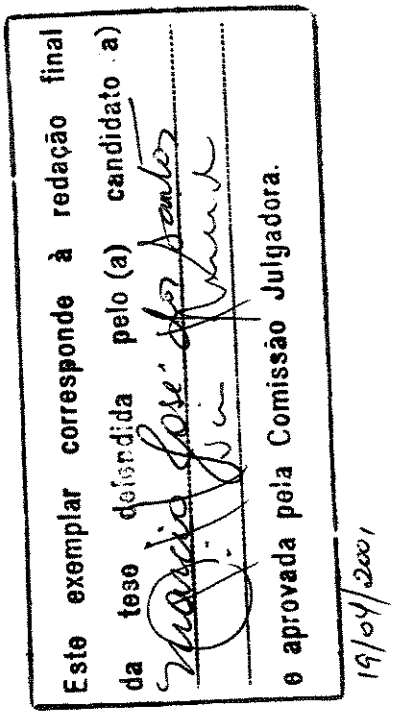
UNIVERSIDADE ESTADUAL DE CAMPINAS

MARCIO JOSÉ DOS SANTOS

O Controle dos Movimentos Voluntários do Ombro em Nadadores com Instabilidade Glenoumeral



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Orientador: Prof. Dr. Gil Lúcio Almeida

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RESUMO

A Síndrome do Impacto do Ombro (SIO) é freqüentemente observada em movimentos repetitivos do ombro acima de 60 graus durante a realização de atividades atléticas ou profissionais. Essa disfunção afeta uma grande número de atletas, em especial 80% dos nadadores profissionais. Nesta população a instabilidade glenohumeral quase sempre é apontada como causa primária da SIO. A proposta desta investigação foi analisar os possíveis déficits de controle motor em nadadores com Historia de Síndrome do Impacto do Ombro e Instabilidade glenoumeral, quando comparados aos nadadores normais, desta forma aprimorar as bases para um melhor diagnóstico e tratamento desta patologia. **Métodos**. Participaram deste experimento oito nadadores sem qualquer patologia ortopédica ou neurológica na articulação do ombro (NN) e oito nadadores com História de Síndrome do Impacto do Ombro e Instabilidade Glenoumeral (HSIO). Cada sujeito realizou cinco movimentos, bilateral e simultâneos, de elevação do ombro no plano escapular, "o mais rápido possível" para três diferentes distâncias (30^0, 90^0, and 150^0). Foram registradas o angulo, velocidade e aceleração dos movimentos da articulação do ombro, assim como as atividades eletromiográficas dos músculos glenoumerais (deltóide anterior, peitoral maior, grande dorsal), escapulotorácicos (serrátil anterior, trapézio superior e inferior) e os músculos da articulação do cotovelo (bíceps e tríceps). **Resultados.** O movimento angular e a velocidade aumentaram com a distância dos alvos para ambos os grupos de sujeitos. Para as três distâncias analisadas os movimentos de translação do ombro não tiveram variação entre os grupos, entretanto as translações do ombro aumentaram de forma linear conforme aumentou-se a distância dos alvos. A intensidade com que os músculos agonistas foram ativados não mudou com a distância dos alvos e foram similares para ambos os grupos de sujeitos. A quantidade das atividades musculares tanto agonistas como antagonistas aumentou com a distância dos alvos, e

os grupos não mostraram diferenças significantes segundo a ANOVA "two way". Somente com exceção do trapézio inferior cujo atividade permaneceu constante entre as duas últimas distâncias. Para as três distâncias analisadas os músculos antagonistas não modularam suas latências em ambos os grupos. Em relação a ordem de recrutamento, todos os músculos de ambos os grupos tiveram e mesmo tempo e seqüência de recrutamento. **Conclusão e Discussão**. Os nadadores com HSIO realizaram os movimentos da articulação do ombro de forma similar aos nadadores normais, em termos de amplitude angular, deslocamentos translacionais e velocidade. Portanto, contrário a alguns experimentos (Lukasiewicz, et al, 1999; Ludewig & Cook, 2000), os movimentos do úmero não afetaram os movimentos da escápula e vice-versa. Ambos os grupos de sujeitos usaram estratégias similares ao modularem as atividades agonistas e antagonistas, usando as mesmas estratégias de modulação já descritas anteriormente como "Estratégia da velocidade Insensível" (Gotllieb, et al, 1989). A partir dos nossos resultados, nós podemos concluir que a História da Síndrome do Impacto do Ombro e a presença de Instabilidade em nadadores, não altera a habilidade do sistema de controle motor, desses indivíduos, em ativar e modular os músculos glenoumerais e escapulotorácicos, ao realizarem movimentos voluntários, sem carga, o mais rápido possível.

ABSTRACT

The Shoulder Impingement Syndrome (SIS) is often observed in repetitive movements of the shoulder above 60° during the performance of occupational or athletic activities. This dysfunction affects a great number of athletes, and in special 80% of the professional swimmers. In this population the glenohumeral instability is frequently associated with this syndrome. The purpose of this investigation was to analyze possible deficits of the motor control in the subjects with History of Shoulder Impingement Syndrome and Instability when compared with Normal subjects, in this way can provide better basis for evaluation of pathology. **Methods**. Eight swimmers without any neurological or orthopedic disorder (NN) and history of pain in the shoulder, and eight swimmers with (HSIS) took part in this experiment. Each subject performed bilateral and simultaneous elevation movements of the shoulder, "as fast as possible" in the scapular plane into three different target distances (30^0, 90^0, and 150^0). The Angle, velocity and acceleration , such as EMG muscle activities of the shoulder joint were recorded. The muscles available were anterior deltoid, pectoralis major, latissimus dorsi from the glenohumeral joint and the scapulothoracic joint muscles were serratus anterior, upper and lower trapezius. The biceps and triceps long head muscles also were recorded. **Results.** The angular excursion and the angular velocity increase with distance for both groups of the subjects. The linear translational movements of the shoulder did not varied between groups for each of the three directions analyzed. However, the shoulder linear translation at each of the three direction increased with target distances. The intensity, in which each of the agonist muscle of the glenohumeral and scapularthoracic joints were activated did not change with the target distances, and were similar for both groups of subjects. The amount of muscle activities of the agonist and antagonist muscles increased with target distance, the only exception was for the amount of muscle activity

of the lower trapezius that did not increase with target distances. The two way ANOVA did not showed significant difference between two groups. The antagonist latency did not moduleted for three distances analized in the both groups. A despite of the recruitment ordem, all muscles of the two groups had the same time of the activation. **Conlusion and Discussion.** Compared with normal individuals, the swimmers with HSIS performed these movements with similar amplitude of movement and velocity and accuracy. The movements of the scapula was not affected by the movements of the humerus and vice-versa, contrary the any studies (Lukasiewicz, et al, 1999; Ludewig & Cook, 2000). Both groups of subjects used similar strategies to modulate the activities of the agonist and antagonist muscles using the same rules of modulation that were described as "Speed Insensitivity Strategy" (Gotllieb, et al, 1989).

So we can conclude that the history of shoulder impingement and/or the presence of shoulder instability did not affect the ability of the motor control system to activate and modulate the glenohumeral and scapulotoracic muscles to generate unloaded voluntary fast movements at the shoulder joint.

I- INTRODUÇÃO

Revisão anatômica do ombro

O complexo do ombro, pelas suas características anatômicas, é a articulação mais móvel encontrada no corpo humano, possibilitando uma grande amplitude de movimentos. Esses movimentos ocorrem nas quatro articulações que compõem o complexo do ombro (figura 1): 1) a glenoumeral, do tipo esferóide, onde o movimento se dá entre a cabeça do úmero e a cavidade glenóide da escápula; 2) a esternoclavicular, do tipo esferóide com disco articular, onde o movimento ocorre entre o manúbrio do esterno e a porção proximal da clavícula; 3) a acromioclavicular, também esferóide com disco articular incompleto, ocorrendo o movimento entre a porção distal da clavícula e o processo acromial da escápula e a 4) escapulotorácica que ocorre entre a face costal da escápula com a face externa do gradio costal ao nível torácico. Essa última é considerada uma articulação no sentido fisiológico e não anatômico.

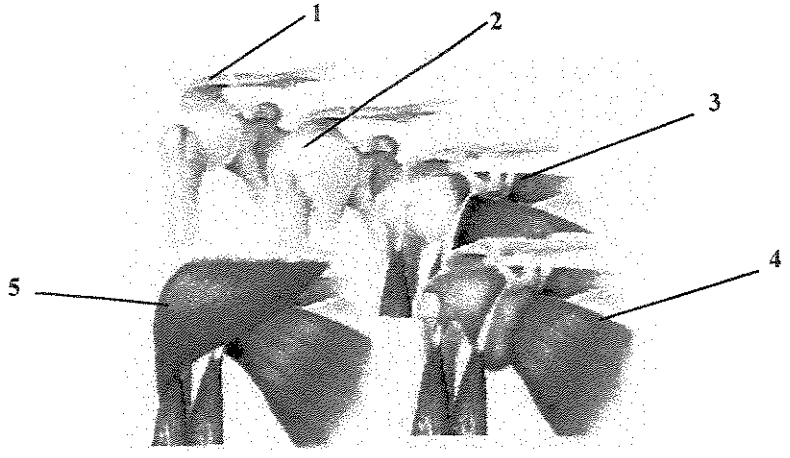

Figura 1: Representação esquemática do complexo articular do ombro, vista anterior da esquerda superior para direita: 1) articulação glenoumeral descoberta, 2) cápsula articular, ligamentos coraroacromial, coracoclavicular, 3) músculos supraespinhoso, subescapular e bíceps cabeça longa e curta (mostrando o túnel do supraespinhoso), 4) bursa subacromial e 5) músculo deltóide recobrindo todas as estruturas. (Adaptado de Netter, Frank, H. : Atlas de Anatomia Humana, 1º ed. Artes Médicas, Porto Alegre, 1996.)

Os movimentos desse complexo podem acontecer em uma das articulações ou envolver as quatro simultaneamente. A combinação coordenada desses movimentos possibilita a execução de tarefas amplas e complexas (Corso, 1995; Lephart, et al., 1997).

A articulação glenoumeral, a mais móvel deste complexo, devido à falta de restrições ósseas, é estabilizada pela cápsula articular, ligamentos glenoumerais, coracoumeral e pela atividade sincrônica dos músculos do manguito rotador e das três porções do deltóide (Biglani at al. 1996). Os ligamentos glenoumerais, destacando-se o ligamento glenoumeral ântero-inferior, servem como estabilizadores estáticos, impedido movimentos acessórios de forma excessiva nos extremos da amplitude de articular (Blasier & Goldenberg, 1992; Bigliane, et al., 1992). Já o manguito rotador, junto com o tendão bicipital cabeça longa, é um importante estabilizador dinâmico, impedindo a translação superior excessiva da cabeça umeral (Biglane at al. 1996).

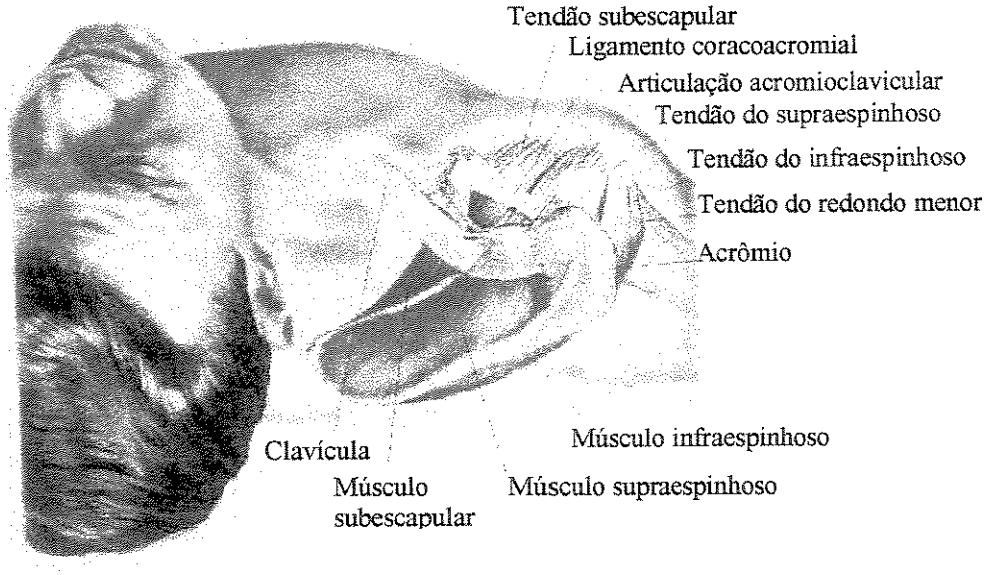

Figura 2: Vista superior do conjunto de músculos e seus tendões que fazem parte do manguito rotador. (Adaptado de Netter, Frank, H. : Atlas de Anatomia Humana, 1º ed. Artes Médicas, Porto Alegre, 1996.)

O manguito rotador é composto pelos músculos supraespinhoso, subescapular, infraespinhoso e redondo menor (figura 2).

Acima da cabeça do úmero está o arco coracoacromial (composto pelo processo coracóide, o acrômio e o ligamento coracoacromial) e o músculo deltóide. Entre a cabeça do úmero e o arco coracoacromial existe um espaço, mais precisamente um túnel, por onde passam os tendões do músculo supraespinhoso e da porção longa do bíceps. Esse último, por sua vez, é intracapsulado, inserindo-se no tubérculo supraglenoidiano, atravessando toda cabeça umeral e saindo da articulação pelo sulco bicipital. Nesse espaço, existe também uma bolsa serosa subacromial e, um pouco mais distalmente, outra bolsa chamada subdeltoídea (figura 3)

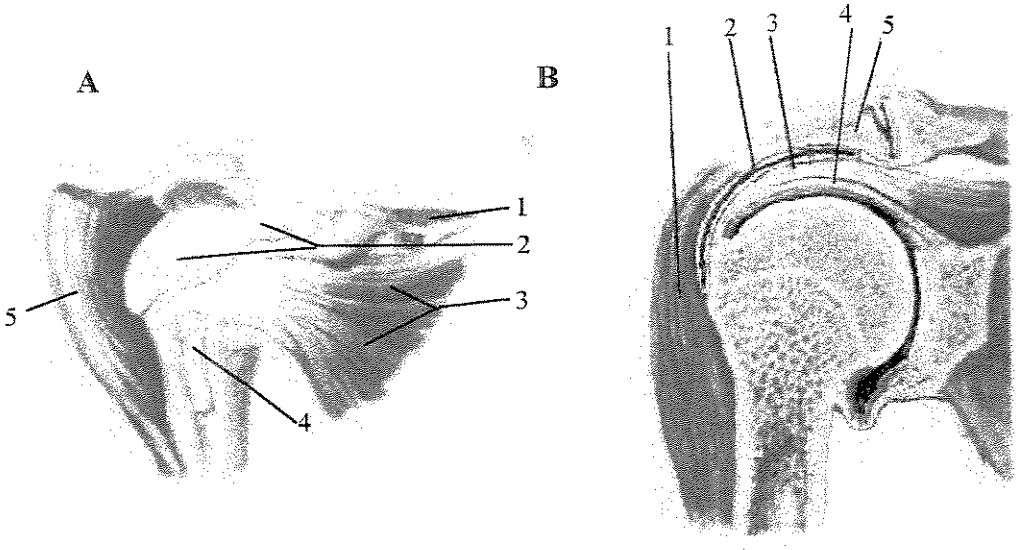

Figura 3: Figura esquemática mostrando o espaço subacromial. A- 1) músculo supraespinhoso, 2) bolsa subdeltoídia com extensão sob o acrômio, 3) músculo subescapular, 4) tendão bicipital porção longa, 5) músculo deltóide (rebatido). B - secção frontal da articulação do ombro, 1) músculo deltóide, 2) bursa subdeltoídea, 3) tendão do supraespinhoso, 4) cápsula articular, 5) acrômio. (Adaptado de Netter, Frank, H. : Atlas de Anatomia Humana, 1ª ed. Artes Médicas, Porto Alegre, 1996.)

