Estratégias de Controle Motor utilizadas por indivíduos com lombalgia crônica submetidos a um auto-distúrbio postural

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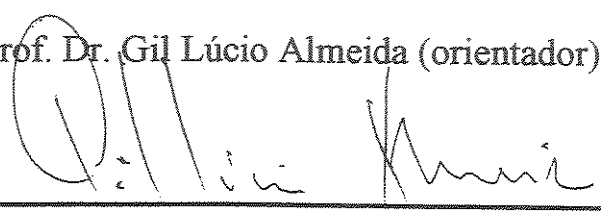

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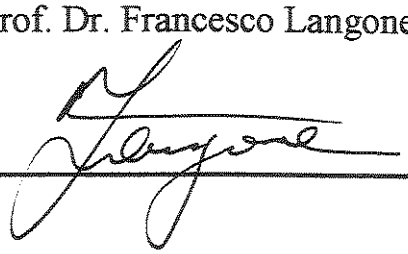

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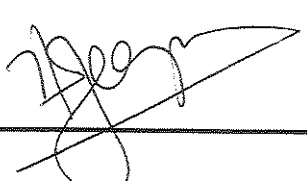

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Dedico esse trabalho a minha esposa Márcia e a minha filha Marina

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RESUMO

Introdução. O objetivo desse estudo foi investigar as diferenças de estratégias de controle motor utilizadas por portadores de lombalgia crônica e sujeitos normais durante um auto-distúrbio postural. **Sujeitos**. Dezesseis sujeitos participaram deste estudo sendo divididos em dois grupos de oito. O grupo I era composto de quatro homens e quatro mulheres com idades entre 23 a 40 anos(M=25, SD=6,19) que não apresentavam história de lombalgia, ou patologias neurológicas, ou ortopédicas. O grupo II apresentou o mesmo número de sujeitos, quatro homens e quatro mulheres com idades entre 21 a 30 anos (M=26, SD=3,92) que apresentavam lombalgia crônica. **Metodologia.** Pedimos que os sujeitos segurassem um bastão com uma carga de 2 Kg, elevando e mantendo acima da cabeça com os ombros em 130° de flexão com os cotovelos estendidos, movendo em direção a um alvo à 9O graus em extensão, o mais rápido possível, estando em posição ortostática sobre uma plataforma de força. Os ângulos das articulações do ombro, quadril, joelho e tornozelo, foram registrados com um sistema óptico-elétrico de análise do movimento (Optotrak 3020), a atividade eletromiográfica dos músculos deltóide anterior, deltóide posterior, reto abdominal, multifidus, reto femural, bíceps femural, gastrocnêmio lateral e tibial anterior com eletrodos de superfície (Delsys) e o deslocamento do centro de pressão através de uma plataforma de força (AMTI OR-6). **Resultados.** Sujeitos sem e com lombalgia crônica apresentaram estratégias semelhantes de controle motor. No início eles apresentaram atividade eletromiográfica dos músculos focais Deltóide Anterior e Posterior e os outros permaneceram em atividade basal, com o movimento dos membros superiores em direção ao alvo, vários distúrbios ocorreram na postura provocando várias mudanças nos padrões cinemáticos e eletromiográficos dos sujeitos. **Discussão e Conclusão.** Os indivíduos com e sem

lombalgia crônica produziram rápidos movimentos bilaterais dos ombros de cima para baixo com padrões cinemáticos e eletromiográficos semelhantes. Indivíduos com lombalgia crônica não mudam suas estratégias de controle motor em reagir a um auto-distúrbio postural.

ABSTRACT

The purpose of this study was to investigate the differences of motor control in subjects with and without chronic low back pain during self-inflicted postural disturbance. **Subjects**. Sixteen subjects participated in this study divided into two groups of eight subjects. Group I included four males and four females (M=25, SD=6.19) who had never low back pain or neurologic or orthopedic diseases. Group II included the same number of subjects four males and four females (M=26, SD 3.92) who had chronic low back pain and did not have apparent limitations of motion at the major joints and any other complaint of neurological or orthopedic disorders in addition to the history of low back pain. **Methods.** The subjects were asked to hold the bar with two kilograms of weight and elevate it up and forward (overhead) keeping the upper-limb extended with shoulders at 130° moving toward a target simultaneously both shoulders at 90° (from upper to down position) The subjects also stood on a force platform at the upper right position. The movement of shoulder, hip, knee and ankle joints were recorded by the Optotrak 3020, three-dimensional, optoelectric motion analysis, the EMG activity of the anterior deltoid, posterior deltoid, recto abdominis, multifidus, rectus femoris, biceps femoris, lateralis gastrocnemius e tibialis anterior were detected by bipolar surface electrodes (Delsys), and the displacement of center of pressure were registered by biomechanical platform (AMTI OR-6). **Results**. Subjects with and without low back pain showed similar strategies. In the beginning of the task they showed focal muscles EMG activity of the anterior and posterior deltoid the other muscles were in basal level. When the movement of the shoulders occurred in the target direction, postural disturbance occurred and many changes in cinematic of shoulder, hip,

knee and ankle and EMG activities of focal and non-focal joints. **Discussion and Conclusion.** Both Individuals with and without chronic low back pain generated fast bi-lateral shoulder movements with similar kinematic and pattern of muscle activities, from the overhead position to a downward target. Individuals with chronic low-back pain did not change their strategy to react to self-inflicted disturbance.

I. INTRODUÇÃO

A coluna vertebral lombar é composta de 5 vértebras que se localizam abaixo da região torácica; é mantida sobre a cintura pélvica por um conjunto de músculos e ligamentos que suportam todo o peso da coluna na postura ereta. Na parte anterior se localizam os corpos vertebrais, discos e ligamentos longitudinais; na posterior, o arco vertebral, suas articulações, ligamentos e os músculos posteriores; entre os corpos se encontram os discos intervertebrais.

Os corpos vertebrais e os discos intervertebrais da região lombar suportam mais de 80 % da carga de compressão axial. Os corpos, com exceção das bordas, são revestidos por uma fina camada de membrana hialina que entra em contato com o anel fibroso do disco intervertebral. Esse contato tem uma importância vital na nutrição do disco.

O arco vertebral, formado pelos pedículos e pela lâmina posterior, protege as estruturas do canal espinhal e forame intervertebral. Cada arco apresenta um número de processos que limitam anatomicamente os movimentos em determinada direção. A coluna lombar tem que combinar mobilidade com estabilidade. Mobilidade e estabilidade são geralmente inversamente proporcionais. Na região lombar o disco e o arco intervertebral, duas articulações sinoviais e os ligamentos geram a estabilidade necessária, possibilitando a mobilidade.

O disco intervertebral, que é composto por um núcleo pulposo e um anel fibroso. A principal função do disco é suportar as pressões de compressão e cisalhamento e manter a estabilidade e a rigidez no movimento entre os segmentos vertebrais. O anel fibroso, graças às suas propriedades elásticas, suporta as pressões transmitidas ao núcleo pulposo. O disco é avascular, sua nutrição ocorre por difusão e apresenta inervação apenas da parte externa do anel fibroso (Bogduk, 1983).

Os ligamentos da coluna vertebral têm como função manter a estabilidade entre os segmentos; os ligamentos longitudinais anterior e posterior localizam-se na parte anterior e posterior dos corpos vertebrais. O ligamento amarelo é o mais elástico, na região lombar ele é mais fino e suas fibras passam verticalmente entre as lâminas. Os ligamentos supraespinhoso e interespinhoso, reforçados pelas fibras mediais do erector da espina e os interespinhais, completam a parte posterior da coluna lombar.

Os músculos são responsáveis pela manutenção da postura e pelos movimentos. Existe importante relação entre a coluna lombar e o ângulo de inclinação pélvica: quanto maior a inclinação pélvica, maior o aumento da curvatura anterior lombar (lordose lombar). Os músculos responsáveis pela manutenção da inclinação pélvica são: os músculos abdominais (reto abdominal e oblíquos abdominais), os eretores da espinha (sacroespinhais), o glúteo máximo, os ísquiostibiais e o psoas maior. A inclinação pélvica é determinada principalmente pela interação desses músculos. Os músculos eretores da coluna e o psoas maior aumentam o ângulo de inclinação pélvica levando ao aumento da lordose, e os abdominais, glúteos e isquiotibiais diminuem a inclinação, diminuindo a lordose. O encurtamento desses músculos pode alterar a posição de repouso e a dinâmica dos movimentos nessa região (Taylor & Twomey, 2000).

Os músculos abdominais possuem importante função na flexão do tronco contra a ação da gravidade, principalmente o reto abdominal. Os oblíquos atuam na flexão e também na rotação; não existem evidências de que somente o fortalecimento dos músculos abdominais tenha influência sobre o ângulo de inclinação pélvica (Day, Smidt & Lehman, 1984; Taylor & Twomey, 2000). Os eretores da coluna são responsáveis principalmente pela extensão do tronco. O mais importante extensor é o multifidus, o eretor espinhal, que é dividido em longuíssimo do tórax e ileocostal lombar são mais importantes nos movimentos de inclinação lateral (Ng,

Richardson & Jull, 1997). O glúteo máximo é extensor do quadril, junto com os isquiotibiais, que, além de extensores do quadril, são flexores do joelho. O psoas maior é um músculo flexor do tronco e, associado com o ilíaco, também é flexor do quadril.

Movimentos da coluna lombar

Para ocorrer o movimento na região lombar deve existir uma interação entre todos os seus segmentos. No entanto, o movimento entre cada seguimento vertebral é mínimo, mas somando-se todos os movimentos entre as vértebras, consegue-se uma boa amplitude de flexão e extensão. A orientação das facetas articulares facilita os movimentos no plano sagital: geralmente 80% do movimento é para flexão e 20% para extensão. As amplitudes de movimento variam de autor para autor devido a diferentes métodos de mensuração (Taylor & Twomey, 2000). Uma amplitude média de 70° de flexão-extensão é considerada normal para pessoas adultas.

O movimento de rotação ainda foi pouco estudado. Porém, para Kapandji (1990) essa rotação na região lombar é mínima, devido ao fato de o posicionamento das facetas articulares permitir o mínimo desse movimento. Outras fontes alegam uma amplitude que varia de 5° a 36° de movimento (Loebl, 1973; Horak, 1987)

Lombalgia

Dor segundo a International Association for Study of Pain (IASP) é "uma experiência sensorial e emocional desagradável, associada a um dano real ou potencial dos tecidos, ou descrita em termos de tais lesões.", pode ser conseqüência de um estímulo nocivo, lesão do sistema nervoso ou a associação de vários mecanismos.

