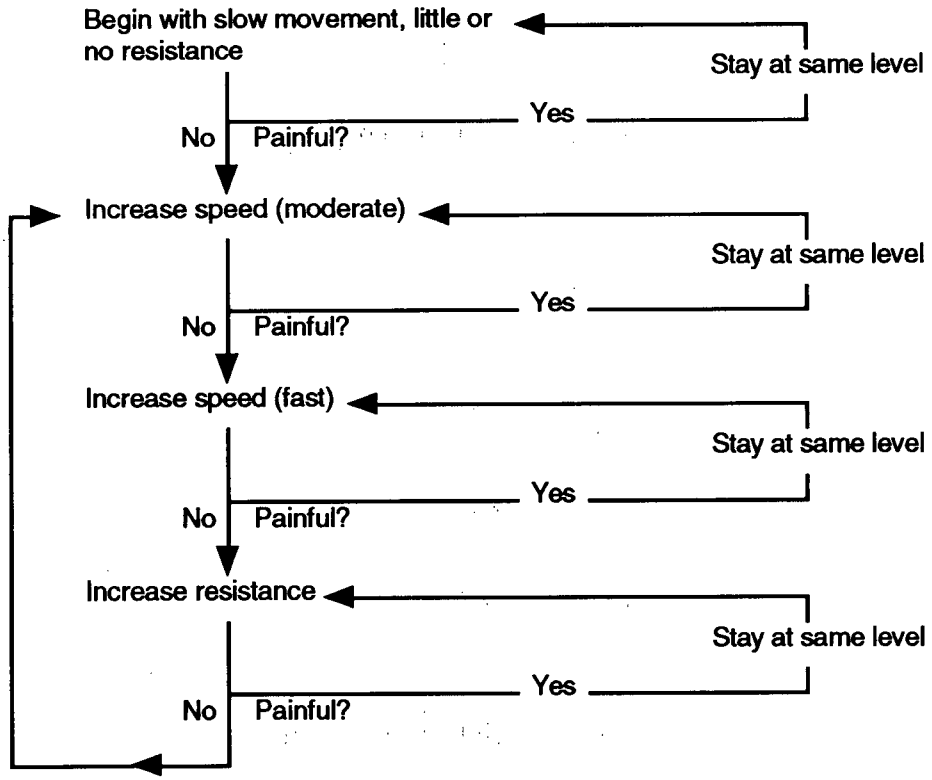


3) Hamstring stretch - keep your back straight so the movement comes from your hip only.



Eccentric Squats:

Three sets of 10 repetitions of the "drop" squat are performed. During the "drop" squat exercise keep your feet shoulder width apart, your trunk upright, and when looking down you should see the centre of your knee cap is over the second toe (performing the exercise in front of a mirror may help you to maintain the correct alignment). The exercise is progressed according to your knee pain during the exercise. You are performing the exercise at the correct intensity if knee pain is experienced between the 20th and 30th repetition. Once it becomes painfree to perform 3 sets of 10 repetitions you may progress to the next level. Hand-weights will provided once you reach that level in program. The progression of weight held in the hands is 10% of body weight (example: a 60 kg person would use 6 kg - 3 kg held in each hand). An outline of the progression of exercise is given below:



Stretching:

The same stretches are performed following the "drop" squats.

Application of Cold:

At the end of the program apply cold to the affected knee for 15 minutes. Try to use a consistent cold source (ie. gel pack, bag of ice or frozen vegetables).

Appendix 9

Physical Examination Procedure

Subjective:

Current History:

Symptoms: pain (location, type, constant / intermittent)?

swelling (location)?

locking, grinding, catching, or giving way of knee?

symptoms worsening, improving, staying the same?

onset of symptoms (insidious, trauma)?

aggravating or relieving factors?

functional limitations (walking, running, jumping, stairs, sitting)?

Activities: current level of physical activity? type of activity?

level of participation (competitive, recreational)?

desired level of activity?

footwear / orthotics?

Medications: use of medications for this condition?

General Health: other conditions? age?

Occupation: type of activity?

Past Medical History:

Other neurological / orthopaedic conditions?

Prior knee injuries? Treatment?

Objective:

Observation:

Standing: (equal weight bearing, genu varus / valgus / recurvatum, patella position, swelling, muscle wasting, pes planus / cavus, pronation or supination of foot)

Lumbar Scanning Examination:

Functional Tests:

Bilateral squat, single-leg squat, bilateral hop, single-leg hop, bilateral calf-raise, single-leg calf-raise, gait, running.

Observation of knee alignment during single and double leg squat / hop.

Active Range of Motion:

Knee: flexion, extension, internal rotation, external rotation.

Passive Range of Motion:

Knee: flexion, extension, internal rotation, external rotation.

Resisted Isometric Testing:

Knee flexion (hamstrings), knee extension (quadriceps femoris), rapid eccentric load applied to extended knee in seated position if necessary

Ligament Stability:

Collateral ligaments of knee.

Cruciate ligaments of knee (Lachman's test, anterior and posterior drawer).

Special Tests:

McMurray meniscus test, Hughston plica test, Wipe test for knee effusion, Apprehension test for patellar dislocation, test for femoral anteversion / retroversion

Joint Play:

Anterior / posterior glide of tibia on femur, medial / lateral / superior / inferior glides of patella, anterolateral and posteromedial glides of fibular head on tibia.

Palpation:

Patella (apex, base, medial and lateral retro-patellar surfaces), patellar ligaments, distal iliotibial band, popliteal fossa, hamstring tendons, and quadriceps muscles.

Measures of Lower Extremity Alignment:

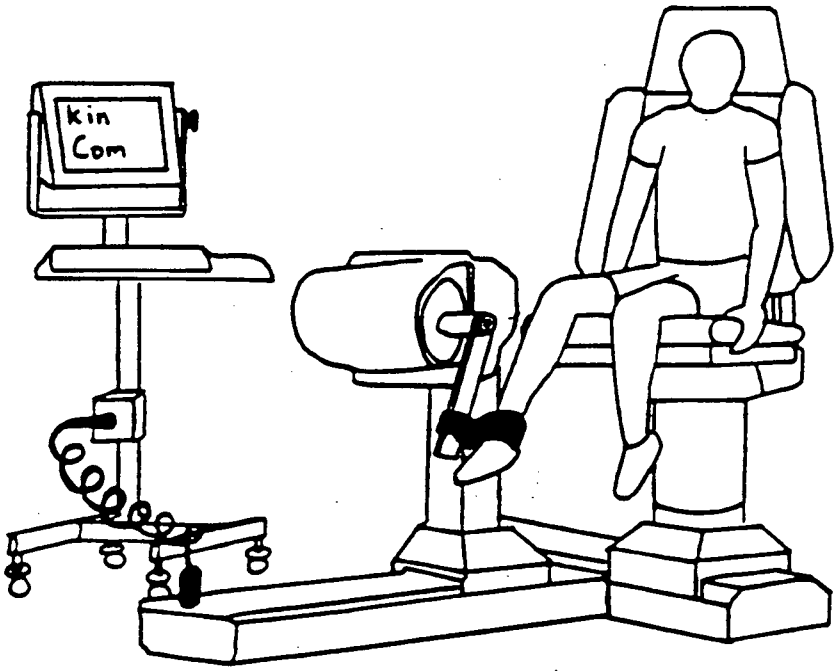
Q-angle

Leg-length

Measures of Height and Weight: using equipment at AMSMC.

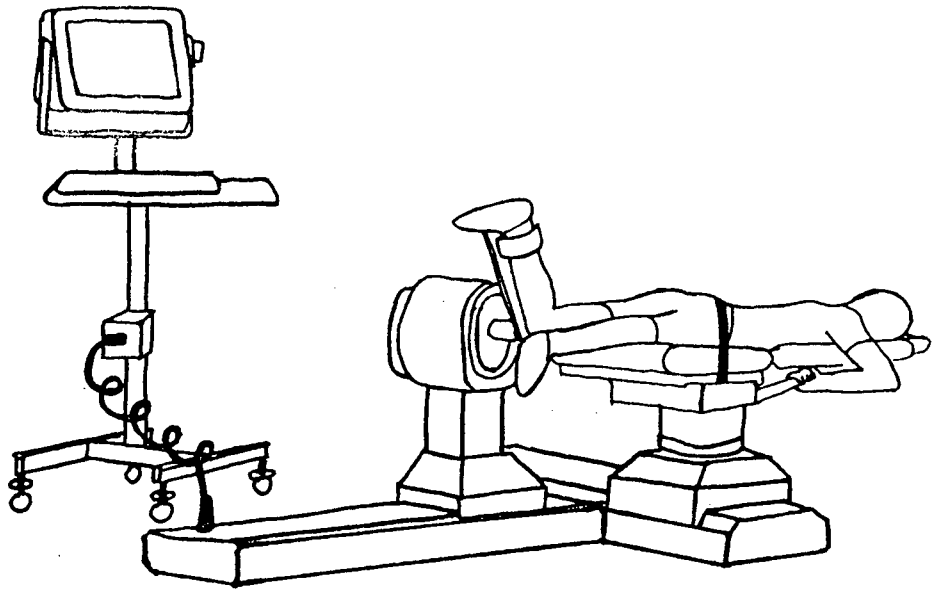
Appendix 10

Kin Com:isokinetic quads/hams and isometric quadriceps test position



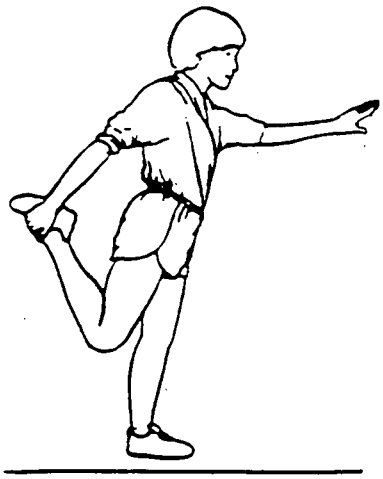
Appendix 11

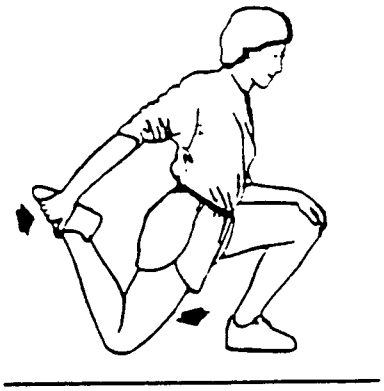
Kin Com: isometric hamstring test position



Appendix 12

Stretching positions for quadriceps femoris and hamstring muscles





Appendix 13

Pilot Study: Isometric Hamstring Testing

Purpose

The purpose of this pilot study was to determine the test-retest reliability of isometric hamstring strength testing in a prone position using the Kin Com isokinetic dynamometer (Chattanooga Group Inc., TN). The prone position has been recommended for hamstring testing due to the stabilization of the subject's thigh[1]. Trudelle-Jackson and colleagues[2] have examined the reliability of isometric hamstring testing in the prone position on the Kin Com isokinetic dynamometer. They reported an r value of 0.94, however, testing was done at 90 degrees of knee flexion. Walmsley & Jang[3] reported that the isometric torque generating capability of the of the hamstrings is greatest at zero degrees of knee extension when the hip is at zero degrees of flexion. In this pilot study, peak isometric hamstring force was tested at 30 degrees of knee flexion with the hip in zero degrees of flexion. Thirty degrees of knee flexion was selected in order to avoid full extension of the knee, and to avoid the potential of active insufficiency of the hamstring muscles at 90 degrees of knee flexion.

Methods

Subjects

Fourteen subjects (7 female, 7 male) between the ages of 19 and 33 years (mean age=24.6 years) participated in this study. All subjects were healthy university students with no prior knee injuries (except one subject; SF, who had an anterior cruciate deficient right knee).

Table 1. Subject Characteristics

Subject	Age (years)	Gender
S.F.	25	male
S.S.	33	female
R.S.	27	male
M.S.	31	male
L.M.	28	female
S.C.	20	female
T.L.	19	female
K.G.	20	female
L.H.	25	female
L.H.	21	female
D.M.	22	male
G.N.	24	male
D.B.	27	male
T.R.	22	male

Setting

The study was conducted at the AMSMC, Vancouver, British Columbia in the exercise physiology lab and physical therapy clinic. The study was conducted by the co-investigator, Tyler Dumont, and was supervised by Dr. Donna MacIntyre.

