THE EFFECT OF INCREASING LEVELS OF EXERTION ON KNEE JOINT PROPRIOCEPTION

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The purpose of this study was to examine the effect of incremental levels of isokinetic concentric muscle exertion on passive reproduction of passive positioning (PRPP) and active reproduction of passive positioning (ARPP) at the knee joint in male and female collegiate soccer and basketball players.

Subjects for this study included 20 (10 males and 10 females) volunteers. Subjects performed knee extension and flexion concentric isokinetic exercise until torque output fell below the 10%, 30%, or 50% of maximum hamstring torque for three consecutive repetitions. Subjects were then tested on either PRPP or ARPP following the isokinetic exercise session. Following testing of the first independent measure, subjects were given a 20 minute rest period. Following the rest period, the procedure was repeated for two more exercise sessions. Testing of PRPP and ARRP was counterbalanced between trials and sessions in order to decrease the chance of a learning effect on the results of each testing session.

The major findings of this study indicate that increasing levels of exertion do not have a significant effect on either active reproduction ability [ARPP-45° ($F_{2,38} = 0.88$, $p = 0.42$), ARPP-30° ($F_{2,38} = 0.69$, $p = .51$), and ARPP-15° ($F_{2,38} = .23$, $p = 0.80$) or passive reproduction ability [PRPP-60°·s^{-1} ($F_{2,38} = 0.25$, $p = .78$) , PRPP-90°·s^{-1} ($F_{2,38} = 0.31$, $p = 0.73$), and PRPP120°·s^{-1} ($F_{2,38} = 1.58$, $p = 0.22$)]. However, the reliability of all PRPP and ARPP measures at 15° demonstrated poor reliability.

Fatigue has long been theorized to be a contributing factor in decreased proprioceptive acuity, and therefore a contributing factor to joint injury. The lack of significant findings may be explained by the idea that as the level of muscle fatigue increases muscle spindle discharge increases. Poor reliability for all PRPP and ARPP at 15° draws into question the meaningfulness of the results for these measures.

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CHAPTER 1

INTRODUCTION

Research Problem

Ligament injuries of the knee represent a significant percentage of lower extremity injuries during athletic participation. Epidemiological studies indicate that female soccer and basketball players have a higher incidence of ligament injury, particularly the anterior cruciate ligament (ACL), when compared to their male counterparts (23, 64, 103). Studies involving college age males and females showed female soccer players were 2.3 and female basketball players were 2.9 times more likely to sustain an ACL injury compared to males in the same sports (3, 4, 17). At the high school level, studies indicate ACL injuries in female basketball players are 3.5 to 3.8 times more common than in male basketball players (45, 101, 120). Other studies indicate that the occurrence of ACL injuries in females are up to eight times that of males (3, 109). Several factors have been suggested for these differences including hormonal factors (2, 56, 64, 84, 140, 158, 159), anatomic factors (3, 65, 104, 138, 147), fatigue (58, 63, 74, 107, 108, 128-130, 139), joint laxity (96, 98, 128, 129), inadequate kinesthetic/proprioceptive sensibility (74, 128, 129), and decreased neuromuscular control (43, 62, 128, 135, 160). Fatigue is perhaps the single most important due to fatigue's manageability via training and fitness. Of particular interest here is the role of fatigue in the underlying mechanisms involved in proprioceptive sensibility.

Kinesthetic sense (kinesthesia) is defined as the awareness of body position and body movement (91, 123, 155). Kinesthesia, which includes proprioceptive sense, is a key factor involved in injury avoidance and skilled movement. Kinesthesia and proprioception are needed to provide crucial position, movement, and force information. As effectors, muscles merely obey the commands of the central nervous system (CNS). A strong fit muscle is of no use if the muscle is not activated in time to protect itself, a joint, or other muscles (138), and, the CNS commands are only as good as the sensory information on which they are based.

Proprioception, the sense of joint position and movement of a joint, is one component of kinesthesia (68, 76-82, 91, 126). Proprioceptors (position sensors) are found in muscles, joints, and skin. These receptors are sensitive to changes in stretch, and relay information regarding joint movement and position to the CNS for interpretation and evaluation. Studies of ligament and muscle receptors indicate that ligament receptors are more sensitive near the end limits of a joint's motion (22, 29, 49, 68, 73, 127, 141, 166), whereas muscle receptors may play a key role in the mid-range of motion (49, 68, 76, 91, 126, 141). Using afferent information provided by proprioceptors, the CNS relays efferent signals to muscles to generate a muscle contraction or does not relay efferent signals resulting in muscle relaxation.

Fatigue is generally associated with failure of muscle to sustain a contraction. Other definitions include, an inability to maintain a given force and an inability to maintain a given exercise intensity (11, 19, 24, 39, 40, 47). Central factors (CNS factors), however, should not be overlooked. At the very least, painful input due to exertion from free nerve endings (nociceptors) may reduce an athlete's motivation to continue. At the extreme, reduced output from sensory receptors and delayed output from CNS integration centers, and/or the alpha and gamma motor systems may cause a reduction in the protective reflex mechanisms of muscles (11, 14) (Figure 1).

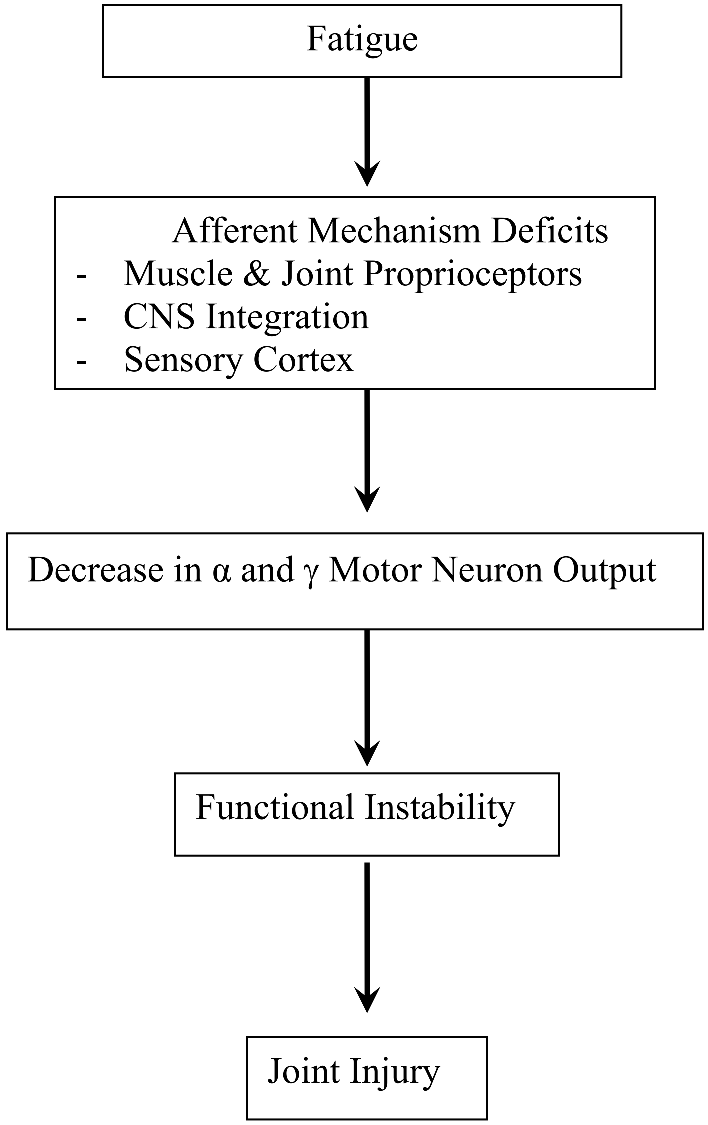


Figure 1. The Effect of Fatigue on Proprioception

Previous research has shown a decrease in neuromuscular control (104, 105, 128, 129, 160) and proprioception (74, 100, 104, 105, 130, 139, 153) following maximal fatigue of muscles surrounding the knee joint, during mild fatigue in anterior cruciate ligament deficient (ACL-D) individuals (75), and following low intensity work to fatigue (18). During athletic competition, athletes commonly slow down or are unable to generate the same amount of force that they could during earlier phases of the activity. In this investigator's opinion, however, it is rare to see an athlete fatigued to the point of collapse (maximum fatigue) in sports such as soccer or basketball. Fatigue, therefore, should be viewed as a continuum ranging from minimal to maximum levels (Figure 2).

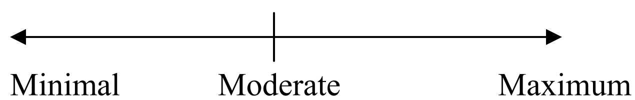


Figure 2. Fatigue continuum

There is a link between information provided by sensory inputs to the CNS and the performance of an appropriate motor task. This link requires proper functioning of sensory receptors, CNS integrating centers, sensory cortex, cerebellum, spinal cord, alpha motor neurons, gamma loops, motor end plates, and muscle cells, for appropriate performance of motor tasks (10, 14, 24, 36, 43, 93, 122, 141). Deficiencies, related to fatigue at any of these levels may result in decreased motor performance (18, 86, 88, 145).

Statement of Purpose

The purpose of this study was to examine the effect of incremental levels of isokinetic concentric muscle exertion on passive reproduction of passive positioning (PRPP) and active reproduction of passive positioning (ARPP) at the knee joint in male and female collegiate soccer and basketball players.

Significance of Study

The effect of increasing levels of exertion on knee joint proprioception has not been investigated to date. Information obtained from this study will add to the general database of knowledge concerning the effects of exertion on proprioception. Specifically, this study hopes to establish that PRPP and ARPP scores plateau between moderate and maximum levels of exertion. If a plateau relationship is demonstrated, it may provide evidence that proprioceptive abilities are decreased to a level that might prevent muscles from reacting to injurious forces at the knee joint prior to maximum muscle fatigue. If correct, this study would provide a potential link between non-maximal exertion levels, diminished proprioception, and knee joint ligament injury.

Hypotheses

It is expected that:

1. A difference in PRPP and ARPP scores for all conditions will be found between males and females.
2. Ratings of perceived exertion will not be different between males and females for each level of exertion.
3. Passive Reproduction of Passive Positioning ability at a knee joint velocity of 60°/s will be different at exertion levels of 10% below maximum isokinetic torque, 30% below maximum isokinetic torque, and 50% below maximum isokinetic torque.

4. Passive Reproduction of Passive Positioning ability at a knee joint velocity of 90°/s will be different at exertion levels of 10% below maximum isokinetic torque, 30% below maximum isokinetic torque, and 50% below maximum isokinetic torque.
5. Passive Reproduction of Passive Positioning ability at a knee joint velocity of 120°/s will be different at exertion levels of 10% below maximum isokinetic torque, 30% below maximum isokinetic torque, and 50% below maximum isokinetic torque.
6. Active Reproduction of Passive Positioning ability at a knee joint angle of 15° will be different at 10% below maximum isokinetic torque, 30% below maximum isokinetic torque, and 50% below maximum isokinetic torque.
7. Active Reproduction of Passive Positioning ability at a knee joint angle of 30° will be different at 10% below maximum isokinetic torque, 30% below maximum isokinetic torque, and 50% below maximum isokinetic torque.
8. Active Reproduction of Passive Positioning ability at a knee joint angle of 45° will be different at 10% below maximum isokinetic torque, 30% below maximum isokinetic torque, and 50% below maximum isokinetic torque.

Limitations

1. Experience in sport varied among subjects.
2. Maximum torque production varied between testing trial and/or test day for each subject.
3. Testing was not conducted at every possible isokinetic speed.
4. Testing was not conducted across a wide spectrum of subjects.
5. Results cannot be generalized to non-isokinetic movements.
6. The training level of each subject was not controlled.

7. Motivation and anxiety related to testing may have affected the subject's torque production and PRPP and ARPP scores.
8. Isokinetically induced concentric hamstring muscle fatigue may not involve the same mechanisms involved in muscle fatigue induced by sport participation.

Delimitations

1. Subjects were confined to isokinetic movement patterns of knee flexion and extension.
2. Subjects' muscle contractions were confined to concentric tension.
3. Subjects were confined to a seated position for isokinetic exercise and proprioception testing procedures.
4. Study was confined to a small subset of isokinetic speeds.

Assumptions

It was assumed that:

1. Each subject produced maximum effort during each isokinetic exercise session.
2. The subjects honestly reported their leg dominance.
3. Subjects honestly reported any orthopedic or other health related problems that might prevent inclusion in this study.

CHAPTER 2

REVIEW OF LITERATURE

Anatomy and Biomechanics of the Knee Joint

The knee joint is a compound synovial joint which functions as a modified hinge and is comprised of three bones, the femur, tibia, and patella, and three articulating surfaces, the medial tibiofemoral, lateral tibiofemoral, and the patellofemoral articulations. The three articulating surfaces are enclosed in a common joint capsule with the tibiofemoral articulations involved in movement of the knee joint (125, 142). Motion of the knee joint is constrained to six degrees of freedom: flexion-extension, axial rotation, varus-valgus, anterior-posterior translation, medial-lateral translation, and compression-depression (83, 102, 142). Movements of flexion and extension involve a combination of the femoral condyles rolling and gliding over the tibial articulating surfaces.

The knee functions to withstand large forces, provide great stability, and afford a large range of motion (41, 142, 155-157). The combination of anatomical mobility and high movement demand causes the knee joint to be the "weak link" in the lower extremity (89, 98, 142, 155, 156). Mobility is primarily provided by the bony structure, whereas, stability is primarily provided by muscles, ligaments, and cartilage (25, 66, 142). Injury to these stabilizing structures is common in athletics (5, 156).

Bony Anatomy and Biomechanics

The superior portion of the knee joint is made up of the distal enlargements of the medial and lateral femur known as the condyles. The condyles are convex both longitudinally and transversely and are separated by the intercondylar fossa (125, 142). The femoral condyles articulate with the two smaller tibial condyles which are both slightly concave with the lateral tibial condyle also convex anteriorly and posteriorly (142). Together with the intercondylar eminence, which helps to increase the congruity of the joint, these structures make up what is referred to as the tibial plateau (125, 142). Compressive forces created by the "closed-pack-position", have been found to be an important factor in providing stability to the knee joint and preventing shear forces that create anterior-posterior translation of the tibia relative to the femur (38).

The longitudinal articulating surface of the femoral condyles is approximately twice the length of the surface of the tibial condyles (142). This difference does not allow for pure rolling motion to occur during the motions of knee flexion and extension. Instead, the condyles execute both rolling and gliding movements with varying ratios throughout the range of motion (142). Rolling is predominate at the initiation of flexion and gliding occurs more at the end of flexion (142).

<u>Soft Tissue Anatomy and Biomechanics</u>

Musculature of the Knee. The knee musculature of the lower extremity is located in the anterior and posterior compartments of the thigh and posterior compartment of the lower leg (125, 142, 149). The extensor muscles of the knee occupy the anterior compartment of the thigh and are innervated by the femoral nerve. The primary flexor and rotator muscles are located in the posterior compartments of the thigh and the lower leg and are innervated by branches of either the sciatic or tibial nerve depending on their location (125, 149).

The primary knee extensor muscles, commonly referred to as the quadriceps femoris, consist of the vastus medialis, vastus intermedius, vastus lateralis, and the rectus femoris, which have independent origins and a common insertion at the base of the patella known as the quadriceps tendon (125). The vastus medialis originates from the lower portion of the intertrochanteric line, the medial lip of the linea aspera, and the medial intermuscular septum. The vastus intermedius has its origin at the anterior and lateral surface of the femur, and the vastus lateralis originates from the greater trochanter and the linea aspera of the femur (149). The rectus femoris originates from the ilium by two separate heads, a straight head arises from the anterior inferior iliac spine and a reflected head which originates just above the margin of the acetabulum (125). The action of the quadriceps is applied to the knee through the patellar ligament, which attaches the apex of the patella to the tibial tuberosity (125, 149). All four of the quadriceps femoris muscles receive innervation from branches of the femoral nerve (125, 142, 149). Research indicates that the quadriceps muscle group provides a significant anterior shear force to the anterior cruciate ligament (ACL), particularly when the hamstring group is in a fatigued state (6, 7, 35).

The primary knee flexor muscles of the posterior compartment of the thigh are known collectively as the hamstrings, which include the biceps femoris, semitendinosus, and the semimembranosus. The biceps femoris consists of a long and short head. The long head arises from the ischial tuberosity, and the short head originates from the lateral lip of the linea aspera and the lateral intermuscular septum. The two heads converge in the lower third of the thigh and insert on the head of the fibula. The semitendinosus originates from the ischial tuberosity and its tendon of insertion passes behind the knee and inserts on the proximal medial surface of the tibia just below the medial condyle. The semimembranosus arises from the lower portion of the ischial tuberosity and inserts at the posteromedial side of the medial tibial condyle. The three muscles of the hamstrings receive innervation from branches of the sciatic nerve (125, 142, 149).

Carlsöö (28) noted that the mechanical effect of the hamstrings differed from conditions of free hanging (open kinetic chain) and when the foot was in contact with the ground. The investigators found that during the free hanging (open kinetic chain) condition the hamstrings created a flexor moment, whereas during the closed kinetic chain condition an extensor moment was created. Escamilla (38), however, noted that the hamstrings exert a posterior force throughout the range of motion.

