UNIVERSIDADE ESTADUAL DE CAMPINAS

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ESTUDO DO CONTROLE MOTOR E DA BIOMECÂNICA NA LESÃO E RECONSTRUÇÃO DO LIGAMENTO CRUZADO ANTERIOR

Tese apresentada ao Instituto de Biologia da Universidade Estadual de Campinas, para obtenção do título de Doutor em Biologia Funcional e Molecular na área de Fisiologia.

Orientador: Prof. Dr. Gil Lúcio Almeida

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Este trabalho possibilitou conhecer os caminhos da sabedoria, da solidão, da transmissão do conhecimeto, da desmistificação do ceticismo, do respeito, da perda, da razão, da ignorância..... e desta conquista

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RESUMO

Introdução: Não existe um consenso na literatura sobre as mudanças cinemáticas e eletromiográficas (EMGs) que podem ocorrer devido a lesão do ligamento cruzado anterior (LCA) ou pela sua reconstrução. No entanto, o entendimento das modificações e adaptações do sistema de controle motor é crucial para a tomada de decisões sobre a reconstrução cirúrgica ou a reabilitação deste ligamento. **Hipótese:** A lesão do LCA provocaria alterações nos padrões cinemáticos dos movimentos articulares do quadril, joelho e tornozelo e dos músculos que agem diretamente nestas articulações. Essas alterações seriam refletidas na dificuldade desses indivíduos em realizar movimentos de extensão do joelho (cadeia cinética aberta) e de agachamento (cadeia cinética fechada). A lesão do LCA também comprometeria a capacidade desses indivíduos para manter a perna estendida contra a ação da gravidade, devido a uma possível perda de propriocepção. Já a reconstrução cirúrgica do LCA promove a estabilidade da articulação do joelho gerando uma melhora nos padrões cinemáticos e EMGs dos movimentos descritos acima. **Métodos:** Os movimentos do joelho, quadril e tornozelo foram registrados usando um sistema de análise do movimento (OPTOTRAK 3020) numa frequência de 100 Hz e calculado o deslocamento, a velocidade e a aceleração angular do quadril, joelho e tornozelo. Estes valores cinemáticos foram sincronizados com os dados das atividades dos músculos reto femural, vasto medial, vasto lateral, semitendinoso, bíceps femural, gastrocnêmio medial, gastrocnêmio lateral e tibial anterior. Para o registro da atividade EMG foram usados eletrodos de

superfície – DELSYs (model DE2.2L). Os sinais EMGs foram coletados na frequência de 1000Hz, amplificados (X 2000), filtrados (20-450Hz) e registrados. Esses dados foram analisados em indivíduos controle, com lesão do LCA e com reconstrução com o terço central do ligamento patelar. Em média, em cada experimento foram investigados 9 indivíduos para cada grupo. Os movimentos analisados foram os de extensão do joelho (Capítulo II), do agachamento (Capítulo III) e da manutenção da perna estendida contra a ação da gravidade (Capítulos IV e V). Para análise estatística entre e intra-grupos foi utilizado o modelo ANOVA. **Resultados:** Os resultados destes trabalhos revelaram que apesar da lesão do LCA provocar na maioria das vezes, instabilidade articular no joelho detectada pelas manobras clínicas, não existem alterações nas estratégias motoras utilizadas para executar os movimentos de extensão, do agachamento ou da manutenção da perna estendida contra a gravidade. Os três grupos investigados utilizaram a mesma estratégia em termos dos padrões cinemáticos das articulações do quadril, joeho e tornozelo e dos padrões EMGs dos músculos que influenciam diretamente nos movimentos destas articulações. Já a reconstrução ligamentar também não provocou alterações nos padrões EMGs e cinemáticos durante estas tarefas. **Conclusões:** Tanto a lesão como a reconstrução do LCA não afetam as estratégias de controle motor usadas pelo sistema nervoso central para modular a atividade EMG durante o movimento de extensão do joelho, do agachamento ou na manutenção da perna estendida contra a ação da gravidade. Não foram encontradas alterações proprioceptivas em indivíduos com lesão ou reconstrução do LCA

ABSTRACT

Introduction: There is no consensus in the literature regarding the cinematic and electromyographic (EMG) changes that can occur due to a lesion of the anterior cruciate ligament (ACL) or its reconstruction. However, the understanding of the modifications and adaptations of the motor control system is crucial for making decisions on surgical reconstruction or rehabilitation of this ligament. **Hypothesis:** The lesion of the ACL would provoke alterations in the cinematic patterns of the articular movements of the hip, knee and ankle and of the muscles that act directly on these joints. These alterations would be reflected in the difficulty of these individuals to realize extension movements of the knee (open kinetic chain) and squatting (closed kinetic chain). The lesion of the ACL would also compromise the capacity of these individuals to keep the leg extended against gravity, due to a possible loss of proprioception. On the other hand, the surgical reconstruction of the ACL promotes stability of the knee joint, improving the cinematic patterns and EMGs of the above-described movements. **Methods:** The hip, knee and ankle movements were registered using a movement-analysis system (OPTOTRAK 3020) at a frequency of 100 Hz and the dislocation, speed and angular acceleration were calculated. These cinematic values were synchronized with the activity data of the rectus femoris, vastus medialis, vastus lateralis, semitendinosus, biceps femoris, gastrocnemius medialis, gastrocnemius lateralis and tibialis anterior muscles. To register the EMG activity, surface electrodes (DELSYs - model DE2.2L) were used. The

EMG signals were collected at a frequency of 1000 Hz, amplified (X 2000), filtered (20-450 Hz) and registered. These data were analyzed in control individuals with ACL lesion and with reconstruction of the central third of the patellar ligament. On the average, in each experiment nine individuals were investigated for each group. The movements analyzed were knee extensions (Chapter II), squatting (Chapter III) and maintenance of the leg extended against gravity (Chapters IV and V). For statistic analysis between and among groups, the ANOVA model was utilized. **Results:** The results of this work revealed that, in spite of the fact that the ACL lesion generally causes articular instability in the knee, detected by clinical maneuvers, there are no alterations in the motor strategies utilized to execute the extension movements, squatting or maintenance of the leg extended against gravity. The three groups investigated utilized the same strategy in terms of the cinematic patterns of the hip, knee and ankle articulations and of the EMG patterns of the muscles that are directly influential in the movement of these joints. On the other hand, ligament reconstruction did not provoke alterations in the EMG and cinematic patterns during these tasks. **Conclusions:** Neither the lesion nor the reconstruction of the ACL effect the motor control strategies used by the central nervous system to modulate the EMG activity during knee-extension movement, squatting or the maintenance of the leg extended against gravity. No proprioceptive alterations were encountered in individuals with lesion or reconstruction of the ACL.

APRESENTAÇÃO

No Capítulo I dessa tese é apresentada uma introdução em forma de revisão de literatura referente aos conhecimentos mais recentes dos aspectos biomecânicos e do controle motor em indivíduos com lesão e com reconstrução do ligamento cruzado anterior (LCA). Com base nesta revisão de literatura são apresentadas várias hipóteses para explicar as possíveis alterações ou adaptações cinemáticas e nos padrões eletromiográficos (EMGs) em indivíduos com lesão ou reconstrução do LCA.

Em especial, no Capítulo II é analisado um movimento em cadeia cinética aberta do joelho (extensão), onde foi verificada a hipótese que a instabilidade mecânica secundária à lesão do LCA afetaria os padrões cinemáticos e das atividades musculares. Para investigar esta hipótese, três grupos de indivíduos (controle n=10, lesados n=10 e reconstruídos n=10) realizaram movimentos de extensão do joelho em três amplitudes angulares distintas, o mais rapidamente possível.

No Capítulo III é verificada a hipótese de que a lesão do LCA provocaria uma modificação no acoplamento cinemático dos movimentos do quadril, joelho e tornozelo durante o movimento do agachamento. A mudança nesse acoplamento estaria relacionada às alterações nos padrões eletromiográficos. Já a reconstrução do LCA promoveria a estabidade articular regularizando o acoplamento dos movimentos nas três articulações. Esta normalização seria refletida nos padrões cinemáticos e eletromiográficos. Foram investigados três

grupos de indivíduos (controle n=9, lesados n=9 e reconstruídos n=9) e quantificados os dados cinemáticos e EMGs durante o agachamento. Como no movimento de extensão, os padrões cinemáticos e EMGs dos três grupos investigados durante o agachamento, foram idênticos.

No Capítulo IV é descrita a estratégia de controle motor utilizada por indivíduos sem história de lesão no joelho para manter o joelho estendido contra a ação da gravidade, imediatamente após a realização de uma contração isotônica. Para esta análise foram investigados 8 indivíduos sem história de lesões nos membros inferiores e quantificados os dados cinemáticos e EMGs (músculos: reto-femural e bíceps femural) durante a manutenção da perna estendida contra a ação da gravidade. Durante os experimentos, os indivíduos foram incapazes de manter a perna totalmente estacionada contra a ação da gravidade, aumentando a extensão do joelho em poucos graus. O aumento da extensão do joelho com consequente incremento do torque muscular, foi acompanhado por uma diminuição da quantidade da atividade eletromiográfica. Esses dados são discutidos no contexto das teorias de controle motor.

Finalmente, no Capítulo V é analisada a hipótese que a lesão do LCA modificaria as estratégias descritas no Capítulo IV para manter o joelho estendido contra a ação da gravidade, depois de uma contração isotônica. Essas alterações estariam associadas a uma possível perda proprioceptiva provocada pela lesão do LCA. No entanto, a reconstrução deste ligamento promoveria a estabilidade articular. Devido a isto, a estratégia postural para manutenção da perna contra a ação da gravidade seria normalizada no grupo

reconstruído. Para esta análise foram investigados indivíduos controle (n=8), lesados (n=8) e reconstruídos (n=8) e quantificados os dados cinemáticos e EMGs (músculos: reto-femural e bíceps femural) durante a manutenção do joelho estendido contra a ação da gravidade.

Finalmente no capítulo VI é feita uma discussão geral dos dados dos três experimentos, no contexto da literatura revisada e é apresentada uma conclusão geral.

CAPÍTULO I – Artigo: "Lesão e reconstrução do LCA: Uma revisão biomecânica e do controle motor"

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Palavras chave: ligamento cruzado anterior, controle motor, biomecânica, revisão

RESUMO

Este artigo apresenta uma revisão dos conhecimentos mais recentes sobre as implicações dos aspectos biomecânicos, do controle motor e da neurofisiologia do ligamento cruzado anterior. Estes aspectos são contextualizados analisando o ligamento normal e as consequências provocadas devido à lesão ou mesmo à reconstrução deste órgão. São descritos o mecanismo da lesão, as cargas de deformação, o papel dos receptores proprioceptivos, os comportamentos cinemáticos, cinéticos e eletromiográficos encontrados na população controle, com lesão ou com reconstrução do LCA. Com base nestas informações foram elaborados os princípios a serem considerados para o tratamento fisioterapêutico do ligamento cruzado anterior.

SUMARY

This article presents a revision of the most recent knowledge on the implications of the biomechanical aspects, of motor control, and of neurophysiology of the anterior cruciate ligament. These aspects are put into context by analyzing the normal ligament and the consequences provoked by a lesion or even the reconstruction of this organ. The article describes the lesion mechanism, the burdens of deformation, the role of the proprioceptive receptors, and the cinematic, kinetic and electromyographic behaviors encountered in the control population, with lesion or ACL reconstruction. Based on this information, the principles to be considered in the physiotherapeutic treatment of the anterior cruciate ligament were elaborated.

A função mecânica do LCA

O ligamento cruzado anterior (LCA) tem uma propriedade de resistência tensil bastante considerável (Woo et al. 1991; Beynnon et al. 1996). Woo et al (1991) mensuraram que a carga necessária para romper o LCA em cadáver, com o joelho a 30 graus de flexão, foi de aproximadamente 2160N. Os valores de ruptura do LCA foram menores em cadáveres com idade mais avançada.

Beynnon et al. (1996) colocaram um transdutor *in vivo* diretamente no ligamento cruzado anterior em humanos e quantificaram a tensão anterior exercida no LCA durante o movimento do joelho. Foi detectado que essa tensão é pequena e aumenta, com a extensão do joelho, em ligamentos intactos. Interessante, também, foi o fato dessa tensão não mudar com o tipo de movimento do joelho (cadeia cinética fechada – agachar – *versus* cadeia aberta – extensão). De fato, Wascher et al (1993) comprovaram, através de mensuração direta da força em cadáveres, que a tensão no LCA aumenta nas amplitudes finais da extensão e na hiperflexão do joelho.

Há um consenso na literatura e entre os clínicos de que o ligamento cruzado anterior atua como um estabilizador mecânico, restringindo a anteriorização e a rotação da tíbia com relação ao fêmur. Para Fu et al. (1993) e Fukubayashi et al. (1982), a principal função do LCA é prevenir o deslocamento anterior da tíbia em relação ao fêmur. Kennedy et al. (1974) e Wascher et al. (1993) acrescentaram que este ligamento age no mecanismo de rotação interna e externa do joelho e na restrição do estresse em valgo e varo (ver Fu et al.

1993, para uma revisão). Apesar de mecanicamente limitar principalmente o deslocamento anterior da tíbia em relação ao fêmur, funcionalmente a maior vulnerabilidade do LCA ocorre em mecanismos rotacionais, visto que 70% das rupturas foram relacionadas ao mecanismo rotacional (Mello et al. 1999).

A lesão do LCA

Baseado em estudos feitos para uma população de uma cidade americana (Daniel et al. 1994), estimamos que podem ocorrer 24 lesões do LCA ao ano para cada 10000 habitantes. Se aplicarmos esse índice para a população brasileira em 2002, teremos cerca de 40.800 indivíduos sofrendo por ano uma lesão do LCA.

Como pode um ligamento tão forte romper com essa frequência? Apesar de ser bastante resistente, a ruptura do ligamento ocorre devido a uma sobrecarga máxima que ultrapasse os limites de resistência do LCA. Normalmente, as rupturas desta estrutura originam-se em atividades esportivas (Daniel et al., 1994; Fatarelli, 1997; Noyes et al. 1983c) e, na maioria das vezes, limitam ou impedem a prática das mesmas (Abdalla et al. 1995; Hernandez et al. 1996; Johnson et al. 1992; Noyes et al. 1983c). Funcionalmente, podem ser identificados dois grupos de indivíduos com lesão no LCA. O primeiro tem sintomas clínicos como edema, dor e falseios durante movimentos do joelho e apresenta dificuldade em realizar algumas atividades da vida diária (Noyes et al. 1983c). Para os indivíduos deste grupo, frequentemente é recomendada a reconstrução cirúrgica do LCA (Gomez-Castresana & Bastos, 1992; Mott, 1983; Noyes et al. 1983; Paulos et al. 1983).

Entre as técnicas mais utilizadas para esta cirurgia, destacam-se as reconstruções do LCA utilizando o terço central do ligamento patelar (Noyes et al. 1983; Paulos et al. 1983) e a reconstrução anatômica com o tendão do músculo semitendinoso (Larson, 1985; Mott, 1983). Outra possibilidade é a utilização deste último juntamente com o tendão do músculo grácil (Gomez-Castresana & Bastos, 1992; Karlson et al., 1994). A finalidade da reconstrução do LCA é de restaurar a estabilidade articular (Fu et al. 1993) e prevenir deslocamentos excessivos da tíbia em relação ao fêmur (Lane et al. 1994).

Por outro lado, há um grupo de indivíduos que tem a lesão do LCA, mas não refere sintomas clínicos como edema e dor (Noyes et al. 1983c). Os indivíduos deste grupo podem realizar tarefas motoras envolvendo a articulação do joelho, sem nenhum déficit motor aparente (Wojtys & Huston, 1994) e podem ser considerados como adaptados à lesão.

Existem vários fatores de risco que predispõem os indivíduos à lesão do LCA. Entre esses fatores de risco destacam-se os anatômicos (Harner et al. 1994), neuromusculares (Wojtys & Huston, 1994; Ciccotti et al. 1994), e biomecânicos (Marans et al. 1989; Devita et al. 1997; Brandsson et al. 2002; Andriacchi 1990).

A. Aspectos Anatômicos

Aspectos como formato ou tamanho de estruturas ósseas ou ligamentares têm sido associados como fatores predisponentes à lesão do LCA (Schickendantz & Weiker, 1993). Harner et al. (1994) observaram que indivíduos

com história de lesão bilateral do LCA apresentavam um aumento na largura do côndilo femural lateral comparados ao grupo controle. Houve também uma maior presença de lesões ligamentares nos indivíduos com histórias familiares de lesões ligamentares do joelho. Por outro lado, Hernandez et al. (1995) não observaram diferenças anatômicas significativas entre indivíduos com e sem lesão do LCA entre os ângulos do teto do intercôndilo femoral - LCA, do teto do intercôndilo femoral-platô tibial, entre os 2/3 proximais com o 1/3 distal do LCA. Também não foram observadas diferenças na altura do intercôndilo femoral e do índice de largura no intercôndilo femoral.

B. Fatores Neuromusculares

Capacidade proprioceptiva

Acredita-se que a lesão do LCA produz perda da propriocepção, deteriorando a capacidade destes indivíduos para detectar alterações estáticas (Barrack et al. 1989; Borsa et al. 1997) ou dinâmicas na posição da perna (Corrigan et al. 1992). A perda da propriocepção tem sido também associada a mudanças no tempo de latência dos músculos nas respostas reflexas (Beard et al. 1993; Wojtys & Huston, 1994), na ordem (Lass et al. 1991; Kålund et al. 1990) e padrão de atividade eletromiográfica (EMG) durante movimentos voluntários (Solomonow et al. 1987).

Corrigan et al. (1992) verificaram a habilidade dos indivíduos com LCA lesado para detectar a posição estática e pequenas alterações na angulação articular (Figura 01). Os autores identificaram que existe uma relação entre a

força da musculatura posterior e da anterior com os mecanismos proprioceptivos. Os sujeitos que não tiveram um predomínio da musculatura posterior do joelho sobre a anterior apresentavam uma diminuição na propriocepção da posição e da mudança articular. No entanto, os que mostravam predomínio da musculatura dos ísquios-tibiais em relação ao quadríceps tiveram uma melhor performance proprioceptiva. Este desequilíbrio entre a musculatura anterior e posterior foi devido a uma diminuição da força gerada pelo quadríceps. Desta forma, a hipotrofia do quadríceps, após a lesão do LCA, funcionaria como mecanismo adaptativo.

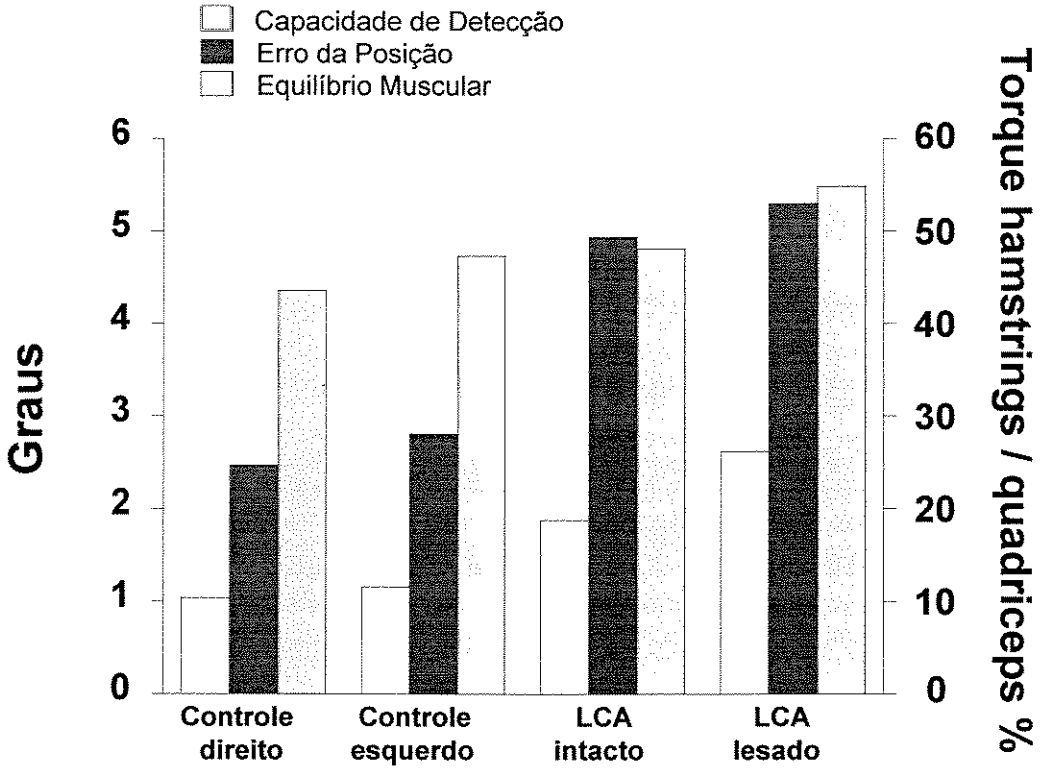

FIGURA 01: Limiar de detecção da posição articular e equilíbrio muscular. Grupos LCA rompido, não rompido e controle. Variáveis detecção do movimento, erro da posição e equilíbrio muscular (Corrigan et al. 1992).

No estudo de Barrack et al. (1989), como no de Corrigan et al. (1992), foi documentado que existe uma dificuldade dos indivíduos em detectar o movimento passivo no membro lesado entre 30° a 40° de extensão do joelho. No entanto, Borsa et al. (1997) observaram essa dificuldade nestes indivíduos apenas na amplitude final da extensão.

Barrack et al. (1989) e Borsa et al. (1997) usaram o membro não lesado como controle. Por outro lado, Corrigan et al. (1992) compararam o membro lesado e contra-lateral com o membro de um grupo controle sem história de lesão no joelho. Comparado com sujeitos sem lesão do LCA, Corrigan et al. (1992) demonstraram perda proprioceptiva na articulação de ambos os joelhos em sujeitos com lesão unilateral do LCA. Esses dados mostram que devemos ser cautelosos ao assumirmos o lado não lesado como parâmetro de normalidade para o lado lesado. Por ser uma circuitaria complexa, as informações provenientes dos receptores de um ligamento podem também projetar, via polissináptica, nos neurônios motores que inervam a musculatura contra-lateral à lesão.

Apesar de vários estudos mostrarem o papel do LCA relacionado à capacidade de detecção do membro espacialmente, é fundamental destacar que outros estudos falham em detectar alterações proprioceptivas em indivíduos com lesão (Good et al 1999; Jennings & Seedhom, 1994) ou reconstrução do LCA (MacDonald et al. 1996).

Mais recentemente, Fremerey et al. (2000) moveram a articulação do joelho numa velocidade de 0.5°/s e mediram a capacidade dos indivíduos para

reproduzir passivamente a posição que sua articulação foi alterada. Os indivíduos com lesão aguda do LCA cometeram erros de 6.7 e 2.2°, respectivamente, para detectar a posição do membro lesado e não lesado. Já os indivíduos com lesão crônica apresentaram uma melhora na percepção da posição angular do membro no lado lesado e não apresentaram alteração no lado não lesado.

Fremerey et al. (2000) observaram que, após seis meses da reconstrução cirúrgica do LCA, houve uma melhora significativa na capacidade de detecção angular no membro reconstruído. Interessante que, ao contrário dos dados obtidos por Borsa et al. (1997), no estudo de Fremerey et al. (2000), os indivíduos com lesão crônica do LCA apresentaram uma menor dificuldade para detecção da posição espacial no final da extensão do joelho.

Vale ressaltar que a substituição protética total unilateral do quadril não provocou uma perda proprioceptiva substancial (Grigg et al. 1973). Já Clark et al. (1979) anestesiaram a articulação do joelho e estruturas adjacentes para diminuir a ação dos receptores articulares, não observando alterações da percepção da posição articular estática.

Concluímos, portanto, que a capacidade de detecção da posição articular na ausência dos elementos sensoriais capsulares poderia ser compensada por informações provenientes, por exemplo, do fuso neuro-muscular localizado nas fibras extra-fusais dos músculos. Baseado na revisão da literatura apresentada acima, devemos ser bastante cautelosos ao assumirmos que uma lesão do LCA provoca perda proprioceptiva que não possa ser compensada por informações

provenientes de outros receptores da cápsula articular ou dos fusos musculares. Matthews (1964) demonstrou, em um elegante estudo, que as informações provenientes do fuso neuro-muscular são essenciais na detecção estática e dinâmica dos movimentos. Skoglund (1956) também demonstrou que as informações provenientes da cápsula articular informam apenas as amplitudes máximas dos movimentos de flexão e extensão.

Em nossos estudos, evidenciamos que a lesão do LCA ou a sua reconstrução não influenciam a capacidade dos indivíduos para manter o membro inferior estendido contra a ação da gravidade *("ver Capítulo V – The lesion of anterior cruciate ligament and its surgical reconstruction do not affect the modulation of electromyographic activities during isometric contraction")*. Para manter o membro estendido contra a ação da gravidade, é preciso que o indivíduo tenha uma boa percepção estática da força.

Recentemente, tem sido demonstrado a capacidade de regeneração dos receptores presentes no interior do LCA. Barrack et al. (1997) mostraram que, após uma reconstrução do LCA, os receptores presentes no interior deste ligamento podem regenerar. Porém, antes da cirurgia, a população neural era composta predominantemente por mecanoreceptores (89%), restando 11% de terminações nervosas livres. Após a reconstrução, o ligamento passou a ser composto por 90% de terminações nervosas livres e 10% de mecanoreceptores.

Surpreendentemente, Ochi et al. (1999) demonstraram reinervação sensorial em cachorros que foram submetidos à reconstrução do LCA, mostrando que potenciais evocados somatosensoriais foram detectados devido

à estimulação direta dos receptores do LCA, após a cirurgia de reconstrução ligamentar. A possível regeneração dos receptores do LCA pode explicar a diferença dos resultados registrados na literatura.

Respostas reflexas da musculatura

Os receptores proprioceptivos, no interior do LCA, ocupam uma extensão entre 1 a 2,5% do volume total deste ligamento (Zimny et al. 1986; Schutte et al. 1987). A função destes receptores seria sinalizar as tensões a que esse ligamento é submetido durante os movimentos do joelho. Ao serem estimulados, estes receptores geram potenciais de ação que podem ser transmitidos pelas vias aferentes para a medula e retransmitidos para os centros superiores do SNC. As informações aferentes também podem ativar, via reflexa, as vias eferentes que inervam as fibras extra-fusais, promovendo a contração dos músculos. A estimulação destes receptores geraria, então, uma resposta reflexa (Johansson et al. 1991).

Evidências em estudos com animais (Solomonow et al. 1987; Miyatsu et al. 1993) e em humanos (Tsuda et al. 2001) submetidos à cirurgia do joelho mostram que uma tração mecânica do LCA gera uma ativação reflexa dos ísquios-tibiais (Solomonow et al. 1987; Miyatsu et al. 1993; Tsuda et al. 2001). Já um aumento da pressão articular do joelho provoca uma inibição reflexa do quadríceps (Kennedy et al. 1982; de Andrade et al. 1965). Apesar de alguns estudos em animais demonstrarem uma possível circuitaria neural para esta ação reflexa (Solomonow et al. 1987; Miyatsu et al. 1993), nos humanos esta circuitaria ainda não foi mapeada. No entanto, baseando-se no tempo de

latência entre a tração mecânica do LCA e o início da atividade muscular, pode-se afirmar que se trata de uma circuitaria envolvendo várias sinapses (polissináptica) (Tsuda et al. 2001). Uma das dificuldades em identificar esta circuitaria neural é que os neurônios motores alfa, que inervam a musculatura, também recebem projeções provenientes de outras estruturas, como, por exemplo, os receptores da cápsula articular. De fato, Solomonow et al. (1987) demonstraram que a ativação de receptores da cápsula articular também pode ativar reflexamente a musculatura.