Instrumentation

All testing was conducted on the Kin Com Isokinetic Dynamometer (model 125AP)(Chattanooga Group Inc., TN).

Procedures

Study Design

A test-retest design was implemented where all testing was repeated within 28 hours of the initial test.

Test Administration

All testing procedures were identical for all subjects. The order of testing was as follows: left hamstring testing followed by right hamstring testing on day 1, left hamstring testing followed by right hamstring testing on day 2.

All subjects were tested in a prone position (Appendix 10) and were stabilized via a belt across the pelvis. The subjects were also instructed to hold on to the back support of the Kin Com during testing. The axis of the Kin Com lever arm was aligned with the axis of the knee (lateral tibiofemoral joint line). Lever arm length was set as 75% of the fibula length for each subject (measured from the superior aspect of head of fibula to inferior aspect of lateral malleolus). For each test the subject performed 3 warmup isometric contractions of 5 seconds duration each. The subjects were instructed to give an effort of approximately 50% of maximum for each warmup contraction. The warmup contractions were performed at 25-30 degrees of knee flexion (25-30 degrees from horizontal). The subject then performed 3 maximal isometric contractions of 5 seconds duration each. The subjects were instructed to gradually increase their effort to maximum within the first 2-3 seconds and then sustain the effort for the last 2-3 seconds. The maximal contractions were performed at 30 degrees of knee flexion (30 degrees from horizontal). A 30 second rest was given between all warmup and maximal contractions. The gravity compensation feature of the kin Com was applied to all tests.

Data Collection

During peak isometric force measures, all data was saved on the Kin Com computer system. A backup copy of all data (floppy disk) was stored in a locked filing cabinet at the SRS.

Data Management and Analysis

The data collected during peak isometric force testing were analyzed by the Kin Com computer system and an average peak force (N) was calculated from the 3 maximal efforts for the hamstring muscles. A Pearson product moment coefficient of correlation was calculated for the group's left and right hamstring test-retest measures.

Results

The r values for left and right hamstring testing were 0.986 and 0.927 respectively. Richman and Makides[4] have suggested a scheme to rank the correlation values; 0.80 to 1.0 is "very reliable", 0.60 to 0.79 is "moderately reliable", and 0.59 and less has "questionable reliability". The results of this pilot study suggest the protocol used was very reliable.

Table 2. Peak Force (N) and r values.

Subject	Left: Day 1	Left: Day 2	Right: Day 1	Right: Day 2
S.F.	199	198.3	189.3	216
S.S.	194.7	192.7	203.7	208.3
R.S.	269.7	260.7	187.7	246.3
M.S.	197	205	249.3	252.7
L.M.	161.7	152	164	150.7
S.C.	262	254.7	217.7	230
T.L.	124	111.7	147.7	101.7
K.G.	124.7	136.7	95.7	126
L.H.	133.7	131.7	166.3	164
L.H.	150.7	137.7	181	166
D.M.	290	313.7	308.3	276.7
G.N.	341.3	369.7	331.3	338.7
D.B.	132.3	147.3	151	169
T.R.	319	306.7	323.3	309.7

r= 0.986 r=0.927

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Appendix 14

Pilot Study: Alignment Measures

Purpose

The purpose of this pilot study was to determine the intra-tester reliability for measurements of Q-angle, true leg length (TLL), and sub-talar joint neutral (STJN). Q-angle was defined as the angle between the line of pull of the quadriceps muscle and the longitudinal axis of the patellar tendon[1]. Tomisch et al.[2] have reported an intra-tester intra-class correlation coefficient (ICC) of 0.63 for Q-angle measurements. TLL was defined as the distance between the anterior superior iliac spine (ASIS) and the medial malleolus by Hoyle et al.[3]. Because this measurement value may be influenced by muscle wasting or obesity, measurement to the lateral malleolus is less likely to be affected by muscle bulk[4]. For the purposes of this pilot study TLL was defined as the distance between the ASIS and most inferior aspect of the lateral malleolus. Hoyle et al.[3] have reported intra-tester r values ranging from 0.993 to 0.997 for measures of TLL (ASIS to medial malleolus). STJN was defined as the position in which the foot is neither pronated or supinated[5]. Elveru et al.[6] have reported an ICC value of 0.77 for intra-tester measures of STJN while Smith-Oricchio[7] reported an ICC value of 0.60 for intra-tester measures of STJN.

Methods

Subjects

Ten healthy university students (5 male, 5 female) with a mean age of 22.5 years (range: 19-27 years) participated in this study.

Table. 1 Subject Characteristics

Subject	Age (years)	Gender
S.C.	20	female
T.L.	19	female
K.G.	20	female
L.H.	25	female
L.H.	21	female
S.F.	25	male
D.M.	22	male
G.N.	24	male
D.B.	27	male
T.R.	22	male

Setting

The study was conducted at the AMSMC, Vancouver, British Columbia in the exercise physiology lab and physical therapy clinic. The study was conducted by the co-investigator, Tyler Dumont, and was supervised by Dr. Donna MacIntyre.

Instrumentation

A standard tape measure (Invacare, Elyria, OH) was used for all measures of TLL. A 12 inch goniometer (Fred Sammons Inc., Brookfield, IL) was used for all measures of Q-angle. A 6 inch goniometer (MJ Therapy and Associates Inc., Waukesha, WI) was used for all measures of STJN.

Procedures

Study Design

A test-retest design was implemented where all testing was repeated within 28 hours of the initial test.

Test Administration

All testing procedures were identical for all subjects. The order of testing TLL, Q-angle, and STJN was conducted in a random manner. The following measures were collected: left and right TLL, left and right Q-angle, left and right STJN.

TLL: All subjects wore shorts and were positioned supine with a pillow supporting the head and neck. Each subject's legs were separated at the midline point of each heel by the distance measured between the subject's anterior superior iliac spines (ASIS). During measurement, the investigator palpated the ASIS and held one end of the tape measure with one hand while the other hand positioned the tape measure at the most distal aspect of the lateral malleolus. The measurement was taken to the nearest millimeter and each leg was measured in alternate order three times.

Q-angle: All subjects wore shorts and were positioned supine with a pillow supporting the head and neck. The feet were positioned vertically using sand-bag weights to minimize femoral rotation during testing. A pen mark was placed on the skin overlying the centre of the patella which was determined via palpation of the patella and visual estimation of the centre position. The goniometer axis was aligned over the centre position mark. The stationary arm of the goniometer was aligned with the ASIS which was unmarked, but exposed and palpated by the investigator. The mobile arm of the goniometer was aligned with the most prominent palpable point of the tibial tuberosity. Q-angle was measured to the nearest degree three times on each leg in random order.

STJN: All subjects wore shorts and were positioned in prone with the foot and ankle of the side to be measured hanging 15-20 cm over the end of the plinth. The opposite lower extremity was placed into hip flexion, external rotation, and abduction with the knee flexed and the plantar surface of the foot

resting against the medial aspect of the test-side knee. This was done to ensure that the subjects remained in a static position during measurement. The midline of the posterior aspect of the lower one third of the test leg was visually estimated ignoring the Achilles tendon, and a narrow line was drawn to represent the midline using a straightedge. The midline of the posterior aspect of the calcaneus was visually estimated ignoring any exostoses, and a narrow line was drawn to represent the midline using a straightedge. To place the the foot in the STJN position the examiner placed the thumb of the medial hand under the medial aspect of the head of the talus and the index finger over the lateral aspect of the head of the talus. The thumb of the examiner's other hand was placed on the plantar surface of the fourth and fifth metatarsal heads, and the examiner's index and middle fingers were placed on the dorsal aspect of the fourth and fifth metatarsal heads. To palpate the medial head of the talus the foot was maximally pronated. To palpate the lateral head of the talus the foot was maximally supinated. The STJN position was considered the position where the examiner was able to palpate the head of the talus equally, both medially and laterally. The STJN position was maintained with the examiner's lateral hand and the goniometer was aligned with the examiner's medial hand to measure the angle between the longitudinal midlines of the calcaneus and lower leg. The goniometer was read to the nearest degree and the measurement was repeated three times (random order of foot testing).

Data Collection

All measurement data were collected, written on paper, and entered into a computerized spreadsheet program. A backup copy of all data (floppy disk) was stored in a locked filing cabinet at the SRS.

Data Management and Analysis

A mean score was calculated for the three trials taken of each measurement (Q-angle, TLL, STJN). A Pearson product moment coefficient of correlation was calculated to determine intra-tester reliability for each of the alignment measures.

Results

The TLL measurements produced r values of 0.998 for both the left and right sides and were considered "very reliable" according to the guidelines of Richman & Makrides[8]. The Q-angle measurements resulted in r values of 0.873 for the left side and 0.825 for the right side and were considered "very reliable" according to the guidelines of Richman & Makrides[8]. The STJN measurements resulted in r values of 0.539 for the left side and 0.429 for the right side, thus the reliability was considered "questionable". The results of this pilot study determined that the primary investigator would use only measurements of TLL and Q-angle to document lower extremity alignment in the subjects involved in the thesis research project.

Table 2. TLL (cm) and r values.

Subject	Left: Day 1	Left: Day 2	Right: Day 1	Right: Day 2
S.C.	88.9	88.7	89.1	88.8
T.L.	83.8	84	83.7	83.9
K.G.	89.3	88.9	89.5	89
L.H.	88.9	88.8	89.2	89.2
L.H.	83.6	83.4	83.5	83.2
S.F.	93.9	94.1	94.2	93.8
D.M.	88	88.1	88.5	88.6
G.N.	88.7	89	88.5	88.3
D.B.	91.9	92.1	92	92.3
T.R.	97.8	97.6	97.7	97.7
		r=0.998		r=0.998

Table 3. Q-angle (degrees) and r values.

Subject	Left: Day 1	Left: Day 2	Right: Day 1	Right: Day 2
S.C.	17	17	16	16
T.L.	19	20	21	23
K.G.	14	14	15	16
L.H.	18	19	19	19
L.H.	14	13	17	13
S.F.	11	14	12	13
D.M.	11	14	13	14
G.N.	15	15	13	14
D.B.	16	17	15	16
T.R.	12	11	14	11
		r=0.873		r=0.825

Table 4. STJN (degrees) and r values

Subject	Left: Day 1	Left: Day 2	Right: Day 1	Right: Day 2
S.C.	4	4	10	6
T.L.	3	4	3	6
K.G.	0	2	4	2
L.H.	2	6	10	5
L.H.	7	5	6	8
S.F.	7	11	9	3
D.M.	3	2	2	4
G.N.	5	4	14	8
D.B.	8	6	3	5
T.R.	6	3	4	4
		r=0.539		r=0.429

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Appendix 15

Physical Examination of each subject

Subject #1

Date: April 4, 1997
Subjective Examination:
HPI:

In May 1996, subject #1 developed left anterior knee pain which occured on take-off in high jumping, run-ups in high jumping, while driving, and while using stairs. She also reported "noises" from the left knee. The onset was gradual (during training for high jumping May / June 1996). No locking or giving way of the left knee was reported. She took the months of July and August off due to the knee pain. In September training resumed. In January 1997 subject #1 sought physiotherapy treatment at the University of British Columbia. She was diagnosed with patellar tendinitis and treatment consisted of ultrasound, ice, and squatting exercises. She was also taking Ibuprofen (discontinued at the end of February 1997). Her high jump season ended in March 1997 (stopped all training). Her knee condition has fluctuated over the past 6-8 months but has plateaued the past 1-2 months. Current symptomology consists of the following: aching of the left knee at night and while sitting, left knee pain on stairs, left knee "noises" and "catching" sensations, and mild swelling on the medial aspect of the patellar tendon. She uses ice occasionally if particularly sore.

PMH:

No prior left knee injuries reported. Moderate right ankle sprain in 1994.

General Health:

Good, no other medical conditions, age 20 years, no orthotics.

Occupation:

University of British Columbia student (biology).