A dor é classificada como aguda ou crônica. A determinação da dor aguda é precisa e está relacionada com o mecanismo causador, havendo expectativa de desaparecimento após a cura da lesão. Na presença da dor pode haver repostas neurovegetativas tais como: elevação da pressão

arterial, taquicardia e outras, como ansiedade e agitação. A dor crônica é aquela que se mantém por mais de três meses, geralmente não tem mais a função de alerta, é mal delimitada e pode levar a sérios transtornos psicológicos como: alteração do humor, ansiedade, depressão e outros. Essas características podem ser atenuadas, acentuadas ou perpetuadas pelas variáveis socioculturais e psíquicas do indivíduo e do meio (Pimenta,1999).

A dor na região lombar ou lombalgia é a causa mais comum de incapacidade músculo-esquelética nos países industrializados em indivíduos abaixo de 45 anos (Biering-Sorensen, 1983). Atualmente ela assume proporções de uma verdadeira epidemia, ocasionando grandes custos sociais e econômicos. Apesar de ter baixa letalidade, a lombalgia possui uma altíssima morbidade (Nachensom, 1996), diminuindo a qualidade de vida da população. Sua incidência não aparenta depender somente do grau de carga a que a coluna é submetida, uma vez que sua freqüência não aumenta em ocupações que requerem esforços (Dales, Macdonald & Porter, 1986). Contudo, numa meta análise do National Institute for Occupational Safety and Health (NIOSHI) mostra exatamente o contrário. A associação entre esforços físicos e lombalgias vêm sendo demonstrada na literatura ocupacional há décadas. Por mecanismos diferentes tanto as profissões sedentárias quanto as de trabalho pesado são considerados de risco para lombalgia ocupacional.

A etiologia da lombalgia aguda, na maioria dos casos, é associada a fatores mecânicos (Gallienne & Gallou,1991). Porém, existem inúmeras causas de lombalgia aguda que muitas vezes não são determinadas. Deyo & Tsui-Wu (1987) relataram que a causa da lombalgia aguda é desconhecida em 80-85% dos casos. As dores lombares agudas também são caracterizadas como somática e radicular. A dor somática é causada por estimulação nociva de estruturas ou tecidos intrínsecos da região lombar. Geralmente ela provém da vértebra, das articulações

zigoapofisárias, do disco intervertebral, dos ligamentos, dos músculos e da fáscia lombar. Já a dor lombar aguda radicular é causada por irritação dos nervos que passam pelo canal vertebral ou pelos forames intervertebrais lombares (Bogduk, 2000). Finalmente, existem também as lombalgias exógenas, causadas por modificações na cinética de outras articulações não lombares (coxo-femural, joelhos, tornozelos e pés), e as lombalgias de origem reflexa, como as causadas por distúrbios nos rins, intestinos e outros órgãos. O diagnóstico das lombalgias agudas exógenas e de origem reflexa é ainda mais complicado.

Por outro lado, vários fatores têm sido sugeridos para determinar a causa da lombalgia crônica. Entre esses fatores destacam-se os psicológicos (Murray, 1982; Sternbach, 1973; Frymoyer, 1985; Turk, Okifuji & Sherman, 2000; Roland & Morris, 1983), os socioeconômicos (Vallfors, 1985; Natvig, 1970, Magora, 1975; Snook, 1982) e os fisiológicos (Biering-Sorenson, 1984; Pope et. al., 1985; Burton et. al., 1989; Esola, et. al., 1996; McClure et. al., 1997).

Vários estudos demonstraram que fatores psicológicos podem ser causa ou conseqüência da lombalgia (Murray, 1982; Sternbach, 1973; Frymoyer, 1985), isto é, a dor poderia levar à alteração psicológica ou essa alteração psicológica levar à dor. Pacientes com lombalgia crônica não se confrontam somente com o sintoma, mas também com muitos outros problemas, como os financeiros e familiares. Os fatores psicossociais e comportamentais favorecem a manutenção e o aumento desse problema. (Turk, Okifuji & Sherman, 2000). Porém, Roland & Morris (1983) demonstraram que as alterações psicológicas são consequências da dor crônica e não a sua causa.

Os estudos de Vallfors (1985) e Natvig (1970), demonstraram que a maioria dos trabalhadores que desenvolvem dor lombar crônica apresentam baixo nível educacional, falta de atividade esportiva e movimentos de inclinação e elevação repetidos por longo período. Porém,

para Magora (1975) e Snook (1982), a prevalência da lombalgia está mais ligada à insatisfação com o trabalho do que com os fatores de carga ou sedentarismo.

Independentemente da causa da lombalgia crônica, vários estudos demonstraram modificações na cinemática dos movimentos da coluna lombar nos indivíduos com história de lombalgia (Biering-Sorenson, 1984; Pope et. al., 1985; Burton et. al., 1989; Esola, et. al., 1996; McClure et. al., 1997). No entanto, não existe um consenso sobre essas modificações cinemáticas. Para alguns autores, como Biering-Sorenson (1984), Pope et. al. (1985) e Burton et. al. (1989), durante episódios de dores lombares existem pequenos movimentos vertebrais, e a qualidade desses parece estar alterada. Pessoas com disfunções lombares crônicas têm uma restrição da amplitude dos movimentos, mesmo durante os períodos não dolorosos, comparados com as pessoas sem essas disfunções Para outros, como Esola, et. al. (1996), McClure et. al. (1997) os movimentos de pessoas com história de lombalgia são amplos e rápidos durante a fase inicial da extensão da coluna a partir da posição fletida. Essa estratégia pode contribuir para a recorrência da dor ou resultar dela. Já durante o movimento de flexão essas pessoas tendem a movimentar antes a coluna do que o quadril. Esses autores encontraram também uma flexibilidade inadequada dos músculos do quadril, associada à excessiva mobilidade lombar.

A literatura também registra modificações no padrão eletromiográfico dos indivíduos com lombalgia crônica (Oddsson et al, 1997; Roy & Oddsson, 1998; Alexiev, 1994). Oddsson et al (1997) e Roy & Oddsson (1998) encontraram sinais de fadiga muscular nos músculos paravertebrais. Alexiev (1994) encontrou assimetria nas atividades entre os lados direito e esquerdo dos músculos paravertebrais. No entanto, em nosso trabalho, quando observamos indivíduos com lombalgia crônica comparados com indivíduos que não apresentavam esse sintoma, durante um auto-distúrbio postural causado pelos movimentos dos ombros em extensão,

com uma carga de 2 Kg nas mãos, não encontramos diferenças significativas entre as atividades eletromiográficas desses músculos.

Controle postural

Postura é o arranjo relativo de todos os segmentos corporais é uma resposta neuromecânica relacionada com a manutenção do equilíbrio. Um sistema está em equilíbrio mecânico quando a somatória de forças que agem sobre ele é igual a zero e esse sistema apresenta estabilidade após uma perturbação (interna ou externa) e se consegue retornar a sua posição de equilíbrio (Enoka, 2000).

O centro de gravidade localizado ao nível de S2 e a pequena base de suporte colocam o corpo, ereto e na posição ortostática, em permanente equilíbrio instável. Isto é, ocorrem freqüentemente oscilações posturais, frutos dos constantes desvios e correções da posição do centro de gravidade dentro da base de apoio (Latash, 1993).

Existe um controle das oscilações posturais a partir dos sistemas sensoriais: visual, vestibular e somatosensorial. O sistema vestibular fornece informações relativas à posição da cabeça em relação à gravidade e ao movimento linear e rotatório da cabeça. O sistema somatosensorial fornece informações sobre a posição e movimento dos segmentos corporais. O sistema visual fornece informações sobre a posição do corpo em relação ao meio ambiente.

O controle postural é a habilidade de manter o equilíbrio no campo gravitacional. Esse equilíbrio é obtido pela manutenção do centro de gravidade corporal sobre a base de suporte (Horak, 1987; Massion, 1992; Gurfinkel, et al., 1995). Nessa condição, todas as forças agindo no corpo estão balanceadas, de forma que o centro de gravidade é controlado em relação à base de suporte, tanto em uma posição estática como em uma dinâmica (Pedotti, et al.,1989).

A eficiência desse controle depende de uma complexa interação do sistema neural e músculo-esquelético com as forças internas e externas que atuam no corpo. Os componentes do sistema neural englobam o processamento motor de toda atividade neural proveniente do sistema nervoso. Os componentes do sistema músculo-esquelético incluem as propriedades viscoelásticas dos músculos e ligamentos, a configuração anatômica dos ossos , músculos, articulações e a maquinaria contrátil.

O sistema nervoso considera todos estes componentes no controle de determinada resposta postural. Na posição bípede sobre uma superfície rígida observa-se uma pequena atividade eletromiográfica dos músculos posturais (Gurfinkel et al.,1974). Nesse caso, o Sistema Nervoso Central deixa que as propriedades viscoelásticas dos músculos e ligamentos se encarreguem da manutenção do equilíbrio estático.

Durante a posição neutra, sem ação de forças externas ativas, Cholewicki, et al. (1997), usando um aparelho que fixa a pelve, encontraram coativação entre os músculos paravertebrais e reto abdominal em indivíduos normais. Mas em sujeitos com lombalgia crônica essa coativação poderia aumentar e esse aumento seria para compensar a falta de firmeza muscular "stiffness". Esse conceito de firmeza parte da teoria da estabilidade espinhal apresentada por Panabi (1992), sobre as causas da lombalgia, a estabilidade espinhal é obtida através da função coordenada de três sub-sistemas, o sub-sistema passivo (estruturas osteoarticulares), o sub-sistema ativo (músculos espinhais) e o sub-sistema neural (o controle dos músculos através do sistema nervoso central e periférico). Esses três sub-sistemas não podem atuar independentes e são considerados uma única entidade na estabilização espinhal. Um distúrbio em um dos sub-sistemas pode ser compensado por outro sub-sistema para manter a estabilidade da coluna, essa estabilidade é requisito básico para a ausência de dor na coluna.

Com a falta da firmeza o sistema neuro-muscular teria uma dificuldade maior de resistir a um desequilíbrio. Porém, o aumento exagerado da atividade muscular levaria à fadiga e à dor. Para esses autores, esse aumento dos níveis de coativação muscular poderia se constituir em um indicador objetivo da disfunção no sistema de estabilização passiva da coluna lombar.

As forças internas com as quais os sistemas neural e músculo-esquelético reagem são representadas pelo desequilíbrio postural gerado pelo movimento focal nas articulações não focais (auto-distúrbio). Para reagir a esse desequilíbrio, o sistema de controle motor deve ser capaz de prever o distúrbio e enviar uma resposta antecipatória (feedforward). Essa resposta é caracterizada pela ativação da musculatura não focal (músculos posturais), que geram uma força que previne o desequilíbrio. A resposta é enviada para a musculatura não focal, antes mesmo do início da atividade muscular focal (Aruin & Almeida, 1997).

