UNIVERSIDADE ESTADUAL DE CAMPINAS

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Controle Motor de Movimentos de Reversão em Indivíduos Neurologicamente Normais e Portadores da Síndrome de Down: O Efeito do Feedback Intrínseco

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SUMÁRIO

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Resumo

Neste trabalho, investigamos o efeito do feedback intrínseco nos padrões cinemáticos, cinéticos e eletromiográficos em movimentos de reversão do braço. Estes movimentos foram realizados em três distâncias angulares (108°, 126° e 144°) e em três orientações espaciais (180°, 90° e 0°) por indivíduos neurologicamente normais e portadores da síndrome de Down. As distâncias angulares foram definidas com base na quantidade de deslocamento do cotovelo. Durante a realização das tarefas, os indivíduos não tiveram a informação visual do braço. Para identificar a quantidade correta de deslocamento do cotovelo, os indivíduos tiveram que comparar as informações provenientes de um goniômetro fixo nesta articulação, com um conjunto de luzes, as quais acendiam com o aumento do deslocamento do cotovelo em extensão.

Indivíduos neurologicamente normais apresentaram um acoplamento entre variáveis intrínsecas (i.e. atividade eletromiográfica, torques musculares) e extrínsecas (velocidade linear do dedo indicador). Do ponto de vista do controle motor, este acoplamento simplificaria o número de variáveis que o Sistema Nervoso teria que controlar durante a execução dos movimentos. Este acoplamento entre variáveis intrínsecas e extrínsecas foi denominado como "Building Block Strategy". Além disso, indivíduos neurologicamente normais, modularam as atividades eletromiográficas e os torques musculares com o aumento da distância do movimento de acordo com as regras previstas pela "Speed Insensitive Hyphotesis" (Gottlieb, et al., 1989). Esta modulação da atividade eletromiográfica e das forças musculares foi independente da orientação espacial.

Indivíduos portadores da síndrome de Down também foram capazes de realizar a tarefa proposta; no entanto, eles apresentaram diferenças sutis na forma em que controlaram os

movimentos. Primeiro, o acoplamento entre variáveis intrínsecas e extrínsecas foi menor para esta população. Segundo, eles foram incapazes de modular as atividades eletromiográficas e os torques musculares com o aumento da distância, como observado para os indivíduos neurologicamente normais. Terceiro, estes indivíduos apresentaram um padrão muscular de co-ativação da musculatura agonista-antagonista. Dessa forma, podemos concluir que, a "Building Block Strategy" não é uma estratégia universal, a qual poderia ser aplicada a todas populações.

Com relação à precisão dos movimentos, ambos os grupos cometeram quantidades de erros similares ao realizarem movimentos de reversão utilizando apenas o feedback intrínseco. Este achado poderia sugerir que o Sistema Nervoso não utiliza este tipo de informação para controlar os movimentos.

Abstract

In this study, we investigated the effect of the intrinsic feedback in the kinematic, kinetic and, eletromyographic (EMG) patterns in reversal movements of the arm. This movements were performed in three angular distances (108°, 126° and 144°) and in three spatial orientations (180°, 90° and 0°), by normal (NN) and Down Syndrome (DS) individuals. The angular distances were defined based in the quantity of the elbow excursion. During the performance of the tasks, the subjects did not have the visual information of the arm. To identify the correct quantity of the elbow excursion, the subjects had to associate the information provided from the goniometer placed on their joint with a visual information from ligths, which turned on with the increase of the elbow excursion.

Normal individuals showed a coupling between intrinsic (i. e. muscle activity, muscle torque) and extrinsic (linear velocity of the fingertip) variables. From the motor control point of view, this coupling reduces the number of the variables that the Central Nervous System (CNS) had to control during the execution of movements. This coupling between intrinsic and extrinsic variables was denominated here “Building Block Strategy”. However, normal individuals modulated the EMG activities and the muscle torques with an increase in the angular distance, according to the “Speed Insensitive Hyphotesis” (Gottlieb *et al*, 1989).

Down syndrome individuals also were able to perfom the proposed task, however, they showed subtle differences in the manner they controled the movements. First, the coupling between intrinsic and extrinsic variables was less significant for this population. Second, they were unable to modulate the EMG activities and the muscle torques with an increase in the angular distance as observed for the NN individuals. Third, this individuals showed co-activation patterns of the agonist and antagonist muscles. In this way, we can conclude that the

conclude that the “Building Block Strategy” can not be considered an universal strategy to be applied to all populations.

Regarding the accuracy of the movements, both groups obtained similar quantities of error when performing reversal movements using just the intrinsic feedback. This found suggests that the CNS does not use this type of information to control the movements.

I. Introdução

Primeiramente iremos apresentar uma breve revisão da literatura sobre teorias de controle motor. Especificamente iremos descrever as regras descritas pela "Dual Strategy Hypothesis" (Corcos *et al,* 1989; Gottlieb *et al,* 1989a) utilizadas pelo Sistema Nervoso (SN) para o controle dos movimentos. A seguir iremos descrever a contribuição das variáveis intrínsecas (ângulo, velocidade, aceleração, torque muscular) e extrínsecas (deslocamento linear, velocidade linear) no controle motor.

Segundo Gottlieb *et al,* (1989a), estratégias são descritas como um conjunto de regras que podem ser utilizadas para explicar uma série de tarefas motoras em termos de padrões de ativação muscular. Quando realizamos uma tarefa motora simples, como por exemplo, flexão da articulação do cotovelo, a ativação muscular é caracterizada pela geração de um padrão recíproco de atividade (agonista-antagonista-agonista), o qual recebeu o nome de padrão trifásico (Hallett *et al,*1975; Hannaford, 1985).

Na tarefa citada acima, quando uma pessoa precisa mover o braço de um ponto A a um ponto B, o músculo agonista é ativado primeiramente para gerar uma força que irá lançar o membro em direção ao alvo. Neste momento, é possível identificar um primeiro "burst" de atividade muscular agonista. Cerca de algumas dezenas de milisegundos após o início desta atividade, é possível identificar um "burst" de atividade antagonista, o qual irá frear o movimento no alvo. O tempo entre o início da atividade muscular agonista e o início da atividade muscular antagonista é chamado de latência antagonista. Finalmente, é possível identificar um segundo "burst" de atividade agonista, o qual ajudará a "travar" o membro no alvo.

Sendo assim, estratégias têm sido usadas para descrever como o Sistema Nervoso Central (SNC) modularia este padrão de ativação muscular para gerar movimentos uni-articulares restritos em diferentes distâncias, velocidades e cargas ("Dual Strategy Hyphotesis") (Corcos *et al*, 1989; Gottlieb *et al*, 1989a). Estas estratégias assumem que o comando central (potencial de ação), enviado aos neurônios motores alfa na medula, poderiam ser modulados como um filtro de baixa frequência. Isto é, os neurônios motores alfa além de transmitir os impulsos nervosos efetores, também modulariam estes impulsos.

De acordo com a "Speed Insensitive Strategy" (Gottlieb *et al*, 1989a), padrões de ativação muscular agonista de movimentos realizados "o mais rápido possível" são caracterizados pela geração de um "burst" de intensidade constante para diferentes amplitudes angulares. Já a duração deste "burst" agonista aumentaria com a distância movida. Com relação a musculatura antagonista, o início de sua atividade ocorreria mais tarde para movimentos realizados em distâncias maiores.

Por outro lado, para movimentos realizados em diferentes velocidades, a estratégia utilizada pelo SN para modular a atividade eletromiográfica (EMG) agonista e antagonista mudaria. A intensidade de ativação da musculatura agonista seria maior para movimentos mais rápidos. Por outro lado, a duração do primeiro "burst" de atividade agonista permaneceria constante. Com relação à atividade antagonista, esta ocorreria mais cedo para movimentos com velocidades maiores. Esta estratégia de controle para movimentos realizados sob a demanda de diferentes urgências de velocidade angular foi denominada "Speed Sensitive Hyphotesis" (Corcos *et al*, 1989).

Esses achados nos mostram que, dependendo da instrução dada ao sujeito para a realização de uma tarefa motora, o SNC pode utilizar estratégias diferentes para controlar padrões de ativação muscular. Estes padrões parecem ser modulados de forma a garantir eficiência ao sistema de controle motor. Por exemplo, a modulação da latência antagonista para movimentos realizados em diferentes amplitudes articulares prolonga a aplicação do freio, favorecendo a geração de movimentos mais velozes para distâncias maiores.

As estratégias descritas acima foram utilizadas para explicar a geração de padrões musculares de movimentos uni-articulares de flexão do cotovelo restritos em um manipulando. Almeida et al, (1995) compararam tarefas uni-articulares de flexão do cotovelo realizados em um manipulando no plano horizontal, com tarefas uni-articulares de flexão do cotovelo e ombro, realizadas sem restrições. Estas tarefas foram executadas em diferentes distâncias angulares. Os resultados deste estudo demonstraram que para ambas as tarefas os padrões de ativação muscular foram similares aos descritos pela "Speed Insensitive Hyphotesis" (Gottlieb *et al,* 1989a). A única exeção foi para a latência antagonista do músculo deltóide posterior na tarefa não-restrita de flexão do ombro. Nesta condição, não houve a modulação da latência antagonista com o aumento da distância do movimento. Em outras palavras, as regras estabelecidas pela "Speed Insensitive Hyphotesis" (Gottlieb *et al,* 1989a) não poderiam ser usadas para explicar a modulação da atividade EMG de movimentos não-restritos realizados contra a ação da gravidade.

Em estudos mais recentes, Gottlieb (1998) comparou movimentos uni-articulares de apontar com movimentos uni-articulares de reversão do cotovelo realizados em um manipulando. O objetivo principal deste estudo foi verificar se as regras descritas previamente são específicas para movimentos uni-articulares de apontar ou se elas poderiam ser aplicadas a outros tipos de movimentos uni-articulares, como por exemplo, mover em direção a um alvo com retorno imediato à posição inicial. O raciocínio por trás deste estudo é que movimentos uni-articulares de reversão apresentariam padrões cinemáticos (ângulo, velocidade, aceleração) e eletromiográficos idênticos aos movimentos uni-articulares de apontar, pelo menos, no que diz respeito ao movimento de ida em direção ao alvo.

O padrão trifásico de ativação muscular também foi observado para movimentos uni-articulares de reversão. A novidade é que, neste caso, o primeiro "burst" de atividade agonista foi abruptamente "desligado", sendo possível distinguir dois "bursts" de atividade agonista claramente separados. Resultados similares foram observados por Schmidt *et al* (1988) e Sherwood *et al* (1988). Segundo Gottlieb (1998) este fenômeno permite que o torque flexor diminua mais rapidamente, já que o sujeito terá que retornar o membro à posição inicial e para isso ele precisará gerar um torque em direção oposta (torque extensor). As regras estabelecidas pela "Speed Insensitive Hyphotesis" (Gottlieb *et al,* 1989a) também foram capazes de descrever os padrões de ativação muscular de movimentos uni-articulares de reversão.

Resultados similares têm sido demonstrados por Almeida *et al* (2000), em movimentos multi-articulares de reversão, não restritos, envolvendo as articulações do ombro e cotovelo em diferentes orientações espaciais. O achado adicional foi que a orientação espacial influencia a quantidade de força gerada pelos músculos em relação ao total de atividade EMG.

Este conjunto de regras é baseado na idéia de que atividade eletromiográfica (EMG) e força muscular são variáveis fisiologicamente relacionadas; isto é, ambas são geradas por um pulso de excitação (potencial de ação) (Gottlieb *et al*, 1989b) e que padrões EMGs refletem a convergência, nos neurônios motores e interneurônios na medula, de comandos centrais e periféricos.

A favor desta idéia existem vários estudos (Almeida et al, 1995; Gottlieb *et al*,1996) demonstrando a existência de uma forte correlação entre atividade EMG e forças musculares geradas nas articulações (torque muscular). Como vimos anteriormente, movimentos uni-articulares são caracterizados por um padrão trifásico de atividade muscular e em parte, é esta atividade que irá gerar as forças nas articulações. Assim como a atividade muscular, os torques musculares gerados em movimentos uni-articulares, também apresentam um padrão, caracterizado por um pulso bifásico, com dois tempos de reversão. Dessa forma, o primeiro "burst" agonista, geraria o primeiro impulso do torque muscular, lançando o membro em direção ao alvo. Aproximadamente na metade do movimento, este impulso reverteria a direção, graças à ativação da musculatura antagonista. Da mesma forma, o "burst" de atividade antagonista, geraria o segundo impulso.

Todos os estudos citados anteriormente revelam comportamentos observados ao nível articular (informação intrínseca). No entanto, não podemos deixar de mencionar os estudos que acreditam que o sistema nervoso controla os movimentos a partir de variáveis extrínsecas e não intrínsecas. (Abend *et al*, 1982; Bernstein, 1967; Kaminski & Gentile, 1989; Morasso, 1981). Vários estudos têm demonstrado que o SNC utilizaria as informações extrínsecas (por exemplo, velocidade linear do dedo) e não as informações intrínsecas (por exemplo, ângulo articular) para controlar os movimentos.

Morasso (1981) estudou movimentos de alcançar envolvendo as articulações do ombro e cotovelo em diferentes orientações espaciais e alvos, sem que os sujeitos tivessem a informação visual do seu braço. Neste experimento, foi observado que, apesar das diferentes combinações de ângulo articular (informação intrínseca), a velocidade linear da mão (informação extrínseca), apresentou sempre o mesmo perfil, ou seja, um único pico. Baseado nas invariâncias da velocidade linear, Morasso (1981) propôs a "Hipótese do Controle Espacial", onde os comandos centrais para os movimentos são no sentido de especificar a trajetória da mão e não os movimentos das articulações. Este controle implicaria na existência de um mecanismo para transformar comandos motores espaciais em padrões de ângulos articulares. Conclusões similares foram feitas em outro estudo (Abend *et al*, 1982).

Como vimos no modelo da "Dual Strategy Hypothesis" (Corcos *et al*, 1989; Gottlieb *et al*, 1989a) para o controle dos movimentos, as regras são definidas a partir de parâmetros intrínsecos (i.e. padrões de atividade EMG). No entanto, invariâncias observadas tanto para parâmetros intrínsecos como extrínsecos, não respondem a questão se o SNC planeja os movimentos em termos de padrões de ativação muscular ou do deslocamento linear do membro no espaço. A verdade é que qualquer modelo de controle motor utilizado, seja intrínseco ou extrínseco, exigiria uma transformação interna do SNC (Sainburg *et al*, 1995). Por exemplo, vamos assumir que o SNC planeja os movimentos em termos de deslocamento linear do membro no espaço. Neste caso, ele teria que em algum nível determinar os padrões de atividade EMG, computar os torques e gerar o deslocamento angular nas articulações.

Baseado em estudos recentes, demonstramos que o SNC utiliza tanto as informações intrínsecas como as extrínsecas no controle dos movimentos (Almeida *et al*, 2000). Solicitamos aos indivíduos neurologicamente normais (NN) que realizassem movimentos de reversão em várias regiões do espaço. Durante a execução destes movimentos, os indivíduos tiveram informações apenas sobre o deslocamento angular do cotovelo (feedback intrínseco). Demonstramos a existência de um acoplamento entre os "bursts" de atividade EMG e os impulsos gerados nas articulações do ombro e cotovelo. Este acoplamento entre atividade EMG e torque muscular poderia ser definido como uma preferência do SNC pelo controle intrínseco dos movimentos.

No entanto, neste mesmo estudo também demonstramos existir um acoplamento entre estas atividades musculares e estes impulsos com a velocidade do deslocamento do membro no espaço. Este acoplamento foi denominado "Building Block Strategy". A idéia básica desta estratégia é que existiria uma co-variação linear entre os parâmetros intrínsecos (atividades e impulsos musculares) e extrínsecos (velocidade do dedo indicador). Do ponto de vista do planejamento do movimento, este estudo demonstrou que o SNC deve utilizar as informações extrínsecas para controlar os movimentos. Suportes à esta idéia foram obtidos com a análise da acuracidade na execução dos movimentos. A invariância na trajetória e na velocidade do dedo indicador também dá suporte à idéia de que o SNC planeja os movimentos a partir de variáveis extrínsecas.

Do ponto de vista da execução a "Building Block Strategy" simplificaria a tarefa do SN em controlar movimentos ao nível articular, ao nível da coordenação entre as articulações e ao nível da transformação intrínseca para extrínseca. A correlação linear entre estas variáveis é compatível com a sugestão de que em algum nível superior, as propriedades cinemáticas dos movimentos poderiam ser planejadas em termos experimentais, tais como aquelas fornecidas por observação visual das trajetórias.