Activity:

Currently: playing tennis occasionally, walking on campus.
Usual training pattern: Fall - general conditioning (weights, speed / tempo work, weights, technical). Progresses into more weights and sprints (technical 3x/week, weights 3 days/week, sprints 1-2x/week). Competitive season begins January and continues until March. Summer season peaks in July.

Objective Examination:
Observation:

Normal lower extremity alignment. No gait faults noted. Slight wasting of left quadriceps muscles observed.

Lumbar Scanning exam:

Negative.

Functional Tests:

Bilateral squat: full ROM, left knee pain on rising.
Unilateral squat left: half-squat = left anterior knee pain.
Bilateral + unilateral calf raises: normal.

Active Range of Motion:

Normal active knee ROM bilaterally.

Passive Range of Motion:

Normal passive knee ROM bilaterally.

Resisted Isometric:

Right knee flexion and extension = strong + painfree
Left knee flexion = strong + painfree, Left knee extension at 0 , 45, and 90 degrees = strong + painful (weakness near full extension).

Ligament Stability:

Cruciate and collateral ligament tests of both knees negative.

Special Tests:

McMurray's test, Hughston plica test, Apprehension test, Patellar grind test, and Wipe test negative bilaterally. (noted crepitus left knee on patellar grind test)
Flexibility: Right straight leg raise = 100 degrees, left straight leg raise = 120 degrees. Slight decreased left prone knee bend ROM (tight quadriceps/rectus femoris). Ober's slightly tight on left. Janda Hip flexor position: slight lateral position of left leg (tight iliotibial band). Calf flexibility good bilaterally.

Joint Play:

Patellar glides normal bilaterally. Superior tibio-fibular joint glides normal bilaterally.

Palpation:

Left inferior patellar pole tender on palpation.

Measures:

Q-angle: Left = 16 degrees (16,15,16), Right = 17 degrees (19, 16,15)
Leg length: Left = 101.1 cm (101.0, 101.1, 101.1), Right = 99.9 cm (99.9, 100.0, 99.8)
Height: 177 cm
Weight: 63 kg

Diagnosis:

Left chronic patellar tendinitis.

Subject #2

Date: September 18, 1997

Subjective examination:
HPI: Subject #2 reported a 2 year history of right anterior knee pain with occasional left anterior knee pain. She reported her knee pain was worst during the summers and was related to playing ultimate. Current symptomatology consisted of right knee crepitus with squatting, right knee pain with downhill hiking, jogging > 20min, and jumping, and occasional right infrapatellar swelling. She reported having past physiotherapy treatment in August, 1996. She reported no night pain, locking, or paraesthesia.

PMH: Subject #2 reported that while in grade 11 she had a temporary bout of right jumper's knee.

General health: good, no other medical conditions, age 22 years, no orthotics.

Occupation: Obtained geography degree from University of British. Recently returned from travel in Australia and currently looking for work.

Activity: Subject #2 reported she likes to participate in the following activities: cycling, ice hockey (played at university level), volleyball, swimming, rollerblading, and ultimate.

Objective examination:
Observation:
Normal lower extremity alignment. No gait faults noted.

Lumbar scanning examination:
Negative.

Functional tests:
Bilateral squat: full ROM.
Unilateral half-squats: no pain.
Right hop: pain. Left hop: pain-free.
Bilateral + unilateral calf raises: normal.

Active Range of Motion:
Normal active knee ROM bilaterally.

Passive Range of Motion:
Normal passive knee ROM bilaterally.

Resisted Isometric:
Knee extension at 0,45, and 90 degrees of flexion strong and pain-free bilaterally. Knee flexion strong and pain-free bilaterally.

Ligament stress tests:

Cruciate and collateral ligament tests normal bilaterally.

Special tests:

McMurray's test, Hughston plica test, Apprehension test, Patellar grind test, and Wipe test negative bilaterally. Flexibility: straight leg raise 60 degrees bilaterally (tight hamstrings), restricted prone knee bend right >left (tight quadriceps / rectus femoris), positive Thomas test bilaterally (right=left), Ober's test negative bilaterally. Janda hip flexor position normal bilaterally. Calf flexibility normal bilaterally.

Joint Play:

Normal patellar glides bilaterally. Normal superior tibio-fibular joint glides bilaterally.

Palpation:

Right patellar pole tender on palpation.

Measures:

Q-angle: Left=11 degrees (10, 11, 12), Right=8 degrees (7, 9, 8)
Leg length: Left=93.8 cm (93.7, 93.8, 94.0), Right=94.1 cm (94.3, 94.1, 93.9)
Height: 176 cm
Weight: 74kg

Diagnosis:

Right chronic patellar tendinitis.

Subject #3

Date: September 25, 1997

Subjective Examination:
HPI: In October 1996, subject #3 developed left >right anterior knee pain and stiffness (located at the patellar poles), possibly related to participation in volleyball. He reported stiffness after prolonged sitting and some peri-patellar tendon swelling in both knees. By September 1997 the symptoms began to settle, however, pain and stiffness still resulted from running or volleyball. He reported no night pain, paraesthesia, clicking or locking of the knee. He tried using ice after activity but reported little benefit. He reported that he had not recieved any other form of treatment for his knees. He reported he was not taking any medications and had no prior investigations of his knees (i.e. x-rays).

PMH: He reported that during high school he suffered a mild hyperextension injury to the left knee and did not require any treatment (full recovery reported).

General Health: good, no other medical conditions, age 28 years, no orthotics.

Occupation: Landscaper: full-time seasonal work.

Activity: Subject #3 reported relative inactivity the past few months. He reported that he would go on occasional hikes. He also stated he was coaching volleyball 3 times per week and playing volleyball (BCVA A league) twice per week.

Objective Examination:
Observation:
Normal lower extremity alignment. No gait faults noted.

Lumbar scanning exam:
Negative.

Functional tests:
Bilateral squat: full ROM, no pain.
Left and right unilateral half-squats produced knee pain.
Left and right unilateral hops produced knee pain (right > left).

Active Range of Motion:
Normal active knee ROM bilaterally.

Passive Range of Motion:
Normal passive knee ROM bilaterally.

Resisted isometric:
Left and right knee extension and flexion strong and pain-free.

Ligament stress tests:

Cruciate and collateral ligament tests negative bilaterally.

Special tests:

McMurray's test, Hughston plica test, Apprehension test, and Wipe test negative bilaterally. Positive patellar compression test on left knee. Flexibility: left straight leg raise = 80 degrees, right straight leg raise = 90 degrees, decreased prone knee bend left > right (tight quadriceps / rectus femoris). Positive Ober's test right leg (tight iliotibial band). Janda hip flexor position normal bilaterally. Calf flexibility normal bilaterally.

Joint play:

Normal patellar glides bilaterally. Normal superior tibio-fibular joint glides bilaterally.

Palpation:

Lateral aspect left patella and inferior pole left patella tender on palpation. Medial and lateral aspect of right patella tender on palpation.

Measures:

Q-angle: Left =11 degrees (11, 11, 12), Right =12 degrees (12, 11, 12)
Leg length: Left =96.4 cm (96.2, 96.5, 96.3), Right =96.6cm (96.4, 96.7, 96.7)
Height: 185 cm
Weight: 78 kg

Diagnosis:

Left chronic patellar tendinitis, right patello-femoral pain syndrome.

Subject #4

Date: October 6, 1997.

Subjective Examination:
HPI: Subject #4 reported that approximately 2 year ago (Fall 1995) she developed bilateral (right > left) knee symptoms. The symptoms consisted of anterior knee pain, crepitus, and occasional mild swelling distal to the patellae. These symptoms were aggravated by running, strength training, and rowing. The symptoms worsened from October 1996 to February 1997. She underwent a short period of physiotherapy treatment (2 visits, minisquats tried but too painful) during March 1997. At this time symptoms have settled approximately 50% since February 1997. Current symptoms include general anterior knee pain bilaterally and some right medial knee pain. She reported some stiffness of the knees during the start of runs. Aggravating activities include: Russian lunges, quads stretching, running, and cycling. She reported no night pain, clicking or locking, or paraesthesia at the knees. She reported she was not taking any medications and had had no recent knee investigations (i.e. x-rays).

PMH: She reported that when she was in high school she had 3 right lateral patellar subluxations. She reported having sustained a left ankle inversion sprain in September, 1997 (full recovery).

General health: excellent, no other medical conditions, age 25 years, no orthotics.

Occupation: Graduate student in electrical engineering at the University of British Columbia.

Activity: International level rower (single sculler) - currently in off-season. Currently running 4-5x per week, daily commuting to University of British Columbia on bicycle, and weight lifting up to 4x per week.

Objective Examination:
Observation:
Mild genu varum noted bilaterally. Mild left pes planus. No gait faults noted. Reduced right vastus medialis muscle bulk observed.

Lumbar scanning examination:
Negative.

Functional tests:
Bilateral squat: full ROM, R knee uncomfortable at bottom of squat.
Left half squat: good. Right half squat: "unsteady" and "uncomfortable".
Single leg hops produced no symptoms.

Active Range of Motion:
Normal active knee ROM bilaterally.

Passive Knee Range of Motion:

Normal passive knee flexion bilaterally.
Mild knee hyper-extension bilaterally.

Resisted isometric:

Right knee extension at 45 degrees and 90 degrees of flexion produced knee pain. Left knee extension painfree at 0,45, and 90 degrees flexion.

Ligament stress tests:

Cruciate and collateral ligament tests negative bilaterally.

Special tests:

McMurray's test, Hughston plica test, Apprehension test, and Wipe test negative bilaterally. Positive patellar compression test bilaterally. Flexibility: straight leg raise 90 degrees bilaterally, prone knee bend within normal limits bilaterally, positive Ober's test right leg (tight iliotibial band), Janda hip flexor position normal bilaterally, calf flexibility normal bilaterally.

Joint play:

Normal patellar glides bilaterally. Normal superior tibio-fibular joint glides bilaterally.

Palpation:

Right patellar pole and left medial patellar retropatellar surface tender on palpation.

Measures:

Q-angle: Left=13 degrees (13,14,13), Right=13 degrees (14,13,13)
Leg length: Left=90.3 cm (90.1, 90.5, 90.4), Right= 90.0 cm (89.9, 90.0, 90.2)
Height: 170.7 cm
Weight: 65kg

Diagnosis:

Right chronic patellar tendinitis, left patello-femoral pain syndrome.