A irrigação do ombro é feita via artéria axilar e suas comunicantes, as artérias circunflexas posterior e anterior do úmero e também pelos ramos da artéria toracoacromial. A inervação do ombro é feita pelas raízes de C5 e C6 que vão formar o plexo Braquial. Do tronco

superior do plexo braquial saem os ramos que vão inervar as estruturas do ombro (nervo axilar, subescapular e o supraescapular).

Biomecânica

Os movimentos, no complexo articular do ombro, podem ser classificados basicamente em três planos: no plano sagital, temos o movimento de extensão e flexão (45 a 180 graus), no plano coronal, a adução e abdução (-30 a 180 graus) e, no plano transversal, a rotação interna e externa (80 a 110 graus). Vários autores (Poppen & Walker, 1976; Otis, et al, 1994; Neer, 1995; Malanga, et al., 1995) têm empregado o termo elevação para indicar o movimento do braço acima da cabeça, no plano da escápula, ou seja, num plano oblíquo (ver figura 4 na página 11). Esse movimento é realizado de 30 a 40 graus anterior ao plano coronal. Nesse plano, o ombro permite maior amplitude de movimento devido a uma tensão menor na cápsula articular como também uma otimização dos músculos deltóide e supraespinhoso durante o movimento de elevação do braço (Poppen & Walker, 1976). Freqüentemente, esse é o plano mais usado para elevar o braço acima da cabeça, a partir da posição ortostática (Neer, 1995; Poppen & Walker, 1976; Otis, et al., 1994).

Os movimentos de elevação do braço, no plano escapular, estão presentes em várias atividades do cotidiano e em várias práticas desportivas. A combinação dos movimentos de elevação e rotação externa, como acontece ao pegar um objeto no alto, ocorre principalmente na articulação glenoumeral. Porém, durante a realização dessas tarefas, observamos também movimentos nas outras três articulações que compõem o complexo articular do ombro (Corso, 1995; Norkin & Levangie, 1988; Neer, 1995; Poppen & Walker, 1976; Otis, et al., 1994).

Na elevação do braço, a amplitude total de movimento é em torno de 180 graus, sendo que 120 graus desse movimento ocorrem na articulação glenoumeral (Poppen & Walker, 1976;

Otis, et al., 1994; Neer, 1995; Corso, 1995; Norkin & Levangie, 1988; Tomberlin & Saunders, 1994) e os outros 60 graus ocorrem principalmente na articulação escapulotorácica. Porém, as articulações esternoclavicular e a acromioclavicular também vão participar nesses movimentos. A articulação esternoclavicular se eleva, enquanto que a acromioclavicular roda posteriormente durante a elevação do braço (Corso, 1995; Kambar, et al., 1993). A partir da posição ortostática, os primeiros 30 graus de movimento ocorrem na articulação glenoumeral. Esse movimento é executado principalmente pela ativação dos músculos supraespinhoso e deltóide anterior (Thompson, et al., 1996; Bassett, et al., 1990; Otis, et al., 1994). Entre os 30 e 180 graus de elevação, o movimento ocorre sincronicamente entre as articulações glenoumeral e escapulotorácica, numa razão de 5:4 (Poppen & Walker, 1976), isto é, para cada 5 graus de movimento na articulação glenoumeral ocorrem 4 graus de movimento escapulotorácico. Durante a execução desse movimento, os músculos mais atuantes na articulação glenoumeral são o deltóide porção anterior, suparespinhoso e bíceps (Neer, 1995). Os músculos serrátil anterior, trapézio superior e inferior atuam na articulação escapulotorácica (Paine & voight, 1993; Ludewig &Cook, 2000;) .

Segundo James Otis e colaboradores (1994), os músculos infraespinhoso e subescapular também colaboram na elevação do braço no plano escapular. O músculo infraespinhoso é bastante ativado durante a rotação externa da cabeça umeral após os 90 graus de elevação (Neer, 1995; Poppen & Walker, 1976; Otis, et al., 1994). Essa rotação externa evita o impacto da tuberosidade maior do úmero contra o arco coracoacromial (Neer, 1995; Bigliani, et al., 1997). Os movimentos entre as articulações glenoumeral e escapulotorácica são executados de forma sinérgica. Ao mesmo tempo que o úmero se eleva na articulação glenoumeral, a articulação escapulotorácica abduz, roda para cima e inclina-se posteriormente, mantendo a cabeça do

úmero alinhada com a cavidade glenóide (Corso, 1995; Ludewig & Cook, 2000). Os músculos, que atuam nos movimentos dessa articulação, o fazem de maneira conjunta e combinada. São eles o serrátil anterior e as porções superior e inferior do trapézio (Corso, 1995; Paine & Voight, 1993; Wadsworth & Bullock-Saxton, 1997)

Síndrome do Impacto do Ombro

O complexo articular do ombro pode ser acometido por diversas disfunções, entre elas, a Síndrome do Impacto do Ombro (SIO). Essa Síndrome é caracterizada por microtraumas repetitivos nas estruturas dentro do espaço subacromial (figura 4), em particular, no tendão do músculo supraespinhoso, no tendão da cabeça longa do bíceps e na bursa subacromial (Corso, 1995).

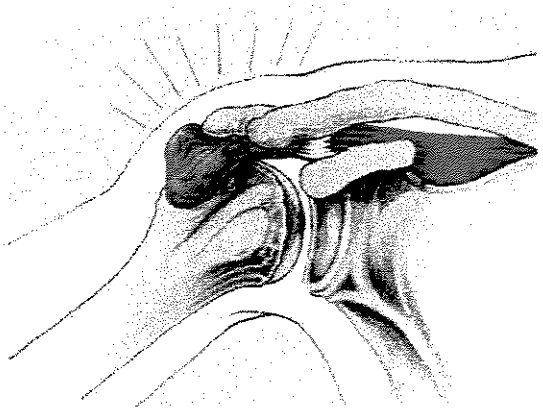

Figura 4: Mecanismo da Síndrome do Impacto do ombro, durante a elevação do braço, a bursa e o tendão do supraespinhoso são pressionados entre a tuberosidade maior do úmero e a borda acromial antero-inferior. (Adaptado de Peterson, L.;Renstron, P.: Sports Injuries: Their Prevention and Treatment, 1986)

Esses microtraumas são provocados pelo esmagamento destas estruturas contra o arco coracoacromial durante a elevação do braço (Kambar, et al., 1993; Rockwood & Lyons, 1993).

Neer descreveu três estágios desta disfunção. O estágio I é quando ocorre edema e hemorragia dentro do espaço subacromial, freqüentemente provocados por traumas e uso excessivo no trabalho ou nos esportes. Usualmente acomete indivíduos com até 25 anos de

idade. O estágio II representa a progressão dos sintomas anteriores com fibrose e espessamento das estruturas dentro do espaço subacromial, caracterizando tendinite crônica do supraespinhoso. O estágio II é mais comum entre os indivíduos de 25 a 40 anos de idade. O estágio III envolve ruptura parcial ou total dos tendões do manguito rotador e tendinite do tendão bicipital, podendo também ocorrer mudanças ósseas na borda anterior do acrômio e no tubérculo maior do úmero. Esse estágio da SIO é mais comum nos indivíduos acima de 40 anos (Neer, 1983).

Fatores estruturais e funcionais são descritos como causadores desse microtrauma. Entre os fatores estruturais, destacam-se as anomalias ósseas do acrômio (Kambar, et al., 1993; Rockwood & Lyons, 1993; Neer, 1995), degeneração e osteofitoses na articulação acromioclavicular (Neer, 1983). Entre os fatores funcionais, os mais comuns são fraquezas e desequilíbrios musculares, uso excessivo, isquemia, instabilidade glenoumeral, falta de sincronismo entre a articulação glenoumeral e escapulotorácica (Poppen & Walker, 1976; Paine,R.M. & Voight, M., 1993; Bigliane & Levine 1997; McMaster, et al, 1998; Ludewig & Cook, 2000) e entre a articulação glenoumeral e outras articulações não focais, por exemplo: a falta de flexibilidade do quadril e/ou da coluna, assim como retrações dos isquios tibiais, pode mudar o acoplamento entre os movimentos do quadril, coluna e ombro durante o arremesso. A mudança no acoplamento dos movimentos entre essas articulações pode impor um deslocamento angular excessivo na articulação do ombro, podendo acarretar sobrecarga nesta articulação que predisponha a SIO (Kibler, 1998) .

Investigações epidemiológicas revelaram que 16% a 40% dos indivíduos acima de 40 anos desenvolveram a SIO através de atividades ocupacionais nas quais o braço é mantido ou elevado freqüentemente acima dos 60 graus (Hagberg & Wegman, 1987). As disfunções da articulação do ombro afetam também um número elevado de atletas, especialmente os que

praticam esportes de sobrecarga no ombro, tais como, 66% dos nadadores de elite, 57% dos arremessadores profissionais de beisebol, 44% dos jogadores de vôlei, 29% dos arremessadores de dardo. (Johnson, 1988). Levantamentos epidemiológicos mais recentes mostram que 80% dos nadadores de alta performance apresentam disfunções na articulação do ombro (Bak, 1997; Bak & Magnusson, 1997, McMaster, et al. 1998). McMaster, et al (1998) acharam correlações significantes entre a instabilidade e a dor no ombro de nadadores.

Vários estudos têm mostrado modificações nos padrões cinemáticos e eletromiográficos nos movimentos do ombro em pessoas portadoras da SIO, incluindo nadadores.

Modificações nos padrões de ativação muscular

Estudos eletromiográficos sob a água, entre nadadores normais e nadadores com dor, têm relatado alterações da ativação muscular, em 7 de 12 músculos pesquisados (Scovazzo, et al, 1991). Mudanças nos padrões de ativação dos músculos da cintura escapular têm sido observadas em arremessadores de beisebol, com instabilidade crônica da articulação glenoumeral, quando comparados a indivíduos normais (Glousman, et al, 1988).

Durante o arremesso, os atletas tiveram um aumento na atividade eletromiográfica dos músculos bíceps e supraespinhoso, simultâneo a uma diminuição da atividade dos músculos peitoral maior, subscapular, longo dorsal e serrátil anterior. Os autores sugerem que esse aumento de atividade do bíceps e supraespinhoso seria para compensar a frouxidão dos ligamentos anteriores do ombro. A acentuada diminuição da ativação muscular do segundo grupo, principalmente na fase de engatilhar o arremesso, levando o braço em rotação externa máxima, contribuiria para a instabilidade anterior da articulação glenoumeral.

Ludewig & Cook (2000) também encontraram mudanças nos padrões eletromiográficos (EMG), durante movimentos de elevação do ombro no plano escapular, comparando indivíduos

normais a indivíduos acometidos pela SIO atribuídas ao esforço repetitivo durante a execução de trabalhos braçais. Em especial, os indivíduos portadores da SIO apresentaram um aumento das atividades EMG dos músculos trapézio superior e inferior e diminuição dessa atividade para o serrátil anterior.

Contrário a esses achados, mostramos recentemente (Santos & Almeida, 2001) que os indivíduos com História de Síndrome de Impacto no Ombro e instabilidade glenoumeral geram os mesmos padrões de EMG, em termos de intensidade e quantidade de atividade EMG, durante a execução de movimentos voluntários normais de elevação do ombro no plano escapular. Nesse estudo, foi registrada a atividade EMG dos agonistas e antagonistas da articulação glenoumeral (deltóide anterior, peitoral maior, grande dorsal), dos escapulotorácicos (serrátil anterior, trapézio superior e inferior) e dos músculos que participam da articulação do cotovelo (bíceps e tríceps). A presença de diferenças nos padrões eletromiográficos observados nesses indivíduos em outros estudos (Scovazzo, et al, 1991; Glousman, et al, 1988; Ludewig & Cook, 2000) foi atribuída ao método de registro eletromiográfico.

Ordem de Recrutamento da musculatura

Wadsworth e Bullock-Saxton (1997) investigaram a ordem de recrutamento dos músculos que movem a cintura escapular em nadadores portadores de história de Síndrome do Impacto do Ombro. Esses indivíduos realizaram a tarefa de elevação máxima do braço no plano da escápula em um tempo de 4 segundos. Os resultados foram colhidos durante os primeiros 30 graus de elevação. Nadadores sem história da Síndrome do Impacto do Ombro iniciam o recrutamento do trapézio superior 217ms antes do início do movimento. O serrátil anterior é ativado 53ms após o início do movimento, seguido 296ms pela ativação do trapézio inferior. Por outro lado, para os indivíduos portadores da Síndrome do Impacto do Ombro, o tempo de

ativação desses músculos é modificado. Respectivamente, 137, 131 e 365ms para os músculos trapézio, serrátil anterior e trapézio inferior.

Os nossos estudos (Santos & Almeida, 2001) também não suportam a observação de que indivíduos com história da SIO tenham uma modificação na ordem de recrutamento da musculatura do ombro. A latência no início do recrutamento entre os músculos glenoumerais (deltóide, peitoral maior, grande dorsal), os músculos que participam da articulação do cotovelo (bíceps e tríceps), incluindo os três músculos escapulares (serrátil anterior, trapézio superior e inferior), não excedeu a 40 milisegundos. No estudo de Wadsworth & Saxton (1997) não foram usadas informações cinemáticas acopladas às atividades musculares, como em nosso estudo, dificultando a identificação do início da atividade EMG que seja associada ao movimento. Note que a latência na ordem de recrutamento dos músculos registrados por estes autores (Wadsworth & Saxton, 1997) foi muito maior do que a latência fisiológica (Latash, et al, 1993; Almeida, et al, 1994). Em especial, Irman & Saunders (1944) demonstraram que, durante o movimento do ombro no plano da escápula, os três músculos escapulares são ativados simultaneamente, caracterizando um padrão de co-ativação.