Neste estudo utilizamos a "Building Block Strategy" para estudar os movimentos de reversão em indivíduos portadores da SD. De um modo geral, os movimentos destes indivíduos são mais lentos (Almeida *et al*, 1994; Latash & Corcos, 1991) e desengonçados (Frith & Frith, 1974) quando comparados com os dos indivíduos NN. Uma revisão de literatura dos mecanismos de controle motor utilizados pelos indivíduos SD é apresentada na seção IV.I (Almeida *et al*, 1999). De um modo geral, observamos um padrão de co-ativação generalizada da musculatura agonista e antagonista nos indivíduos portadores da SD (Almeida, 1993; Almeida *et al*, 1991; Almeida *et al*, 1994; Latash *et al*, 1993).

Estes indivíduos também demonstraram serem incapazes de modular a atividade muscular agonista e antagonista em termos dos "bursts" descritos anteriormente. Também observamos uma diminuição no acoplamento entre atividade EMG e os torques gerados nas articulações do ombro e cotovelo. A atividade EMG e as forças musculares também foram pobremente correlacionadas com a velocidade do dedo indicador. Estes dados demonstram que a origem da falta de coordenação motora nestes indivíduos pode estar relacionada à inabilidade em modular a atividade EMG de forma a gerar os torques musculares necessários à execução de movimentos com determinadas velocidades lineares.

O resultado deste estudo, incluindo uma discussão sobre a "Building Block Strategy" é apresentado na seção II, no artigo: "Building Block Strategy during reversal movements performed with intrinsic information about elbow excursion". Nesta seção, apresentamos também outro artigo a ser submetido e cujo título é: "Control of reversal movements in normal individuals and with Down syndrome: The effect of intrinsic feedback".

Na seção III, apresentamos uma discussão geral da tese envolvendo os dois artigos apresentados na seção II. Finalmente na seção IV, apresentamos os trabalhos já publicados e os resumos submetidos em congressos científicos.

II. Trabalhos a serem submetidos à publicação

Os dados parciais obtidos durante o desenvolvimento desta tese foram organizados em dois trabalhos a serem submetidos à publicação.

Building Block Strategy during reversal movements performed wih intrinsic information about elbow

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Running Title: Building Block Strategy

Summary

Neurologically normal individuals performed reversal movements over three target distances and three spatial orientations. The target distances were based on the amount of elbow excursion. During the performance of these movements the individuals did not have visual information about the moving limb. However, they could identify the amount of elbow excursion comparing the information from a goniometer, fixed at this joint, with a set of lights showed in the direction of the movement. All individuals were able to perform the reversal movements, but failed to use the intrinsic information to move the elbow joint at the required angular excursion. These results favor the idea that the Central Nervous System (CNS) did not use intrinsic information to plan movements.

At the execution level, the two agonists and the antagonist EMG bursts of muscle activities and the muscle impulses increased with target distance independently of the changing in spatial orientations. Also, there was a linear-covariance between intrinsic (muscle activities and muscle impulses) and extrinsic (linear speed of the fingertip) variables. This linear coupling between the intrinsic and extrinsic variables was termed "Building Block Strategy". This strategy facilitates the task of the CNS to control movements.

Supplementary Key Words: reversal movements, muscle activity, muscle torque, spatial orientation.

Introduction

The performance of a single-joint elbow flexion movement involves a generation of a reciprocal pattern of muscle activity (EMG) characterized by sequential bursts of agonist-antagonist-agonist activities. This reciprocal pattern of muscle activities was called triphasic pattern of muscle activity (Hallett, Shahani, & Young, 1975; Hannaford & Stark, 1985). Gottlieb, Corcos, & Agarwal (1989a); Corcos, Gottlieb, & Agarwal, (1989) described how the CNS could modulate the intensity and/or duration of the first burst of agonist EMG activity and the time between the onset of the agonist and antagonist EMG burst of activities (antagonist latency) to generate constrained single-joint movements over different distances, speeds and loads. The intensity of the first agonist EMG burst (for ballistic movements performed under the instruction to "move as fast as possible") was kept constant and its duration increased with angular distance. The antagonist latency for these movements was delayed for longer distances.

This simple-set of rules has also observed for unconstrained single-joint movements (Almeida, Hong, Corcos, & Gottlieb, 1995), and could also describe the burst of muscle activities for constrained reversal movements involving elbow single-joint. Gottlieb (1998), showed that the intensity for the first EMG burst observed for of these reversal movements was not modulated with angular distance. This first EMG burst increased with target distance and ends abruptly in a silent period, and the antagonist latency increased with target distance.

The coordination between the generation of both elbow and shoulder muscle torque could be described by the principle of linear co-variance between them. This principle states that both elbow and shoulder muscle torques are coupled on time and in magnitude during the performance of unconstrained pointing (Gottlieb, Song, Hong, Almeida, & Corcos, 1996). It can also explain the coordination between both elbow and shoulder muscle torques for movements performed over different spatial orientation and for reversal movements (Almeida, Hasan, & Corcos, 1999).

Long ago Bernstein (1935) advanced the idea that the CNS should have higher level of projections of space and not of muscle force or muscle activities. Since then, Morasso (1981) proposed the so-called "spatial control hypothesis". This hypothesis was based on observed invariance of the fingertip trace and linear velocity (extrinsic information) for planar pointing movements of the upper limb to targets at different spatial locations. For the same movements the author failed to observe invariance for the angular excursion and angular velocity (intrinsic information) of elbow and shoulder joints. The basic temporal pattern of the hand trace and its linear velocity were preserved across several handwriting movements with differents amplitudes. Supports favoring the idea that the CNS uses extrinsic information to control movement can be found in several others studies (Lashley, 1951; Russell, 1976). The idea of "spatial control hypothesis" implies the existence of a mechanism for transforming spatial motor commands into coordinated joint angular patterns (Pellionisz & Llinas, 1980; Pellionisz, 1980).

The straight hand paths are not necessary the primary concern in the control of arm movements. Indeed, Kaminski & Gentile (1989) showed some degree of curvature of hand path that could be partially attributed to difference in time of movement onset of the elbow and shoulder joint. Also optimization procedures including minimization of energy, of the jerk, stiffness and the onset between movements across different joints can generate smooth angular velocity at the joint level (intrinsic level).

This debate is more than theoretical and has a huge impact in clinical application and in sport activities. Those that work with human movement face the dilemma about what kind of information (intrinsic versus extrinsic) should provide to the performer. Also, several of the experiment reported in the literature used computer guided tasks that involve the visual transformation based on the intrinsic information. In this experiment first is showed the effect of intrinsic feedback on movement accuracy and on the pattern of muscle activation and muscle torque for planar reversal movement. Second it is also showed a linear coupling between the intrinsic (muscle torque and muscle activities) and extrinsic (speed of the fingertip) variables. Third, we called this coupling as the "Building Block Strategy". Finally, it is discussed how this strategy could be used to control reversal movements.

Methods

Subjects

Eight neurologically normal subjects (NN), 4 male and 4 female took part in this experiment after given formal consent approved by the State University of Campinas. All subjects were right-hand and were between 15 and 30 years old.

Tasks

Each subject performed reversal planar movements, involving elbow and shoulder excursion with the wrist and hand immobilized by a thermoplastic splint. The movements we divided into three tasks that varied with the spatial orientations (180°, 90° e 0°). The line in the saggital plane, crossing the middle line of the subject's body, in the antero-posterior direction, defined the spatial orientation of the 90° task. The line defining the spatial orientation for the

0° (right) and for the 180° tasks (left) was orthogonal to the line of the 90° task (figure 1). The initial position was set at the intersection between the lines of 90° task with the line of the 0° task. At the initial position, the fingertip was kept over the line of the 90° task, with the elbow joint at 90° of extension (full flexion being equal to 0°).

Insert here figure 1

The subjects were seated in an adjustable chair, with their trunks strapped in the straight back of the chair. A cable fixed at the ceiling of the experiment room sustained the proximal part of forearm. At this position their right limb could move freely around 10 cm above the top of a closed table (1.5x1x1 meter).

For each task the subject performed three series of movements defined according to one of the three elbow angular excursions. We used the following steps to define the angular target distance of the elbow joint. First, a homemade goniometer was fixed on the elbow joint. This goniometer was connected to a set of ten small lights, fixed on series in a metal bar (15 X 2 cm). For each 9° of elbow excursion in extension one light was turned on. This set of lights was put on the task spatial orientation, one meter away from the initial position, in the subject's field of vision. Second, keeping the wrist immobilized, the experimenter passively moved the subject's upper limb from the initial position to the direction of the spatial orientation line of the task. The upper limb was positioned in such a way that the fingertip laid on the line of the task with the elbow joint extended at one of the three target distances (108°, 126° and 144°, in figure 1). The elbow angular target distance was checked with simple acrylic gonimometer. Forth, at the target position, the homemade goniometer was adjusted to turn on the appropriate number of lights. The number of lights turned on were two, four, and six, respectively for the 108°, 126° and 144° elbow target distance (figure 1).

During the performance of the tasks the subjects used a glass without lenses that avoid any view of their moving limb (finger, hand, forearm and upper arm). The only information the subjects had was the number of lights turned on and the direction of the movement. So, to perform the task well the subjects first had to match the required elbow excursion at the required spatial orientation of the task with the correspondent number of lights. Thus, for each task the subject had to perform a series of five movements to a target that would require 108°, 126°, and 144° of elbow extension at the final position. The trunk line was kept orthogonal to the line of the 90° task during all experiments (figure 1).

Before data recording each subject had 5 trials of practice for each experimental condition (task and amount of elbow excursion). The subjects were instructed to move "as fast as possible" to and from the target, without stressing reaction time. They were also required to try to perform the movements "as accurately as possible". Five movement trials were recorded for each experimental condition during two seconds each one.

Recording and processing of EMG data

We recorded the EMG activity of the biceps long head, lateral head of triceps, anterior deltoid and posterior deltoid muscles using a DelSYS (model DE2.2L) EMG amplifiers with surface electrodes, with the total gain of 2000 and band pass of 20-450 Hz. All EMG data were digitilized at 1000 frames/second using Optotrak software and synchronization unit. These EMG data were rectified and filtered using 20 ms moving size window. The processed EMG data were used for the quantification purpose.

Recording and processing of kinematic data

We fixed LEDs (light emotion diode) as closely as possible to center of the shoulder, elbow and wrist joints, and on the fingertip. The X and Y coordinates of these LEDs were recording using Optotrak Motion Analysis System 3020 at 100 frames per second. From these data, the orientations of the two segments were calculated. The upper and forearm orientations with respect to the medial lateral axis (X axis in figure 1) in the horizontal plane are denoted respectively by ø1 and ø2. The elbow angle was defined as 180° minus the difference between ø2 and ø1. From angle we obtained angular velocity and acceleration. Angle, velocity and acceleration were smoothed using 10 ms moving size window. The linear velocity of the fingertip was obtained from the X and Y coordinates of the fingertip.

Using inverse dynamics to calculate muscle torque

Using the subject's weight and the regression equation of Winter (1979) we obtained the inertial parameters of the upper arm and forearm and the location of the center of mass of these segments. In addition to the measured lengths of the proximal and distal segments (L1 and L2 respectively), the following inertial parameters were estimated: the masses of the segments (m1, m2); the distances from the proximal end of the segment to its center of mass (c1, c2); and the moments of inertia about the center of mass (I1, I2). At each moment of time, and for each segment, given the current coordinates of one of the markers fixed to the segment and the orientation of the segment, the known distance of the marker from the center of mass was used to determine the coordinates of the center of mass. The center of mass coordinates are denoted by (x1, y1) for the proximal segment, and (x2, y2) for the distal segment.

The equations of motion, derived from first principles, relating the torque at each joint to kinematic variables and inertial parameters, are as follows. T1 and T2 represent, respectively, the muscle torque at the shoulder and elbow joints. Some authors refer to this as the "generalized muscle torque (moment)" (Schneider, Zernicke, Schmidt, & Hart, 1989).

Insert here the equation

In deriving these equations of motion, we have not assumed that the shoulder remains fixed in space. Note that if this assumption were made, and the center of mass coordinates were expressed in terms of joint angles by appropriate trigonometric relations, the equations of motion would be transformed into the more commonly employed form in which joint angles rather than center of mass coordinates appear as the kinematic variables.

Quantification

For the purpose of analysis all movements were divided into three phases based on the muscle torque profile. The first phase was identified as the interval between the onset and the first time the muscle torque reversed direction. The second phase was identified as the interval between the end of the first phase until the time the muscle torque reversal direction for the second time. The third phase identified was the time interval between the end of the second phase and the time the muscle torque reversal direction for the third time. The four broken vertical lines at the shoulder muscle torque (see figure 3) illustrate the three landmarks that encompass the three movement phases. These three phases were defined for both elbow and shoulder muscle torque.

EMG agonist and antagonist burst

We plotted on a monitor screen the fingertip velocity, muscle torque and the corresponding EMG activity for the agonist and antagonist muscle for each analyzed joint. Using a computer cursor we visually identified the onset of the agonist and antagonist EMG bursts for each joint. The onset was taken as the first sustained rise above the baseline. We integrated the agonist EMG activity from its onset until the first 30 ms. This activity was used to identify the intensity in which the muscle was activated. We also integrated the agonist EMG activity during two others intervals of time encompassing two agonist bursts of muscle activities. The first agonist burst was defined as the integrated muscle activity, from its onset until the first time that the muscle torque reversed direction. The second agonist burst was defined as the integrated muscle activity during the time interval between the second and the third time that muscle torque reversed direction. The bursts of the antagonist muscle activity were integrated from the onset of the first agonist muscle to the time the muscle torque reversed direction for the second time. All quantified values of the EMG bursts of muscle activities were normalized. For the normalization we divided the quantified value of the amount of EMG activity during each phase by its EMG activity during the baseline. The baseline was defined as the time interval between 200 and 300 ms before movement onset.

Muscle impulse

We integrated the muscle torque during the time of the three movement phases described above and called them as the first, the second, and the third impulses.

Elbow and shoulder excursion and movement error

We measured the elbow and shoulder angular excursion at the reversal time. We calculate the percentage of constant error using the following equation:

$$CE=(PTD-RTD)/RTD*100$$

Where CE is the constant error, PTD is the performed target distance at the reversal time, and RTD is the target distance required by the task. The amount of error was measured at the direction of the spatial orientation for each task (180°, 90° and 0°).

Data analysis

An one way ANOVA was used to test the effect of target distance for the kinematic, kinetic and EMG dependent variables for the elbow and shoulder joints. An one way ANOVA was also used to test the effect of target distance on the movement error. We ran a three way ANOVA to test the effect of target distance, and type of peaks (first versus the second), and presented the results of this ANOVA in the results session. We explored the interactions observed between and among some of the variables tested in the ANOVAs, using a Post hoc comparison based on the modified Bonferroni procedure (the overall error rate was set at 0.1). We ran a factorial analysis using the Principal Component as an extraction method with the three landmarks of the elbow and shoulder muscle torques, and with the intrinsic (the three EMG bursts and the three muscle impulses of both elbow and shoulder joints) and extrinsic (the two peak speeds of the fingertip) variables.

Results

The subjects were able to perform all tasks at the required spatial orientation

The tasks were designed to constrain the elbow joint to a specific angular excursion at the target distance. Despite this fact, all subjects were free to choose any combination of elbow and shoulder movements to perform the tasks. The subjects had (figure 2a) moved the elbow joint at required target distance (108°, 126° and 144°). As expected an Anova showed that the elbow excursion increased with target distance for 180° (F= 76.551, p=0.0001), 90° (F= 37.655, p=0.0001), and 0° (F= 62.426, p=0.0001) spatial orientations.

Insert here figure 2

Movement accuracy

We quantified the percentage of error between the performed and the required elbow excursion for each spatial orientation (see figure 2b). A repeated measured ANOVA showed that for all three spatial orientations the amount of error decreased with target distance. The main effect of target distance was (F= 3.57, p=0. 0558), (F=12.00, p=0.0009), and (F= 6.080, p=0.0126) respectively for 180°, 90°, and 0° spatial orientations. For 180° spatial orientation the averaged errors were 23.52%, 7.46%, -5.19%, respectivelly for 108°, 126° and 144° target distances, and for 90° spatial orientation these values were 60.38%, 11.35%, -3.10%. For 0° spatial orientation these mean values were 32.80%, 5.14%, -6.065%, respectively for 108°, 126° and 144° target distances.