Appendix 16

Raw Data

Table 9. DDS Data and Knee Function VAS Data - Subject #1

Date	Day	Intensity	Unpleasantness	VAS Score
10-Apr	1	10.2	5.3	32.5
13-Apr	4	8	5.6	33.9
15-Apr	6	8	6.9	31.1
19-Apr	10	10.8	8.1	35.4
22-Apr	13	9.3	6.3	37.5
25-Apr	16	10.7	7.3	37.5
28-Apr	19	9.3	6.5	37.1
1-May	22	9.6	7.8	39.3
4-May	25	11.5	9.1	43.9
7-May	28	9.4	5.8	40.0
12-May	33	11	7	40.4
15-May	36	11.8	8.4	36.1
19-May	40	6.6	4.4	31.4
22-May	43	9.3	5.8	36.1
25-May	46	8.7	6.4	31.4
28-May	49	7.4	5.9	32.1
3-Jun	55	7.5	5.1	27.5
6-Jun	58	9.3	6.6	29.3
16-Jul	98	8	5.3	27.1

Note: baseline phase = days 1-13
intervention phase = days 16-58
follow-up = day 98

Table 10. Isometric Peak Force (N) - Subject #1

Date	Day	R Quads	L Quads	R Hams	L Hams
10-Apr	1	543	416.25	204	181
13-Apr	4	534	514	159	233
15-Apr	6	548.75	530	252.75	166.5
19-Apr	10	551.5	486	265.75	177.5
22-Apr	13	576.25	484.5	224.5	220.5
25-Apr	16	560.25	526.75	235.5	176.75
28-Apr	19	565	527.5	221.25	203.75
1-May	22	632.5	505.25	208	204.5
4-May	25	558.5	533.5	207.25	219.5
7-May	28	605.75	560.5	209.75	206
12-May	33	615.5	568	195.75	208.75
15-May	36	640.25	561.5	201.5	209
19-May	40	631.75	584	232.25	239.25
22-May	43	624.25	603	213.25	222
25-May	46	647.75	646.5	223.5	223.5
28-May	49	646.5	581.75	207	203
3-Jun	55	599.5	650.5	201.5	207
6-Jun	58	659.75	612.25	204.25	224.5
16-Jul	98	618.5	616.75	196	228.25

Note: baseline phase = days 1-13
intervention phase = days 16-58
follow-up = day 98

Table 11. Isokinetic Average Torque (Nm) - Subject #1

Date	Test	R quad conc	R quad ecc	L quad conc	L quad ecc	R ham conc	R ham ecc	L ham conc	L ham ecc
9-Apr	Pre	111	139	87	103	70	87	76	84
9-Jun	Post	139	165	117	140	89	106	68	74

Table 12. Daily Activity Levels - Subject #1

Date	Day	Time (min)	Date	Day	Time (min)
10-Apr	1	50	17-May	38	65
11-Apr	2	60	18-May	39	110
12-Apr	3	30	19-May	40	275
13-Apr	4	130	20-May	41	185
14-Apr	5	170	21-May	42	515
15-Apr	6	50	22-May	43	515
16-Apr	7	30	23-May	44	515
17-Apr	8	30	24-May	45	335
18-Apr	9	60	25-May	46	65
19-Apr	10	40	26-May	47	685
20-Apr	11	30	27-May	48	505
21-Apr	12	40	28-May	49	515
22-Apr	13	60	29-May	50	515
23-Apr	14	60	30-May	51	515
24-Apr	15	40	31-May	52	125
25-Apr	16	50	1-Jun	53	65
26-Apr	17	50	2-Jun	54	755
27-Apr	18	130	3-Jun	55	545
28-Apr	19	195	4-Jun	56	545
29-Apr	20	40	5-Jun	57	545
30-Apr	21	60			
1-May	22	40			
2-May	23	50			
3-May	24	70			
4-May	25	40			
5-May	26	60			
6-May	27	50			
7-May	28	120			
8-May	29	70			
9-May	30	185			
10-May	31	335			
11-May	32	50			
12-May	33	640			
13-May	34	475			
14-May	35	525			
15-May	36	515			
16 May	37	515			

Table 13. DDS and Knee Function VAS score - Subject #2

Date	Day	Intensity	Unpleasantness	VAS Score
25-Sep	1	9.3	8	32.5
28-Sep	4	9.9	11.2	29.2
2-Oct	8	9.4	10.4	29.6
5-Oct	11	10	9.3	24.6
7-Oct	13	9.5	11.3	27
9-Oct	15	8.8	11.5	26.2
14-Oct	20	10.5	6.8	18.6
16-Oct	22	10.5	10.7	18.2
19-Oct	25	10	7.6	27.3
21-Oct	27	10.2	7.8	35.2
23-Oct	29	10.6	10.3	38.9
26-Oct	32	9.9	9.7	33.5
28-Oct	34	10.7	10.3	31.5
30-Oct	36	11.1	8.7	25.5
2-Nov	39	10.9	9.3	20.9
4-Nov	41	10.3	10	22
9-Nov	46	11.5	9.8	22.6
13-Nov	50	10.6	11	20.4
16-Nov	53	11.8	14.8	26.2
20-Nov	57	11.7	9.9	23.8
23-Nov	60	12	9.3	17.3
27-Nov	64	11.4	6.3	24.4
13-Jan	111	10.1	2.9	15.4

Note: baseline phase = days 1-20
intervention phase = days 22-64
follow-up = day 111

Table 14. Isometic Peak Force (N) - Subject #2

Date	Day	R quads	L quads	R hams	L hams
25-Sep	1	415	401.25	174.75	174.75
28-Sep	4	352.25	309.75	179.5	164.25
2-Oct	8	504.25	415.25	156.5	159
5-Oct	11	393.5	481.25	165	149
7-Oct	13	386.75	488	163	174.25
9-Oct	15	432.5	397.75	156.75	176.5
14-Oct	20	389.5	354.75	169	163.75
16-Oct	22	406.25	438.5	167	163.75
21-Oct	27	425.75	393.25	160.25	146.5
23-Oct	29	408	392	157.5	143.25
26-Oct	32	337.75	368.25	169.5	157
28-Oct	34	415.5	383.5	176.25	160.75
30-Oct	36	482	478.25	191.25	179.25
2-Nov	39	486.75	417.25	196.25	192.5
4-Nov	41	595.75	463.5	188.5	189.5
9-Nov	46	552	515.75	200.5	172.25
13-Nov	50	556.75	536.75	202.25	183.25
16-Nov	53	611.25	587.75	196.75	164.5
20-Nov	57	610.5	580	199.25	187.75
23-Nov	60	665.5	711.25	209	192.5
27-Nov	64	666	632.75	188	173

Note: baseline phase = days 1-20
intervention phase = days 22-64

Table 15. Isokinetic Average Torque (Nm) - Subject #2

Date	Test	R quad conc	R quad ecc	L quad conc	L quad ecc	R ham conc	R ham ecc	L ham conc	L ham ecc
18-Sep	Pre	85	95	84	108	36	44	65	75
2-Dec	Post	82	111	89	115	46	60	40	45
13-Jan	Follow -up	94	112	79	102	46	54	43	47

Table 16. Daily Activity Levels - Subject #2

Date	Day	Time	Date	Day	Time
26-Sep	2	17	1-Nov	38	90
27-Sep	3	50	2-Nov	39	145
28-Sep	4	90	3-Nov	40	60
29-Sep	5	52	4-Nov	41	30
30-Sep	6	32	5-Nov	42	85
1-Oct	7	54	6-Nov	43	65
2-Oct	8	30	7-Nov	44	30
3-Oct	9	32	8-Nov	45	130
4-Oct	10	127	9-Nov	46	115
5-Oct	11	240	10-Nov	47	20
6-Oct	12	22	11-Nov	48	85
7-Oct	13	127	12-Nov	49	110
8-Oct	14	30	13-Nov	50	20
9-Oct	15	30	14-Nov	51	35
10-Oct	16	30	15-Nov	52	70
11-Oct	17	10	16-Nov	53	140
12-Oct	18	60	17-Nov	54	62
13-Oct	19	10	18-Nov	55	15
14-Oct	20	50	19-Nov	56	40
15-Oct	21	32	20-Nov	57	25
16-Oct	22	32	21-Nov	58	35
17-Oct	23	92	22-Nov	59	25
18-Oct	24	35	23-Nov	60	135
19-Oct	25	200	24-Nov	61	25
20-Oct	26	22	25-Nov	62	35
21-Oct	27	32	26-Nov	63	40
22-Oct	28	117	27-Nov	64	30
23-Oct	29	225	28-Nov	65	65
24-Oct	30	135	29-Nov	66	60
25-Oct	31	60	30-Nov	67	30
26-Oct	32	125			
27-Oct	33	35			
28-Oct	34	120			
29-Oct	35	30			
30-Oct	36	30			
31-Oct	37	170			

Table 17. DDS and Knee Function VAS Score - Subject #3

Date	Day	Intensity	Unpleasantness	VAS score
13-Oct	1	0	0	25.7
16-Oct	4	0.8	0.3	23.9
19-Oct	7	0.3	0.5	21.4
22-Oct	10	0.4	0	21.8
27-Oct	15	3.2	1.7	30.7
1-Nov	20	3.8	1.7	28.6
4-Nov	23	2.2	1.2	26.1
6-Nov	25	2.8	1.4	26.1
8-Nov	27	1.8	1.3	27.1
12-Nov	31	1.4	0.2	27.9
15-Nov	34	2.4	0.4	31.8
19-Nov	38	1.7	0.3	31.4
22-Nov	41	2.8	0.8	28.6
25-Nov	44	0.4	0.1	28.6
28-Nov	47	2	0.5	25.7
2-Dec	51	0.5	0.2	20.0
5-Dec	54	4.8	1.8	30.7
8-Dec	57	2	0.4	29.6
11-Dec	60	1.3	0.3	22.5
15-Dec	64	1.4	0.4	23.2
18-Dec	67	0.6	0.1	22.9
20-Dec	69	0.6	0.1	22.5
20-Jan	100	0	0	12.9

Note: baseline phase = days 1-27
intervention phase = days 31-69
follow-up = day 100

Table 18. Isometic Peak Force (N) - Subject #3

Date	Day	R quads	L quads	R hams	L hams
13-Oct	1	888	761.5	260.25	312.25
16-Oct	4	847.5	628.25	278	295.75
22-Oct	10	985.25	762.75	267.25	311.25
27-Oct	15	977.75	969.25	291.25	318.5
1-Nov	20	900.75	788.75	301.25	334.5
4-Nov	23	1051.25	972.5	309.5	312.25
6-Nov	25	1018.5	940.25	292	322.75
8-Nov	27	966.75	741.25	305.5	308
12-Nov	31	970	875.5	278.5	304.75
15-Nov	34	993.25	713.75	304.25	325.75
19-Nov	38	1044	862	310	308.75
22-Nov	41	914.75	790	286.5	286.5
25-Nov	44	957.25	865.75	278.5	308.75
28-Nov	47	1003.75	859	274	356.75
2-Dec	51	975.75	920.5	270.5	323.75
5-Dec	54	928.5	921	287	326.75
8-Dec	57	998.75	971.25	273.75	333
11-Dec	60	1000.75	900.5	286.75	324
15-Dec	64	1015	886.25	288	333
18-Dec	67	970.5	882.25	309.75	334.5
20-Dec	69	1027.75	891	282.75	316.75

Note: baseline phase = days 1-27
intervention phase = days 31-69

Table 19. Isokinetic Average Torque (Nm) - Subject #3

Date	Test	R quad conc	R quad ecc	L quad conc	L quad ecc	R ham conc	R ham ecc	L ham conc	L ham ecc
10-Oct	Pre	107	153	96	110	88	131	89	122
21-Dec	Post	144	209	112	170	90	131	92	123
20-Jan	Follow -up	137	184	126	171	82	129	93	125

Table 20. Daily Activity Levels - Subject #3

Date	Day	Time	Date	Day	Time
13-Oct	1	425	18-Nov	37	25
14-Oct	2	370	19-Nov	38	70
15-Oct	3	185	20-Nov	39	170
16-Oct	4	440	21-Nov	40	110
17-Oct	5	220	22-Nov	41	105
18-Oct	6	10	23-Nov	42	20
19-Oct	7	15	24-Nov	43	20
20-Oct	8	420	25-Nov	44	80
21-Oct	9	335	26-Nov	45	10
22-Oct	10	330	27-Nov	46	105
23-Oct	11	330	28-Nov	47	150
24-Oct	12	270	29-Nov	48	20
25-Oct	13	12	30-Nov	49	30
26-Oct	14	20	1-Dec	50	50
27-Oct	15	290	2-Dec	51	125
28-Oct	16	160	3-Dec	52	115
29-Oct	17	285	4-Dec	53	100
30-Oct	18	330	5-Dec	54	160
31-Oct	19	390	6-Dec	55	45
1-Nov	20	190	7-Dec	56	30
2-Nov	21	25	8-Dec	57	65
3-Nov	22	35	9-Dec	58	50
4-Nov	23	120	10-Dec	59	50
5-Nov	24	60	11-Dec	60	70
6-Nov	25	40	12-Dec	61	165
7-Nov	26	150	13-Dec	62	45
8-Nov	27	70	14-Dec	63	40
9-Nov	28	45	15-Dec	64	65
10-Nov	29	70	16-Dec	65	60
11-Nov	30	190	17-Dec	66	65
12-Nov	31	295	18-Dec	67	160
13-Nov	32	20	19-Dec	68	180
14-Nov	33	280	20-Dec	69	145
15-Nov	34	60			
16-Nov	35	15			
17-Nov	36	25			