As forças externas são representadas pelos distúrbios posturais gerados pela interação com o meio ambiente. Para o desequilíbrio postural gerado por forças externas, o sistema utiliza reações compensatórias (Oddsson, 1990). Essas reações compensatórias são disparadas pelas informações aferentes na forma de um feedback.

Baseados nesses estudos nós esperávamos observar mudanças nas estratégias utilizadas pelo Sistema Nervoso Central de um indivíduo com lombalgia crônica para reagir a um auto-distúrbio postural, isto é, causado por forças internas. O auto-distúrbio é a causa mais comum de desequilíbrio de nosso cotidiano e é provocado pelo movimento focal de uma articulação. Esse movimento gera uma força nas articulações não focais, produzindo um distúrbio postural.

Nesta dissertação apresentamos um estudo sobre o efeito dos distúrbios posturais gerados por movimentos bilaterais dos ombros com uma carga de dois quilos. Neste estudo demonstramos primeiramente que os indivíduos com lombalgia crônica não alteravam a

estratégia usada para controlar os movimentos focais do ombro. Nesta posição os membros superiores foram mantidos contra a gravidade com uma contração isométrica dos músculos do ombro. Para executar os movimentos dos ombros os indivíduos normais e com lombalgia crônica desligaram os músculos deltóide anterior e posterior. Esse mesmo padrão de atividade eletromiográfica gerou movimentos dos ombros com cinemática semelhante. Também observamos que os indivíduos com lombalgia crônica reagem aos distúrbios posturais provocados pelos movimentos dos membros superiores usando os mesmos padrões cinemáticos e eletromiográficos.

Tratamento da dor lombar

Existem diversas técnicas de tratamento para a dor lombar. Na fase inflamatória, o tratamento medicamentoso é o primeiro a ser utilizado e tem importante função no combate à inflamação e ao edema. Geralmente são utilizados antiinflamatórios não hormonais, que tratam principalmente os sintomas e geralmente são associados a outras formas de tratamento conservador.

As cirurgias são realizadas apenas quando o tratamento conservador falha. Todos os candidatos à cirurgia devem passar por profunda avaliação psiquiátrica e psicológica (Vilela Filho & Corrêa, 1988). Pacientes com transtorno do humor, caracterizado por profunda depressão, com transtornos da personalidade, caracterizados por hipocondria e histeria, devem ser desencorajados a prosseguir com a cirurgia.

O repouso é importante na fase aguda da patologia. Porém, após essa fase ele pode ser prejudicial. Longos períodos de repouso levam à fraqueza e à hipotrofia muscular, diminuindo a densidade óssea e levando à perda da flexibilidade do tecido conjuntivo (Taylor & Twomey, 2000).

Diversas técnicas também são recomendadas para tratamento da lombalgia, tais como os exercícios (Taylor & Twomey, 2000; Carpenter & Nelson, 1999; McGill, 1998), a manipulação vertebral (Herzog, Scheele & Conway, 1999; Di Fabio, 1992), a mobilização vertebral (Twomey, 1992), a abordagem ergonômica (Bullock & Bullock-Saxton, 2000), a escola de postura (Berqguist-Ullman & Larsson, 1977); o tratamento comportamental (Slater et al., 1997) e outros recursos tais como a eletroterapia, a massoterapia e a tração lombar (Werners et al., 1999).

Os exercícios são essenciais na restauração da função e bem estar do indivíduo, tendo como principal objetivo minimizar a carga sobre a coluna para reduzir o risco de exacerbação da lesão. Para evitar os mecanismos de lesão existe importante necessidade de força muscular, flexibilidade, resistência e treinamento, para que se acentuem a estabilidade da coluna vertebral. Existem diversas técnicas e métodos de exercícios para a coluna lombar. Dentre eles podemos mencionar (McGill, 1998): treinamento da capacidade funcional, marcha e exercícios aeróbicos, programas de estabilização lombar focalizando exercícios de cocontração dos músculos do tronco, programas usando uma variedade de bolas, programas direcionados aos desequilíbrios musculares, programas direcionados a uma variedade de músculos do tronco com objetivo de minimizar a carga na coluna, programas para manter o paciente sempre ativo e programas de alongamento dos músculos posteriores.

A manipulação vertebral acompanhada da mobilização é um método utilizado para tratamento das dores de coluna. A mobilização é feita lentamente, trabalhando nos limites articulares fisiológicos; já a manipulação é um movimento passivo de alta velocidade e pequena amplitude que trabalha além dos limites fisiológicos articulares. Não existe ainda uma clara explicação sobre os efeitos da manipulação na dor lombar, mas acredita-se que, na maioria das

dores lombares, existe uma má posição ou subluxação vertebral, redução da hérnia discal, liberação das aderências ao redor dos discos ou facetas articulares e desbloqueio das articulações interapofisárias, o que pode acentuar as respostas reflexas e a estimulação mecânica das fibras nociceptivas das articulações (Herzog, Scheele & Conway, 1999).

Trabalhos mostrando o efeito positivo da manipulação vertebral nas lombalgias demonstraram sérios problemas metodológicos (Di Fabio,1992), sendo que os resultados não são reproduzíveis. Também não foram encontradas diferenças entre a manipulação e a fisioterapia no tratamento da lombalgia no trabalho realizado por Cherkin et. al. (1998).

A mobilização passiva é realizada com movimentos lentos de pequena amplitude e baixo estresse. Acredita-se que a mobilização produza uma distribuição do líquido sinovial e estiramento parcial dos ligamentos e estruturas articulares, necessários para a recuperação das estruturas envolvidas. Geralmente, é associada a outras técnicas terapêuticas (Twomey, 1992).

A abordagem ergonômica possui uma função importante não só na recuperação do indivíduo com lombalgia, mas também em sua prevenção. A ergonomia busca adaptar o trabalho ao homem, procurando evitar posturas inadequadas, esforços repetitivos e mal realizados, iluminação inadequada, ruídos excessivos, vibração exagerada. Ela se baseia na análise do trabalho, educação em princípios ergonômicos, programas de instrução para novos funcionários, adaptação do equipamento e do ambiente de trabalho, mudanças na organização e métodos de trabalho, relaxamento e exercícios (Bullock & Bullock-Saxton, 2000).

A escola de postura tem como objetivo fazer com que o indivíduo tenha a habilidade de cuidar de sua própria coluna vertebral. Em um estudo controlado, Berqguist-Ullman e Larsson (1977) demonstraram redução temporária nas faltas ao trabalho após a implantação da Escola de Postura. Em primeiro lugar, ela se preocupa em orientar o indivíduo em relação à anatomia da

coluna, a sua fisiologia, a como utilizar corretamente as alavancas do corpo humano, à prática de exercícios de relaxamento, de alongamento, de fortalecimento e condicionamento, que são realizados em grupo.

O tratamento comportamental é uma abordagem que se preocupa não somente com a dor, mas na restauração da função das atividades diárias e melhora da qualidade de vida O tratamento é baseado nos seguintes componentes: educação sobre a dor nas costas e função; aumento sistemático de exercícios físicos (caminhadas e fortalecimento); contingente reforço para a melhora da função e atividade e não reforço para os comportamentos dolorosos; auto-manuseio e treinamento para resolver problemas. Existe real efetividade no tratamento comportamental, principalmente quando os sintomas estão relacionados a incapacidade e depressão (Slater et al., 1997).

Devido à natureza recorrente das dores lombares em uma proporção significante da população (Troup, 1981), a limitação dos testes de diagnósticos em identificar a origem dessas dores (Bergquist-Ullman, 1970; Waddel, 1987; Nachemson, 1985), a influência de vários fatores, a ineficácia para verificar a eficiência dos tratamentos (Deyo, 1983) e a diversidade de programas de exercícios, não existindo uma abordagem lógica para a prescrição dos exercícios terapêuticos em indivíduos com lombalgia (Richardson, Jull & Hides, 2000), podemos afirmar que ainda não existe um tratamento ideal para a lombalgia.

Os fatores causadores dessa deficiência não estão sendo considerados nas investigações ou nos tratamentos nas disfunções crônicas lombares, por isso a prevenção é um fator muito importante. Porém, mesmo assim, o que não falta no mercado é a propagação de técnicas ditas como terapêuticas. Um dos possíveis candidatos para a causa da lombalgia pode ser um déficit na estratégia de ajustes posturais, o que será o objetivo específico de nosso trabalho.

II. TRABALHO A SER SUBMETIDO À PUBLICAÇÃO

Os dados obtidos durante o desenvolvimento desta tese foram organizados em um artigo a ser submetido à publicação.

Strategy of Motor Control used by chronic low back pain individuals to avoid self inflicted postural disturbance

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ABSTRACT

Study Design. This study analyzed the strategy of motor control used by two groups of subjects to avoid self-inflicted postural disturbance. Group I (n = 8) included individuals who did not have any history of low back pain, and Group II (n = 8) which included individuals with chronic low back pain.

Objectives. To determine if here are differences in the strategy of motor control in subjects with chronic low back pain to avoid self inflicted disturbance.

Summary of Background Data. There are not any studies in the available literature about self-inflicted disturbance with individuals having chronic low back pain.

Methods. The subjects were asked to lift a bar weighting two kilograms, and hold overhead moving towards a target within 90° of extension, standing on a force platform at the upper right position. The movement of the shoulder, hip, knee and ankle joints, and EMG activity and the displacement of the center of pressure were registered.

Results. Subjects with and without low back pain showed similar strategies. At the beginning of the task they showed only focal muscles EMG activity of the anterior and posterior deltoid. When the movement of the shoulders occurred toward the target, postural disturbance occurred, as well, causing many changes in, the cinematic of the shoulder, hip, knee and ankle, and, also in the EMG activities of focal and non-focal joints.

Conclusions. The results showed, that the Central Nervous System of the subjects with low back pain reacts to a self-inflicted disturbance similarly to the of subjects without chronic low back pain.

Key words. Low back pain, kinematic, EMG, postural disturbance

Introduction

The low back pain is the most common causes of disability, producing a large social and economic burden on society.[13,44] The idea that low back pain is provoked by the degree of loading is still a debatable. Some will argue that its incidence does not appear to depend simply on the degree of loading, since it does not increase necessarily in occupations that require stronger muscle force.[9] However, in one meta-analysis study, the National Institute for Occupational Safety and Health (NIOSHI)[29] reported a relationship between loading and low back pain. The etiology of acute low back dysfunction generally is associated mechanic factors. This is in contrast to chronic low back pain, where even after many episodes of incapacitation it may be impossible to determine conclusively the cause of the pain.[26] Many factors have been suggested as determinants of whether a person becomes a sufferer of chronic low back pain such as psychological,[25,38,14] socioeconomical,[28,41] vocational,[38] and physiological [2,6,7, 24, 31,36] factors.