Nevertheless, we observed some invariance in the intrinsic (joint excursion, muscle torque and EMG activity) and in the extrinsic (linear velocity and the pathway of the fingertip) variables that characterized these movements. Figure 3 illustrate the reversal movement performed by one subject over three elbow angular distance, at the 0° spatial orientation. There was an increase in the linear displacement of the fingertip with the target distance. The linear speed of fingertip was characterized by a double bell-shape with two maximum peaks (figure 3). The first peak occurred around the middle distance from the home to the target position, when the limb moved towards the target. The second peak occurred around the middle distance from the target and the home position. Also, the speed of the fingertip was higher for the longer target distance and for the first peak.

Insert here figure 3

These movements were performed with abduction of the shoulder joint and extension of the elbow joint. The excursion in both joints increased with the required elbow target distance. The angular excursions at these two joints were generated by elbow and shoulder muscle torques. These muscle torques were characterized by three impulses (see method). The first impulse thrown the limb towards the target. The second impulse decelerated the limb on the target, reversal its direction, throwing it back into the home position. The third impulse decelerated and stopped the limb at the home position.

The first bursts of the shoulder (posterior deltoid) and the elbow (triceps) agonist EMG muscle activity initially rise at similar slope for different target distances (108°, 126°, and 144°). However, the first agonist bursts were prolonged for the longer distances (see method for the definition of the two agonist and the one antagonist EMG burst). These first EMG agonist bursts ended abruptly around the time in which the muscle torque reversed direction for the first time (approximately, the time of the first linear peak velocity). After a silent period of agonist muscle activity, there was a second agonist EMG burst, which was smaller than the first agonist burst. Also, there was an antagonist burst that started after the beginning of the first agonist burst. This antagonist EMG burst ended around the time when the muscle torques reversed direction for the second time (approximately the time of the second peak linear velocity). The amount of muscle activity of both agonist EMG bursts and of the antagonist EMG burst increased with the increasing in target distance.

The generation of the three muscle impulses was coupled with the muscle activities of the three EMG bursts. The first EMG agonist bursts generated initially a muscle impulse moving the shoulder joint into abduction and the elbow joint into extension. The EMG activity of the antagonist bursts generates a muscle torque in the opposite direction of the first agonist bursts. So, the antagonist activities generated a second impulse. These impulses decelerated the limb at the target and reversed the movement direction of the two joints, throwing the limb back into the home position. The second agonist EMG burst reversed the direction of these muscle torques for the third time, generating the third muscle impulse. These third muscle impulses decelerate the limb towards the home position.

Overall, the invariance of the intrinsic and extrinsic variables described above was observed for all subjects. We will show next the quantification of the kinematic, kinetic and EMG variables for all subjects and for the three spatial task orientations.

Quantification of the EMG muscle pattern

Intensity of the agonist muscle activity

The intensity (see method) in which the first agonist burst were activated did not increase with the target distance for both elbow and shoulder muscles for all three spatial orientation tasks (figure 4). This is, the first bursts of the shoulder and the elbow agonist EMG muscle activity initially rise at similar slope for different target distances (108°, 126°, and 144°) during the first 30 milliseconds (see ANOVA in table I). The only exception was for the elbow and shoulder muscles at 180° spatial orientation.

Insert here Table I

Insert here Figure 4

Note that for 180° spatial orientation (figure 5) the onset of biceps EMG activity occurred before the onset of the triceps EMG activity, even though the elbow joint moved into extension. In single-joint elbow extension the biceps acts as antagonist. However, the biceps is a bi-articular muscle, also acting at the shoulder joint as an adductor. So, the reversal in the order of the muscle recruitment of the elbow muscle at 180° task is related with the generation of the muscle torque into adduction at the shoulder joint.

Insert here Figure 5

Amount of the EMG agonist and antagonist activity

The amount of EMG activities of the first and the second agonist (figure 4) and the antagonist EMG bursts for elbow and shoulder muscles increased with target distance. This scaling of agonist and antagonist muscle activities with increased target distance was observed for the movements performed over the three spatial orientations (see ANOVA results in table I).

Linear Peak velocity of the fingertip

We ran a three way repeated ANOVA to test the effect of the linear peak speed of the fingertip for spatial orientations (180°, 90°, and 0°), target distances (108°, 126° and 144°), and the type of the linear peak speed (first versus the second peak). The first and the second peak speed of the fingertip increased with target distance (F= 183.3 p= .0001) for all three spatial orientation (F= 64.8 p= .0001). The first peak was faster than the second one (F13.9= p= .0073). The speed of first and the second peak was greater for 0°, compared with 90° and 180° spatial orientations.

Muscle impulse

The muscle torques for both elbow and shoulder joints were characterized by three impulses and each impulse increased with target distance for the 0° and 180° tasks (figure 4). The only exception was the first shoulder muscle impulse for 90° spatial orientation that did not increased with distance (see ANOVA in table I).

Coupling between EMG muscle activity and muscle torque

The three landmarks which are the three times that the muscle torque reversed direction of both elbow and shoulder were linear related among them. The factor analysis using the Principal component as the extract method showed that all these six landmarks contributed to define the first factor, and that this factor could explain 95% of the population variance (figure 6).

Insert here Figure 6

Overall the EMG bursts of elbow and shoulder muscle activities and the three elbow and shoulder impulses increased with target distance. We also implied in the description of the figure 3 that there is a coupling among the three EMG bursts of muscle activities (two agonist and one antagonist bursts), the three muscle impulses and the two linear peak speed of the fingertip. We investigated this coupling running a factor analysis using the Principal Component as extraction method with these variables. In Figure 7 we plotted the first factor against the second factor of the unrotated matrix with the weight values for each variables (the three muscle impulses and three EMGs for both elbow and shoulder joint and the two peak of the fingertip linear speed) for the each spatial orientation.

The total variance proportions for the first and the second factor was 0.7 and 0.16 for the 0° task, 0.62 and 0.12 for the 180° task, and 0.53 and 0.22 for the 90° task. Note that all these variables contribute to define the variance proportion of the first factor, mainly the three impulses and the three EMG bursts of the elbow joint and the two peaks of the fingertip linear speed. For the 90°task the muscle impulses were very small and varied in direction (six subjects moved the shoulder initially into adduction, whereas the other two moved it into abduction). This may explain the decrease of the variance proportion for the first factor for the 90° task. The weight values were smaller for the EMG burts of the shoulder muscles.

Insert here Figure 7

Discussion

The CNS does not seems to use the intrinsic information to plan reversal movements

Traditionally the integrity of the joint static position sense is tested asking the subject to match the angle between two joints. The dynamical position sense is tested asking the subject to detect the minimal velocity threshold in which the passive movement of the joint is detected. However, during these tests, the individual could focus the attention on the extrinsic information about the limb displacement, instead of using the intrinsic information per se to perform the task. For example, when asked to move the joint at certain angle (intrinsic information), the individual could imagine where the fingertip should move in the space (extrinsic information). These tests also avoid the complexity involving multi-joint movements (i.e., interaction torques, the gravitational force and the coupling between joints).

The present results sugested that the nervous system has great difficult in using the intrinsic information to perform accurately a complex task as the reversal movements. This difficult can be observed by comparing the percentage of error obtained from similar reversal tasks using the extrinsic information about the finger tip linear displacement (extrinsic information - Almeida, Ferreira, Marconi, 2000). First, here we reported that the the error in performing the required elbow excursion was on average 14% across all tasks, compared with 8%, when the subjects had extrinsic information about the finger tip. Second, there was a great error variability among subjects when they performed the tasks with the intrinsic information about the elbow excursion. The standand error of this percentage of error across subjects was 9.4% (figure 2) for the task with the intrinsic information compared with the 3% of the task with the extrinsic information how reported in other study (Almeida, Ferreira, Marconi, 2000).

These results suggest that during complex movements the CNS cannot relay on the intrinsic information to plan fast reversal movements. The comparison of the accuracy observed in these two experiments (intrinsic versus extrinsic information) supports the idea that the CNS uses the extrinsic information (i.e., linear displacement of the limb) to plan a motor task (Abend, Bizzi, & Morasso, 1982; Bernstein, 1967; Kaminski & Gentile, 1989; Lashley, 1951; Morasso, 1981; Russell, 1976).

Like for the unconstrained single-joint movements (Almeida, et al., 1995) the angular trajectories at the individual joints were quite variable. Nevertheless, the fingertip paths were invariant with a gently curves. A double bell shaped profile also characterized the fingertip speed. Each peak speed resembled the one observed for pointing movements (Atkeson & Hollerbach, 1985; Morasso, 1981; Soechting & Lacquaniti, 1981). This is remarkable given the great dynamical complexity of the reversal movements. This invariance of the fingertip path and speed across different target distances and spatial orientations also favor the idea that the CNS plans the movement based on the extrinsic information (Morasso, 1981). The additional finding here is that at the extrinsic level the CNS does not have to plan the movements into phases (to and from the target). Note the two peak speed were linear correlated.

The modulation of the muscle activities

The strategy used by the CNS to generate and modulate the first agonist bursts of the EMG activity, for all tasks, was the same as described by the "Speed Insensitive Strategy" (Gottlieb, et al., 1989a). These first agonist bursts was generated with similar intensity, across different angular distances and spatial orientation for both elbow and shoulder muscles [1]. The total amount of EMG activity of the first agonist burst increased with target distance for all spatial orientations (see figure 4 and Table I). The modulation of the first agonist EMG burst was observed for several other movements (Almeida, et al., 1995).

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[1] The only exception was for the triceps and anterior deltoid muscles at 180° spatial orientation (see table I). For this task the elbow joint moved into extension, but the biceps long head muscles was activated before the triceps. The biceps long head crosses both shoulder and elbow joints and acted as the agonist muscle at the shoulder joint adducting it. We do not have any explanation for why the intensity in which triceps and anterior deltoid muscles was activated increased with target distance. These data showed that we have to be very careful in defining the agonist or the antagonist rules of one muscle.

Overall, the pattern of modulation of the first EMG agonist burst with distance was also observed for constrained reversal movements (Gottlieb, 1998). Like this author we also observed that the first EMG burst ends abruptly in a silent period. The additional finding here is that the modulation of the agonist burst with increased angular distance was also observed for the second agonist and for the antagonist EMG bursts. Also this modulation of the EMG muscle activity with target distance does not depend on the workspace.

However, the modulation of the amount of antagonist activity with target distance is different from what has been reported for constrained reversal movements (Gottlieb, 1998). This author showed a weak correlation of the antagonist activity with target distance for the reversal movements. We considered the antagonist EMG activity from its onset until the time of muscle torque had reversed direction for the second time (see methods). In Gottlieb's study the antagonist activity was integrated from the onset until the velocity towards the target had fallen to 5% of its first peak. However, we observed the duration of this antagonist burst to end around the second peak of the fingertip speed (time around which the muscle torque reverse direction for the second time). So, we believe that our data better represent the correct measure of the antagonist EMG activity for the reversal movements.

In the pointing movements the only function of the antagonist muscle activity is to decelerate the limb towards the target (Hallett, et al., 1975). However, in the reversal movements this antagonist burst has a double function. It first decelerates the limb into the target, and second it has to reverse its direction and bring the limb back to the home position. This double function may explain why for constrained (Gottlieb, 1998) and unconstrained (Almeida, et al., 1995) single-joint movements the antagonist EMG burst did not scale with distance, but for the reversal movement it does. For single-joint movements the antagonist muscles act just in an eccentric contraction decelerating the limb at the target position. The capability of the muscle to generate relative force at certain change of the muscle length on time is not linear when the muscle contracts eccentricaly (Gordon, 1982). Under eccentric contraction one could not observe an increasing of muscle activity with target distance, even if the decelerating muscle impulse scaled with target distances (Almeida, et al., 1995). In the reversal movements, the antagonist EMG burst first has to contract eccentricaly to decelerate the limb into the target, but then it has to continue with concentric contraction to reverse movement direction and bring the limb to the home position.

In summary, the simple-set of rules stated by the "Speed Insensitive Strategy" (Gottlieb, et al., 1989a) can be used to describe the modulation with target distance of the EMG muscle activities of both elbow and shoulder muscles. The additional finding here is that the modulation of the agonist and antagonist EMG activity with target distance did not depend on the spatial orientation of the target and did not change when the individual used intrinsic information to perform the task. Finally, note in figure 4 that the amount of muscle activities of the EMG bursts varied with changing in spatial orientation. This observation supports several others findings showing the effect of workspace on the amount of EMG activity generated by the muscles (Gabriel, 1997).

The modulation of the muscle torques

It has been showed that for pointing movements the elbow and shoulder muscle torque rise and reverse direction at the same time (Gottlieb, et al., 1996) reveling a temporal coupling between them. Here we expand this observation to show that all three landmarks of the elbow and shoulder muscle torques of the reversal movements co-varied linearly (figure 6). It has also been showed that the acceleration impulse increased with target distance for constrained (Gottlieb, et al., 1989a) and unconstrained (Almeida, et al., 1995) single-joint movements, and for pointing and reversal constrained movements (Gottlieb, 1998) Here, we expand this observation showing that all three muscle impulses of the elbow and shoulder joints increased with target distances. The modulation of these muscle impulses with target distances was not dependable on spatial orientations.

"Building Block Strategy"

It has been showed that the acceleration impulse is linearly related with the amount of the first EMG agonist burst (Gottlieb, et al., 1996). The idea behind this coupling between muscle activity and muscle impulse came from the observation that both are variables physiologically linked. The action potentials that produce the EMG activities also activate the formation of the actin-myosin cross-bridges that generates the muscle forces.

Here we advance this idea to incorporate new observations and new strategies of movement control. First, we showed that the three bursts of the muscle activities and the three muscle impulses at both joints increased with target distance (figure 4). We also showed that the modulation of both muscle activities and muscle impulses with target distance did not depend on spatial orientation. Second, we expand the idea of the coupling between muscle activities and muscle torques to show that the three muscle impulses, and the three EMG bursts of muscle activities of both elbow and shoulder joints were linearly correlated. Third, we also showed the muscle activities and the muscle torques were correlated with the two peak speeds of the fingertip (figure 7). We are calling this linear coupling between the intrinsic (muscle activities and muscle torques) and extrinsic (speed of the fingertip) variables as the

"Building Block Strategy". This strategy would facilitate the CNS task in terms of movement control by reducing the amount of variables that has to be independently controlled (Bernstein, 1967).

This simplification of the control would occur at the intrinsic and extrinsic level of movement control. At the intrinsic level, the CNS has to decide how to control the movement at each joint and how to coordinate the movement between joints. At the extrinsic level the CNS has to decide how to control the linear displacement of the limb in the space. For each joint, we are learning that the CNS does not have to deal with the whole movement extension. Since the two agonist and the one antagonist EMG bursts of muscle activities are linearly correlated, it would be enough for the motor control system to define, for example, just the first burst of the EMG agonist. The same reasoning is true for the three muscle impulses, since they were linearly related. For the coordination between two joints, we are learning that the CNS does not have to deal with movement at two joints separately. As we showed, muscle activities and muscle torques are also linearly correlated between two independent joints. It has been showed that the elbow and shoulder muscle torques are linearly coupled in time. This coupling between these two torques were termed Principle of Linear Co-variance (Gottlieb, et al., 1996). Here we expand this idea to incorporate the linear coupling among muscle impulses and muscle activities.

Our data suggested that the motor control system planned the movements at the extrinsic level. If this is the case, the CNS has to transform at some level, the extrinsic information into appropriate pattern of muscle activities and muscle torques at the joint level (intrinsic level) (Sainburg, Ghilardi, Poizner, & Ghez, 1995). The coupling between the intrinsic (muscle torque and muscle activities) and the extrinsic (speed of the fingertip) variables showed that the transformation of the extrinsic into intrinsic variables would require a simple linear transformation.

In summary, the "Building Block Strategy" would simplify the task of the CNS to control movements, at the joint level, at the level of the coordination between joints and at the extrinsic to intrinsic transformation level. The linear correlation between these variables is compatible with the suggestion that at some higher level, kinematic properties of movements may be planned in experiential terms such as those provided by visual observation of the trajectories (Wolpert, Ghahramani, & Jordan, 1994; Wolpert, Ghahramani, & Jordan, 1995a). The execution of these kinematic plans however, is in terms of the forces expected to produce them. These forces may be estimated from an internal model of the dynamic of the limbs and its external load (Shadmehr & Mussa-Ivaldi, 1994; Wolpert, Ghahramani, & Jordan, 1995b).

Limitations

One could argue that the "Building Block Strategy" is an expression of mechanical constraints represented by byproducts of the Newtonian mechanics. First, it has been showed that for simple movements performed with one unusual path, the linear co-variance between elbow and shoulder muscle is abandoned (Gottlieb, et al., 1996). Second, we have showed that the linear coupling between the intrinsic (muscle activities and muscle impulses) and extrinsic (speed of the fingertip) variables is decreased for the reversal movements performed by individuals with Down syndrome (Marconi, Almeida, & Ferreira, 2000) .