Table 21. DDS and Knee Function VAS Score - Subject #4

Date	Day	Intensity	Unpleasantness	VAS Score
19-Oct	1	7.8	9	34.4
23-Oct	5	9.1	7.5	29.3
26-Oct	8	8.3	7.4	33.6
29-Oct	11	13.4	10.1	40.4
1-Nov	14	9.9	8	26.9
5-Nov	18	12.8	7	33.9
7-Nov	20	8.7	7.3	35.5
10-Nov	23	9.1	6.3	31.4
13-Nov	26	11.6	11.6	32.3
16-Nov	29	9.3	9.9	29.3
19-Nov	32	12.8	15.4	33.9
23-Nov	36	10.9	7	24.4
26-Nov	39	9.8	8.1	27.1
30-Nov	43	16.4	15.1	32.6
3-Dec	46	12.5	10.6	30.8
6-Dec	49	8.6	7.9	22.1
10-Dec	53	9.6	6.3	17.9
15-Dec	58	10.5	7	21.1
18-Dec	61	10.9	8.6	18.9
6-Jan	80	9.3	6	20.7
8-Jan	82	12.2	6.8	20.4
10-Feb	115	11	9.7	27.1

Note: baseline phase = days 1-32
intervention phase = days 36-82
follow-up = day 115

Table 22. Isometric Peak Force (N) - Subject #4

Date	Day	R quads	L quads	R hams	L hams
19-Oct	1				
23-Oct	5	372.5	461.25	260.5	218.75
26-Oct	8	430	403	261.25	237
29-Oct	11	421.5	434.25	266.25	268.5
1-Nov	14	446.5	524.75	248.75	247.5
5-Nov	18	441.75	548.5	264.5	273
7-Nov	20	443.5	516.25	261.25	272.75
10-Nov	23	418	514.25	231	237.25
13-Nov	26	399	594	222.75	251.25
16-Nov	29	334.75	486.25	211	236.75
19-Nov	32	414	542.25	219.25	219.25
23-Nov	36	290.25	501.25	203.25	204.5
26-Nov	39	317.75	466.75	224	224.5
30-Nov	43	188	359.25	210.25	209.5
3-Dec	46	291	595.75	214.75	239.75
6-Dec	49	510.25	612.75	183.25	206.25
10-Dec	53	616	666.75	237.25	255.25
15-Dec	58	451.75	535.75	246.25	234.75
18-Dec	61	551	612.25	235	240.75
6-Jan	80	293.25	432.5	241.75	243
8-Jan	82	488	676.75	239.75	247.25

Note: baseline phase = days 1-32
intervention phase = days 36-82
day 1: isometric data not collected

Table 23. Isokinetic Average Torque (Nm) - Subject #4

Date	Test	R quad conc	R quad ecc	R ham conc	R ham ecc	L quad conc	L quad ecc	L ham conc	L ham ecc
15-Oct	Pre	61	94	72	87	89	132	86	116
10-Jan	Post	57	82	94	117	103	137	64	84
10-Feb	Follow-up	46	70	74	86	56	84	88	107

Table 24. Daily Activity Levels - Subject #4

Date	Day	Time (min)	Date	Day	Time (min)
23-Oct	5	250	28-Nov	41	90
24-Oct	6	210	29-Nov	42	180
25-Oct	7	140	30-Nov	43	140
26-Oct	8	205	1-Dec	44	80
27-Oct	9	300	2-Dec	45	N/A
28-Oct	10	170	3-Dec	46	325
29-Oct	11	290	4-Dec	47	185
30-Oct	12	220	5-Dec	48	N/A
31-Oct	13	190	6-Dec	49	N/A
1-Nov	14	270	7-Dec	50	175
2-Nov	15	110	8-Dec	51	220
3-Nov	16	180	9-Dec	52	60
4-Nov	17	270	10-Dec	53	215
5-Nov	18	170	11-Dec	54	110
6-Nov	19	145	12-Dec	55	60
7-Nov	20	195	13-Dec	56	300
8-Nov	21	145	14-Dec	57	150
9-Nov	22	190	15-Dec	58	290
10-Nov	23	130	16-Dec	59	185
11-Nov	24	225	17-Dec	60	190
12-Nov	25	160	18-Dec	61	N/A
13-Nov	26	300	19-Dec	62	90
14-Nov	27	70	20-Dec	63	130
15-Nov	28	215	21-Dec	64	150
16-Nov	29	190	22-Dec	65	0
17-Nov	30	140	23-Dec	66	170
18-Nov	31	N/A	24-Dec	67	120
19-Nov	32	160	25-Dec	68	90
20-Nov	33	90	26-Dec	69	170
21-Nov	34	120	27-Dec	70	115
22-Nov	35	225	28-Dec	71	280
23-Nov	36	150	29-Dec	72	60
24-Nov	37	160	30-Dec	73	210
25-Nov	38	290	31-Dec	74	140
26-Nov	39	130	1-Jan	75	130
27-Nov	40	200	2-Jan	76	270

Table 24 cont'd. Daily Activity Levels - Subject #4

Date	Day	Time (min)
3-Jan	77	135
4-Jan	78	60
5-Jan	79	140
6-Jan	80	105
7-Jan	81	70
8-Jan	82	0
9-Jan	83	200
10-Jan	84	240
11-Jan	85	175
12-Jan	86	170
13-Jan	87	0
14-Jan	88	150
15-Jan	89	70
16-Jan	90	130

Appendix 17

C-statistic calculation

(Tryon, 1982)

1. Using the baseline data points, obtain a difference score by subtracting the first score from the second, the second from the third, and so on until all the baseline scores are used.

2. Square each of the values obtained in step 1. Add these squared values to obtain the "sum of squared difference scores".

3. Calulate the mean of the baseline data points.

4. Calculate the mean difference scores by subtracting each baseline raw score from the mean score.

5. Square each of the mean difference scores. Add these squared values to obtain the "sum of squared mean difference scores".

6. Insert the appropriate values in the formula below:

$$C = 1 - \frac{\text{sum of squared difference scores}}{(\text{sum of squared mean difference scores X } 2)}$$

7. Compute the standard error (SE) for the C-statistic using the formula below:

$$SE = \text{square root of } (n - 2 / (n - 1) \times (n + 1))$$

(where n = number of scores in baseline data series)

8. Determine significance of C-statistic by computing Z score from formula below:

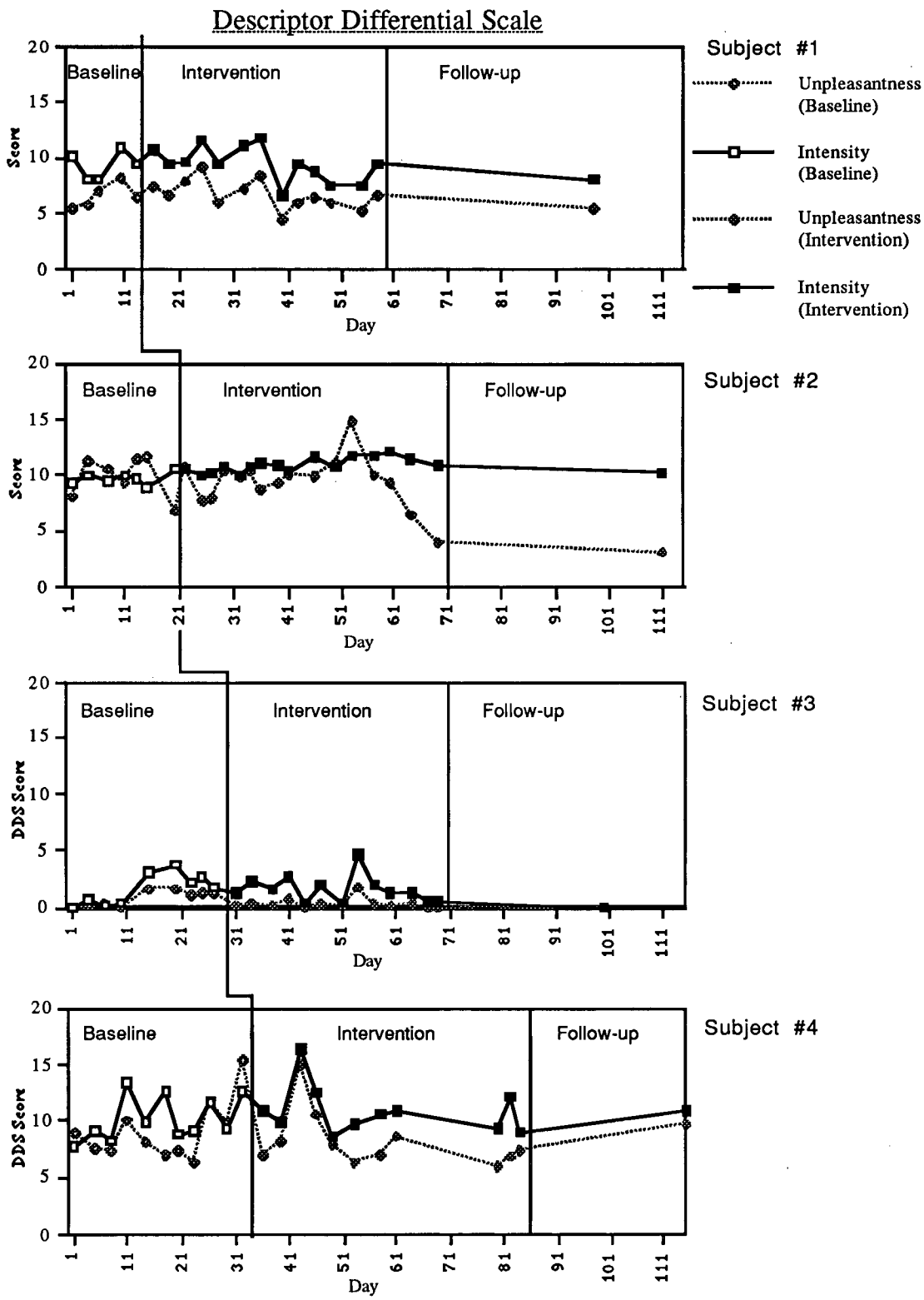
$$Z = \frac{\text{C - statistic value}}{\text{SE}}$$

(for any data series with 8 or more data points, a Z of 1.64 is statistically significant at a level of $p < 0.05$, one-tailed test)

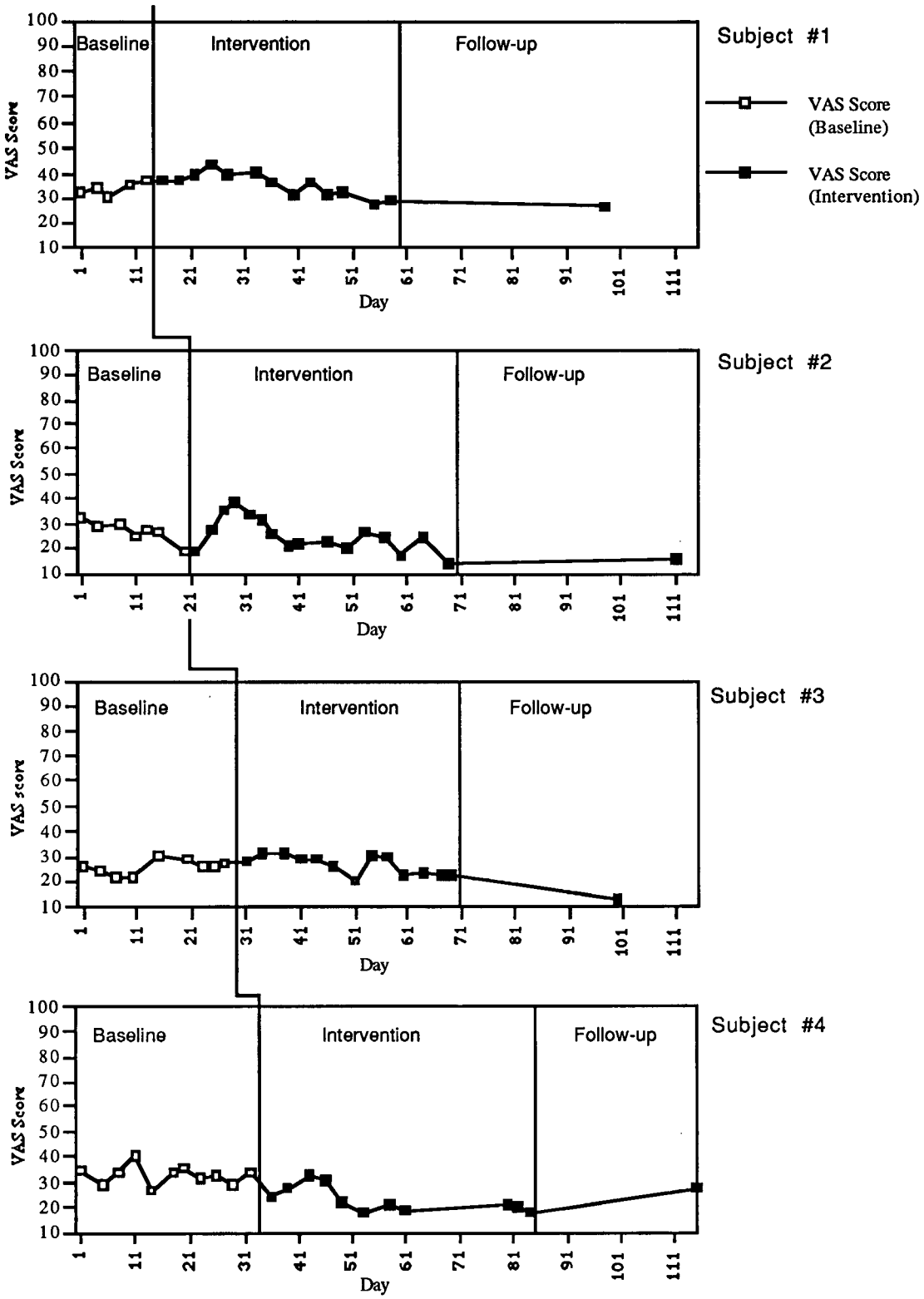
9. If the baseline C-statistic is not signicant, then a C-statistic can be calculated for the entire data series using the above formulae. A significant C-statistic ($p < 0.05$) calculated from the entire data series suggests a statistically significant trend across baseline and intervention phases.