The knee flexors located in the posterior compartment of the lower leg are the gastrocnemius, popliteus and plantaris. The two heads of the gastrocnemius have their proximal attachment above the femoral condyles and a distal attachment via the tendocalcaneus (Achilles tendon) to the calcaneus. Innervation of the gastrocnemius is from branches of the tibial portion of the sciatic nerve. The popliteus attaches proximally to the lateral condyle of the femur and distally to the posterior-medial surface of the tibia above the soleal line. It receives innervation from the tibial nerve (125, 142, 149).

Muscles that act as internal rotators of the tibia with respect to the femur are the semitendinosus, semimembranosus, popltieus, gracilis, and sartorius. External rotation of the tibia with respect to the femur is produced by the biceps femoris (142). The gracilis originates from the body and inferior ramus of the pubis and inserts on the medial surface of the tibia just below the medial condyle and is innervated by the obturator nerve. The sartorius originates from the anterior superior iliac spine and inserts on the upper medial surface of the tibial shaft near the gracilis and semitendinosus. Innervation of the gracilis is via the femoral nerve (125, 149).

Menisci of the Knee. The medial and lateral menisci are semilunar shaped fibrocartilage that increase the congruency of tibiofemoral articulations and help to distribute pressure by spreading joint reaction forces over a larger surface area of the articulating surfaces (142). Both menisci are attached to the tibia at their anterior and posterior horns and through the coronary ligaments. The coronary ligaments are a part of the joint capsule that attaches the peripheral edges of the menisci to the margin of the tibia (125). Further support for the menisci is provided by ligamentous and tendonous attachment to the menisci.

Collateral Ligaments of the Knee. The medial and lateral collateral ligaments prevent passive movement of the knee in the frontal plane (142). The medial collateral ligament prevents abduction of the tibia on the femur and the lateral collateral prevents adduction.

The medial collateral ligament is a broad flat band that extends from the medial epicondyle to the medial aspect of the tibia below the medial tibial condyle (125, 155). The lateral collateral ligament is cord-like and runs between the lateral femoral epicondyle and the apex of the fibula (125). The attachments of the collateral ligaments on the femoral epicondyles are offset posteriorly and superiorly to the axis of rotation, which causes the ligaments to become taut when the knee is extended and slack when the knee is flexed (142). The collateral ligaments help to prevent anterior and posterior displacement and tibial rotation when the knee is extended and allow these movements as the knee is flexed.

Cruciate Ligaments of the Knee. The anterior and posterior cruciate ligaments provide control and stability to the knee throughout the motions of flexion and extension (151). These ligaments lie within the femoral intercondylar fossa in the center of the joint. Although the cruciate ligaments are intimately related to the joint capsule, they are extracapsular structures (142). The cruciate ligaments maintain a relatively constant length throughout the motions of flexion and extension even though not all parts are taut at the same time. The relatively constant length of the cruciate ligaments helps to force the sliding motions of the condylar surfaces to occur (121, 155). In addition to their primary functions, both cruciate ligaments function secondarily as internal collateral ligaments, preventing varus and valgus rotation of the knee (103, 110).

The anterior cruciate ligament (ACL) consists of two distinct bundles, the anteromedial bundle which is tight in knee flexion and lax in knee extension, and the posterolateral bundle which is tight in knee extension and lax in knee flexion (8, 12, 85, 131, 136, 144, 154, 156, 161). The ACL attaches to the anterior intercondyler fossa of the tibia and runs laterally and superiorly to attach on the inside of the lateral condyle of the femur and is the primary stabilizer preventing anterior displacement of the tibia on the femur (152). The ACL is innervated by a neurovascular bundle in the subsynovial connective tissue, and is innervated primarily at its end regions (166).

The posterior cruciate ligament (PCL) attaches on the posterior intercondylar fossa of the tibia and runs medially to attach on the inside of the medial femoral condyle. The PCL consists of two bundles, the anterolateral bundle is tight in flexion and lax in extension while the posteromedial bundle is tight in extension and lax in flexion (156, 161). The PCL limits posterior displacement of the tibia on the femur (99).

Proprioception

Sir Charles Bell first described the sense of position and actions of the limbs, referring to it as the "sixth sense" (91). This "sixth sense" concerns perceived sensations about the static position or velocity of movement of body parts and perceived sensation about the forces generated during muscular contractions (91).

Input from a variety of sources contributes to kinesthetic sense (155). Some aspects of kinesthetic sensibility, such as a sense of effort and heaviness, and a sense of timing, are generated by sensory centers that monitor motor commands sent to muscles (155). Other aspects of kinesthesia are generated largely by input from peripheral receptors that monitor the execution of the motor command (155). For example, input from the eyes and ears are responsible for generating a sense of body position in space and time (155). The sense of joint movement and joint position is generated by receptors located in the skin, muscles, tendons, joint capsules, and ligaments surrounding the joints (11, 22, 68, 91, 122, 126, 141, 155, 166). These receptors are referred to as proprioceptors and the sensation they provide is called proprioception (91).

Proprioceptive organs signal to the central nervous system information about the relative position of body parts. Apart from pressure receptors in the soles of the feet, proprioceptive organs do not supply any information as to the orientation of the body with respect to gravity; proprioceptors only signal the position of one body part with respect to another (126). The receptors involved lie in muscles and musculotendinous junctions (muscle spindles and Golgi tendon organs), joint capsules and ligaments (Pacinian corpuscles and Ruffini end organs), and the skin (126). Research indicates that the function of these sensory organs may be affected by the type of activity being performed (59, 77, 93, 94, 122, 132), joint hypermobility (55, 96), tendon vibration (31), joint effusion (53), joint anesthesia (29), gender (56, 63, 128, 129), injury to joint (9, 20, 72, 78, 80, 82, 138) or musculotendinous structures (80), nervous system injury (49), elastic bandages, neoprene sleeves taping and bracing (16, 21, 27, 111), and fatigue (10, 11, 18, 32, 58, 74, 100, 104-108, 132, 139, 165). Limb dominance (52, 65) however, does not appear to play a role in proprioceptive abilities.

Joint Capsule and Ligament Proprioceptors

Several sensory receptors have been identified in the joint capsule. These sensory receptors have been named based on similarities to receptors found in other tissue and include: Ruffini endings which are found in the joint capsule, Golgi endings located in the ligaments of the joint, encapsulated Paciniform endings found in the fibrous periosteum near articular attachments, and free nerve endings found in the joint capsule (68, 155). The distribution of these receptors is nonuniform within the joint, which may reflect the location of areas of increased stress on the joint capsule and ligaments during movement (29, 49, 166).

Two main types of proprioceptors in the joint capsule and ligaments are the Ruffini end organs (Ruffini corpuscles) and Pacinian corpuscles. Ruffini end organs consist of a small number of main terminal branches with a profuse system of small branches on the end of each main branch (76, 81, 91, 122, 123, 155). The end of each main branch and its smaller branches are enclosed within a connective tissue covering and the entire corpuscle is located between the collagenous fibers of the capsule and ligament (91, 155). Ruffini end organs are reported to be mostly responsive to tension. Pacinian corpuscles consist of a single terminal branch surrounded by several concentric layers of Schwann cells, all of which are enclosed in a connective tissue cover (76, 81, 91, 122, 123, 155). Pacinian corpuscles appear to be responsive to compression forces, and are widely distributed between the collagenous fibers of the joint capsule and surrounding fascia (76, 81, 91, 122, 123, 155).

The role of ligaments in proprioception is unclear. Because tension in ligaments may be caused by movement in a number of different directions, proprioceptors within ligaments most likely cannot provide information on movement in any specific direction (155). Ligament proprioceptors may make a significant contribution to joint stabilization in reflexive muscular activity (22, 29, 49, 67, 142, 155, 166).

Joint capsule receptors are innervated by distinct nerve branches as well as by branches from nerves supplying adjacent muscles and overlying skin. Most joint capsule receptors appear to discharge at the extremes of movement, typically during flexion and extension, providing information relative to joint position. These response properties suggest that proprioceptors located in the joint capsule function as limit detectors, whose role is to signal extreme positions of the joint, and in so doing, prevent damage to the joint (49, 63, 67, 76, 91, 155).

According to Jones (68), joint receptors in the hand appear to play a more important role in proprioception than joint receptors do in the proximal areas of an extremity such as the knee. Zimny et al. (166), however, found that the anterior cruciate ligament contained not only free nerve endings but also Ruffini end organs and Pacinian corpuscles as well. Zimny et al. (166) also indicated that the greatest populations of receptors were in the femoral and tibial attachment areas of the ligament, and that the receptors constituted 2.5% of the ligament. From these findings, Zimny et al. (166) concluded that the human anterior cruciate ligament is capable of discriminating afferent outflow to the central nervous system. In cats, Krauspe (73) found that most afferent fibers arising from the ACL were activated by the application of local pressure near the femoral attachment. The afferents were activated when the knee was extended, flexed, externally rotated, and internally rotated with the greatest activity during hyperextension with either external rotation or internal rotation. Receptor discharge activity was not found at a resting length of 30 degrees of flexion. Krauspe (73) concluded that these results suggested that the sensory innervation of the ACL plays an important role in joint stability and maintaining the integrity of the ligament.

A direct reflex arc between the ACL and the hamstring has been proposed (67, 143, 150). Solomonow et al. (142) documented that hamstring EMG activity was significantly increased during low to moderate load application to the ACL. Similarly, Johansen (67) found that knee joint afferents frequently and powerfully influence fusimotor neurons (γ-motor neurons). Tsuda et al. (150) reported decreased muscle activity in the anesthetized knee due to removal of impulses from the neural elements of the ACL. In contrast, Grabiner et al. (46) refutes a direct reflex arc. During isometric extension exercise of the knee these investigators found no significant difference in hamstring excitation with regard to muscle or joint angle.

Muscle Receptors

The Muscle Spindle. Muscle spindles are elongated structures ranging from 4 to 10 mm in length, and consist of a bundle of specialized muscle fibers (intrafusal muscle fibers) lie in parallel with the fibers of the extrafusal muscle and are classified by the arrangement of their nuclei into nuclear bag fibers and nuclear chain fibers (37, 49, 67, 68, 76, 79, 80, 155). The nuclei of the nuclear bag fibers are clustered in a group, whereas the nuclei of nuclear chain fibers are arranged in a line parallel to the long axis of the muscle fiber. The regions of the fibers on each side of the central region contain a large number of myofibrils and are referred to as the "polar regions" (37). The typical muscle spindle consists of two bag fibers and four chain fibers (37). Central regions of both fiber types are supplied with Type Ia and Type II sensory nerve endings (68, 126, 155). The endings of Type Ia fibers spiral around the intrafusal fibers and the endings of type II fibers consist of a number of branches comprised of end bulbs which adhere closely to the sarcolemma of each fiber.

Polar regions of the intrafusal fibers are supplied with motor endings from A-beta (A-β) and A-gamma (A-γ) motor nerve fibers (49, 67, 68, 126, 155). Stimulation of the A-β and A-γ nerves results in contraction of the polar regions, stretching the polar region and exciting the spiral and end bulb nerve endings resulting in sensory discharge via the Type Ia and Type II sensory nerve fibers (9, 49, 67, 68, 126, 155). Type Ia and Type II sensory fibers synapse in the spinal cord directly with A-alpha motor neurons that supply the extrafusal fibers (49, 67, 68, 126, 155). The level of contraction of the extrafusal fibers depends on the level of activation, which depends on the degree of tension in the intrafusal fibers. Under resting conditions there is always a certain amount of tension in the intrafusal fibers, which is caused by activation by the sensory centers of the brain via the A-beta and A-gamma fibers, resulting in a certain amount of tension in the extrafusal muscle fibers (49, 67, 68, 126, 133, 155).

Sensory output from muscle spindles can also be generated by stretching the muscle as a whole, as this will cause stretch of the muscle spindle and excite the spiral and end bulb nerve endings (49, 67, 68, 126, 155). Eccentric muscle contraction is an essential component of normal movements. It is thought that sensory information provided by muscle spindles as a result of stretching during eccentric tension provides a sense of joint position and joint movement, especially during mid-range movements (42, 91, 139). The rate of sensory information generated depends on the muscle tone (resistance to stretch by a relaxed muscle) of the intrafusal fiber, increasing intrafusal fiber muscle tone increases the sensitivity of the spindles to stretch and increases of spindle sensitivity cause an increase in intrafusal fiber muscle tone (37). Rapid stretch results in reflex contraction of muscles, which is caused by the spindle afferent to muscle efferent loop, to prevent subluxation of associated joints. These stretch reflexes are important in protecting joints from injury (54, 60, 94, 108, 133). Excitation of muscle spindles appears to be primarily responsible for initiating reflexive muscle contraction (50, 67, 93).

The Golgi Tendon Organ. Golgi Tendon Organ's (GTO) are located at the junction between the muscle tendon and the extrafusal fibers (musculotendinous junction) and are said to be in series with the extrafusal fibers (68, 126, 142, 155). Golgi Tendon Organs are simple receptor organs containing a single afferent connection and no efferent connections. The terminal sensory organ of the afferent neuron of the GTO is located within a capsule and branches to encircle several strands of collagen in the musculotendinous junction (37). Each tendon organ is innervated by a single Group Ib axon and is attached to 10 to 20 muscle fibers (68, 155).

When a muscle and its associated connective tissue are stretched either passively or actively the strands of collagen pinch and excite the Group Ib afferent. Given the means by which the GTO is activated, it is commonly described as a monitor of muscle force (37). The level of force needed to active a GTO is dependent on the mode of activation. Passive stretching requires greater force than does active stretching to active the tendon organ (37, 68, 155).

Proprioception Assessment Techniques

The most common method for testing knee proprioception involves a seated (open-chain) position (9, 13, 16, 76-80, 112, 124, 138). However, some previous studies examining the effect of fatigue have tested proprioception in an open-chain position (100, 104, 105, 128-130, 139). Given that the majority of ligament injuries at the knee occur with the foot in contact with the ground and the knee internally or externally rotating around a fixed foot, it would seem that the most appropriate means for proprioception testing would be in a closed kinetic chain position. Recent studies, however, indicate a strong positive correlation between open and closed-kinetic chain measurement techniques (90, 112).

Procedures for the assessment of joint movement and position sensibility have been well documented (9, 16, 20, 27, 31, 52-54, 58, 59, 65, 74, 76-81, 90, 100, 104, 105, 111, 112, 124, 128-130, 139). Barrack and Skinner (9, 139) originally described assessment techniques for both phenomenons. Joint movement sensibility is assessed by measuring threshold to detection of passive movement (TTDPM). Joint position sense is determined using either reproduction of passive positioning (RPP) or reproduction of active positioning (RAP).

Measurement of joint movement sensibility typically requires the subject to determine when a slow (0.5 degrees per second) angular displacement of the joint has occurred. The slow speed of movement is an attempt to eliminate sensory information from muscle receptors (9, 76-81, 128-130, 139). Movement is typically restricted to agonist-antagonist patterns (i.e. – flexion and extension). In an attempt to eliminate external stimuli, the subject is blindfolded to eliminate visual cues, wears headphones that play "white noise" to eliminate auditory cues, and wears a pneumatic compression boot to reduce the activation of sensory receptors in the skin (9, 76-81, 128-130, 139). Studies indicate that kinesthetic awareness in the knee is enhanced near the limits of the range of motion (9, 139). These findings parallel neurophysiological studies which indicated the existence of a disproportionate distribution of sensory receptors at ligament attachment sites and that these receptors produce a maximal discharge at the end limits of motion (9).

Joint position sensibility is assessed by having the subject's ability to reproduce a presented joint angle (9, 76-81, 128-130, 139). This assessment can be performed passively or actively, and in an open- or closed-kinetic chain motion. The subject is blindfolded and is instructed to hold the presented position for 2-4 seconds before returning to the start position, the subject is then instructed to reproduce the angle he/she was presented (9, 76-81, 128-130, 139). The measures of RPP and RAP stimulate sensory receptors within the joint and the muscle. These measures provide a more functional assessment of the afferent pathways (76-81, 128-130).

Debate has arisen concerning whether testing for position sensibility should be performed in a closed- or open-kinetic chain position. Previous studies have found that testing proprioception in a weight bearing (closed-kinetic chain) position improves the accuracy of reproducing joint angles compared to the non-weight-bearing position (open-kinetic chain) (59, 100). Others have found sensation of limb position to be more accurate with an active muscle contraction (74). Some investigators have also suggested that open-kinetic chain assessment appears to isolate the cutaneous and joint receptors, whereas closed-kinetic chain assessment may isolate the muscle mechanoreceptors (74, 90). Higgins and Perrin (59) examined joint reproduction sensibility in weight bearing and non-weight bearing test situations. The results indicated no difference in position reproducibility sense between testing situations.

Fatigue

Fatigue has been defined as an inability to maintain a given exercise intensity (24) or as a failure to maintain the required amount of force (47). These definitions, however, imply that fatigue is an event that occurs at a specific point in time and fails to consider changing conditions within the muscle that ultimately lead to a point of failure (44, 97). It is possible to have muscle fatigue without impaired function of the muscle itself (24). Proper functioning of sensory receptors, the central nervous system (CNS), integrating centers, sensory cortex, spinal and α-motor neurons, γ-loops, motor end plates, muscle cells, and the cerebellum are frequently required in the performance of motor tasks (24). These factors may be related to factors specific to the muscle itself (peripheral fatigue) or to factors related to the central nervous system (central fatigue) (97, 130).