The fact that the first factor of the factorial analysis explained just 70% of the total variance population could de assumed as a limitation of the "Building Block Strategy" to predict an unified rule used by the CNS to control movements. We would like to argue the contrary. Given all technical problems related with the recording, processing and quantification of the EMG muscle activities and the fact that we recorded just a small fraction of the total muscle activities involved in the execution of the reversal movements. Given the complexity involved in the inverse dynamics to obtain the muscle torque and all its approximation, we can conclude that 70% of the total possible variance population represented a lot communality for one factor.

The coupling among these variables showed that the CNS does not have to plan each variable separately. Indeed, the coupling among these variables is stronger if we do not consider the three EMG bursts of shoulder muscle activities. The better correlation between the elbow EMG bursts of activity with the three elbow impulses could be attributed to the fact that we provide intrinsic information (angular excursion) about this joint and not about the shoulder joint. However, we do not think that this could be the case. Other muscles than the anterior and posterior deltoid (i.e., pectorals and trapezium) act at the shoulder joint. We could not rule out the possibility that the EMG burst of these other shoulder muscles could better correlate with the observed muscle torque at this joint.

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Figure Legends

Figure 1a

Illustration of the task setup. A homemade goniometer was fixed at the elbow joint and connected to a set of small lights. The subjects wore glasses without lenses that allowed the view of the set of the lights, but not of the moving limb. The movements were performed into three target distances and over three spatial orientations (180°, 90°, and 0°). The target distances were defined by the amount of elbow angular excursion as 108°, 126°, and 144°. The initial position of the upper limb was set with the shoulder joint at 45° (abduction) and elbow at 90° (extension). The limb was supported against gravity with a cable fixed at the proximal part of the forearm and connected to the ceiling.

Figure 1b

Illustration of the pathway of fingertip in the X and Y axe for all spatial orientations and all target distances. The solid line represents the movements performed at the 108°, the semi-broken line at the 126° and broken line at the 144°. The closed circles represents the required elbow angular excursion.

Figure 2

Elbow excursion (a) and the percentage of error between the required and the executed elbow angular excursion (b). The data were averaged across all eight normal subjects for each of the three target distance (108°, 126°, and 144°) over the three spatial orientations (0°, closed circle, 90°, closed squared, and 180°, closed triangle). T.D. was the expected target distance and is showed in gray diamond. The elbow excursion was given in degrees, the error in percentage. Vertical bars represent standard error and the negative values represent undershooting.

Figure 3

Time series of the extrinsic (fingertip linear velocity) and intrinsic (angular excursion, angular velocity, muscle torque at the joint, and EMG muscle activity) variables describing one movement performed over the 0° spatial orientation task. This movement was performed by one subject over the three required elbow angular excursions (108° - solid line, 126° - semi-broken line, 144° broken line). The intrinsic variables are showed for the shoulder (on the left panel) and elbow (right panel) joints. Linear velocity is given in millimeters per second, angle excursion in degrees, angular velocity in degrees per second, muscle torque in Newton meters per second, and muscle activity in volts. The shoulder muscles are the posterior deltoid (PD) and the anterior deltoid (AD), and the elbow muscles are triceps lateral head (TRIC) and biceps (BIC). The four vertical lines in the muscle torques represent the three landmarks when the muscle torques reversed direction.

Figure 4

The intensity (ITEN) and the amount of the first EMG agonist burst (F. AGO), of the antagonist EMG bursts (ANT), of the second agonist EMG bursts (S. AGO), and the muscle impulses during the three movement phases: the interval of the first (F. impulse), second (S. impulse), and third impulse (T. impulse). These parameters are showed for shoulder and elbow joints, for the three angular distances (108°, 126°, and 144°) for the three spatial orientation (180°, open circle, 90°, open squared, and 0°, open triangle). The values of EMG activities are normalized and the impulses are given in Newton meters per second.

Figure 5

Time series of the intrinsic (muscle torque at the joint, and EMG muscle activity) variables describing one movement over the 180°spatial orientation task. This movement was performed by one subject over the three required elbow angular excursions (108°- solid line, 126°- semi-broken line, 144°- broken line). These variables are showed for the shoulder (on the left panel) and elbow (right panel) joints. The muscle torque is given in Newton meter per second, and muscle activity in volts. The shoulder muscles are the posterior deltoid (PD) and the anterior deltoid (AD), and the elbow muscles are triceps lateral head (TRIC) and biceps (BIC).

Figure 6

The first (X-axis) and the second (Y-axis) factors obtained from the Factor Analysis using the Principal Component as the extraction methods. Each symbol represents one of the three landmarks of both elbow and shoulder joints: The first and the fourth landmarks are the time in which the elbow and shoulder reversed direction for the first time (open circle and open diamond). The second and fifth landmarks are the time when both elbow and shoulder muscle torque reverse direction for the second time (open triangle and open inverted triangle). The third and the sixth landmarks are the time when both elbow and shoulder muscle torque reverse direction for the third time (open square and axe).

Figure 7

The first (X-axis) and the second (Y-axis) factors obtained from the Factor Analysis using the Principal Component as the extraction methods. Each symbol represents: FPLS (the first peak of the linear speed of the fingertip - closed circle), SPLV (the second peak of the linear speed of the fingertip - open square), FISH (first impulse of the shoulder - open circle), SISH (second impulse of the shoulder - axe), TISH (third impulse of the shoulder - open square with a back slash), FASH (first agonist burst of the shoulder - closed triangle), SASH (second agonist burst of shoulder - plus signal), ANSH (EMG antagonist burst of the shoulder - inverted open triangle), FIEL (first elbow impulse - open square with a single cross), SIEL (second elbow impulse - closed square), TIEL (third elbow impulse - closed diamond), FAEL (first agonist burst of the elbow - open triangle), SAEL (second agonist burst of the elbow - closed inverted triangle), and ANEL (antagonist burst of the elbow - open diamond). The data are presented for each of the three spatial orientation (180°, 90° e 0°).

Table I

The effect of target distance on the three impulses and EMG muscle activities of both elbow and shoulder joints, tested using one way ANOVA for each of the three spatial orientation (180°, 90°, and 0°). The muscle impulses were the first, second, and the third. The EMG activities were: 1) The intensity in which the agonist EMG burst was activated, integrated during the first 30 milliseconds (INTEN). The first (integrated at the time of the first impulse) and the second (integrated at the time of the second and the third impulse) EMG agonist bursts (AGO). The antagonist EMG activity, integrated from the time of the first and the second impulse (ANT). The plus signal showed that the effect of target distance were significant at the level of $p<0.05$, and the minus signal, that these effect was not significant.

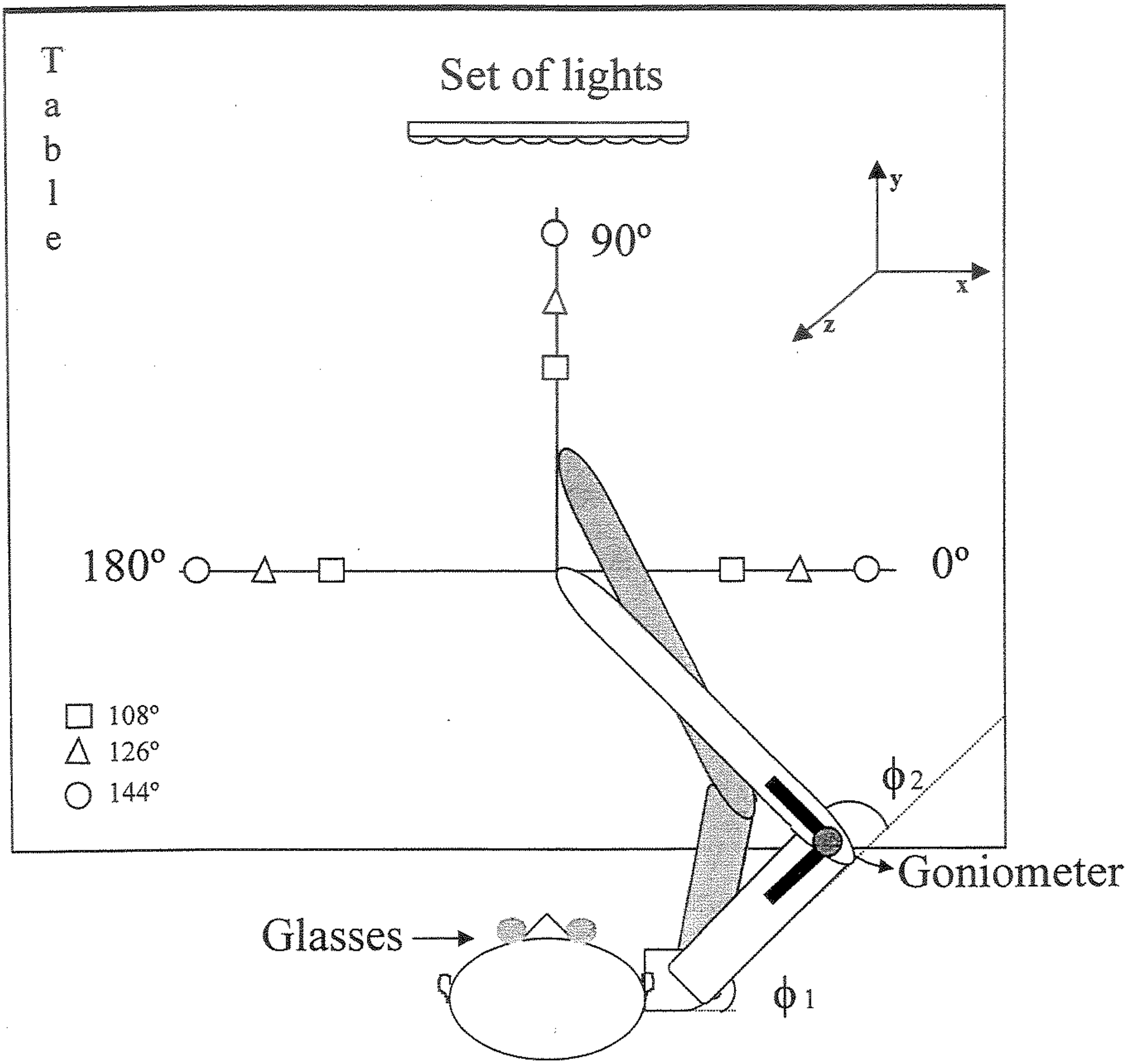

T
a
b
l
e
Set of lights
y
x
z
90°
180°
0°
□ 108°
△ 126°
○ 144°
ϕ2
Goniometer
Glasses
ϕ1

Figure 1a

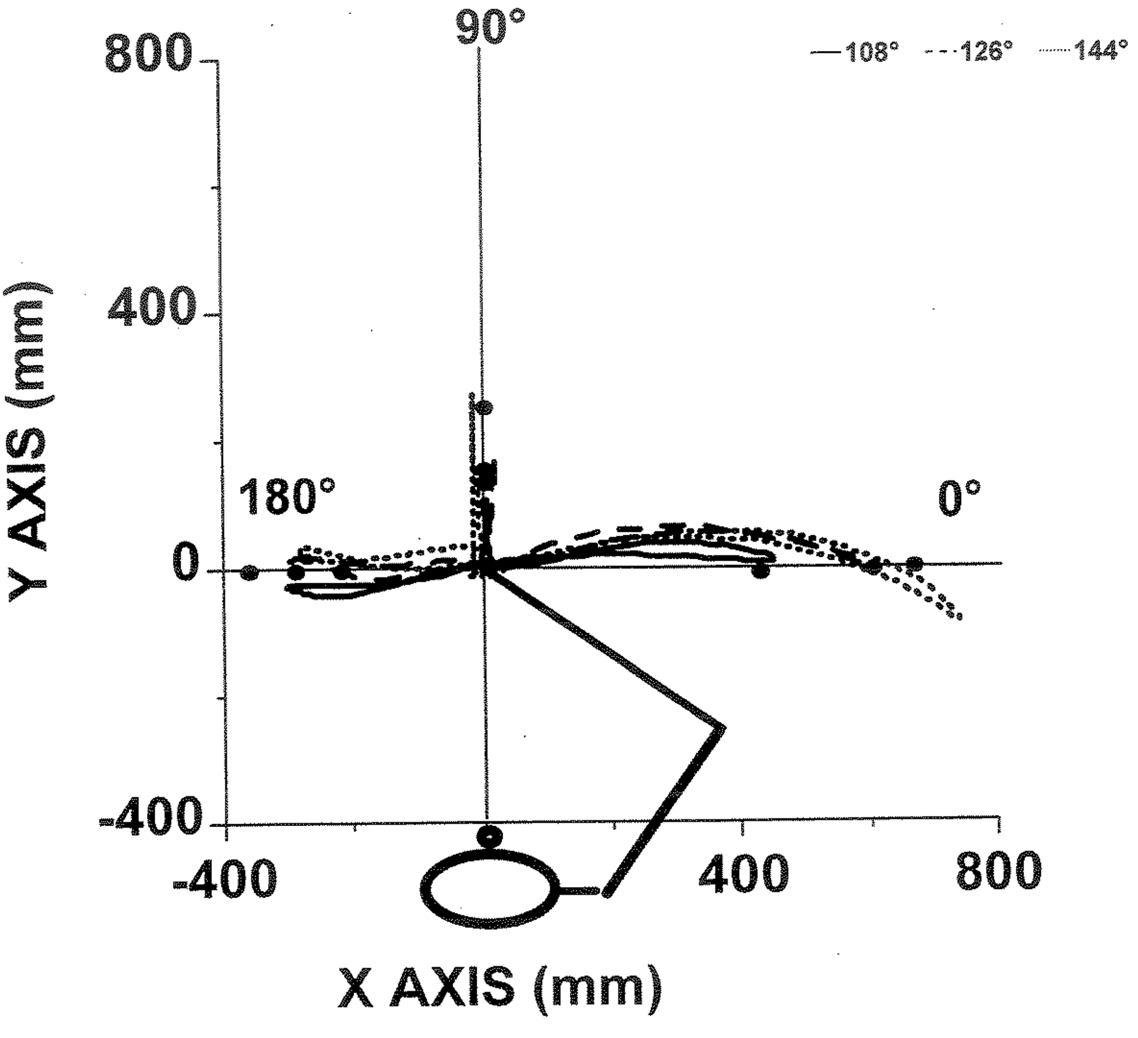


Figure 1b

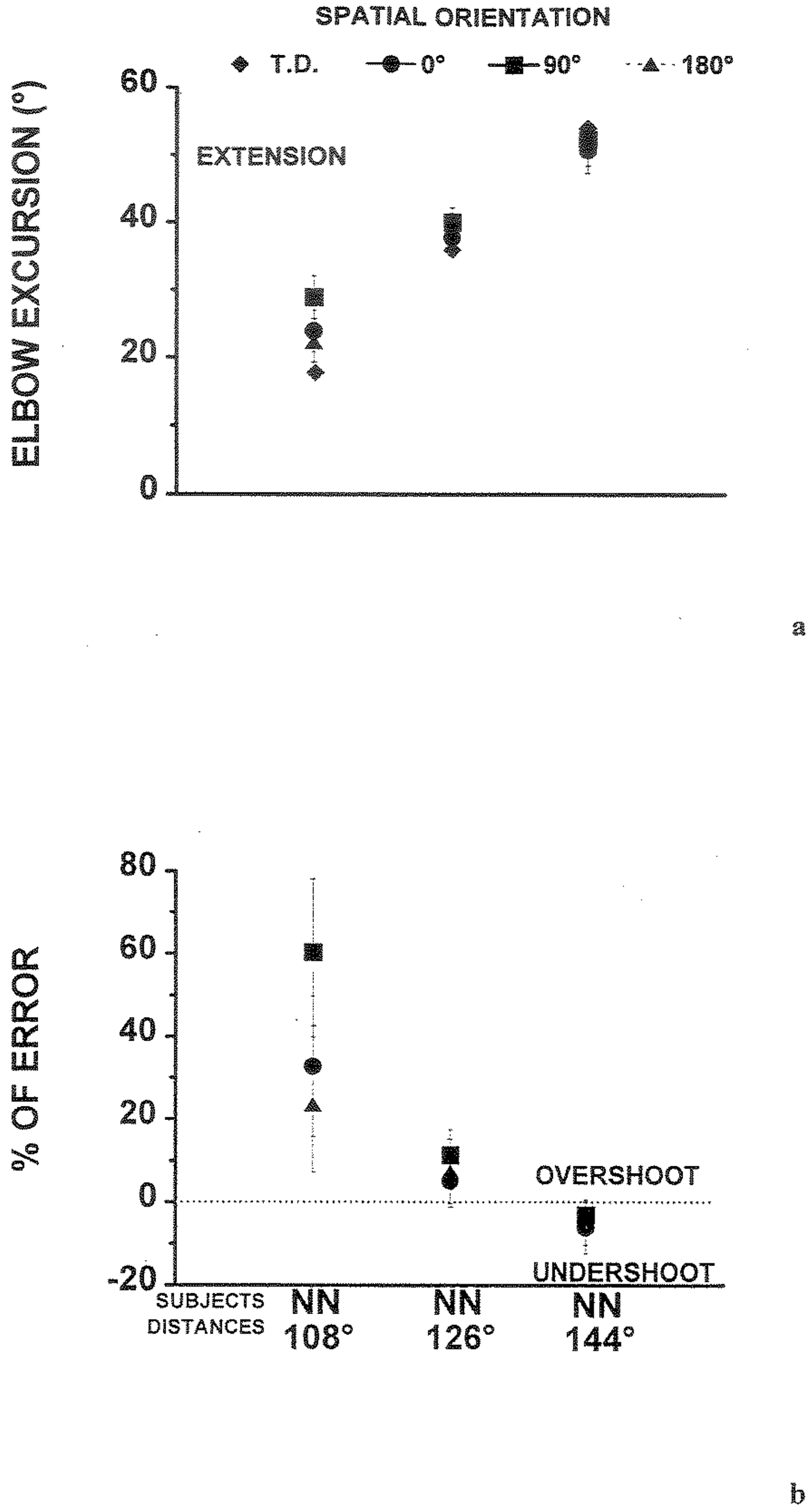

SPATIAL ORIENTATION
T.D.
0°
90°
180°
ELBOW EXCURSION (°)
60
40
20
0
EXTENSION
a
% OF ERROR
80
60
40
20
0
-20
OVERSHOOT
UNDERSHOOT
SUBJECTS
DISTANCES
NN
108°
NN
126°
NN
144°
b