Probably the chronicity of low back pain has multifactorial origin, even though it has been traditionally viewed as a collection of musculoskeletal disorders. This view, however, has not led to the development of useful or widely agreed upon diagnostic criteria for low back dysfunction[26] or to very effective treatment protocols.[10]

During episodes of low back dysfunction there is a paucity of vertebral movement and the quality of these movements appear to be altered. Persons with chronic low back dysfunction are thought to have less amplitude of movement even during non-painful periods than persons without low back conditions.[4,6,7] However, the other studies showed that the movements of the persons with low back pain history are ample and fast during the

beginning of the extension while rising from a forward flexed position.[24] This strategy may have been resulting or contributing in the recurrent low back pain. During the forward bending, those persons tend to move the spine before then the hip.[12] These authors said that an inadequate flexibility of hip muscle, associated with a large low back motion, results in low back pain.

The recurrent nature of low back pain in a significant proportion of the population,[41] and the ongoing inability of diagnostic tests to find pathology in many of these persons,[5] suggest that some causative factors are not being considered in investigations and treatment of chronic low back dysfunction.

In particular, we tested the hypothesis that in addition of the signals of the history of low back pain, these individuals would also have problems that would irradiate to other joints. In order to test this hypothesis we asked both with and without low back pain individuals to perform fast bi-lateral loaded shoulder movements as fast as possible from overhead position.

In a sense these movements could be characterized as unconstrained single-joint. Studies[15,17,18] about pointing single-joint movement has been characterized by triphasic burts of muscle activities identified by sequential agonist-antagonist-agonist EMG burts of muscle activities. The first EMG burst of the agonist muscle moved the limb towards the target and, after some latency the antagonist muscle was activated to decelerate the limb towards the target. Finally, the second EMG bust of the agonist muscle locked the limb at the target position. All these studies [1,15,17,18] were restricted to unloaded single-joint movements performed from relaxed position.

So, in the first part of this study we address the question of how the load would affect the motor control strategy used by neurological normal individuals to perform bi-lateral fast shoulder movements. We than used this normative data to study possible change or adaptation in the motor control strategy as a function of the history of low back pain. We did not find any change in the pattern of modulation of the shoulder muscles and in the shoulder kinematic that could be related with chronic low back pain.

In the second part of this study, we analyzed the possibility that the focal bi-lateral loaded movements of the shoulder would change the motor strategy used by the individuals with low back pain to react to a postural disturbance. The postural disturbance could be divided into self-inflicted[3] and environment-induced disturbance[11,16,20], such as those provoked by platform perturbation. The self-inflicted disturbance is provoked by the reaction forces generated by the focal voluntary movement on the non-focal (postural) joints.

We have not found any study in the available literature about self-inflicted disturbance with individuals with chronic low back pain. However, the self-inflicted disturbance is the most common form of postural imbalance that we have to deal with on the everyday.[3,21]

Cholewicki et. al. (1997)[8] studied individuals on a neutral position without external loads, using one apparatus that restrained pelvis motion. They found the EMG coactivation of the paraspinal and recto abdominis muscles on subjects without low back pain. Based on this data it was hypothesized that individuals with low back pain would have increased coactivation of the muscles to compensate for loss of stiffness in the spinal column as

proposed initially by Panjabi [31]. However, the increased co-activation would predispose this individuals to fatigue and pain. This idea could also be supported by other studies showing a change in the muscle fibers composition in individuals with low back pain LBP.[35]

Based on the hypothesis that individuals with low back pain would have increased co-activation, we would expect to observe changes and adaptation in the postural strategy used by these individuals to react to a self-inflicted perturbation.

In the special we expected to observe a decreased postural sway on the ankle, knee and hip joints due to an increased muscle co-activation in individuals with low back pain. Contrary to this hypothesis we showed that both individuals with and without low back pain used similar kinematics and muscle activities response.

Methods

Subjects. Sixteen subjects participated in this study. The subjects were divided into two groups based on presence or absence of chronic low back pain. Group I did not have any history of low back pain or any other neurological or orthopedic disorders. Group II had history of low back pain for at least 6 months or more, and did not have apparent limitations of motion at the major joints and any other complain of neurological or orthopedic disorders in addition to the history of low back pain. A student t-test did not reveal group differences for age ($t=0.898$. $p= 0.400$), height ($t=0.361$ $p= 0.729$), and weight ($t= -1.077$ $p= 0.317$). These results are presented in Table 1. All subjects gave informed consensus according to the procedures approved by the Ethics Committee of the Medical Sciences of the University of Campinas.

Insert here Table 1

Instrumentation. The subjects stood on a biomechanical platform (AMTI OR-6) that measures the ground reaction. Two marks shaped like a foot were drawn on the platform surface. The marks were fixed on the top of the platform 12 centimeters a part, and each one 6 centimeters away from the center of the platform (figure 1). The subjects were asked to stand easy on the top of these marks with one foot inside one mark, in such a way that their weight were equally distributed in the antero-posterior and in the lateral medial direction in relation to the center of the platform.

Insert here figure 1

We recorded the reaction force (Fx, Fy and Fz) and the moments of force (Mx, My and Mz) in three orthogonal directions. The force platform data were collecting at 1000 Hz; amplified (X4000) and a filtered band pass (10-1050 Hz). Horizontal displacements of the center of pressure (CP) in anterior- posterior direction were calculated using the following equation:

Center of Pressure = My/Fz,

Which My is the moment of force into the antero posterior direction and Fz is the force measured along the gravitational direction.

A set of infrared emitting diodes was taped to bony landmarks near the shoulder, hip, knee, ankle, elbow, wrist and head of V metatarsal. These LEDs were fixed on the left side of the subject's body. The displacement of the marks were recorded by the Optotrak 3020

(Northern Digital Inc), three-dimensional, optoeletric motion analysis system sampling at 200 Hz. Using the X and Y coordinates of these marks we calculated the angular displacements of the shoulder, hip, knee and ankle joints. The angles of the shoulder joint were derived in order to get the angular velocity.

EMGs were recorded from 8 muscles. The signals were detected by bipolar surface electrodes of 1-cm diameter (Delsys); amplified 2000x by a filtered band-pass 45-450 Hz recorded at 1000 Hz of frequencies placed over the muscle bellies. The recorded muscles were the anterior deltoid (AD), posterior deltoid (PD), recto abdominis (RA), multifidus (MF), rectus femoris (RF), biceps femoris (BF), lateralis gastrocnemius (LG), and tibialis anterior (TA). The signal were rectified and smoothed before analysis.

Procedure. The subjects held a bar made of wood (70 centimeters of length by 3X2 centimeters of width) with 2 kilograms of weight fixed in its center. The bar was hold with the two hands, one in each extremity of the bar. At the initial position the subjects were asked to hold the bar and elevate it up and forward (overhead). At this initial position the shoulders were kept at 130° of flexion (measured at the counterclockwise direction from the orthostatic position, with the upper-arm parallel to the trunk), elbow joint in full extension (upper-arm aligned to the forearm), and the wrist at neutral position. The angles of the shoulder, elbow and wrist at the initial position were measured with a manual goniometer. At the initial position the subjects also stood on a force platform at the upper right position. From this initial position the subjects were asked to perform 90° of extension movements (from upper to down position) using simultaneously both shoulder joints. These movements were performed "as fast as possible", and accuracy and reaction

time were not stressed. The targets at the initial and final positions were set using 15 centimeter stickers by 2 centimeter of radius made of soft plastic that could easily bend when touched. These targets were fixed in a metal bar and perpendicularly positioned to the bar hold by the subjects, in such a way that the wood bar could touch the target keeping the elbow and wrist joints at the angles defined for the initial position (figure 1). Before data recording the subject tried three times to get familiar with the task, after that they performed movements that were recorded for further analysis. On average, the subjects had 10 movements and 10 seconds of interval between each movement.

Data analysis All kinematics (ankle, knee, hip and shoulder), Kinetic (Center of pressure) and EMG data (muscle activities of anterior deltoid, posterior deltoid, rectus femoris, multifidus, rectus femoris, biceps femoris, lateralis gastrocnemius and anterior tibialis) were plot on the computer monitor for each movement. Using the cursor, we identified on line the start point of the velocity, its peak and end for each trial. Every movement was divided into 3 phases based on the three landmarks of the shoulder velocity profile (figure 2C). The First Phase (I) was the interval of 200 milliseconds just before the time of the beginning of the velocity (from line 1 to line 2). The Second Phase (II) started at the end of the Phase I, and extended until the time of peak velocity (from line 2 to line 3). The Third Phase (III) stated at the end of the Phase II and ended at the time when the velocity returned to zero (from line 3 to line 4). For the time interval of each of these three phases we integrated the EMG muscle of all eight recorded muscles. We also integrated the muscle activities of each muscle from 50 to 150 milliseconds, after the beginning of the data record to establish the base line for each muscle at the initial position. Before data analysis, the

values of EMG muscle activities of the eight-recorded muscles were normalized by dividing the muscle activity of each of these muscles, obtained at each of the three phases, by its base line value calculated at the initial position. We also calculated the difference between the minimum and maximum angular excursion and the linear displacement of the center of pressure during movement time (from the beginning to the end of the shoulder velocity). The angular excursion was calculated for the shoulder, hip, knee and ankle joints. The maximum peak velocity of the shoulder was also calculated. Group differences were assessed using analysis of variance (ANOVA), and variations of Student's t-test. Before the statistical analysis we tested the homogeneity of the sample and the sample were considered homogeneous.

Results

Figure 2 illustrates how one neurologically normal (NN) and one subject with low back pain (LBP) performed the task. Both subjects were able to perform the task very well, moving the shoulder close to the required target distance, even though the movement was larger to the LBP subject. The shoulder movement velocity of both subjects (figure 2C) was characterized by a bell-shape profile, and its peak was a little bit faster for the LBP subject, compared to the NN subject. The focal shoulder movement generated a self-inflicted postural disturbance at the hip, knee and ankle joints affecting the displacement of the center of pressure.

Insert here figure 2

This self-inflicted postural disturbance varied with the shoulder movement phase. During the first movement phase (before the onset of the focal shoulder movement), the anterior and the posterior deltoids were initially activated isometrically to keep the upper-arms at the initial over and forehead position (figure 2D). During this phase, the EMG activities of the postural muscles (rectus abdominis, multifidus, rectus femoris, biceps femoris, tibialis anterior, and lateralis gastrocnemius) were at the basal level (figure 2F, H, J). At this time interval the hip, knee and ankle joints were also stationed (figure 2E, G, I).