Kennedy et al. (1982) provocaram a distensão capsular infundindo 60 cc de solução salina intra-articular e, como consequência, ocorreu uma inibição do quadríceps em humanos. Para estes autores, a estimulação dos mecanoreceptores, via aumento da pressão intra-articular, seria o motivo da geração do reflexo de inibição do quadríceps. A infusão plasmática (de Andrade et al. 1965), a efusão aguda devido a uma menisectomia medial (Stokes & Young, 1984) e a efusão crônica do joelho também provocaram uma inibição do quadríceps com diminuição da força muscular (Fahrer et al. 1988; Jones et al. 1987). Assim, quando se tensiona anteriormente o LCA com o objetivo de ativar reflexamente a musculatura, também pode haver uma estimulação dos receptores da cápsula articular contribuindo para a ativação ou inibição reflexa da musculatura do joelho (Solomonow et al. 1987; Guanche et al. 1995).

Beard et al. (1993) e Wojtys & Huston (1994) demonstraram que indivíduos com lesão do LCA apresentam um atraso na ativação reflexa dos

ísquios-tibiais quando submetidos a uma tração mecânica no LCA, devido a um deslocamento passivo da tíbia no sentido anterior.

Wojtys & Huston (1994) deslocaram a tíbia anteriormente, aplicando uma carga de 13,5 Kg ao nível da região póstero-proximal, e observaram o tempo de reação muscular nos indivíduos com lesão do LCA (tempo entre o início do deslocamento passivo da tíbia e o início da atividade muscular do gastrocnêmio, ísquios-tibiais laterais e mediais, quadríceps medial e lateral). Comparado aos sujeitos controle, os tempos de reação foram maiores para os portadores de lesão do LCA para o músculo bíceps femural (Figura 02).

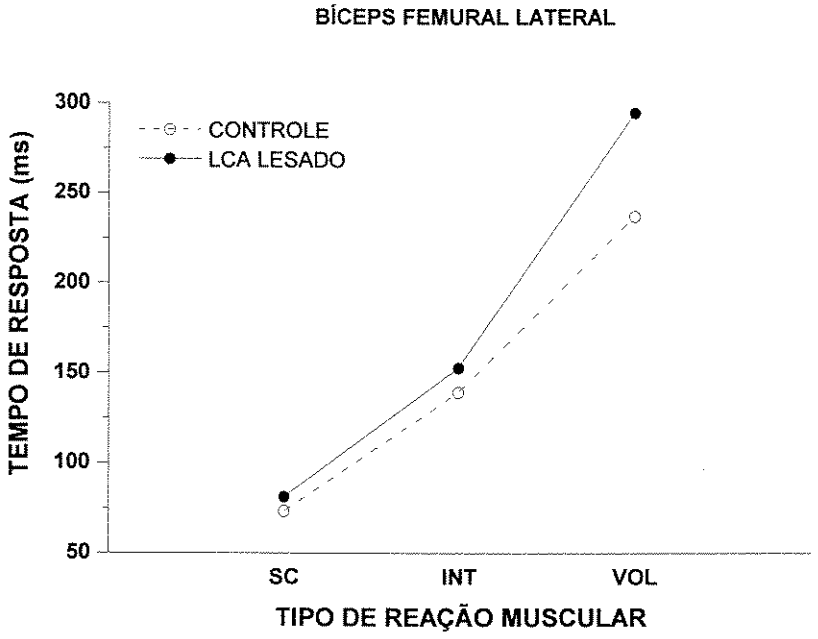

FIGURA 02: Tempo de reação muscular. Respostas de acordo com o tempo de latência em: i) reflexo monosináptico (SC); latência entre 20 a 119 msecs ii) resposta intermediária (INT); latência entre 130 a 170 msecs e iii) resposta voluntária (VOL); latência entre 220 a 369 msecs. Grupo controle (linha tracejada), Grupo com lesão do LCA (linha cheia). O tempo é dado em milisegundos. Adaptado dos dados de Wojtys & Huston (1994).

Resultados similares foram obtidos para os outros músculos estudados. Esses autores também observaram uma variabilidade em termos dessas

respostas nos diferentes grupos de lesionados do LCA (agudo, semi-agudo e crônico).

No entanto, o atraso na ativação reflexa dos ísquios-tibiais não foi observado em indivíduos com lesão do LCA testados em experimento similar (Jennings & Seeldhom, 1994). Dado a provável complexidade da circuitaria neural envolvida nesta resposta reflexa, e a variação no tipo de manipulação mecânica (deslocamento passivo da tíbia) usada para eliciar esta resposta reflexa, é natural que diferentes autores tenham observado resultados conflitantes.

Apesar da disputa da existência ou não de déficit na resposta reflexa devido à lesão do LCA, esses estudos têm tido uma grande influência na avaliação clínica da lesão do LCA e na implementação de tratamentos fisioterapêuticos para estes indivíduos. Acredita-se que a falta das informações proprioceptivas originadas do LCA provoque duas respostas distintas. Primeiro, ocorreria uma inibição reflexa do quadríceps, diminuindo a massa desta musculatura, fato que também é observado clinicamente (Lorentzon et al. 1989). Essa inibição também diminuiria a capacidade do quadríceps em gerar um torque extensor, que provocasse uma anteriorização excessiva da tíbia (Andriacchi 1990). Segundo, a falta dessas informações também provocaria uma ativação reflexa dos ísquios-tibiais (Konishi et al. 2002; Tsuda et al. 2001), o que promoveria o fortalecimento dessa musculatura (Branch et al. 1989). O objetivo dessas duas respostas seria o de minimizar a geração de uma força de translação na articulação do joelho, que provocasse a anteriorização da tíbia

(Branch et al. 1994; Tsuda et al. 2001). Assim, com a lesão do LCA, o fortalecimento dos ísquios-tibiais e a atrofia reflexa do quadríceps seriam uma resposta adaptativa para substituir a ação estabilizadora do LCA.

Essa lógica tem influenciado sobremaneira o tratamento fisioterapêutico de indivíduos com lesão e/ou submetidos à reconstrução cirúrgica do LCA, caracterizado por um fortalecimento seletivo dos ísquios-tibiais (Solomonow et al. 1989).

Ordem de recrutamento

A sequência na qual uma musculatura é recrutada tem sido estudada para inferir a ordem em que são gerados os torques nas articulações durante uma ação reflexa (Wojtys & Huston, 1994) ou voluntária (Lass et al. 1991; Kålund et al. 1990; Wojtys & Huston, 1994). A lesão do LCA provoca atraso na ativação do quadríceps, ísquios-tibiais e gastrocnêmio, durante a atividade reflexa induzida pelo deslocamento anterior da tíbia. No estudo de Wojtys & Huston (1994), esses atrasos foram observados nas fases aguda, semi-aguda e crônica da lesão do LCA. Entretanto, durante tarefas reflexas e voluntárias em indivíduos com lesão aguda, o quadríceps foi ativado antes dos ísquios-tibiais, enquanto nos indivíduos com lesão crônica os ísquios-tibiais precederam a ativação do quadríceps. Para esses autores, a antecipação da ativação do quadríceps seria uma resposta inapropriada, que ocorreria devido à perda das informações proprioceptivas provenientes do LCA lesado. Já os indivíduos crônicos teriam tempo para adaptar o sistema de controle motor à falta dessas informações.

No entanto, para Kålund et al. (1990), a ordem de recrutamento dos músculos que atuam no joelho parece estar preservada em indivíduos com lesão do LCA durante movimentos voluntários, como o andar num plano e numa rampa. O que mudaria, nos sujeitos portadores de lesão do LCA, seria o tempo de latência entre o início da atividade dos músculos, quando esses indivíduos são submetidos a uma carga maior. Assim, durante a marcha em terreno inclinado, ocorreria uma antecipação na ativação dos ísquios-tibiais, impedindo o deslocamento anterior da tíbia.

Note, na Figura 03, que, para o caminhar em esteira inclinada, os indivíduos com LCA lesado anteciparam a ativação dos ísquios-tibiais em relação ao quadríceps. A antecipação da ativação dos ísquios-tibiais, anterior ao contato do calcanhar ao solo, gera um torque flexor que evita a extensão do joelho e o deslocamento anterior da tíbia.

No entanto, é preciso lembrar que outros estudos não confirmaram a presença de alterações na ordem de recrutamento em indivíduos com lesão do LCA nas respostas reflexas (Jennings & Seedhom, 1994) e voluntárias (Lass et al. 1991). Uma comparação entre esses estudos é comprometida, uma vez que a ordem de recrutamento é estudada durante diferentes tarefas reflexas (Wojtys & Huston, 1994) e voluntárias (Kålund et al. 1990; Lass et al. 1991), envolvendo diferentes grupos de lesionados do LCA (agudo, semi-agudo e crônico) e usando diferentes manipulações.

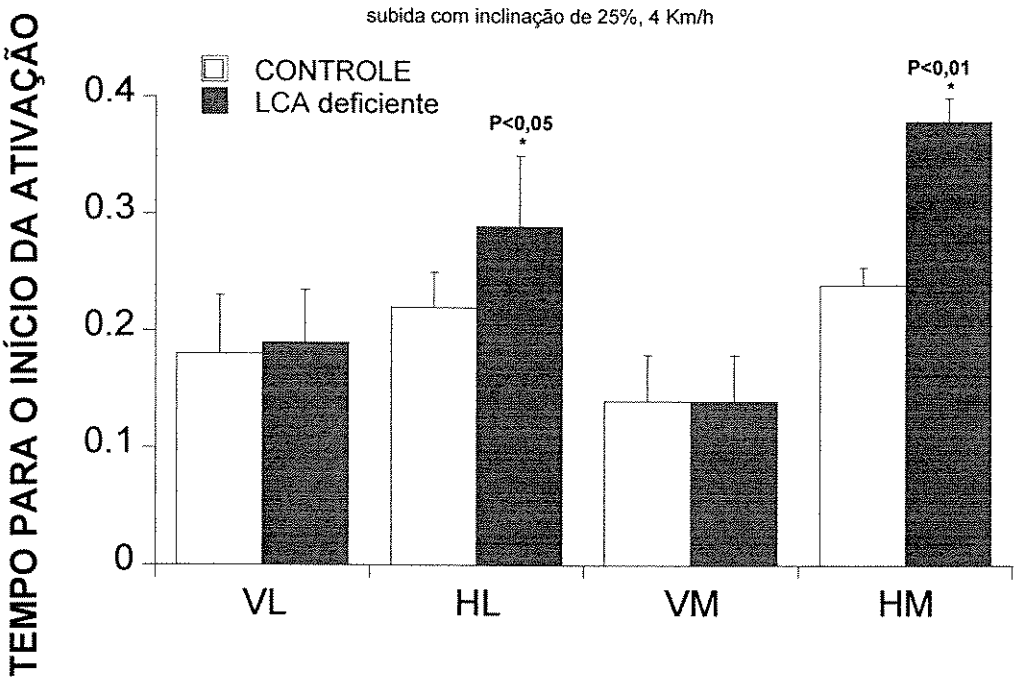

FIGURA 03: Tempo para o início da ativação dos músculos vasto lateral (VL), ísquio-tibial lateral (bíceps femural, HL), vasto medial (VM), e ísquio-tibial medial (semitendinoso, HM), durante a marcha a 4 km/h com inclinação de 25% no grupo controle (barra vazia) e com lesão no LCA (barra Çlistada). O tempo é dado em milisegundos e representa o período em que o músculo foi ativado antes que o calcanhar tocasse o solo. Asteriscos (*) indicam diferenças significantes entre os dois grupos e as barras o desvio padrão Kålund et al. (1990).

Quantidade e padrão de atividade muscular

Se a ordem de recrutamento determina quando a força será aplicada, a análise da quantidade e do padrão de atividade eletromiográfica (EMG) gerada pode revelar a quantidade de força muscular (Gottlieb et al. 1989).

Vários estudos observaram uma diminuição da quantidade de atividade EMG do quadríceps (Gauffin & Tropp, 1992; Branch et al. 1989) e um aumento dessa atividade para os ísquios-tibiais (Branch et al. 1989; Ciccotti et al. 1994) em indivíduos com lesão do LCA.

Em indivíduos submetidos à reconstrução cirúrgica do LCA, os padrões de atividade EMG do vasto medial, vasto lateral, reto femural, bíceps femural, semi-membranoso, tibial anterior, gastrocnêmio e soleus seriam normais durante várias atividades funcionais (i.e., caminhar, descer rampa, subir escadas e correr) (Ciccotti et al. 1994). No entanto, os indivíduos com lesão do LCA, submetidos a seis meses de programa de reabilitação, apresentaram um aumento da ativação do vasto lateral, bíceps femural e tibial anterior nessas atividades funcionais. A maior atividade desses músculos poderia evitar a rotação interna e a anteriorização da tíbia. A rotação tibial poderia provocar o fenômeno conhecido como "*pivoteamento*" (Ciccotti et al. 1994).

É possível que uma redução da atividade EMG do quadríceps (Branch et al. 1989), associada a um aumento da atividade do bíceps femoral (Ciccotti et al. 1994) e semitendíneo (Branch et al. 1989), evite o deslocamento anterior da tíbia durante o caminhar (Andriacchi, 1990; Ciccotti et al. 1994; Branch et al. 1989), pular (Gauffin & Tropp, 1992) e na flexo-extensão do joelho (Tibone et al. 1986). Já a maior atividade do bíceps femural e vasto lateral evitaria uma rotação interna da tíbia (Ciccotti et al. 1994; Zhang et al. 2002). Assim, a modulação da atividade desses músculos substituiria a ação que seria exercida pela LCA intacto. Porém, em movimentos simples, como a extensão do joelho (ver Capítulo II) e o agachamento (ver Capítulo III), não observamos mudanças, na ordem de recrutamento e na quantidade de atividade EMG da musculatura que cruza o joelho, em indivíduos com lesão do LCA ou com a reconstrução cirúrgica desse ligamento.

C. Fatores Biomecânicos

Mudanças cinemáticas

A lesão do LCA pode provocar mudanças em pequenos movimentos translacionais (Marans et al. 1989; Brandsson et al. 2001; Brandsson et al. 2002) e em movimentos amplos e rotacionais do joelho e quadril (Devita et al. 1997; Bull et al. 2002).

Movimentos translacionais e rotacionais e a firmeza do joelho

Os movimentos que ocorrem na articulação do joelho foram reconstruídos em seis graus de liberdade – sendo três translacionais (ântero-posterior, médio-lateral e súpero-inferior) e três rotacionais (interna-externa, valgo-varo e flexo-extensão) – utilizando um eletrogoniômetro específico para esta articulação. Porém, durante a fase de balanço da marcha, os portadores de lesão do LCA apresentaram cerca de 4,2 mm a mais de translação ântero-posterior da tíbia em relação ao fêmur, quando comparados com a média de 11,8 mm dessa translação observada nos indivíduos normais (Marans et al. 1989). Outros estudos demonstraram a presença de laxidão da articulação do joelho depois da ruptura do LCA (Gauffin & Tropp, 1992; Yack et al. 1993; Markolf et al. 1981; Lane et al. 1994).

Recentemente, Brandsson et al. (2001), usando a técnica de "análise radioesteriométrica dinâmica", mostraram que os indivíduos portadores de lesão do LCA mudam a cinemática dos movimentos do joelho. No entanto, esses autores, usando essa mesma técnica, não observaram mudanças na cinemática

dos movimentos do joelho entre os indivíduos com lesão do LCA e aqueles submetidos à reconstrução do LCA com o terço central do ligamento patelar (Brandsson et al. 2002), durante a movimentação do joelho de 55 graus de flexão até a extensão total. A variação média da amplitude nesses dois grupos de indivíduos foi de 6 e 2 graus, respectivamente, para a rotação interna/externa e a abdução/adução. Já os movimentos de translação foram de 2, 6, 5 mm, respectivamente, para a translação médio/lateral, proximal/distal, ântero/posterior.

Por outro lado, Bull et al. (2002) quantificaram a amplitude de movimento da tíbia em relação ao fêmur, nos testes específicos do LCA e de instabilidade articular do joelho, antes e depois da cirurgia de reconstrução do LCA, com o terço central do ligamento patelar. Após a cirurgia, a translação anterior da tíbia em relação ao fêmur foi reduzida no Teste da Gaveta Anterior de 13.2 para 5.9 mm (55%) e no de Lachman de 16 para 4.5 mm (72%). A reconstrução cirúrgica também provocou uma diminuição de 5 graus na rotação interna/externa passiva e a eliminação da subluxação anterior da tíbia com rotação interna, durante o teste clínico do *Pivot Shift*. Experimentos como esse mostram que a reconstrução cirúrgica do LCA pode promover uma melhora na firmeza articular durante toda a flexão do joelho (Gillquist & Messner, 1995; Bull et al. 1999).

Porém, não se pode negligenciar o elegante estudo de Brandsson et al. (2002) mostrando que a cirurgia de reconstrução do LCA não aumenta o grau de firmeza articular quando comparada com indivíduos lesados. No estudo de Brandsson et al. (2002), os sujeitos foram testados um ano após a cirurgia.

Logo, não se pode descartar a possibilidade de que o grau de estabilidade articular conseguido com a reconstrução cirúrgica diminui com o tempo.

Existe também uma variabilidade muito grande na amplitude e direção dos movimentos de translação do joelho entre indivíduos com lesão do LCA. Por exemplo, Bull e colaboradores (2002) demonstraram recentemente que o grau de translação e/ou rotação articular, para eliciar a positividade no teste do *Pivot Shift* (Galway et al. 1972; Losee, 1983), varia de indivíduo para indivíduo com lesão do LCA. Esta instabilidade, pode ser, inclusive, eliciada apenas pela translação ou pela rotação articular em alguns indivíduos e não necessariamente pela combinação dos dois movimentos, como demonstrado em cadáveres por Lane et al. (1994).

Não se pode negar a presença de instabilidade articular medida por testes clínicos (Losee, 1983; Kennedy et al. 1978; Galway et al. 1972) e pela reconstrução cinemática dos movimentos de translação de pequena amplitude em indivíduos com lesão do LCA (Bull et al. 2002; Brandsson et al. 2001; Marans et al. 1989). Porém, os estudos de um modo geral, nem sempre revelam uma correlação entre os resultados dos testes clínicos com a mensuração cinemática desses movimentos (Brandsson et al. 2002; Bull et al. 2002). Uma explicação para esse fenômeno é que os testes clínicos são feitos de forma passiva, impondo um distúrbio na articulação, que pode variar de examinador para examinador e que não necessariamente representam os distúrbios impostos na articulação durante os movimentos ativos.

A explicação para esses diferentes resultados deve estar em diferenças metodológicas observadas entre os estudos, como diferenças anatômicas entre os sujeitos, grau e extensão da lesão do LCA, envolvimento ou não de outras estruturas, tempo de lesão e, principalmente, falta de padronização na aplicação das forças durante os testes clínicos (Brandsson et al. 2002).

Apesar destas controvérsias, há um senso comum de que joelhos fortes, saudáveis e, principalmente, estáveis são essenciais para o sucesso de algumas funções motoras (Beard et al. 1993; Tibone et al. 1986). Porém, baseando-se na literatura citada acima, a instabilidade imposta pela lesão do LCA não necessariamente compromete a execução de todas as tarefas motoras. O que ainda não sabemos é se existiria um grupo específico de tarefas motoras capazes de impor uma anteriorização e rotação da tíbia, ao ponto de comprometer a performance motora.

Padrão flexor

De um modo geral, os indivíduos com lesão do LCA apresentam um padrão flexor do quadril e joelho durante a marcha (Berchuck et al. 1990; Devita et al. 1997). Devita et al. (1997) demonstraram que indivíduos com lesão do LCA e indivíduos com 3 e 5 semanas pós-reconstrução caminham com um padrão flexor durante a fase de apoio nas articulações do joelho e quadril. Com 5 semanas após a reconstrução, foi evidente uma tendência da cinemática articular do joelho de retornar a angulações próximas do observado para o grupo controle. Posteriormente, foi observado que, com 3 semanas pós-reconstrução cirúrgica do LCA, a amplitude angular dos movimentos durante a fase de

balanço da marcha foi, em média, 10% menor no quadril e 39% e 32% maior, respectivamente, para o joelho e tornozelo. Após 6 meses da cirurgia, essas amplitudes angulares também retornaram aos valores observados em indivíduos sem lesão do LCA (Devita et al. 1998). Em outras palavras, com o tempo, os indivíduos submetidos à reconstrução cirúrgica do LCA corrigiriam o padrão flexor da marcha, passando a adotar um padrão mais ereto. Resultados similares foram descritos por Gauffin & Tropp (1992) durante o salto.

Se existe um padrão flexor da marcha em indivíduos com lesão do LCA, ainda não está claro como a reconstrução cirúrgica do LCA, com o tempo, favorece o aparecimento de uma marcha mais ereta. Como veremos abaixo, é possível, inclusive, ocorrer uma mudança no padrão dos torques gerados na articulação do joelho, sem mudanças no padrão cinemático (Devita et al. 1998).

Porém, vários outros estudos não observaram mudanças na cinemática dos movimentos do joelho em indivíduos com lesão do LCA nos movimentos de extensão *("ver Capítulo II")*, na marcha (Devita et al. 1997; Roberts et al. 1999) e no agachar *("ver Capítulo III")*.

Mudanças cinéticas

Falseio

Solomonow et al. (1987) mostraram uma "falha no torque" extensor em alguns indivíduos com lesão no LCA, durante movimento isocinético concêntrico com força de extensão máxima do joelho. Esta queda do torque foi acompanhada por uma queda na atividade EMG do quadríceps na amplitude entre 37° a 46° de flexão, com um aumento na atividade eletromiográfica dos

ísquios-tibiais, para tentar evitar esta "falha do torque". Para Solomonow et al. (1987), esta queda no torque seria resultado de uma subluxação articular.

No entanto, em movimento isocinético concêntrico de flexo-extensão do joelho a 30°/s, não observamos um padrão de queda no torque, que pudesse estar associado ao comportamento descrito por Solomonow et al. (1987) nos cinco indivíduos portadores de lesão do LCA testados (Figura 04). Encontramos, também, resultados similares nas velocidades de 60, 90, 120 e 150°/s. Porém observamos essa queda no torque resultante em um indivíduo portador de lesão no LCA (dado não mostrado na Figura 04). Não sabemos a explicação para o fato de apenas alguns indivíduos apresentarem a queda no torque durante os movimentos isocinéticos concêntricos. Preferimos denominar o que Solomonow et al (1987) chama de falseio, apenas como uma queda abrupta do torque extensor resultante, pois estes autores não quantificaram o mecanismo translacional ou rotacional da tíbia em relação ao fêmur.

É possível que diferenças anatômicas ou no mecanismo de controle neuro-muscular, possam predispor alguns indivíduos a apresentar a queda do torque extensor resutante durante os exercícios isocinéticos, enquanto outros não apresentam esta queda.

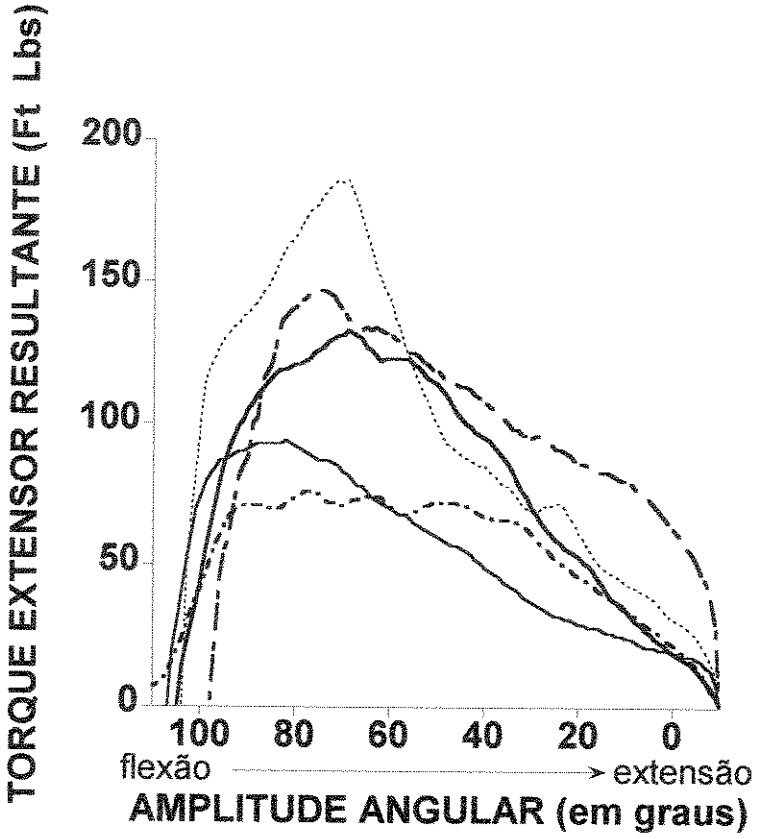

FIGURA 04: Torque extensor resultante no movimento isocinético concêntrico de extensão do joelho a 30°/s. Cada curva representa os dados de um dos seis indivíduos com lesão do LCA. Na amplitude angular de 0 graus, a articulação do joelho está estendida.

Predomínio de um padrão de torque flexor

Andriacchi (1990) e Berchuck et al. (1990) quantificaram, na plataforma de força de reação do solo, as alterações na magnitude do momento de flexo-extensão do joelho, durante a fase de suporte da marcha. Conforme ilustrado na Figura 05, os autores observaram que, durante a marcha, 75% dos indivíduos com lesão no LCA usam um torque interno na articulação do joelho predominantemente em flexão, evitando contrair o quadríceps, quando o joelho se encontra próximo ao momento de extensão total, durante a fase de apoio da marcha. Dessa forma, diminui-se a tração anterior da tíbia. Porém, os 25%

restantes dos indivíduos com lesão do LCA usaram o mesmo padrão de torque do joelho identificado nos indivíduos normais.