Peripheral Fatigue

Fitts (39) states that the primary cause of muscle fatigue in highly trained athletes appears to be peripheral in nature. Peripheral fatigue involves the function of the neuromuscular junction and the processes related to excitation-contraction (EC) coupling (39, 40). Several experts have debated the exact location/process within EC coupling that causes muscle fatigue.

The process of EC coupling involves the activation of the surface membrane, propagation of the signal down the T-tubles, bringing the activation into the depths of the cell leading to the release of calcium and finally the activation of the contractile elements (39, 40). The release of inorganic phosphate (92, 130), decreased hydrolysis of ATP (92) and a decreased availability of calcium (24) have been linked to decreased force output of muscle. Shifts in the levels of calcium (40, 47), potassium (24), lactate (33); enzymes, and metabolites (92, 148) have also been thought to play a role in muscle fatigue. Shalin (134) however, has stated that no one element, enzyme, or metabolite can be independently responsible for peripheral fatigue. Mair et al. (95) suggested that fatigue alone does not play a role in muscle injury at any tissue length. These investigators also suggest that fatigued muscles are able to absorb less energy before reaching the degree of stretch that caused injury.

Isokinetic dynamometry has been shown to be an effective means for producing muscle fatigue in isolated muscle groups. Douris (33) found that blood lactate accumulation increased significantly as isokinetic velocity increased. The researcher concluded that increased blood lactate levels found as isokinetic velocity increased may be due to greater recruitment of fast twitch muscle fibers. Studies investigating the measurement of isokinetically induced fatigue have indicated that the use of a muscle fatigue index is the most reliable method for measuring isokinetically induced muscle fatigue (33, 69, 113, 116, 117). Fatigue indexes are determined by dividing the last five torque curves by the first five then multiplying by 100 to obtain a percentage value (33, 69, 113). Douris (33) reported a significant positive correlation between muscle fatigue index and blood lactate. Pincivero et al. (113) found high intraclass correlation coefficients for the fatigue index in the non-dominant leg, but not in the dominant leg.

Central Fatigue

Central fatigue has been identified as a progressive reduction in voluntary drive to motor neurons during exercise (42). Central fatigue appears to be related to afferent and efferent signals to higher brain centers, central command, recruitment of the α-motor neuron pool, and the α-motor nerves themselves (42). Motor cortical output eventually becomes suboptimal for recruitment of motor neurons. At least some of this central fatigue occurs at sites "upstream" from the motor cortex (42).

Belhaj-Saif et al. (10) found that firing frequency during fatigue demonstrated the active participation of the motor cortex in the control of compensation for peripheral adjustments due to muscle fatigue. Taylor et al. (145) found that fatigue altered EMG responses to transcranial stimulation during fatigue suggesting both increased excitation and increased inhibition in the motor cortex. Taylor et al. concluded that since these changes were not affected by manipulation of afferent input, the increased excitation and increased inhibition of the motor cortex must result from intrinsic cortical processes and/or altered voluntary drive to the motor cortex (145).

Electrically induced fatigue has been shown to affect muscle afferents, supraspinal tracts, and motoneurons. Following one minute of electrical stimulation, Loscher et al. (87) demonstrated that even though metabolic stress and contractile fatigue were still present, supraspinal, muscle spindle, and motoneuronal recovery were allowed. Darques and Jammes (32) established the activation of group IV muscle afferents during electrically induced muscle fatigue. These researches concluded that the mechanisms related to muscle afferent stimulation is not solely due to increased extracellular potassium concentrations, but also by the efflux of muscle metabolites present during fatiguing contractions.

During fatiguing isometric contractions, at 30 % of MVC, Loscher et al. (88) found that the excitatory drive to the triceps surae alpha motoneuron pool increased which the investigators proposed was a compensatory mechanism to recruit new unfatigued motor units and/or increase motor unit firing rates. Loscher et al. (88) concluded that muscle twitch at endurance limits and an EMG that does not attain its unfatigued MVC level are strong indicators that central fatigue occurred during a sustained submaximal contraction (88). Macefield et al. (93, 94) found that during an isometric contraction, the discharge frequency of muscle spindle afferents progressively declined as the EMG progressively increased. The researchers concluded that although many factors may contribute to the decline in muscle spindle discharge, the most likely result is a progressive disfacilitation of the alpha motoneuron, which may contribute to the decrease in motor unit firing rates during sustained contractions.

Effect of Fatigue on Proprioception

Studies have suggested that a decrement in proprioception with respect to a loss in joint (58, 74, 104, 105, 128, 130) and/or muscle (11, 32, 74, 94, 130, 132, 139) receptor function may occur with the onset of fatigue. It has been proposed that joint injuries might be more prevalent as a result of a reduction in joint proprioception (9, 20, 21, 78, 79, 81, 82, 138). In general, it is believed that a decline in muscle spindle discharge is accompanied by a progressive increase in EMG, representing a clear dissociation between EMG activity and fusimotor-driven spindle discharge (94).

Electrically induced fatigue in cats has shown increased resting discharge and frequency of discharge to slow stretch and vibration during fatigue in Type Ia and Type II spindle afferents (11). While another study demonstrated depressed or abolished Ib afferent responses during fatigue in static, dynamic, and peak frequency which lasted 10 to 20 seconds indicating slow recovery (11). In human studies, Lattanzio et al. (74) found a decrease in kinesthetic sensibility in males following three maximal fatigue protocols (ramp test, continuous test, and interval test), and during the continuous and interval tests in females. Decreases in joint angle and force proprioception were also found to be significantly impaired following eccentric exercise to fatigue (132). Skinner et al. (139) found a decrease in the ability to reproduce joint angles after a series of interval running sprints to fatigue, suggesting that this decreased ability was either due to a loss of efficiency of muscle spindles, or decreased muscle function. Others, however, have suggested that exercise to fatigue is associated with increased muscle spindle activity in response to stretch or vibration and reduced GTO activity, resulting in unopposed alpha and gamma motor neuron stimulation to motor units (11).

In contrast to the findings that fatigue affects proprioception, Marks and Quinney (100) ascertained that following 20 maximal isokinetic quadriceps contractions in sedentary females, no significant decrease in knee proprioception occurred. However, others have found that isokinetic exercise to fatigue has a significant effect on reproduction sensibility (104, 105, 128, 129, 153). Further, it has been demonstrated that low-intensity work to fatigue diminished proprioceptive acuity, which could lead to impaired motor control further diminishing position sensibility (18).

Studies of hamstring and quadriceps muscle fatigue have shown decreases in peak knee moment at heel strike (107) and delayed muscle activation during fatigue (108). Mair et al. (95) found that fatigued muscles absorb less energy before reaching the degree of stretch of soft tissue that causes injury. Mair et al. indicated a significantly decreased force to failure at 50 percent fatigue at isokinetic velocities of 1 and 10 $cm \cdot s^{-1}$, which the investigators concluded was a sufficient level of fatigue to interfere with the storage and retrieval of elastic energy (95).

During fatigue, biodynamical compensations in the mechanical properties of the knee extensor musculature, as evidenced by differences in knee kinematics and muscle activation times, may occur to enhance knee stability (108). Evidence suggests that fast-twitch muscle fibers have a greater Type Ia afferent innervation as compared to slow-twitch fibers (24, 61, 87, 94, 97, 130, 139, 141, 148, 163). This finding, along with the fact that fast-twitch fibers fatigue faster, would seem to suggest that as muscle fatigue increases there will be a decrease in proprioceptive awareness which could lead to a decrease in efferent responses.

Previous research has also indicated that decrements in proprioceptive acuity play a role in decreased functional ability. Wojtys et al. (160) found a 32.5% increase in anterior tibial translation (ATT) following fatigue. This finding indicated that muscle responses of the knee musculature originating at the spinal cord and cortical level showed significant slowing, and in some cases, an absence of activity following quadriceps and hamstring muscle fatigue. Muscle recruitment patterns have also been shown to be altered following fatigue, particularly in females (128, 160).

Isokinetic Dynamometry

Strength, endurance, and power measurement by isokinetic dynamometry are well documented in the literature (2, 26, 51, 70, 71, 115, 118, 119, 146). Isokinetic measures such as torque, work, and power on the Biodex dynamometer have been found to be highly reproducible. Pincivero et al. (119) found ICC values ranging from r = 0.88 to r = 0.97 and r = 0.82 to r = 0.96 for isokinetic variables measured at $60°\cdot s^{-1}$ and $180°\cdot s^{-1}$ respectively between 2 tests separated by seven days. Gross et al. (51) examined peak extension torque (PET), peak flexor torque (PFT), knee extension angular work (EW), and knee flexion angular work (FW) at $180°\cdot s^{-1}$ on a Biodex isokinetic dynamometer. They obtained ICC values of 0.97 for PET, 0.91 for PFT, 0.97 for EW, and 0.89 for FW.

The influence of hip position and isokinetic velocity on quadriceps and hamstring isokinetic measures has been documented in previous studies (70, 162, 164). Worrell et al.. (164) found higher peak torque values for the quadriceps and hamstrings in a seated position when compared to a supine testing position in both male and female subjects. In a study comparing average torque of the hamstring muscle group in a supine versus prone position it was found that the average torque for the hamstrings was higher in a prone position (162). Comparison between seated, supine, and prone positions indicated that hamstring peak torque is higher when tested in a seated position as opposed to either prone or supine positions (164). Kannus and Beynnon (70) ascertained that peak torque values and angle of peak torque changed with increases in isokinetic velocity. Their results indicated that peak hamstring torque values decreased from 121 ($\pm$ 25) Nm at 60 $°\cdot s^{-1}$ to 88 ($\pm$ 20) Nm at 180 $°\cdot s^{-1}$ for males and from 62 ($\pm$ 19) Nm to 42 ($\pm$ 15) Nm for females at $60°\cdot s^{-1}$ and $180°\cdot s^{-1}$, respectively. Angle of hamstring peak torque was also found to be seven degrees greater for both males and females as speed increased from $60°\cdot s^{-1}$ to $180°\cdot s^{-1}$ (70). Worrell et al. found a similar decrease in peak torque for the hamstring as isokinetic velocity increased (162, 164).

Summary and Conclusion

The function of the knee is to withstand large forces, provide stability to the lower extremity, and afford the lower extremity with a large potential range of motion. The knee is considered the "weak link" in the lower extremity kinetic chain due to the relationship between high anatomical mobility and the high movement demands. Stability of the knee is achieved by both passive (ligaments, joint capsule, and cartilage) and active (musculotendinous) components.

Proprioceptive organs located in musculotendinous structures, ligaments, joint capsules, and skin provide information relative to the joints position and motion in space to the CNS. This information, along with feed-forward mechanisms, helps to regulate muscle responses aimed at protecting the joint from injury.

Several factors which may affect the functioning of proprioceptive organs have been identified and investigated. The role of fatigue on diminished proprioceptive acuity is of great interest given that the onset of fatigue can be altered through training. Maximum levels of fatigue have been found to diminish joint movement and joint position abilities in athletic and non-athletic populations. Additional evidence suggests that low levels of fatigue may affect proprioception in anterior cruciate ligament deficient individuals.

In conclusion, the role of fatigue on proprioceptive abilities is unclear at this time. There is little information available regarding how proprioceptive abilities decline as the amount of exertion increases during exercise. The purpose of the present study is to determine the role of increasing exertion on proprioceptive measures.

CHAPTER 3

METHODOLOGY

Experimental Design

The study utilized a multifactoral repeated measures design. Subjects were assessed on two dependent measures. The dependent variables included passive reproduction of passive positioning (PRPP) and active reproduction of passive positioning (ARPP). The independent variable was muscle fatigue, which was operationalized as a percentage of peak isokinetic hamstring muscle torque (10%, 30%, and 50% below peak hamstring torque). Testing of the dependent measures were conducted prior to (pre-test) and following (post-test) the exercise intervention. Change scores for the dependent variables were calculated and used in further statistical manipulations. Exercise intervention and testing of the two dependent measures were conducted using a counterbalanced design to control the occurrence of a learning effect on the outcomes of the study.

Subject Characteristics

Subjects for this study included 10 male (20.8 ± 1.1 years of age, 190.0 ± 8.9 cm, 91.1 ± 11.9 kg, and 12.30 ± 3.6 years in sport) and 10 female (19.5 ± 1.2 years of age, 166.1 ± 6.9 cm, 64.5 ± 7.1 kg, and 12.3 ± 2.0 years in sport) volunteers. Subjects were recruited from NCAA Division III soccer and basketball athletes. Individuals with a history of cardiovascular disease, diabetes, hypertension, pregnant and/or orthopedic pathology or injury of the knee were excluded from participating in this study. Written informed consent was obtained from each subject in accordance with the Institutional Review Board at California Lutheran University and the University of Pittsburgh (Appendix A).

Isokinetic Exercise Procedures

Fatigue of the quadriceps and hamstring muscle groups of the subject's non-dominant leg was induced utilizing a Biodex System 3 Isokinetic Dynamometer (Biodex Medical Inc., Shirley, NY, U.S.A.). The decision to use the non-dominant leg for all testing procedures was made due to results of earlier work showing that the non-dominant leg is more reliable in similar measurements than the dominant leg (114).

Subjects performed the isokinetic exercise protocol in a seated position with knees and hips at 90-degree angles on the Biodex dynamometer chair. Subjects were secured to the chair by means of thigh, pelvic, and torso straps to minimize extraneous body movements. Subjects crossed their arms across their chest to further prevent extraneous movements from the upper extremity. The lateral femoral epicondyle of the test leg was used to align the axis of rotation of the knee joint with the axis of rotation of the dynamometer resistance adapter. The resistance adapter was strapped into placed approximately three cm above the medial malleolus. Gravity correction was obtained by measuring the torque with the knee in a relaxed state at zero degrees of flexion. The Biodex System 3 Advantage Software Program (version 3.2, Biodex Medical Inc., Shirley, NY, U.S.A.) automatically adjusted torque values for gravity. Calibration of the Biodex dynamometer was performed prior to each testing session according to the specifications outlined in the manufacturer's user manual (15).

Following the setup procedures, isokinetic exercise was performed through an angular range of motion of 90 degrees. Motion stops were set at 0 degrees and 90 degrees of knee flexion. During the testing procedure, the cushion setting on the control panel for the ends of range of motion was set to their lowest (hard) setting in order to reduce the effect of limb deceleration on the reciprocal motion. Reciprocal concentric isokinetic knee extension and flexion was performed at the pre-set angular velocities of 90 degrees$\cdot s^{-1}$ for 10 repetitions, 180 degrees$\cdot s^{-1}$ for 15 repetitions, 240 degrees$\cdot s^{-1}$ for 20 repetitions, and 300 degrees$\cdot s^{-1}$ for 25 repetitions. Prior to each set, subjects performed 5 sub-maximal and 2-3 maximal repetitions for warm up and familiarization purposes. A rest period of 40 seconds was provided between each of the four sets (115). Presentation of isokinetic angular velocities was randomized and counter-balanced. Following a rest period of 40 seconds, subjects then performed the reciprocal flexion and extension movement at 180 degrees$\cdot s^{-1}$ until the hamstring (flexion movement) peak torque value fell to 90%, 70%, or 50% of the subject's peak isokinetic torque for three consecutive repetitions. Peak isokinetic torque values were determined by selecting the highest torque value during the first five contractions for each trial (33, 100, 116, 117, 139).

Ratings of perceived exertion (RPE) were collected during the isokinetic exercise protocol. Subjects were asked to determine their level of exertion following the exertion protocol to see if their level of perceived exertion matched the level of physiological exertion demonstrated by the decrease in isokinetic torque values. Subjects were read a script prior to testing as outlined in the American College of Sports Medicine's Guidelines for Exercise Testing and Prescription (2). Please see Appendix B for a copy of the script and data recording sheet.

Proprioception Testing Procedures

Immediately prior to and following each isokinetic fatigue session, subjects were tested on one of the dependent measures (PRPP or ARPP). Testing of these measures was conducted using the

Biodex isokinetic dynamometer. Subjects were blindfolded during testing to eliminate any visual cues related to joint position.

Passive Reproduction of Passive Positioning Protocol

Testing for PRPP was conducted using the Biodex isokinetic dynamometer. The subject's leg was placed at an inital angle of 90 degrees of knee flexion for each trial. The subject's leg was then passively moved to the test angle of 20 degrees of knee flexion by the examiner at an angular velocity of 10 degrees$\cdot s^{-1}$. Subjects concentrated on the sensation of the presented angle (20 degrees of knee flexion) for 3 seconds. The subject's leg was then returned passively to the starting position by the examiner. Following a three second rest period the dynamometer passively moved the subject's leg at one of the test velocities (60 degrees$\cdot s^{-1}$, 90 degrees$\cdot s^{-1}$, or 120 degrees$\cdot s^{-1}$). The subject attempted to stop the dynamometer movement at the presented joint angle (20 degrees of knee flexion) before the dynamometer initiated the flexion movement at the end of the range of motion. Once the subject felt the test leg was in the position of the presented angle, the subject depressed the "hold/resume" switch preventing the dynamometer from further movement. If the subject failed to stop the dynamometer movement prior to moving back into flexion, the investigator recorded the end range position (0 degrees) for that trial. The Biodex System 3 software interface recorded the absolute angular difference (AAD) between the presented and reproduced angles. The investigator also recorded the actual angle of detection in order to determine if the subjects were stopping the movement prior to, or after, the test angle (Appendix C). Each subject performed three trials at each angular velocity. The three values were averaged and the average AAD was used for further analysis (57).