Padrões cinemáticos

Mudanças nos padrões cinemáticos dos movimentos das articulações glenoumeral e escapulotorácica têm sido reportadas em indivíduos com SIO, por exemplo: existiria uma translação superior excessiva da cabeça umeral dentro da cavidade glenóide, quando o braço fosse elevado (Flatow, et al, 1994). Lukasiewicz (1999) e Ludewig (2000) mostraram mudanças nos movimentos da escápula durante a elevação do braço em indivíduos com SIO quando comparados aos indivíduos normais. Essas mudanças implicariam num maior contato da

tuberosidade maior do úmero contra o arco coracoacromial, predispondo a microtraumas os tecidos moles presentes nesse espaço.

Em nossos estudos (Santos & Almeida, 2001), os indivíduos com história de Síndrome do Impacto do Ombro e instabilidade glenoumeral não apresentaram qualquer mudança na cinemática durante os movimentos voluntários de elevação do ombro sem carga. Esses indivíduos executaram os movimentos do ombro com a mesma amplitude, velocidade e precisão do que os indivíduos normais. Nós acreditamos que os movimentos realizados com carga (Lukasiewicz,1999 e Ludewig & CooK, 2000) poderiam afetar a instabilidade do ombro.

Avaliação da SIO

Na maioria das vezes, os diagnósticos da SIO são estabelecidos pelo simples fato de o paciente apresentar dor na região ântero-superior do ombro. Para maior precisão da causa primária do SIO, é necessária uma investigação muito apurada. A complexidade anatômica e funcional da articulação do ombro, como a própria apresentação clínica do paciente, é muito variável, dificultando o diagnóstico (Bigliani & Levine, 1997).

Para um diagnóstico preciso da SIO, primeiramente realiza-se uma coleta da história da patologia, passando para o exame físico (inspeção, palpação, mobilidade, força e testes específicos). Os exames complementares, tais como, Radiografias, Ultra-sonografia e Ressonância Magnética, podem ajudar a confirmar e classificar o diagnóstico.

História

O sintoma mais comum é a dor, podendo vir acompanhada de fraqueza e rigidez. A dor normalmente é agravada com movimentos do braço acima da cabeça, podendo ser irradiada no braço (Clarnette & Miniace, 1998). A duração, a qualidade, os traumas e as atividades associadas à dor ajudam a esclarecer a disfunção e servem para eliminar patologias associadas (Neer,

1995). O conhecimento das atividades da vida diária (AVDs), como o tipo de trabalho, esporte que o indivíduo pratica e as atividades que envolvem movimentos do braço acima da cabeça, são essenciais para identificar os fatores predisponentes. Um destes fatores é a instabilidade glenoumeral que pode ser classificada de acordo com a direção (anterior, posterior, inferior ou multidirecional), etiologia (traumática ou não) e freqüência (aguda, recorrente ou crônica) (Clarnette & Miniace, 1998)

Exame Físico

Através da história do paciente, o clínico já poderá sugerir o diagnóstico e focar o exame físico. Como veremos abaixo, o exame físico consta de inspeção, palpação, ADM (amplitude de movimento), testes neurológicos e testes especiais para o ombro. Como é comum os problemas cervicais causarem dor na região do ombro (Palmer & Epler, 1990), é necessária uma avaliação física da mesma.

Inspeção

O ombro deve ser inspecionado a partir de três posições: anterior, posterior e perfil. Deve-se observar a presença de cicatrizes, mudanças de cor, sudorese, atrofias musculares, assimetrias e deformidades. Deformidades e assimetria anterior e superior podem indicar lesões esternoclavicular, clavicular e acromioclavicular. Mudanças no contorno da articulação glenoumeral podem indicar luxações. A escápula deve ser inspecionada para verificar a possível existência de anomalias ou posicionamentos anormais.

Palpação

A palpação deve ser focada sistematicamente nas estruturas anatômicas, particularmente nas estruturas que estão sob suspeitas de alterações. No caso da SIO, as estruturas mais comprometidas são a bursa subacromial e tendão do supraespinhoso. Essas duas estruturas são melhor palpadas quando o terapeuta estende o braço do paciente enquanto palpa, descendo distalmente e ântero-lateral ao acrômio (Palmer & Epler, 1990).

Amplitude do Movimento

Essa avaliação envolve três movimentos básicos: elevação, rotação externa e rotação interna. A elevação normal varia entre 150^0 e 170^0. A rotação externa, avaliada com o braço ao longo do corpo e cotovelo fletido a 90^0, varia entre 50^0 e 80^0. Já a rotação interna é avaliada pedindo ao paciente alcançar os processos espinhosos da coluna vertebral com o polegar, o nível considerado normal é entre T5 e T10 (Neer, 1995). A diminuição dessas amplitudes, em comparação com o lado oposto, é um sinal importante de disfunções na articulação do ombro. Nos testes de amplitude de movimento, é importante notar em qual amplitude o paciente percebe a dor; nos casos de SIO, o paciente geralmente relata a presença de dor entre 60^0 e 120^0. Por essa razão, essa amplitude é denominada como "Arco Doloroso" (Palmer & Epler, 1990).

Teste de força muscular

Os testes de força muscular devem ser executados isolando-se o máximo possível o músculo a ser avaliado. Nos casos da SIO, os músculos principais envolvidos são o supraespinhoso e o bíceps cabeça longa. O supraespinhoso é melhor isolado a 90^0 de elevação

com o braço rodado internamente (figura 7) (Jobe, et al, 1997), já o bíceps cabeça longa pode ser testado com o braço ao longo do corpo e o cotovelo fletido a 90^0 (Palmer & Epler, 1990).

Testes específicos

Existem dois testes principais na investigação da SIO, que localizam diferentes áreas de impacto.

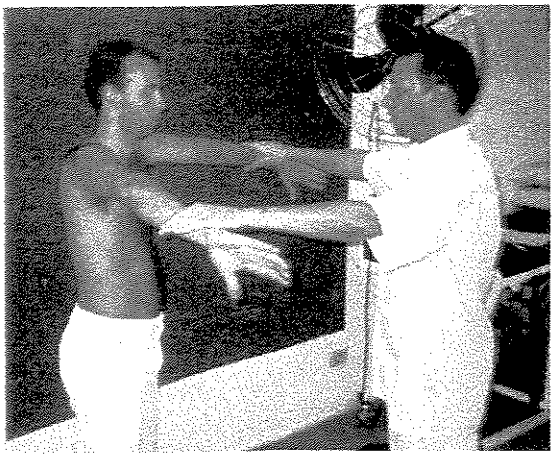

Figura 7: Representação do Teste de Jobe. O paciente mantêm o braço rodado internamente à 90^0 de elevação, enquanto o terapeuta emprega uma força para baixo sobre os seus antebraços. Essa manobra testa a força muscular e a integridade do tendão do supraespinhoso.

O primeiro deles é o teste de *Neer* que é realizado elevando-se passivamente o braço do paciente no plano escapular. Com a outra mão, o terapeuta estabiliza a escápula. O teste é positivo quando a dor normalmente é elicitada na amplitude entre 70^0 e 120^0 (figura 8A). O segundo teste é uma modificação da manobra de Neer, em que o braço é elevado passivamente até 90^0 no plano escapular. Nessa posição, mantendo o cotovelo fletido a 90^0, o braço é então rodado internamente. Esse teste é denominado de *Hawkins*, sendo positivo quando a dor é sentida pelo paciente durante esta manobra (figura 8B) (Neer, 1983; Hawkins & Bokor, 1990). A instabilidade pode ser investigada, movendo-se a cabeça umeral nas três direções das possíveis instabilidades.

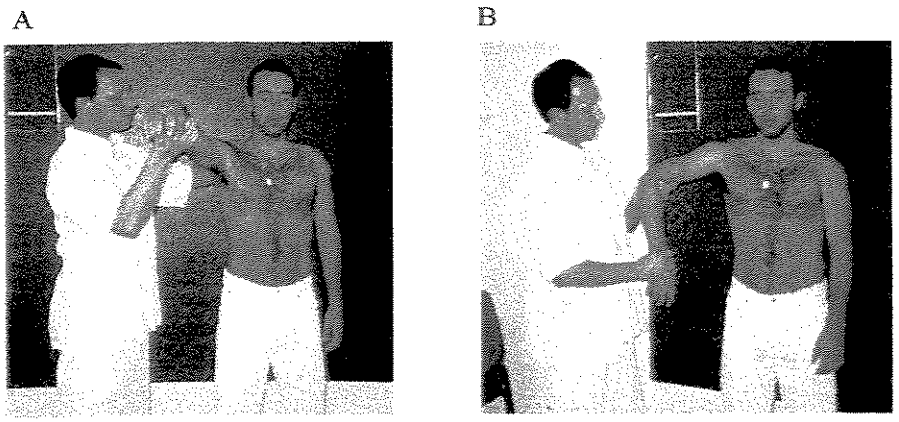

Figura 8: Representação dos testes específicos para evidenciar diferentes áreas de pinçamento da Síndrome do Impacto do Ombro. A- O terapeuta fixa a escápula e, com a outra mão, eleva o braço do paciente passivamente. B- O terapeuta abduz e roda internamente o braço do paciente passivamente. Os testes são considerados positivos quando provocam dor.

O teste de *Drawer* avalia a instabilidade ântero-posterior do ombro. Nesse teste, o terapeuta segura o braço do paciente, em posição neutra, ao longo do corpo e em posição ortostática, executando gentilmente translações nas direções anterior e posterior. A instabilidade inferior é testada com o teste de *sulcu*. Na mesma posição inicial do teste de *Drawer,* o terapeuta aplica no braço uma tração no sentido inferior. Ambos os testes são positivos quando é percebido um deslocamento da cabeça umeral dentro da cavidade glenóide. Quando a cabeça umeral é deslocada em todas as direções, a instabilidade é denominada multidirecional (Hawkins & Bokor, 1990; Neer & Foster, 1980).

Exames Complementares

As radiografias (RX) devem ser solicitadas, principalmente em lesões agudas, para descartar a possibilidade de fraturas, luxações glenoumeral ou acromioclavicular (figura 9). O RX também é necessário em dores crônicas, quando não há progressos com o tratamento conservador. As vistas Ap (ântero-posterior), axilar e do túnel do supraespinhoso podem identificar mudanças degenerativas, calcificações, tipo de acrômio e deslocamentos da cabeça umeral (Lyons & Orwin, 1998).

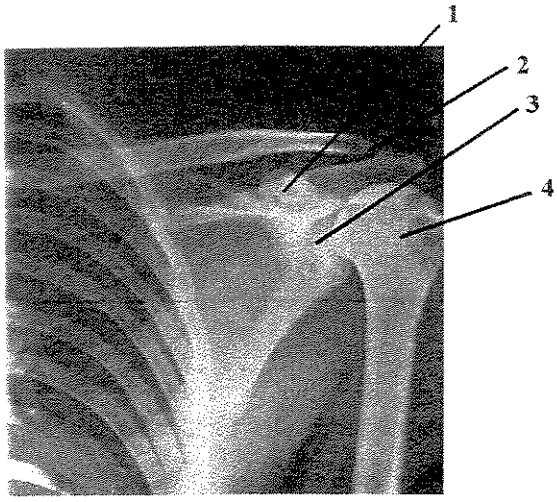

Figura 9: Representação de um RX da articulação do ombro, vista anterior: 1) processo coracóide, 2) articulação acromioclavicular, 3) articulação glenoumeral, 4) cabeça umeral. (Adaptado de Gardner, E. : Estudo Regional do Corpo Humano, 1 º ed. Guanabara Koogan, Rio de Janeiro, 1988.

Sob suspeitas de ruptura do manguito rotador, os exames por imagem do tipo Ultra-som ou Ressonância Magnética (RM) devem ser utilizados (Bigliani & Levine, 1997). A RM tem ganhado popularidade na última década e tem sido útil para detectar cistos, rupturas parciais e diferentes locais de lesão do manguito rotador. Rossi, em 1998, através da RM, classificou quatro principais áreas de lesão: 1) Subacromial anterior, 2) Borda glenoidal posterosuperior, 3) Subcoracoídea e 4) Área entre o ligamento espinoglenoidal e a fossa do infraespinhoso.

Tratamento

Os tratamentos conservadores mais comuns na Síndrome do Impacto do Ombro incluem a interrupção ou modificação das atividades físicas, uso de antinflamatórios não esteróides, injeções subacromial de esteróides e fisioterapia (Bigliane et al 1997). Se durante seis meses o tratamento conservador não for satisfatório, a descompressão cirúrgica é indicada (Norlim, 1989), embora a decisão de se fazer uma intervenção cirúrgica dependa das circunstâncias e esteja associada à individualidade de cada paciente. No entanto, a maioria dos pacientes não necessita de cirurgia para se recuperar da SIO. Em pacientes cuja causa do impacto é secundária a instabilidade, como em atletas jovens, os resultados da descompressão subacromial não são satisfatórios (Burns,et al, 1992).

Fisioterapia

Somente com um diagnóstico preciso teremos chances de sucesso em um programa de reabilitação. Nos primeiros estágios, a dor pode ser controlada, evitando-se a posição que causa dor e os movimentos no plano do arco doloroso. Outras estratégias podem ser usadas para o alívio da dor, tais como, aplicações de gelo, calor, ultra-som e estimulação elétrica (Kibler, 1996). No entanto, essas prescrições dependem da fase da patologia da SIO.

Como vimos anteriormente, a cabeça umeral é estabilizada pelos componentes estáticos (passivos) e dinâmicos (ativos). Esse mecanismo necessita de um equilíbrio entre mobilidade funcional e estabilidade (Jobe, 1995). Além desses componentes, é necessário um movimento sincrônico acoplado à articulação escapulotorácica (Poppen & Walker, 1976). Alguns protocolos enfatizavam principalmente o fortalecimento dos músculos do manguito rotador (Jobe & Bradley, 1989), outros incluem os músculos escapulotorácicos (Wilk & Andrews, 1996). Desequilíbrios entre esses músculos provocaria um distúrbio no sincronismo entre os movimentos da escápula e do úmero, predispondo à impactação do ombro. Para a realização desses trabalhos de fortalecimento, estudos eletromiográficos trouxeram grande colaboração, dando condições para a escolha das posições que melhor isolem os músculos a serem trabalhados (Malanga, et al, 1995; Kelly,et al 1996).

Os programas de tratamento, geralmente, incluem restauração da mobilidade passiva e ativa, exercícios de força e resistência enfatizando os músculos do manguito rotador e escapulotorácicos (Kambar, et al, 1993), exercícios de coordenação e propriocepção (Lephart, et al, 1997; Kibler, 1998). Esses exercícios visam, principalmente, ao restabelecimento do ritmo escapuloumeral, ao equilíbrio muscular, à estabilidade dinâmica articular e aos padrões de

sincronização neuromuscular. O comprometimento desses fatores pode ser participante da causa ou das conseqüências da SIO (Hawkins & Kennedy, 1980).