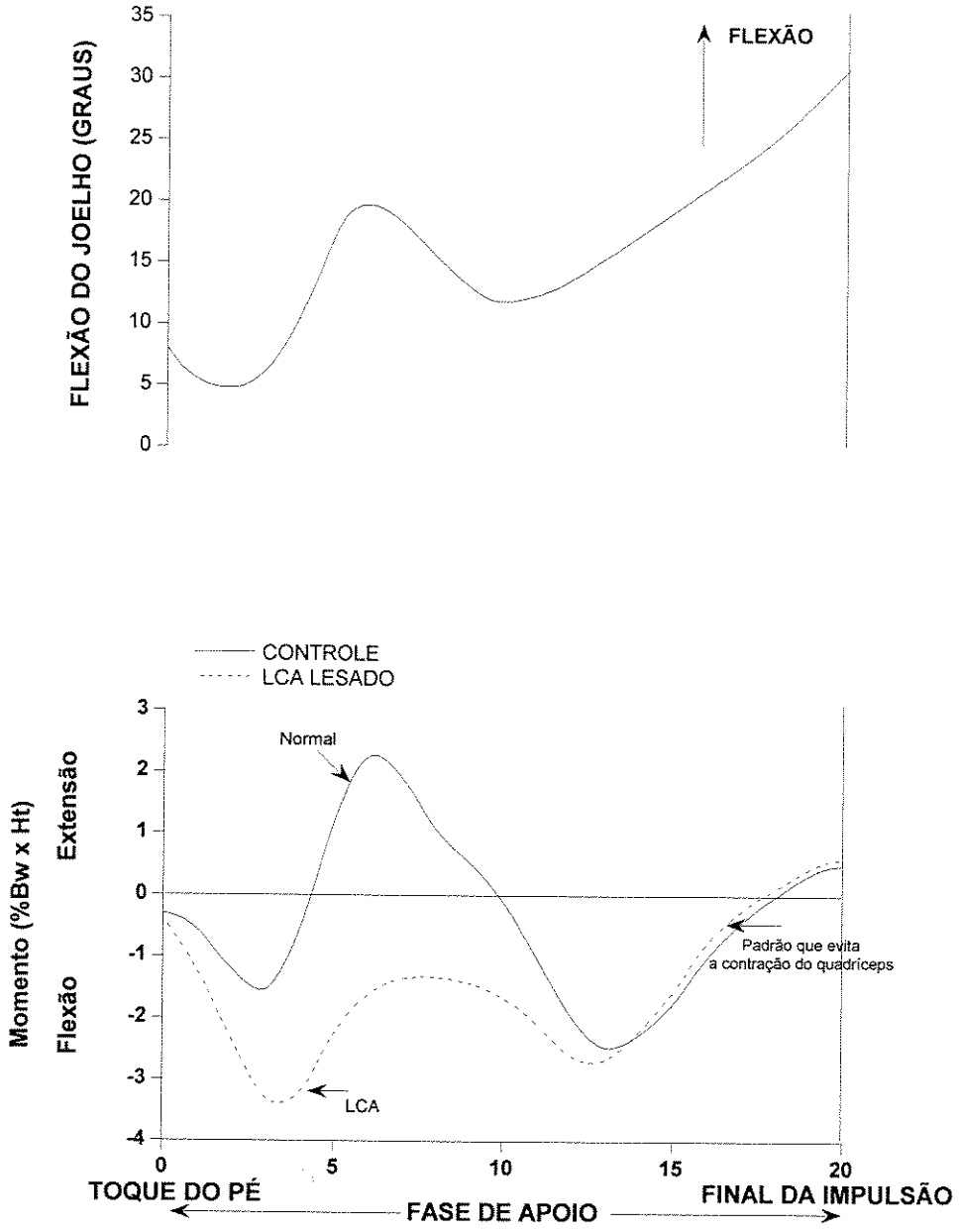

FIGURA 05: Relação entre o ângulo de flexão e o momento externo (flexão e extensão) do joelho durante a fase de apoio. A linha tracejada revela o padrão registrado em 75% dos indivíduos com LCA-deficiente. A linha cheia mostra o padrão do torque observado em indivíduos normais. Este padrão foi definido como "*quadríceps avoidance*" já que o momento externo não requer contração do quadríceps. (Berchuck et al. 1990).

Por outro lado, Devita et al. (1997) não identificaram em indivíduos com 2 semanas após a lesão do LCA o mesmo padrão de torque flexor do joelho descrito por Andriacchi (1990). Devita et al. (1998) mostraram também que os padrões cinemáticos do deslocamento do quadril, joelho e tornozelo foram similares durante a fase de apoio e de balanço entre indivíduos normais e com 6 meses após a reconstrução ligamentar. Porém, o padrão da variação do torque extensor do joelho no tempo (impulso), nos indivíduos com lesão do LCA na fase inicial do apoio, foi 57% inferior ao observado nos indivíduos normais. Resultados similares foram observados por Timoney et al. (1993) em indivíduos após 9 meses de reconstrução cirúrgica do LCA.

Assim, Devita et al. (1998) demonstraram que talvez o importante a se avaliar, nos indivíduos com lesão ou reconstrução do LCA, seja o impulso dos torques e não a cinemática (variação angular) dos movimentos.

A lesão do LCA tem sido também associada com o desequilíbrio entre grupos musculares (Zhang et al. 2002). Esses autores encontraram que o torque isométrico, nos indivíduos com lesão crônica, é 20% maior na rotação interna, quando comparado com a rotação externa. Já em indivíduos controle e com lesão aguda do LCA, essa relação é invertida, sendo o torque rotacional externo 20% maior do que o interno. Em outras palavras, a lesão crônica do LCA geraria uma fraqueza isométrica da musculatura que faz a rotação interna, ou, um fortalecimento dos rotatores externos. Estes dados suportam as observações de que ocorre um aumento da atividade do vasto lateral e do bíceps femural durante a marcha, na fase de suporte, nos indivíduos com lesão do LCA (Ciccotti

et al. 1994). Houve um predomínio do torque isométrico em abdução e extensão, respectivamente, sobre os torques isométricos de adução e flexão tanto nos indivíduos normais como nos com lesão crônica ou aguda do LCA. A reconstrução cirúrgica do LCA também ajudou na recuperação do desequilíbrio da força muscular. Esses autores atribuíram a fraqueza dos rotatores internos, nos indivíduos com lesão crônica, a um mecanismo de adaptação que evitaria uma sobrecarga no LCA e/ou a instabilidade articular.

Um desequilíbrio ou fraqueza muscular tem também sido atribuído à lesão do LCA em outros estudos (Tibone et al. 1986; Gauffin & Tropp, 1992; Kannus et al. 1992). Na avaliação isocinética, foi detectado uma perda da força de 14% para o quadríceps e 4% para os ísquios-tibiais (Tibone et al. 1986). Porém, depois de oito anos da lesão do LCA (Kannus et al. 1992), os torques isocinéticos, extensor e flexor retornaram aos valores observados em indivíduos normais. No entanto, comparado com o lado não lesado em velocidade média-alta (180°/s), houve uma perda de até 81% da força isocinética dos ísquios-tibiais e 39% do quadríceps (Kannus et al. 1992). Esses dados poderiam sugerir uma perda seletiva das fibras de contração rápida devido à lesão do LCA. Entretanto, é preciso salientar que, nesses estudos (Kannus et al. 1992; Tibone et al. 1986; Gauffin & Tropp, 1992), foi comparado o membro lesado com o contra-lateral. Porém a lesão ligamentar em um joelho pode também interferir na funcionalidade do membro contra-lateral (Corrigan et al. 1992). Também é preciso considerar que, nos experimentos isocinéticos em sistemas de

dinamometria computadorizados, são avaliados os movimentos em planos padronizados e normalmente simulando esforços máximos e não funcionais.

Implicações Práticas

Existem vários protocolos de atendimento fisioterapêutico para os indivíduos com lesão do LCA (Blackburn Jr, 1985; Wilk et al. 1997) ou submetidos à reconstrução cirúrgica desse ligamento (Irrgang & Harner, 1997; De Carlo et al. 1997; Mangine & Krechek, 1997). De um modo geral, esses programas podem ser divididos em tratamento fisioterapêutico conservador (Blackburn Jr, 1985) ou acelerado (Shelbourne & Nitz, 1990; De Carlo et al. 1997).

O tratamento fisioterapêutico conservador e o acelerado visam a reabilitação de indivíduos com lesão ou reconstrução do LCA. A diferença do tratamento conservador para o acelerado é que, no acelerado, o tratamento inicia logo após a lesão e visa o retorno do indivíduo que foi submetido à reconstrução ligamentar, à prática esportiva ou às suas atividades funcionais, o mais brevemente possível. Infelizmente, a maioria desses protocolos não foi testada cientificamente para se comprovar a sua eficácia. Os autores desses protocolos, muitas vezes, sequer apresentam uma explicação técnico-científica que justifique o tratamento proposto. No máximo, argumentam que o protocolo é baseado na experiência clínica do fisioterapeuta e que "funciona". Portanto, somos chamados a creditar na experiência empírica do autor. No entanto, mais recentemente, foi demonstrado cientificamente o efeito benéfico da aplicação de um programa acelerado sobre o tradicional (De Carlo et al. 1997).

Com base na revisão de literatura citada acima, gostaríamos de sugerir os seguintes princípios gerais para o tratamento fisioterapêutico dos indivíduos portadores de lesão do LCA ou submetidos à reconstrução desse ligamento.

1) Princípio da individualização do tratamento

Como demonstrado anteriormente, apesar do LCA ser um ligamento altamente resistente, vários fatores podem causar sua lesão (Johnson et al. 1992). A ruptura do LCA desencadeia respostas neuromusculares e biomecânicas bastante complexas e não totalmente esclarecidas (Wojtys & Huston, 1994). Uma das explicações mais plausíveis para os conflitantes achados neuromusculares e biomecânicos, observados na literatura, em decorrência da lesão ou reconstrução cirúrgica do LCA, é a variabilidade entre os indivíduos. Essa variabilidade não é apenas anátomo-fisiológica, mas também está presente nos agentes causadores da própria lesão e na sua extensão.

Portanto, o primeiro princípio, para se estabelecer um programa de tratamento fisioterapêutico para essa clientela, é realizar uma avaliação individualizada, que considere os dados da anamnese e os testes específicos e complementares da clínica médica e fisioterapêutica.

1.1 Exame físico

No exame físico, a presença de edema, hipotrofia do quadríceps, cicatrizes de traumas ou cirurgias anteriores, bem como alterações na marcha e limitação de movimento, é típica de lesões ligamentares do joelho. Na palpação,

possível presença de dor, edema, relações anormais das superfícies articulares, crepitações também são informações expressivas. A presença de crepitação é um sinal típico de degeneração articular, a qual, a longo prazo, pode ser uma complicação das lesões ligamentares.

Pode haver limitação da mobilidade articular devido à dor e ao espasmo, debilidade muscular, rigidez, contratura articular ou bloqueio articular. O movimento passivo pode estar diminuído, por alguma das razões anteriores, ou aumentado, no caso de frouxidão ou ruptura ligamentar. Assim, uma hiperextensão do joelho será possível e deve ser investigada bilateralmente para distinguir se o aumento da translação anterior da tíbia em relação ao fêmur, investigada nos testes da Gaveta Anterior e de Lachmann, ocorre devido à lesão do LCA ou devido à frouxidão ligamentar bilateral.

1.2 Anamnese

Na anamnese, deve ser investigada a história do paciente/cliente, com ênfase no acidente que gerou a lesão e na queixa principal do paciente, relacionando os sintomas como dor, edema, falseios ou relato de instabilidade no joelho. A seguir, investiga-se as queixas secundárias relacionando-as à história pregressa, direcionando-as a possíveis associações com outras afecções ou, mesmo, uma possível recidiva. Com relação à gênese da lesão, como normalmente, a lesão ocorre nos esportes, o mecanismo causal da lesão deve ser investigado. Normalmente, são movimentos típicos que sobrecarregam a articulação, com ou sem contato físico, ou, mesmo, por traumas diretos em acidentes automobilísticos e motociclísticos.

História familiar, com relação às alterações congênitas ou adquiridas do sistema músculo-esquelético, deve ser considerada. Uma história familiar de lesões ligamentares pode ser um fator de risco de origem hereditária.

1.3 Evolução Clínica

Com relação aos sinais e sintomas das lesões agudas e crônicas do LCA, NOYES et al. (1983c) demonstraram que a lesão aguda do LCA, na maioria das vezes, apresenta sinais e sintomas característicos como dor até 24 horas, edema até uma hora após a lesão inicial, estalido no momento da lesão, limitação de movimento até 24 horas da lesão inicial e dificuldade ou incapacidade para continuar a atividade esportiva.

Entretanto, após melhora do quadro, a pessoa poderá voltar à prática esportiva ou às atividades da vida diária e, posteriormente, apresentar recidivas. Deste modo, a ocorrência das torções secundárias é comum nas manifestações crônicas das lesões ligamentares.

A lesão crônica é caracterizada no relato pelo paciente de falseio ou instabilidade articular. O problema principal é que as recidivas podem afetar outras estruturas, como meniscos e/ou ligamentos, que não foram atingidos na lesão inicial, os quais atuam auxiliando o papel biomecânico do LCA. Com o retorno do cliente à atividade física, principalmente àquelas que solicitam mudanças de direção, saltos e giros, pode haver um alto risco de recidiva (NOYES et al., 1983c).

Com o decorrer do tempo, o indivíduo poderá apresentar agravo do quadro e relatar incapacidade funcional, a qual está relacionada a episódios de

falseio e instabilidade articular. Estes episódios de subluxações repetidas com suporte de peso causam dano significante para a articulação, originando defeitos na cartilagem articular, lesões meniscais e frouxidão dos restritores secundários, tanto os capsulares como os ligamentares (NOYES et al., 1985).

1.4 Testes específicos

Os testes de Lachman (Torg et al., 1976; Wroble & Lindelfeld, 1988) e da Gaveta Anterior (Hughston et al., 1976a) caracterizam-se como manobras específicas para investigação da integridade do LCA. O teste clínico utilizado para investigar a estabilidade da articulação do joelho e integridade do LCA é o *Jerk Test de Hughston* (Hughston et al. 1976b), o qual é empregado para avaliação da instabilidade ântero-lateral do joelho. Esta manobra consiste no deslocamento anterior excessivo e rotação interna do platô lateral da tíbia sobre o fêmur. A posivitidade desta manobra provavelmente indica, ruptura do LCA (Hughston et al., 1976b). O *Pivot Shift Test* ou *Teste de Mac-Intosh* (Losee, 1983; Galway et al. 1972) é o inverso do *Jerk Test*", provocando não uma subluxação articular, mas uma redução.

Para Bull et al. (2002), o *Pivot Shift Test* é mais consistentemente descrito como uma translação do platô tibial do que como uma rotação. Apesar destes testes serem indicativos da integridade do LCA ou da estabilidade articular, Brandsson et al. (2002) e Bull et al. (2002) não observaram uma relação significante entre os testes clínicos (*Teste de Lachman* e o *Pivot Shift Test*) e a medida da translação da tíbia. O maior problema, nesses experimentos, é que

as cargas impostas para realizar esses testes variam muito entre examinadores. O segundo maior problema são as diferenças individuais.

1.5 Testes complementares

Exames de radiografia e ressonância magnética podem auxiliar na investigação da lesão ou da reconstrução ligamentar. Exames radiográficos contribuem para detecção de fraturas condrais ou para a análise do ponto de fixação do enxerto.

Os exames de imagens de ressonância magnética do joelho auxiliam na comprovação da lesão do ligamento cruzado anterior. O índice de acerto (validade interna) destes exames é de 92%. Caracteriza-se por um exame não invasivo, sem riscos aos sujeitos pesquisados. Apesar de sua eficácia ser menos efetiva em relação ao exame clínico, conforme evidenciaram recentemente Liu et al. (1995) e Gelb et al. (1996), este exame facilita a confirmação da lesão ligamentar.

1.6 Avaliações biomecânicas

Os exames biomecânicos e de controle motor ainda estão restritos principalmente a laboratórios bastante sofisticados e são usados principalmente na investigação científica. Estas análises, fundamentam os padrões cinemáticos (ângulo, velocidade e aceleração do movimento), cinéticos (torque), da atividade eletromiográfica (ordem de recrutamento, quantidade e padrão de atividade muscular) gerando informações individualizadas sobre a quantificação de movimentos funcionais como a marcha, salto, agachamento, movimento de flexo-extensão e rotacionais do joelho entre outros. Estas análises devem ser

comparadas com o início, as fases intermediárias e o final do tratamento para possibilitar a quantificação das alterações motoras. Apesar destas avaliações não serem usadas no dia a dia da prática médica e fisoterápica, a importância destas análises para acompnahar a evolução do tratamento não carece de contestação.

2. Princípio do condicionamento pré-cirúrgico

Na fase pré-reconstrução, algumas medidas devem ser tomadas para promover uma adequada reabilitação pós-reconstrução (De Carlo et al. 1997). Entre estas se destacam: restabelecimento total da amplitude de movimento, ausência de edema e dor, diminuição do padrão de marcha flexora, recuperação da força muscular e preparação mental para a reconstrução. Essas intervenções fisioterapêuticas, implementadas na fase pré-cirúrgica, têm mostrado uma aceleração na fase de recuperação pós-cirúrgica (De Carlo et al. 1997).

3. Princípio da atrofia reflexa do quadríceps

Logo após a lesão inicial e a subsequente cirurgia é comum observar edema e dor na articulação do joelho. Existe uma grande possibilidade de que a atrofia do quadríceps, observada nesses indivíduos (Kennedy et al. 1982; de Andrade et al. 1965) seja causada por estímulos oriundos dos receptores da cápsula articular, que são ativados pelo aumento da pressão intra-articular provocada pelo edema (de Andrade et al. 1965).

Na fase aguda da lesão e no pós-cirúrgico, a fisioterapia deve objetivar principalmente diminuir o edema de forma a evitar a possível inibição reflexa do quadríceps. Para tanto, recomenda-se o uso de gelo e a manutenção do

membro inclinado contra a ação da gravidade, combatendo principalmente o edema.

4. Princípio da dor na limitação da amplitude de movimento

A limitação da amplitude de movimento observada no pós-lesão e no pós-cirúrgico pode também ser provocada pela presença do edema e da dor. Com a diminuição do edema e da dor, pode-se começar a trabalhar os movimentos passivos para recuperar a amplitude de movimento.

Mais raramente, também podem ser observados episódios de bloqueio na amplitude articular. Nesse caso, deve-se investigar a origem do bloqueio articular, o qual pode ser de origem verdadeira (causado por corpo livre, menisco ou o próprio LCA) ou falso (causado por manifestações antálgicas, como por exemplo, devido à lesão do ligamento colateral medial). No caso dos bloqueios de origem verdadeira, a intervenção cirúrgica pode ser necessária.

5. Princípio da graduação da tração no LCA

A tração a que o LCA é submetido varia com o tipo de tarefa e o modo de contração (Johnson et al. 1996) e aumenta com a extensão do joelho (Beynnon et al. 1997).

Na fase inicial da lesão do LCA e/ou da sua reconstrução cirúrgica, recomenda-se prescrever tarefas que envolvam pouca ou nenhuma tração no LCA. Por exemplo, uma contração isométrica dos ísquios-tibiais e do quadríceps próximo à flexão do joelho geraria uma tração mínima no LCA.

Nos indivíduos lesionados, o objetivo dessas tarefas é evitar gerar subluxação adicional do joelho que agrave o quadro clínico e provoque lesões

associadas. Nos indivíduos com reconstrução, as tarefas com pouca tração no LCA protegeria o neoligamento de uma sobrecarga que pudesse produzir um comprometimento no implante.

Nas fases mais avançadas do tratamento desses indivíduos, pode-se gradativamente introduzir tarefas que exijam mais esforço, como a contração isométrica feita nas amplitudes próximas da extensão a 15 graus.

6. Princípio do equilíbrio muscular

Um quadríceps mais fortalecido em relação aos ísquios-tibiais pode predispor o indivíduo a uma anteriorização da tíbia, colocando uma grande tração no LCA, o que pode gerar uma tensão excessiva no ligamento (Andriacchi 1990). Da mesma forma, predomínio dos rotatores externos sobre os internos poderá predispor ao entorse e a uma lesão no LCA (Zhang et al. 2002).

Para prevenir a lesão do LCA, deve-se evitar o agente causador, como movimentos intensos ou traumas, que geram a abrupta anteriorização e rotação interna da tíbia provocando a lesão do LCA (Ciccotti et al. 1994). Geralmente esses estresses não podem ser previstos, principalmente nos esportes de contato e durante pivoteamentos em terrenos irregulares. Sempre que o impacto for maior do que a capacidade do LCA de suportá-lo ocorrerá inevitavelmente a lesão.

Para prevenir a lesão do LCA, recomenda-se uma análise criteriosa da existência de desequilíbrios musculares entre os grupos agonistas e antagonistas. Um fortalecimento seletivo e redução do tempo de resposta muscular dos ísquios-tibiais e rotatores externos do joelho podem, dependendo

do grau do impacto, evitar uma sobrecarga no LCA, evitando a lesão do ligamento (Ciccotti et al. 1994).

A atrofia seletiva do quadríceps é uma resposta adaptativa do sistema de controle motor para evitar a anteriorização da tíbia (Andriacchi 1990). Assim, deve-se tolerar e até promover um fortalecimento seletivo dos isquíos-tibiais na fase aguda da lesão do LCA. Esse fortalecimento ajudará a evitar sobrecargas adicionais no joelho que geram uma anteriorização da tíbia.

Na reconstrução cirúrgica do LCA, só faz sentido promover um fortalecimento seletivo dos ísquios-tibiais por precaução. O neoligamento deve ser mais resistente que o LCA e, com a cicatrização total do procedimento cirúrgico (por volta de 3 meses segundo Abe et al. 1993), deve estar preparado para suportar as constantes trações no joelho promovidas pela anteriorização da tíbia.

7. Princípio da periodização do treinamento

Para respeitarmos os princípios da periodização do treinamento (Zatsiorsky, 1993), exercícios de resistência, com séries de longas repetições e baixas cargas, devem ser introduzidos na fase inicial do tratamento, e exercícios de força com poucas repetições e altas cargas, posteriormente. Esse princípio é válido tanto para os indivíduos com lesão do LCA como para os reconstruídos.

Em indivíduos após três semanas de lesão do LCA, deve-se investigar se o indivíduo relata instabilidade articular ou se ocorrem os indesejáveis reentorses. É uma fase na qual é possível começar a introduzir exercícios de resistência, enfatizando toda a musculatura da perna.

O treinamento nessa fase deve enfatizar o fortalecimento dos ísquios-tibiais e rotatores externos para que esses músculos possam gerar um torque flexor e de rotação externa no joelho, que evite a anteriorização da tíbia com rotação interna. Em outras palavras, a ação desses músculos poderia substituir a função do LCA, promovendo a estabilidade articular com a finalidade de evitar possíveis falseios (Ciccotti et al. 1994; Bull et al. 2002).

No final do processo de reabilitação, os exercícios devem ser voltados para a funcionalidade do indivíduo e devem ser trabalhadas as mais variadas velocidades de movimento, a fim de preparar o sistema músculo-esquelético e de controle motor a reagir a uma variabiliade de estímulos.

8. Princípio da eficiência máxima

Funcionalmente, a importância do quadríceps para um esportista de alto nível do futebol, com grande explosão muscular e potência nos chutes, não pode ser desprezada. Neste sentido, se for estabelecido um programa de reabilitação específico para indivíduos com lesão do LCA, estimulando o fortalecimento seletivo dos ísquios-tibiais, certamente que suas qualidades funcionais serão alteradas e, provavelmente, prejudicadas devido ao novo jogo de forças enter agonstas e antagonistas. Então, a grande perspicácia do fisioterapeuta será identificar até que ponto o programa deste indivíduo pode ser voltado para preservar as suas qualidades motoras e promover padrões de segurança, a fim de evitar novas lesões no joelho com o retorno ao esporte.

O fisioterapeuta deve sempre manter em mente que exercícios que impõem rotação no joelho e anteriorização da tíbia, como, por exemplo, a corrida

em zigue-zague, devem ser introduzidos apenas na fase final da reabilitação dos indivíduos com reconstrução cirúrgica do LCA. Estes movimentos são caracteristicamente de risco à lesão ou relesão deste ligamento (Fatarelli, 1997) e devem ser evitados pelos indivíduos com lesão do LCA em fases iniciais do tratamento.

Se houver história de recidiva na atividade esportiva de hábito do indivíduo, orientações devem ser promovidas para a mudança de atividade física.

9. Princípio da recuperação proprioceptiva

Existe muita controvérsia sobre a possível perda ou não da propriocepção em indivíduos com lesão do LCA (Corrigan et al. 1992 *versus* Jennings & Seedhom, 1994). Porém, não temos nenhum dado científico mostrando que essa possível perda de propriocepção possa comprometer a funcionalidade do joelho em indivíduos com lesão e reconstrução cirúrgica do LCA. Portanto, precisamos ser bastante conservadores ao propor um treinamento proprioceptivo para esses indivíduos como meio de se recuperar a funcionalidade do joelho. Estes treinamentos proprioceptivos são fundamentados em exercícios que estimulam a reposta muscular, provocando uma diminição na latência ou mesmo uma otimização na modulação desta resposta. Como exemplo, podem ser gerados distúrbios mecânicos com uma carga imposta posteriormente na tíbia. Esta carga pode provocas a translação anterior da tíbia em relação ao fêmur e como consequência aumentar o estresse gerado no LCA devido a esta translação, promovendo a resposta reflexa dos

ísquio-tibiais (Wojtys & Huston, 1994). Outra possibilidade é promover o ganho da propriocepção através de exercícios que estimulem a capacidade do indivíduo detectar a posição estática ou dinâmica da articulação no espaço (Corrigan et al. 1992).

10. Princípio da velocidade do movimento

As velocidades funcionais do indivíduo devem ser enfatizadas na fase anterior ao retorno à prática esportiva. Ou seja, indivíduos que fazem esportes de explosão, como 100 metros rasos no atletismo, devem ser trabalhados nas fases avançadas com exercícios que promovam o ganho de força em velocidades angulares altas, como por exemplo 400°/s.

11. Princípio do tratamento bilateral

Como vimos, não existe um único membro prejudicado (Corrigan et al. 1992) e os efeitos da lesão de um membro podem afetar o desempenho do lado contra-lateral (Fremerey et al. 2000; Corrigan et al. 1992). Visto que, parte das lesões do LCA são bilaterais (Fatarelli, 1997), o tratamento do membro contra-lateral deve ser enfatizado no sentido de promover padrões cinemáticos e cinéticos semelhantes entre os dois membros.

12. Princípio da autonomia do paciente

Vários indivíduos com lesão do LCA adaptam-se muito bem à lesão e conseguem realizar as atividades da vida diária com desenvoltura (Noyes et al. 1983c). Não sabemos ainda porque essa adaptação ocorre muito bem com um grupo desses indivíduos. Nesse contexto, não surpreende o fato da indicação

cirúrgica ser baseada em critérios subjetivos como funcionalidade do paciente e histórias de reentorses.

No caso de estar prejudicada a funcionalidade do indivíduo, com manifestações como falseios e dor na marcha, ou com o comprometimento da performance no caso dos esportistas, a reconstrução cirúrgica do LCA é melhor justificada.

No entanto, deve-se respeitar o princípio da autonomia do paciente na decisão sobre a cirurgia. O cirurgião mostrará para o paciente os prós e contras da cirurgia, baseando-se no conhecimento científico, para que o paciente possa tomar uma decisão mais sábia.

HIPÓTESES A SEREM TESTADAS COM BASE NA REVISÃO DA LITERATURA

Considerando os conhecimentos mais recentes da literarura discutidos no Capítulo I e as divergências verificadas nos experimentos que analisam as alterações cinemáticas e eletromiográficas em indivíduos com lesão ou reconstrução do LCA, foram realizados quatro procedimentos experimentais para investigar as modificações e adaptações do sistema de controle motor devido a lesão ou a reconstrução do LCA.

No Capítulo II foram formuladas as hipóteses de que a instabilidade mecânica secundária à lesão do LCA, afetaria os padrões cinemáticos e da atividade muscular levando a um déficit motor funcional durante movimentos de extensão do joelho. Já a reconstrução normalizaria estas variáveis devido ao retorno da estabilidade articular, a qual é provocada pela cirurgia.

No Capítulo III foram analisadas as hipóteses que a lesão do LCA causaria um acoplamento cinemático distinto entre os movimentos das articulações do quadril, joelho e tornozelo. Já a recosntrução do LCA melhoraria a estabilidade articular, normalizando os padrões cinemáticos e eletromiográficos ao nível observado em indivíduos controle.

Na sequência, é proposto no Capítulo IV um modelo experimental para entender a estratégia de controle motor utilizada por indivíduos controle para manter o joelho estendido contra a ação da gravidade durante uma contração isométrica logo após o movimento isotônico.