Active Reproduction of Passive Positioning Protocol

Testing of ARPP was conducted using the Biodex isokinetic dynamometer. Subjects were blindfolded to eliminate visual cues related to joint position. The subject's leg was placed at a starting angle of 60 degrees of knee flexion for each trial. The subject's leg was then passively moved to one of the test angles (45 degrees, 30 degrees, or 15 degrees of knee flexion) by the examiner. Subjects concentrated on the sensation of the presented angle for three seconds. The subject's leg was then returned passively to a different position than the starting position by the examiner. Following a three second rest period the subject attempted to actively reproduce the presented joint angle. Once the subject felt the test leg was in the position of the presented angle the subject depressed the hold/resume switch preventing the dynamometer from further movement. The Subject had 5 seconds to reproduce the presented angle.

The Biodex System 3 software package recorded and stored the absolute angular difference (AAD) between the presented and reproduced angles. Each subject performed three trials at each angle and the average of the trials was recorded for statistical interpretation (57).

Test Procedures

Pre-testing evaluation was conducted in the Athletic Training Lab at least 24 hours prior to the first testing session. During the initial pre-testing evaluation, a medical history and demographic information (mass, height, age, and years in sport) were obtained. Leg dominance was determined at this time by asking the subject "if you were to kick a soccer ball, with which leg would you kick the ball?" The leg indicated as the non-dominant (non-kicking) leg served as the test leg for all testing sessions. Subjects were also introduced to the Biodex System 3 Isokinetic machine and the testing procedure to be used in the study at this time. During the initial pre-testing evaluation, each subject was also introduced to the testing procedures for PRPP and ARPP. Subjects began testing sessions at least 24 hours following the pre-testing evaluation.

Upon entering the Athletic Training Lab for each testing session, subjects were provided 5 to 10-minutes for stretching prior to testing. Following stretching, the subject was pre-tested for one of the PRPP or ARPP measures. Subjects then performed knee extension and flexion concentric isokinetic exercise as described above until torque output fell below the predetermined percentage of peak torque for three consecutive repetitions. Subjects were then post-tested on the same PRPP or ARPP measure as the pre-test following the isokinetic exercise session. Following testing of the first independent measure, subjects were given a 20 minute rest period. Following the rest period, subjects were pre-tested on a different PRPP or ARPP measure. The subject then performed knee extension and flexion concentric isokinetic exercise until torque output fell below the predetermined percentage of peak torque for three consecutive repetitions at the same angular velocity used in the first session. Following the second isokinetic exercise session, subjects were post-tested again on the same PRPP or ARPP measure used in the pre-test. This procedure was repeated for a third exercise session.

Exercise sessions were separated by a minimum of 48 hours to allow complete recovery. Table 1 provides an example of one testing protocol used for one subject. Exercise intensity was counter-balanced between sessions. Testing of PRPP and ARRP was counterbalanced between trials and sessions in order to decrease the chance of a learning effect on the results of each testing session. Subjects were also asked to refrain from participating in any lower extremity exercise routines for the remainder of the study.

Table 1. Sample testing protocol

Test Session	% Peak Isokinetic Torque	Test Variable #1	Test Variable #2	Test Variable #3
1	70%	ARPP 45°	PRPP 90°/s	ARPP 30°
2	90%	PRPP 120°/s	ARPP 30°	PRPP 60°/s
3	50%	ARPP 15°	ARPP 45°	PRPP 90°/s
4	90%	ARPP 15°	ARPP 45°	PRPP 90°/s
5	70%	ARPP 15°	PRPP 120°/s	PRPP 60°/s
6	50%	PRPP 120°/s	ARPP 30°	PRPP 60°/s

Data Analysis

Mean AAD values for ARPP and PRPP were used for further data analyses. Analysis of variance with repeated measures for exertion level at ARPP angles of 15°, 30°, and 45° by gender and exertion level at PRPP of $60°\cdot s^{-1}$, $90°\cdot s^{-1}$, and $120°\cdot s^{-1}$ by gender were utilized to determine statistical significance. Ratings of perceived exertion were analyzed using a two way analysis of variance (RPE x gender). All tests of significance were carried out at an alpha level of $P < 0.05$. Bonferroni pairwise comparisons were used to determine which findings are significant at the 0.05 level. Reliability of the PRPP and ARPP were estimated by computing Chronbach's alpha coefficients for pre-exertion and post-exertion test values.

CHAPTER 4

RESULTS

Rating of Perceived Exertion

Mean RPE values for exertion levels of 10%, 30%, and 50% below maximum isokinetic hamstring torque for males and females are presented in Table 2. Two-way analysis of variance demonstrated a statistically significant main effect for RPE ($F_{2,36} = 328.06$, $p = 0.00$). Post-hoc analysis revealed a statistically significant difference between RPE at 10% and 30% exertion ($F_{1,18} = 110.05$, $p = 0.00$) and between RPE at 30% and 50% exertion ($F_{1,18} = 447.426$, $p = 0.00$) A statistically significant main effect was also found for gender ($F_{1,18} = 5.92$, $p = 0.03$). Post-hoc analysis revealed a statistically significant difference for gender at an exertion level of 30% below maximum isokinetic hamstring torque ($t = 2.80$, $p = 0.03$). A statistically significant interaction effect was not found between RPE and gender ($F_{2,36} = 1.60$, $p = 0.22$). (Table 3).

Table 2. RPE at exertion levels of 10%, 30%, and 50% below max. hamstring torque.

% Exertion		N	Mean	Std.Dev.	Std.Error
10%	0	10	10.64	1.46	0.46
	1	10	9.70	1.25	0.40
	Total	20	10.17	1.41	0.32
30%	0	10	14.06	1.05	0.33
	1	10	12.77	1.02	0.32
	Total	20	13.42	1.21	0.27
50%	0	10	18.00	0.75	0.24
	1	10	17.77	0.80	0.25
	Total	20	17.88	0.77	0.17

0 = Male, 1 = Female

Table 3. Two-Way ANOVA Table for RPE at 10%, 30%, and 50% max. hamstring torque.

	Df	Mean Square	F	Sig.
Gender	1	3.371	5.923	.026
Error (Gender)	18			
RPE	2	299.961	328.055	.000
RPE*Gender	2	1.460	1.597	.217
Error (RPE)	36	.914		

Passive Reproduction of Passive Positioning

Table 4 presents mean change score values for PRPP absolute angular difference by test angular velocity for males and females. Figures 3-5 display the mean change AAD scores for PRPP by gender at joint angular velocities of $60°\cdot s^{-1}$, $90°\cdot s^{-1}$, and $120°\cdot s^{-1}$.

Table 4. Mean Change Scores for PRPP Absolute Angular Difference*

Joint Angular Velocity	Gender	N	Exertion Level (% below maximum hamstring torque)					
			10%		30%		50%	
			Mean	Std. Dev	Mean	Std. Dev	Mean	Std. Dev
$60°\cdot s^{-1}$	Male	10	-0.51	2.45	-0.82	2.12	-0.31	4.10
	Female	10	-0.33	2.53	0.56	2.89	-1.07	2.22
	Total	20	-0.42	2.43	-0.13	2.57	-0.69	3.23
$90°\cdot s^{-1}$	Male	10	-0.32	2.82	-0.27	2.00	1.14	4.22
	Female	10	-1.09	4.15	-1.38	4.44	-1.08	3.45
	Total	20	-0.75	3.47	-0.83	3.40	0.03	3.92
$120°\cdot s^{-1}$	Male	10	-0.09	2.59	-0.66	5.07	1.40	5.71
	Female	10	-0.93	3.76	-4.13	3.41	-1.38	4.44
	Total	20	-0.51	3.17	-2.40	4.57	0.01	5.18

*(-) values indicate a decrease in pre-test to post-test (pre-post = change score) values

PRPP at Angular Velocity of $60°\cdot s^{-1}$

Reliability for PRPP at $60°\cdot s^{-1}$ was estimated by computing Chronbach's alpha coefficients for pre-exertion and post-exertion test values. As can be seen in Table 5 these coefficients ranged from -0.08 to 0.74 and had a mean value of 0.53. Maulchy's Test of Sphericity ($\varepsilon = 0.85$, $p = 0.24$) revealed that the assumption of sphericity had been met for PRPP at an angular velocity of

$60°\cdot s^{-1}$ between exertion levels of 10%, 30%, and 50% below maximum hamstring torque. Repeated measures analysis of variance for PRPP at $60°\cdot s^{-1}$ (Table 6) did not reveal a statistically significant main effect for exertion ($F_{2,36} = 0.25$, $p = 0.78$). Neither a statistically significant main effect for gender ($F_{1,18} = 0.10$, $p = 0.76$), nor an interaction effect between exertion and gender for PRPP at $60°\cdot s^{-1}$ ($F_{2,36} = 0.90$, $p = 0.41$) were found.

Table 5. Measurement Reliability of PRPP at $60°\cdot s^{-1}$

% of Maximum Exertion		F	p	Chronbach' Alpha
10% below max	pre-test	1.61	0.21	0.66
	Post-test	0.52	0.60	0.65
30% below max	pre-test	0.29	0.75	0.73
	Post-test	4.66	0.02	0.74
50% below max	pre-test	0.11	0.90	0.47
	Post-test	0.71	0.50	-0.08

Table 6. PRPP at $60°\cdot s^{-1}$ Repeated Measures ANOVA Table

Source	df	Mean Square	F	Sig.
Gender	1	1.08	0.10	0.76
Error (Gender)	18	10.73		
Exertion	2	1.58	0.25	0.78
Exertion * Gender	2	5.77	0.90	0.41
Error (Exertion)	36	6.39		

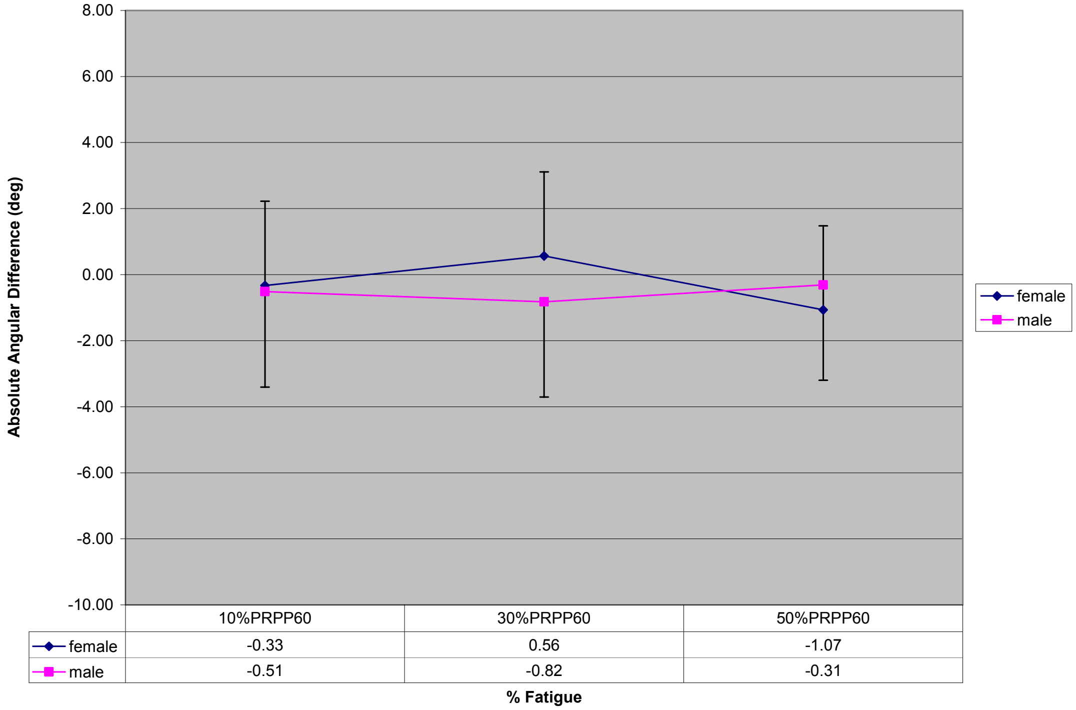


Figure 3. Absolute Angular Difference for PRPP at $60°\cdot s^{-1}$.
(Values in lower panel are means)

<u>PRPP at Angular Velocity of $90°\cdot s^{-1}$</u>

Reliability for PRPP at $90°\cdot s^{-1}$ was estimated by computing Chronbach's alpha coefficients for pre-exertion and post-exertion test values. As can be seen in Table 7 these coefficients ranged from 0.43 to 0.67 and had a mean value of 0.56. Maulchy's Test of Sphericity ($\varepsilon = 0.99$, $p = 0.93$) revealed that the assumption of sphericity had been met for PRPP at an angular velocity of $90°\cdot s^{-1}$ between exertion levels of 10%, 30%, and 50% below maximum hamstring torque. Repeated measures analysis of variance for PRPP at $90°\cdot s^{-1}$ (Table 8) did not reveal a statistically significant main effect for exertion ($F_{2,36} = 0.30$, $p = 0.74$). Neither a statistically significant main effect for gender ($F_{1,18} = 2.65$, $p = 0.12$), nor an interaction effect between exertion and gender for PRPP at $90°\cdot s^{-1}$ ($F_{2,36} = 0.20$, $p = 0.82$) were found.

Table 7. Measurement Reliability of PRPP at $90°\cdot s^{-1}$

% of Maximum Exertion		F	p	Chronbach's Alpha
10% below max	pre-test	0.20	0.82	0.64
	Post-test	1.77	0.18	0.67
30% below max	pre-test	0.41	0.66	0.46
	Post-test	2.17	0.13	0.66
50% below max	pre-test	0.23	0.79	0.43
	Post-test	0.43	0.66	0.49

Table 8. PRPP at $90°\cdot s^{-1}$ Repeated Measures ANOVA Table

Source	df	Mean Square	F	Sig.
Gender	1	28.02	2.65	0.12
Error (Gender)	18	10.57		
Exertion	2	4.29	0.30	0.74
Exertion * Gender	2	2.88	0.20	0.82
Error (Exertion)	36	14.35		

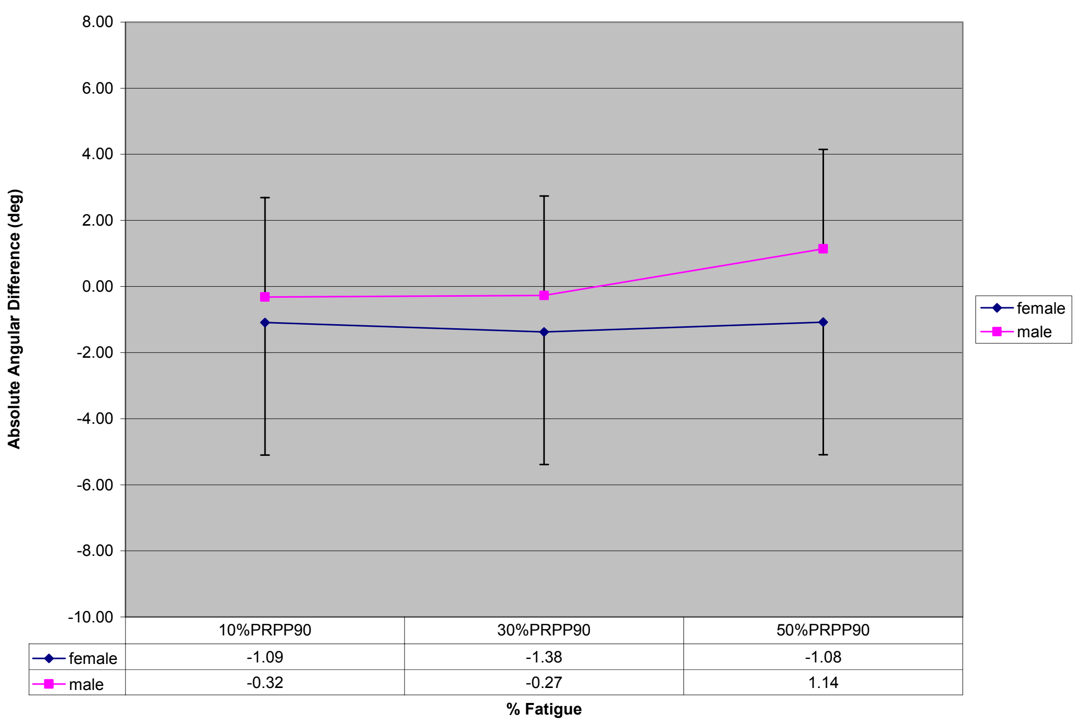


Figure 4. Absolute Angular Difference for PRPP at $90°\cdot s^{-1}$.
(Values in lower panel are means)

PRPP at Angular Velocity of $120°·s^{-1}$

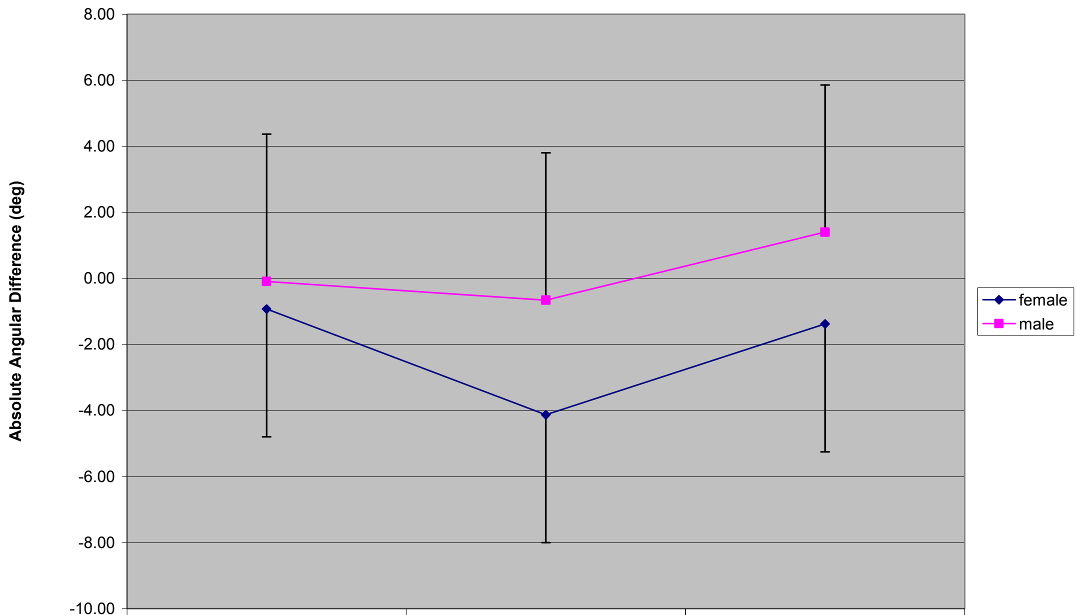
Reliability for PRPP at $120°·s^{-1}$ was estimated by computing Chronbach's alpha coefficients for pre-exertion and post-exertion test values. As can be seen in Table 9 these coefficients ranged from -0.42 to 0.56 and had a mean value of 0.31. Maulchy's Test of Sphericity ($\varepsilon = 0.76$, $p = 0.10$) revealed that the assumption of sphericity had been met for PRPP at an angular velocity of $120°·s^{-1}$ between exertion levels of 10%, 30%, and 50% below maximum hamstring torque. Repeated measures analysis of variance for PRPP at $120°·s^{-1}$ (Table 10) did not reveal a statistically significant main effect for exertion ($F_{2,36} = 1.53$, $p = 0.23$). A statistically significant main effect for gender ($F_{1,18} = 6.22$, $p = 0.02$) was found. Pairwise comparisons between gender for PRPP at $120°·s^{-1}$ showed a mean difference between males and females of 2.36 ($p = 0.02$). A statistically significant interaction effect between exertion and gender for PRPP-$120°·s^{-1}$ was not found ($F_{2,36} = 0.45$, $p = 0.64$).