Existem vários protocolos propostos para maximizar a reabilitação do ombro (Brewster, 1993; Kambar,1993; Wilk & Andrews, 1995; Kibler, 1998). Como ilustração, mostramos, no ANEXO, o protocolo proposto por Wilk & Andrews, 1995. No entanto, achamos que esses protocolos não devem ser aplicados de forma indiscriminada. Muitas das técnicas fisioterapêuticas utilizadas nesses protocolos ainda precisam passar pelo crivo científico.

Os programas de reabilitação não são necessariamente rígidos, vão depender das causas e da fase da patologia, assim como as condições individuais de cada paciente. De maneira geral, a fase inicial enfatiza a eliminação da dor e ganho da amplitude de movimento, a segunda fase concentra-se na manutenção da amplitude completa do movimento e na recuperação da força muscular e a fase final focaliza exercícios isotônicos e isocinéticos progressivos, como também a preparação para o retorno das atividades profissionais ou desportivas (Kambar, et al, 1993).

II. OBJETIVOS

A prevalência da Síndrome do Impacto do Ombro e Instabilidade Glenoumeral é muito grande em atletas, especialmente em nadadores. O objetivo desse estudo foi ampliar os conhecimentos e o entendimento de como o sistema nervoso controla os movimentos, ditos "naturais", de elevação do ombro no plano escapular em nadadores normais. A partir desse conhecimento, compara-los aos movimentos de nadarores com História de Síndrome do Impacto no Ombro e Instabilidade Glenoumeral.

III. TRABALHO A SER SUBMETIDO Á PUBLICAÇÃO

The control of shoulder voluntary movements in swimmers with shoulder glenohumeral instability

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The control of shoulder voluntary movements in swimmers with shoulder glenohumeral instability

SUMMARY

Eight swimmers without any neurological or orthopedic disorders (NN) or history of pain in the shoulder, and eight swimmers with HSIS took part in this experiment. Each subject performed bilateral and simultaneous elevation movements of the shoulder in the scapular plane and into three different target distances (30^0, 90^0, and 150^0) "as fast as possible". The angular excursion, angular velocity and linear translational movements increased with distance for both groups of subjects. The activities of the agonist and antagonist muscles did not show significant difference between the two groups. Compared with normal individuals, the swimmers with HSIS performed these movements with similar amplitude of movement, velocity and accuracy. Both groups used similar strategies to modulate the activities of the agonist and antagonist muscles by applying the same modulation rules described as "Speed Insensitivity Strategy" and the recruitment order for all muscles of the two groups had the same time of activation. The history of shoulder impingement and/or the presence of shoulder instability did not affect the ability of the motor control system to activate and modulate the shoulder muscles in order to generate unloaded voluntary fast movements at the shoulder joint.

Key words: Motor control, shoulder, swimmers and Impingement Syndrome.

INTRODUCTION

Shoulder dysfunction is often observed in repetitive movements of the shoulder above 60° during the performance of occupational or athletic activities (Hagberg & Wegman, 1987). This dysfunction affects a great number of athletes, and in particular, 80% of professional swimmers (Bak & Faun□, 1997). Nevertheless, there is no clear explanation for the cause of the shoulder impingement syndrome. Anatomic abnormalities of the coroacromial arch or humeral head, overload, ischemia, degeneration of the rotator cuff tendons, decreased muscle activity, abnormalities of the movements at the glenohumeral and scapulothoracic joints and glenohumeral instability are of the causes associated with this syndrome (Poppen & Walker, 1976; Jobe & Bradley, 1989; Kambar, et al., 1993; Rockwood & Lyons, 1993, McMaster, Roberts & Stoddard, 1998; Lukasiewicz, et al, 1999; Ludewig & Cook, 2000).

There are several changes in the movements of the glenohumeral and scapulo-thoracic joints in individuals with shoulder impingement. Upper-arm elevation associated with excessive superior or anterior translation of the humeral head on the glenoid fossa, inadequate external rotation of the humerus, decrease in the normal scapular upward rotation, and posterior tipping of the scapula on the torax are some of these abnormal movements (Poppen & Walker, 1976; Kambar, 1993 Paine & Voight, 1993: Ludewig, 2000).

Changes in the pattern of muscle activities have also been reported in several other studies on swimmers with shoulder impingement (Scovazzo, at al, 1997, Wadsworth & Bullock-Saxton, 1997; Klaus, 1997; Ludewig, 2000) and baseball players with shoulder instability. Glousman (1988) recorded the shoulder muscle activities under water of swimmers without and with pain due to the shoulder impingement syndrome. These authors reported significant group differences in the pattern of muscles activities of the anterior and middle deltoid, infraspinatus, subscapularis,

upper trapezius, rhomboids, and the serratus anterior during freestyle stroke. However, the muscles firing pattern of the posterior deltoid, supraspinatus, teres minor, pectoralis major and latissimus dorsi was similar in both groups. Wadsworth and Bullock-Saxton (1997) also asked freestyle swimmers with history of shoulder impingement syndrome and subacromial impingment to elevate the shoulder at the scapular plane outside water. These individuals displayed increased variability and delay in the time of recruitment of the upper and lower trapezius and serratus anterior.

Some authors tried to associate these kinematic changes in the shoulder movement with the changes in the pattern of muscle activities in individuals with shoulder dysfunctions (Lukasiewicz, et al, 1999; Ludewig, 2000). For instance, during free style, the swimmers with painful shoulder entered the hand further away from the middle line, with the humerus lower to the water. This pattern, also called dropped elbow, was associated with decreased the muscle activity of the anterior deltoid. Others showed that the change in the motion of the scapula during arm elevation is caused by a decrease in the muscle activity of the serratus anterior (Ludewig, 2000) and deficient synergy between the muscle activities of the upper and lower trapezius (Kambar, 1993; Paine & Voight, 1993).

The interpretation of these kinematic and electromiographic findings is limited by three factors. First, a variety of movement disorders could be associated with shoulder impingement. For instance, subacromial impingement has been often diagnosed with painful shoulder. Nevertheless, shoulder impingement is not the only cause of pain in the antero-superior aspect of the shoulder (Bigliani, 1997). Second, we cannot partial-out the effect due to joint instability to the effect due to pain. Third, generally these studies quantified the shoulder and/or the scapula muscle activities at static position or during a phase of complex athletic activities (i.e, freestyle

swim) (Glousman, 1988; Scovazzo, at al, 1991, Ludewig, 1996;). An important exception is the recent study of Ludewig (2000).

Despite the possible cause for the changes in muscle activities and in the glenohumeral and scaputhoracic movements, there is an accepted common mechanism that would cause shoulder impingement. As the upper-arm moves overhead it brings the great tuberosity in closer contact with the coracromial arch (Flatow, et al, 1994), decreasing its space and exposing the rotator cuff tendons (Neer, CS Jr 1983, Zuquerman at al 1992). Some authors believe that shoulder instability would hinder or prevent the proper synergy between the scapulae and the upper-arm movements, generating additional stress and micro-traumas of the rotator cuff tendons (Bak & Fauno, 1997; Bak & Magnusson, 1997; McMaster, et al, 1998). These micro-traumas could also cause tendinitis of the supra-spinatus muscle and non-specific shoulder pain.

Here, we tested the hypothesis that shoulder instability per se would change the muscle activation pattern of the glenohumeral and scapulothoracic muscles. These hypothesis was based on the evidence described above which show the changes in the pattern of muscle activities in individuals with shoulder instability (Glousman, 1988; Bak & Fauno, 1997). We used the simple rules identified as the "Speed Insensitivity Strategy" (Corcos, et al, 1989; Gottlieb, et al, 1989a, b) to test the changes and modification in the modulation of the glenohumeral and scapulothoracic muscles with an increase in movement distance. The rules are based on the modulation of the amount of EMG burst of muscle activity and of the antagonist latency with movement distance. These rules were observed for constrained elbow (Gottlieb, et al, 1996) and unconstrained voluntary shoulder movements (Almeida, at al, 1995).

Contrarily to our initial hypothesis, we showed that the motor control system is functionally intact in swimmers with a history of shoulder impingement and shoulder instability,

but without pain during the test. In other words, compared with the general population, these individuals use the same motor control strategy to generate voluntary overhead shoulder movement at the scapula plane. By selecting only individuals with shoulder instability and with a history of shoulder impingement, but without a painful shoulder, we avoid the diagnosis problem in studies on the shoulder. By limiting individuals with shoulder instability to swimmers, we avoid the effect of occupational exposure on the diagnosis activities (Hagberg at al 1987, Rosecrance at al 1994). We also chose swimmers because shoulder instability is the most common cause associated with shoulder impingement in this population (Bak & Fauno, 1997).

OBJECTIVES

The prevalence of the Shoulder Impingement Syndrome and Glenohumeral Instability is high among athletes, especially in swimmers. This study aimed at improving the knowledge and understanding of how the nervous system controls natural movements of shoulder elevation in the scapular plane in normal swimmers. Once this knowledge is established, it will be compared with that on the movements of swimmers with a History of the Shoulder impingement Syndrome and Glenohumeral Instability.

METHODS

Subjects

Eight swimmers without any neurological or orthopedic disorders (NN) or a history of pain in the shoulder, and eight swimmers with a history of the Shoulder Impingement Syndrome (HSIS) took part in this experiment after giving their formal consent and the experiment had been approved by the State University of Campinas. Four males and four females composed each group. The average age was 20.87 (SE=0.58) and 20.62 (SE=0.75), respectively for the NN and the HSIS group.

All swimmers with HSIS underwent clinical examination and were interviewed by the same physician at the State University of Campinas, using a protocol defined by Klaus Bak (1997). In table 1, we showed the assessment for coracoacromial impingement, glenohumeral instability, general joint hypermobility, and the presence of a history of shoulder impingement for each subject in the HSIS group. The history of shoulder impingement was assessed by interview. The subject was considered as having a history of shoulder impingement if she/he referred to a history of pain localized in the proximal anterolateral region of the shoulder and to pain during elevation or abduction of the upper-limb overhead (from 60° to 120° of upper-limb elevation, which characterizes the painful arch of movement). All HSIS subjects referred to feeling this pain several times during the last six months or even for a longer period of time.

Insert here Table I

Shoulder coracoacromial impingement was assessed by the Neer's and Hawkins's tests (Neer CS II, 1983; Hawkins & Bokor, 1990). For the Neer's test, the examiner forcefully elevated the shoulder into flexion, causing a "jamming" of the greater tuberosity against the inferior acromion. For the Hawkins' test the shoulder and elbow were passively flexed to 90^0 and then forcefully rotated internally. No HSIS subjects reported any pain during these two tests and were, therefore, considered as not presenting "impingement signs" during this study even though they had reported a history of shoulder impingement in the past.

The antero-posterior glenohumeral instability was assessed by the Drawer's test as described by Hawkins and Bokor (1990). This test was performed by holding the upper-arm at the orthostatic position and then gently translating the humeral head passively into the anterior and posterior directions. Inferior instability was tested with the sulcus test (Neer & Foster, 1980) by applying a downward traction on the arm at neutrally rotated position. General joint

hypermobility was evaluated using the criteria described by Carter and Wilkinson (Carter & Wilkinson, 1964). The person was classified as having general hypermobility if at least three out of five assessed joints (elbow, knee, thumb, fingers and ankle) showed it.

No subjects in the HSIS group had i) a history of traumatic injury or surgery in the shoulder, or ii) pain during examination. However, all HSIS subjects presented at least one type of glenohumeral instability. It must be noted that all the subjects in the HSIS group had at least one kind of glenohumeral instability, as measured by the antero-posterior and inferior instability. Six subjects showed antero-posterior and inferior instability, one subject showed only antero-posterior instability and one presented only inferior instability. (Table I). Nevertheless, none of the subjects in this group showed a sign of impingement and three out of eight subjects presented hypermobility of the joints (Table I).

Tasks

Each subject performed bilateral and simultaneous elevation of both shoulders in the scapular plane. At the initial position, the subjects stood upright at the orthostatic position with their arms hanging relaxed at the side of their bodies (figure 1). The scapular plane was defined at 30 degrees in front of the coronal plane of the subjects' body. This scapular plane was measured with a manual goniometer, as the angle between the line crossing the humerus and the line crossing both shoulders, with the shoulder kept at 90^0 of abduction, the elbow full extended and the wrist at the neutral position. Keeping the upper-arm at this position, the experimenter put a circular metal bar (2.2 meters in length by 2 centimeters in radius) in the vertical position, in such a way that the hand would move in front of and close to it (figure 1). In other words, the vertical bar guided shoulder movement in the scapular plane. There was one bar on each side of the subject.

Using a manual goniometer, we measured the target distance (30^0, 90^0, and 150^0), as the angle between the humerus and the medial line of the body. Each target distance was set by asking the subjects to move the upper limb into the scapular plane, with the elbow completely extended and the wrist at the neutral position. At each target distance, we fixed a small piece of cotton on the bar a small that could easily bend when touched by the hand. The subject performed elevation movements of the shoulder into three different target distances (30^0, 90^0, and 150^0). The subjects were instructed to move "as fast as possible" keeping the elbow extended. Five movement trials were recorded for each experimental condition, for two seconds each.

Insert figure 1

Recording and processing of EMG data

The EMG muscle activities were recorded from the glenohumeral joint muscles (anterior deltoid, pectoralis major, latissimus dorsi) and the scapulothoracic joint muscles (serratus anterior, upper and lower trapezius). The biceps and triceps long head muscles were also recorded. Surface electrodes were used to record muscle activities, which were aligned in the direction of the muscle fibers. These activities were recorded using DelSYS (model DE2.2L) EMG amplifiers with the total gain of 2000 and frequency of 20-450Hz. All EMG data were digitalized at 1,000 frames/second using Optotrak software and a synchronization unit. These EMG data were rectified and filtered using a 20ms moving-size window. The processed EMG was used for quantification purposes.

Recording and processing of kinematic data

The LEDs (light-emitting diode) were fixed in the anterior superior iliac spine (ASIS) and center of the shoulder, elbow and wrist joints. The X (medial-lateral), Y (cephalic-caudal) and Z (anterior-posterior) coordinates of these LEDs were recorded using Optotrak Motion Analysis

System 3020, at 200 frames per second. The translation of the shoulder was obtained using the coordinates of the shoulder's LED. From LED marks, we obtained the angle of the shoulder and elbow joints. From the shoulder angle, we calculated its velocity and acceleration. Angle, velocity and acceleration were smoothed using a 5 ms moving-size window.