Com base na estratégia encontrada em indivíduos controle durante o modelo experimental investigado no Capítulo IV, o Capítulo seguinte analisa a hipótese que indivíduos lesados teriam dificuldades para modular a atividade eletromiográfica durante a contração isométrica devido aos danos causados pela lesão nos receptores proprioceptivos presentes no interior do LCA. Estes receptores exerceriam papel crucial na modulação desta atividade muscular. Já os indivíduos reconstruídos não apresentariam alterações cinemáticas ou eletromiográficas durante este tipo de contração, devido à restauração da estabilidade articular provocada pela cirurgia ligamentar, ou devido à regeneração dos receptores presentes no interior do neoligamento. Os resultados destas análises poderão conduzir a tomada de decisões relacionadas às intervenções fisioterápicas em indivíduos com lesão ou reconstrução do LCA.

CAPÍTULO II – "The lesion and reconstruction of the Anterior Cruciate Ligament do not affect the control of fast voluntary knee extension movements"

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Key words: anterior cruciate ligament, motor control

ABSTRACT

Lesion of the ACL can generate mechanical instability of the knee and changes in the pattern of muscle activities with functional deficits in the knee movements. The decision for reconstruction surgery of this ligament is made based mainly on the clinical judgment of existing joint instability and functional motor deficit. We tested a hypothesis whereby the mechanical instability secondary to the lesion of the ACL would affect the patterns of muscle activity leading to a functional motor deficit. Subjects without lesion (n=10), with lesion of ACL and mechanical instability of the knee (n=10) and with surgical reconstruction of the ACL (n=10) performed knee movements over three angular distances. Movement kinematics and activation patterns (electromyography, EMG) of leg muscles were recorded. The movement speeds were bell-shaped and increased with movement distance. All three groups of subjects showed similar indices of accuracy. The amount of agonist and antagonist muscle activity increased with movement distance for all three groups of subjects. Our data show that the mechanical instability of the knee joint does not affect motor strategies used to perform fast voluntary extension movements of the knee joint.

INTRODUCTION

The main function of the anterior cruciate ligament (ACL) is to prevent the anterior displacement of the tibia over the femur (Fu et al. 1993; Fukubayashi et al. 1982). This ligament also acts on the internal rotational mechanism of the knee (Fukubayashi et al. 1982; Kennedy et al. 1974) and prevents the valgus and varus angulation of the knee (see, Fu et al. 1993, for reviews).

Lesion of this ligament provokes several changes in the motor control system that can affect motor performance. Proprioceptive deficits (Barrack et al. 1997; Beard et al. 1993; Borsa et al. 1997; Corrigan et al. 1992), abnormal electromyographic (EMG) patterns of the knee muscles (Ciccotti et al. 1994; Wojtys & Huston, 1994) and increased reaction time (Beard et al. 1993; Kålund et al. 1990; Wojtys & Huston, 1994) are some of the changes reported in the literature for individuals with ACL lesions. These changes have been associated with abnormal kinematics of the knee movements, such as modifications of the gait cycle (Andriacchi 1990; Berchuck et al. 1990), differences in dynamic balance (Gauffin and Tropp, 1992; Hoffman et al. 1999) and in the amplitude and pattern of anterior/posterior translation of the tibia (Marans et al. 1989).

However, these changes can be ameliorated with surgical reconstruction of the ACL (Ciccotti et al. 1994; Heier et al. 1997; Timoney et al. 1993). For example, Ciccotti et al. (1994) did not observe changes between control individuals and those submitted to surgical reconstruction of the ACL in the amount of EMG activity in several leg muscles during the performance of a

number of motor tasks. Other studies showed an improvement in various motor functions in individuals submitted to a surgical reconstruction of the ACL (Ciccotti et al. 1994; Heier et al. 1997; Timoney et al. 1993). On the other hand, a great proportion of the individuals with ACL lesion does not show functional deficit during the performance of tasks involving the affected knee joint (Andriacchi 1990; Berchuck et al. 1990).

Nevertheless, the decision for surgical reconstruction of the ACL is made based mainly on a clinical judgment of the existing joint instability and functional deficit (Christer et al. 1989; Clancy, 1985; Lysolm & Gillquist, 1982). The basic idea is that the reconstruction of the ACL would produce enough mechanical stability of the knee joint to allow it to overcome the loss of the proprioception. Compared to individuals with intact ACL, individuals submitted to surgery are expected to show normal patterns of muscle activity and indistinguishable motor function.

If this were the case, we would expect to observe changes in the electromyographic (EMG) patterns of knee muscle activity in individuals with lesion of the ACL that were not submitted to surgical reconstruction of this ligament and had mechanical instability of the joint. So, we hypothesized, first, that the lesion of the ACL would affect the patterns of muscle activity leading to a functional motor deficit in individuals with mechanical instability of the joint. Second, a surgical reconstruction of the ACL would prevent the mechanical instability of the joint and result in a more normal pattern of muscle activity and movement kinematics. We addressed these hypotheses by asking individuals

without lesion, with lesion and mechanical instability of the knee joint, and individuals with surgical reconstruction of the ACL to perform unconstrained fast knee movements over different distances. The findings are discussed in terms of functional changes observed in knee movements of individuals with ACL lesion and with surgical reconstruction of the ACL. The discussion is focused on biomechanical and neurophysiological models (Andriacchi 1990; Ciccotti et al. 1994; Fukubayashi et al. 1982; Fuss et al. 1992; Marans et al. 1989; Solomonow et al. 1989; Wojtys & Hustin, 1994) to explain the motor deficits observed in the individuals with lesion of the ACL.

METHODS

Subjects

We tested ten subjects without any neurological or orthopedic dysfunction (*Control Group*), ten subjects with lesion of the ACL that were not submitted to the reconstruction of this ligament and had mechanical instability of the knee (*Lesioned Group*), and ten subjects that had the ACL reconstructed surgically (*Reconstructed Group*) with a patellar tendon graft. All subjects of the three groups were male and tested after giving formal consent according to procedures approved by the State University of Campinas. Information about each of the subjects is shown in Table 01

TABLE 01: Clinical examination of all individuals of the three groups

Group of Subjects		Subjects									
		1	2	3	4	5	6	7	8	9	10
•	*Age (years)*										
CON		25.6	29.3	25.8	34.7	36.4	33.6	24	28.9	23.8	23
LES		19.9	27.5	40	35.6	27.8	33	28.6	26.5	27.3	24.9
REC		21.9	26.2	37.9	19.2	20.9	30.2	30.4	21	24.6	31.2
•	*Side of lesion or tested*										
CON		R	R	R	L	L	L	R	L	L	L
LES		R	R	L	L	L	R	L	R	L	L
REC		L	R	L	L	L	R	R	R	R	L
•	*Side of dominance*										
CON		R	R	R	R	R	R	R	R	R	R
LES		R	R	R	R	R	R	L	L	L	L
REC		R	R	R	R	R	R	R	R	R	R
•	*Edema and pain 24 hours after the lesion*										
LES		+	-	+	+	+	+	-	-	+	-
REC		-	-	-	+	+	+	+	+	+	+
•	*Associated lesions*										
LES		-	-	-	-	-	-	-	MM*	-	-
REC		-	-	BM	BM	BM	BM	-	MM	MM*	LM
•	*History of giving way*										
LES	*(BS)*	+	-	-	+	+	-	+	-	-	+
REC	*(BS)*	+	+	+	+	+	+	+	+	+	+
REC	*(AS)*	-	-	-	-	-	-	-	-	-	-
•	*Anterior Drawer Test*										
LES	*(BS)*	+	+	+	+	+	+	+	+	+	+
REC	*(BS)*	+	+	+	+	+	+	+	+	+	+
REC	*(AS)*	-	-	-	-	-	-	-	-	-	-
•	*Pivot Shift Test*										
LES	*(BS)*	+	+	+	+	+	+	+	+	+	+
REC	*(BS)*	+	+	+	+	+	+	+	+	+	+
REC	*(AS)*	-	-	-	-	-	-	-	-	-	-
•	*Lachman Test*										
LES	*(BS)*	+	+	+	+	+	+	+	+	+	+
REC	*(BS)*	+	+	+	+	+	+	+	+	+	+
REC	*(AS)*	-	-	-	+	-	-	-	-	-	-
•	*Time until the test (months)*										
LES	*(IL)*	48	48	60	25	70	40	69	60	61	16
REC	*(IL)*	40	34	20	21	41	45	24	19	26	59
REC	*(FS)*	38	32	10	10	20	28	21	17	25	47
•	*Type of reconstruction*										
REC		PL	PL	PL	PL	PL	PL	PL	PL	PL	PL

CON=control; LES=lesioned; REC=reconstructed *R=right; L=left*

BS=before surgery; AS=after surgery *IL= from the initial lesion; FS= from surgery*

*MM=medial meniscus; LM=lateral meniscus; BM=both meniscus; PL= patellar ligament graft; *condral lesion;*

All subjects of the *Lesioned Group* showed positive signs of lesion of the ACL in the anterior drawer test (Hughston et al. 1976) and Lachman's test (Torg et al. 1976; Wroble and Lindfeld, 1988). The pivot shift test (Galway et al. 1972; Losee, 1983) was positive for all subjects of the *Lesioned Group*. In addition to these specific tests, all subjects of the *Lesioned Group* were examined in the following complementary tests: abduction at 0 and 30° (Hughston et al. 1976), adduction at 0 and 30° (Hughston et al. 1976), McMurray's test (Crenshaw, 1989), and the posterior drawer test (Hughston et al. 1976). McMurray's test is designed to assess the integrity of the meniscus, whereas the other complementary tests are designed to assess the ligament integrity. Five out of ten subjects of the *Lesion Group* had a history of the knee giving way with recurrent sprain. The ligament rupture of the ACL was verified through magnetic resonance imaging. The results of the specific and the complementary tests are also presented on Table 01.

All subjects of the *Control Group*, and all subjects of the *Reconstructed Group* were tested negatively for all the specific and complementary tests. After the surgery, none of the subjects of the *Reconstructed Group* presented signs of giving way, new lesion, or arthrofibrosis of the reconstructed knee (Shelbourne et al. 1991). None of the subjects of the three groups felt pain during the test sessions, showed signs of ligament laxity, or decreased amplitude of movement of the knee joint. Also, none of the subjects of the three groups had any history of lesion of the meniscus or of ligaments of the contra-lateral knee. The dominant limb was determined by asking the participant which leg he preferred for kicking.

Procedure

At the initial position, the subject was comfortably seated on a chair, with the hip, knee, and ankle joints flexed at 90° (Figure 06). The foot was resting on the floor, in such way that it was not lifted when the knee extended, so the movement was one open chain. The hip movements were not constrained and only the trunk of the subjects was fixed with Velcro straps to the chair. In this position, the subject performed knee extension movements with the dominant limb over three target positions (30, 55 and 80°). He was asked to perform the movements "as fast as possible" and to keep the limb segment at the target position without making any voluntary correction. Reaction time was not stressed. Seven trials for each target distance were recorded.

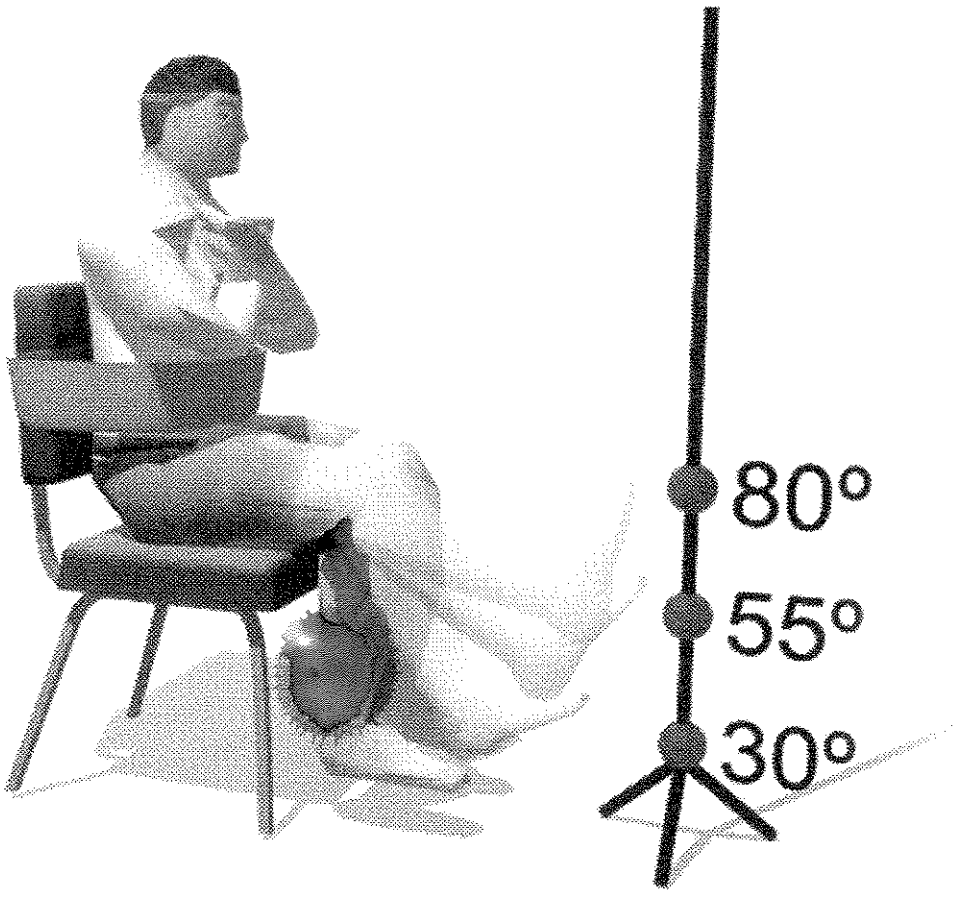

FIGURE 06: Illustration of the initial position of the subject and the three distances of the knee extension movements (30°, 55°, 80°).

Kinematic data

We recorded the X, Y and Z coordinates of the Light-Emission Diode (LED) markers, using a three-dimensional motion analysis system (OPTOTRAK3020-Northern Digital Inc.) at 100 frames per second. The LED markers were attached to the hip (superior iliac crist), the knee (lateral condyle) and the ankle joints (external malleolus). These coordinates were used to calculate the angular displacement of the knee in sagital plane. Angular velocity and acceleration were derived from the angle.

EMG recordings

We also recorded the muscle activity of the rectus femoris-RF, vastus medialis-VM, vastus lateralis-VL, semitendinosus-ST, biceps femoris-BF, gastrocnemius medialis-GM, gastrocnemius lateralis-GL and tibialis anterior-TA. These EMGs were recorded using bipolar surface EMG electrodes (DeLSys-model DE2.2L). The EMG signals were collected at 1000Hz, amplified (X 2000), band-pass filtered (20-450Hz) and recorded. Before data analyses, the EMG signals were rectified and low-pass filtered with a Butterworth filter with a cut-off frequency of 20 msecs.

QUANTIFICATION OF DATA

For each trial, the angle, velocity, and acceleration of the knee movements, and the EMGs of the eight recorded muscles were displayed on a screen monitor. The initial and final angles were visually identified to obtain the total angular excursion. We also identified the maximum peak velocity of the knee. The onset of each recorded muscle was taken from the point where the EMG signal first increased and was sustained above the baseline. The agonist EMG activity was integrated, for each muscle, from its onset until the time of the first zero crossing of the acceleration (at the time of the peak velocity). The antagonist EMG activity was integrated, for each muscle, from its onset until the time of the second cross zero of the acceleration (approximately at the end of the movement). These EMG indices were used to characterize the amount of activity for each muscle.

We calculated the baseline activity level for each muscle by integrating the EMG during the first recorded 100 ms (more than one second before the onset of the movement). We normalized each of the integrated EMG values, cited above, by dividing it by the corresponding baseline value. The time of the recruitment order for each muscle was calculated in relation to the time of the onset of the rectus femoris muscle.

STATISTIC ANALYSIS

The effects within subjects (target distances and type of muscles) and between subjects (the three groups) were analyzed for several of the dependent variables studied using a mixed ANOVA model. The alfa was set at .05.

RESULTS

Figure 07 illustrates the knee-extension movements performed over three angular distances by one subject of each group (A-Control, B-Lesioned, C-Reconstructed). The angular velocities of the knee were characterized by a bell shape and their peak increased with movement distance for all three subjects. Also, note that the angular excursions and velocities were smooth for all three subjects, even for the largest distance of 80°.

The patterns of the first EMG burst of the agonist (rectus femoris, vastus medialis, vastus lateralis) muscles were similar among these three subjects. The quadriceps muscles (rectus femoris, vastus medialis and vastus lateralis) were activated simultaneously and the amount of their EMG activity increased with the distance. Note that the onset of the EMG activity of these muscles occurred

around 100 ms (the vertical broken line) on the time scale. The EMG activity of the quadriceps was responsible for the generation of the muscle torque that accelerated the limb segment towards the target.

The antagonist muscles (biceps femoris and semitendinosus) were activated, in general, around 60 to 100 ms after the onset of the knee agonist muscles. The delay between the onset of the agonist and antagonist muscles is also called antagonist latency. The antagonist latency of the biceps femoris and semitendinosis muscles was not prolonged with target distances. The limb segment was decelerated towards the target by the action of the knee antagonist muscles, and by the action of the gravitational force.

The onsets of lateral and medial gastrocnemius, two other knee flexors, were anticipated to be similar to those of biceps femoris and semitendinosus. The medial and lateral gastrocnemius are bi-articular muscles and also act at the ankle joint. Note that both gastrocnemius muscles were activated simultaneously with the tibialis anterior, showing a pattern of co-activation of the muscles acting at the ankle joint. The data presented in Figure 07 for the three subjects are representative of the data obtained for all subjects of the three groups.

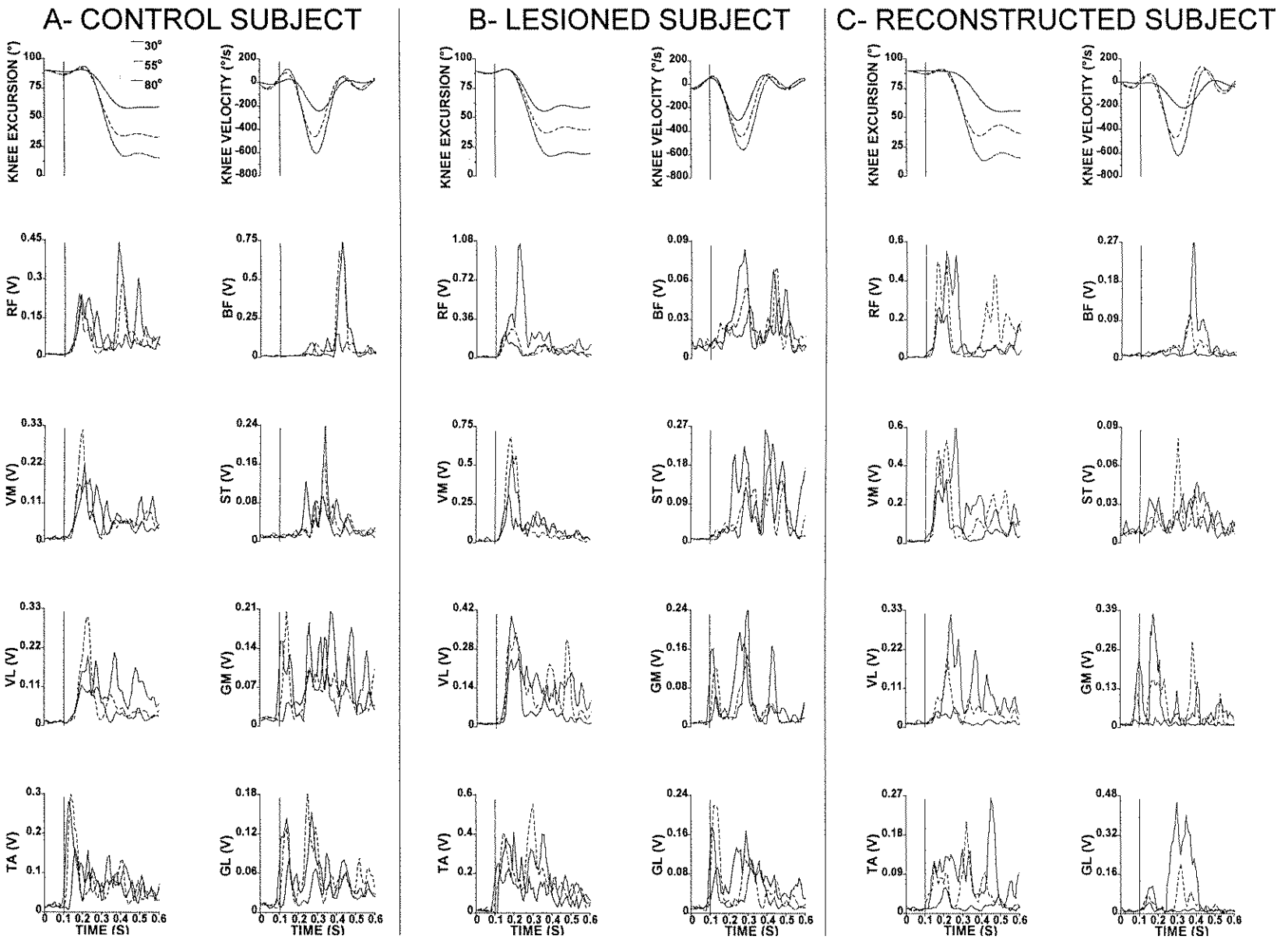

FIGURE 07: Angle and velocity of knee extension movements performed over three target distances (30º-solid line; 55º-broken line; 80º-dotted line) and the EMGs of Rectus Femoris (RF), Vastus Medialis (VM), Vastus Lateralis (VL), Tibialis Anterior (TA), Biceps Femoris (BF), Semitendinosus (ST), Gastrocnemius Medialis (GM) and Gastrocnemius Lateralis (GL). The data were aligned based on the onset of Rectus Femoris (vertical broken line). The data represent one trial performed by one subject of each group (A-control subject-left panel), (B-lesioned subject-middle panel) and (C-reconstructed subject-right panel). The time is in seconds, the angle in degrees, the velocity in degrees per second and the electromyographic activity in Volts.

All subjects performed the extension of the knee joint using a similar amount of EMG activity

Figure 08 shows that all individuals of the three groups performed the knee extension movements with similar angular excursion and peak velocity, which increased with movement distance. Also, note that the variability, as measured by the standard deviation of the angular excursion and velocity did not vary across all groups of subjects. The hip movement of all subjects of the three groups was below five degrees and did not vary across the three groups (data not shown here). The amount of agonist (rectus femoris, vastus medialis, vastus lateralis) and antagonist (semitendinosus, biceps femoris) EMG activity increased with distance and did not differ across the three groups. The subjects of the three groups also had similar amounts of EMG activity of the lateral and medialis gastrocnemius, and of the tibialis anterior.

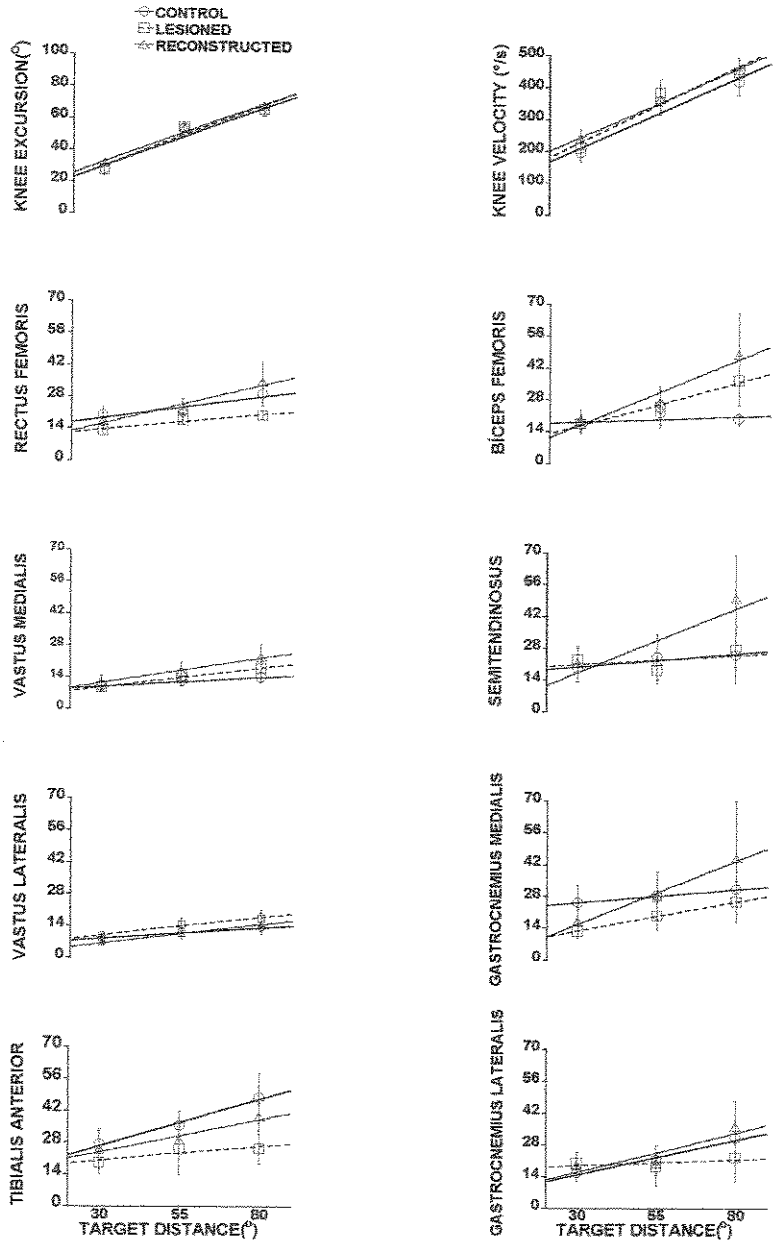

FIGURE 08: Angular excursion and velocity of the knee extension movements and the muscle activities of Rectus Femoris, Vastus Medialis, Vastus Lateralis, Tibialis Anterior, Biceps Femoris, Semitendinosus, Gastrocnemius Medialis and Gastrocnemius Lateralis. The data were plotted for the three target distances (30°, 55°, 80°) and were averaged for each of the three groups of subjects (open circle - control group; open square – lesioned group; open triangle – reconstructed group). The angles are in degrees, the velocity in degrees per second and the EMG activity is dimensionless.

The ANOVA tests revealed that the angular excursions and velocities of the knee movements and the amount of EMG activity of all recorded muscles did not change across the three groups. Note in Table 02 that there were no main effects on the groups of subjects (control *versus* lesioned *versus* reconstructed) for angular excursion, peak velocity, and the amount of EMG activity of the eight recorded muscles. However, all these variables increased with movement distance. For all these kinematic and EMG variables, there were no interactions between groups of subjects and target distances.

TABLE 02: Analysis of variance (ANOVA) for several quantities of the agonist and antagonist EMG activities, angular excursion and velocity of the knee movements.