Table 9. Measurement Reliability of PRPP at $120°·s^{-1}$

% of Maximum Exertion		F	p	Chronbach's Alpha
10% below max	pre-test	3.10	0.06	0.33
	post-test	0.48	0.62	0.56
30% below max	pre-test	0.91	0.41	-0.42
	post-test	0.36	0.70	0.31
50% below max	pre-test	1.61	0.21	0.55
	post-test	2.00	0.15	0.55

Table 10. PRPP at $120°·s^{-1}$ Repeated Measures ANOVA Table

Source	df	Mean Square	F	Sig.
Gender	1	83.70	6.22	0.02
Error (Gender)	18			
PRPP - 120°/s	2	32.04	1.53	0.23
PRPP - 120°/s * Gender	2	9.32	0.446	0.64
Error (PRPP - 120°/s)	36	20.90		



	10%PRPP120	30%PRPP120	50%PRPP120
female	-0.93	-4.13	-1.38
male	-0.09	-0.66	1.40

% Fatigue

Figure 5. Absolute Angular Difference for PRPP at $120°\cdot s^{-1}$.
(Values in lower panel are means)

Active Reproduction of Passive Positioning

Table 11 presents mean change score values for ARPP absolute angular difference by test angle for males and females. Figures 6-8 display the mean change AAD scores for ARPP by gender at joint angles of 15°, 30°, and 45° of knee flexion.

Table 11. Mean Change Scores for ARPP Absolute Angular Difference*

Joint Angle	Gender	N	Exertion Level (% below maximum hamstring torque)					
			10%		30%		50%	
			Mean	Std. Dev	Mean	Std. Dev	Mean	Std. Dev
15°	Male	10	-0.09	0.74	-0.71	2.20	-0.61	1.45
	Female	10	1.15	2.24	0.60	2.24	0.22	1.70
	Total	20	0.53	1.74	-0.06	2.26	-0.20	1.59
30°	Male	10	-0.47	2.57	0.05	1.90	-1.08	2.84
	Female	10	-0.88	2.76	-0.48	1.80	-0.89	2.97
	Total	20	-0.68	2.60	-0.22	1.82	-0.99	2.83
45°	Male	10	-0.81	1.21	-0.20	1.71	-0.16	1.45
	Female	10	-1.19	1.99	-1.14	2.63	-1.07	1.91
	Total	20	-1.00	1.61	-0.67	2.21	-0.62	1.71

*(-) values indicate a decrease in pre-test to post-test (pre – post = change score)values

ARPP at Angular Position of 15°

Reliability for ARPP at 15° was estimated by computing Chronbach's alpha coefficients for pre-exertion and post-exertion test values. As can be seen in Table 12 these coefficients ranged from 0.28 to 0.80 and had a mean value of 0.57. Maulchy's Test of Sphericity ($\varepsilon = 0.80$, $p = 0.14$) revealed that the assumption of sphericity had been met for ARPP at a knee joint angle of 15° between exertion levels of 10%, 30%, and 50% below maximum hamstring torque. Repeated measures analysis of variance for ARPP at 15° (Table 13) did not reveal a statistically significant main effect for exertion ($F_{2,36} = 0.84$, $p = 0.44$). A statistically significant main effect for gender ($F_{1,18} = 6.10$, $p = 0.02$) was found. Pairwise comparisons between gender for ARPP at 15° showed a mean difference between males and females of -1.13 ($p = 0.02$). A statistically significant interaction effect between exertion and gender for ARPP at 15° was not found ($F_{2,36} = 0.10$, $p = 0.91$).

Table 12. Measurement Reliability of ARPP at 15°

% of Maximum Exertion		F	p	Chronbach's Alpha
10% below max	pre-test	1.15	0.23	0.80
	post-test	2.26	0.12	0.67
30% below max	pre-test	2.71	0.08	0.41
	post-test	3.34	0.05	0.80
50% below max	pre-test	0.85	0.44	0.47
	post-test	2.19	0.13	0.28

Table 13. ARPP at 15° Repeated Measures ANOVA Table

Source	df	Mean Square	F	Sig.
Gender	1	19.00	6.10	0.02
Error (Gender)	18	3.11		
Exertion	2	2.97	0.84	0.44
Exertion * Gender	2	0.34	0.10	0.91
Error (Exertion)	36	3.54		

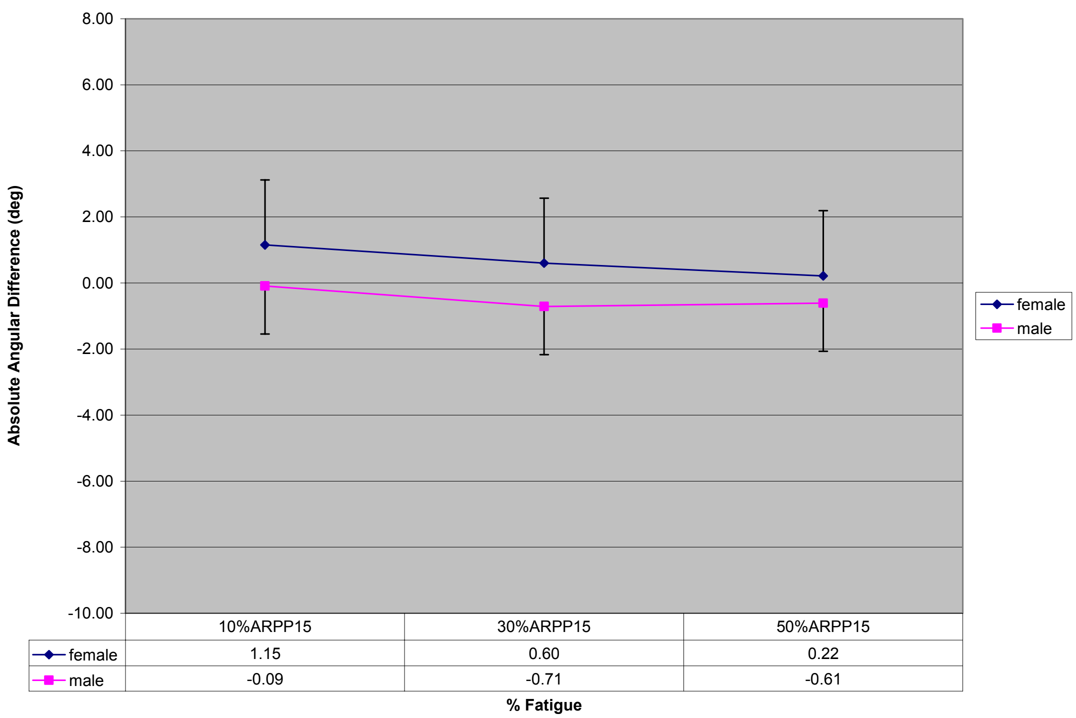


Figure 6. Absolute Angular Difference for ARPP at 15°.
(Values in lower panel are means)

<u>ARPP at Angular Position of 30°</u>

Reliability for ARPP at 30° was estimated by computing Chronbach's alpha coefficients for pre-exertion and post-exertion test values. As can be seen in Table 14 these coefficients ranged from 0.56 to 0.83 and had a mean value of 0.73. Maulchy's Test of Sphericity ($\varepsilon = .75$, $p = 0.09$) revealed that the assumption of sphericity had been met for ARPP at a knee joint angle of 30° between exertion levels of 10%, 30%, and 50% below maximum hamstring torque. Repeated measures analysis of variance for ARPP at 30° (Table 15) did not reveal a statistically significant main effect for exertion ($F_{2,36} = 0.66$, $p = 0.53$). Neither a statistically significant main effect for gender ($F_{1,18} = 0.10$, $p = 0.76$), nor an interaction effect between exertion and gender for ARPP at 30° ($F_{2,36} = 0.16$, $p = 0.85$) was not found.

Table 14. Measurement Reliability of ARPP at 30°

% of Maximum Exertion		F	p	Chronbach's Alpha
10% below max	pre-test	1.15	0.33	0.79
	post-test	0.95	0.39	0.69
30% below max	pre-test	2.91	0.07	0.79
	post-test	0.70	0.50	0.56
50% below max	pre-test	4.50	0.02	0.72
	post-test	0.38	0.69	0.83

Table 15. ARPP at 30° Repeated Measures ANOVA Table

Source	df	Mean Square	F	Sig.
Gender	1	0.94	0.10	0.76
Error (Gender)	18	9.83		
Exertion	2	3.00	0.66	0.53
Exertion * Gender	2	0.74	0.16	0.85
Error (Exertion)	36	4.58		

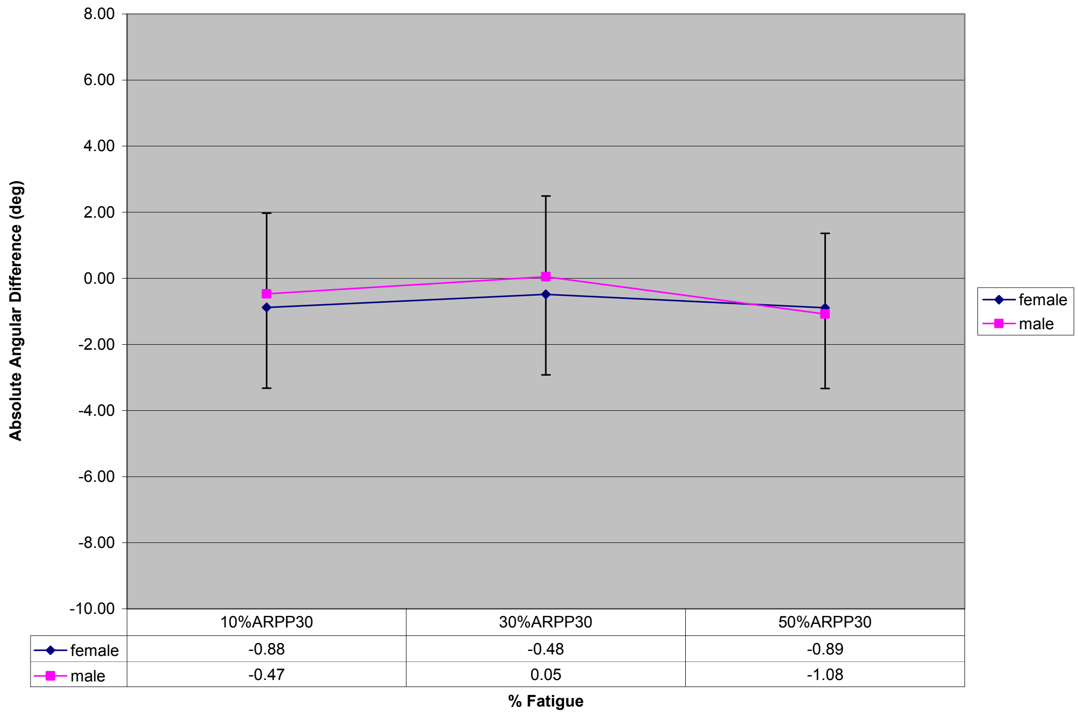


Figure 7. Absolute Angular Difference for ARPP at 30°.
(Values in lower panel are means)

ARPP at Angular Position of 45°

Reliability for ARPP at 45° was estimated by computing Chronbach's alpha coefficients for pre-exertion and post-exertion test values. As can be seen in Table 16 these coefficients ranged from 0.56 to 0.90 and had a mean value of 0.79. Maulchy's Test of Sphericity ($\varepsilon = 0.78$, $p = 0.13$) revealed that the assumption of sphericity had been met for ARPP at a knee joint angle of 45° between exertion levels of 10%, 30%, and 50% below maximum hamstring torque. Repeated measures analysis of variance for ARPP at 45° (Table 17) did not reveal a statistically significant main effect for exertion ($F_{2,36} = 0.22$, $p = 0.81$). Neither a statistically significant main effect for gender ($F_{1,18} = 3.26$, $p = 0.09$), nor an interaction effect between exertion and gender for ARPP at 45° ($F_{2,36} = 0.13$, $p = 0.88$) were found.

Table 16. Measurement Reliability of ARPP at 45°

% of Maximum Exertion		F	p	Chronbach's Alpha
10% below max	pre-test	1.26	0.30	0.86
	post-test	0.51	0.61	0.81
30% below max	pre-test	3.10	0.06	0.56
	post-test	0.14	0.87	0.79
50% below max	pre-test	0.76	0.47	0.84
	post-test	2.81	0.07	0.90

Table 17. ARPP at 45° Repeated Measures ANOVA Table

Source	df	Mean Square	F	Sig.
Gender	1	8.29	3.26	0.09
Error (Gender)	18	2.55		
Exertion	2	0.87	0.22	0.81
Exertion * Gender	2	0.50	0.13	0.88
Error (Exertion)	36	3.97		

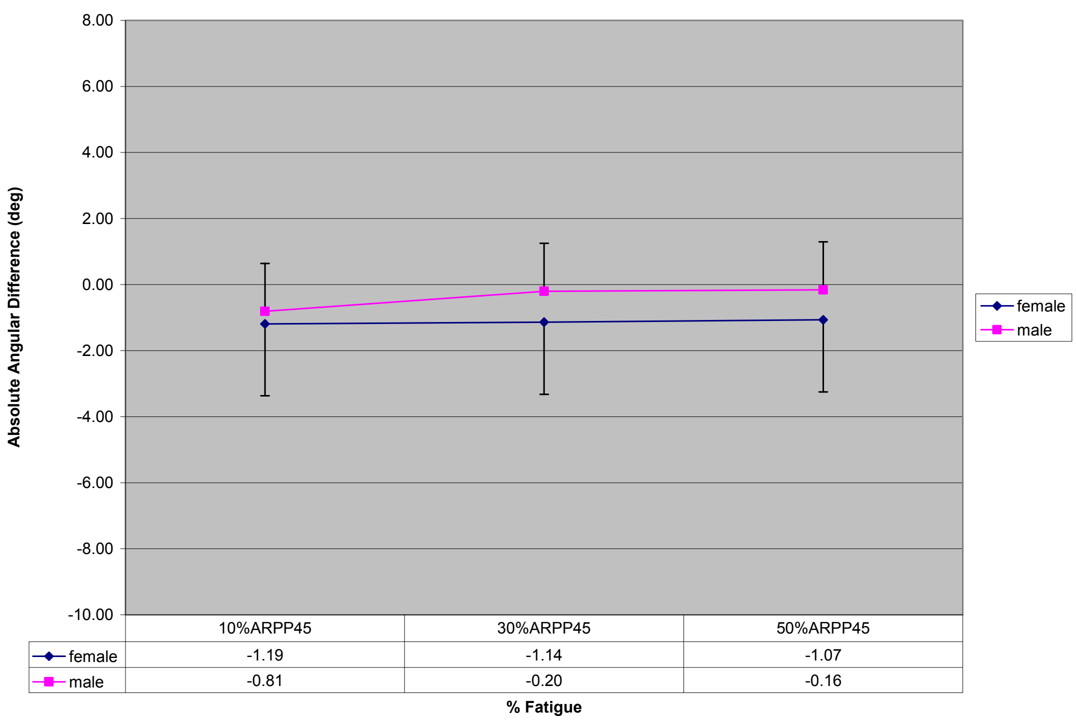


Figure 8. Absolute Angular Difference for ARPP at 45°.
(Values in lower panel are means)

Summary

Chronbach's alpha for PRPP scores indicate these measures should be interpreted cautiously. Active reproduction of passive positioning demonstrated adequate to good reliability at joint angles of 30° and 45°, but poor reliability at a joint angle of 15°. The results of the current study showed a statistically significant gender main effect for PRPP at $120°\cdot s^{-1}$ and for ARPP at a joint angle of 15°. Chronbach's alpha coefficients for those two measures, mean α for PRPP at $120°\cdot s^{-1} = 0.31$ and mean α for ARPP at a joint angle of $15° = 0.57$, indicate that caution must be used when interpreting these findings. Statistically significant interaction effects for PRPP by gender and ARPP by gender were not found. The major findings of this study indicate that increasing levels of exertion do not have a statistically significant effect on either passive or active reproduction of passive positioning ability. Poor Chronbach's alpha for all PRPP measures and ARPP at 15° cause the findings for those measures to be inconsistent.