Quantification

The angle, velocity, acceleration of the shoulder joint, and the EMG activity for the eight recorded muscles for each trial were plotted on a monitor screen. The initial and final angle were visually identified to obtain the total angular excursion. We also identified the maximum peak velocity of the shoulder. The onset of the agonist (anterior deltoid, biceps, upper and lower trapezius and serratus anterior) and antagonist (latissimus dorsi, pectoralis major and triceps) muscle activities were taken from the point of the first sustained rise above the baseline. The agonist EMG activity was integrated during 30 milliseconds from its onset. This activity was used to identify the intensity in which the muscle was activated (Gottlieb, et al, 1989). The agonist EMG activities were also integrated from their onset until the time the movement acceleration reversed direction for the first time (during the time of the peak velocity). The antagonist EMG activities were also integrated from their onset until the time the acceleration reversed direction for the second time (approximately at the end of movement). The integrated values of the agonist and antagonist muscles, based on the acceleration profiles, were used to identify the amount of muscles activity observed during the movement. Each of the integrated EMG values cited above was normalized by dividing it by its correspondent baseline value. The baseline of each muscle was calculated by integrating the EMG activities during the first recorded 100 milliseconds. The time of the recruitment order for each muscle was defined as the difference between the time of the beginning of the acceleration and the time of the onset of

muscle activity.

Data analysis

A two-way ANOVA was used to test the effect of group, distance and interaction on the kinematics and EMG-dependent variables studied. A multiple t- test was used to find out the difference between recruitment orders of all the muscles.

RESULTS

All the subjects in both groups were able to performe the tasks very well. They elevated the shoulder joint "as fast as possible" into the scapular plane (30 degrees in front of the coronal plane, see figure 1). Figure 2 depicts the angular excursion and velocity, and the electromyographic (EMG) muscle activities for the shoulder movements performed over three angular target distances. The data are presented for one normal subject (NN, figure 2a) and for one subject with a History of the Shoulder Impingement Syndrome (HSIS, figure 2b) and illustrate how the task was performed. The angular excursion and the angular velocity increased with distance for both subjects and the velocities were characterized by bell-shape profiles.

EMG activities are presented for the muscles of the glenohumeral (anterior deltoide, pectoralis major, and latissimus dorsi), the scapularthoracic (upper and lower trapezius, and anterior serratus) and the elbow (biceps long head and triceps) joints. The agonist EMG muscle activities of the glenohumeral (deltoide and biceps[1]) and the scapularthoracic (upper and lower trapezius, serratus anterior) muscles initially risen at the same slope for all three target distances. The number of EMG activities of these agonist muscles increased with target distances.

The antagonist muscles of the glenohumeral (pectoralis major, and latissimus dorsi) were activated around 40 millisecond after the beginning of the agonist muscle onset. The latency

[1] The biceps long head is a bi-articular muscle acting at the flexion of the elbow

between the onset of the agonist and that of antagonist muscles is called antagonist latency. The antagonist latency between the onset of the antagonist glenohumeral muscles and that of the agonists of the glenohumeral and the scapularthoracic muscles did not change with target distances. However, the antagonist latency between the biceps and triceps muscles was prolonged for longer target distance. The recruitment order of all agonist muscles of the three joints did not vary for either subject. The upper trapezius was the first muscle to be activated, followed by the anterior deltoid, anterior serratius and lower trapezius. However, the latency between these agonist muscles was less than 20 milliseconds.

The agonist activities of the glenohumeral and the scapularthoracic muscles were characterized by two bursts of EMG activities. The first agonist bursts accelerated the limb against gravity towards the target using concentric contractions. These first agonist bursts ended abruptly around the time that peak velocity occurred and were salient for a short period of time. After this short period, there was a second occurrence of agonist bursts. They sustained the limb at the target position against gravity, using isometric contractions. The antagonist burst of the glenohumeral muscles (pectoralis major, and latissimus dorsi) generally started around 50 milliseconds after the onset of the first agonist burst. The antagonist muscles were activated excentrically and were responsible for the deacceleration of the limb at the target position.

Note that the duration of the second agonist burst of the lower trapezius and the duration of the antagonist burst of the pectoralis were different between thetwo groups of subjects. The normal subjects "turned off" the antagonist pectoralis major abruptly before the end of the movement, and the agonist lower trapezius around the middle of the movement excursion. However, the HSIS subjects prolonged the activation of these two muscles beyond the end of the movement.

Insert here figure 2

Angular excursion and angular velocity of the shoulder

Figure 3 depicts the angular excursion and the velocity of the shoulder elevation movements over the three target distances for the two groups of subjects. One could think that all subjects tended to undershoot the target distances, and the amount of this undershooting was larger for the longer distances. However, this is not necessarily the case. First, in figure 3a, the real angular excursion is shown. This is the final minus the initial position, and the initial position was not zero. Second, there was a considerable amount of shoulder translation which is shown below (figure 4). This translation affected the position of the LED mark on the shoulder joint and therefore the measured shoulder angle was smaller than the one measured with the manual goniometer used to set the target. Notice that the difference between the measured and the required movement excursion increased with target distance. This is because shoulder translation was larger for the longer target distance (figure 4).

The results of a two-way ANOVA did not revealed major effects for the group of subjects (NN versus HSIS) for the angular excursion and movement velocity (see table II). However, both angular excursion and peak velocity increased with target distance, and there was no interaction between the effect of group and distance for these two variables.

Insert here figure 3

Translational movements of the shoulder

The linear translational movements of the shoulder on the X, Y and Z axes correspond respectively to the movements of the medial translation (A), cephalic translation (B) and the posterior translation (C) (see figure 4). The results of the two-way ANOVA showed that the linear translation of the shoulder did not vary between groups for each of the three directions

analyzed (see Table II). However, the shoulder linear translation in each of the three directions increased with target distances. The interaction between the group and the target distance was not significant for each of the three directions analyzed (Table 1). Note that the translation of the shoulder at the medial and cephalic direction was around 10 centimeters for the larger target distance.

Insert here figure 4

Pattern of modulation of the electromyographic activities

Figure 5 depicts the intensity of activation of the agonist muscles of the glenohumeral and scapularthoracic joints. The two-way ANOVA showed that the intensity, in which each of the agonist the glenohumeral (anterior deltoide, pectoralis major and biceps long head) and scapularthoracic (upper and lower trapezius, anterior serratus) muscles was activated did not change with the target distances and were similar for both groups of subjects. Also, there was no interaction between group and target distance for each of the muscles analyzed (Table II). The lack of modulation of the intensity of the agonist muscles with target distance can also be observed in figure 2. As pointed before, the first agonist bursts of these muscles rose at the same slope for all three target distances for the two subjects.

Insert here figure 5

Quantity of agonist activity

Both groups of subjects performed all tasks using a similar number of EMG muscle activities of the first agonist and antagonist bursts (figure 6). As revealed by the two-way ANOVA there was no significant group effect for the amount of muscle activity of each agonist and antagonist muscle of the glenohumeral joint, scapularthoracic and elbow joints. The amount of muscle activity of the anterior deltoid, pectoralis major, latissimus dorsi, biceps long head,

upper trapezius, serratus anterior, and triceps increased with target distance (see ANOVA results in Table II and figure 6). The only exception was for the amount of muscle activity of the lower trapezius, which did not increase with target distances, as revealed by the ANOVA test. The interaction between group and target distance was not significant for all the eight muscles analyzed (table II).

Insert here figure 6

Antagonist latency and order of recruitment of all muscles

The antagonist latency between the onset of the first burst of the glenohumeral, scapularthoracic and the onset of the antagonist latissimus dorsi did not increase with target distances (see table III). The ANOVAs did not show any group difference for these antagonist latencies either and nor were the interactions between group and target distances significant for these antagonist latencies.

Figure 7 depicts the recruitment order of all muscles in relation to the onset of the movement measured based on the acceleration profiles (see method). The first muscles to be activated were the upper trapezius (UT) and anterior deltoid (AD), followed by the lower trapezius (LT), serratus anterior (SER) and biceps (BIC). These muscles are agonists and act on the glenohumeral, scapularthoracic and elbow joints and were activated between 20 to 40 milliseconds before the beginning of the movement. A multi student t-test showed that UT and AD were activated at similar times, which was around 40 milliseconds before the onset of the movement. This test also showed that LT, SER and BIC were activated around 30 milliseconds before the movement onset. The time of activation of these three muscles is statistically different from the time of activation of UT and AD. The antagonist muscles latissimus dorsi (LD), pectoralis major (PEC) and triceps (TRIC) were initially activated at the time of the beginning of

the movement, around 40 milliseconds after the onset of the UT and DA agonist muscles. A multi student t-test also showed that the onset did not differ among these antagonist muscles and that these antagonist latencies were different in comparison with the onset of all six agonist muscles.

DISCUSSION

There is no group difference in terms of movement performance

Contrarily to our initial hypothesis, individuals with a history of shoulder impingement and with shoulder instability did not present any functional deficit in their motor mechanism during the performance of overhead fast voluntary bi-lateral movements of the shoulder joints in the scapula plane. Compared with normal individuals, they performed these movements with similar amplitude of movement and velocity and accuracy (figure 3).

Why did we fail to observe any change in the movement kinematic of the shoulder in individuals with a history of shoulder impingement and with shoulder instability? Recently, Ludewig and Cook (2000) showed a coupling between the movements of the humerus and the scapula for individuals without shoulder impingement. Especially as the humerus moved into lateral elevation, the scapula moved into upward rotation and as the humerus moved into lateral rotation, the scapula moved into a tipped posterior position (see figure 4 in this study). This coupling between the scapula and humerus movements prevents the contact of the great tuberosity with the coracromial arch (Flatow, et al, 1994). On the other hand, Ludewig and Cook (2000) showed that individuals with shoulder impingement change the coupling between the scapula and the humerus movements. These individuals have more anterior tipping of the scapula at maximum shoulder elevation with a decrease of the coracromial arch, which exposes the rotator cuff tendons to additional stress (Neer, CS II 1983, Zuquerman at al 1992).

In this our study, the scapula movements or the rotation of the humerus were not recorded. However, we could assume that the movements of the scapula would be affected by the movements of the humerus and vice-versa. Therefore, the translation of the LED mark placed on the center of the shoulder joint (figure 4) would be affected by the movement of the scapula. We can assume this association between the movement of the scapula and the shoulder translation for two reasons. First, the sensors fixed to pins embedded in the underlying bones or placed at the skin surface give similar measure of the scapula displacement (cf. AR Karduna and collegues, umpublished research, 1999). Second, as shown above, the movements of the scapula and humerus are coupled (Ludewig and Cook, 2000).

In figure 4C, it was shown that the shoulder joint of both groups of individuals moved into posterior direction at the three amplitude of movements and the amount of this posterior translation was similar in both groups. In other words, shoulder instability probably did not affect the movement of the tipping of the scapula. Similar reasoning could be used to analyze the group difference in terms of scapular medial rotation and upward rotation and the humeral rotation observed in individuals with shoulder impingement.

In summary, the subjects in both groups performed the shoulder movements, at the scapula plane with similar shoulder translation at the medial, cephalic and posterior direction (figure 4). Shoulder translation increased with the amplitude of the movements for both group of individuals. Because the shoulder voluntary movements were very similar among the individuals with and without shoulder instability, one would also expect to observe a similar pattern of muscle activities in both groups, and this is exactly what we reported. Both groups of subjects used similar strategies to modulate the activities of the agonist and antagonist muscles. They kept the intensity in which the agonist muscles (AD, UT, LT, SER and BIC) were activated constant

over different target distances and prolonged the number of activities of these muscles for longer distances (figure 5).

These simple rules of modulation of the agonist EMG bursts have been observed for other muscles during elbow (Almeida, et al, 1994; Almeida, et al, 1995, Corcos, et al, 1995) and shoulder voluntary movements (Gotlieb, et al, 1997) and were described as "Speed Insensitivity Strategy" (Gotllieb, et al, 1989). Our data extended this observation by showing that this pattern of modulation of the agonist muscles could also be observed for the muscles of the glenohumeral (anterior deltoide), the scapularthoracic (upper and lower trapezius, and anterior serratus) muscles. The increase in the amount of muscle activity with the increase in the shoulder angular position was also observed for the upper trapezius, levator scapulae and serratus anterior for normal individuals performing isometric maximum voluntary contraction at the scapulae plane (Ludwing, et al, 1996). The only exception was for the muscle activity of the lower trapezius, which did not increase with movement distance. These findings are in agreement with our data. Note that as shown in Table II the lower trapezius was the only muscle that did not have its activity modulated with an increase in the angular excursion of the shoulder joint. However, we have to keep in mind that the type of muscle contraction was different in the two studies. In our study the movement was voluntary and the muscles were contracted isotonically, whereas in Ludwing 's study (1996), there was no movement and the muscles contracted isometricaly.

So we can conclude that the history of shoulder impingement and/or the presence of shoulder instability did not affect the ability of the motor control system to activate and modulate the glenohumeral and scapulotoracic muscles to generate unloaded voluntary fast movements at the shoulder joint.

Joint instability does not seem to affect the control of the unloaded voluntary shoulder

movements

It is difficult to compare the results of our experiment with literature data because the muscles recorded, the type of tasks, and the characterization of the groups in terms of occupation and history of shoulder impingement varied across the experiments (Glousman, 1988; Scovazzo, at al, 1991; Wadsworth & Bullock-Saxton, 1997; Ludewig and Cook, 2000). Different tasks imposed different mechanical demands to the motor control system. For example, Glousman (1988) compared the performance of athletes skilled in throwing while pitching a baseball with and without a history of chronic anterior shoulder instability. As compared with the normal group, the latter of individuals showed decreased muscle activities of the pectoralis major during the late cocking phase and decreased muscle activities of the latissimus dorsi during the acceleration and follow-through phases (the end of the pitching). These authors associate the decreased activities of these muscles to the instability of the shoulder joint. In our study, we failed to observe group differences for all the muscles studied, including the pectoralis and the latissimus dorsi, even though our group presented shoulder instabiltiy.

It has been shown that the stimulation of the afferent anterior and the infeiror axillary articular nerves elicited EMG activity in the biceps and in the rotator cuff muscles in cats (Guanche, et al, 1995). Let us assume that joint stability will generate less intra-capsular pressure in the shoulder joint. Based on Guanche's study, one could expect to observe less EMG activity in the shoulder muscles, as reported for the pectoralis major and the latissimus dorsi during pitching (Glousman, 1988). On the other hand, Glousman (1988) also observed an increase in the EMG activities of the supraspinatus and biceps muscles. So, it is possible that pitching generated excessive muscle torque at the shoulder joint, which compromised the joint stability to a level that would affect the generation and the modulation of the EMG activities of these muscles.

However, we do not know how shoulder instability could, at the same time, enhance the activation of some shoulder muscles and inhibit the activation of others. Based on our experiment, joint stability does not seem to affect the reflex mechanism connecting the afferents from the shoulder capsule to the alfa motor neuron pool innervating the shoulder muscles.