During the second shoulder movement phase (from the beginning of the shoulder movement to the time of its peak velocity) the centers of pressure of both subjects were displayed anteriorly around two centimeters. This center of pressure anterior displacement occurred mainly due to shoulder motion. As the shoulder moved downward it displayed the center of mass anteriorly (figure 2B). Notice that during this second phase the hip, knee and ankle also were kept almost stationed (figure 2E, G, I). The peak of the anterior displacement of the center of pressure occurred when the shoulder joint of both subjects was closed to 90° (upper limb parallel to the floor). At this position the effect of gravity was most pronounced on the upper-limb.

As the shoulder joint started to move downward[1], the anterior and posterior deltoid were turned off until the end of this second phase (time of the shoulder peak velocity). Note that the LBP subject failed to turn off the anterior deltoid and posterior deltoid completely figure 2D). During this second movement phase the rectus abdominis and the rectus

[1] In one pilot experiment we observed that upper limb moved downward due to the action of the pectoralis major and the latissimus dorsi. Also, the force of gravity forced the limb into downward position.

femoris were activated (figure 2F, H). The activation of the rectus femoris seems to have acted mainly extending the knee joint, whereas the activation of the rectus abdominis during this movement phase could have avoided the hyperextension of the hip. The multifidus was activated to fix the trunk when the upper limbs moved downward.

During the third movement phase, a second burst of muscle activities was observed for the anterior and posterior deltoid muscles for both subjects. Note that during this third phase the shoulder was decelerating towards the target. These second EMG bursts were characterized by an eccentric contraction and were responsible to decelerate and hold the upper-limb at the target position against gravity (figure 2D).

During this third shoulder movement phase the hip moved into flexion. This hip movement would bring the center of pressure anterioly. However, the center of pressure of both subjects moved into the posterior direction during this third phase. This posterior displacement of the center of pressure could be attributed to a translational displacement of the pelvic girdle into the posterior direction. This displacement was measured by the linear displacement of the LED placed on the hip (see figure 3). Note that the pelvic girdle of both subjects was translated around 5 centimeters into the posterior direction (backward). Thus, even though the hip joint moved into flexion bringing the trunk anteriorly the whole pelvic girdle moved backward displacing the center of pressure posteriorly. That is, the anterior displacement of the trunk and upper-limb were probably compensated by the posterior translation of the pelvic girdle.

During this third shoulder movement phase, the hyperextension of the knee and the plantar flexion of the ankle were very small. However, the hip moved around 10° of

flexion and was kept at this flexed position even after the end of the shoulder movement. During the third movement phase the multifidus was activated eccentrically avoiding even more flexion of the hip due to the effect of gravity on the trunk. The biceps femoris was activated to avoid the flexion of hip. The gastrocnemius is a bi-articular muscle, its activity could be related to the plantar flexion of the ankle and the flexion of the knee. Note that during this phase the knee moved from hyperextension to flexion position, and this movement could also be related to the increase of the muscle activity of the gastrocnemius (figure 2F, H, J).

Insert here figure 3

Focal movements of the shoulder joint

Both groups of subjects performed the shoulder movements with similar angular amplitude (figure 4) and velocity. The t-test did not reveal group difference for both shoulder angular excursion (t_1= -0.725 p = 0.496) and shoulder angular velocity (t_1 = 0.758 p = 0.477). Note also that the variability of the shoulder excursion and shoulder velocity as measured by the standard deviation (vertical bars) were similar between both groups.

Insert here figure 4

Non focal movements of postural joints and the displacement of the center of pressure

The self-inflicted postural disturbance had similar effect on the postural joints between both groups of subjects. The major angular displacements of the hip, knee and

ankle, for all subjects and groups, occurred mainly during the third shoulder movement phase (see also figure 2E, G, I). Because of that we are reporting just the angular displacement of three joints during the third movement phase. Between both groups, these angular displacements were on average 15° into flexion, 3° into extension, and 3° in plantar flexion, respectively for the hip, knee and ankle joints (figure 5). The t-test did not reveal group differences for the angular displacement of the hip (t_1 = -0.728 p= 0.494), knee (t_1 =0.661 p= 0.533), and ankle joints (t_1 = -0.364 p= 0.428).

Insert here Figure 5

At the first phase the shoulders were stationed and the displacement of the center of pressure were minimum and did not vary between subjects and groups. The center of pressure moved into anterior direction during the second movement phase, and posterior direction during the third movement phase (figure 2B). The ANOVA test did not reveal group difference for the displacement of the center of pressure (figure 6) (see Table 2). However, there was a main effect of movement phase, showing that the displacement of the center of pressure was larger at the posterior than to the anterior direction (F=1.18 p = 0.29). The interaction between group and movement phase was not significant.

Insert here table 2

Focal e non-focal muscle activities

The ANOVA test showed did not reveal group differences in the amount of the EMG activities of the focal (anterior deltoid, posterior deltoid) and non-focal (postural) muscles (see Table 2 and figure 6). However, as reveled by ANOVA test, the amount of

EMG activities of these muscles changed with movement phase. As compared with the first movement phase, during the second movement phase the muscle activities of the two focal muscles decreased. However, these muscles activities increased during the third phase as compared with the one observed during the second phase. Also there was no group differences for the amount of EMG activities of all postural muscles (rectus abdominis, rectus femoris, tibialis anterior, multifidus, biceps femoris, gastrocnemius lateralis) as showed by ANOVA test presented in Table 2 (figure 6). The movement phase also influenced the amount of the EMG activities of these postural muscles. Overall, the total amount of these postural muscle EMG activities increased during the second movement phase, as compared with the first movement phase. The EMG activities of the anterior postural muscles (rectus abdominis, rectus femoris, tibialis anterior) decreased during the third movement phase as compared with the second movement phase. On the other hand, as compared with the second movement phase, during the third movement phase the EMG activities of the posterior postural muscles (multifidus, biceps femoris , gastrocnemius lateralis) increased or were kept at similar level.

Inset here Figure 6

Discussion

The control of focal shoulder movements from overhead to downward position

Both Individuals with and without chronic low back pain generated fast bi-lateral shoulder movements, holding a two-kilogram bar using similar kinematic and pattern of

muscle activities (figure 4 and 6). Like many others single joint movements[15,17,18] the velocities of the focal shoulder movements were characterized by a bell shape profile (figure 2C). However, the patterns of agonist muscle activities observed in our experiment differed from what has been reported for pointing single-joint movements [15,17,18] including shoulder joint.[1] The muscle activities of these pointing single-joint movements have been characterized by a typical triphasic bursts of muscle activities identified by sequential agonist-antagonit-agonist EMG burts of muscle activities. The first EMG burst of the agonist muscle moved the limb towards the target and, after some latency the antagonist muscle was activated to decelerate the limb towards the target. Finally, the second EMG burst of the agonist muscle locked the limb at the target position.

In our experiment, the individuals started the shoulder movements from an isometric contraction of the anterior and posterior deltoids. These isometric contractions were mainly responsible to hold the upper-limb and the bar at the initial overhead position. In this sense our experiment differed from the other single-joint experiments in terms of initial state of contraction of the muscles. Usually, the movements of the single-joint experiments are performed with the muscles relaxed initial position. [15,17, 18]

In our experiment just before the beginning of the movements the EMG activities of the anterior and posterior deltoids muscles were turned-off. The deactivation of the anterior and posterior deltoids allowed the force of gravity to act in synergy with the shoulder extensor muscles (major pectoralis and latissimus dorsis) accelerating the upper-limb towards the target. Just before the upper-limb started to decelerate towards the target these two muscles were again activated generating a second EMG bursts. These second EMG

bursts were generated by an eccentric contraction. Whereas, the first isometric bursts of the two focal shoulder muscles had one agonist function, the second burst of these muscles had one antagonist function in a sense that they decelerated the upper-limb towards the target.

In summary, we showed that the type of contraction of the focal muscle and its function could change during single-joint movements depending on the type of task done. Nevertheless, despite the kind of the task we can say that the muscle activities are modulated to fulfill the mechanical demand of the task. Also, we showed that the chronic low-back pain did not affect the strategy used by the Central Nervous System to modulate the pattern of shoulder muscle activities during the performance of loaded shoulder movements from overhead to downward position.

Individuals with chronic low-back pain did not change their strategy to react to self-inflicted disturbance

The self-inflicted disturbance is the most common form of postural disturbances that we have to deal with in our everyday life. It has been showed that normal individuals react to this kind of self-disturbance using anticipatory response, characterized by changes in the background of the postural muscles EMG activities. [3,21] This anticipatory response occurred prior on the onset of the activation of the focal muscles. The focal shoulder movements were preceded by anticipatory postural responses just in the rectus abdominis. The increased EMG activities of the rectus abdominis muscles would stabilize the toraxic and abdominal cavities fixing the insertion of the pectoralis major. In this way, the anticipatory reaction in the rectus abdominis would allow the pectoralis major to develop its force

moving the upper-limb downward. However, new studies are necessary to prove this hypothesis.

In other experiment involving bi-lateral movements of the shoulder, the anticipatory response was also observed in the rectus abdominis and rectus femoris muscles. [3] Why we failed to observe the anticipatory response in the other hip muscles? Note in figure 2B that at the initial position the center of pressure was displaced one centimeter at the anterior direction. This occurred because the postural equilibrium of the individuals was charged by the elevation of the upper-limb at the overhead position kept against gravity. At this initial position, an increase on the background EMG activities at the postural muscles probably stabilized the postural joints (hip, knee, and ankle), which would increase the muscle stiffness. So, there was no need for the anticipatory responses in the other postural muscles.

The self-inflicted studies also reported a tri-phasic pattern EMG activity for the postural muscles, after the anticipatory responses. [3,21,40] We also observed a kind of agonist-antagonist-agonist bursts (triphasic pattern) of EMG activities in the postural muscles (figure 2F,H,J). However, we believe that the characterization of the postural muscle activities in a triphasic pattern is not the most appropriate form to understand the rules of these EMG activities to provide the postural adjustment. During the second shoulder movement phase, the muscle activities of all four postural muscles (rectus abdominis, multifidus, rectus femoris, biceps femoris, tibialis anterior and lateralis gastrocnemius) increased. That is, there was one co-activation of the anterior (rectus abdominis, rectus femoris and tibialis anterior) and posterior (multifidus, biceps femoris and lateralis gastrocnemius) muscles. This co-activation would stabilize the pelvis girdle, knee, and

ankle joints by increasing the stiffness of these muscles. These co-activation was enough to prevent large displacement at the hip, knee, ankle joints, and of the center of pressure due to the accelerating torque generated by the abrupt bi-lateral movements of the shoulders (figure 6).