CHAPTER 5

DISCUSSION AND CONCLUSIONS

The purpose of this study was to examine the influence of increasing levels of physical exertion on passive and active reproduction of passive positioning ability. The results of the current study showed a statistically significant gender main effect for PRPP at $120°\cdot s^{-1}$ and for ARPP at a joint angle of 15°. Chronbach's alpha for those two measures, mean α for PRPP at $120°\cdot s^{-1} = 0.31$ and mean α for ARPP at a joint angle of $15° = 0.57$, indicate that the findings associated with these measures should be interpreted cautiously. Statistically significant interaction effects for PRPP by gender and ARPP by gender were not found. The major findings of this study indicate that increasing levels of exertion do not have a statistically significant effect on either passive or active reproduction of passive positioning ability. Poor Chronbach's alpha coefficients for all PRPP measures and ARPP at 15° indicate the reliability of these findings is not as strong as previously reported. Passive and active reproduction of passive position measures demonstrated large variability. The large variability in these measures may be linked to the generally poor reliability of these measures.

Rating of Perceived Exertion

The results from the present study showed a statistically significant difference for RPE and for gender at an exertion level of 30%. The RPE values reported corresponded to the level expected during the exercise interventions. Although RPE corresponded to the level of expected exertion during the exercise trials, isokinetically induced muscle exertion of the hamstring and quadriceps muscles may be too localized to cause neuromuscular deficits at a level sufficient to create changes in proprioceptive acuity. Subjects indicated their overall feeling of exertion for a given exercise session.

A gender effect for RPE was not found at exertion levels of 10% and 50%. A statistically significant effect for gender was found at an exertion level of 30%. Previous studies have shown differences in RPE values between males and females at maximum exertion (113, 115, 116). However, these studies used untrained subjects. Green et al. (48) did not demonstrate a gender effect during exercise at a moderate work rate. Green et al. indicated that the use of subjects involved in the same sport or activity will result in similar RPE values between gender groups (48). The current study used subjects who participate in soccer and basketball at the collegiate level. The findings from the current study are in agreement with the findings of Green et al. (48).

Passive Reproduction of Passive Positioning

The results from the present investigation demonstrated that isokinetic hamstring exercise to exertion levels of 10%, 30%, and 50% below maximum hamstring torque did not have a significant effect on PRPP ability at angular velocities of $60°\cdot s^{-1}$, $90°\cdot s^{-1}$, and $120°\cdot s^{-1}$. A statistically significant gender main effect was found at an angular velocity of $120°\cdot s^{-1}$.

The apparent lack of a gender difference may be due in part to the poor reliability of these measures. However, other investigators have also failed to show a gender difference for PRPP measures (16, 58, 129). Callaghan, et al. (16) did not find a statistically significant difference in PRPP between males and females. However, these investigators did not report the reliability of the PRPP measure. The finding of a gender effect at an angular velocity of $120°\cdot s^{-1}$ should be interpreted cautiously due to the poor reliability associated with this angular velocity.

To date, no study has examined the effect of increasing levels of exertion on passive reproduction ability at the knee joint. Previous studies have suggested that a decrement in proprioception as measured by a loss in joint and muscle receptor function may occur with the onset of maximum fatigue (128, 130). Bentley (11) suggested that exercise to fatigue is associated with increased muscle spindle activity in response to stretch or vibration and reduced GTO activity, resulting in unopposed alpha and gamma motor neuron stimulation to motor units. Sensory output from muscle spindles can be generated by stretching the muscle as a whole, as this will cause stretch of the muscle spindle and excite the spiral and end bulb nerve endings (49, 67, 68, 126, 155). In general, the faster the rate of stretch, the greater the discharge from the muscle spindles. The current study demonstrated no increase in PRPP for pre to post-test values at all angular velocities and levels of exertion except for angular velocities of $90°·s^{-1}$ and $120°·s^{-1}$ at an exertion level of 50% below maximum hamstring torque. These later angular velocities did not change in post-test reproduction ability. As muscle fatigue increased and the speed of joint motion (stretch) increased, there was no change in pre-test to post-test passive reproduction ability. These results are in agreement with those of Bentley (11).

Sensory input to the CNS from cutaneous and joint proprioceptors may significantly affect passive reproduction ability. McCabe et al. (90) hypothesized that cutaneous, joint, and muscle mechanoreceptors may act independently in transmitting proprioceptive information to the CNS. Cutaneous and joint proprioceptors consist primarily of Ruffini end organs and Pacinian corpuscles, which are sensitive to tension and compression forces respectively and produce discharges at the extreme limits of the joints range of motion (76, 81, 91, 122, 123, 155). The test angle of 20 degrees of flexion for the current study may not have been sufficient to cause a discharge of the receptors located in the joint and joint capsule (46). As such, sensory receptors located in the ligaments and joint capsule of the knee joint may not have played a significant role in position sensation.

The lack of demonstrated reliability for PRPP measures means that the present findings must be interpreted cautiously. Reliability data for these measures is not present in proprioception literature. However, the PRPP test used presently has been used by other investigators with varying findings (16, 27, 90). McCabe, Myers, and Lephart (90) demonstrated moderate correlation coefficients for PRPP (r= 0.52 and r= 0.58). Correlation coefficients, however, are statistical tests used to determine the relation between two or more different measures (heart rate and RPE for example) not measures of the same task. The methods used in the current study were similar to these previous studies in the areas of instrumentation, setup, and movement of the limb into extension. The current study did investigate different velocities of joint movement which was different from those reported previously.

The lack of reliability for PRPP measures may be due to either the reaction or anticipation skill level of the individual subject, or a combination of both factors. Reaction time is an indication of the speed with which one can perceive and respond to the environment (34). Anticipation time requires the subject to coordinate and synchronize a motor response with an external event (34). Birmingham et al. (16) attempted to address the problem of reaction time and PRPP by asking each subject if they were satisfied that their knee position reflected the target angle and allowed the subject to correct the position if desired. These previous investigations do not indicate if corrections were actually made by subjects during passive testing. In the current study, it would seem that both reaction time and anticipation time played a role in PRPP ability as the subjects were required to perceive the appropriate angle and coordinate the appropriate response to stop limb movement. An area of particular interest would seem to be latency time. Latency time is the time between perceiving the stimulus and reacting to it. Subjects in the current study were required to stop dynamometer movement by depressing the hold/resume switch for the dynamometer. The times between sensory receptor activation, position recognition, and depressing the switch were not accounted for in the present study and may have contributed to the decreased reliability of PRPP measures.

Active Reproduction of Passive Positioning

Active reproduction of passive positioning measures has been shown to be a valid and reliable measure when testing absolute error scores (13, 90). The methods used in the current study were similar to these previous studies in the areas of instrumentation, setup, movement of the limb into extension only, and using test angles of 15° and 45°. The current study did investigate an additional test angle, 30° of joint flexion. The lack of demonstrated reliability for ARPP at a joint angle of 15° indicates that the findings for this joint angle should be interpreted cautiously. A possible reason for the decreased reliability at a joint angle of 15° might be that the knee attachment for the Biodex System 3 Isokinetic Device produced 6 N·m of torque at this angle. The torque produced by the knee attachment may have been sufficient to cause a decrease in trial to trial reliability especially following the 50% below hamstring maximum torque exercise sessions. Chronbach's alpha for joint angle of 30° and 45° from the current investigation indicate that these measures have good reliability (mean values = 0.73 and 0.79 respectively).

The apparent lack of a gender difference may be due in part to the poor reliability of these measures. However, other investigators have also failed to show a gender difference in ARPP measures (74, 100, 129). Marks and Quinney, did not find a gender effect for ARPP following isokinetically induced fatigue. The current study failed to demonstrate a gender effect for joint angles of 30° and 50°. The findings from the current study are in agreement with those of Marks and Quinney (100). The gender effect found at 15° should be interpreted cautiously due the the poor reliability of this test angle.

To date, no study has examined the effect of increasing levels of exertion on ARPP at the knee joint. The results from the present investigation demonstrated that isokinetic hamstring exertion at levels of 10%, 30%, and 50% below maximum hamstring torque did not have a significant effect on ARPP ability at joint angles of 45°, 30°, and 15° of knee flexion. In an earlier study of the effect fatigue has on knee joint proprioception, Lattanzio et al. (74) found a decrease in kinesthetic sensibility in males following three maximal fatigue protocols (ramp test, continuous test, and interval test), and during continuous and interval tests in females. Decreases in joint angle and force proprioception were also found to be significantly impaired following eccentric exercise to fatigue (132). Skinner et al. (139) found a decrease in the ability to reproduce joint angles after a series of interval running sprints to fatigue, suggesting that this decreased ability was either due to a loss of efficiency of muscle spindles, or decreased muscle function. Bentley (11), however, suggested that exercise to fatigue is associated with increased muscle spindle activity in response to stretch or vibration and reduced GTO activity, resulting in unopposed alpha and gamma motor neuron stimulation to motor units. The current study did not demonstrate a statistically significant relation between ARPP for pre to post-test values for all joint angles and levels of exertion.

During fatigue, biodynamical compensations in the mechanical properties of the knee extensor musculature, as evidenced by differences in knee kinematics and muscle activation times, may enhance knee stability (108). Evidence suggests that fast-twitch muscle fibers have a greater type Ia afferent innervation as compared to slow-twitch fibers (24, 61, 87, 94, 97, 130, 139, 141, 148, 163). This finding, along with the fact that fast-twitch fibers fatigue comparatively faster, would suggest that as muscle fatigue increases there will be a decrease in proprioceptive awareness which could lead to a decrease in efferent responses. The findings from the current study are not in agreement with these previous findings.

In contrast to the findings that fatigue affects proprioception, Marks and Quinney (100) ascertained that following 20 maximal isokinetic quadriceps contractions in sedentary females, no significant decrease in knee proprioception occurred. However, others have found that isokinetic exercise to fatigue has a significant effect on reproduction sensibility (104, 105, 128, 129, 153). The findings of the current study seem to agree with those of Marks and Quinney (100), as non-significant differences in ARPP were found between pre- and post-test measurements for all levels of exertion.

Summary and Conclusion

Fatigue has long been theorized to be a contributing factor in decreased proprioceptive abilities, and therefore a contributing factor to joint injury. The results from the present study indicate that three levels of isokinetically induced exertion do not affect PRPP or ARPP. The lack of significant findings may be explained by Bentley's (11) theory that as the level of muscle fatigue increases muscle spindle discharge increases. This increase in muscle spindle discharge as muscle fatigues may actually provide a protective mechanism producing increased joint stability. The findings in the current study, however, may not relate to sport activity situations since the induced exertion was localized to the musculature of one thigh. Further, Chronbach's alpha coefficients for all PRPP measures and ARPP at a joint angle of 15° demonstrated that the reliability of these measures was not strong.

In conclusion, the role of fatigue on proprioceptive abilities is unclear at this time. There is little information available regarding how proprioceptive abilities decline as a function of increasing levels of physical exertion. The isokinetic exercise intervention used in the current study did not produce significant differences in PRPP and ARPP. An investigation examining the effect of increasing levels of exertion using methods that more closely approximate sport activities may produce levels of fatigue that are sufficient to alter PRPP and ARPP measures. The inclusion of reaction time and/or anticipation time as a covariable for PRPP may provide improved reliability of these measures.

Research Recommendations

It is recommended that future research of PRPP focus on the roles reaction time and anticipation time play in PRPP measurements. These may be important covariables that could improve the reliability of the measures used for PRPP. However, it is unclear if the primary covariable in this regard is reaction time or anticipation time as both might affect PRPP outcomes. It is recommended that methods in future investigations incorporate the movement of knee extension, the angular velocities of the movements, and the use of a hand switch in the determination of reaction and anticipation time in order to closely replicate the PRPP skill tested. It is also recommended that it be determined if the torque produced by the knee attachment caused a decrease in reliability at angular positions greater than 30°.

It is recommended that future research focus on an exercise protocol that produces increasing levels of exertion that more closely replicate sport and athletic activities. Significant differences in proprioception measures were found by Lattanzio et al. (74) and Skinner et al. (139) while using fatigue interventions that replicate sport activities and activities that were longer in duration than the current exertion protocol. These investigators only examined the effect of fatigue interventions following maximum exertion levels. It is recommended that the effect of increasing levels of exertion be reexamined using methods that more closely approximate sport activities. It is a further recommendation that the assessment of exertion level include measures that can ascertain overall body exertion and not rely only on anatomically localized perception. Measures such as oxygen consumption to target specific percentages of maximum oxygen consumption and electromyographic activity of the muscles tested may provide better assessments of the amount of exertion attained at each exertion level tested.

APPENDIX A

CONSENT TO ACT AS A SUBJECT IN AN EXPERIMANTAL STUDY

TITLE: The Effect of Increasing Levels of Exertion on Knee Joint Proprioception

INVESTIGATOR: William S Gear, MS, ATC
Chair, Exercise Science and Sports Medicine; Director, Athletic Training Education
MC #3400
California Lutheran University
(805) 493-3547

SOURCE OF SUPPORT: None

DESCRIPTION:

I understand that I have been asked to participate in this research study because I am a volunteer male or female between 18-30 years of age and have no previous history of knee injury. I understand that I will be assessed on my ability to reproduce joint angles passively and actively before and after an exercise period to induce varying levels of muscle exertion. The investigator in this study hopes to determine if varying levels of exertion affect knee joint proprioception which may provide information as to these variables role in joint injury.

Prior to testing, your leg dominance will be determined by asking you which leg you would use to kick a ball, this leg will be used as the test leg for the proprioception and single leg hop tests. Base line measures for active and passive repositioning sensibility tests will be conducted two days prior to the start of exercise interventions.

You will be familiarized with the testing procedures prior to initiation of testing. You will be asked to perform a joint position sense test actively and passively prior to and following the exercise session. For the muscle exertion inducing exercise session, you will be asked perform knee joint flexion and extension exercise on the Biodex Isokinetic Dynamometry. The exercise session will be stopped when a torque production value of 90%, 70%, or 50% below the maximum torque output occurs for three consecutive trials. You will be asked to report to the Exercise Science Laboratory, F-4 for six testing sessions over a period of three weeks.

RISKS AND BENEFITS:

I understand that all tests will be implemented within my discretion and that I may choose to withdraw from the study at any time. Possible risks from this study may include mild to moderate muscle soreness during and immediately following the exercise sessions. If this discomfort exceeds my tolerance, testing will be discontinued. I understand that the investigator is a Certified Athletic Trainer, and as such can provide me with treatment options if needed. I understand that there may be no direct benefit to me for participating in this study. However, the results of this study may lead to a better understanding of how moderate levels of fatigue relate to joint injury.

ALTERNATIVE TREATMENTS:

No additional treatments will be available.

COST AND PAYMENTS:

I understand that I will not be charged nor paid for participation in this study

CONFIDENTIALITY:

All records pertaining to my involvement in this research study will be stored in a locked file cabinet in the investigator's office. My identity on these records will be indicated by a case number. This information will only be accessible to the investigator listed on the first page of this document. I understand that any information about me or my treatment will be handled in a confidential (private) manner consistent with other hospital records. I will not be specifically identified in any publication or the research results. However, in unusual cases, my research records may be inspected by appropriate government agencies or released to an order from a court of law.

RIGHT TO WITHDRAW:

I understand I do not have to take part in this research study and, if I change my mind, that I can withdraw at any time. My other care and benefits will be the same whether I take part in this study or not. I also understand that I may be removed from this research study by the investigator in the event of my inability to complete the testing procedures.

VOLUNTARY CONSENT:
The above information has been explained to me and all of my questions have been answered. I understand that I will receive a copy of this consent form signed by myself and a witness. If I have any questions I may contact the investigator, William S. Gear at (805) 493-3547. By signing this form I agree to participate in this study.

____________________________________ __________
Subject Signature Date

____________________________________ __________
Witness Signature Date

INVESTIGATOR'S CERTIFICATION:

I certify that I have explained to the above individual the following aspects of this study: The nature and purpose, the potential benefits and possible risks associated with participating in this study and that I have answered any questions that have been raised and witnessed the above signature.