As in our experiment, Ludwing (2000) also asked subjects to perform voluntary shoulder movement of elevation at the scapula plane. The performance of normal individuals was compared with the performance of individuals that were routinely exposed to overhead work and had impingement symptoms. Compared with normal individuals, the individuals with the Shoulder Impingement Syndrome showed a decreased upward rotation of the scapula at the completion of the first phase (60° of humeral elevation) and a more anterior tipped position at the end of the third phase (120° of humeral elevation) and increased medial rotation under load condition. These changes in the scapula movement was associated with a decreased muscle activity in the serratus anterior. These individuals also showed increased EMG activities of the upper and lower trapezius. Our results do not support these findings, since we did not observe group differences for the EMG activities of these muscles.

Several studies showing kinematic changes in the movements of the scapula and humeus in individuals with shoulder disfunctions used static or slow movements (Lukasiewicz, et al, 1999; Ludewig & Cook, 2000). In our experiment the movements were performed as fast as possible (around 0.6 seconds) (figure 2), whereas in the other studies the shoulder movements lasted 4 seconds (Wadsworth & Bullock-Saxton, 1997; Ludewig and Cook, 2000). A faster movement would charge the motor control system more in terms of mechanical demands than a slow one and, as a result, muscle activities are larger for fast movements. In this sense, the signals of the EMG activities for fast movements have better resolution because the relation of

the signal to noise is increased (De Luca, 1972). A fast movement would also charge the shoulder joint more in terms of stability than a slow one. So, based on our experiment, we could rule out the possibility that the changes in the muscle activities of some shoulder muscles could be attributed to increased shoulder joint instability if the movement is performed with load.

In normal individuals, it was shown that for each 30° of shoulder movement, there was less than 1.5 millimeters of displacement of the humeral ball on the face of the glenoid (Poppen & Walker, 1976; Bigliani, et al, 1996.). These authors also showed that these accessory movements would increase in individuals with shoulder instability. We did not measure the displacement of the humeral ball on the face of the glenoid in our subjects. However, we could assume based on this literature that this displacement was larger for the group with shoulder instability as compared with the normal group. So, our study did not support the belief that the incongruente movements of the humeral head in the glenoid fossa would be detrimental to voluntary movement of the shoulder joint (Moseley, 1962; Neer & Foster, 1980; Bowen, et al, 1991; Bigliani, 1996).

We should keep in mind that, in individuals with shoulder instability, these incongruent movements between the humeral head and the glenoid fossa are less than 3 millimiters (Bigliani, et al, 1996). In our study the individuals performed the movements without any additional load on the upper-arm. The additional load could increase the number of these incongruent movements. Several studies have also shown that the kinematic and eletromyographic differences between the movements of the normal individuals and individuals with the Shoulder Impingement Syndrome increased with the load of the upper-arm (McQuade & Smidt, 1998; Michaels, 1995; Ludewig, 2000). Taken together, these findings could show that joint instability would affect the performance of voluntary movements at the shoulder only if the limb is loaded

with additional weight. We are planning to test this hypothesis in the future.

The recruitment order of the scapulothoracic muscles

The recruitment order of the scapulothoracic muscles was investigated in swimmers with Subacromial Impingement (Wadsworth & Saxton, 1997). The shoulder muscles were recruited in the same sequence as the upper trapezius, to the serratus anterior and finally the lower trapezius for individuals with and without subacromial impingement. The upper trapezius was activated 217 ms prior to the onset of the movement for the normal individuals and 137 ms for the individual with subacromial impingement. The serratus anterior was activated after the onset of the shoulder movement, respectively 53 and 131 ms for the normal individuals and for those with subacromial impingement. The lower trapezius was activated 319 ms also after the onset of the movement for the normal individuals and 496 ms for thosel with subacromial impingement. In our experiment, the onset of all muscles recorded, including the upper and lower trapezius and the serratus anterior, did not exceded 40 ms after the onset of the movement, which is far below the values reported by Wadsworth & Saxton, 1997.

The kinematic of the shoulder movements was not recorded in this experiment and the onset of muscle activity was not visually identified using this kinematic information, as we did in our experiment. In the study of Wadsworth & Saxton, 1997 the onset was defined as the time in which the muscles first achieve 5%, above the baseline, of its maximum amplitude. We believe that this procedure is not adequate to identify the onset of muscle activities since it yielded excessively large values to make any physiological sense. Also, these values cannot explain the coupling between the movements at the glenohumeral and the scapulo-thoracic joints (Ludewig & Cook, 2000), which would require a shorter time interval between the activation of these muscles even for the normal individuals. Also, there is no physiological or biomechanical need

for the upper trapezius to be activated 217 ms before the onset of the shoulder movement. The same reasoning can apply for the onset reported for the other two muscles.

Indeed, all agonist glenohumeral (anterior deltoid and biceps) and scapulotoracic (upper and lower trapezius, serratus anterior) muscles we activated almost simultaneouly (figure 7). These data showed a co-activation pattern of the recruitment order of these muscles. Note that the difference in the onset in which these muscles were recruited was less that 10 ms. The co-activation of these muscles increased joint stiffeness, allowing more joint stability (Aruin & Almeida, 1997). This increase in joint stability is necessary since the glenohumeral and scapulotoracic joints are very unstable due to their anatomic configuration (Bigliane, et al, 1996). The antagonist latissimus dorsi, pectoralis major and triceps muscles were activated around 40 ms after the onset of the agonist muscles, around the onset of the movement.

Finally, we have shown that shoulder joint stability does not affect the recruitment order since we failed to observe group differences (Table III, figure 7). Also, some studies suggest the protocal, based on the co-activation of the glenohumeral and scapulothoracic muscles (Inman, et al, 1944).

Clinical implication

The rehabilitation of individuals with the Shoulder Impingement Syndrome depends on its degree of severity, including the type of pain reported by the client. Several physical therapy protocols have been designed to treat the Shoulder Impingement Syndrome (Brewster & Schwab, 1993; Kamkar, et al, 1993; Wilk & Andrews, 1996; Kibler, 1998). In general the goal of these treatments is to strengthen the rotator cuff muscles, restore the normal shoulder range of motion, and decrease pain. A training program has been suggested to restore the scapulo-humeral rhythm (Jobe & Bradley, 1989), by strengthening the rotator cuff and the scapular rotators. The basic

idea behind this kind of kinesiotherapy treatment is that the strengthening of the rotator cuff and the scapular rotator will ensure that the scapula will follows the humerus, thus preventing the contact of the great tuberosity with the coracromial arch. The closer contact between the coracacromial arch and great tuberosity will cause the impingement syndrome (Flatow, et al, 1994).

In our experiment, the individuals had a history of shoulder impingement. They did not report pain, but had shoulder instability. Despite the fact that the major function of the rotator cuff is to provide shoulder stability, studies have also shown the involvement of the other glenohumeral and scapulotoracic muscles in addition to that of the rotator cuff muscles in individuals with shoulder dysfunction (Glousman, et al, 1988; Scovazzo, et al, 1991). For instance, compared with the control group, the individuals with a painful shoulder during freestyle swimming showed changes in the pattern of activation of the rotator cuff muscles (infraspinatus, subescapulares) and the anterior and middle deltoid, upper trapezius, romboids, and serratus anterior muscles (Scovazzo, et al, 1991).

We did not record the EMG activities of the rotator cuff muscles, but as pointed above, the change in the pattern of activities of these muscles was also accompained by changes in the glemuhumeral and scapulothoracic muscles. In our study, there were no changes in the pattern of muscle activities of the glemuhumeral and scapulothoracic muscles for individuals with shoulder instability. Even if there were only the involvement of the rotator cuff muscles, we would expect a change in the kinematic of the shoulder movements. Also note that we did not observe any kinematic changes in the shoulder movements between the groups with and without shoulder instability (figure 3).

However, we should be very careful in not recommending any physical therapy protocal

aimed at strengthening the rotator cuff and the scapular rotators if individuals have a history of shoulder impingement and shoulder instability. We showed that he capability of these individuals to properly activate their muscles to generate appropriate isotonic force is preserved during the performance of the unloaded fast movements. The increase in muscle strength due to isometric training it is not necessarily transferred to isotonic contraction (Jones, et al, 1989; Sale & MacDougall, 1981). So, the strength of the shoulder muscles would not improve the ability of these individuals to generate the unloaded fast shoulder movements. However, under load condition, we could observe the change in the pattern of the EMG of the recorded muscles, which would affect the kinematic movement. Let us assume that future studies will prove that this hypothesis is correct. Therefore, the strengthening training for individuals with shoulder instability would be highly recommended, mainly if they usually perform loaded voluntary movements or isometric contraction of the shoulder muscles on a daily basis.

Other studies have suggested the proprioceptive training for individuals with the Shoulder Impingement Syndrome (Lephart, et al, 1997). In our experiment, we did not observe any change in the recruitment order of the glenohumeral and scapulothoracic muscles for individuals with shoulder instability. Also, there was no change in the kinematic of the shoulder and the accuracy of these movements that could be related to any proprioceptive defict. So, based on these findings we do not recommend any specific training protocal aimed to improve the proprioception of individuals with shoulder instability.

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Figures legends

Figure 1

Illustration of the task setup. From the arms handing relaxed at the side of body, the subjects performed bilateral and simultaneous elevation of both shoulders in the scapular plane (30 degrees in front of the coronal plane), the movements was performed into three different target distances (30^0, 90^0 and 150^0).

Figure 2

Temporal series for the shoulder movements over three angular target distances, performed of one normal subject (A) and one subject with History of Shoulder Impingement Syndrome (HSIS). The figure depicts the angular excursion, velocity, and eletromiographic (EMG) muscle activities.

Figure 3

The angular excursion and the velocity of the shoulder elevation movements over the three targets distance (30^0, 90^0 and 150^0). The average values obtained for the two groups of subjects (NN and HSIS).

Figure 4

The linear translational movements of the shoulder for the two groups of subjects (NN and HSIS) over three angular target distances (30^0, 90^0 and 150^0), in X, Y e Z-axis, corresponding respectively to the movements of the medial translation (A), cephalic translation (B) and the posterior translation (C).

Figure 5

The average of the intensity of activation the agonist muscles for the two groups of subjects (NN and HSIS) over three angular target distances (30^0, 90^0 and 150^0). The agonistas recorded was the glenohumeral (anterior deltoid and biceps long head) and scapulothoracic (upper and lower trapezius, anterior serratus) joints.

Figure 6

The amount of the muscle activities of the first agonist (anterior deltoid, biceps long head, upper and lower trapezius, serratus anterior) and antagonist (pectoralis major, latissimus dorsi and triceps) bursts. The muscle activities were recorded for the both groups of subjects (NN and HSIS) over three angular target distances (30^0, 90^0 and 150^0).

Figure 7

The recruitment order of all muscles in relation to the onset of the movement, for the two groups of subjects (NN and HSIS). Muscles recorded: anterior deltoid (AD), pectoralis major

(PEC), latissimus dorsi (LT), upper trapezius (UP), lower trapezius (LT), serratus anterior (SER), biceps long head (BIC) and triceps (TRIC).

Table I

The assessment for presence of history of shoulder impingement, coracoacromial impingement, glenohumeral instability and general joint hypermobility for each subjects of the HSIS group.

Table II

ANOVA results ($p<0,05$) for the angle and velocity of the shoulder, shoulder movement translational, intensity of activation the agonist muscles and quantity of muscles activity.

Table III

ANOVA results ($p<0,05$) for the antagonist latency in relation of the latissimus dorsi, showing both group of the subjects (NN and HSIS) did not modulate the antagonist latency.