Figure 2

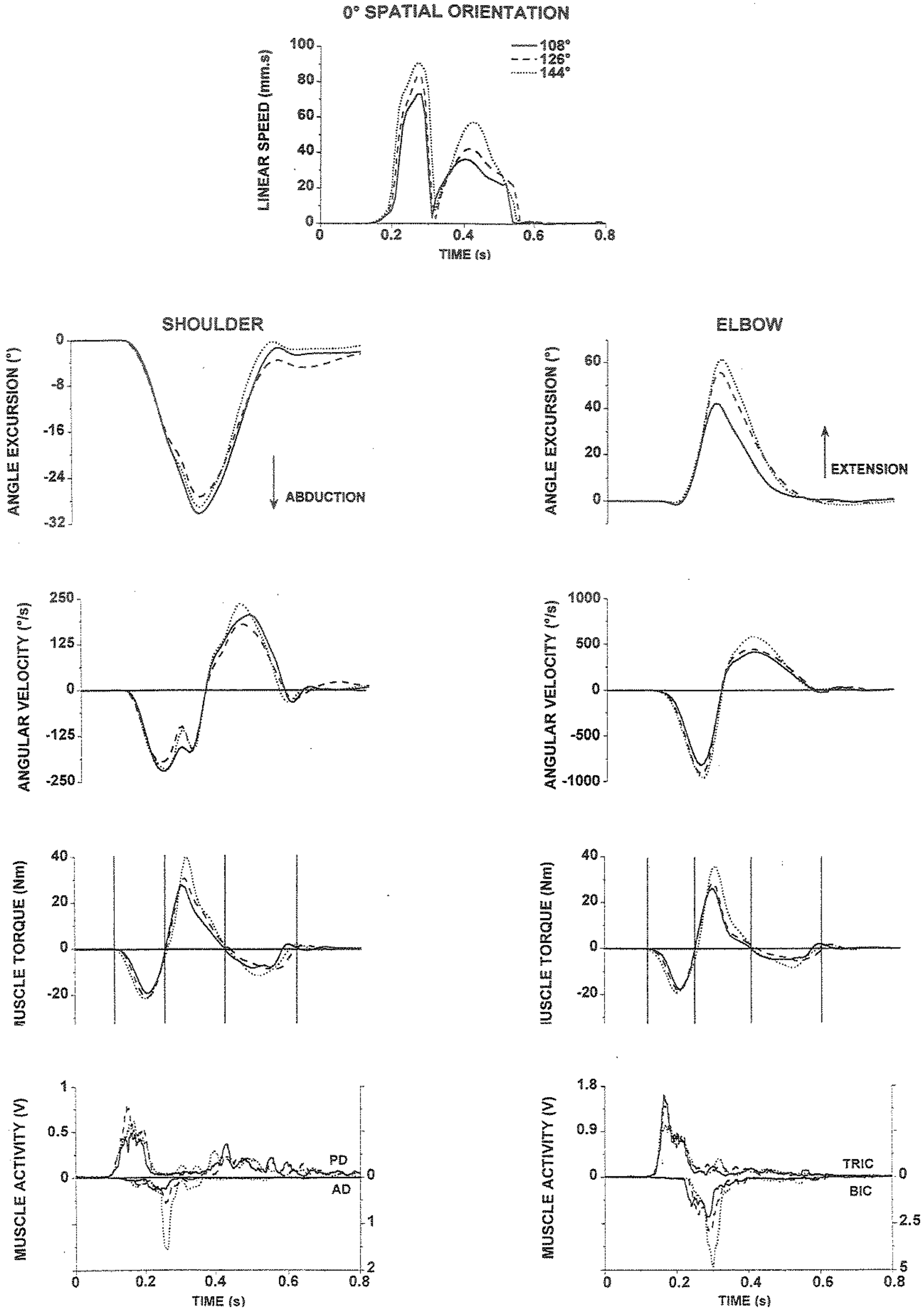

0° SPATIAL ORIENTATION
LINEAR SPEED (mm.s)
TIME (s)
108°
126°
144°
SHOULDER
ELBOW
ANGLE EXCURSION (°)
ABDUCTION
EXTENSION
ANGULAR VELOCITY (°/s)
MUSCLE TORQUE (Nm)
MUSCLE ACTIVITY (V)
PD
AD
TRIC
BIC
TIME (s)

Figure 3

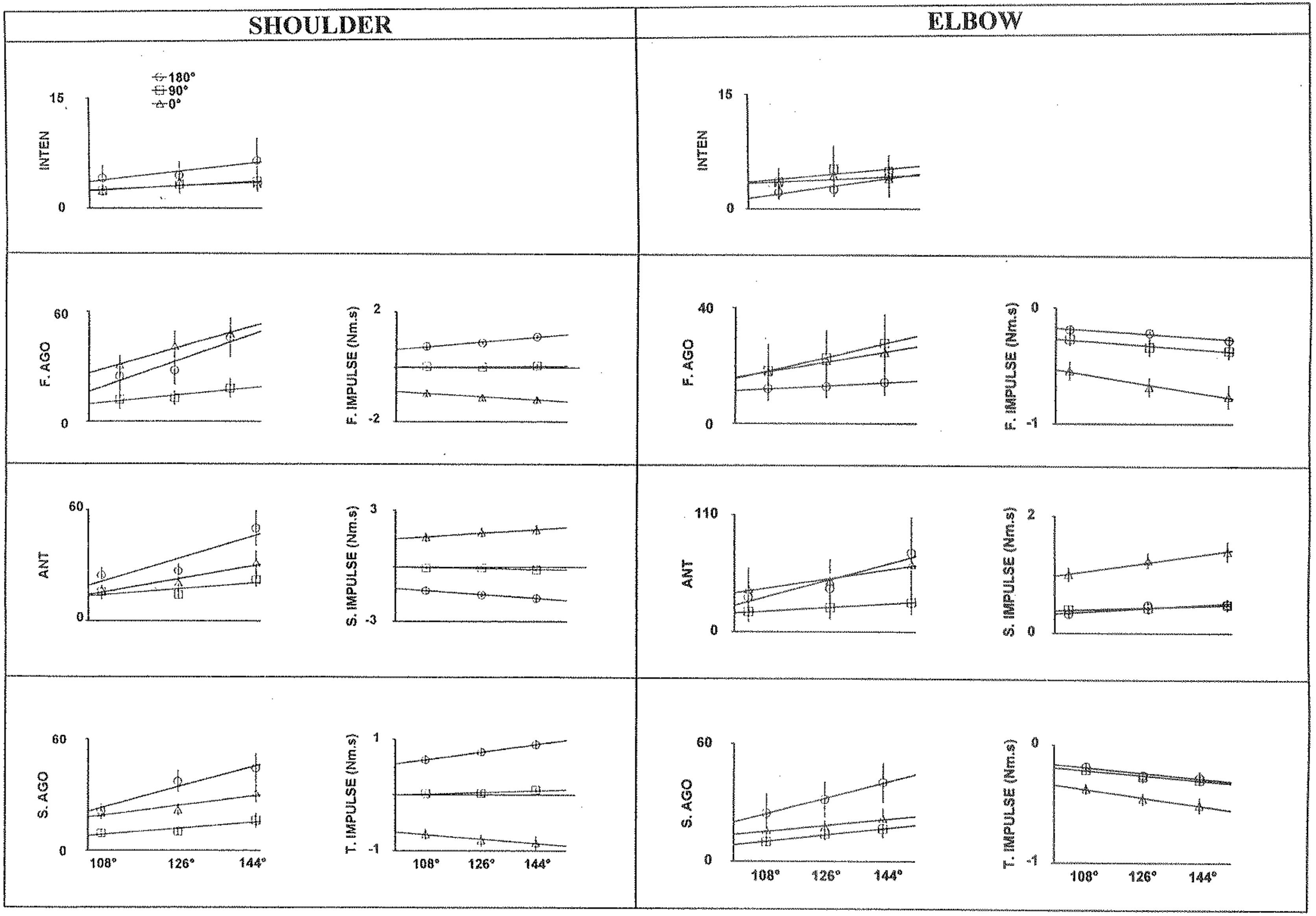

SHOULDER
ELBOW
180°
90°
0°
INTEN
15
0
F. AGO
60
0
40
F. IMPULSE (Nm.s)
2
-2
0
-1
ANT
60
0
110
S. IMPULSE (Nm.s)
3
-3
2
0
S. AGO
60
0
T. IMPULSE (Nm.s)
1
-1
0
108°
126°
144°