____________________________________ __________
Investigator's Signature Date

APPENDIX B

RPE Script

"During the exercise test I want you to pay close attention to how hard you feel the exercise work rate is. This feeling should reflect your total amount of exertion and fatigue, combining all sensations and feelings of physical stress, effort, and fatigue. Don't concern yourself with any one factor such as leg pain, shortness of breath or exercise intensity, but try to concentrate on your total, inner feeling of exertion. Try not to underestimate or overestimate your feeling of exertion; be as accurate as you can."

RPE Data Sheet

Subject ID____

Test Intensity	RPE post 1	RPE post 2	RPE post 3

APPENDIX C
DATA COLLECTION SHEETS

PRPP Data Sheet

Subject ID ____

Exertion level ________________________

Test Speed	Rep 1	Rep 2	Rep 3
Pre			
Post			

Test Speed	Rep 1	Rep 2	Rep 3
Pre			
Post			

Test Speed	Rep 1	Rep 2	Rep 3
Pre			
Post			

ARPP Data Sheet

Subject ID ____

Exertion level ______________________

Test Angle	Rep 1	Rep 2	Rep 3
Pre			
Post			

Test Angle	Rep 1	Rep 2	Rep 3
Pre			
Post			

Test Angle	Rep 1	Rep 2	Rep 3
Pre			
Post			

BIBLIOGRAPHY

1. Aagaard P, Simonsen EB, Trolle M, Bangsbo J, Klausen K. Specificity of training velocity and training load on gains in isokinetic knee joint strength. ***Acta Physiol Scand***. 156: 123-129, 1996.

2. American College of Sports Medicine. ***ACSM's guidelines for exercise testing and prescription***. 6th Edition. Lippincott Williams and Wilkens, Baltimore. 2000. pp 78-79.

3. Arendt EA, Agel J, Dick R. Anterior cruciate ligament injury patterns among collegiate men and women. ***J Ath Train***. 34(2): 86-92, 1999.

4. Arendt E, Dick R. Knee injury patterns among men and women in collegiate basketball and soccer: NCAA data and review of literature. ***Am J Sports Med***. 23(6): 694-701, 1995.

5. Arnheim DD, Prentice WE. ***Principles of Athletic Training***. 10th Edition. McGraw Hill, Boston. 2000. pp 515-522.

6. Aune AK, Ekeland A, Nordsletten L. Effect of quadriceps or hamstring contraction on the anterior shear force to anterior cruciate ligament failure: an *in vivo* study in the rat. ***Acta Orthop Scand***. 66(3): 261-265, 1995.

7. Aune AK, Nordsletten, Ekeland A. Structural capacity of the knee to anterior cruciate ligament failure during quadriceps contraction. An *in vivo* study in the rat. ***J Biomechanics***. 29(7): 891-897, 1996

8. Bach JM, Hull ML, Patterson HA. Direct measurement of strain in the posterolateral bundle of the anterior cruciate ligament. ***J Biomechanics.*** 30(3): 281-283, 1997.

9. Barrack RL, Skinner HB, Buckley SL. Proprioception in the anterior cruciate deficient knee. ***Am J Sports Med***. 17(1): 1-6, 1989.

10. Belhaj-Saïf A, Fourment A, Maton B. Adaptation of the precentral cortical command to elbow muscle fatigue. ***Exp Brain Res***. 111: 405-416, 1996.

11. Bentley S. exercise-induced muscle cramp: proposed mechanisms and management. ***Sports Med***. 21(6): 409-420, 1996.

12. Beynnon BD, Fleming BC, Johnson RJ, Nichols CE, Renström PA, Pope MH. Anterior cruciate ligament strain behavior during rehabilitation exercises in vivo. ***Am J Sports Med***. 23(1): 24-34, 1995.

13. Beynnon BD, Renström PA, Konradsen L, Elmqvist LG, Gottlieb D, Dirks M. Validation of techniques to measure knee proprioception. In: Lephart SM, Fu FH, eds. ***Proprioception and Neuromuscular control in joint stability***. Human Kinetics, 2000. pp 127-138.

14. Biedert RM. Contribution of the three levels of nervous system motor control: spinal cord, lower brain, cerebral cortex. In: Lephart SM, Fu FH, eds. ***Proprioception and Neuromuscular control in joint stability***. Human Kinetics, 2000. pp 23-29.

15. Biodex Advantage Software Operations Manual (version 3.2). Biodex Medical Systems, Inc.

16. Birmingham TB, Inglis T, Kramer JF, Vandervoort AA. Effect of a neoprene sleeve on knee joint kinesthesis: influence of different testing procedures. ***Med Sci Sports Exerc***. 32(2): 304-308, 2000.

17. Bjordal JM, Arnøy F, Hannestad B, Strand T. Epidemiology of anterior cruciate ligament injuries in soccer. ***Am J Sports Med***. 25(3): 341-345, 1997.

18. Björklund M, Crenshaw AG, Djupsjöbacka M, Johansson H. Position sense acuity is diminished following repetitive low-intensity work to fatigue in a simulated occupational setting. ***Eur J Appl Physiol***. 81(5): 361-367, 2000.

19. Bochdansky T, Kollmitzer J, Ebinbechler G. The role of electromyography in the assessment of neuromuscular control. In: Lephart SM, Fu FH, eds. ***Proprioception and Neuromuscular control in joint stability***. Human Kinetics, 2000. pp 145-159.

20. Borsa PA, Lephart SM, Kocher MS, Lephart SP. Functional assessment and rehabilitation of shoulder proprioception for glenohumeral instability. ***J Sport Rehabil***. 3(1): 84-104, 1994.

21. Branch TP, Hunter R, Donath M. Dynamic EMG analysis of anterior cruciate deficient legs with and without bracing during cutting. ***Am J Sports Med***. 17(1): 35-41, 1989.

22. Brand RA. A neurosensory hypothesis of ligament function. ***Med Hypotheses***. 29: 245-250, 1989

23. Brezzo RD, Oliver G. ACL injuries in active girls and women. ***JOPERD***. 71(6): 24-27, 2000.

24. Brooks GA, Fahey TD, White TP. ***Exercise Physiology: Human Bioenergetics and Its Applications***. 2nd Edition. Mayfield Publishing Company, 1996. pp 701-721.

25. Buchanan TS, Lloyd DG. Muscle activation at the human knee during isometric flexion-extension and varus-valgus loads. ***J Orthop Res***. 15: 11-17, 1997.

26. Burdett RG, Van Swearingen J. Reliability of isokinetic muscle endurance tests. ***J Orthop Sports Phys Ther*** 8(10): 484-488, 1987.

27. Callaghan MJ, Selfe J, Bagley PJ. The effects of patellar taping on knee joint proprioception. ***J Athl Train***. 37(1): 19-24, 2002.

28. Carlsöö S, Nordstrand A. The coordination of the knee-muscles in some voluntary movements and in the gait in cases with and without knee joint injuries. ***Acta Chir Scand***. 134: 423-426, 1968.

29. Clark FJ, Grigg P, Chapin JW. The contribution of articular receptors to Proprioception with the fingers in humans. ***J Neurophysiol***. 61(1): 186-192, 1989.

30. Collins JJ, O'Connor JJ. Muscle-ligament interactions at the knee during walking. ***Proc Instn Mech Engrs***. 205: 11-18, 1991.

31. Cordo P, Gurfinkel VS, Bevan L, Kerr GK. Proprioceptive consequences of tendon vibration during movement. ***J Neurophysiol***. 74(4): 1675-1688, 1995.

32. Darques JL, Jammes Y. Fatigue induced changes in group IV muscle afferent activity: differences between high- and low- frequency electrically induced fatigues. ***Brain Res***. 750: 147-154, 1997.

33. Douris PC. The effect of isokinetic exercise on the relationship between blood lactate and muscle fatigue. ***J Orthop Sports Phys Ther***. 17(1): 31-35, 1993.

34. Drowatzky JN. ***Motor Learning: Principles and Practices***. 2nd Edition. Burgess Publishing Company, Minneapolis. 1981. pp. 122-126.

35. Dürselen L, Claes L, Kiefer H. The influence of muscle forces and external loads on cruciate ligament strain. ***Am J Sports Med***. 23(1): 129-136, 1995.

36. Dye SF. Functional anatomy of the cerebellum. In: Lephart SM, Fu FH, eds. ***Proprioception and Neuromuscular control in joint stability***. Human Kinetics, 2000. pp 31-35.

37. Enoka RM. ***Neuromechanics of Human Movement***. 3rd Edition. Human Kinetics, Champaign, IL. 2002. pp 232-239.

38. Escamilla RF, Fleisig GS, Zheng N, Barrentine SW, Wilk KE, Andrews JR. Biomechanics of the knee during closed kinetic chain and open kinetic chain exercises. ***Med Sci Sports Exerc***. 30(4): 556-569, 1998.

39. Fitts RH. Muscle fatigue: the cellular aspects. ***Am J Sports Med***. 24(6): S-9 – S-13, 1996.

40. Fitts RH, Balog EM. Effect of intracellular and extracellular ion changes on E-C coupling and skeletal muscle fatigue. ***Acta Physiol Scand***. 156: 169-181, 1996.

41. Frigo C, Crenna P, Jensen LM. Moment-angle relationship at lower limb joints during human walking at different velocities. ***J Electromyogr Kinesiol***. 6(3): 177-190, 1996.

42. Gandevia SC. Insights into motor performance and muscle fatigue based on transcranial stimulation of the human motor cortex. ***Clin Exp Pharmacol Physiol***. 23: 957-960, 1996.

43. Garrett WE, Kirkendall DT. Motor learning, motor control, and knee injuries. In: Lephart SM, Fu FH, eds. ***Proprioception and Neuromuscular control in joint stability***. Human Kinetics, 2000. pp 53-57.

44. Glace BW, McHugh MP, Gleim GW. Effect of a 2-hour run on metabolic economy and lower extremity strength in men and women. ***J Orthop Sports Phys Ther***. 27(3): 189-196, 1998.

45. Gomez E, DeLee JC, Farney WC. Incidence of injury in Texas girls' high school basketball. ***Am J Sports Med***. 24(5): 684-687, 1996.

46. Grabiner MD, Koh TJ, Miller GF. Further evidence against a direct automatic neuromotor link between the ACL and hamstrings. ***Med Sci Sports Exerc***. 24(10): 1075-1079, 1992.

47. Green HJ. Mechanisms of muscle fatigue in intense exercise. ***J Sports Sci***. 15: 247-256, 1997.

48. Green JM, Crews, TR, Bosak AM, Peveler WW. Overall and differentiated ratings of perceived exertion at the respiratory compensation threshold: effects of gender and mode. ***Eur J Appl Physiol***. 89(5): 445-450, 2003

49. Grigg P. Biophysical studies of mechanoreceptors. ***J Appl Physiol***. 60(4): 1107-1115, 1986.

50. Grill SE, Hallett M, McShane LM. Timing of onset of afferent responses and of use of kinesthetic information for control of movement in normal and cerebellar-impaired subjects. ***Exp Brain Res***. 113: 33-47, 1997.

51. Gross MT, Huffman GM, Phillips CN, Wray JA. Intramachine and intermachine reliability of the biodex and cybex II for knee flexion and extension peak torque and angular work. ***J Orthop Sports Phys Ther***. 13(6): 329-335, 1991.

52. Guadagnoli MA, Kleiner DM, Holcomb WR, Miller MG. The assessment of leg dominance by motor function, proprioception and strength. ***J Athl Train***. 3(2): S-29, 1998.

53. Guido J, Voight ML, Blackburn TA, Kidder JD, Nord S. The effects of chronic effusion on knee joint Proprioception: a case study. ***J Orthop Sports Phys Ther***. 25(3): 208-212, 1997.

54. Hagood S, Solomonow M, Baratta R, Zhou BH, D'Ambrosia R. The effect of joint velocity on the contribution of the antagonist musculature to knee stiffness and laxity. ***Am J Sports Med***. 18(2): 182-187, 1990.

55. Hall MG, Ferrell WR, Sturrock RD, Hamblen DL, Baxendale RH. The effect of the hypermobility syndrome on knee joint proprioception. ***Br J Rheumat***. 34: 121-125, 1995.

56. Heitz NA, Eisenman PA, Beck CL, Walker JA. Hormonal changes throughout the menstrual cycle and increased anterior cruciate ligament laxity in females. ***J Ath Train***. 34(2): 144-149, 1999.

57. Henry, F. M. "Best" versus "Average" individual scores. ***The Research Quarterly***. 38(2): 317-320, 1967.

58. Hiemstra LA, Lo IK, Fowler PJ. Effect of fatigue on knee proprioception: implications for dynamic stabilization. ***J Orthop Sports Phys Ther***. 31(10): 598-605, 2001.

59. Higgins MJ, Perrin DH. Comparison of weight-bearing and non-weight-bearing conditions on knee joint reposition sense. ***J Sports Rehab***. 6: 327-334, 1997.

60. Horita T, Komi PV, Nicol C, Kyröläinen H. Stretch shortening cycle fatigue: interactions among joint stiffness, reflex, and muscle mechanical performance in the drop jump. ***Eur J Appl Physiol***. 73: 393-403, 1996.

61. Hortobágyi T, Hamilton JT, Lambert J. Fatigue effects on muscle excitability. ***Int J Sports Med***. 17: 409-414, 1996.

62. Huston LJ, Wojtys EM. Neuromuscular performance characteristics in elite female athletes. ***Am J Sports Med***. 24(4): 427-436, 1996.

63. Ireland ML. Proprioception and neuromuscular control related to the female athlete. In: Lephart SM, Fu FH, eds. ***Proprioception and Neuromuscular control in joint stability***. Human Kinetics, 2000. pp 291-299.

64. Ireland ML. Anterior cruciate ligament injury in female athletes: epidemiology. ***J Ath Train***. 34(2): 150-154, 1999.

65. Jacks TK, Shaeffer WP, Francis KK, Mogol GD, Kleiner DM. The effects of leg dominance on joint position sense of the lower extremity. ***J Ath Train***. 35(2): S-80, 2000.

66. Jacobs R, Bobbert MF, van Ingen Schenau GJ. Mechanical output from individual muscles during explosive leg extensions: the role of biarticular muscles. ***J Biomechanics***. 29(4): 513-523, 1996.

67. Johansson H, Sjölander P, Sojka P. A sensory role for the cruciate ligaments. ***Clin Orthop and Related Research***. 268: 161-178, 1991.

68. Jones LA. Somatic senses 3: proprioception. In: Cohen H, eds. ***Neuroscience for Rehabilitation***, 2nd edition. Lippincott Williams and Wilkins, Philadelphia. pp. 111-130, 1999

69. Kaminski TW, Godfrey MD, Braith RW, Stevens BR. Measurement of acute dynamic anaerobic muscle fatigue using a novel fatigue resistance index. ***Isokinet Exerc Sci***. 8: 95-101, 2000.

70. Kannus P, Beynnon B. Peak torque occurrence in the range of motion during isokinetic extension and flexion of the knee. ***Int J Sports Med***. 14(8): 422-426, 1993.

71. Kannus P, Järvinen M, Johnson R, Renström P, Pope M, Beynnon B, Nichols C, Kaplan M. Function of the quadriceps and hamstrings muscles in knees with chronic partial deficiency of the anterior cruciate ligament. ***Am J Sports Med***. 20(2): 162-168, 1992.

72. Khalsa PS, Grigg P. Responses of mechanoreceptor neurons in the cat knee joint capsule before and after anterior cruciate ligament transection. ***J Orthop Res***. 14: 114-122, 1996.

73. Krauspe R, Schmidt M, Schaible H-G. Sensory innervation of the anterior cruciate ligament: an electrophysiological study of the response properties of single identified mechanoreceptors in the cat. ***J of Bone and Joint Surg***. 74-A(3): 390-397, 1992.

74. Lattanzio PJ, Petrella RJ, Sproule JR, Fowler PJ. Effects of fatigue on knee proprioception. ***Clin J Sport Med***. 7: 22-27, 1997.

75. Lent van MET, Drost MR, Wildenberg FAJM. EMG profiles of ACL-deficient patients during walking: the influence of mild fatigue. ***Int J Sports Med***. 15: 508-514, 1994.

76. Lephart SM, Fu FH, Borsa PA. Proprioception in sports medicine. ***Adv in Operat Orthop***. 2: 77-94, 1994

77. Lephart SM, Giraldo JL, Borsa PA, Fu FH. Knee joint Proprioception: a comparison between female intercollegiate gymnasts and controls. ***Knee Surg, Sports Traumatol, Arthroscopy***. 4: 121-124, 1996.

78. Lephart SM, Kocher MS, Fu FH, Borsa PA, Harner CD. Proprioception following anterior cruciate ligament reconstruction. ***J Sports Rehab***. 1: 188-196, 1992.

79. Lephart SM, Pincivero DM, Giraldo JL, Fu FH. The role of Proprioception in the management and rehabilitation of athletic injuries. ***Am J Sports Med***. 25(1): 130-137, 1997.

80. Lephart SM, Pincivero DM, Rozzi SL. Proprioception of the ankle and knee. ***Sports Med***. 25(3): 149-155, 1998.

81. Lephart SM Swanik CB, Fu, FH. Reestablishing neuromuscular control. In: Prentice WE, ed. ***Rehabilitation techniques in sports medicine***, 3rd edition. McGraw Hill, Boston. 1999. pp 88-106.