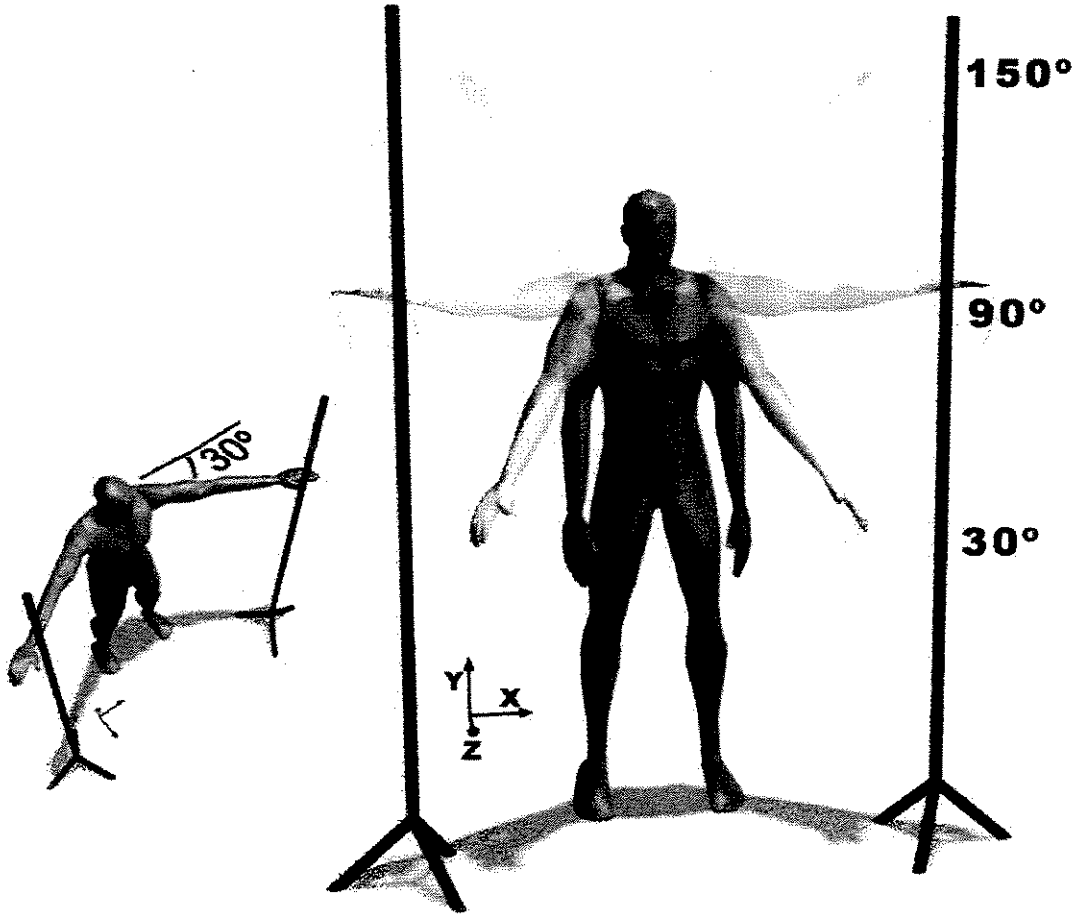


Fig. 1

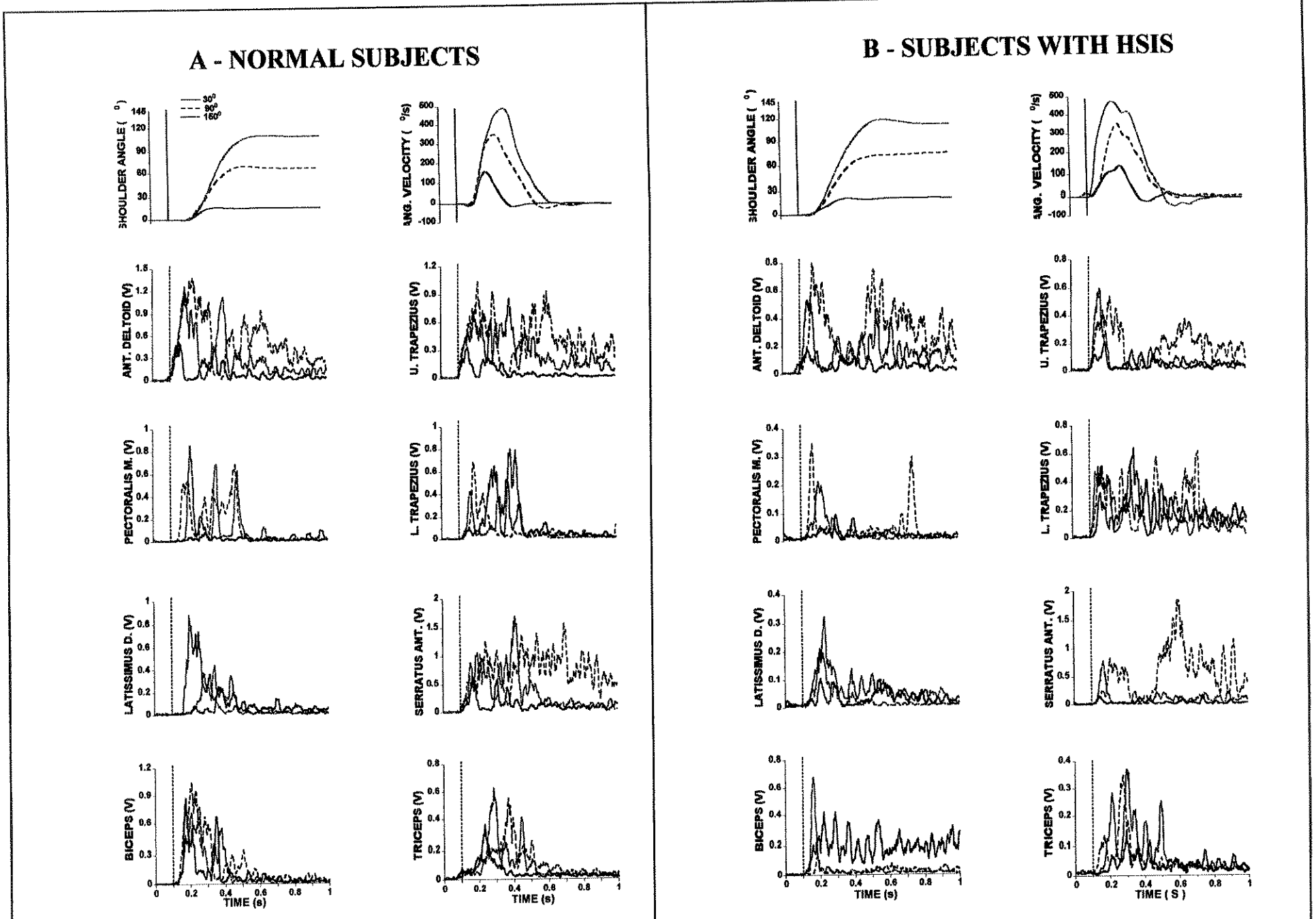
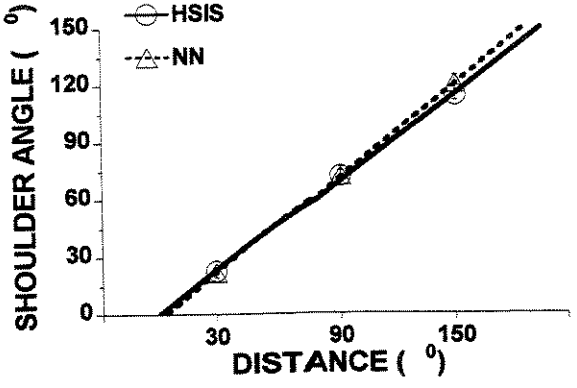

A - NORMAL SUBJECTS
B - SUBJECTS WITH HSIS
30°
90°
150°
SHOULDER ANGLE (°)
ANG. VELOCITY (°/s)
ANT. DELTOID (V)
U. TRAPEZIUS (V)
PECTORALIS M. (V)
L. TRAPEZIUS (V)
LATISSIMUS D. (V)
SERRATUS ANT. (V)
BICEPS (V)
TRICEPS (V)
TIME (s)
TIME (S)

Fig.2

ANGULAR KINEMATIC AND VELOCITY



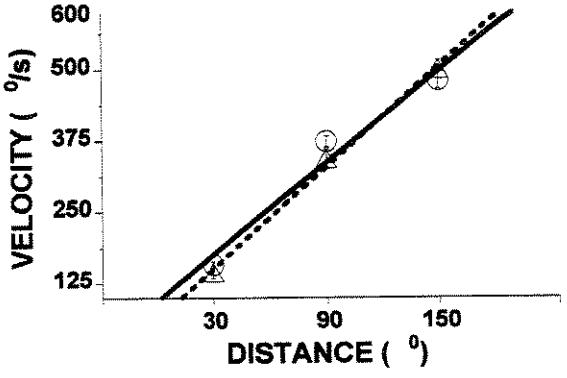


FIG.3

SHOULDER TRANSLATIONAL MOVEMENT

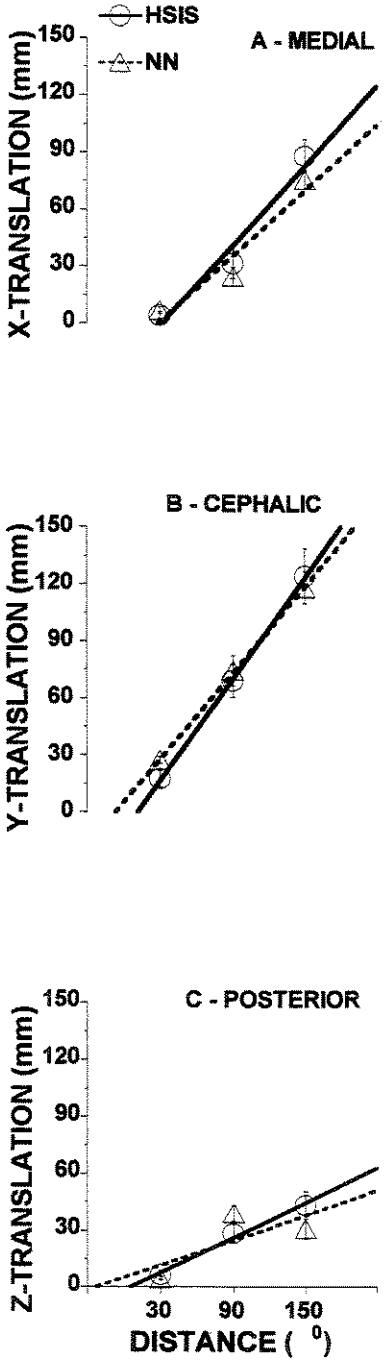


FIG.4

INTENSITY OF MUSCLE ACTIVATION

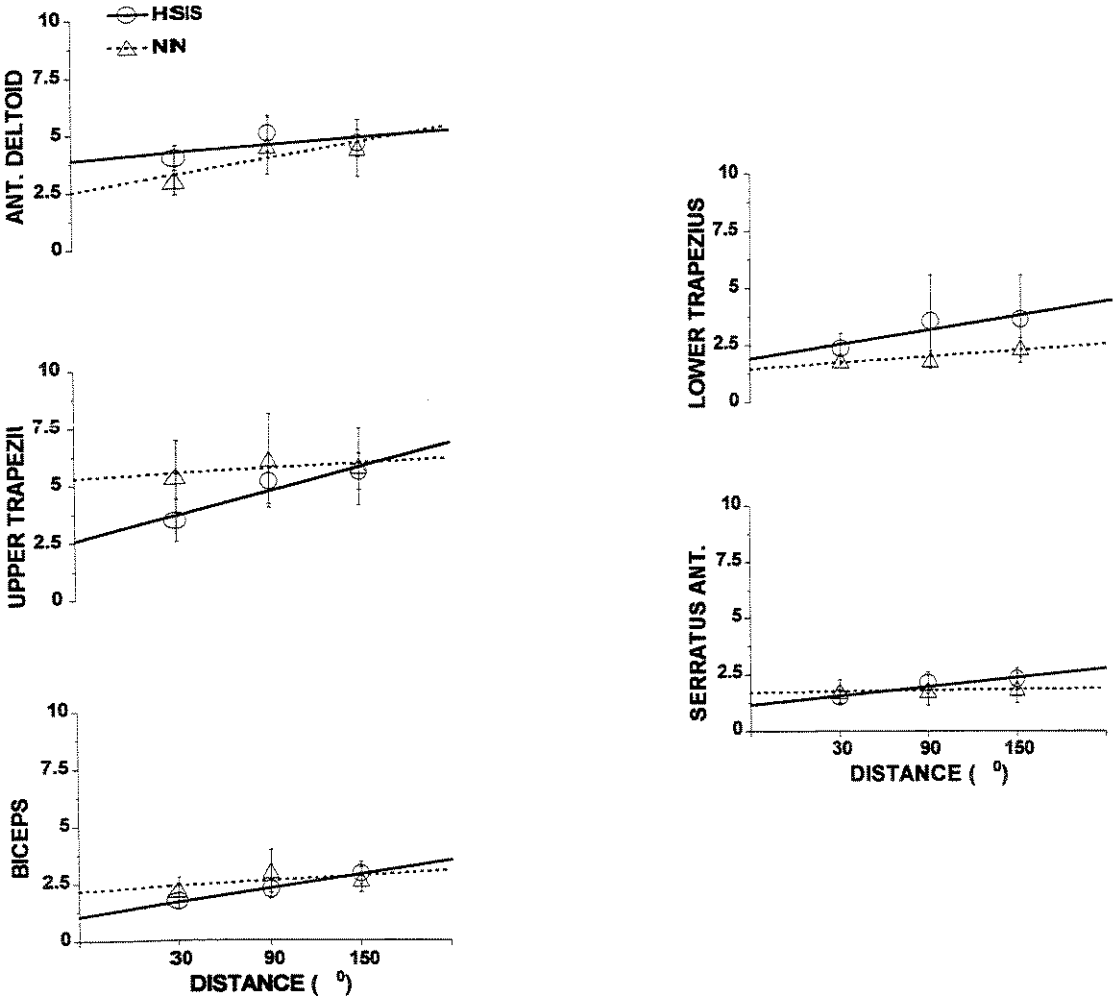


FIG.5

AMOUNT OF MUSCLE ACTIVITY

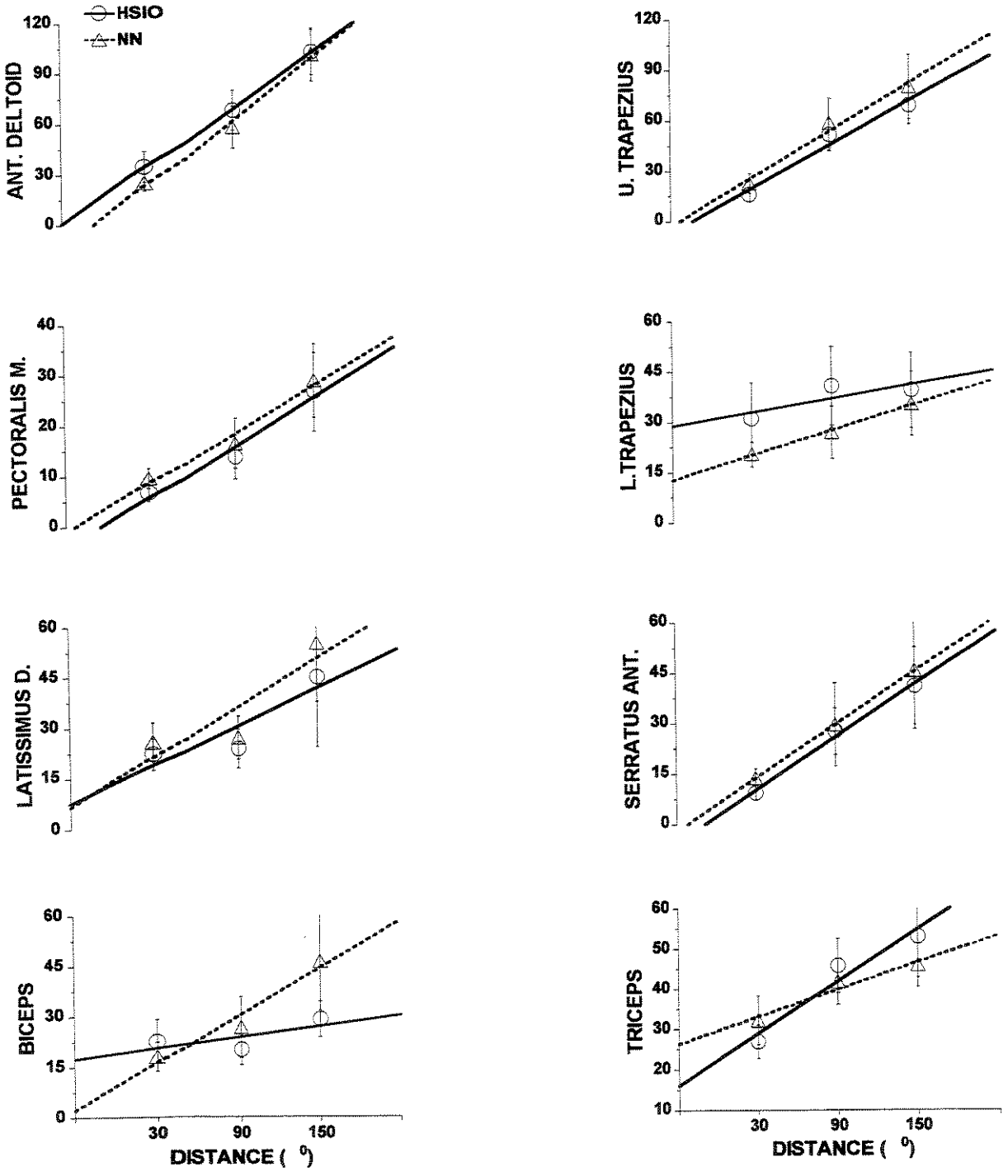


FIG.6

RECRUITMENT ORDER

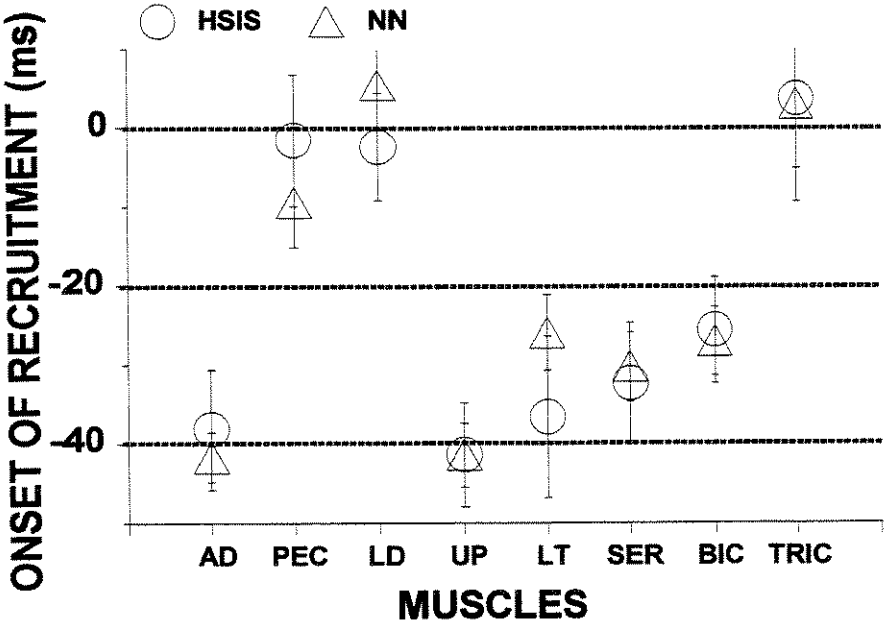


FIG.7

TABLE I - Clinical assessment of the subjects with History of Shoulder Impingement Syndrome (HSIS)