		Group effect		Distance effect		Interaction group effect x distance	
		$F_{(2,54)}$	p	$F_{(2,27)}$	p	$F_{(4,54)}$	p
Knee angle		0.15	0.8639	586.20	0.00001	1.04	0.3921
Knee velocity		0.69	0.5075	256.79	0.00001	0.73	0.5772
Agonist and Antagonist burst	RF	0.595	0.5562	10.865	0.0001	0.985	0.4209
	VM	1.769	0.1829	57.406	0.0001	1.102	0.3611
	VL	1.763	0.1840	26.961	0.0001	0.263	0.9008
	TA	0.238	0.7893	8.324	0.0005	1.295	0.2799
	BF	0.772	0.4693	5.767	0.047	1.798	0.1381
	ST	0.990	0.3810	4.020	0.0219	0.57	0.9939
	GM	0.130	0.8782	5.116	0.0082	0.761	0.5537
	GL	0.176	0.8390	15.791	0.0001	1.164	0.3332

The effect of time of surgery

We compared the data from two sub-groups of subjects with ACL reconstructed with patellar ligament graft. One sub-group of six subjects was tested 10 to 21 weeks after surgery and the other, also composed of six subjects, 32 to 50 weeks from the time of surgery. The ANOVA showed no difference

between the two sub-groups, and the main effect of movement distance on all parameters is presented in Table 02. Also, there were no interactions between the sub-groups and the movement distances for all the parameters studied. The result of these ANOVAs are not presented here.

Recruitment order of all muscles

The onsets of the rectus femoris, vastus medialis, vastus lateralis, tibialis anterior, biceps femoris, semitendinosus, gastrocnemius medialis and gastrocnemius lateralis were calculated in relation to the rectus femoris. These data are shown in Figure 09. We ran three-way ANOVA with one factor among groups (Control *versus* Lesioned *versus* Reconstructed Group) and two factors within groups (movement distances and muscle groups). These analyses showed that movement distance did not affect the recruitment order of the muscles ($F_{2,204}$=0,489 p=0,617) and the results were also similar across the three groups of subjects ($F_{2,17}$=0,265 p=0,77). However, the recruitment order varied across muscles ($F_{6,204}$=85,866 p=0,0001). None of the interaction effects were significant. Note in Figure 09 that the onsets of the biceps femoris and semitendinosus were delayed by about 60 ms in relation to the rectus femoris burst. The biceps femoris and the semitendinosus acted as antagonists, in synergy with gravity, braking the knee movement. The gastrocnemius medialis, gastrocnemius lateralis and tibialis anterior were activated around 10 to 20 ms after the onset of the rectus femoris burst. The interactions among the effects of groups, target distances and the type of muscles were not significant.

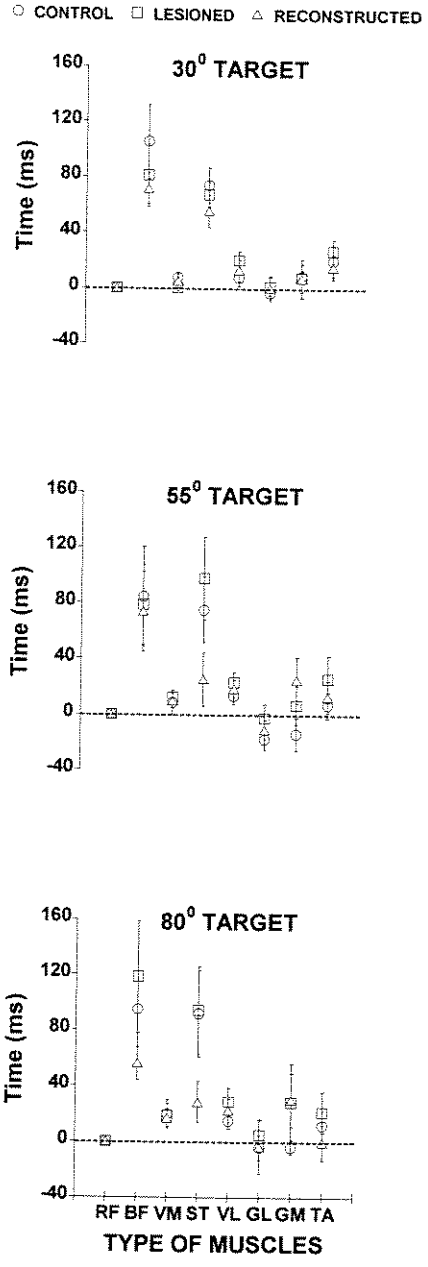

FIGURE 09: Recruitment order of Biceps Femoris, Vastus Medialis, Semitendinosus, Vastus Lateralis, Gastrocnemius Lateralis, Gastrocnemius Medialis and Tibialis Anterior in relation to the Rectus Femoris. The data were averaged across each of the three groups (open circle - control group; open square – lesioned group; open triangle – reconstructed group), and are plotted for the three movement distances (30°-upper panel, 55°-middle panel and 80°-lower panel).

DISCUSSION

The strategies used by normal individuals to control voluntary movements

Single-joint movements of normal individuals have been characterized by bell-shaped velocities with triphasic agonist-antagonist-agonist bursts of muscle activity (Almeida et al. 1995; Angel, 1974; Gottlieb et al. 1989; Hallett et al. 1975; Hannaford & Stark, 1985). The first agonist burst generates a muscle torque accelerating the limb towards the target. After some latency, the antagonist muscle is activated, generating a decelerating muscle torque, whose function is to brake the limb at the target. The second agonist burst would lock the limb at the target position. The peak velocities and the amount of agonist muscle activity during such single-joint movements scale with movement distance (for review, see also Gottlieb et al. 1989).

In the present study, these kinematic and EMG features were preserved during the knee-extension movements of the individuals of the control group. The velocities of these movements were also characterized by a bell-shaped profile and increased with movement distances (Figure 07). The amount of agonist and antagonist EMG activity of all the recorded muscles (rectus femoris, vastus medialis, vastus lateralis, semitendinosus, biceps femoris, gastrocnemius medialis, gastrocnemius lateralis, tibialis anterior) also increased with movement distance (Figure 08). In a sense, these kinematic and EMG patterns replicate and expand what has been observed for several ballistic single-joint movements of

the upper-arm of normal individuals (Almeida et al. 1995; Angel, 1974; Gottlieb et al. 1989; Hallett et al. 1975; Hannaford & Stark, 1985).

However, contrary to the pointing movements involving the upper limb (Almeida et al. 1995 - for review, see also Gottlieb et al. 1989), we observed an increase in the amount of antagonist muscle activities with movement distance (Figure 08). The lack of modulation of the antagonist muscle activity with movement distance has been attributed to the visco-elastic components of the muscles (Gottlieb, 1996; Gottlieb et al. 1989). Our data do not support this hypothesis. We are unaware of any findings showing a difference in the visco-elastic components between the leg and the arm muscles that could account for the modulation of the antagonist muscle activity with movement distance observed in our experiment.

Lesion of the ACL or its reconstruction do not affect the strategy used to perform voluntary knee-extension movements

The movements of all individuals with lesion of the ACL and submitted to surgery were indistinguishable when compared with the movements of control individuals. All three groups of individuals used similar patterns of muscle activity and had comparable movement kinematics (Figure 08). So, we can conclude that the lesion of the ACL or its reconstruction by surgery did not affect the strategy used by the central nervous system to modulate the amount of muscle activity during the performance of single-joint knee extension movements.

Our data do not support the hypothesis that the lesion of the ACL would affect the patterns of muscle activity leading to a functional motor deficit (Ciccotti at al. 1994) during the performance of knee-extension movements. Also, our data do not support the idea that mechanical instability of the knee joint, provoked by the ACL lesion, would compromise the performance of knee movements (Noyes et al. 1983c) at least for the movements studied.

We did not observe any signal in the kinematics of the movements that could be associated with the positive "Pivot Shift Test or Jerk Test" verified for all individuals with lesion of the ACL (Galway et al. 1972; Losee, 1983; Solomonow et al. 1987), despite the fact that all individuals with lesion of the ACL (see Table I) had knee-joint instability as measured by the "Pivot Shift Test". However, we can also conclude that knee-joint instability as mensured by the "Pivot Shift Test", did not affect the performance of fast voluntary knee-extension movements.

Our data also do not support several observations showing that individuals with lesion of the ACL, as compared with individuals without lesion or with surgical reconstruction of the ACL, used more muscle activity to perform several motor tasks. For example, Ciccotti et al. (1994) observed that individuals with lesion of the ACL used proportionally more vastus lateralis muscle activity to perform several tasks (i.e., walking, ramp and stair ascending and descending, and running) when compared to both controls and individuals with ACL reconstruction. On the other hand, the patterns of EMG activity in individuals submitted to surgical reconstruction of the ACL were indistinguishable from those of normal individuals during the performance of these tasks.

Several other studies showed atrophy of the quadriceps in individuals with lesion of the ACL (Andriacchi, 1990; Berchuck et al. 1990). This muscle atrophy has been attributed to an inhibitory reflex mechanism (deAndrade et al. 1965; Fahrer et al. 1988; Jones et al. 1987; Synder-Mackler et al. 1994). Functionally, it is believed that the quadriceps atrophy would decrease the extension torque at the knee, avoiding the anterior displacement of the tibia (Andriacchi 1990). So, the quadriceps atrophy would favor the action of the hamstring muscles that work in synergy with the ACL, avoiding the anteriorization of the tibia. One could expect to observe a decreased amount of muscle activity of the quadriceps if the individual with lesion of the ACL had increased inhibition of these muscles. However, we did not find group differences for the quadriceps muscles during the isotonic contraction (Figure 08). Our conclusion is that the atrophy of the quadriceps if exist, does not affect the ability of the motor control system to generate isotonic contractions to perform fast voluntary movements in individuals with lesion and with surgical reconstruction of the ACL.

The lesion of the ACL does not affect the recruitment order of muscles of knee-extension movements

The torque generated by muscles at a joint depends on the way the agonist and antagonist muscles are modulated, including the sequence in which they are activated. All three groups of individuals recruited all leg muscles in the same order (Figure 09). These data are consistent with the study of Ciccotti et al. (1994) showing that, during the *cross-cutting* activities, normal individuals and

individuals with lesion of the ACL, and with surgical reconstruction of this ligament, activate the quadriceps-hamstrings muscles in the same order. The simultaneous activation of the Gastrocnemius Medialis, Gastrocnemius Lateralis and Tibialis Anterior muscles characterized a pattern of co-activation. This co-activation increased muscle stiffness, generating more stability of this joint. Also, this is consistent with the instruction of the task. We asked the individuals to move the knee joint while keeping the ankle stationary.

LIMITATIONS OF THE STUDY

The decision for surgical reconstruction of the ACL is made based mainly on the clinical judgment of existing joint instability and functional deficit (Christer et al. 1989; Clancy, 1985; Lysolm and Gillquist, 1982). Our study showed that we have to be very cautious about judging the degree of modification or adaptation in the motor control system due to the presence of the ACL lesion. However, we cannot neglect that the maximum anteriorization with rotation of the tibia could overload the knee joint to the point where one could observe a functional deficit due to lesion of the ACL (Fu et al. 1993; Fukubayashi et al. 1982). These movements must be tested to allow us to make more general statements about the ability of individuals with lesion of the ACL to control their movements.

Our data also show that we have to be careful before prescribing physical therapy treatment to ameliorate proprioception and muscle force, if the goal is to improve the control of voluntary movements of these individuals. An analysis of our data did not show any signs that could be attributed to loss of proprioception

or a decreased ability to generate isotonic muscle force during the performance of fast voluntary movements.

If the individuals with and without lesion of the ACL used similar strategies to modulate the pattern of muscle activity, how can several functional disabilities reported in the literature for the individuals with lesion of the ACL be explained? (Andriacchi. 1990; Barrack et al. 1989; Beard et al. 1993; Berchuck et al. 1990; Borsa et al. 1997; Gauffin and Tropp, 1992; Hoffman et al. 1999; Noyes et al. 1983c; Solomonow et al. 1987; Tibone et al. 1986; Timoney et al. 1993). A direct comparison between our data and the data about motor dysfunction in individuals with lesion of the ACL is limited by the kind of task used in these experiments and by the type of analysis.

In our study, the individuals performed fast single-joint movements, whereas in the studies reported in the literature, the movements were part of a complex motor task, such as walking (Andriacchi 1990; Berchuck et al. 1990; Timoney et al. 1993), jumping (Gauffin and Tropp, 1992), and maintaining equilibrium on a platform (Hoffman et al. 1999). In these experiments, involving complex motor tasks, the real-time kinematics and/or the kinetics of the movements were not reported. Without a simultaneous recording of the movement kinematics, it is difficult to give a meaningful interpretation of the EMG activity patterns.

On the other hand, one could argue that single-joint movements are too simple to reveal any important differences in the strategies used to control complex movements (Gottlieb et al. 1989). First, there is no such thing as "single-

joint movements". Any focal movement involving only one joint will generate disturbance in non-focal joints, forcing the motor control system to deal with a very complex problem (Almeida et al. 1995). Second, all knee-extension movements belong to a natural class of movements performed daily by our motor system. The knee-extension movement is also tested by clinicians to evaluate the integrity of this joint. Third, before trying to understand very "ecological tasks", such as walking, one should first try to understand how part of this task is performed. Now that we understand how single-joint knee-extension movements are controlled, we can add complexity to the task and observe at what level function starts to deteriorate, if it does.

For cerebral palsy, we showed that the analysis of single-joint movements, such as the one presented here, could predict deficits in the everyday activities (Almeida et al. 1997). It will not surprise us if we find that this analysis will also very well predict the "ecological tasks" in individuals with lesion or reconstruction of the ACL.

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CAPÍTULO III – "Does the lesion of the anterior cruciate ligament or its surgical reconstruction affect the performance of deep squatting?"

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ABSTRACT

Deep squatting is a complex task, involving movements mainly in joints of the thigh. Lesion of the anterior cruciate ligament (ACL) could generate changes in the kinematics and in the pattern of muscle activities with functional deficits in the knee movements during deep squatting. We tested the hypothesis that the lesion of the ACL would provoke a distinct kinematic coupling between the movements of the hip, knee and ankle joints and that the surgical reconstruction of the ACL would increase joint stability, improving the kinematic and EMG pattern to the level observed in healthy individuals.

Subjects without lesion (n=9), with lesion of the ACL (n=9) and with surgical reconstruction of the ACL (n=9) performed deep squatting movements. Movement kinematics and electromyographic (EMG) activation patterns of leg muscles were recorded. All three groups of subjects showed similar results in movement excursion across the three joints. There was great coupling between the movement excursion among the three joints. All anterior muscles (rectus femoris, vastus lateralis, tibialis anterior) were activated before the posterior muscles (semitendinosus, biceps femoris, gastrocnemius medialis) and the amount of agonist and antagonist EMG activities of all muscles analyzed was similar across all three groups of subjects. All muscles crossing the hip, knee and ankle joints were activated in a reciprocal pattern, in a specific order, to generate the coupled-movement kinematics in these three joints.

We then showed that the lesion of the ACL or its surgical reconstruction do not affect the kinematic patterns (angular excursions and velocities) of the hip, knee, and ankle movements, nor the coupling of these movements during deep squatting. We also showed that the recruitment order and the amount of EMG activities of the anterior and posterior muscles crossing these joints did not vary across all three group of subjects. We recommend the use of deep squatting in the Physical Therapy Programs for individuals with lesion and with surgical reconstruction of the ACL.

INTRODUCTION

It is believed that the major function of the anterior cruciate ligament (ACL) is to prevent the anterior displacement of the tibia over the femur (Fu et al. 1993; Fukubayashi et al. 1982). This ligament also acts on the rotational mechanism of the knee (Fukubayashi et al. 1982; Kennedy et al. 1974) and prevents valgus and varus angulation (see Fu, Harner et al. 1993 - for reviews).

There is a general agreement that the lesion of the ACL provokes joint laxity (Brandsson et al. 2002; Gauffin and Tropp, 1992), increasing the instability of the knee. This lesion has also been associated with several changes in movement kinematics (Gillquest and Messner, 1995; Brandsson et al. 2001), in the pattern of joint torque (Andriacchi 1990; Gauffin and Tropp, 1992), in the patterns of electromyographic (EMG) activities of the muscles crossing this joint, during both voluntary (Ciccotti et al. 1994; Gauffin and Tropp, 1992; Kålund et al. 1990) and reflex movements (Wojtys and Huston, 1994; Beard et al. 1993) and in the dynamic postural control system (Hoffman et al. 1999).

In particular, changes in muscle activities have been attributed to an adaptive response that would generate the net muscle torque that could avoid the anterior displacement of the tíbia (Wojtys & Huston, 1994; Gauffin and Tropp, 1992) or its internal rotation (Zhang et al. 2002; Ciccotti et al. 1994), compensating for the deficient action of the ACL. Thus, both the reduction of the amount of EMG activities of the quadriceps (Branch et al. 1989) and the increase of the EMG of the biceps femoris and semitendinosus muscles (Branch et al.

1989; Ciccotti et al. 1994) are believed to avoid the anterior displacement of the tibia during the gait (Andriacchi, 1990; Ciccotti et al. 1994; Branch et al. 1989), jump (Gauffin and Tropp, 1992) and knee flexion-extension (Tibone et al. 1986).

It is also believed that the lesion of the ACL produces loss of proprioception, deteriorating the capability of these individuals to detect static (Barrack et al. 1989; Borsa et al. 1997) and dynamic changes in position of the leg (Corrigan et al. 1992). This lesion also changes the latency time of the knee muscles due to a reflex response (Beard et al. 1993; Wojtys & Huston, 1994) or voluntary movements (Lass et al. 1991; Kålund et al. 1990).

These deficits in movement kinematics and kinetics, EMG patterns and proprioception can be ameliorated with the surgical reconstruction of the ACL (Ciccotti et al. 1994). On the other hand, other authors failed to observe changes in movement kinematics (Devita et al. 1998), EMG patterns (Bulgheroni et al. 1997; Ciccotti et al. 1994) and proprioception (Good et al. 1999; Fridén et al. 1998; Jennings and Seedhom, 1994; MacDonald et al. 1996) due to the lesion or surgical reconstruction of the ACL.

Brandsson et al. (2001), using "*dynamic radiosteremetric analysis*" showed that the lesion of the ACL changed the movement kinematics of the knee. Nevertheless, more recently, these authors did not observe kinematic changes between individuals with lesion of the ACL and individuals submitted to ligament reconstruction.

Despite this controversy, there is a common opinion that a strong stable knee is paramount to the success of several motor functions (Beard et al. 1993;

Tibone et al. 1986). Nevertheless, based on the literature cited above, we can conclude that it is still not clear how the increment in joint instability would affect movement kinematics and the control of the muscle activities.

During squatting, the antero-posterior and medial-lateral shear forces and the compressive forces increase with the flexion of the knee (Hattin et al. 1989). However, during this task, the ACL is submitted to anterior strain forces that decreased when parallel squatting was attained (Beynnon et al. 1996). These strain forces are opposed mainly by the mechanical restriction of the ACL (Hughston et al. 1976; Fu et al. 1993; Fukubayashi et al. 1982).

One possibility for the disagreement about the kinematics and EMG changes cited above for the individuals with lesion of the ACL and with surgical reconstruction would be a compensatory mechanism involving changes in the coupling of the movement of the hip, knee and ankle, connected by a chain. The EMG patterns of the leg muscles would also be modified to reflect the alterations in the coupling of the movement crossing the three joints.

In this study, we describe the kinematics and the EMG patterns of muscle activities of the ankle, knee and hip joints in healthy subjects during deep squatting. Then, we first tested the hypothesis that the lesion of the ACL would provoke a distinct kinematic coupling between the movements of the three joints. This fact is based on the idea that the lesion of the ACL would compromise the ability to keep a strong stable knee, making the performance of squatting difficult. Second, the surgical reconstruction of the ACL would increase joint stability,

improving the kinematics and EMG pattern to the level observed in healthy individuals.

METHODS

Subjects

We tested nine subjects without any neurological or orthopedic dysfunction (control group), nine subjects with lesion of the anterior cruciate ligament that were not submitted to the surgical reconstruction (lesioned group), and nine subjects that had the anterior cruciate ligament reconstructed surgically (reconstructed group). All subjects were tested after giving formal consent according to the procedures approved by the State University of Campinas. The average ages, with the standard errors were 28.5 (±2.07); 29.03 (±1.97) and 26.13 (±2.25), respectively, for the control, lesioned and reconstructed groups.

All subjects of the lesioned group showed positive signs of lesion of the anterior cruciate ligament to the specific tests of the anterior drawer (Hughston et al. 1976), Lachman´s Test (Torg et al. 1976; Wroble and Lindfeld, 1988) and pivot shift tests (Galway et al. 1972; Losee, 1983). In addition of these two specific tests, all subjects of the lesioned group were submitted to the following complementary tests: abduction at 0 and 30° (Hughston et al. 1976), adduction at 0 and 30° (Hughston et al. 1976), Mc Murray´s test (Crewshaw, 1989), and the posterior drawer test (Hughston et al. 1976) (see Table 03). Finally, the ligament rupture of the ACL was verified through magnetic resonance imaging.

TABLE 03: Clinical examination of all individuals of the three groups

Group of Subjects		Subjects								
		1	2	3	4	5	6	7	8	9
•	*Age (years)*									
CON		25.6	29.3	20.5	34.7	36.4	33.6	24	28.9	23.8
LES		19.9	27.5	40	35.6	22.9	33	28.6	26.5	27.3
REC		21.9	26.2	37.9	19.2	20.9	30.2	30.4	21	27.5
•	*Side of lesion or tested*									
CON		R	R	L	L	L	L	R	L	L
LES		R	R	L	L	L	R	L	R	L
REC		L	R	L	L	L	R	R	R	R
•	*Side of dominance*									
CON		R	R	R	R	R	R	R	R	R
LES		R	R	R	R	R	R	L	L	L
REC		R	R	R	R	R	R	R	R	R
•	*Edema and pain 24 hours after the lesion*									
LES		+	-	+	+	+	+	-	-	+
REC		-	-	-	+	+	+	+	+	+
•	*Associated lesions*									
LES		-	-	-	-	-	-	-	MM*	-
REC		-	-	BM	BM	BM	BM	-	MM	-
•	*History of giving way*									
LES	*(BS)*	+	-	-	+	+	-	+	-	-
REC	*(BS)*	+	+	+	+	+	+	+	+	+
REC	*(AS)*	-	-	-	-	-	-	-	-	-
•	*Anterior Drawer Test*									
LES	*(BS)*	+	+	+	+	+	+	+	+	+
REC	*(BS)*	+	+	+	+	+	+	+	+	+
REC	*(AS)*	-	-	-	-	-	-	-	-	-
•	*Pivot Shift Test*									
LES	*(BS)*	+	+	+	+	+	+	+	+	+
REC	*(BS)*	+	+	+	+	+	+	+	+	+
REC	*(AS)*	-	-	-	-	-	-	-	-	-
•	*Lachman Test*									
LES	*(BS)*	+	+	+	+	+	+	+	+	+
REC	*(BS)*	+	+	+	+	+	+	+	+	+
REC	*(AS)*	-	-	-	+	-	-	-	-	-
•	*Time until the test (months)*									
LES	*(IL)*	48	48	60	25	12	40	69	60	61
REC	*(IL)*	40	34	20	21	41	45	24	19	22
REC	*(FS)*	38	32	10	10	20	28	21	17	12
•	*Type of reconstruction*									
REC		PL	PL	PL	PL	PL	PL	PL	PL	PL

CON=control; LES=lesioned; REC=reconstructed *R=right; L=left*

BS=before surgery; AS=after surgery *IL= from the initial lesion; FS= from surgery*

*MM=medial meniscus; LM=lateral meniscus; BM=both meniscus; PL=patellar ligament; *condral lesion*

Four out of nine subjects of the lesioned group had a history of knee giving way with recurrent sprain. All subjects of the control group and the reconstruction group tested negatively for all the specific and complementary tests pointed out above. All tests were performed by the same physician (the third author). After surgery, none of the subjects of the reconstructed group presented signs of giving way, new lesion, or artrofibrosis of the reconstructed knee (Shelbourne et al. 1991). None of the subjects of the three groups felt pain or decreased range of motion of the knee joint movements during the test sessions. The subjects of the three groups were all males and did not have any history of lesion of the meniscus or of ligaments of the contra-lateral knee.

PROCEDURE

Initially, the subject stayed in the upright position (Figure 10). Feet were positioned parallel on an inclined wooden board that constrained the ankle joint to 20 degrees of plantarflexion (zero degrees being with the feet parallel to the floor). In this initial position, the subject kept arms crossed about the shoulders.

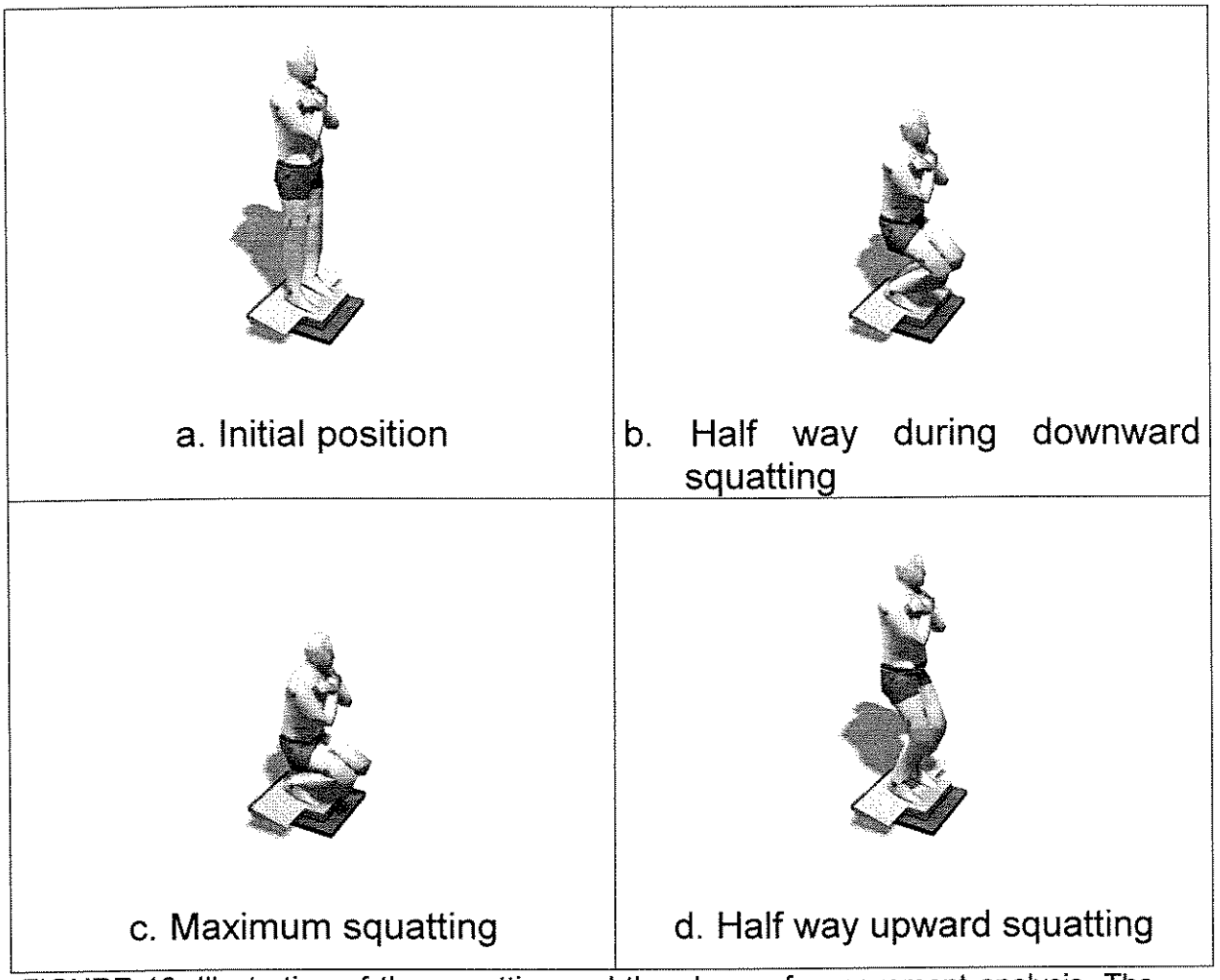

FIGURE 10: Illustration of the squatting and the phases for movement analysis. The squatting was divided into in two parts. The initial phase (picture a) is the initial position until maximum knee angle in squatting. The second phase (picture b) is the moment in which knee articulation reached the peak of velocity in the descendent phase. The third phase (picture c) is the moment in which knee articulation reached the maximum angle at the end of movement. The last (picture d) is the moment in which knee articulation reached the ascendant phase.