Figure 4

180° SPATIAL ORIENTATION

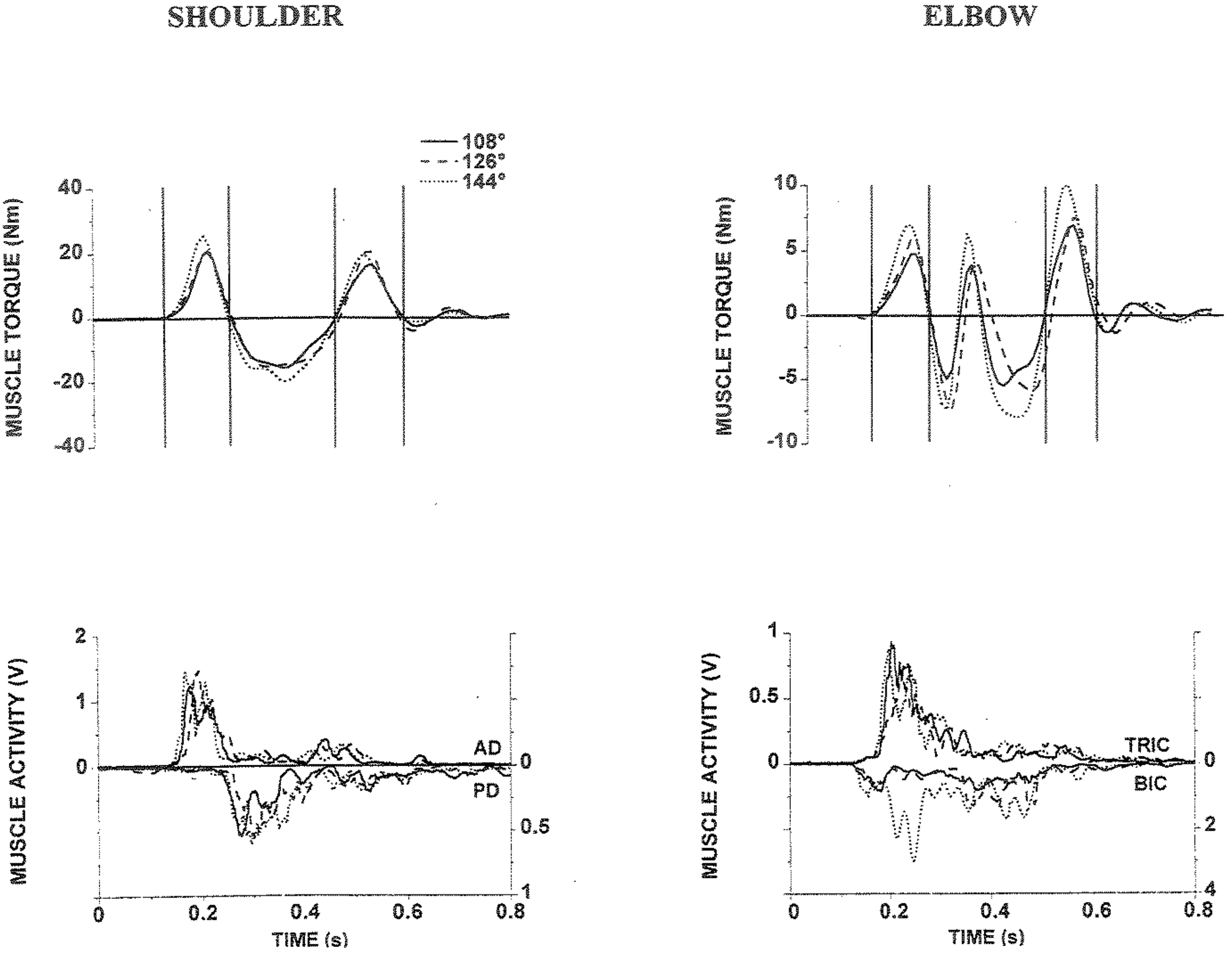


Figure 5

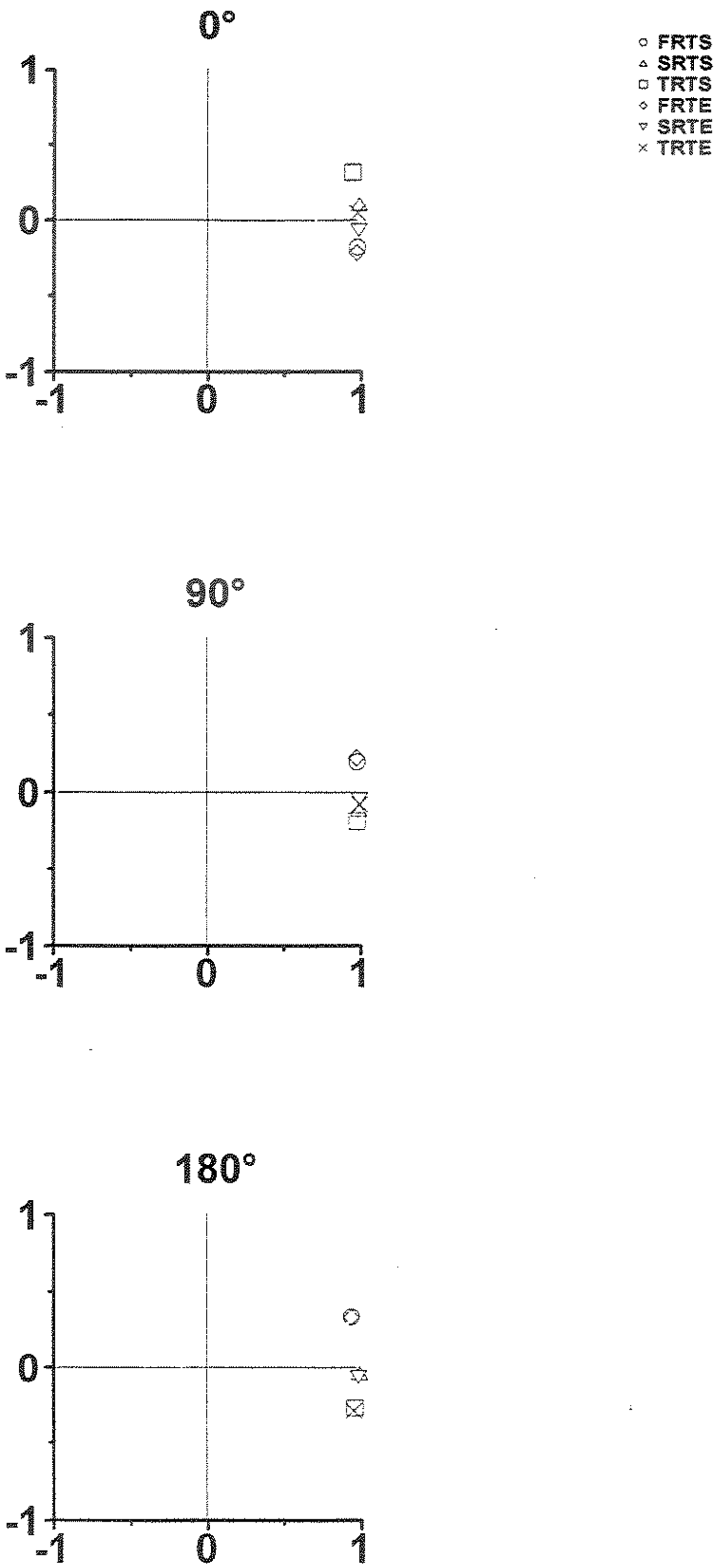


Figure 6

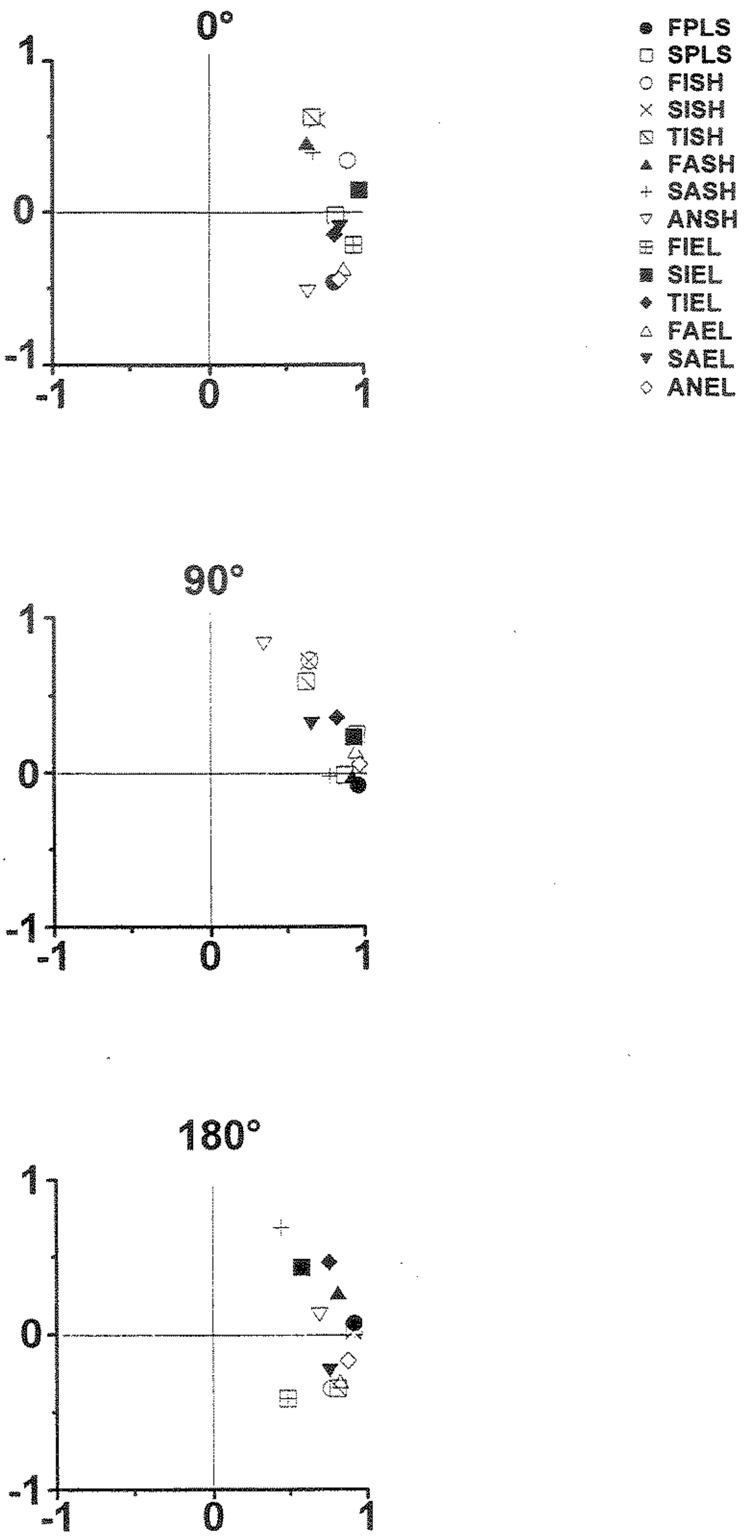
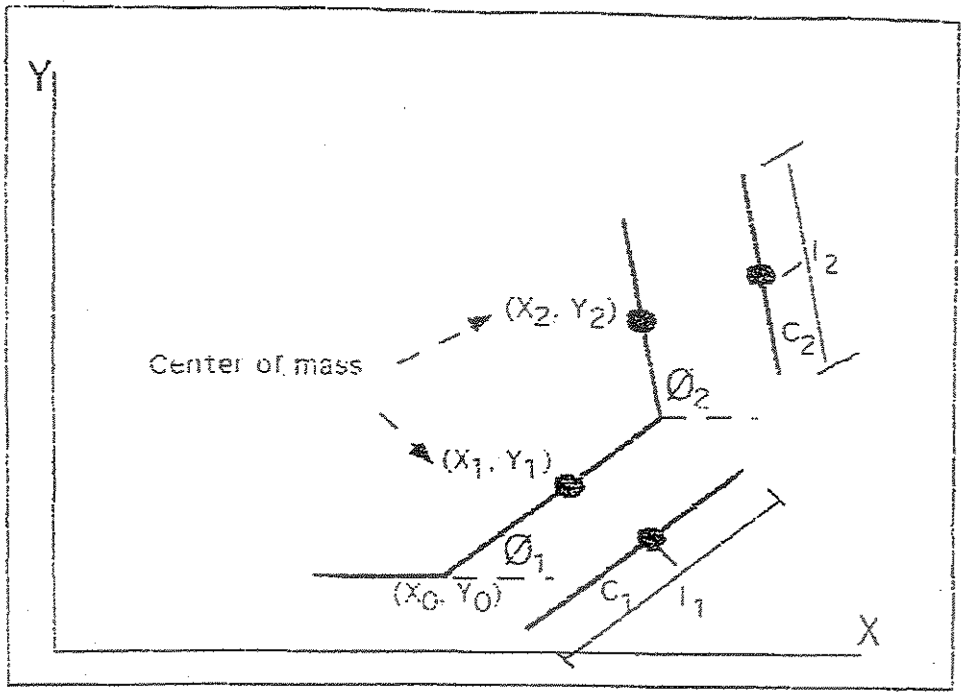

0°
90°
180°
1
0
-1
-1
0
1
● FPLS
□ SPLS
○ FISH
× SISH
⧄ TISH
▲ FASH
+ SASH
▽ ANSH
⊞ FIEL
■ SIEL
◆ TIEL
△ FAEL
▼ SAEL
◇ ANEL

Figure 7

		TIME OF THE FIRST IMPULSE		**TIME OF THE SECOND IMPULSE**		**TIME OF THE THIRD IMPULSE**	
		Shoulder	Elbow	Shoulder	Elbow	Shoulder	Elbow
IMPULSE	180°	43.9 +	3.6 +	26.9 +	3.4 +	47.4 +	5.4 +
	90°	1.2 -	3.1 +	3.9 +	3.3 +	2.8 +	3.5 +
	0°	7.1 +	17.5 +	5.4 +	12.3 +	5.9 +	10.0 +

		TIME OF FIRST IMPULSE		**TIME OF THE FIRST + SECOND IMPULSE**		**TIME OF THE SECOND + THIRD IMPULSE**	
		Shoulder	Elbow	Shoulder	Elbow	Shoulder	Elbow
INTEN	180°	3.2 +	6.3 +				
	90°	1.8 -	.58 -				
	0°	2.7 -	2.1 -				
AGO	180°	10.4 +	4.5 +			9.5 +	3.7 +
	90°	6.2 +	11.1 +			7.5 +	4.6 +
	0°	14.3 +	6.0 +			15.2 +	6.8 +
ANT	180°			7.3 +	4.2 +		
	90°			10.4 +	8.4 +		
	0°			13.4 +	6.4 +		

Table I



$$\mu_2 = m_2c_2\,[-\ddot{X}_1\sin\varnothing_2 + \ddot{Y}_2\cos\varnothing_2] + I_2\ddot{\varnothing}_2$$

$$\mu_1 = \mu_2 + m_1c_1\,[-\ddot{X}_1\sin\varnothing_1 + \ddot{Y}_1\cos\varnothing_1] + m_2l_1\,[-\ddot{X}_2\sin\varnothing_1 + \ddot{Y}_2\cos\varnothing_1]$$

$$\text{elb}_{INT} = I\ddot{\varnothing}_2 - \mu_2$$

$$\text{Sho}_{INT} = \mu_1 - [I_2 + m_2c_2^2 + I_1 + m_1c_1^2 + m_2l_1^2 + 2m_2l_1c_2\cos\varnothing_2]\ddot{\varnothing}_1$$

Where:

1 = shoulder
2 = elbow
I = moment of inertia in relation to the center of mass.
¨ acceleration
μ_2 = elbow muscle torque
μ_1 = shoulder muscle torque
Elb_{INT} - elbow interaction torque
Sho_{INT} = shoulder interaction torque

Equation of Motion

Control of reversal movements in normal individuals and Down syndrome: The effect of intrinsic feedback

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Running Title: Normal and Down Syndrome individuals: the effect of intrinsic feedback

Summary

Normal and individuals with Down syndrome performed reversal movements over three different target distances and three spatial orientations. The target distances were based on the amount of elbow excursion. During the performance of these movements the subjects did not have visual information about the moving limb. However, they could identify the amount of elbow excursion comparing the information from a goniometer, fixed at this joint, with a set of lights showed in the direction of the movement. Both individual groups were able to perform the reversal movements, but failed to use the intrinsic information to move the elbow joint at the required angular excursion. These results favor the idea that the Central Nervous System (CNS) did not use intrinsic information to plan movements.

Normal subjects performed the reversal movements using a linear-covariance between intrinsic (muscle activities and muscle impulses) and extrinsic (linear speed of the fingertip) variables. This linear coupling between the intrinsic and extrinsic variables was termed "Building Block Strategy". On the other hand, the Down syndrome individuals were less efficient in coupling muscle activities, with muscle impulses and with the fingertip speed. The pattern of muscle activities of these subjects was characterized by a generalized co-activation. They also failed to modulate the bursts of muscle activities with changing in target distance and spatial orientation. The data are discussed in terms of possible impairments in the motor control system of the Down syndrome individuals.

Supplementary Key Words: reversal movements, Down syndrome, intrinsic feedback, spatial orientation, motor control.

Introduction

The performance of a single-joint elbow flexion movement involves a generation of a reciprocal pattern of muscle activity (EMG) characterized by sequential bursts of agonist-antagonist-agonist activities, called the triphasic pattern of muscle activity (Hallett, et al, 1975; Hannaford & Stark, 1985). Since then, a simple set of rules has been used to describe how Central Nervous System (CNS) could modulate this pattern of muscle activity to generate constrained single-joint movements over different distances, speeds and loads (Corcos, et al., 1989; Gottlieb, et al., 1989). This pattern has been described in terms of modulation of the intensity and/or duration of the first EMG agonist burst and the time between the onset of this burst and antagonist EMG burst of muscle activities (antagonist latency). This simple-set of rules was generalized to explain how the agonist and antagonist bursts of the EMG activities could be modulated with changing in angular distance for unconstrained elbow and shoulder flexion (Almeida, et al, 1995), for pointing (Gottlieb, et al, 1996) and for constrained reversal movements (Gottlieb, 1998).

This set of rules are based on the assumption that the EMG activities and the muscle force are variables physiologically linked, in the sense, that both are generated by a common excitation pulse (action potential) driven to the motor neuron pool (Gottlieb, et al., 1989). Because of that muscle activity and muscle force would share a causal relationship. The linear relationship between the EMG agonist burst and the muscle impulse has been showed for several movements (Almeida, et al., 1995; Gottlieb, et al., 1996).

Additional studies showed that the muscle torques at the elbow and shoulder joints are coupled during pointing movements (Gottlieb, et al., 1996). The coordination between the generation of both elbow and shoulder muscle torque could be described by the Principle of Linear Co-variance between them (Gottlieb, et al., 1996). This principle states that both elbow and shoulder muscle torques are coupled on time and in magnitude during the performance of unconstrained pointing movements. The linear co-variance could also explain the coupling between elbow and shoulder muscle torques for movements performed over different spatial orientations (Gottlieb, et al., 1997) and for reversal movements (Almeida, et al., 1999).

However, all these rules were formulated at the execution level and dealt with observed behavior that occurred at the joint level (intrinsic level). Long ago, Bernstein (1935) advanced the idea that the CNS should have higher level of projections of space and not of force joint or muscles. Since than Morasso (1981) proposed the so-called "spatial control hypothesis". This hypothesis was based on observed invariance of the fingertip trace and linear velocity (extrinsic information) for planar pointing movements of the upper limb to targets at different spatial locations. Supports favoring the idea that the CNS uses extrinsic information to control movement can be found in several others studies (Lashley, 1951; Russell, 1976).

The idea of "spatial control hypothesis" implies in the existence of a mechanism for transforming spatial motor commands into coordinated joint angular patterns (Pellionisz, 1980). In this sense, the modulation of the muscle patterns and the muscle torque is not in contradiction with the idea of the "spatial control hypothesis". However, more recently, Gottlieb (1998) compared constrained elbow flexion and elbow reversal movements and concluded that the "knowledge of net muscle torque and limb kinematics, is not adequate to fully predict those rules or the muscle activation patterns they produce" (page 1). If this observation was correct, the CNS would have to transform the spatial motor commands into appropriate level of muscle activity and into appropriate level of muscle torque, at two separate stages. However, we showed that the CNS use a "Building Block Strategy" to generate and couple the patterns of muscle activities, with muscle impulses at both elbow and shoulder joints, and with the linear speed of the fingertip. This "Building Block Strategy" was observed during unconstrained reversal movements performed with intrinsic (Almeida, et al., 2000; Marconi, et al., 2000) and extrinsic information about the target (Almeida, et al., 2000). However, the amount of error observed in these experiments favor the idea that the CNS uses extrinsic information to plan a motor task.

In this study we used the "Building Block Strategy" to compare how individuals with Down syndrome control reversal movements. It has been showed that these individuals can shift from a typical triphasic to a generalized pattern of muscle activity, characterized by a co-activation during the performance of several movements (for a review, see Almeida, et al., 1999). Their movements are clumsy and slower compared with overall population (Almeida, et al., 1994). We also showed that DS individuals can perform reversal movements at different spatial orientations. However, they spent more time reversing the movement at the target and used proportional more elbow than shoulder muscle torque (Almeida, et al., 1999).

Methods

Subjects

Eight neurologically normal subjects (NN), 4 male and 4 female, and eight subjects with Down Syndrome (DS) took part in this experiment after given formal consent approved by the State University of Campinas. All subjects were right-hand and were between 15 and 30 years old of age. The subjects of both groups were paired by gender and age.

Tasks

Each subject performed reversal planar movements, involving elbow and shoulder excursion with the wrist and hand immobilized by a thermoplastic splint. The movements were divided into three tasks that varied with the spatial orientations (180°, 90° e 0°). The line in the saggital plane, crossing the middle line of the subject's body, in the antero-posterior direction, defined the spatial orientation of the 90° task. The line defining the spatial orientation for the 0° (right) and for the 180° tasks (left) was orthogonal to the line of the 90° task (figure 1). The initial position was set at the intersection between the lines of 90° task with the line of the 0° task. At the initial position, the fingertip was kept over the line of the 90° task, with the elbow joint at 90° of extension (full flexion being equal to 0°).

Insert here figure 1

The subjects were seated in an adjustable chair, with their trunks strapped in the straight back of the chair. A cable fixed at the ceiling of the experiment room sustained the proximal part of forearm. At this position their right limb could move freely around 10 cm above the top of a closed table (1.5x1x1 meter).