The duration of deceleration phase (phase III) of the shoulder movement was longer than its acceleration phase (phase II) (figure 2C). So, the postural disturbance generated by the shoulder movements was prolonged during the third phase. The pattern of the EMG activities of the postural muscles during the third phase shift from the co-activation (observed during the second phase) to a reciprocal pattern. This is the EMG activities of the anterior postural muscles (rectus abdominis, rectus femoris and tibialis anterior) decreased during the third shoulder movement phase, whereas the EMG activities of the posterior postural muscles (multifidus, biceps femoris and lateralis gastrocnemius) increased (figure 6). The decreasing in the muscle EMG activities of the anterior postural muscles during the third phase would avoid to add muscle force to the gravitational force, which could force one abrupt flexing of the trunk. The posterior postural muscles were activated eccentrically, since the hip was flexed and the center of mass moving anteriorly during the third phase. These eccentric contractions of the multifidius and biceps femoris also avoided an abrupt flexion of the trunk.

The individuals with chronic low-back pain did not change this strategy to react to self-inflicted disturbance generated by bi-lateral shoulder movements. On the other hand, compared with normal individuals it has been showed that individuals with chronic low back pain has increased the co-activation of the trunk flexor and extensor muscles,[9,31] the

elevation of the coativation was to compensate the lack of stiffness. With that the neuromuscular system would have more difficulty to resist to the imbalance. However, the higher muscle activity would take to fatigue and pain.[9] In this study the individuals were tested without load and at the neutral position. We did not measure the muscle activity at the relaxed position, and the individuals were not tested without load and in standing position, it is possible that the background postural muscle activities could have been larger for the individuals with chronic low-back pain as proposed by Panjabi (1992).[31] However, the co-activation observed during the second movement phase for all postural muscles was similar between groups, and during the third movement phase both groups used a reciprocal pattern of muscle activities (figure 6). So, it is possible that the co-activation observed during the neutral position for the individuals with chronic low back pain does not have any functional effect in the ability of the Central Nervous System to react to self-inflicted postural disturbance.

Several studies showed kinematic changes in the lumbar and hip movements in individuals with low back pain.[6,7,12,24] For example, compared with normal, individuals with history of low-back pain move the lumbar spine during early forward bending and had a significantly lower lumbar-to-hip flexion ratio during middle forward bending.[12] In our experiment we did not dissociate the hip and the lumbar movements and it is possible that later hip movements could have compensated one early forward bending during the self-inflicted perturbation. However, based on the postural EMG muscle activities between both individuals we would not expect to observe early forward bending of the lumbar spine. So, our findings support the observation such the degree of lumbar mobility could not be

related to history of low back pain.[7] Nevertheless, we cannot make any direct comparison between the angular displacement of the postural joints reported in our experiment (figure 2E, G, I) with voluntary movements involving total bending of the trunk.[7,12] The movements at the postural joints (hip, knee, and ankle) were induced by the self-inflicted disturbance of the upper-limb, and were very small.

An increased fatigability of the erector spinae muscles has been reported as good indicator of the development of the low back dysfunction.[30,36,37] The muscle fatigability has been measured using the rate of declination in the median frequency of the surface EMG power spectrum.[23] In our experiment the individuals performed ten trials of shoulder movements, which were not enough to produce any observed signal of fatigue in the EMG activities of the muscles.

Rose and Rothstein[35] suggested that one cause of low back pain was an inappropriate mix of muscle fiber types in the back muscles. These back muscles would be unable to generate adequate tension under various demand conditions. They believed that because of an inadequate supply of fatigue resistant, rapidly contracting fatigue resistant or rapidly contracting fiber, depending on the demands of the load, persons would be unable to either generate quickly, or maintain tension. The functional demands of the task would place the low back at risk. In our study individuals with chronic low back pain showed the same pattern of EMG activity and the inappropriate mix of fiber types did not alter the kinematic of the postural joints and the function of the spine was preserved.

The low back pain does not change the strategy to control the movements. The individual with chronic low back pain does not change the strategy of the Central Nervous System to react to a self-inflicted disturbance.

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FIGURE LEGENDS

Figure1

Experimental setup for study of postural adjustment on the platform of force

Figure 2

Temporal series of the kinematics of shoulder, hip, knee and ankle, displacement of center of pressure and EMG activities of the Anterior Deltoid, Posterior Deltoid, Rectus Abdominis, Multifidus, Rectus Femoris, Biceps Femoris, Tibialis Anterior and Gastrocnemius Lateralis of the a normal subject without low back pain (continue lines) and a subject with history chronic low back pain (broken lines). On the left side: shoulder excursion, shoulder velocity and the phases of analysis, hip excursion, and knee excursion and ankle excursion. On the right side: Displacement of the Center of Pressure, EMG muscles activities of Anterior Deltoid and Posterior Deltoid, Rectus Abdominis and Multifidus, Rectus Femoris and Biceps Femoris, Tibialis Anterior and Gastrocnemius Lateralis. The numbers (1 to 4) represent 200 ms before the beginning of the movement, 2 vertical broken lines beginning of the movement, 3 peak of velocity and 4 final from the movement.

Figure 3

The displacement of the hip landmark in the X axis of the one subject without low back pain (continuos lines) and one subject with chronic low back pain (broken lines). The negative value means displacement in posterior direction.

Figure 4

Means of the shoulder excursion and shoulder velocity of all trials of the subjects.

Figure 5

Means of the displacement of the center of pressure, excursions of the hip, knee and ankle joint of all trials of the subjects.

Figure 6

Normalized EMG muscles activities of the Anterior Deltoid, Rectus Abdominis, Rectus Femoris and Tibialis Anterior (anterior muscles on the left side), Posterior Deltoid, Multifidus, Biceps Femoris and Gastrocnemius Lateralis (posterior muscles on the right side). The muscle activity is without unit because of the normalization.

Table I

Gender, age, height and weight of the subjects.

Table II

ANOVA results ($p < 0.05$) for the displacement of center of pressure (CP), and EMG activities of the Anterior Deltoid, Posterior Deltoid, Rectus Abdominis, Multifidus, Rectus Femoris, Biceps Femoris, Tibialis Anterior and Gastrocnemius Lateralis.