The subjects were instructed to perform the deep squatting downward until the knee was flexed 100°, and then return to the initial position. Only individuals with maximum angular excursion of the knee between 90-110 degrees were included in each of the three groups. The target was set with marks fixed in front of the subjects in such way to constrain the movements to the desired excursion. All movements to and from the target were performed

according to the instruction "to move as fast as possible". Each subject performed 11 trials of movements.

Kinematic data

We recorded the X, Y and Z coordinates of the LED marks using a tridimensional motion-analysis system (OPTOTRAK 3020) at 100 frames per second. The LED marks were attached to the center of the shoulder, hip (superior iliac crist), knee (lateral condyle), ankle (external malleolus), and to the fifth toe. The X and Y coordinates of these marks were used to calculate the angular displacement and velocity of the three joints in sagital plane.

The EMG muscle activities

We also recorded the muscle activities of the rectus femoris-RF, vastus medialis-VM, vastus lateralis-VL, semitendinosus-ST, biceps femoris-BF, gastrocnemius medialis-GM, gastrocnemius lateralis-GL and tibialis anterior-TA. These EMGs were recorded using bipolar surface EMG electrodes DELSys (model DE2.2L). The EMG signals were collected at 1000Hz, amplified (X 2000), band-pass filtered (20-450Hz) and recorded. Before data analysis, the EMG signals were rectified and low-pass filtered with a Butterworth filter with a cut-off frequency of 20 msecs.

Quantification of data

The movement was divided into two phases, the downward phase, from the initial position to the target position (when velocity first crossed zero), and the upward phase, from the target to the initial position (when velocity crossed zero for the second time - Figure 11).

The difference between the initial and final positions was defined as the angular excursion. The maximum peak velocity of the knee was calculated for the two phases. The time series of the EMGs, and the angular excursion and velocity were plotted in a computer monitor for each trial. Then, using a computer mouse, the onset of each EMG signal was visually identified as the first time the signal increased and was sustained above the baseline (Gotttlieb, 1996). The EMG activities were integrated for each muscle from the onset of the muscle activity to the first cross zero of the acceleration, and from this time to the second cross zero of the acceleration. These integrals were used to characterize the amount of activity for each muscle during the upward and downward phases.

The EMG activities of each muscle were also integrated over a 100 ms interval, that occurred at 1000 ms, just before the onset of each muscle's EMG activitiy. These integrals were used to define the baseline activity levels for each muscle. We normalized each of the integrated EMG values calculated during the two phases by dividing it by its corresponding baseline value. The normalization using the EMG recorded during maximum voluntary contraction (MVC) did not affect the results reported here.

The time of recruitment for each muscle was calculated in relation to the time of the onset of the rectus femoris muscle (time zero).

STATISTIC ANALYSIS

The effects within subjects (phases of movement and type of muscles) and between subjects (the three groups) were analyzed for several of the dependent variables studied using a mixed ANOVA model.

RESULTS

All subjects of the three groups performed the tasks very well with similar movement excursions and velocities. Note that the ANOVA test did not reveal main effect for group for either peak velocity or angular excursion, when the subjects moved downward and upward during the deep squatting (see table 04).

TABLE 04: Analysis of angular excursion and angular velocity (ANOVA)

Phase of movement	Down		Up	
	Group effect		Group effect	
	$F_{(2,24)}$	P	$F_{(2,24)}$	P
knee angle	.7162	.4987	1.7630	.1930
knee velocity	.2535	.7781	1.5374	.2354
hip angle	.0761	.9269	.0618	.9402
hip velocity	2.0876	.1459	.7916	.4646
ankle angle	1.2912	.2934	1.2558	.3029
ankle velocity	.0006	.9994	.9371	.4056

Kinematic coupling between the movements of the three joints

The squatting was performed with different combinations of the hip, knee and ankle movements, but did not vary across all three groups of subjects. On the average across all three groups, the downward phase of the squatting was performed with 112°(±10.1) of hip flexion, 101°(±5.8) of knee flexion and 33°(±6.6) of ankle dorsiflexion. The upward squatting also involved the participation of the three joints with a similar amount of movement excursion across them, but with the movements in opposite directions.

In Figure 12 we plotted the time series of the angular excursion of the knee *versus* hip (a), the knee *versus* ankle (b), and the hip *versus* ankle (c) during the interval of time when the subject moved downward and upward, and calculated the linear regression between each pair of movements. The coefficient of linear regression |r| between each pair of movements was above 0.9 for this subject, showing great coupling in the movement excursion among the three joints.

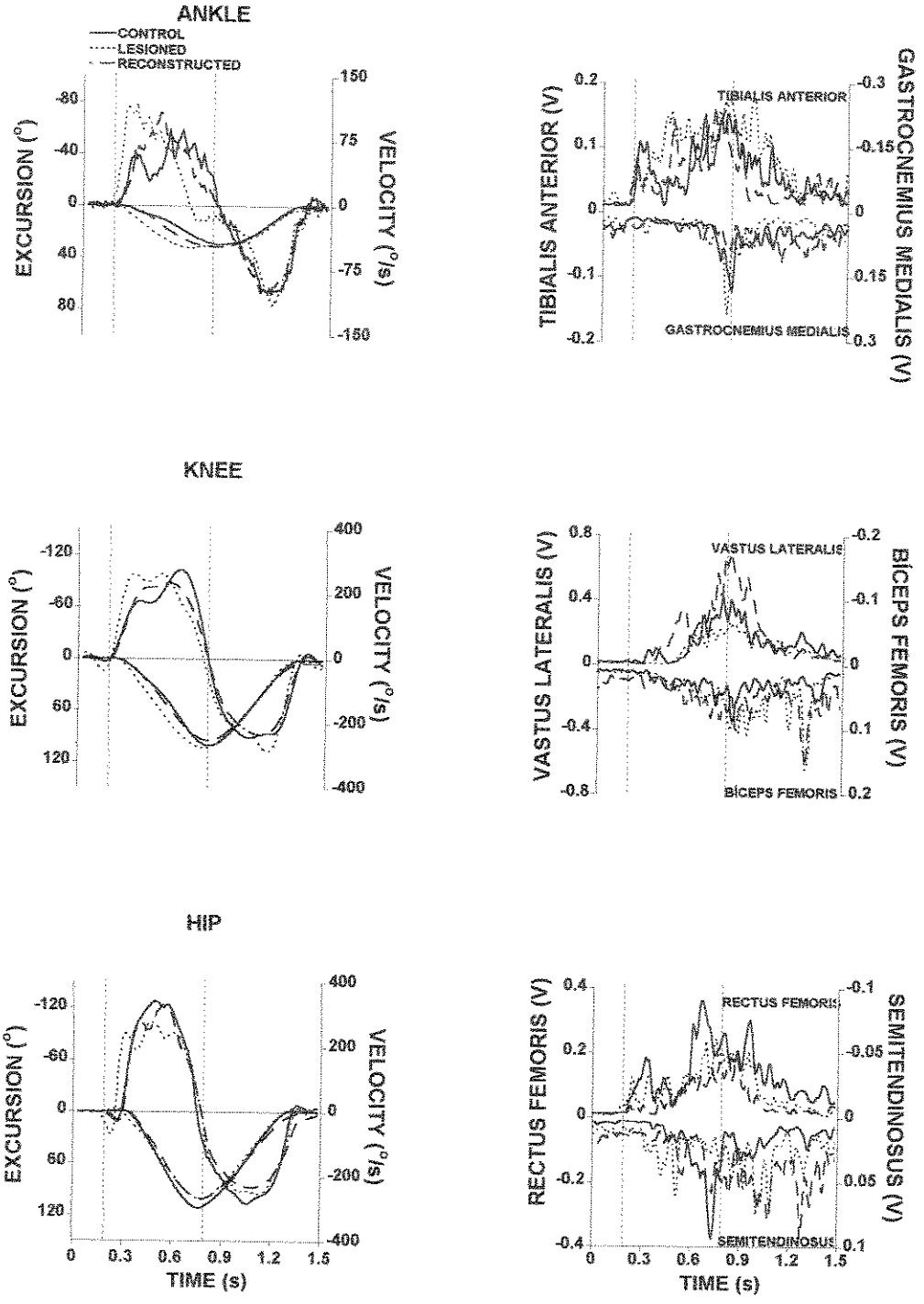

FIGURE 11: Time series of kinematics (in left column) and EMG values (In right column) performed during deep squatting. Excursion and velocity of ankle, knee and hip movements and the EMGs of Rectus Femoris (RF), Vastus Lateralis (VL), Tibialis Anterior (TA), Biceps Femoris (BF), Semitendinosus (ST) and Gastrocnemius Medialis (GM). The data represent one trial performed by one subject of each group. (control group-solid line; lesioned group – stipple line; reconstructed group – discontinuous line). The time is in seconds, the excursion in degrees, the velocity in degrees per second and the electromyographic activity in Volts.

This coupling among the excursions of the three joints was observed for all subjects of the three groups. The ANOVA test did not reveal the effect of group for the coefficient of linear correlations for each pair of comparisons (knee *versus* hip, knee *versus* ankle, and hip *versus* ankle). See table 05 for the ANOVA results.

TABLE 05: Statistical analysis of linear regression for interaction between joints. Correlation and inclination degree between joints. ANOVA Test.

	knee x hip		hip x ankle		knee x ankle	
	$F_{(2,24)}$	p	$F_{(2,24)}$	p	$F_{(2,24)}$	p
correlation	1.2279	.3107	.0812	.9223	.7297	.4925
Slope	.8634	.4344	.5657	.5754	.0401	.9697

The movement of the joints and the length of the muscles

During downward and upward squatting, both knee and hip joints moved proportionally at the rate of one by one degree. This proportionality can be observed by the slope of the linear correlation shown in Figure 12. The ANOVA test showed that the slopes for each pair of movement comparisons were similar across all three groups (see table 05).

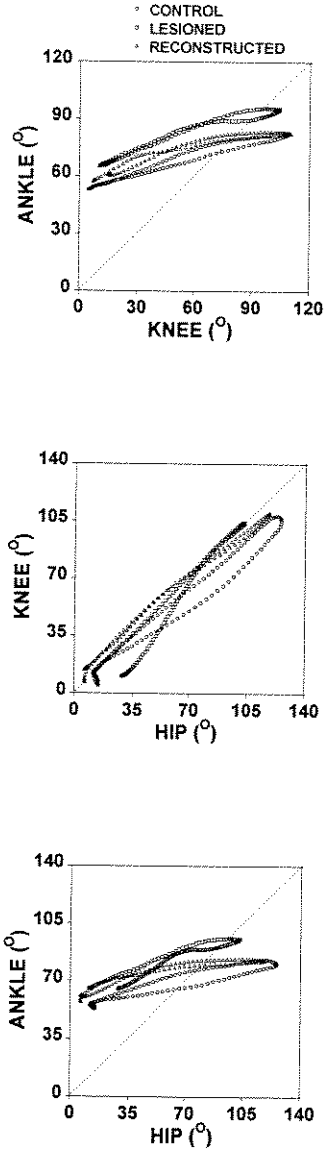

FIGURE 12: Angular interaction of ankle x knee (top); hip x knee (middle) and hip x ankle (lower).

TABLE 06: Analysis of integrals of EMG burst of eight muscles in downward and upward phase (ANOVA) - (group effect)

	PHASE			
Muscles	Integral Downward		Integral Upward	
	$F_{(2,24)}$	p	$F_{(2,24)}$	p
rectus femoris	.9178	.4142	.0540	.9476
vastus medialis	.6026	.5558	.0965	.9084
vastus lateralis	2.646	.0924	.0359	.9648
tibialis anterior	.6765	.5182	.9576	.3986
semitendinosus	.5815	.5671	.1441	.8665
biceps femoris	.9506	.4025	.6522	.5307
gastrocnemius medialis	2.0322	.1539	1.7099	.2031
gastrocnemius lateralis	2.0305	.1551	1.7076	.2045

The flexion of the trunk on the thigh (hip flexion) decreases the length of the rectus femoris and increases the length of the semitendinius and biceps femoris, all bi-articular muscles. However, the flexion of the thigh on the leg (knee flexion) increases the length of the rectus femoris and decreases the length of the semitendinosus and biceps femoris muscles. The opposite is true for hip and knee extension.

The implication of the similar rate of changes of both knee and hip angles is that the length of the bi-articular muscles that cross these joints (rectus femoris, semitendinosus and biceps femoris) may not change during deep squatting.

The gastrocnemius muscles are also bi-articular, crossing both ankle and knee joints. The knee flexion contributes to the decrease in the length of the gastrocnemius, and the dorsiflexion of the ankle to the decrease in the length of the anterior tibialis. During downward squatting the knee is flexing and the ankle

is moving into dorsiflexion, but at different rates. For each degree of ankle dorsiflexion there are 3 degrees of knee flexion. This proportionality can be observed by the slope of the linear correlation shown in Figure 12. See also the ANOVA results in Table 05 in which we did not observe group differences for the slope. So, we can assume that, during downward squatting the gastrocnemius muscles are shortening. The opposite is true when the individuals move upward, when the gastrocnemius muscles are lengthening.

The EMG patterns of muscle activities

Figure 11 shows the time series data of one individual of each group, when moving downward and upward. In general, the EMG patterns of all eight muscles recorded varied across muscles, but were qualitatively similar among all three groups. All anterior muscles (rectus femoris, vastus lateralis, tibialis anterior) were activated before the posterior muscles (semitendinosus, biceps femoris, gastrocnemius medialis).

The recruitment order of the muscles

In Figure 13 we show that, during squatting, the tibialis anterior is the first muscle to be activated, around 80 milliseconds before the rectus femoris. All other muscles, including the vastus medialis and vastus lateralis were activated after the rectus femoris. The posterior muscles are activated after the anterior muscles. The ANOVA test did not reveal group differences for the order of recruitment of all eight muscles (see Table 07).

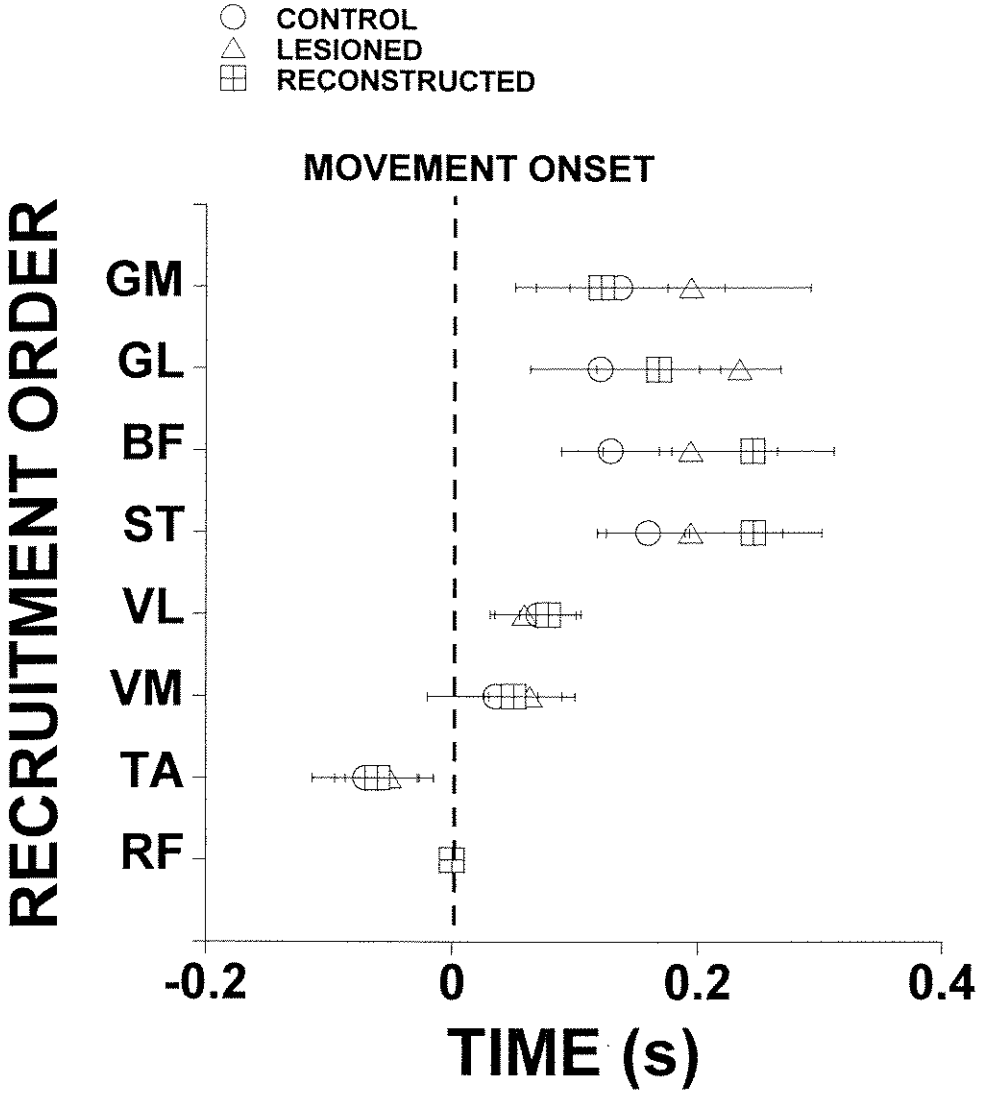

FIGURE 13: Time of recruitment of muscles. The Rectus Femoris (RF) is the reference for moment of onset. EMG activities of Gastrocnemius Medialis (GM), Gastrocnemius Lateralis (GL), Biceps Femoris (BF), Semitendinosus (ST), Vastus Lateralis (VL), Vastus Medialis (VL), Tibialis Anterior (TA) and Rectus Femoris (RF).

TABLE 07: Analysis of recruitment order of all muscles during deep squatting (ANOVA) - (group effect)

Muscles	$F_{(2,24)}$	p
rectus femoris	-	-
vastus medialis	1.4727	.2556
vastus lateralis	.3611	.7018
tibialis anterior	.0214	.9788
semitendinosus	.9812	.3931
biceps femoris	.5355	.5955
gastrocnemius medialis	.2287	.7987
gastrocnemius lateralis	2.5283	.1155

Quantification of the amount of EMG

The amount of agonist and antagonist EMG activities of all muscles analyzed (rectus femoris, vastus medialis, vastus lateralis, semitendinosus, biceps femoris, gastrocnemius medialis, gastrocnemius lateralis and tibialis anterior) were similar across all three groups of subjects (Figure 11). Note that the ANOVA test did not reveal the effect among groups for the integral of the eight muscles during the downward and upward phases of deep squatting (see Table 06).

DISCUSSION

Kinematic and EMG strategy during deep squatting

Deep squatting is a complex task, involving movements mainly of the hip, knee, and ankle joints and the kinematics of these movements are well described in the literature (Wretenber et al. 1996; Isear et al. 1997). The EMG patterns of the major muscles crossing the knee joint are also described during this task

(Isear et al. 1997; MacCaw, 1999; Stuart et al. 1996). Here we have advanced this knowledge in two ways. First, we show that the three joints move in synchrony, displaying a very well coupled behavior during the performance of deep squatting (Figure 12). Second, we show that the muscles crossing the hip, knee, and ankle joints are activated in a reciprocal pattern in a specific order to generate the coupled-movement kinematics in these three joints.

The initial activation of the tibialis anterior provokes the dorsifexion of the ankle joint, disrupting the equilibrium, forcing the body to fall. The force of gravity acting on the body provokes the movement of the knee and hip into flexion. The rectus femoris, vastus medialis and vastus lateralis are than activated around 100 ms after the onset of the tibialis anterior to decelerate the limb at the final position of squatting, avoiding the complete fall of the body, during the downward phase. Finally, the posterior muscles (semitendinosus, biceps femoris, gastrocnemius medialis and lateralis) are activated to reverse movement direction, forcing the ankle into plantarflexion, and the knee and hip into extension, moving the limb at the initial position, until the end of the upward phase of squatting.

The effect of lesion and the surgical reconstruction of the ACL on the kinematic and EMG strategy during deep squatting

We show that the lesion of the ACL or its surgical reconstruction do not affect the kinematic patterns (angular excursions and velocities) of the hip, knee, and ankle movements, nor the coupling of these movements during deep

squatting (Figure 12). We also show that the recruitment order and the amount of EMG activities of the anterior and posterior muscles crossing these joints did not vary across all three groups of subjects (Figures 11 and 13). So, contrary to our initial hypothesis, we can conclude that the lesion and the surgical reconstruction of the ACL do not affect the movement kinematics of the hip, knee and ankle joints, nor the EMG pattern of the major leg muscles analyzed during the performance of deep squatting. Indeed, the individuals of the three groups used similar kinematic and electromyographic strategy to perform the task. As a direct consequence of these observations, we show that there are no compensatory mechanisms involving changes in the kinematics of the hip and ankle joints that could account for possible changes in the kinematics of the knee joint reported in the literature for individuals with lesion of the ACL (Roberts et al. 1999) or with surgical reconstruction of this ligament (Devita et al. 1998).

Indeed, our findings support several observations showing that the lesion of the ACL (Osternig et al. 2001) or its surgical reconstruction (Devita et al. 1998; Bulgheroni et al. 1997; Ciccotti et al. 1994) do not affect either the kinematics of voluntary knee movements nor the pattern of the EMG of the muscles crossing the hip, knee and ankle joints. The lack of difference in movement kinematics of the knee was reported mainly during the gait (Devita et al. 1998; Bulgheroni et al. 1997).

On the other hand, our data are in clear contradiction with several clinical, biomechanical and electromyographic observations showing changes in movement kinematics in individuals with lesion of the ACL (Losee, 1978;

Kennedy et al. 1978; Peterson et al. 1984) or submitted to surgical reconstruction (Bush-Joseph et al. 2001; Gillquest and Messner, 1995). Our data also do not support several studies showing changes in the small translational movements of the knee (Marans et al. 1989; Brandsson et al. 2001; Brandsson et al. 2002) in individuals with lesion of the ACL.

Nevertheless, we cannot neglect the studies showing that individuals with ACL lesion used a more flexed kinematic pattern during walking (Berchuck et al. 1990; Devita et al. 1997) and sometimes have the giving way phenomena during voluntary (Solomonow et al. 1987; Noyes et al. 1983c) and passive movement of the knee (Losee, 1978; Kennedy, et al.1978).

How can these findings be reconciled with the observation that the lesion of the ACL or its surgical reconstruction do not necessarily affect the kinematics of the hip, knee and ankle movements and coupling between these movements, and the EMG patterns of the muscles crossing these joints during deep squatting?

One possibility is that the kinematic changes reported in the literature for a knee with lesion of the ACL or submitted to surgical reconstruction would <u>not</u> be manifest in deep squatting because of the sensitivity of the measurement we used. For example, the increment in translational movements of the knee, due to the lesion of the ACL, could have more effect on the shape of the movement, which would be manifest as more fluctuation in the angle trajectory. However, a visual analysis of the angular excursions and velocities of the movements did not show any abrupt changes that could be characterized as a disruption in

movement smoothness or specific variability that could be associated to any of the three groups (Figure 11).

The lack of group differences in the EMG patterns of the muscles also corroborates the idea that the lesion of the ACL or its surgical reconstruction do not affect the control of voluntary movements during deep squatting. Contrary to common opinion, it has been shown that the ACL is submitted to similar and small strain force during knee flexion movements performed in an open chain and a closed chain, (i. e. parallel squatting) (Beynnon et al. 1996). So, we cannot rule out the possibility that, during deep squatting, the strain to which that the ACL would be submitted is not enough to generate the changes in the movement kinematics reported in the literature (Marans et al. 1989; Brandsson et al. 2001; Brandsson et al. 2002).

If that were true, then for some tasks the knee would not be submitted to a net joint torque that could generate the anteriorization and rotation of the tibia, provoking the so called "*giving way phenomena*" or the kinematic changes reported in the literature for individuals with lesion of the ACL (Marans et al. 1989; Brandsson et al. 2001; Brandsson et al. 2002; Losee, 1978; Kennedy et al. 1978; Peterson et al. 1984). The answer to this question is beyond the scope of this study.

Despite these discrepancies in the literature, we can conclude that the lesion of the ACL does <u>not</u> compromise the ability to keep a strong stable knee, to the point of hindering the performance of squatting.

Our third hypothesis that the surgical reconstruction of the ACL would increase joint stability improving the kinematic and EMG patterns to the level observed in healthy individuals, was also compromised. We do not have any reason to believe that the surgical reconstruction of the ACL would be responsible for the outstanding performance observed in this group of individuals, for the simple reason that there were no group differences in the performance of deep squatting when this group was compared with the lesioned group.

Indeed, this observation corroborates with the observation that, during the stance support phase of the gait, the knee kinematics is similar between individuals with lesion and with surgical reconstruction of the ACL (Devita et al. 1998).

Finally, we cannot rule out the possibility that the group differences would be reflected in the kinetics and not in the kinematics, as described by Devita et al. (1998). These authors showed that, after reconstruction of the ACL and accelerated rehabilitation, these individuals walk with normal kinematic patterns, but altered joint torque. This observation is not trivial, because the kinematic of the movement is used to determine, via inverse dynamics, the torque at the joint. Some theories in motor control have shown variance in the kinematics, but invariance in the shape of the joint torque during voluntary upper-limb movements (Almeida et al. 1997). So, it is possible that the individuals with lesion and with surgical reconstruction of the ACL changed the impulse (i.e, the rate of

change in the muscle torque during the time) to obtain similar movement trajectory shown by normal individuals.

However, we have to consider that, if the individuals with lesion and with surgical reconstruction of the ACL used different kinetic patterns to perform deep squatting, these would be reflected in the EMG patterns of the muscles. This is not what we observed, since all three groups of individuals used similar EMG patterns for all muscles analyzed.

Mode of contraction of the bi-articular muscles during deep squatting

The mode of contraction (isometric, concentric, and eccentric) is determined by the total length of the muscle. Here we show how this length can vary by considering the change in the rate of movement excursions among the involved joints, and the origin and insertion of the bi-articular muscles.

Thus, the length of the rectus femoris, the biceps femoris and semitendineous muscles, that cross both the hip and the knee joint, did not vary during deep squatting, because both hip and knee joints moved at a similar rate (Figure 12). So, we can conclude that the muscles rectus femoris, biceps femoris and semitendinosus probably work in an isometric mode during the upward and downward phases of deep squatting.

The gastrocnemius crosses both the ankle and knee joints. During the downward phase, the length of this muscle decreased due to the rate of change in both ankle and knee joints (Figure 12). So, the gastrocnemius probably works in a concentric mode during downward squatting. The opposite is true during the

upward phase of squatting for the length of the gastrocnemius muscle. Because of that it probably works in an eccentric mode during the upward squatting.