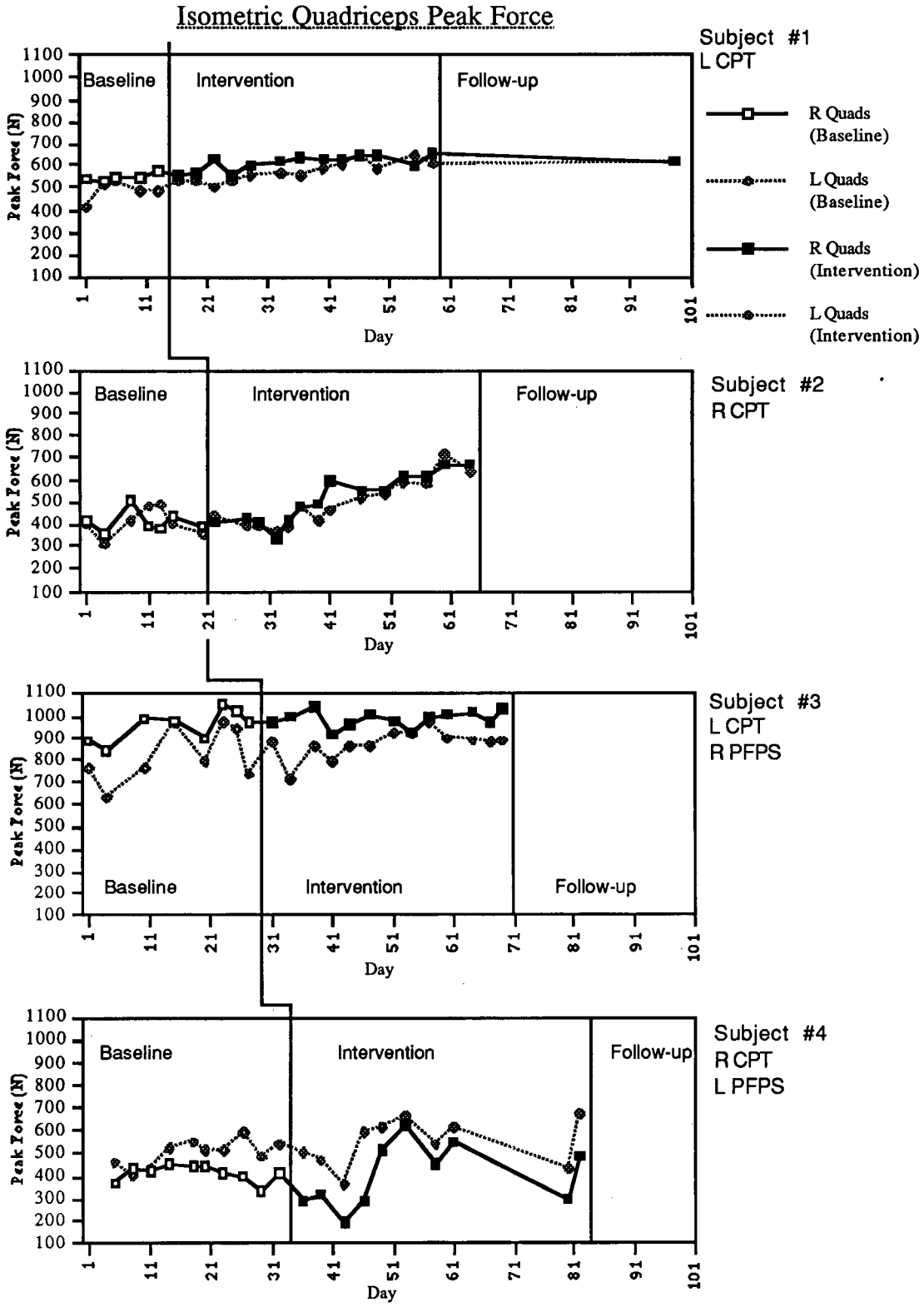
Appendix 18

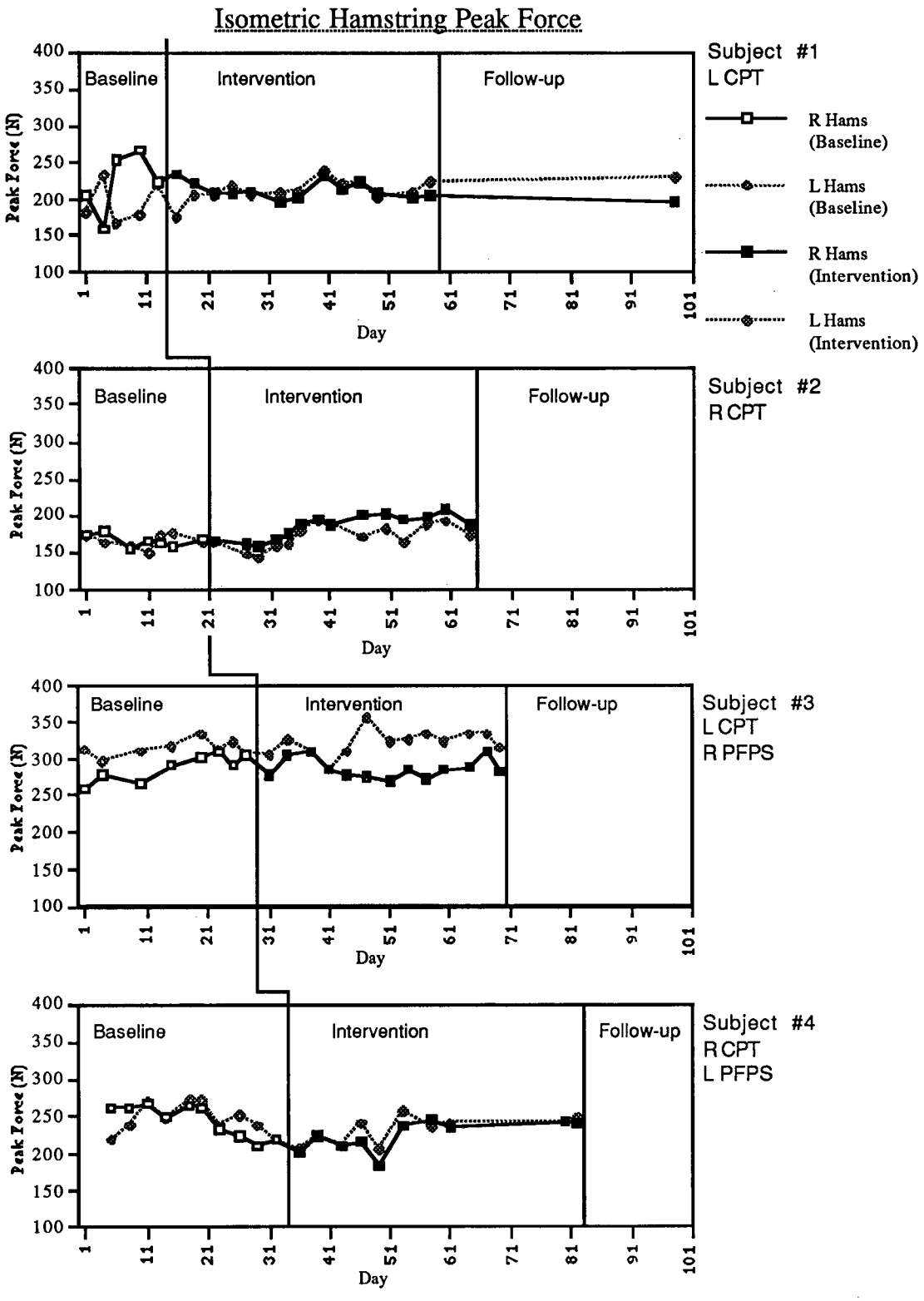
Composite Graphs: Between Subjects / Within Subjects