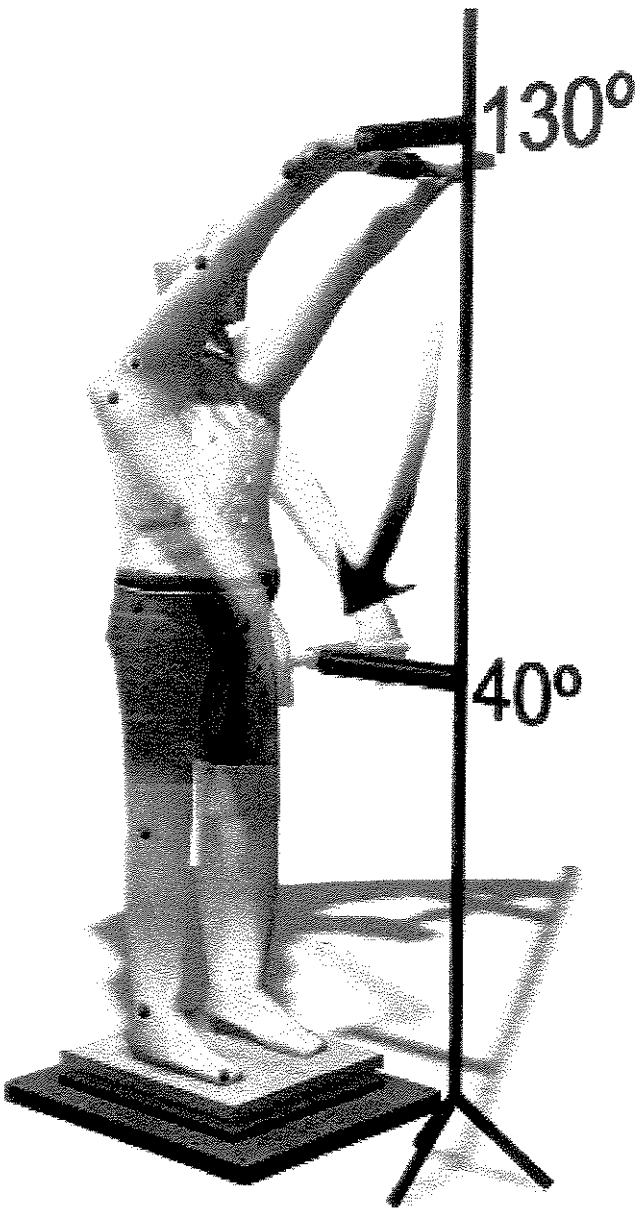

FIGURE 1

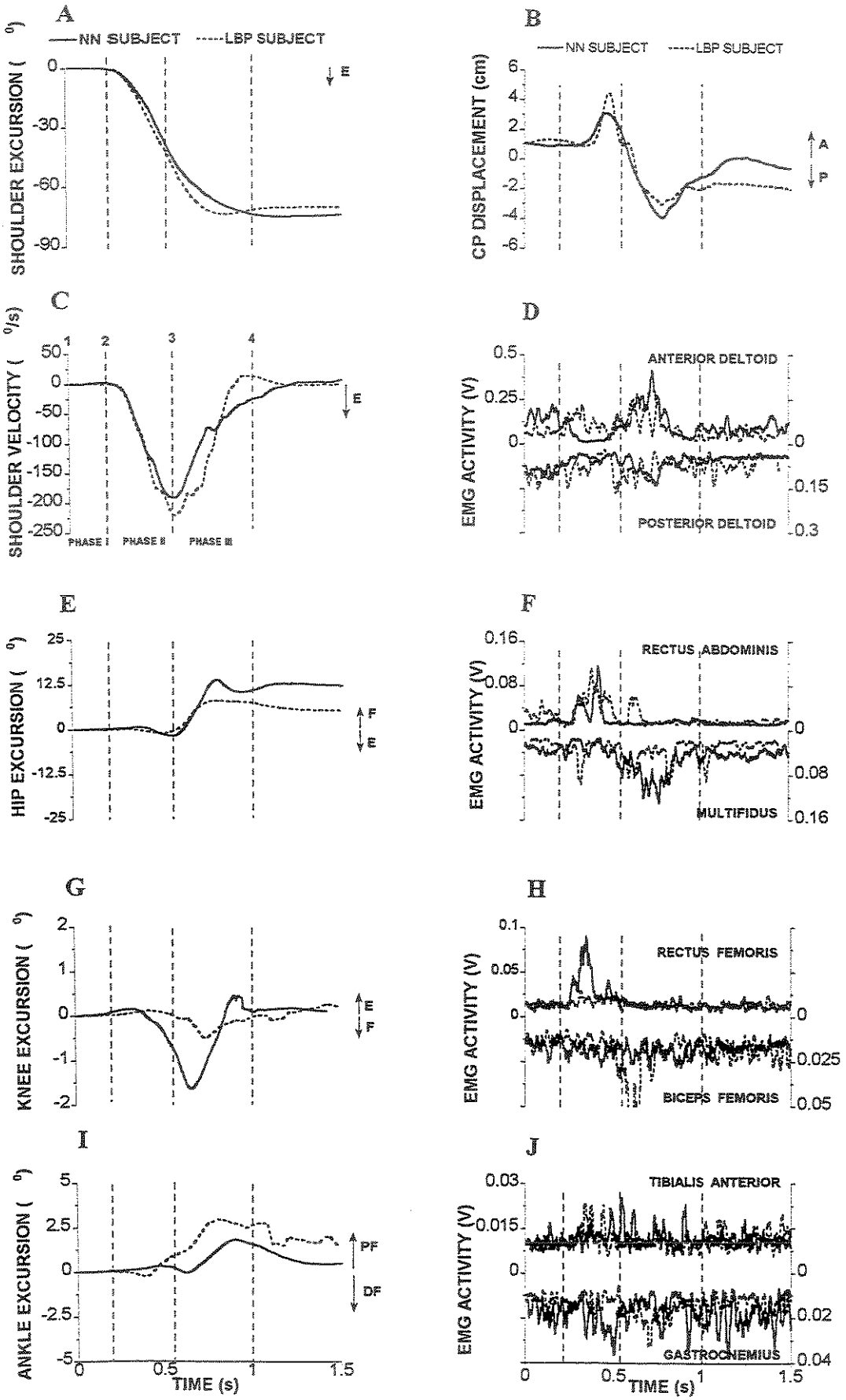
A
NN SUBJECT
LBP SUBJECT
SHOULDER EXCURSION (°)
0
-30
-60
-90
E
B
NN SUBJECT
LBP SUBJECT
CP DISPLACEMENT (cm)
6
4
2
0
-2
-4
-6
A
P
C
SHOULDER VELOCITY (°/s)
50
0
-50
-100
-150
-200
-250
1
2
3
4
E
PHASE I
PHASE II
PHASE III
D
EMG ACTIVITY (V)
0.5
0.25
0
ANTERIOR DELTOID
POSTERIOR DELTOID
0
0.15
0.3
E
HIP EXCURSION (°)
25
12.5
0
-12.5
-25
F
E
F
EMG ACTIVITY (V)
0.16
0.08
0
RECTUS ABDOMINIS
MULTIFIDUS
0
0.08
0.16
G
KNEE EXCURSION (°)
2
1
0
-1
-2
E
F
H
EMG ACTIVITY (V)
0.1
0.05
0
RECTUS FEMORIS
BICEPS FEMORIS
0
0.025
0.05
I
ANKLE EXCURSION (°)
5
2.5
0
-2.5
-5
PF
DF
0
0.5
1
1.5
TIME (s)
J
EMG ACTIVITY (V)
0.03
0.015
0
TIBIALIS ANTERIOR
GASTROCNEMIUS
0
0.02
0.04
0
0.5
1
1.5
TIME (s)

FIGURE 2

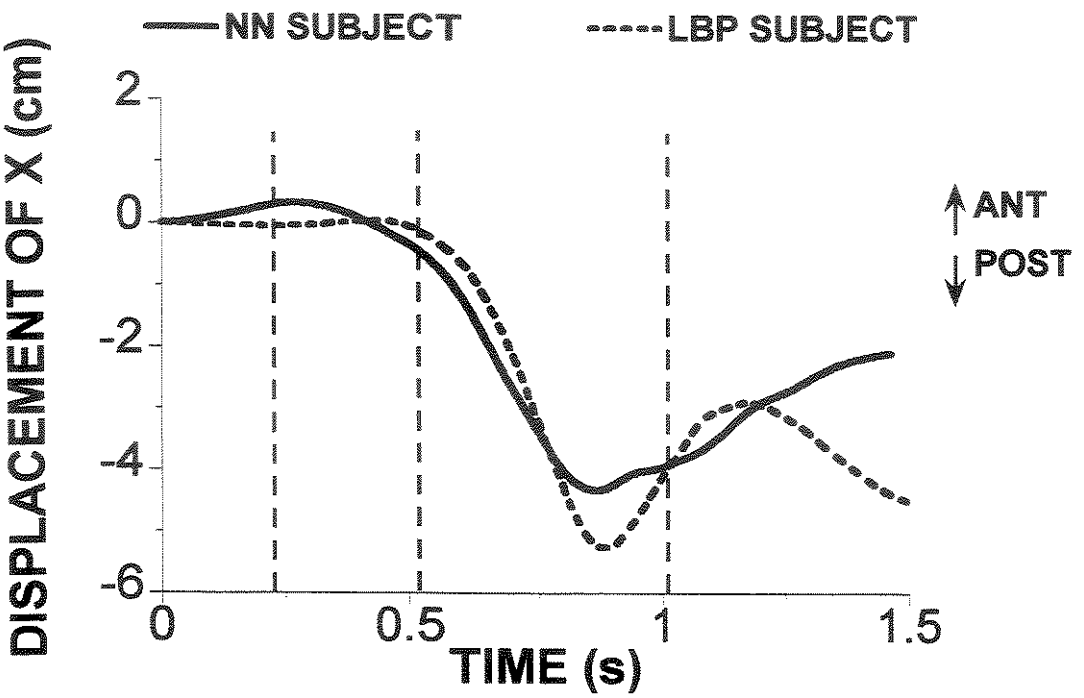
NN SUBJECT
LBP SUBJECT
DISPLACEMENT OF X (cm)
2
0
-2
-4
-6
0
0.5
1
1.5
TIME (s)
ANT
POST

FIGURE 3

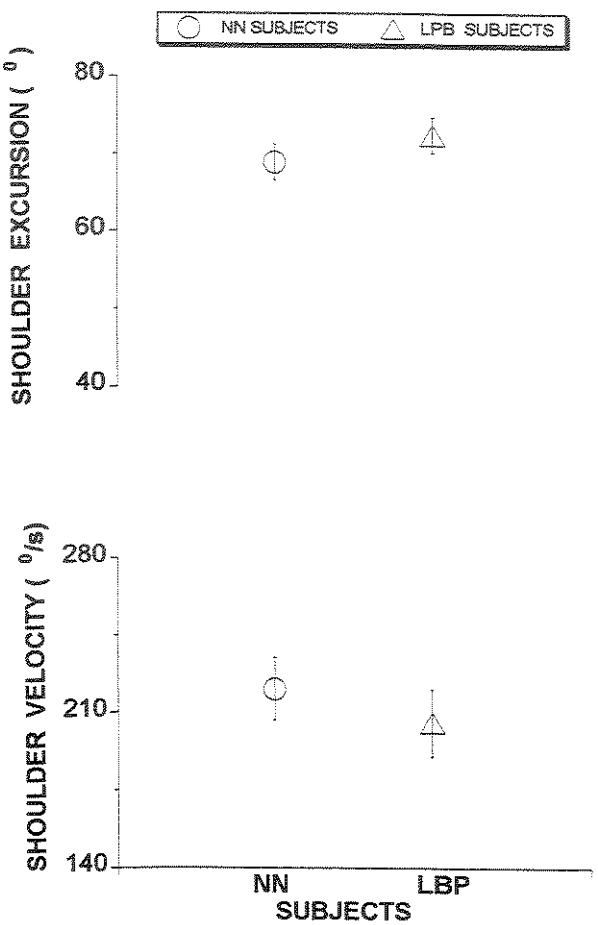
NN SUBJECTS
LPB SUBJECTS
SHOULDER EXCURSION (0)
80
60
40
SHOULDER VELOCITY (0/s)
280
210
140
NN
LBP
SUBJECTS

FIGURE 4

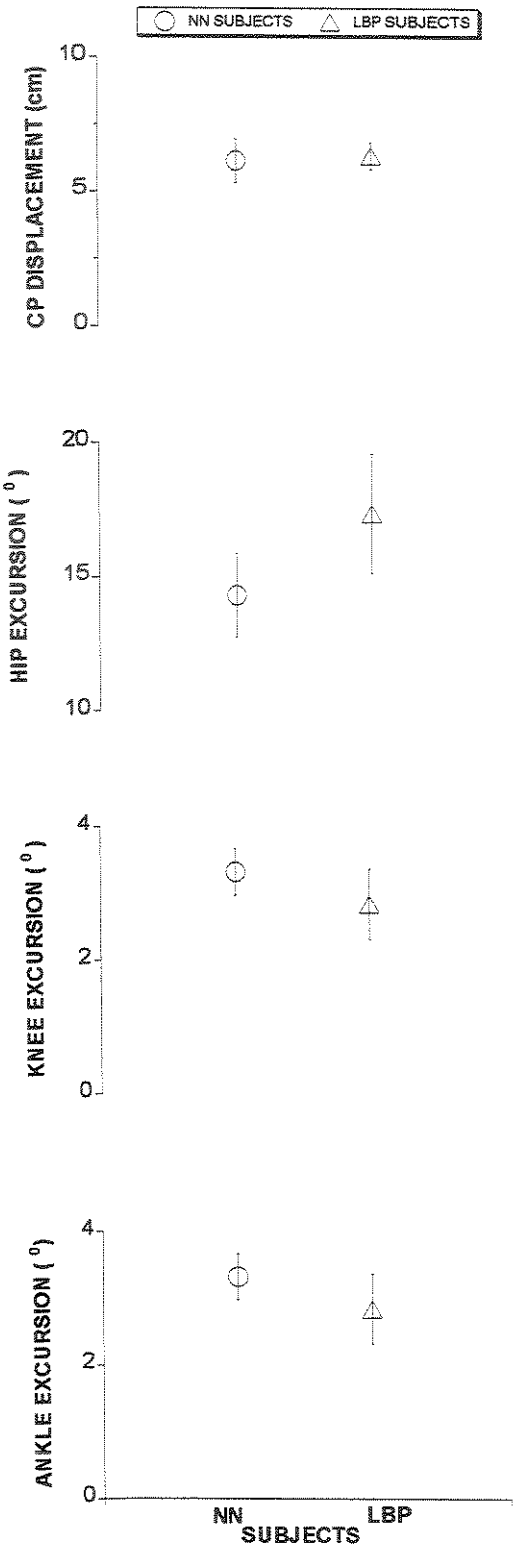
NN SUBJECTS
LBP SUBJECTS
CP DISPLACEMENT (cm)
10
5
0
HIP EXCURSION (°)
20
15
10
KNEE EXCURSION (°)
4
2
0
ANKLE EXCURSION (°)
4
2
0
NN
LBP
SUBJECTS