PRACTICAL IMPLICATIONS

We also show that during deep squatting, the anterior and posterior muscles of the leg generate high levels of EMG activities during different modes of contraction, and with reciprocal pattern activities between the anterior and posterior muscles. During deep squatting, the ACL is submitted to small strain that decreases with flexion of the knee (Beynnon et al. 1996). Also, the ACL is submitted to similar strain when the knee works in open-chain (i.e., free flexion/extension) or in closed-chain (i.e., deep squatting) (Beynnon et al. 1996). Several Physical Therapy Protocols designed to recover functions in individuals with lesion of the ACL (Blackburn, 1985) and in individuals submitted to surgical reconstruction of this ligament (Irrgang and Harner, 1997; Wilk et al. 1997; De Carlo et al. 1997; Shelbourne & Nitz, 1990) recommend that reciprocal activation of muscles be increased. So, based on these findings, we one could recommend the use of deep squatting in the Physical Therapy Program for individuals with lesion and with surgical reconstruction of the ACL. However, it must be proven first that the deep squating is an appropriate treatment for this population.

Aknowledgement

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CAPÍTULO IV - "Modulation of thigh muscle activities when keeping knee extended against gravity"

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Key words: anterior cruciate ligament, motor control

ABSTRACT

In this experiment we first showed that the Central Nervous System modulates the electromyographic (EMG) activities of the thigh muscles to keep the knee joint extended against gravity (isometric contraction) immediately after a voluntary movement (isotonic contraction). At the target, the individuals failed to keep the extended limb completely stationed against gravity. Indeed, they increased the knee excursion by a few degrees and decreased the amount of the agonist and antagonist EMG activities. In addition, at the target, the variability of the angular excursion and EMG activities decreased. Our results will be discussed according to the two theories proposed to explain motor control (Gottlieb et al. 1989) *versus* (Feldman, 1974).

INTRODUCTION

The explanation for the observed relationship between electromyographic (EMG) activities and movement kinematics and kinetics has been addressed by two theories on motor control (Gottlieb et al. 1989 *versus* Feldman, 1974). According to Gottlieb et al. (1989), the physiological linkage between action potential and the formation of a crossing bridge between actin and myosin, which generate the contraction of the muscle extra-fuse fibers, is explained by increasing the amount of EMG activities with an increase in the muscle torque during isotonic (Gottlieb et al. 1989) and isometric contraction (Lawrence, and DeLuca, 1983).

On the other hand, for Feldman et al. (1998), the relationship between EMG activities and movement kinematics and kinetics is a direct consequence of central commands, that determine the new position of the joint. Based on this theory (Equilibrium Point Hypothesis), when a new position of a joint is defined, the state of the joint can be described with two variables (Asatryan and Feldman, 1965; Feldman et al. 1998). The first variable is the reciprocal activation, which could be characterized by the difference in the levels of muscle activation in the agonist and antagonist. This difference is assumed to assure the final position against the external load. The second variable is characterized by the sum of the levels of muscle activation in the agonist and antagonist that defines the state of co-activation of the muscles. The co-activation determines the level of the "joint stiffness" which defines the stability of the final position.

Both theories are based on the movements performed from a static position. In this initial state, the muscle would be completely relaxed. However, human movements very often occur in a chain, with one action immediately following another, forcing the muscle to shift from one state of activation to another. This shift can also involve a change in the mode of contraction of the muscle.

The theories on motor control do not address the behavior of a muscle that is forced to change from one mode of activation to another. Could these two theories account for the kinematic and EMG activities observed during an isometric contraction that occurs immediately after an isotonic contraction?

In this experiment we first showed that the Central Nervous System modulates the EMG activities of the thigh muscles to keep the knee joint extended against gravity (isometric contraction), immediately after a voluntary movement (isotonic contraction). At the target, the individuals failed to keep the limb extended against gravity. Indeed, they increased by a few degrees and decreased the amount of agonist and antagonist EMG activities. In addition, at the target, the variability of the angular excursion and EMG activities decreased. Our results will be discussed according to the two theories proposed to explain motor control (Gottlieb et al. 1989 *versus* Feldman et al. 1974).

METHODS

Subjects

We tested eight healthy subjects after they gave formal consent according to the procedures approved by the State University of Campinas. The average age of the subjects was 29.6 (SE=±1.6).

Procedure

At the initial position, the subject sat comfortably in a chair, with the hip, knee, and ankle joints flexed at 90° (full extension being 0°), and with the foot fully supported on the floor (Figure 14). From this initial position, the subject performed 80° of knee extension movements. The subject was instructed to perform the movement "as fast as possible" and to keep the limb segment stationed at the target position without making any voluntary correction during at least two seconds. Reaction time was not stressed and seven trials were recorded for each subject.

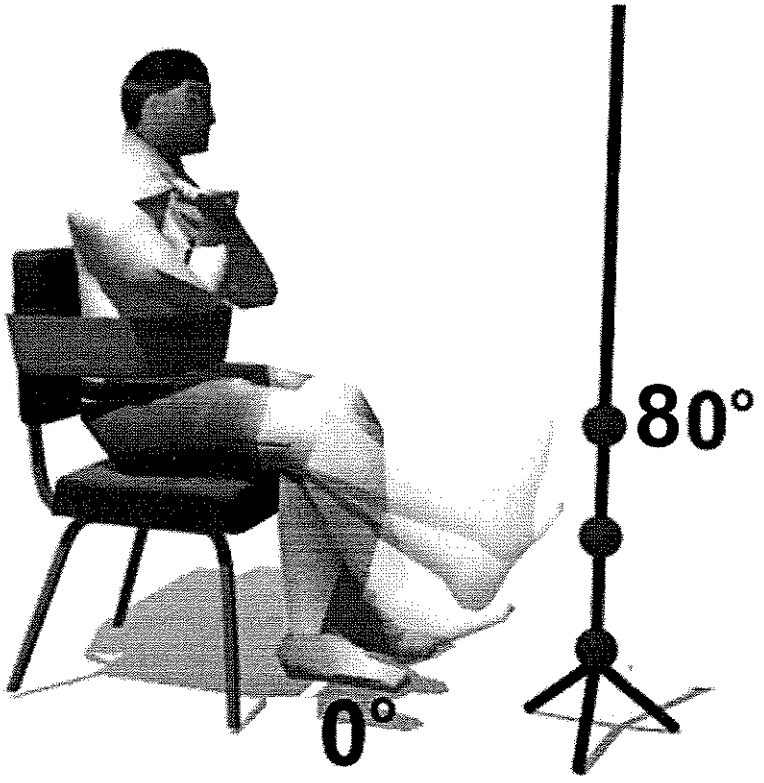

FIGURE 14: Illustration of the initial position of the subject and the distance of the knee extension movements (80°).

Kinematic data

We recorded the X, Y and Z coordinates of the LED marks using a tridimensional motion-analysis system (OPTOTRAK 3020) at 100 frames per second. The LED marks were attached to the hip (superior iliac crist), knee (lateral condyle) and ankle (external malleolus) joints. These coordinates were used to calculate the angular displacement and velocity bi-dimensionally (sagital plane).

The EMG muscle activities

We also recorded the EMG muscle activities of the rectus femoris and the biceps femoris muscles. These EMG activities were recorded using bipolar surface EMG electrodes DeLSys (model DE2.2L). The EMG signals were collected at 1000Hz, amplified (X 2000), band-pass filtered (20-450Hz) and recorded. Before data analyses the EMG signal was rectified and low-pass filtered by means of Butterworth filter with a cut-off frequency of 20 msecs.

QUANTIFICATION OF DATA

The angle excursion was defined as the angular difference between the initial and the final position, when the limb was already at the target. The target was positioned in such way as to constrain the knee (10 degrees of extension, with full extension being zero degrees).

The averaged agonist and antagonist EMG activities and the averaged angular excursion were calculated during ten intervals of 100 milliseconds each (see Figure 15 for illustration). The first interval initiates 150 ms after the first cross zero of the velocity (end of movement time).

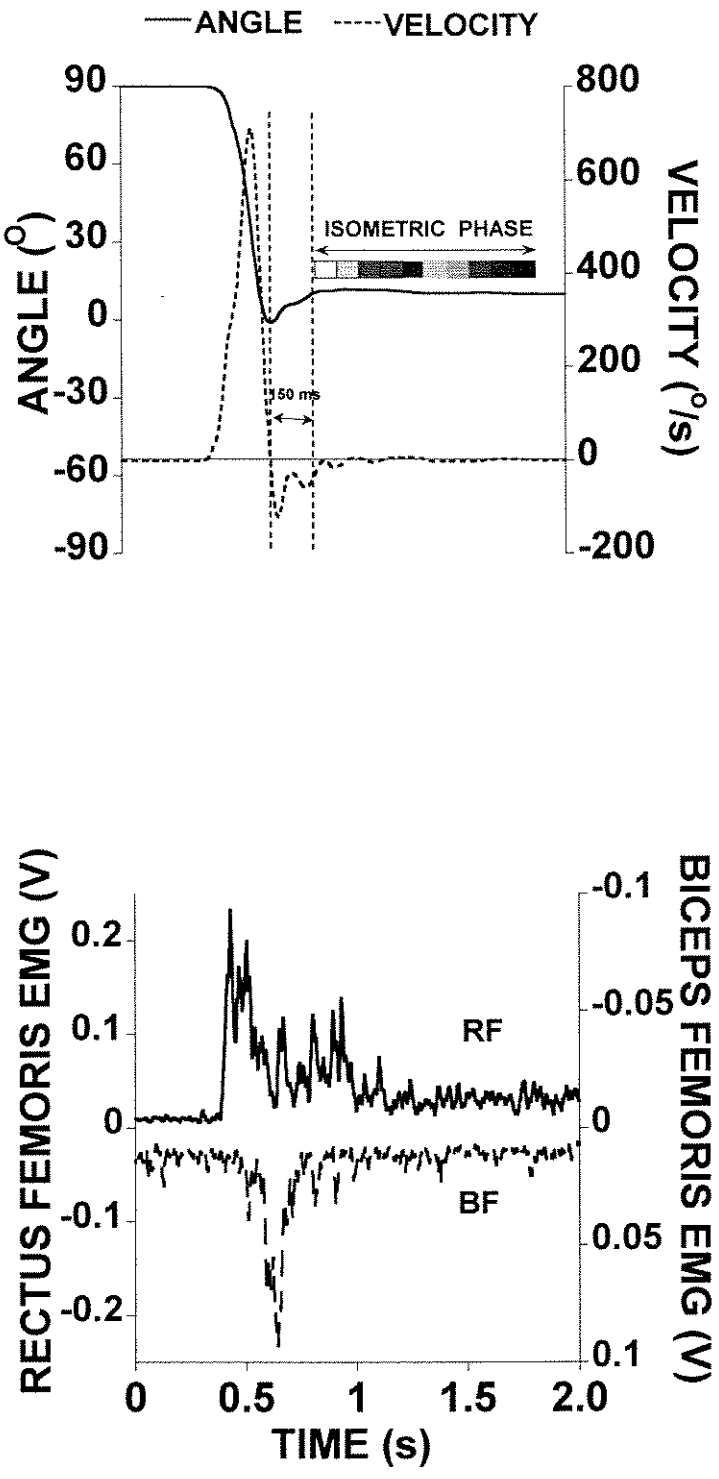

FIGURE 15: Angle and velocity of knee-extension movements performed as fast as possible (above). Muscle activities of rectus femoris- RF (agonist) and biceps femoris – BF (antagonist) (below). The time is in seconds. The angle is in degrees, the velocity in degrees per second and the electromyographic activity in Volts.

STATISTIC ANALYSIS

The effects within subjects (distances and type of muscles) were analyzed for several of the dependent variables studied using a mixed ANOVA model.

RESULTS

All subjects performed the tasks very well with similar movement excursion and velocity to the target. At the target they kept the leg against gravity with similar kinematic and EMG patterns. The subjects moved the leg to the target position, close to full knee extension, as fast as possible, and kept it there with small oscillation. Even though the subjects were instructed not to make any voluntary correction at the target, during the first second at the target they increased the knee extension around 2 degrees (Figure 16a) with an averaged variability of 0.5 degrees (Figure 16b).

These observations were confirmed by the ANOVA tests (see Table 08) that showed main effect for the following variables: time interval, the angular excursion and the variability in which the subjects kept the limb at the target.

TABLE 08: Analysis of variance (ANOVA) of kinematic and EMG. Angular excursion and standard deviation of knee. Average EMG activities of muscles rectus femoris, biceps femoris and standard deviation of the average. The EMG data is not normalized.

	Distance	
	$F_{(9,63)}$	p
knee angle	11.458	0.0001*
standard deviation of angular excursion	11.133	0.0001*
rectus femoris average EMG	5.053	0.0001*
rectus femoris standard deviation of the average	.4.631	0.0001*
biceps femoris average EMG	2.232	0.0336*
biceps femoris standard desviation of the average	.1.917	0.0413*

In other words, at the target, the subjects tended to extend the limb a few degrees and, therefore, the variability of the fluctuation of the limb at the target position decreased with time.

During the first second at the target the amount and the variability of agonist (rectus femoris, Figures 16c and 16d) and antagonist (biceps femoris, Figures 16e and 16f) EMG activities decreased with time intervals (see ANOVA tests).

The levels of muscle activation in the agonist (rectus femoris) and in the antagonist (biceps femoris) were calculated by integrating the EMG activities of these muscles during ten intervals of 100 ms each with the first interval beginning 150 ms after the first cross zero of the velocity (see Figure 13).

The reciprocal level of activation was calculated by subtraction of EMG activity of the rectus femoris with the biceps femoris (Figure 17–top). The level of co-activation was calculated by amount of EMG activity of the rectus femoris with the EMG activity of the biceps femoris (Figure 17–below).

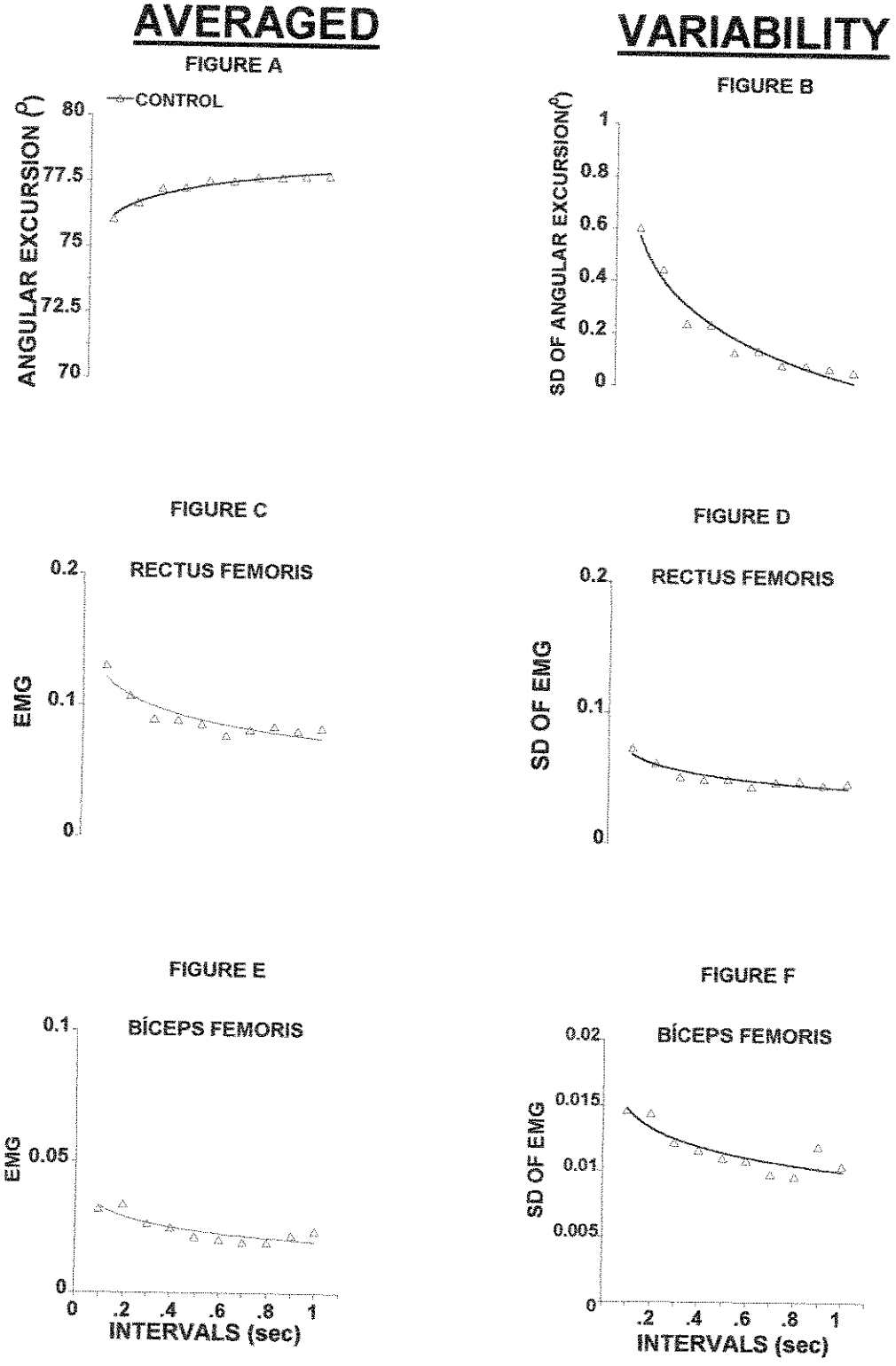

FIGURE 16: The pattern of knee excursion and EMG activities during ten intervals of 100 msecs each. The first interval initiates 150 ms after the first cross zero of the velocity (end of movement time). The angular excursion and the variability in knee joint (Figures a and b). The amount and the variability of agonist (rectus femoris, Figures c and d) and antagonist (biceps femoris, Figures e and f) EMG activities during the first second at the target during isometric contraction.

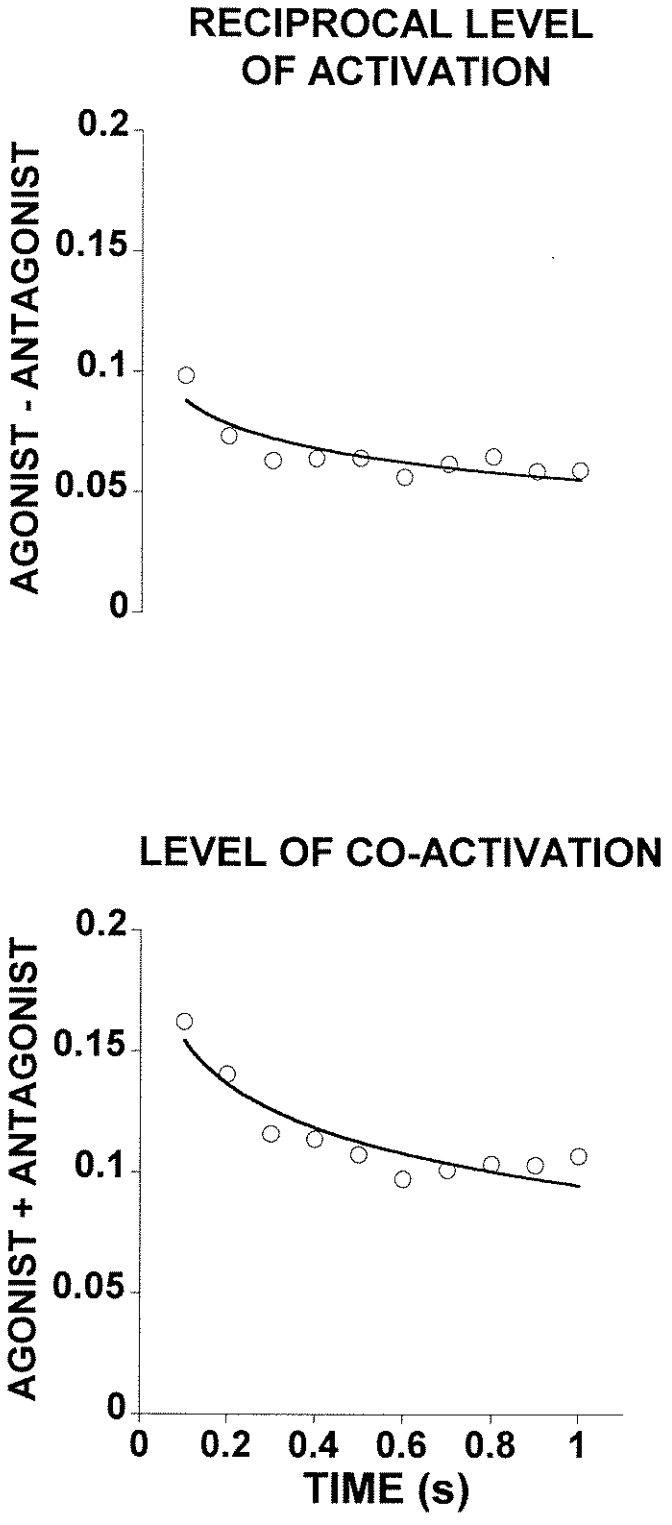

FIGURE 17: The reciprocal level (top) of activation of the rectus femoris muscle (agonist) subtracted from the biceps femoris muscle (antagonist). The level of co-activation (below) of rectus femoris muscle summed with the biceps femoris muscle during isometric contraction. The time is in seconds.

DISCUSSION

It was a surprise to observe that individuals with an intact knee joint increased the knee excursion around two degrees, despite being instructed to keep the leg stationed against gravity at the target position (Figure 16). Also, the observation that the amount of the agonist and antagonist EMG activities decreased when the limb was kept extended at the target differs from other studies, which showed an increase in the amount of agonist EMG activities with movement excursion (Gottlieb et al. 1990; Kirsh and Rymer, 1987). These kinematic and EMG behaviors were very robust across all subjects and the variability of these behaviors decreased with the time in which the leg was kept at the target against gravity.

At the static position, the torque generated in the knee joint varied with the degree of extension of the knee, being maximum at the full extension, and zero at 90 degrees of flexion. If the subjects kept the limb completely stationed at 10 degrees (final position), the muscle torque at the knee joint would be 98.48% of the maximum value required to keep the knee at full extension (zero degrees). With an increase of two degrees of knee extension at the final position, as observed in our data, the knee muscle torque would be 99.02% of the maximum value required to keep the knee at full extension.

So, to keep the leg extended against gravity, the individuals increased the muscle torque at the knee joint, but decreased the amount of agonist and antagonist EMG activities. On the other hand, several studies reported an increase in the amount of EMG activities with increasing muscle torque during

isotonic (Gottlieb et al. 1989) and isometric contraction (Lawrence and DeLuca, 1983).

In the studies reporting a positive correlation between muscle activities and muscle torques (Gottlieb et al. 1990; Kirsh and Rymer, 1987), the individual initiates the activation of the muscle from a relaxed state of activity. In our study, the individuals arose from isotonic contraction to achieve the target position and, there, generated an isometric contraction to keep the limb stationed at the new position (10 degrees - immediately after an isotonic contraction that threw the limb at the target position). In other words, in our study, to keep the limb at the target, the CNS had to send one command to the alfa motorneuron pool to change the mode of contraction (from isotonic to isometric).

Could the theories on motor control explain the results observed in our experiments?

Based on the Equilibrium-Point Hypothesis (Feldman et al. 1974), the final position against the external load of the knee joint should be defined by a reciprocal level of activation, which could be characterized by the difference in the levels of muscle activation in the agonist (quadriceps) and antagonist (hamstrings). The observed increase of two degrees of knee extension could be explained if the antagonist dropped more dramatically than the agonist, generating an additional extensor torque.

In our experiment, the muscle activation of both agonist and antagonist dropped, but at similar rate, during the time in which the weight of the external load (the mass of lower leg plus foot) was kept against gravity (Figure 16).

However, the agonist activity was larger than the antagonist activity. Hence, the difference between the levels of activation of these two muscles also decreased with time. So, based on the reciprocal level of activation, the Equilibrium-Point Hypothesis cannot explain the observed decrement in the EMG activities with an increase in the angular excursion at the final position.

Also, based on the Equilibrium Point Hypothesis, the Central Nervous System also controls the movement based on a second variable, characterized by the sum of the levels of muscle activation in the agonist (quadriceps) and antagonist (hamstrings). The sum of these two muscles would than define the state of co-activation of the muscles. The state of co-activation would than determine the degree of the "joint stiffness", defining the stability of the final position. The variability of the final position was represented by the standard error of the final knee angle, which decreased with time (Figure 16b).

Concomitantly, the sum of the level of both agonist and antagonist muscles decreased with the time in which the leg was kept at the target (Figure 17-below). This fact could be explained by the prevalence of the agonist activities over the antagonist activities. Our conclusion is that the Equilibrium-Point Hypothesis cannot account for the observed stability of the knee joint at the final position.

Otherwise, the idea that muscle activities and muscle torque must be related because the physiological linkage between the cross bridge formation and the muscle extra-fuse fibers contraction <u>also cannot</u> be used to explain the observed EMG and kinetic behavior. Based on this idea, one would expect to

observe the increment (and not a decrement as we reported) in the amount of agonist EMG and/or in the reciprocal level of activities to explain the increased demand for muscle torque with the increase in knee extension.

So, we can conclude that the theories on motor control cannot explain the decrease in the amount of EMG activities with increase of the angular excursion observed during isometric contraction. In our experiment, the subjects used such a strategy to keep the limb at the target position, against gravity, immediately after an isotonic contraction. These data showed that changes from one state of contraction (isotonic) to another (isometric) can exert a great influence on the pattern of the EMG, and subsequently, on the movement. New studies are needed to explore the effects of the isotonic contraction on the isometric contraction as well as the shifts between other states of muscular contraction before we can propose a putative explanation for the observed phenomena.

Aknowledgement

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CAPÍTULO V - "The lesion of anterior cruciate ligament and its surgical reconstruction do not affect the modulation of electromyographic activities during isometric contraction"

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Key words: anterior cruciate ligament, motor control

ABSTRACT

We showed here that the lesion (n=8) and the surgical reconstruction (n=8) of the ACL do not compromise the strategy to keep the leg extended against gravity, after a voluntary movement of the knee. As in the control group (n=8), the subjects of the lesioned and reconstructed group failed to keep the knee completely stationed against gravity. At the target, the subjects of all groups increased knee extension around two degrees during the interval of one second. During the first second at the target the amount and the variability of agonist and antagonist electromyographic (EMG) activities decreased with time intervals in a similar pattern for all three groups of subjects.

INTRODUCTION

Appoximately 1 to 2,5% of the total area of the human anterior cruciate ligament (ACL) is composed of proprioceptive receptors, such as type I (Ruffini endings), II (Pacinian corpuscles), III (the Golgi tendon organ) and IV (free nerve endings) (Schutte et al. 1987; Schultz et al. 1984; Zimny et al. 1986). The function of these receptors and the real mechanism by which the Central Nervous System uses this information to control movement is not well-known.

Nevertheless, there are several studies showing proprioceptive deficits. Some studies showed deficit in the static and dynamic capability of individuals with ACL lesion to detect the position of the limb (Barrack et al. 1989; Borsa et al. 1997; Corrigan et al. 1992). On the other hand, others studies failed to observe these proprioceptive deficits (Good et al. 1999 ; Fridén et al. 1998).

In favor of the proprioceptive deficit in these individuals, there are some studies showing delay in the onset of electromyographic (EMG) activities due to a reflex response generated by muscle stretching (Beard et al. 1993; Wojtys and Huston, 1994). However, Jennings and Seedhom (1994) failed to observe a change in the delay of the onset of muscle activities due to a reflex response in these individuals.

There is robust evidence showing that individuals with ACL lesion have several functional problems (Noyes et al. 1983c; Noyes et al. 1985; Beard et al. 1993; Andriacchi, 1990; Gauffin and Tropp, 1992). Despite the dispute about the

proprioceptive deficit in individuals with lesion of the ACL, some authors associate these deficits with these functional problems (Beard et al. 1993; Wojtys and Huston, 1994).