For each task the subject performed three series of movements defined according to one of the three elbow angular excursions. We used the following steps to define the angular target distance of the elbow joint. First, a homemade goniometer was fixed on the elbow joint. This goniometer was connected to a set of ten small lights, fixed on series in a metal bar (15 X 2 cm). For each 9° of elbow excursion in extension one light was turned on. This set of lights was put on the task spatial orientation, one meter away from the initial position, in the subject's field of vision. Second, keeping the wrist immobilized, the experimenter passively moved the subject's upper limb from the initial position to the direction of the spatial orientation line of the task. The upper limb was positioned in such a way that the fingertip laid on the line of the task with the elbow joint extended at one of the three target distances (108°, 126° and 144° in figure 1). The elbow angular target distance was checked with simple acrylic gonimometer. Forth, at the target position, the homemade goniometer was adjusted to turn on the appropriate number of lights. The number of lights turned on were two, four, and six, respectively for the 108°, 126° and 144° elbow target distance (figure 1).

During the performance of the tasks the subjects used a glass without lenses that avoid any view of their moving limb (finger, hand, forearm and upper arm). The only information the subjects had was the number of lights turned on and the direction of the movement. So, to perform the task well the subjects first had to match the required elbow excursion at the required spatial orientation of the task with the correspondent number of lights. Thus, for each task the subject had to perform a series of five movements to a target that would require 108°, 126°, and 144° of elbow extension at the final position. The trunk line was kept orthogonal to the line of the 90° task during all experiments (figure 1).

Before data recording each subject had 5 trials of practice for each experimental condition (task and amount of elbow excursion). The subjects were instructed to move "as fast as possible" to and from the target, without stressing reaction time. They were also required to try to perform the movements "as accurately as possible". For normal subjects, five movement trials were recorded for each experimental condition during two seconds each one. For Down syndrome individuals the duration of the trials varied from three to four seconds.

Recording and processing of EMG data

We recorded the EMG activity of the biceps long head, lateral head of triceps, anterior deltoid and posterior deltoid muscles using a DelSYS (model DE2.2L) EMG amplifiers with surface electrodes, with the total gain of 2000 and band pass of 20-450 Hz. All EMG data were digitilized at 1000 frames/second using Optotrak software and synchronization unit. These EMG data were rectified and filtered using 20 ms moving size window. The processed EMG data were used for the quantification purpose.

Recording and processing of kinematic data

We fixed LEDs (light emotion diode) as closely as possible to center of the shoulder, elbow and wrist joints, and on the fingertip. The X and Y coordinates of these LEDs were recording using Optotrak Motion Analysis System 3020 at 100 frames per second. From these data, the orientations of the two segments were calculated. The upper and forearm orientations with respect to the medial lateral axis (X axis in figure 1) in the horizontal plane are denoted respectively by ø1 and ø2. The elbow angle was defined as 180° minus the difference between ø2 and ø1. From angle we obtained angular velocity and acceleration. Angle, velocity and acceleration were smoothed using 10 ms moving size window. The linear velocity of the fingertip was obtained from the X and Y coordinates of the fingertip.

Using inverse dynamics to calculate muscle torque

Using the subject's weight and the regression equation of Winter (1979) we obtained the inertial parameters of the upper arm and forearm and the location of the center of mass of these segments. In addition to the measured lengths of the proximal and distal segments (L1 and L2 respectively), the following inertial parameters were estimated: the masses of the segments (m1, m2); the distances from the proximal end of the segment to its center of mass (c1, c2); and the moments of inertia about the center of mass (I1, I2). At each moment of time, and for each segment, given the current coordinates of one of the markers fixed to the segment and the orientation of the segment, the known distance of the marker from the center of mass was used to determine the coordinates of the center of mass. The center of mass coordinates are denoted by (x1, y1) for the proximal segment, and (x2, y2) for the distal segment.

The equations of motion, derived from first principles, relating the torque at each joint to kinematic variables and inertial parameters, are as follows. T1 and T2 represent, respectively, the muscle torque at the shoulder and elbow joints. Some authors refer to this as the "generalized muscle torque (moment)" (Schneider, Zernicke, Schmidt, & Hart, 1989).

Insert here the equation

In deriving these equations of motion, we have not assumed that the shoulder remains fixed in space. Note that if this assumption were made, and the center of mass coordinates were expressed in terms of joint angles by appropriate trigonometric relations, the equations of motion would be transformed into the more commonly employed form in which joint angles rather than center of mass coordinates appear as the kinematic variables.

Quantification

For the purpose of analysis all movements were divided into three phases based on the muscle torque profile. The first phase was identified as the interval between the onset and the first time the muscle torque reversed direction. The second phase was identified as the interval between the end of the first phase until the time the muscle torque reversal direction for the second time. The third phase identified was the time interval between the end of the second phase and the time the muscle torque reversal direction for the third time. The four broken vertical lines at the shoulder muscle torque (see figure 2) illustrate the three landmarks that emcompass the three movement phases. These three phases were defined for both elbow and shoulder muscle torque.

EMG agonist and antagonist burst

We plotted on a monitor screen the fingertip velocity, muscle torque and the corresponding EMG activity for the agonist and antagonist muscle for each analyzed joint. Using a computer cursor we visually identified the onset of the agonist and antagonist EMG bursts for each joint. The onset was taken as the first sustained rise above the baseline. We integrated the agonist EMG activity from its onset until the first 30 ms. This activity was used to identify the intensity in which the muscle was activated. We also integrated the agonist EMG activity during two others intervals of time encompassing two agonist bursts of muscle activities. The first agonist burst was defined as the integrated muscle activity, from its onset until the first time that the muscle torque reversed direction. The second agonist burst was defined as the integrated muscle activity during the time interval between the second and the third time that muscle torque reversed direction. The bursts of the antagonist muscle activity were integrated from the onset of the first agonist muscle to the time the muscle torque reversed direction for the second time. All quantified values of the EMG bursts of muscle activities were normalized. For the normalization we divided the quantified value of the amount of EMG activity during each phase by its EMG activity during the baseline. The baseline was defined as the time interval between 200 and 300 ms before movement onset.

Muscle impulse

We integrated the muscle torque during the time of the three movement phases described above and called them as the first, the second, and the third impulses.

Elbow and shoulder excursion and movement error

We measured the elbow and shoulder angular excursion at the reversal time. We calculate the percentage of constant error using the following equation:

$$CE=(PTD-RTD)/RTD*100$$

Where CE is the constant error, PTD is the performed target distance at the reversal time, and RTD is the target distance required by the task. The amount of error was measured at the direction of the spatial orientation for each task (180°, 90° and 0°).

Data analysis

Statistical analysis consisted of a three way ANOVA to the kinematic, kinetic, EMG, and percentage of the error dependent variables for the elbow joint. For the shoulder joint we ran a two way ANOVA to test the effect of group and target distance on the variables cited above. We did not include the effect of spatial orientation for the dependent variables of shoulder joint in the ANOVA test for two reasons. First, the shoulder movement changed direction with spatial orientation. It initially moved into abduction for 0°, and into adduction for 180° task. For 90° task, 8 subjects moved the shoulder initially into abduction and 8 subjects moved it into adduction. Second, the agonist and antagonist shoulder muscles also changed with movement direction. The anterior deltoid was the shoulder agonist at the 0°, and at the 180° the posterior deltoid was the agonist. These results of the ANOVA test for the elbow and shoulder joints are presented on Table I. We ran a four way ANOVA to test the effect of group, target distance, spatial orientation and type of peaks (first versus the second), and presented the results of this ANOVA in the results session. We explore the interactions observed between and among some of the variables tested in the ANOVAs, using a Pos hoc comparison based on the modified Bonferroni procedure (the overall error rate was set at 0.1).

We ran a factorial analysis using the Principal Component as an extraction method with the three landmarks of the elbow and shoulder muscle torques, and with the intrinsic (the three EMG bursts and the three muscle impulses of both elbow and shoulder joints) and extrinsic (the two peak speeds of the fingertip) variables.

Results

We present here the data for the normal subjects (Almeida, et al., 2000) for two reasons. First, we use these data to analyze the effect of spatial orientation on the muscle activities and on the muscle impulses. Second, we use these data to compare the performance of both normal and with Down syndrome individuals.

General features of the movements

In figure 2 we illustrated some commonalties and differences in way one DS and one NN subjects performed task at 0° spatial orientation. Overall, the behaviors described for these two subjects are representative of their groups. The linear speed of fingertip was characterized by a double bell-shape with two maximum peaks. The first peak occurred around the middle distance from the home to the target position. The second peak occurred around the middle distance from the target to the home position. Compared with the NN subject, the DS subject moved slower to and from the target and spent more time reversing the movement. Note, also that the second peak of the fingertip was smaller than the first one for the NN subject, but not for the DS subject.

The shoulder and elbow muscle torques were characterized by three impulses (see method). The NN subject generated these three muscle impulses using a reciprocal EMG pattern of muscle activity, whereas the DS subject used a pattern of co-activation. The reciprocal pattern of elbow and shoulder EMG muscle activity was characterized by two agonists and one antagonist EMG bursts. The first agonist burst ended abruptly around the time in which the muscle torque reversed direction for the first time (approximately, the time when the first linear peak speed occurred). After a silent period, a second agonist EMG burst started, which was smaller than the first one. The antagonist EMG burst started in the time interval between the beginning of the first and the second agonist burst. The antagonist EMG burst ended around the time when the muscle torque reversed direction for the second time (approximately the time of the second peak linear velocity).

On the other hand, the elbow and shoulder EMG muscle activities of the DS subject were characterized by a generalized activity of both muscles, during the whole movement time. Note figure 2 there was more fluctuation in the EMG activities and the antagonist latencies were very short, which characterizes a pattern of muscle co-activation. The two agonists and the one antagonist EMG bursts could not be visually identified, as we did for the NN subject.

The generation of the three muscle impulses at the two joints seemed to be coupled with the muscle activities of the three EMG bursts for the NN subject, but not for the DS subject. For the NN subject, the first EMG agonist bursts generated initially a muscle impulse throwing the limb into the target position. This was done by moving the shoulder joint into abduction and the elbow joint into extension. The EMG activity of the antagonist bursts generated the second muscle impulse that had two different functions. It first decelerated the limb at the target position. Then, it reversed movement direction of the two joints, throwing the limb back into the home position. The second agonist EMG burst generated the third muscle impulse, that decelerated the limb towards the home position. Because their EMG bursts were not well defined, we cannot visually identify these coupling between the EMG bursts of muscle activity and the muscle impulses for the DS subjects. Finally, note that the muscle torques were smaller and had more fluctuation for the DS, compared with the NN subjects.

Insert here figure 2

kinematics of the elbow and shoulder joints for both groups of subjects

Even though the experiment setup tried to constrain the elbow excursion at certain target distance (108°, 126°, and 144°), all subjects were free to choose any combination of elbow and shoulder movements to perform the tasks. A visual analysis of the fingertip path revealed that all subjects were able to perform the tasks at the three spatial orientations (180°, 90°, and 0°).

The elbow angular excursion was similar for both groups, and increased with target distances and changed with spatial orientations (Table I). On average, the elbow excursion was 34°, 41°, and 38° respectively for 180°, 90°, and 0° spatial orientation (figure 3). However, there was an interaction effect between subject group and spatial orientation. A post hoc comparison showed that this interaction effect was determined mainly by the DS group, that displayed increased amount of error from the 90°, to 0°, and to 180° spatial orientation. On the other hand, the NN group had similar amount of error across all three spatial orientations. The shoulder angular excursion also was similar between both groups of subjects at the three spatial orientations and increased with target distances (figure 4). Note that initially the elbow joint always moved into extension, but the shoulder joint moved into 90° and 180° adduction and into 0° abduction for spatial orientation task.

Insert here figure 3 e 4

Movement speed

We ran a four way ANOVA to analyze the effect of groups, target distances, spatial orientations and type of the fingertip speed (first versus the second). These data are showed in figure 5. The DS subjects moved slower than the NN subjects (F1,14= 33, p=0.000). The fingertip speed increased with target distances (F2,28= 140, p=0.000), and there was an interaction between group and target distance (F2,28= 29, p=0.000). The movement speed also changed with spatial orientation (F2,28= 100, p=0.000), and there was an interaction between group and spatial orientation (F2,28= 29, p=0.000). The speeds of the first peak was faster than the second (F1,14= 12, p=0.004), but there was an interaction between group and peak speed (F1,14= 12, p=0.004). A post hoc comparison showed that the interaction between group and speed was due to the fact that DS group moved to (first peak) and from the target (second peak) at similar speed, but the NN group moved faster to than from the target.

Insert here figure 5

Movement accuracy

The amount of movement error was similar between both subject groups (Table I). The target distance affected the amount and the direction of this error (figure 6). A post-hoc comparison showed that this error differed for the three target distances. On average, the subjects overshoot the target by 41% at the shortest distance (108°) and by 8% at the intermediary distance (126°). For the larger target distance (144°), they undershoot the target by –6.6%. The spatial orientation also affects the amount of error. There was a main effect of spatial orientation and a interaction between spatial orientation and subject group. A post hoc comparison showed that, this interaction was due to a similar amount of error across all spatial orientations for the NN group, but not for the DS group. For the DS group the amount of error increased from the 180°, to 0°, to 90° spatial orientation.

In summary, we showed that both subject groups performed all tasks using similar elbow and shoulder excursion with comparable amount of error, but with different movement speeds. Next we show how DS and NN individuals performed these tasks with different muscle torques and muscle EMG activities across both joints.

Insert here figure 6

The effect of target distance on EMG muscle activities and muscle impulses

Intensity of activation of the agonist muscle

Overall, the intensity of the triceps agonist bursts did not change with target distances for both subject groups (see table I). However, the NN individuals activate their agonist elbow muscles with greater intensity compared with the DS individuals (figure 3). Note that even though the variability of the intensity in which the triceps was activated was larger for the NN group, compared with DS, there was no significant interaction effect between target distances and groups (table I). Compared with DS, the NN subjects activated the shoulder muscles with greater intensity (table I). The intensity in which the shoulder muscles were activated did not change with target distance into all three spatial orientations (figure 4). The only exception was for NN group subjects at 180° in which the intensity of the activation of the anterior deltoid increased with distance (table I).

The amount of the two agonist and the antagonist EMG bursts of muscle activities

The EMG activities of the first burst of the triceps muscle were smaller for the DS group when compared with the NN group (figure 3). However, there was no group difference in the amount of EMG activities of the second agonist and antagonist bursts of the elbow muscles across all tasks (Table I). The amount of these EMG activities of the elbow muscles increased with target distances. A post hoc comparison showed that this increment of the EMG bursts was observed just for the NN group. The DS subjects did not always modulated the EMG bursts of muscle activities with target distances. For example, the first burst of the triceps muscle of DS individuals did not differ between 126° and 144° target distances. Also, the second agonist burst of the triceps muscle and the antagonist burst of the biceps muscles of these subjects did not differ between 108° and 126° target distance.

The first burst of the shoulder agonist muscle was larger for NN subjects just for 0° spatial orientation. At this spatial orientation the amount of muscle activity of the first shoulder agonist burst increased with distance for NN subjects, but not the DS subjects. There was also no group difference for the antagonist EMG bursts of muscle activities (figure 4). However, DS subjects generated more EMG activities for the second agonist muscle (table I).

The three EMG bursts of muscle activities of the shoulder joint increased with target distance. The only exceptions were for the second agonist and the antagonist EMG bursts at the 90° spatial orientation that remained constant over the three target distances.

The three muscle impulses

The three muscle impulses of the elbow joint increased with target distance (table I) and were larger for the NN group compared with the DS group (figure 3). Note that there was an interaction between group of subjects and target distances for all three muscle impulses. A post hoc comparison showed that this interaction was due to the fact that the three elbow muscle impulses increased with distance for the NN group, but not for DS subjects. For example, the three elbow and shoulder muscle impulses of DS subjects did not differ for the two largest target distances (126° and 144°).

The three shoulder impulses increased with target distance into 0° and 180°, but not into 90° spatial orientation. At 90° spatial orientation there was no group difference in the three shoulder impulses, and their values were closed to zero for both group of subjects (figure 4). However, the three shoulder impulses were larger for the NN than for the SD group for 0° and 180° spatial orientations (Table I). A post hoc comparison revealed that the three shoulder muscle impulses into the 180° spatial orientation increased with target distances for NN subjects, but not for DS group.

The effect of spatial orientation on EMG muscle activities and muscle impulses

Intensity of activation of the agonist muscle elbow muscle

The intensity in which the first agonist elbow muscle was initially activated did not change with spatial orientation (table I), but it was larger for the NN compared with the DS individuals (figure 3).