HSIS SUBJECTS	IMPINGEMENT SIGNAL	ANTERIOR-POSTERIOR INSTABILITY	INFERIOR INSTABILITY	HYPER MOBILITY
1	-	1+	1+	0
2	-	2+	2+	0
3	-	0	1+	+
4	-	2+	2+	0
5	-	1+	0	+
6	-	2+	2+	+
7	-	1+	1+	0
8	-	1+	1+	0

The signal + and – represented presence and absent respectively. The signal 0, 1+ and 2+ represented the grade of the glenohumeral instability. 0, the movement of the humeral head is not perceived, 1+, humeral head moves to glenoid rim, but not over it and 2+, humeral head subluxates over glenoid rim and spontaneously reduces by anterior-posterior instability. The instability inferior, 0, 1+ and 2+ represented the translation of the humeral head, no translation inferior, bigger than 5mm and between 5 and 10 mm respectively.

TABLE II – Anova results

DEPENDENT VARIABLES	GROUP (G)		DISTANCES (D)		INTERACT. (G*D)	
	F(1, 14)	p	F (2, 28)	p	F(2, 28)	p
SHOULDER KINEMATIC						
Shoulder angle	0.19	0.67	1045.15	0.00	1.55	0.23
Shoulder velocity	0.26	0.62	480.25	0.00	3.14	0.06
SHOULDER TRANSLATIONAL MOVEMENT						
Lateral-medial	1.68	0.22	91.22	0.00	1.22	0.31
Cephalo-Caudal	0.19	0.67	79.73	0.00	0.86	0.44
Antero-posterior	0.21	0.88	48.00	0.00	2.75	0.09
INTENSITY OF ACTIVATION OF THE AGONIST MUSCLES						
Anterior Deltoid	0.26	0.62	4.6	0.19	0.38	0.69
Pectoralis major	0.17	0.68	4.5	0.21	0.74	0.93
Upper Trapezius	0.249	0.59	3.12	0.60	1.07	0.36
Lower Trapezius	0.56	0.47	104.12	0.26	0.56	0.58
Serratus Anterior	0.06	0.82	2.71	0.08	1.74	0.19
AMOUNT OF MUSCLES ACTIVITY						
Anterior Deltoid	0.24	0.66	480.74	0.00	0.27	0.80
Pectoralis major	**0.25**	**0.51**	5.61	0.01	1.98	0.16
Latissimus dorsi	0.262	0.62	3.358	0.05	0.06	0.94
Upper Trapezius	0.310	0.59	32.33	0.00	0.06	0.94
Lower Trapezius	0.652	0.43	2.297	0.12	0.40	0.67
Serratus Anterior	0.084	0.76	11.60	0.00	0.02	0.94
Biceps	0.448	0.51	5.613	0.01	1.93	0.16
Triceps	0.154	0.70	5.95	0.00	0.45	0.64

Table II

TABLE III – Antagonist latency in relation to the Latissimus Dorsi

DEPENDENT VARIABLES	GROUP (G)		DISTANCES (D)		INTERECT. (G*D)	
	F(1, 14)	p	F (2, 28)	p	F(2, 28)	p
Anterior Deltoid	1.54	0.23	0.84	0.44	0.34	0.71
Pectoralis major	1.45	0.24	0.48	0.62	0.25	0.77
Upper Trapezius	0.72	0.40	2.08	0.14	0.34	0.71
Lower Trapezius	0.06	0.80	0.31	0.73	1.66	0.20
Serratus Anterior	0.35	0.56	0.71	0.50	0.25	0.77

TABLE III

V. CONSIDERAÇÕES FINAIS

Como vimos, vários estudos apontam mudanças biomecânicas, cinemáticas e de atividade muscular em indivíduos que apresentam Síndrome do Impacto do Ombro (SIO). Tais déficits poderiam estar vinculados às causas ou aos efeitos da Síndrome do Impacto do Ombro. Esses achados nos levaram a levantar hipóteses de que os indivíduos com essa Síndrome poderiam apresentar déficit de controle motor. Para testar essas hipóteses, os sujeitos escolhidos foram nadadores, os quais apresentam um alto índice de disfunções na articulação do ombro (Johnson, 1988; Bak & Magnusson, 1997). Durante a avaliação, detectou-se que a grande maioria desses indivíduos apresentava instabilidade glenoumeral.

Contrário às nossas hipóteses iniciais, os nadadores com História de Síndrome do Impacto do Ombro e Instabilidade glenoumeral (HSIO) não apresentaram qualquer déficit no seu mecanismo de controle motor durante a performance dos movimentos requeridos. Primeiramente, nós não observamos qualquer mudança na cinemática dos movimentos do ombro nos indivíduos com HSIO. Estudos recentes de Ludewig e Cook (2000) mostraram haver um desacoplamento entre os movimentos do úmero e da escápula em indivíduos com Síndrome do Impacto do Ombro. Em conseqüência a esse distúrbio, esses indivíduos apresentariam uma redução no espaço subacromial, causando maior impacto entre a cabeça umeral e o acrômio. Nós mostramos que ambos os grupos de sujeitos tiveram a mesma precisão na excursão dos movimentos de elevação do úmero e a mesma translação posterior para os três diferentes alvos. Dessa forma, os indivíduos com HSIO não apresentam alterações no acoplamento entre a escápula e o úmero, quando comparado aos sujeitos normais.

Como os movimentos voluntários do ombro foram similares em indivíduos normais e em

indivíduos com HSIO, era de se esperar que ambos os grupos de sujeitos usaram as mesmas estratégias na modulação das atividades dos músculos agonistas e antagonistas. Em nosso experimento, ambos os grupos de indivíduos tiveram o mesmo padrão de modulação, que pode ser observado nos músculos glenoumerais (deltóide anterior, peitoral maior e grande dorsal), escapulotorácicos (serrátil anterior, trapézio superior e inferior) e nos que participam da articulação do cotovelo (bíceps e tríceps). Portanto, o nosso experimento difere de outros estudos em que os autores encontraram diferenças no padrão das atividades musculares (Glousman, 1988; Scovazzo, et al, 1991; Wadsworth & Bullock-Saxton, 1997; Ludewig & Cook, 2000). Em outro estudo, bastante similar ao nosso, em termos de movimentos, patologia e sujeitos, os autores registraram mudanças na ordem de recrutamento dos músculos da escápula. Para os mesmos músculos pesquisados, nós mostramos que o tempo de recrutamento não excedeu 40ms antes do início do movimento e foram iguais para os dois grupos estudados. Os valores encontrados por Wadsworth & Saxton, (1997) tanto para os indivíduos normais como para os indivíduos com História de Síndrome do Impacto do Ombro, são muito grandes e não podem explicar as funções de acoplamento e coordenação (Inman, et al., 1944; Popen & Walker, 1976) das complexas articulações do ombro.

Exceto esse último estudo (Wadsworth & Saxton, 1997), é difícil a comparação entre os resultados de nossos estudos e os resultados encontrados na literatura (Glousman, 1988; Scovazzo, et al, 1991, Lukasiewicz, et al., 1999; Ludewig & Cook, 2000), pois os músculos registrados, o tipo de tarefas e a caracterização dos grupos, em termos de sujeitos e ocupação, variam entre os experimentos. Dessa maneira, nós poderíamos concluir que a presença de História de Síndrome do Impacto do Ombro e Instabilidade Glenoumeral não afetaria a

habilidade do sistema de controle motor em ativar e modular os músculos glenoumerais e escapulotorácicos ao gerar movimentos rápidos e sem carga. A instabilidade do ombro poderia ser afetada para os movimentos realizados com carga (Lukasiewicz,1999 e Ludewig & CooK, 2000), por isso, nós estamos planejando testar essa hipótese no futuro.

Existem vários protocolos na área de reabilitação para os indivíduos com Síndrome do Impacto do Ombro (Brewster & Schwab, 1993; Kamkar, et al., 1993; Wilk & Andrews, 1996; Kibler, 1998); de maneira geral, esses protocolos preconizam a manutenção da amplitude de movimento, fortalecimento muscular, especialmente para os músculos escapulares e do manguito rotador. O restabelecimento das funções proprioceptivas também é indicado na fase final do tratamento (Lephart, et al, 1997) .

Será que os programas que indicam o fortalecimento generalizado dos músculos do manguito rotador e da cintura escapular podem estar corretos? A partir dos dados obtidos na literatura, essas alterações são bem particulares e dependem do tipo de tarefa realizada. Em nossos experimentos, os indivíduos com História de Síndrome do Impacto e Instabilidade glenoumeral não apresentam déficit muscular e muito menos proprioceptivos ao realizarem movimentos voluntários sem carga. Nesse caso, exercícios de fortalecimento e de treinamento proprioceptivos poderiam não ser indicados.

Até o momento, existem poucos estudos que investigam a cinemática acoplada às atividades musculares da articulação do ombro. Esses estudos deveriam apresentar maior variação de movimentos e de tarefas. Entre os estudos atuais e os programas de reabilitação ainda existe uma lacuna, no sentido de que outras hipóteses precisam ser testadas e diagnosticadas. Partindo de evidências concretas, poderemos estruturar um verdadeiro programa de reabilitação.

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VI. ANEXOS

Protocolo de tratamento da SIO

Protocolo de tratamento da SIO desenvolvido por Wilk & Andrews, 1995.

Fase 1 – fase de proteção. (0 a 6 semanas)

- **Objetivos:**
- Retorno gradual a ADM normal
- Aumento da força muscular
- Diminuição da dor

0 a 3 semanas

- Tipóia para maior conforto (1 a 2 semanas)
- Exercícios pendulares
- Exercícios ativos assistidos para restabelecer a ADM, em amplitudes que não provocam dor, aumentando gradualmente em amplitudes tolerantes.
- Uso de polias somente para a flexão
- Exercícios para ADM de cotovelo, e apreensão palmar
- Inicio dos exercícios isométricos(submáximo e sem provocar dor):

Abdutores, rotadores externos e internos, flexores do cotovelo e flexores do ombro.

- Uso de modalidades para controlar a dor (gelo, estimulação elétrica)

3 a 6 semanas

- Progresso em todos os exercícios
- Exercícios ativos assistidos para restabelecer a ADM, iniciando RE e RI do ombro até 45 graus de abdução.
- Inicio com os exercícios com elástico, com o braço ao lado do corpo
- Inicio dos exercícios de estabilização da cabeça umeral

Fase 2 – Fase intermediária (7 a 12 semanas)

Objetivos

- Completa ADM sem provocar dor
- Aumento da força e potência muscular
- Aumento das atividades funcionais; diminuição da dor residual

7 a 10 semanas

- Exercícios ativos assistidos para restabelecer a ADM

Flexão de 170 a 180 graus

RE de 75 a 90 graus, RI de 75 a 85 graus (com 90 graus de abdução do ombro)

RE de 30 a 40 graus (com 0 graus de abdução do ombro)

- Exercícios de fortalecimento do ombro

Exercícios com elástico, RE/RI com o braço ao lado do corpo.

Exercícios Isotônicos: deltóide, supraespinhoso, flexores do cotovelo e músculos escapulares

- Utilização da bicicleta ergométrica para os braços
- ADM total é a meta entre 8 e 10 semanas.

10 a 12 semanas

- Continuação de todos os exercícios acima
- Inicio dos exercícios isocinéticos no plano escapular
- Inicio dos exercícios Isotônicos de RE/RI
- Inicio dos exercícios de controle neuromuscular da escápula

Fase 3 – Fase avançada (13 a 21 semanas)

Objetivos:

- Manter a ADM completa, sem dor
- Melhorar a força do complexo do ombro
- Melhorar o controle neuromuscular
- Retorno gradual as atividades normais

13 a 18 semanas

- Inicio do programa de alongamentos ativos para o ombro
- Exercícios ativos assistidos para manter a ADM completa
- Alongamentos capsulares (exceto nas instabilidades do ombro)
- Inicio de exercícios de fortalecimento agressivo, programa isotônico:

Flexão do ombro, abdução do ombro, RE/RI, supraspinhoso, flexores e extensores do cotovelo e musculatura escapular.

- Realização do teste Isocinético (até 14 semanas)
- Inicio do programa de condicionamento geral

18 a 21 semanas

- Continuação de todos os exercícios listados acima
- Inicio de programas esportivos intervalados

Fase 4 – Fase de retorno as atividades

Objetivos:

- Retorno as atividades normais

21 a 26 semanas

- Retorno gradual as atividades normais
- Realização do teste Isocinético
- Continuação do programa de esportes intervalado
- Continuação dos exercícios básicos (fortalecimento e flexibilidade)

* - Exercícios básicos:

- Flexão na polia
- Alongamentos de flexão
- Alongamentos de RE/RI
- Fortalecimento de RE/RI
- Elevações laterais até 90 graus
- Exercícios de ADM passiva, abdução horizontal
- Fortalecimento bíceps