There are several sources that could account for the controversy reported in the literature about the existence or not of the proprioceptive deficit in these individuals. It is well-known that the static and dynamic information about limb position originates mainly in the intrafuse fibers of the muscles (spindle) (Matthews, 1972; Matthews, 1964). Thus, it is possible that some individuals with lesion of the ACL have a compensatory mechanism by which the information from the muscle spindle or other joint-capsule receptors (Guanche et al. 1995) could well compensate the lack of information from the ACL.

Also, the tension in the ACL varies with joint excursion, being maximum at the end of knee extension (Beynnon et al. 1997). So, in order to keep the knee joint extended against gravity, the Central Nervous System must know the joint position and the appropriate muscle tension. The muscle tension is signaled by the Golgi tendinius organ (Matthews, 1972). We do not know at this point if the receptors of the ACL are also able to nourish the Central Nervous System (CNS) with information about the tension on this ligament. Because the ACL is submitted to tension, it is plausible to assume that the receptors inside this ligament also fire with increased tension (Miyatsu and Atsuta, 1993). So, the conflicting results reported in the literature about the possible proprioceptive deficits in the lesion of the ACL could also be attributed to different sources of information.

There is also a possibility that some individuals with lesion of the ACL have more joint instability than others with a similar lesion (Wojtys and Huston, 1994; Noyes et al. 1985). So, a more unstable knee would predispose the individual to have more stretching of the muscle for a certain amount of disturbance, generating a great reflex response. Thus, individual variability in the sample of these studies could also account for the difference in the reported results. If that is true, one would expect to observe decreased proprioceptive deficit in individuals with surgical reconstruction of the knee, as reported in the literature (Fremerey and Lobenhoffer, 2000).

Despite also the possible explanations for these controversies, the idea that a lesion of the ACL generates proprioceptive deficits has had great impact on the physical therapy treatment of individuals with lesion or reconstruction of this ligament (Lephart et al. 1997; De Carlo et al. 1997; Mangine and Kremchek, 1997).

In our daily life activities very often we perform a voluntary movement, stopping the limb extended against gravity. This task requires the appropriate sense of joint position and muscle tension to move the limb to the target and to keep it stationed there.

We have shown that individuals with an intact and stable knee were unable to keep their extended thigh completely stationed against gravity, after a voluntary movement. Indeed, they increased the knee excursion in about two degrees and decreased the amount of agonist and antagonist muscle activities, when the leg was kept extended (see Chapter IV - "Modulation of thigh muscle

activities when keeping knee extended against gravity"). The variability of this postural strategy decreased during the first second that the limb was kept extended.

Here we first tested the hypothesis that the possible lack of proprioceptive information, due to the chronic lesion of the ACL, would affect the postural strategy of keeping the leg extended against gravity after a voluntary movement. Second, we tested the hypothesis that the surgical reconstruction of the ACL would increase the mechanical joint stability and, because of that, the postural strategy of keeping the leg extended against gravity would be ameliorated.

METHODS

Subjects

We tested eight subjects without any neurological or orthopedic dysfunction (control group), eight subjects with lesion of the anterior cruciate ligament that were not submitted to the surgical reconstruction (lesion group), and eight subjects that had the anterior cruciate ligament reconstructed surgically (reconstructed group). All subjects were tested after giving formal consent according to the procedures of the State University of Campinas. The average age was 29.1 (SE= 1.9); 29.2 (SE= 2.3) and 26.0 (SE=2.3) respectively for the subjects of the control, lesion and reconstruction groups. Information about each of the subjects is shown in Table 09.

TABLE 09: Clinical examination of all individuals of the three groups.

Groups		Subjects							
		1	2	3	4	5	6	7	8
•	*Age (years)*								
CON		25.6	29.3	20.5	34.7	36.4	33.6	24	28.9
LES		19.9	27.5	40	35.6	22.9	33	28.6	26.5
REC		21.9	26.2	37.9	19.2	20.9	30.2	30.4	21
•	*Side of lesion or tested*								
CON		R	R	L	L	L	L	R	L
LES		R	R	L	L	L	R	L	R
REC		L	R	L	L	L	R	R	R
•	*Side of dominance*								
CON		R	R	R	R	R	R	R	R
LES		R	R	R	R	R	R	L	L
REC		R	R	R	R	R	R	R	R
•	*Edema and pain 24 hours after the lesion*								
LES		+	-	+	+	+	+	-	-
REC		-	-	-	+	+	+	+	+
•	*Associated lesions*								
LES		-	-	-	-	-	-	-	MM*
REC		-	-	BM	BM	BM	BM	-	MM
•	*History of giving way*								
LES	*(BS)*	+	-	-	+	+	-	+	-
REC	*(BS)*	+	+	+	+	+	+	+	+
REC	*(AS)*	-	-	-	-	-	-	-	-
•	*Anterior Drawer Test*								
LES	*(BS)*	+	+	+	+	+	+	+	+
REC	*(BS)*	+	+	+	+	+	+	+	+
REC	*(AS)*	-	-	-	-	-	-	-	-
•	*Pivot Shift Test*								
LES	*(BS)*	+	+	+	+	+	+	+	+
REC	*(BS)*	+	+	+	+	+	+	+	+
REC	*(AS)*	-	-	-	-	-	-	-	-
•	*Lachman Test*								
LES	*(BS)*	+	+	+	+	+	+	+	+
REC	*(BS)*	+	+	+	+	+	+	+	+
REC	*(AS)*	-	-	-	+	-	-	-	-
•	*Time until the test (months)*								
LES	*(IL)*	48	48	60	25	12	40	69	60
REC	*(IL)*	40	34	20	21	41	45	24	19
REC	*(FS)*	38	32	10	10	20	28	21	17
•	*Type of reconstruction*								
REC		PL	PL	PL	PL	PL	PL	PL	PL

CON=control; LES=lesioned; REC=reconstructed *R=right; L=left*

BS=before surgery; AS=after surgery *IL= from the initial lesion; FS= from surgery*

*MM=medial meniscus; LM=lateral meniscus; BM=both meniscus; PL= patellar ligament; *condral lesion*

All subjects of the lesioned group showed positive signs of lesion of the anterior cruciate ligament in the anterior drawer (Hughston et al. 1976), Lachman (Torg et al. 1976; Wroble and Linddenfeld, 1990) and pivot shift tests (Galway et al. 1972; Losee, 1983). In addition of these specific tests, all subjects of the lesioned group were tested in the following complementary tests: abduction at 0 and 30° (Hughston et al. 1976), adduction at 0 and 30° (Hughston et al. 1976), Mc Murray's test (Crewshaw, 1989), and the posterior drawer test (Hughston et al. 1976). The lesion of the ACL was also confirmed by magnetic ressonance imaging.

Four out of eight subjects of the lesion group had a history of knee giving way with recurrent sprain. All subjects of the control group and the reconstructed group were tested negatively for all the specific and complementary tests pointed out above. All tests were performed by the same physician (the third author). After the surgery, none of the subjects of the reconstructed group presented signs of giving way, new lesion, and arthrofibrosis of the reconstructed knee (Shelbourne et al. 1991). None of the subjects of the three groups felt pain during the test sessions, signs of ligament laxity or decreased amplitude of movement of the knee joint. The subjects of the three groups were all males and did not have any history of lesion of the meniscus or of ligaments of the contra-lateral knee. The dominant limb was determined by asking the participant which leg he preferred for kicking.

Procedure

At the initial position, the subject sat comfortably in the chair, with the hip, knee, and ankle joints flexed at 90° (full extension being 0°), and with the foot fully supported on the floor (Figure 18). From this initial position, the subject performed 80° of knee-extension movements. The subject was instructed to perform the movements "as fast as possible" and to keep the limb segment at the target position without making any voluntary correction during at least two seconds. Reaction time was not stressed. We recorded seven trials for each target distance.

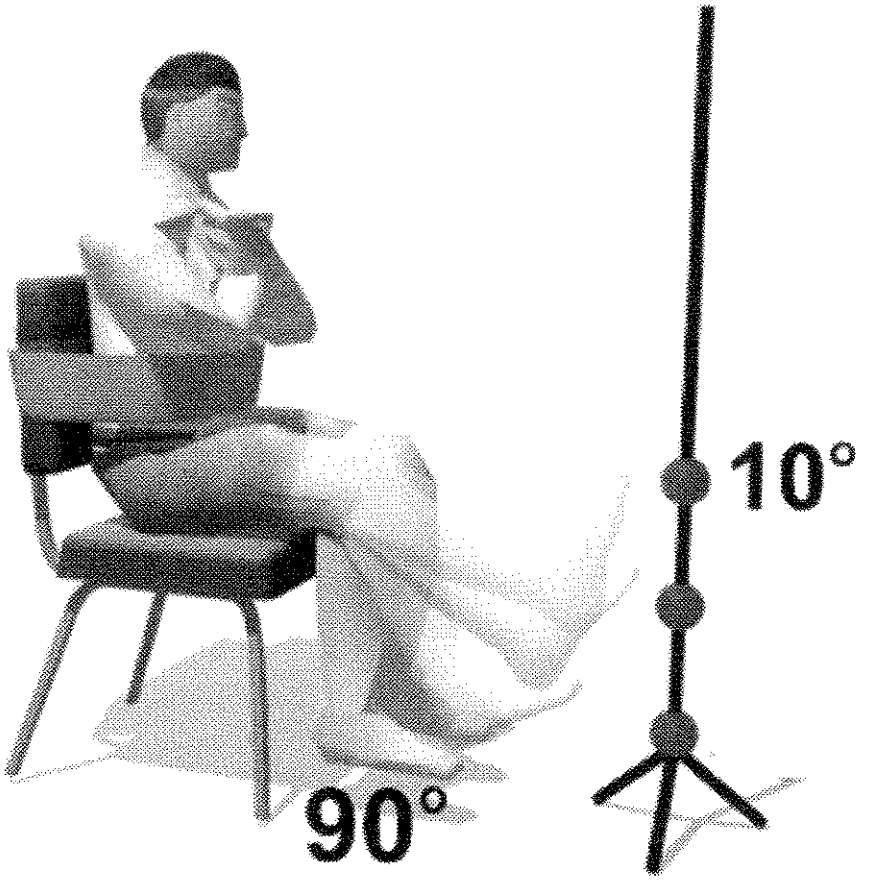

FIGURE 18: Illustration of the initial position of the subject and the distance of the knee-extension movements (80°). The subject was instructed to maintain the limb at the target (10°) at the end of the movements.

Kinematic data

We recorded the X, Y and Z coordinates of the LED marks using a tridimensional motion-analysis system (OPTOTRAK3020) at 100 frames per second. The LED marks were attached to the hip (superior iliac crist), knee (lateral condyle) and ankle (external malleolus). These coordinates were used to calculate the knee's angular displacement and velocity bi-dimensionally (sagital plane).

EMG recordings

We also recorded the EMG muscle activities of the rectus femoris and the biceps femoris muscles. These EMG activities were recorded using bipolar surface EMG electrodes DeLSys (model DE2.2L). The EMG signals were collected at 1000Hz, amplified (X 2000), band-pass filtered (20-450Hz) and recorded. Before data analyses, the EMG signal was rectified and low-pass filtered by means of Butterworth filter with a cut-off frequency of 20 msecs.

QUANTIFICATION OF DATA

The angle excursion was defined as the angular difference between the initial and the final position during the movement.

The averaged agonist and antagonist EMG activities, and the averaged angular excursion were calculated during ten intervals of 100 milliseconds each (for more information, see Figure 15 in Chapter IV – "Modulation of thigh muscle activities while keeping knee extended against gravity"), at the end of movement time. The end of movement time was defined as 150 ms after velocity first crossed zero.

Each of the integrated EMG values cited above was normalized by dividing it by its correspondent baseline value. The baseline was calculated by integrating the EMG activities during the first recorded 100 milliseconds (more than one second before the onset of the movement).

STATISTIC ANALYSIS

The effects within subjects (distances and type of muscles) and between subjects (the three groups) were analyzed for several of the dependent variables studied using a mixed ANOVA model.

RESULTS

All subjects of the three groups performed the tasks very well with similar movement excursion (Figure 19) and velocity to the target, as shown by an ANOVA test for the amount of angular excursion ($F_{(2,21)}$=.1470; p=.8700) and peak velocity of the movement ($F_{(2,21)}$=.1765; p=.8395).

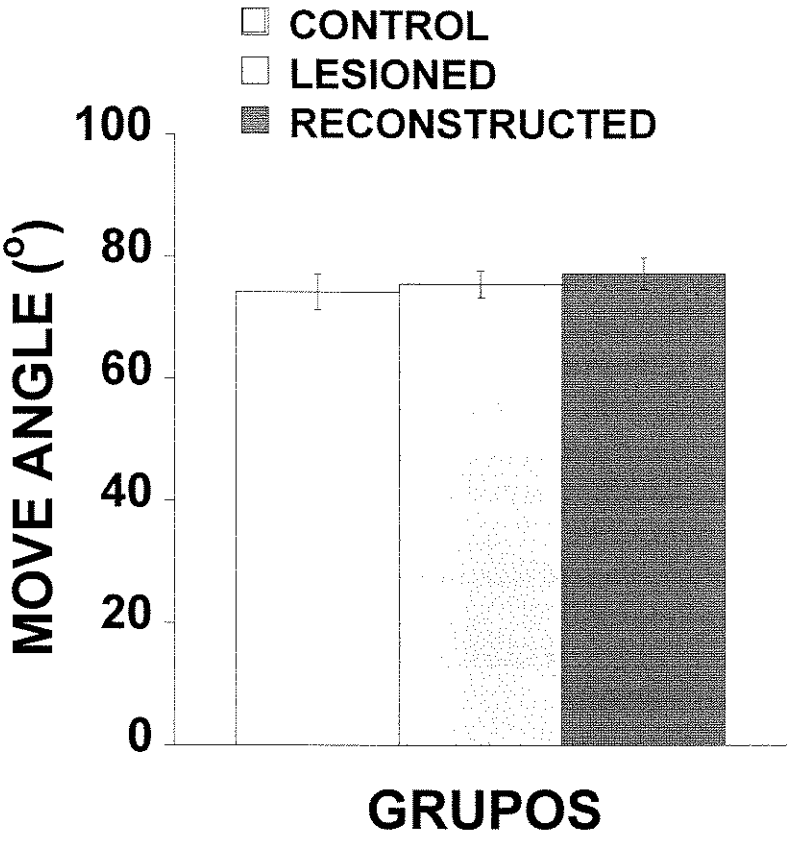

FIGURE 19: Illustration of the movement excursion of the knee to the target (10°). Note that the individuals of the three groups moved the leg to the target position. The bars designate the standard errors for each group.

At the target they kept the leg against gravity with similar kinematic and EMG patterns.The subjects of the three groups moved the leg to the target position, closed to full knee extension, as fast as possible, and kept it there with small oscillation. The subjects were instructed not to make any voluntary correction at the target, during the first two seconds after the end of the movement. Nevertheless, they increased the knee extension by about 2 degrees (Figure 20a) with an average variability of 0.5 degrees (Figure 20b). These observations were confirmed by the ANOVA tests (see Table 10) that show main

effect for time interval, but not for groups of subjects for the angular excursion and its variability.

TABLE 10: Analysis of variance (ANOVA) of kinematics and EMG for three groups. Angular excursion and standard deviation of knee. Average EMG activities of muscles rectus femoris, biceps femoris and standard deviation of the average. The EMG data are not normalized.

	Group effect		*Interval*		*Interaction group effect x interval*	
	$F_{(2,21)}$	p	$F_{(9,21)}$	p	$F_{(18,21)}$	p
Knee angle	.1470	.8700	28.403	.0000	.410	.9844
Angular excursion *(standard deviation)*	.777	.4739	29.437	.0000	.640	.8642
Rectus femoris *(average EMG)*	.772	.9260	8.4388	.0000	.7252	.7817
Rectus femoris *(standard deviation of the average)*	.252	.9751	7.2556	.0000	.3925	.9879
Biceps femoris *(average EMG)*	.6298	.5447	6.0444	.0000	.9676	.4998
Biceps femoris *(standard deviation of the average)*	.2356	.7930	4.4069	.0000	.9128	.5644

In other words, at the target, the subjects of all three groups tended to extend the limb a few degrees, but the variability of limb fluctuation at the target position decreased with time.

During the first second at the target, the amount and the variability of agonist (rectus femoris, Figures 20C and 20D) and antagonist (biceps femoris, Figures 20E and 20F) EMG activities decreased with time intervals in a similar pattern for all three groups of subjects. These observations were confirmed by the ANOVA tests that revealed a main effect for time interval, but not for groups for the amount and variability of agonist and antagonist activity during the time in which the limb was kept at the target.

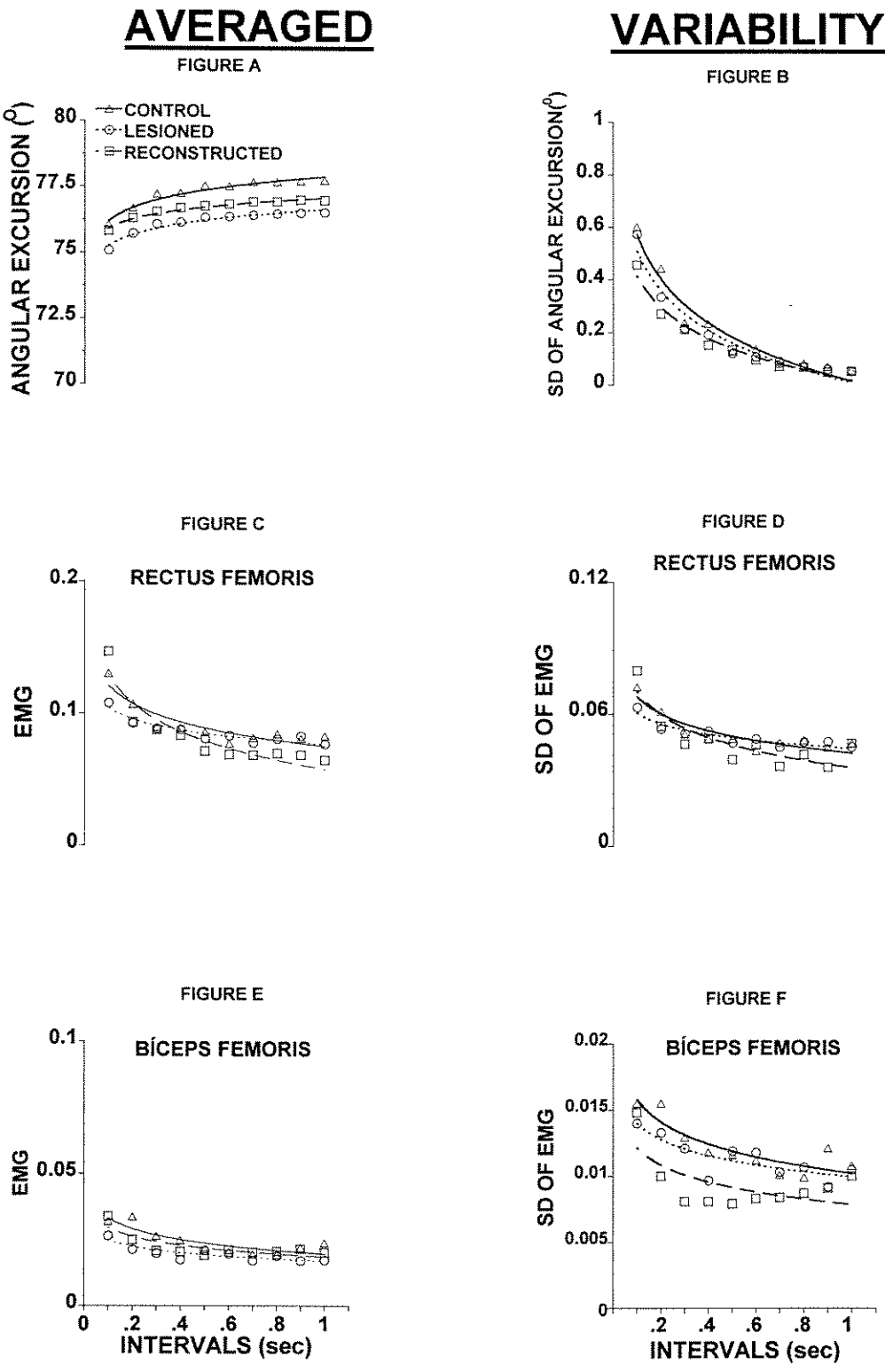

FIGURE 20: The pattern of knee excursion and EMG activities during ten intervals of 100 msecs each. The first interval initiates at 150 ms after the first cross zero of the velocity (end of movement time). The angular excursion and the variability in knee joint of the three groups (Figures a and b). The amount and the variability of agonist (rectus femoris, Figures c and d) and antagonist (biceps femoris, Figures e and f) EMG activities during the first second at the target during isometric contraction of the three groups. Control group (triangle); Lesion group (circle); Reconstructed group (rectangle). The time is in seconds.

DISCUSSION

Contrary to our initial hypothesis, we showed here that the lesion of the ACL does not compromise the strategy of keeping the leg extended against gravity after a voluntary movement of the knee (Figure 20). As in the control group, the subjects of the lesioned group failed to keep the knee completely stationed against gravity at the target. At the target, the subjects of both groups increased knee extension by about two degrees during the interval of one second. Contrary to what has been reported in several studies (Gottlieb et al. 1989; Lawrence, and DeLuca, 1983), the increase in knee extension against gravity was accompanied by a decrease in the amount of agonist and antagonist EMG activities (Figure 20). The variability of the kinematics and EMG also decreased with the time in which the limb was kept at the target for both groups.

One possibility for the increment in knee extension would be a damping phenomena due to the inertia, that would force the limb to oscillate at the target until it could achieve a completely stable final position. We believe that this phenomena could mainly explain the oscillation observed during the first 150 ms after velocity first crossed zero. However, this time was not considered in our analysis. Also, even though the limb was oscillating at the target, the extension of the knee increased in spite of the fact that the individuals were asked to avoid any kind of voluntary correction. So, we do not believe that the increment in knee extension could be associated to a mechanical reaction due to the movement of the shank and foot on the thigh.

With the increase in knee extension there is additional need for more muscle torque (Gottlieb et al. 1989; Lawrence, and DeLuca, 1983). Several

studies reported an increase in the amount of EMG activities with an increase in muscle torque during isotonic (Gottlieb et al. 1989) and isometric contraction (Lawrence and DeLuca, 1983). However, in the study reported here, the individual increased the amount of muscle torque to keep the limb raised against gravity due to the small increase in knee extension at the target, but a decrease in the amount of agonist and antagonist muscle activities (Figure 20).

In the experiment cited in Chapter IV – "Modulation of thigh muscle activities when keeping knee extended against gravity", the individual started the muscle contraction from a relaxed initial position. In our study the individuals moved the limb to the target by activating the quadriceps muscles in a concentric mode, and then kept the limb at the target by shifting the mode of contraction of these muscles to an isometric state. Because the limb oscillated at the target, and increased the knee extension, the exact transition of one mode of contraction (i.e., concentric) to another (i.e., isometric) cannot be well-defined.

We also know that isometric contraction generated more muscle force than concentric isotonic contraction (Westing et al. 1990). So, it is possible that when the activation of the muscle shifts from concentric isotonic contraction to isometric contraction it ends up generating a larger amount of muscle torque for similar amounts of EMG muscle activity. This could explain why the amount of EMG decreased, but the knee extension increased.

Could it be that the increment in the amount of knee extension is due to a miscalculation of the proprioceptive information obtained from the receptors of muscles and joints, including those of the ACL? Several receptors are sensitive

to changes in muscle lengthening (Matthews, 1972; Matthews, 1964) and, at least theoretically, it is possible that the CNS is aware of the increment in the knee extension at the target. However, we do not know why the CNS chooses to increase the knee extension at the target.

Despite the putative explanations for the observed postural strategy, the fact is that the lesion of the ACL did not affect this strategy. Because this strategy must involve a good sense of static and dynamic joint position and of muscle force, our observations support those studies that did not observe proprioceptive deficit in individuals with lesion of the ACL (Good et al. 1999; Fridén et al. 1998).

We have to keep in mind that the lesioned group was composed of individuals tested at least 10 months after the ligament rupture (see Table 09). After that length of time, it is quite possible that information from the receptor of other structures (i.e., muscles and joint capsules) could well compensate for the lack of information from the ACL.

We also showed that the surgical reconstruction of the ACL does not affect the kinematic and electromyographic strategy to keep the limb extended against gravity after a voluntary movement. So, our hypothesis that the surgical reconstruction of the ACL would increase the mechanical joint stability, leading to better control of the extended limb against gravity is compromised. Compared with the subjects of the reconstructed group, lesioned subjects had less stable knees. Nevertheless, the strategy used to keep the limb at the target did not vary across all three groups.

There is some evidence showing that the new ligament used in the surgical reconstruction can recover part of the capability to nourish the CNS with proprioceptive information (Barrack et al. 1997; Ochi et al. 1999). However, based on our data, the idea that the surgical reconstruction of the ACL would improve a possible proprioceptive deficit due to lesion of the ACL must be taken with caution.

We cannot rule out the possibility that the lesioned group learned, after the ligament rupture, how to compensate for the lack of proprioceptive information from the ACL, using information from other sources. The same kind of reasoning could be applied to the reconstructed group. However, we have to at least be cautious before starting to focus the physical therapy treatment of these individuals on the proprioceptive training.

Aknowledgement

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CAPÍTULO VI- CONCLUSÃO

Os experimentos realizados nesta tese não revelaram alterações cinemáticas, cinéticas ou nos padrões da atividade muscular em indivíduos com lesão ou reconstrução do LCA durante movimentos funcionais como a extensão do joelho, o agachamento ou a manutenção do membro estendido contra a ação da gravidade. Estas pesquisas são de certa forma confirmadas por outros estudos, principalmente durante a marcha.

Um dos motivos que explicam a diferença destes achados com os trabalhos que mostram alterações cinemáticas, cinéticas e eletromiográficas, em indivíduos com lesão ou reconstrução do LCA, é a composição dos grupos investigados. Muitas vezes, há uma grande variabilidade intra-grupo com relação ao tempo da lesão ou da reconstrução cirúrgica. Outra possibilidade é a falta de padronização entre os estudos com relação às diferenças nas cargas impostas para gerar distúrbios neuro-musculares; nas velocidades dos movimentos estudados; nas tarefas motoras; no grau de exigência em cada tarefa investigada; nas estruturas lesadas em adição ao LCA; no nível de aderência na atividade esportiva dos indivíduos testados; na precisão dos equipamentos para quantificação do movimento ou, mesmo; em diferenças metodológicas para análise dos resultados.

Nossos dados permitem apontar que, apesar de alguns indivíduos lesados apresentarem alterações na performance durante movimentos funcionais, episódios de falseio ou instabilidade detectada nos testes clínicos,

estes distúrbios articulares não afetam a cinemática do joelho ou os padrões eletromiográficos durante o movimento de extensão do joelho, o agachamento ou na manutenção da perna estendida isometricamente contra a ação da gravidade.

Uma das limitações das análises mostradas nesta tese, é que foram investigados movimentos realizados essencialmente no plano sagital e em condições que não causam tensão acentuada no LCA. É possível que esforços que requeiram, preferencialmente, torques rotacionais ou que provoquem aumento da tensão no LCA, causam uma resposta neuro-muscular mais acentuada. O próximo passo, será analisar estes movimentos rotacionais, reproduzindo as tensões geradas durante as atividades funcionais.

CAPÍTULO VII - REFERÊNCIAS BIBLIOGRÁFICAS

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