FIGURE 5

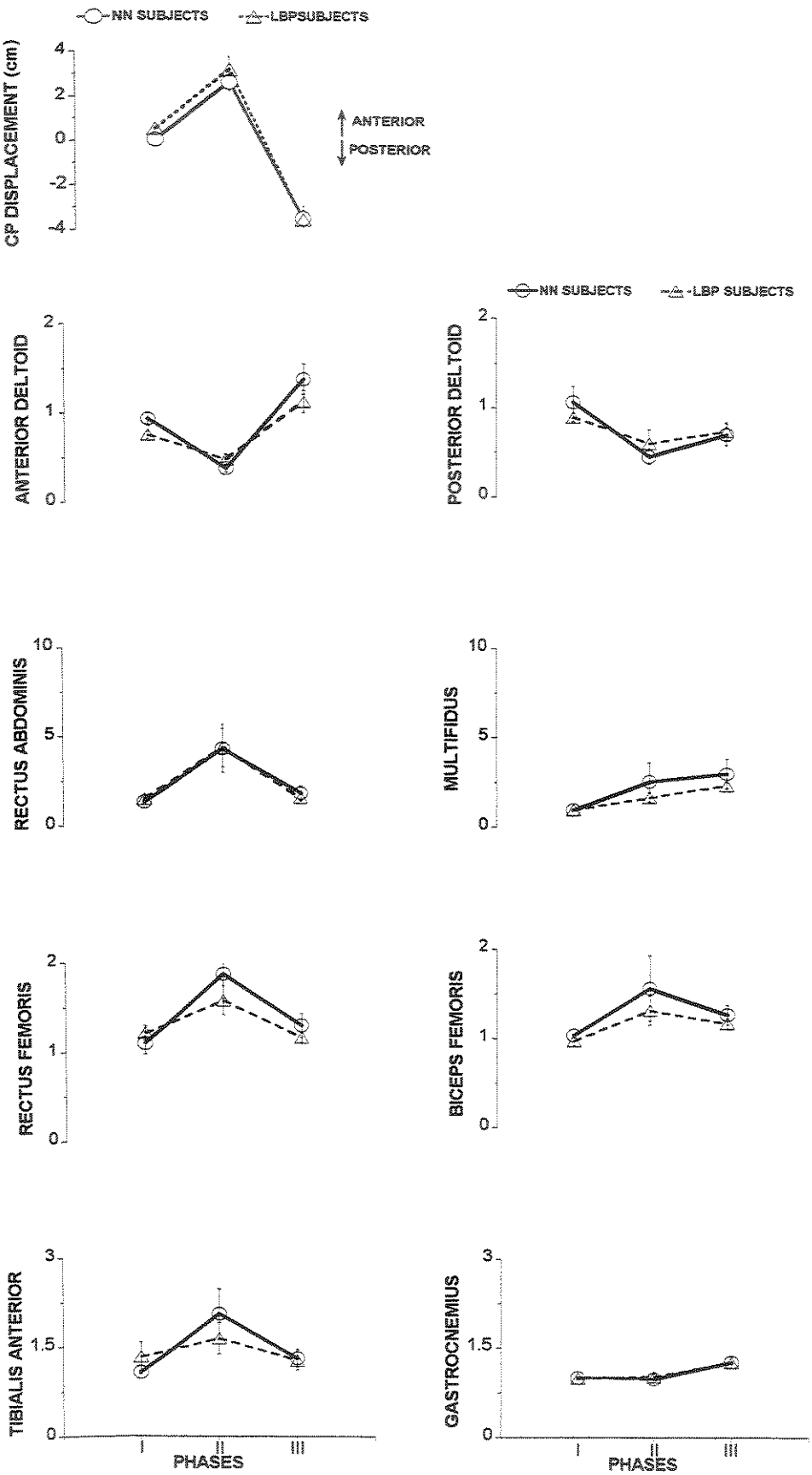
NN SUBJECTS
LBPSUBJECTS
CP DISPLACEMENT (cm)
4
2
0
-2
-4
ANTERIOR
POSTERIOR
NN SUBJECTS
LBP SUBJECTS
ANTERIOR DELTOID
POSTERIOR DELTOID
RECTUS ABDOMINIS
MULTIFIDUS
RECTUS FEMORIS
BICEPS FEMORIS
TIBIALIS ANTERIOR
GASTROCNEMIUS
PHASES
PHASES

FIGURE 6

TABLE 1

GENDER	AGE		HEIGHT (m)		WEIGHT (Kg)	
	NN	LBP	NN	LBP	NN	LBP
M	35	30	1.77	1.66	67	77
M	25	30	1.63	1.83	60	80
M	25	24	1.70	1.77	70	73
M	27	28	1.70	1.65	60	68
F	23	21	1.70	1.55	56	78
F	24	22	1.67	1.64	64	54
F	40	21	1.72	1.78	70	62
F	24	28	1.83	1.72	65	58

M: Male
F: Female

TABLE 2

	GROUP		PHASE		GROUP X PHASE	
MUSCLES	F(1,14)	p	F(2,28)	p	F(2,28)	p
CP	1.18	0.29	127.225	0.00	0.37	0.69
AD	1.51	0.24	40.10	0.00	2.08	0.14
PD	0.00	0.96	14.04	0.00	1.75	0.19
RA	4.00	0.98	12.94	0.00	0.06	0.94
MF	0.60	0.45	8.28	0.00	0.57	0.57
RF	0.40	0.53	12.68	0.00	1.37	0.37
BF	0.71	0.41	3.65	0.04	0.18	0.83
TA	0.04	0.84	8.85	0.00	0.45	0.15
GL	0.03	0.87	14.65	0.00	0.16	0.85

CP: Center of Pressure
AD: Anterior Deltoid
PD: Posterior Deltoid
RA: Rectus Abdominis
MF: Multifidus
RF: Rectus Femoris
BF: Biceps Femoris
TA: Tibialis Anterior
GL: Gastrocnemius Lateralis

GL = 7

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APRESENTAÇÕES EM CONGRESSOS CIENTÍFICOS

FISIOTERAPIA PREVENÇÃO RUMO AO III MILÊNIO

A Conquista do Profissional e a Vitória do Cliente.

13 A 17 DE OUTUBRO DE 1999

XIV CONGRESSO BRASILEIRO DE FISIOTERAPIA

VII EXPO-FISIO-BRASIL 99 * III INFOR-FISIO-BRASIL 99
I SALÃO DE PROTÓTIPOS-BAHIA 99

CERTIFICADO

CONFERIDO A

MARCOS ANTONIO FURUMOTO

POR SUA PARTICIPAÇÃO NO XIV CONGRESSO BRASILEIRO DE FISIOTERAPIA REALIZADO EM SALVADOR, BAHIA, NO PERÍODO DE 13 A 17 DE OUTUBRO DE 1999,

NA QUALIDADE DE

AUTOR DO TEMA LIVRE: ESTUDO DO CONTROLE MOTOR NAS LOMBALGIAS.

MARCOS ANTONIO FURUMOTO (UNIVERSIDADE DE RIBEIRÃO PERTO – UNAERP – RIBEIRÃO PRETO) E GIL LÚCIO ALMEIDA FT, MS, PH.D. (UNIVERSIDADE DE RIBEIRÃO PRETO – UNAERP - RIBEIRÃO PRETO/SP E UNIVERSIDADE ESTADUAL DE CAMPINAS – UNICAMP – CAMPINAS/SP)

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NA QUALIDADE DE

AUTOR DO TEMA LIVRE: DIFERENÇAS NAS ESTRATÉGIAS DE CONTROLE MOTOR NO MOVIMENTO DE EXTENSÃO DO OMBRO ENTRE ADULTOS E IDOSOS

MARIA AUXILIADORA DE OLIVEIRA RODRIGUES (PUC - CAMPINAS - UNICAMP – CAMPINAS/SP), MARCOS ANTONIO FURUMOTO (UNAERP - RIBEIRÃO PRETO E UNICAMP – CAMPINAS/SP) E GIL LÚCIO ALMEIDA (UNICAMP CAMPINAS E UNAERP - RIBEIRÃO PRETO/SP)

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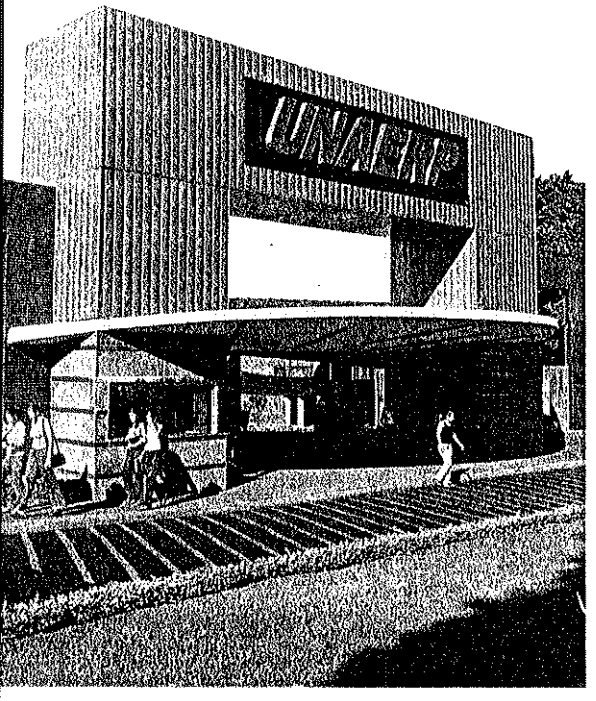

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Elmara Lúcia Bonini Corauci
Reitora

Ribeirão Preto, 08 de novembro de 2000.

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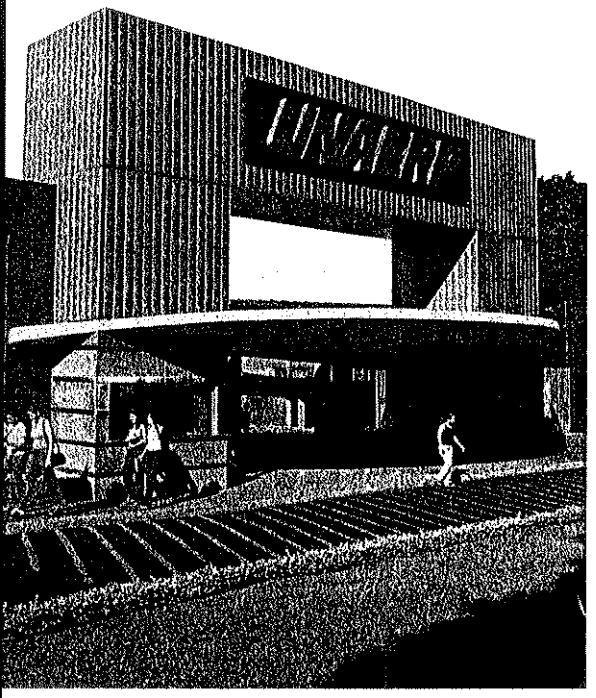

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