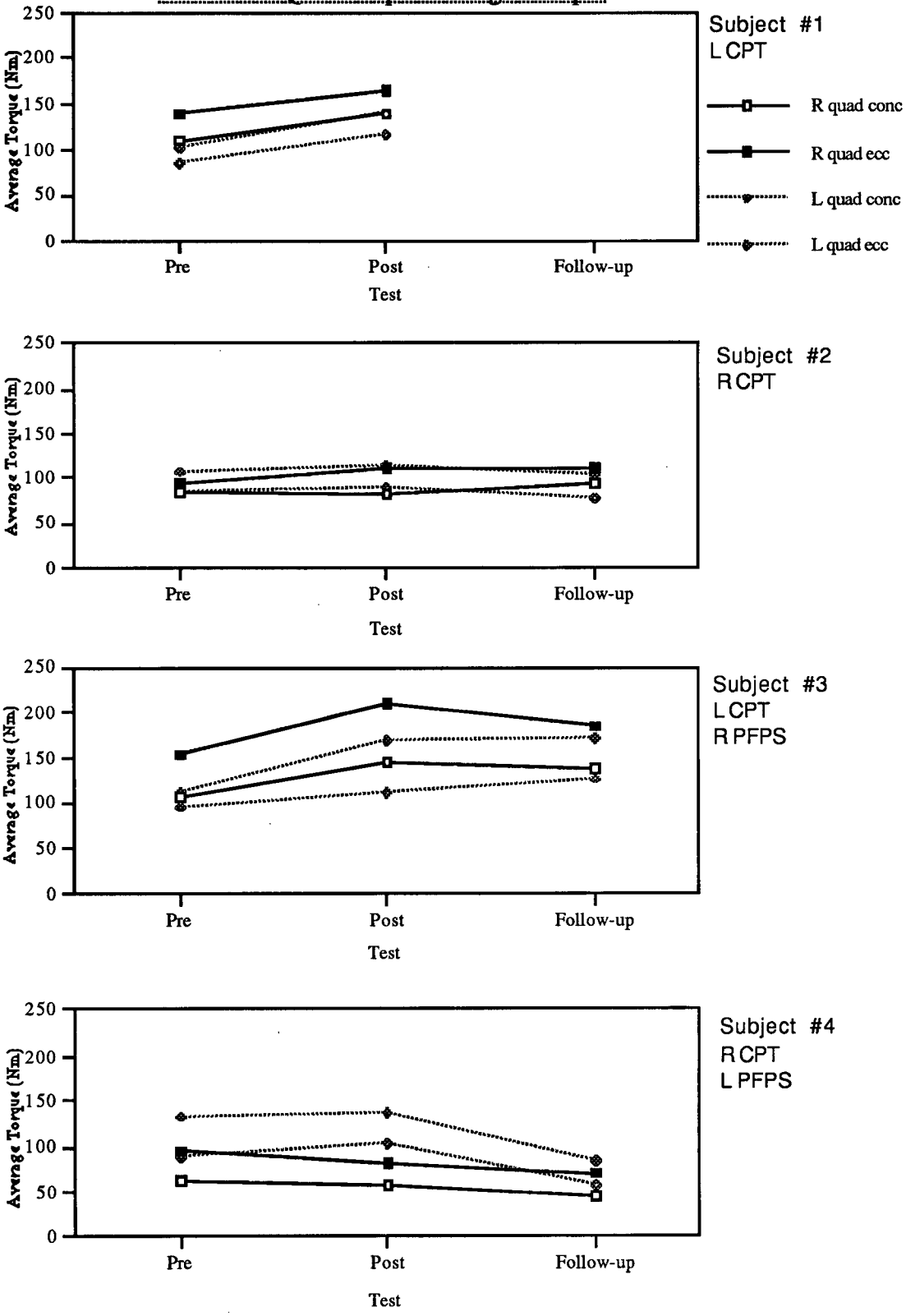
Knee Function VAS



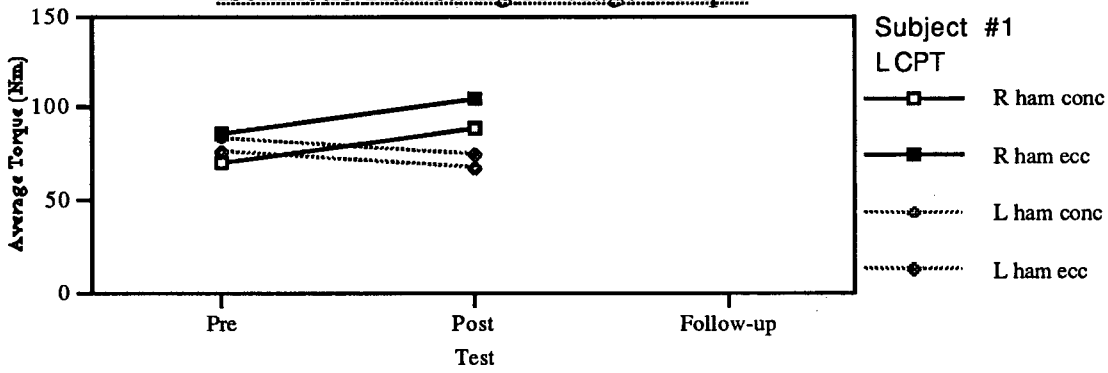

Isometric Quadriceps Peak Force
Subject #1
L CPT
R Quads (Baseline)
L Quads (Baseline)
R Quads (Intervention)
L Quads (Intervention)
Baseline
Intervention
Follow-up
Peak Force (N)
Day
Subject #2
R CPT
Subject #3
L CPT
R PFPS
Subject #4
R CPT
L PFPS

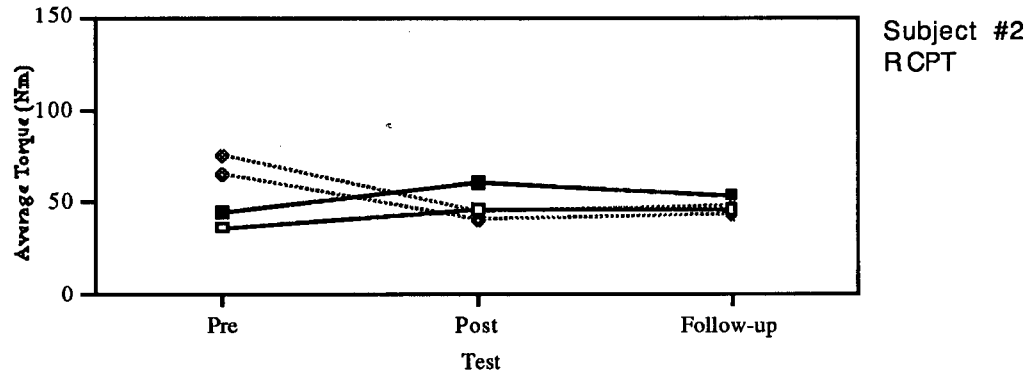

Isometric Hamstring Peak Force
Baseline
Intervention
Follow-up
Peak Force (N)
Day
Subject #1
L CPT
R Hams (Baseline)
L Hams (Baseline)
R Hams (Intervention)
L Hams (Intervention)
Subject #2
R CPT
Subject #3
L CPT
R PFPS
Subject #4
R CPT
L PFPS