82. Lephart SM, Warner JJP, Borsa PA, Fu FH. Proprioception of the shoulder joint in healthy, unstable, and surgically repaired shoulders. ***J Shoulder Elbow Surg***. 3: 371-380, 1994.

83. Ling ZK, Guo HQ, Boersma S. Analytical study on the kinematic and dynamic behaviors of a knee joint. ***Med Eng Phys***. 19(1): 29-36, 1997.

84. Liu SH, Al-Shaikh RA, Panossian V, Finerman GAM, JM. Estrogen affects the cellular metabolism of the anterior cruciate ligament. a potential explanation for female athletic injury. ***Am J Sports Med***. 25(5): 704-708, 1997.

85. Livesay GA, Rudy TW, Woo SL-Y, Runco, TJ, Sakane M, Li G, Fu FH. Evaluation of the effect of joint constraints on the in situ force distribution in the anterior cruciate ligament. ***J Orthop Res***. 15: 278-284, 1997.

86. Ljubisavljević M, Milanović S, Radovanović S, Vukčević I, Kostić V, Anastasijević R. Central changes in muscle fatigue during sustained submaximal isometric voluntary contraction as revealed by transcranial magnetic stimulation. ***Electroenceph Clin Neurophysiol***. 101: 281-288, 1996.

87. Löscher WN, Cresswell AG, Thorstensson A. Central fatigue during a long-lasting submaximal contraction of the triceps surae. ***Exp Brain Res***. 108: 305-314, 1996.

88. Löscher WN, Cresswell AG, Thorstensson A. Excitatory drive to the α-motoneuron pool during a fatiguing submaximal contraction in man. ***J Physiol***. 491(1): 271-280, 1996.

89. Louie JK, Mote CD. Contribution of the musculature to rotatory laxity and torsional stiffness at the knee. ***J Biomechanics***. 20(3): 281-300, 1987.

90. McCabe RE, Myers JB, Lephart SM. The relationship between active and passive assessments of knee proprioception. ***J Ath Train***. 35(2): S-81, 2000.

91. McCloskey DI. Kinesthetic sensibility. ***Physiological reviews***. 58(4): 763-820, 1978.

92. McLester JR. Muscle contraction and fatigue: the role of adenosine 5'-diphosphate and inorganic phosphate. ***Sports Med***. 23(5): 287-305, 1997.

93. Macefield G, Gandevia SC, Burke D. Perceptual responses to microstimulation of single afferents innervating joints, muscles and skin of the human hand. ***J Physiol***. 429: 113-129, 1990.

94. Macefield G, Hagbarth KE, Gorman R, Gandevia SC, Burke D. Decline in spindle support to α-motoneurons during sustained voluntary contractions. ***J Physiol***. 440: 497-515, 1991.

95. Mair SD, Seaber AV, Glisson RR, Garrett WE. The role of fatigue in susceptibility to acute muscle strain injury. ***Am J Sports Med***. 24(2): 137-143, 1996.

96. Mallik AK, Ferrell WR, McDonald AG, Sturrock RD. Impaired proprioceptive acuity at the proximal interphalangeal joint in patients with the hypermobility syndrome. ***Br J Rheumat***. 33: 631-637, 1994.

97. Mannion AF, Dolan P. Relationship between myoelectric and mechanical manifestations of fatigue in the quadriceps femoris muscle group. ***Eur J Appl Physiol***. 74: 411-419, 1996.

98. Markolf KL, Mensch JS, Amstutz HC. Stiffness and laxity of the knee – the contributions of supporting structures. ***J Bone and Joint Surg***. 58-A(5): 583-593, 1976.

99. Markolf KL, Slauterbeck JL, Armstrong KL, Shapiro MW, Finerman GAM. Effects of combined knee loadings on posterior cruciate ligament force generation. ***J Orthop Res***. 14: 633-638, 1996.

100. Marks R, Quinney HA. Effect of fatiguing maximal isokinetic quadriceps contractions on ability to estimate knee-position. ***Percept Motor Skills***. 77: 1195-1202, 1993.

101. Messina DF, Farney WC, DeLee JC. The incidence of injury in Texas high school basketball. ***Am J Sports Med***. 27(3): 294-299, 1999.

102. Mommersteeg TJA, Huiskes R, Blankevoort L, Kooloos JGM, Kauer JMG. An inverse dynamics modeling approach to determine the restraining function of human knee ligament bundles. ***J Biomechanics***. 30(2): 139-146, 1997.

103. Moul JL. Differences in selected predictors of anterior cruciate ligament tears between male and female NCAA division I collegiate basketball players. ***J Ath Train***. 33(2): 118-121, 1998.

104. Myers JB, Guskiewicz KM, Padua DA. Effect of fatigue on proprioception and neuromuscular control of the shoulder. ***J Ath Train***. 34(2): S-9, 1999.

105. Myers JB, Guskiewicz KM, Schneider RA, Prentice WE. Proprioception and neuromuscular control of the shoulder after muscle fatigue. ***J Ath Train***. 34(4): 362-367, 1999.

106. Nyland JA, Caborn DNM, Shapiro R, Johnson DL. Crossover cutting during hamstring fatigue produces transverse plane knee control deficits. ***J Ath Train***. 34(2): 137-143, 1999.

107. Nyland JA, Shapiro R, Caborn DNM, Nitz AJ, Malone TR. The effect of quadriceps femoris, hamstring, and placebo eccentric fatigue on knee and ankle dynamics during crossover cutting. ***J Orthop Sport Phys Ther***. 25(3): 171-184, 1997.

108. Nyland JA, Shapiro R, Stine RL, Horn TS, Ireland ML. Relationship of fatigued run and rapid stop to ground reaction forces, lower extremity kinematics, and muscle activation. ***J Orthop Sport Phys Ther***. 20(3): 132-137, 1994.

109. Oliphant JG, Drawbert JP. Gender differences in anterior cruciate ligament injury rates in Wisconsin intercollegiate basketball. ***J Ath Train***. 31(3): 245-247, 1996.

110. Pandy MG, Shelburne KB. Dependence of cruciate-ligament loading on muscle forces and external load. ***J Biomechanics***. 30(10): 1015-1024, 1997.

111. Perlau R, Frank C, Fick G. The effect of elastic bandages on human knee Proprioception in the uninjured population. ***Am J Sports Med***. 23(2): 251-255, 1995.

112. Perrin DH, Shultz SJ. Models for clinical research involving proprioception and neuromuscular control. In: Lephart SM, Fu FH, eds. ***Proprioception and Neuromuscular control in joint stability***. Human Kinetics, 2000. pp 349-362.

113. Pincivero DM, Gandaio CM, Ito Y. Gender-specific knee extensor torque, flexor torque, and muscle fatigue responses during maximal effort contractions. ***Eur J Appl Physiol***. 89(2): 134-141. 2003.

114. Pincivero DM, Gear WS. Quadriceps activation and perceived exertion during a high intensity, steady state contraction to failure. ***Muscle & Nerve***. 23(4): 514-520, 2000.

115. Pincivero DM, Gear, WS, Moyna NM, Robertson RJ. The effect of rest interval on quadriceps torque and perceived exertion in healthy males. ***J Sports Med Phys Fitness***. 39: 294-299, 1999.

116. Pincivero DM, Gear WS, Sterner RL. Assessment of the reliability of high-intensity quadriceps femoris muscle fatigue. ***Med Sci Sports Exerc***. 33(2): 334-338, 2001.

117. Pincivero DM, Gear WS, Sterner RL, Karunakara RJ. Gender differences in the relationship between quadriceps work and fatigue during high intensity exercise. ***J Strength Cond Res***. 14(2): 202-206, 2000.

118. Pincivero DM, Lephart SM, Karunakara RG. Relationship between open and closed kinematic chain assessment of knee strength and functional performance. ***Clin J Sport Med***. 7: 11-16, 1997.

119. Pincivero DM, Lephart SM, Karunakara RA. Reliability and precision of isokinetic strength and muscular endurance for the quadriceps and hamstrings. ***Int J Sports Med***. 18(2): 113-117, 1997

120. Powell JW, Barber-Foss KD. Sex-related injury patterns among selected high school sports. ***J Athl Train***. 28(3): 385-391, 2000.

121. Race A, Amis AA. Loading of the two bundles of the posterior cruciate ligament: an analysis of bundle function in a-p drawer. ***J Biomechanics***. 29(7): 873-879, 1996.

122. Riemann BL, Guskiewicz KM. Contribution of the peripheral somatosensory system to balance and postural equilibrium. In: Lephart SM, Fu FH, eds. ***Proprioception and Neuromuscular control in joint stability***. Human Kinetics, 2000. pp 37-51.

123. Riemann BL, Lephart SM. The sensorimotor system, part I: the physiologic basis of functional joint stability. ***J Athl Train***. 37(1): 71-79, 2002.

124. Riemann BL, Lephart SM. Sensorimotor system measurement techniques. ***J Athl Train***. 37(1): 85-98, 2002.

125. Rosse C, Gaddum-Rosse P. ***Hollinshead's Textbook of Anatomy***. 5th Edition. Lippincott-Raven Publishers. Philadelphia, PA. 1997. pp. 380-391.

126. Rothwell J. ***Control of human voluntary movement***. 2nd edition. Chapman and Hall, London, 1994. pp. 86-126.

127. Rowinski MJ. Afferent neurobiology of the joint. In: Gould JA, ed. ***Orthopaedic and sports physical therapy***. 2nd edition. Mosby Company, St. Louis, MO, 1990. pp. 49-63.

128. Rozzi SL, Lephart SM, Fu FH. Effects of muscular fatigue on knee joint laxity and neuromuscular characteristics of male and female athletes. ***J Ath Train***. 34 (2): 106-114, 1999.

129. Rozzi SL, Lephart SM, Gear WS, Fu FH. Knee joint laxity and neuromuscular characteristics of male and female soccer and basketball players. ***Am J Sports Med***. 27: 312-319, 1999.

130. Rozzi SL, Yuktanandana P, Pincivero D, Lephart SM. Role of fatigue on proprioception and neuromuscular control. In: Lephart SM, Fu FH, eds. ***Proprioception and Neuromuscular control in joint stability***. Human Kinetics, 2000. pp 375-383.

131. Sakane M, Fox RJ, Woo SL-Y, Livesay GA, Li G, Fu FH. In situ forces in the anterior cruciate ligament and its bundles in response to anterior tibial loads. ***J Orthop Res***. 15: 285-293, 1997.

132. Saxton JM, Clarkson PM, James R, Miles M, Westerfer M, Clark S, Donnelly AE. Neuromuscular dysfunction following eccentric exercise. ***Med Sci Sports Exerc***. 27(8): 1185-1193, 1995.

133. Schäfer SS. Simulation of dynamic fusimotor effects in the discharge frequency of Ia afferents by prestretching the muscle spindle. ***Exp Brain Res***. 108: 297-304, 1996.

134. Shalin K. Muscle fatigue and lactic acid accumulation. ***Acta Physiol Scand***. 128(S556): 83-91, 1986.

135. Shelbourne KD, Davis TJ, Klootwyk TE. The relationship between intercondylar notch width of the femur and the incidence of anterior cruciate ligament tears. ***Am J Sports Med***. 26(3): 402-407, 1998.

136. Shelburne KB, Pandy MG. A musculoskeletal model of the knee for evaluating ligament forces during isometric contractions. ***J Biomechanics***. 30(2): 163-176, 1997.

137. Siff, M. C. Supertraining. Denver, CO: Supertraining Institute; 2000

138. Skinner HB, Barrack RL. Joint position sense in the normal and pathologic knee joint. ***J Electromyogr Kinesiol***. 1(3): 180-190, 1991.

139. Skinner HB, Wyatt MP, Hodgdon JA, Conrad DW, Barrack RL. Effect of fatigue on joint position sense of the knee. ***J Orthop Res***. 4: 112-118, 1986.

140. Slauterbeck J, Fuzie S, Smith M, Clark R, Hardy D. Estrogen and progesterone levels at time of ACL injury. ***J Athl Train***. 36(2): S-61, 2001.

141. Smith LK, Weiss EL, Lehmkuhl LD. ***Brunnstrom's Clinical Kinesiology***. 5th Edition. F.A. Davis Company. Philadelphia, PA, 1996. pp. 90 – 118.

142. Smith LK, Weiss EL, Lehmkuhl LD. ***Brunnstrom's Clinical Kinesiology***. 5th Edition. F.A. Davis Company. Philadelphia, PA, 1996. pp. 301 – 329.

143. Solomonow M, Baratta R, Zhou BH, Shoji H, Bose W, Beck C, D'Ambrosia R. The synergistic action of the anterior cruciate ligament and thigh muscles in maintaining joint stability. ***Am J Sports Med***. 15(3): 207-213, 1987.

144. Takai S, Woo SL-Y, Livesay GA, Adams DJ, Fu FH. Determination of in situ loads on the human anterior cruciate ligament. ***J Orthop Res***. 11: 686-695, 1993.

145. Taylor JL, Butler JE, Allen GM, Gandevia SC. Changes in motor cortical excitability during human muscle fatigue. ***J Physiol***. 490(2): 519-528, 1996.

146. Taylor NAS, Sanders RH, Howick EI, Stanley SN. Static and dynamic assessment of the biodex dynamometer. ***Eur J Appl Physiol***. 62: 180-188, 1991.

147. Teitz CC, Lind BK, Sacks BM. Symmetry of the femoral notch width index. ***Am J Sports Med***. 25(5): 687-690, 1997.

148. Tho KS, Németh G, Lamontagne M, Eriksson E. Electromyographic analysis of muscle fatigue in anterior cruciate ligament deficient knees. ***Clin Orthop Related Res***. 340: 142-151, 1997.

149. Tortora GJ. ***Principles of Human Anatomy***. 4th Edition. Harper and Row, Publishers, Inc, 1986. pp. 260-267.

150. Tsuda E, Okamura Y, Otsuka H, Komatsu T, Tokuya S. Direct evidence of the anterior cruciate ligament-hamstring reflex arc in humans. ***Am J Sports Med***. 29(1): 83-87, 2001.

151. Veltri DM, Deng XH, Torzilli PA, Warren RF, Maynard MJ. The role of the cruciate and posterolateral ligaments in stability of the knee. ***Am J Sports Med***. 23(4): 436-443, 1995.

152. Vilensky JA, O'Connor BL, Brandt KD, Dunn EA, Rogers PI, DeLong CA. Serial kinematic analysis of the unstable knee after transection of the anterior cruciate ligament: temporal and angular changes in a canine model of osteoarthritis. ***J Orthop Res***. 12(2): 229-237, 1994.

153. Voight ML, Hardin JA, Blackburn TA, Tippett S, Canner GC. The effects of muscle fatigue on and the relationship of arm dominance to shoulder proprioception. ***J Orthop Sports Phys Ther***. 23(6): 348-352, 1996.

154. Wang CJ, Walker PS, Wolf B. The effects of flexion and rotation on the length patterns of the ligaments of the knee. ***J Biomechanics***. 6: 587-596, 1973.

155. Watkins J. ***Structure and Function of the Musculoskeletal System***. Human Kinetics. Champaign, IL. 1999. pp. 236-240.

156. Whiting WC, Zernicke RF. ***Biomechanics of Musculoskeletal Injury***. Human Kinetics, Champaign, IL. 1998. pp 113-136.

157. Williams GN, Chmielewski T, Rudolph K, Buchanan TS, Snyder-Mackler L. Dynamic knee stability: current theory and implications for clinicians and scientists. ***J Orthop Sports Phys Ther***. 31(10): 546-566, 2001.

158. Wojtys EM, Huston LJ, Boynton MD, Spindler KP, Lidenfeld TN. The effect of menstrual cycle on anterior cruciate ligament injuries in women as determined by hormone levels. ***Am J Sports Med***. 30(2): 182-188, 2002.

159. Wojtys EM, Huston LJ, Linden TN, Hewett TE, Greenfield MLVH. Association between the menstrual cycle and anterior cruciate ligament injuries in female athletes. ***Am J Sports Med***. 26(5): 614-619, 1998.

160. Wojtys EM, Wylie BB, Huston LJ. The effects of muscle fatigue on neuromuscular function and anterior tibial translation in healthy knees. ***Am J Sports Med***. 24(5): 615-621, 1996.

161. Woo SL-Y, Livesay GA, Engle C. Biomechanics of the human anterior cruciate ligament: muscle stabilization and ACL reconstruction. ***Orthop Review***. August: 935-941, 1992.

162. Worrell TW, Denegar CR, Armstrong SL, Perrin DH. Effect of body position on hamstring muscle group average torque. ***J Orthop Sports Phys Ther***. 11(10): 449-452, 1990.

163. Worrell TW, Perrin DH. Hamstring muscle injury: the influence of strength, flexibility, warm-up, and fatigue. ***J Orthop Sports Phys Ther***. 16(1): 12-18, 1992.

164. Worrell TW, Perrin DH, Denegar CR. The influence of hip position on quadriceps and hamstring peak torque and reciprocal muscle group ratio values. ***J Orthop Sports Phys Ther***. 11(3): 104-107, 1989.

165. Zhou S. Acute effect of repeated maximal isometric contraction on electromechanical delay of knee extensor muscle. ***J Electromyogr Kinesiol***. 6(2): 117-127, 1996.

166. Zimny ML, Schutte M, Dabezies E. Mechanoreceptors in the Human Anterior Cruciate Ligament. ***Anat Rec***. 214: 204-209, 1986.