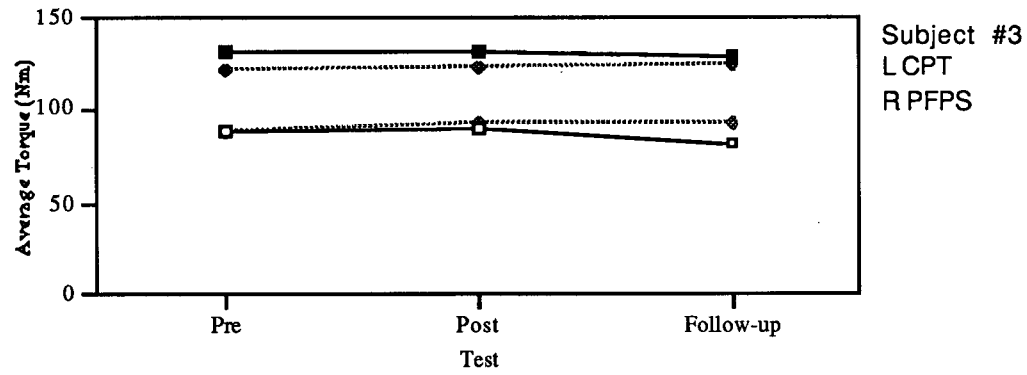
Isokinetic Quadriceps Average Torque

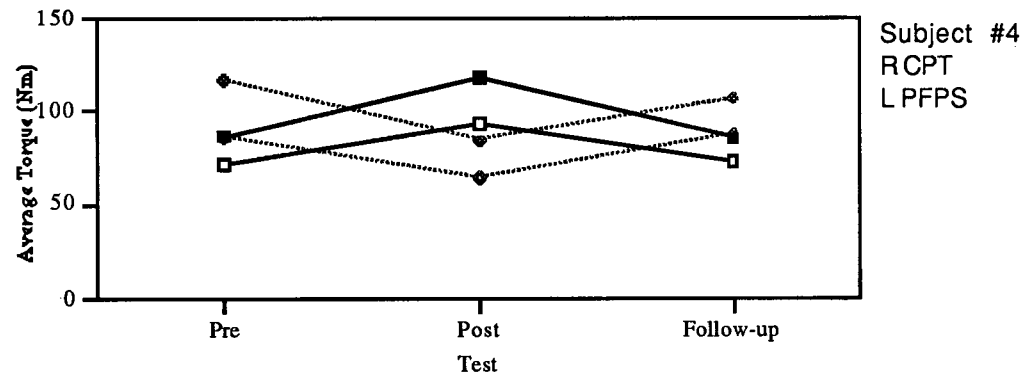


Isokinetic Hamstring Average Torque

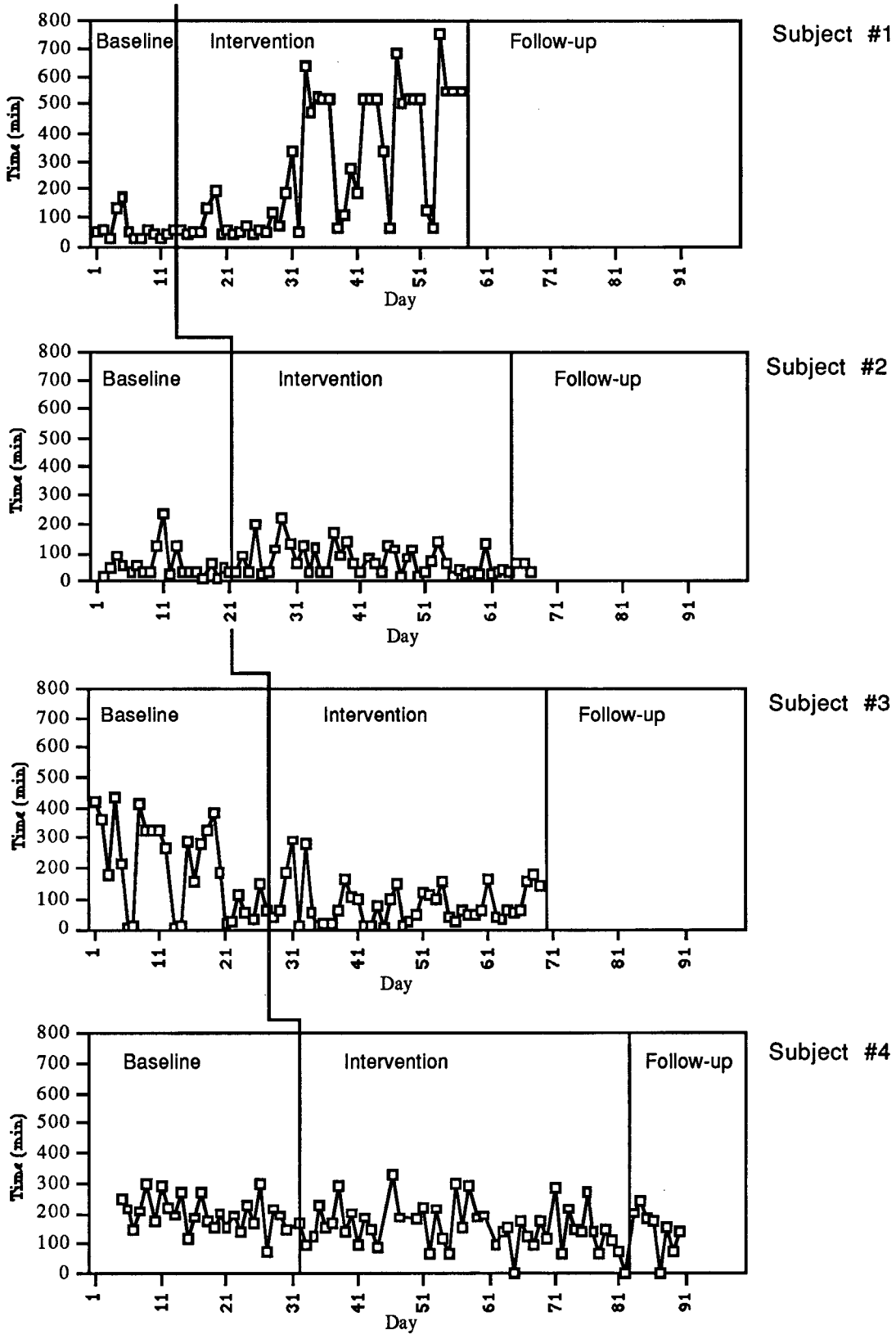






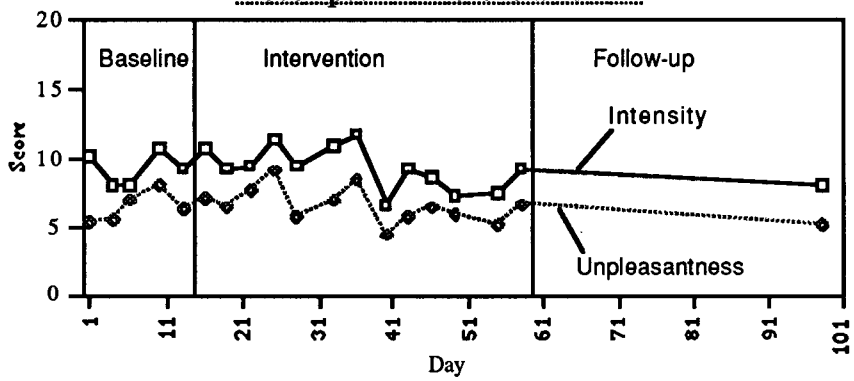


Daily Activity Levels

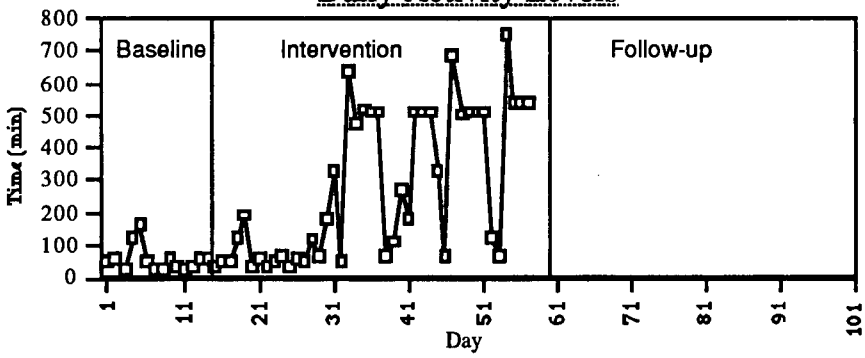


Subject #1
left knee CPT

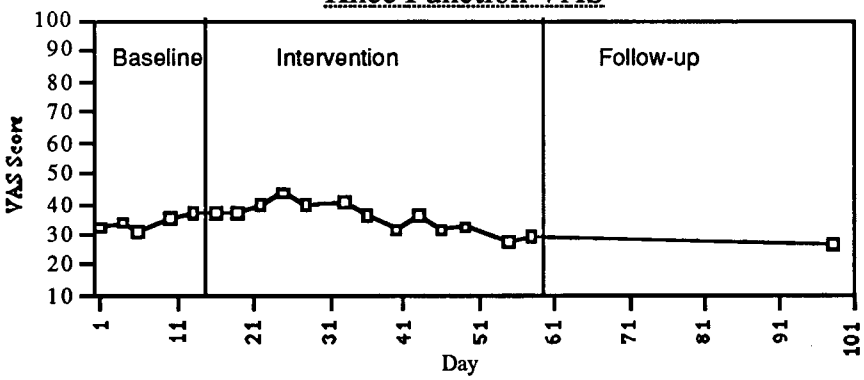
Descriptor Differential Scale



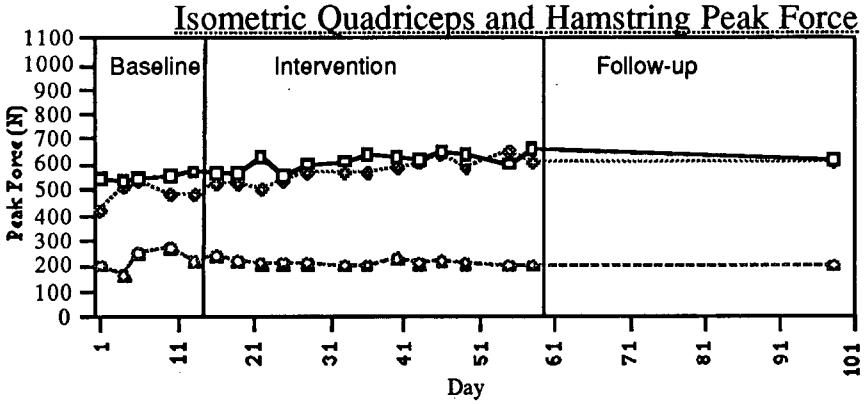
Daily Activity Levels

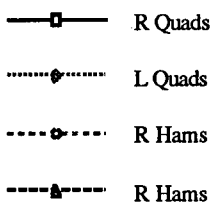


Knee Function VAS

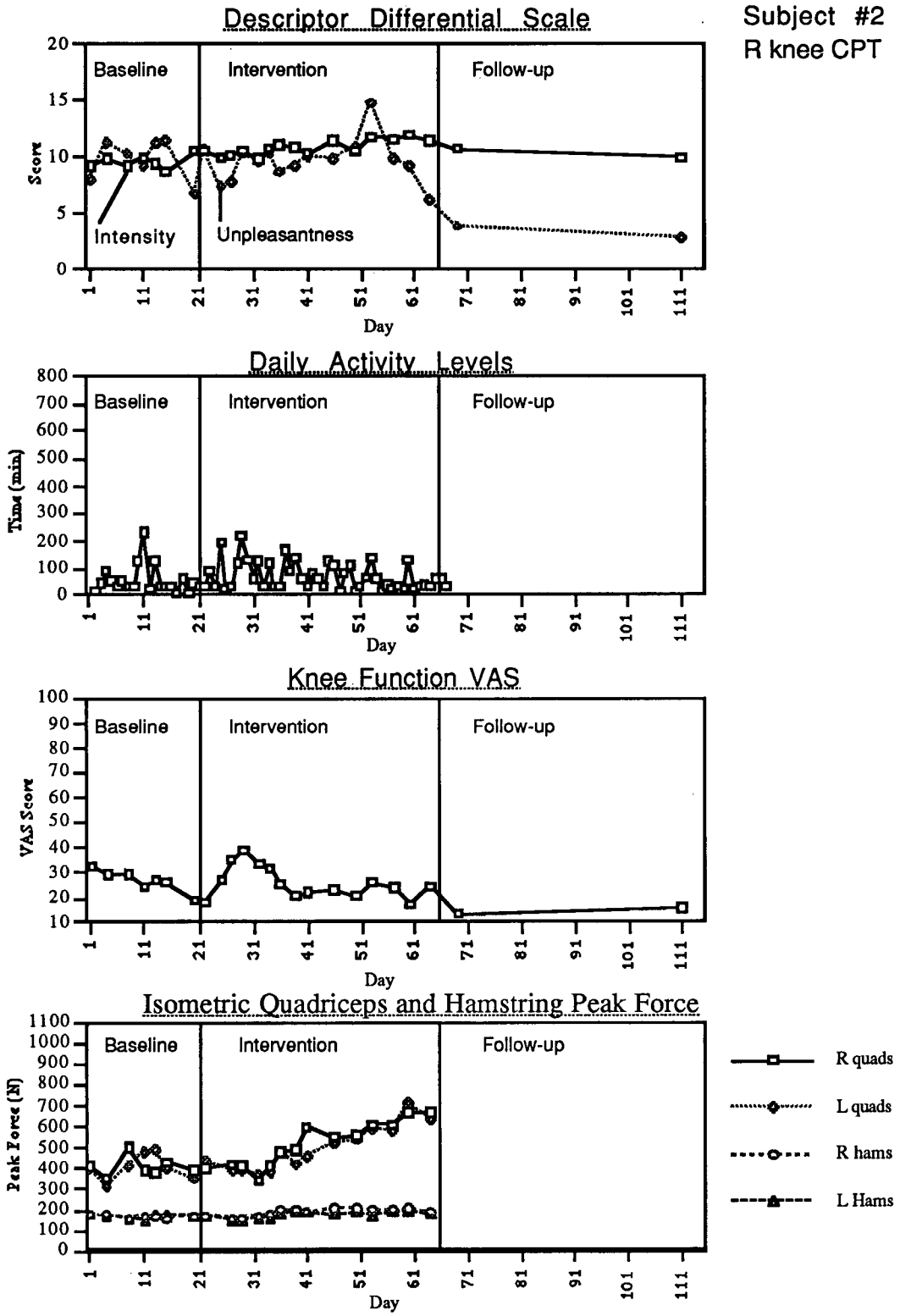


Isometric Quadriceps and Hamstring Peak Force

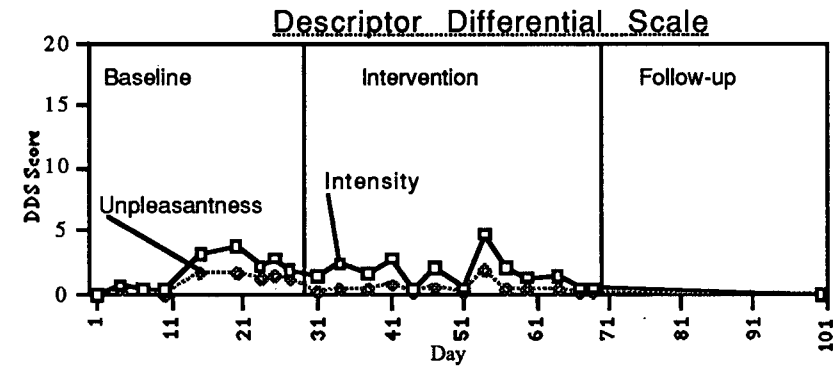


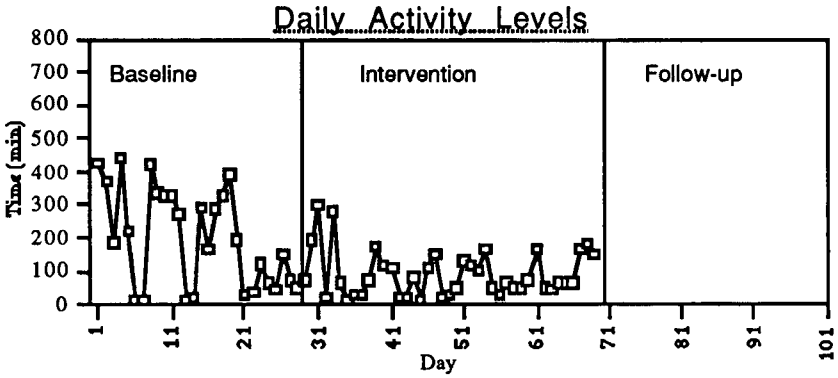


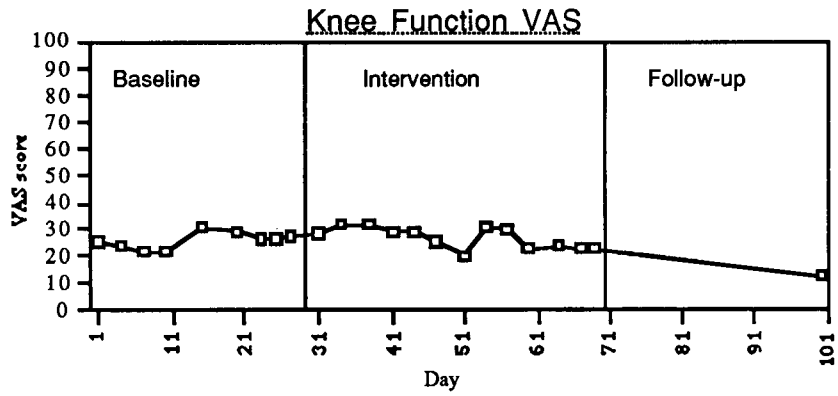
Subject #2
R knee CPT

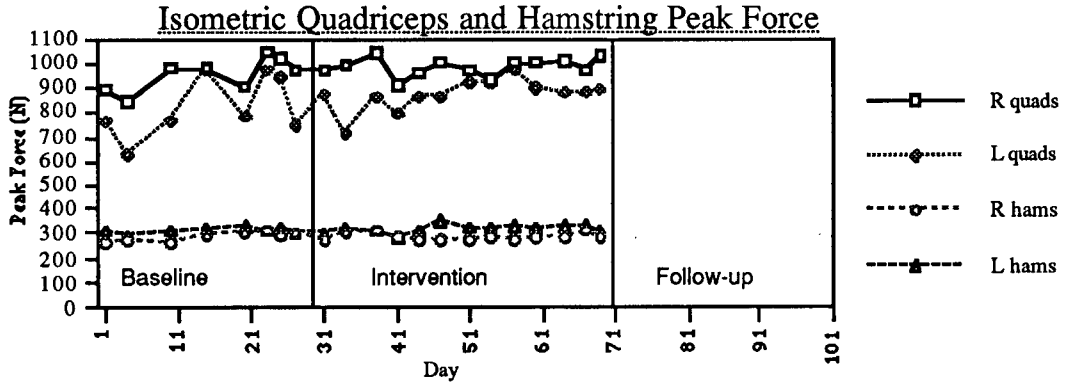


Subject #3
L knee CPT
R knee PFPS









Subject #4
R knee CPT
L knee PFPS