The amount of the two agonist and the antagonist EMG bursts of elbow muscle activities

The spatial orientation affected the amount of muscle activities (figure 3). However, there was a significant interaction between subject groups and spatial orientations for the second agonist and the antagonist EMG burst of muscle activities (table I). A post hoc analysis showed that this interaction was due to qualitative difference between both groups in the way they modulate the bursts of the EMG activities with spatial orientations. For example, the second burst of the triceps muscle of the DS group was comparable for 90° and 180°, but larger than the one at 0°. On the other hand, this second agonist burst of the triceps increased from 90°, to 0°, to 180° spatial orientation for the NN subjects. The burst of the antagonist biceps muscle of the DS group was comparable for 0° and 90°, but smaller than the one at 180° tasks. For the NN group, the antagonist activity was similar for 0° and 180°, but higher than the one of 90° spatial orientation.

The three elbow impulses

The spatial orientation affected the modulation of the muscle impulses in a different way for each subject groups (figure 3, and table I). First, a post hoc analysis showed that the three elbow impulses increased from 180°, to 90°, to 0° spatial orientation for both subject groups. The only exception was the second elbow impulse that was similar between the spatial orientations of 90° and 180° tasks. Second, the group difference for the three impulses was larger for the 0° spatial orientation, as compared with the other two tasks.

Temporal coupling among the times both muscle torques reverse directions

The muscle torques at both joints reversed direction at the same time (figure 2). We ran a factorial analysis using Principal Component Analysis as extraction method among all the three landmarks of the elbow and shoulder muscle torques. The first factor of the factorial analysis explained more than 92% of the population variance for the NN subjects. This results revealed two temporal coupling between elbow and shoulder muscle impulses for the NN subjects. First, both elbow and shoulder muscle impulses reversed direction at the same time. Second, all the three landmarks of the two muscle torques were linear related among them. The DS subjects also reversed elbow and shoulder muscle torque at the same time as can be observed in figure 2. However, for the DS group the explanation of the first factor decreased to 75%. The explanation for the decrease of population variance for the first factor observed for DS subjects has to do with the way they couple all the three landmarks of the two muscle torques (figure 7).

Coupling among muscle impulses and EMG muscle activities

In Figure 8 we plotted the first factor against the second factor of the unrotated matrix. We showed the weight values of each factor for the three muscle impulses and three EMG for both elbow and shoulder joints, and for the two peak of the fingertip linear speed. These weight values were obtained by a factorial analysis using Principal Component Analysis as extraction method. The results for the NN subjects are showed in Almeida et al., (2000) and it is represented here again to allow the comparison between the performance of DS and NN subjects.

The total variance proportions for the first and the second factor was larger for NN than for SD group. This results showed that the DS individuals have difficult in coupling the EMG muscle activities, with muscle impulses and the fingertip speed. For the 0° task the variance proportions 0.7 and 0.16 for the NN subjects, and 0.37 and 0.22 for the DS subjects. For the 90° task the variance proportions were 0.53 and 0.22 for the NN group, and 0.44 and 0.18 for the DS group. Finally, these values for 180° task were 0.62 and 0.12 for NN subjects, and 0.44 and 0.27 for the DS subjects.

The weight values that defined the first factor for each of the variables studied (three EMG bursts, the three muscle impulses of both joints, and the first and the second fingertip peak speeds) were above 0.7 for the NN group for the 0° task. For DS group the weight values of the first factors for the 0° spatial orientation was higher just for the three elbow and shoulder impulses (figure 8). These results show that NN subjects, compared with DS subjects, can better coupling the EMG muscle activities with muscle torque and the speed of the fingertip.

Indeed, the coupling between the three muscle impulses at the elbow and shoulder joints seems to be a common feature of all movements. Overall, the coefficient of linear correlation ($|r|$) was above 0.8 between each pair of the 32 out of 36 variables (3 spatial orientation x 2 groups x 3 muscle impulses x 2 joints) in comparison with all muscle impulses. However, this coefficient of linear correlation was greater for 0°, compared with 180 and 90°

spatial orientations. Also the values for these coefficients of linear correlation were greater for NN subjects at the 0° spatial task compared with DS subjects.

Note that the weights values of the first and the second peak of the linear fingertip speed for the first factor were different between subject groups. They were above 0.8 for the NN subjects, and below 0.5 for DS subjects. This is a reflection of lower linear correlation between both peak speeds for the DS group compared with NN group. The coefficients of linear correlation ($|r|$) between the first and the second peak speed were .37, .31, and .67 respectively for 0°, 90°, and 180° spatial orientation for DS subjects. These values were .76, .93, and .93 for NN subjects.

Discussion

Both normal and Down syndrome individuals do not use the intrinsic information to plan reversal movements

Even though the experiment set up tried to constrain elbow motion at certain angular excursion into workspace of the joints, the subjects were free to choose any combination of elbow and shoulder movements to perform the tasks. Indeed, both individuals groups did not used the expected elbow excursion to perform the tasks. However, the angular excursions of both elbow and shoulder joints were similar for both individual groups, across all tasks (figures 3 e 4). The error for the elbow angular excursion was high, but similar for both groups (figure 6). The results confirm the observation that neither normal (Almeida, et al., 2000), nor Down syndrome individuals (Almeida, et al., 2000) use intrinsic information to plan complex planar reversal movements.

These similar error observed between both groups at the two joints should not be attributed to any technical problem[1]. We do not know the rules that determine choice of the CNS about what range of motion should be used in each of the joints to perform one task. Someone could argue that we were naive when by designing experiments which ignored the constraints of the motor system represented by the joints, nerves and muscles configurations. However, some of these constraints we learned with this study. For example, at 180° spatial orientation the bi-articular biceps worked as the shoulder agonist muscle (Almeida, et al., 2000). The shorting of this muscle would limit the elbow range of motion into extension. This may explain why these movements at 180° spatial orientation were performed with more shoulder than elbow movements.

One could argue that DS individuals traded off speed for accuracy (Kerr & Blais, 1987). Had they moved as fast as the normal individuals, their movement error would be even larger (Fitts & Peterson, 1964). However, we observed that both group of individuals displayed larger error for the shorter target distance than for the longer one.

The effect of target distance on the modulation of the kinematic, kinetic and EMG muscle activities

Even though both individual groups performed the tasks using similar elbow and shoulder movement excursions and with similar movement accuracy, they differ in the strategy used to modulate the three EMG bursts of muscle activities, the three muscle impulses and the two peaks of the fingertip speed.

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[1] The equipments (goniometer and set of lights) used to inform the elbow excursion was the same for all tasks conditions and it worked well in all spatial orientations and target distances used in the experiment.

strategy used to modulate the three EMG bursts of muscle activities, the three muscle impulses and the two peaks of the fingertip speed.

The normal individuals modulate the muscle activities for these reversal movements according to the rules defined by the "Speed Insensitive Strategy" (Gottlieb, et al., 1989), and that was described in Almeida et al., (2000). First, the intensity in which the agonist muscle was activated was kept constant over different target distance. Second, the two agonists and the antagonist EMG bursts of muscle activities increased with target distance. The additional finding here is that the spatial orientation does not affect the way normal individuals modulate the EMG burst of muscle activities and the muscles impulses (figure 3 and 4) with changing in target distance.

However, the strategies used by individuals with Down syndrome to activate the muscles differ in three ways from what was observed for the normal individuals. First, they also kept the intensity in which the agonist muscles were initially activated constant over different target distances. However, this intensity was smaller for Down syndrome individuals, showing their difficult of initially recruiting larger amount of motor units. The implication of that would be a deficit in generating abrupt muscle force and in modulating it. This observation supports several others showing a deficit of these individuals in modulating the grip force (Cole, et al., 1988).

Second, overall the individuals with Down syndrome have problem in modulate the amount of the muscle activities with target distance. This is also reflected in the lack of modulation of the muscle impulses with target distance (figures 3 e 4). Taking together, these findings may show a deficit of individuals with Down syndrome to modulate the amount of muscle force for different movement distances. Nevertheless, the lack of modulation of the muscle forces with target distance did not interfere with the ability of these individuals to move to and from the target. Remember that the participation of the elbow and shoulder joints and the movement accuracy were similar between both individuals groups (figure 6). However, the lack of modulation of the muscle forces with target distances affected movement speed of the individuals with Down syndrome and not their movement accuracy. Observed that the speed of the fingertip were similar for these individuals across different distances (figure 5)[2].

The effect of spatial orientation on the modulation of the kinematic, kinetic and EMG muscle activities

Taking in account these limitations, and considering that both individuals groups had the same angular excursion at both joints, for each spatial orientation (figure 3 e 4), we can draw some conclusions. First, the spatial orientation affects the amount of muscle impulses that can be generated by a certain amount of muscle activities. For example, compared the elbow impulses and the muscle activities at 90° and 0° spatial orientation (figure 3 e 4) for the normal individuals. The first agonist bursts of the EMG triceps activities were similar for both orientations, but the amount of the first elbow impulse differed markedly between both orientations. Also, the elbow movement was larger and faster at 90° than at 0° spatial orientation (figure 5).

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[2] The fact that the amount of muscle activity did not differ between both groups (figure 3 and 4) across different target distances is just a reflection of the time interval used to calculate these activities. Individuals with Down syndrome moved slower, spending greater time to perform the tasks than the normal individuals. Because of that, muscle activities observed for a similar time interval between group.

These findings confirm other observation that the spatial orientation influences the amount of muscle activities and the amount of muscle torque generated at the joint. The amount of force generated by the muscle activity is also affected by its length and speed of shortening. The spatial orientation could affect the length and the speed of shortening a muscle, influencing its capability to generate muscle torque. The effect of the workspace on the amount of the EMG activity generated by the muscles is showed in pointing movements (Flanders, 1991; Gabriel, 1997).

Second, both individual groups differed in the way they scaled elbow muscle impulses and the bursts of the EMG muscle activities with spatial orientation (figure 3 e 4). Overall, the amount of muscle activity and muscle impulse generated by individuals with Down syndrome were less affected by the spatial orientations. Compare for example, the amount of muscle impulse generated by the burst of EMG triceps activity at 90° and 180° spatial orientation. Note that the elbow excursion was similar between both groups at these two spatial orientations, but the change in muscle activities and in muscle impulse was smaller for the individuals with Down syndrome.

Third, the individual with Down syndrome generated proportionally less elbow muscle impulse for all three spatial orientations (table I), but this group difference was greater for the 0° spatial orientation (figure 3). These results showed that their movement performance maybe more impaired in some regions of the workspace than in others. This is exactly what we observed for the fingertip speed (figure 5). Note the group difference to the fingertip speed was longer for 0° spatial orientation.

Why the individuals with Down syndrome have more difficult in generate the muscle impulses at certain region of the workspace? We believe that this has to do with the way they modulate the activation of the muscles. These individuals antecipate the activation of the antagonist muscles independent of the changing in spatial orientation (Ferreira, 2000). Because of that they start to break the movement too earlier. So, this anticipation affects more the spatial orientation in which the elbow moved more, that was at 0° spatial orientation (figure 3). In other words, the individuals with Down syndrome are less efficient in generating muscle torque at 0°, in part, because of the antecipation of the antagonist muscle activation affected more the larger elbow joint excursion at this orientation.

Let us also assume that there is an option amount of EMG activity generated by the muscle at certain region of the workspace that would lead to a proper amount of muscle torque at the joint. Also, assume that the data presented for the normal individuals represent an optimal solution. So, a major group difference observed for the elbow impulse at 0° spatial orientation, could be attributed to a deficit of these individuals in account for the effect of spatial orientation on the generation of the appropriate amount of muscle activity (figure 3).

One could argue that the Down syndrome had problem to modulate the muscle activity with changing in target distance and spatial orientation (Figure 3 e 4) because of their inability to modulate the central motor command sent to the motorneuron pool. Several central deficits could be used to explain the deficits, such as poor mielinization, decreased number of neuron synapses. In this case the lack of modulation of the muscle force with change in target distance and spatial orientation could be attributed to their inability to modulate the muscle activity.

One alternative explanation would be impairment in their ability to sense force. Indeed they failed to modulate the grip force (Cole, et al., 1988) and pressed harder on a surface when asked to move faster (Henderson, et al., 1981). A impairment in their ability to modulate muscle impulses with changing in movement distance and direction could be attributed to a deficit in their proprioceptive sense of muscle force. Without a accurate afferent information of the isotonic forces they could not create an internal model of the exact amount of muscle force need to perform different tasks (Sainburg et al., 1993). As a consequence the individuals with DS would continue to send one sub-optimal command do activate their muscles. Indeed, the Down syndrome affects several areas of the motor control system it is possible that the deficit in generate the reversal movements observed in these individuals has a central and/or a peripheric origin.

Musle activities and muscle torques at elbow and shoulder joints

We have showed that normal individuals coupled the amount of muscle activities, the muscle impulses and fingertip linear speed as a building block (Almeida, et al., 2000). The "Building Block Strategy" would reduce the amount of degree of freedom that the SNC has to deal to perform the reversal movements. In this way the rules of this strategy could represent a powerful synergy used by the CNS to control human movements.

The strong coupling among the EMG muscle activities, muscle impulses and the speed of the fingertip observed for normal individuals, favor the idea that the CNS could use an internal transformation model from the intrinsic information (i.e., muscle impulses and the EMG burst of muscle activity) to the extrinsic information (i.e., peak speed of the fingertip).

Our study showed that the individuals with Down syndrome have a problem in coupling the agonist and antagonist muscle activities with the generation of the appropriate level of muscle impulses. Second, they failed to associate the intrinsic variables with the speed of the fingertip. In this case the individuals with Down syndrome would have impairment in this ability to create this internal model. This fits well with the idea that they would have impairment in their ability to sense change in isotonic force. Their movement error occurred when they had intrinsic information about the target (Ferreira, 2000) and it was similar to normal individuals (figure 6). These data suggest that DS individuals would not have perceptual problems in sense dynamical change in joint position.

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Figures legends

Figure 1

Illustration of the task setup. A homemade goniometer was fixed at the elbow joint and connected to a set of small lights. The subjects wore glasses without lenses that allowed the view of the set of the lights, but not of the moving limb. The movements were performed into three target distances and over three spatial orientations (180°, 90°, and 0°). The target distance was defined by the amount of elbow angular excursion as 108°, 126°, and 144°. The initial position of the upper limb was set with the shoulder joint at 45° and elbow at 90°. The limb was fixed at the ceiling with a cable connected at the proximal part of the forearm.

Figure 2

Time series of the extrinsic (fingertip linear velocity) and intrinsic (muscle torque at the joint, and EMG muscle activity) variables describing one movement performed over 0° spatial orientation task. The data are one trial for one normal (left panel) and one DS subject (right panel). The muscle torques are presented for shoulder (solid line) and elbow (broken line) joints. The EMG muscle activities are presented for shoulder (PD - posterior deltoid, AD - anterior deltoid) and elbow (TRI - tricpes, BIC - biceps) joints. The antagonist muscles (AD and BIC) are plotted with inverted signals. The fingertip speed is given in millimeter per second, the muscle torque in Newton meter per second, and muscle activity in volts. The time is given in milliseconds.

Figure 3

Left panel: Elbow excursion and the first (F.), second (S.), and third (T.) elbow muscle impulses. Right panel: The intensity of the first agonist burst, the amount of muscle activity for the first agonist (F. AGO), for the antagonist (ANT), and for the second agonist (S. AGO) EMG bursts. The data were averaged across all eight normal (NN) and eight Down syndrome (DS) subjects for each of the three target distance (108°, 126°, and 144°) over the three spatial orientation (0°, closed circle, 90°, closed squared, and 180°, closed triangle). T.D. was the expected target distance. The elbow excursion was given in degrees, the muscle impulses in Newton meters per second. The amount of EMG activities was normalized by the baseline and because of that there is no units.

Figure 4

Same variables as presented in figure 3 but for shoulder joints, except that we did not have target distance for the shoulder joints.

Figure 5

The first (upper panel) and second (lower panel) peak of the fingertip speeds are presented for the two subjects groups, for the three target distances and the three spatial orientations. Figure caption is the same as presented in figure 3. The data were averaged across all subjects. The speed is given in millimeters per second.

Figure 6

Percentage of error presented for the two subjects groups, for the three target distances and the three spatial orientations. Figure caption is the same as presented in figure 3. The data were averaged across all subjects. Negative values means that the subjects undershoot the target, and positive values means that the subjects overshoot the target.

Figure 7

The first (X-axis) and the second (Y-axis) factors obtained from the Factor Analysis using the Principal Component as the extraction methods. Each symbol represents one of the three landmarks of both elbow and shoulder muscle joints: The first and the fourth landmarks are the time in which the elbow and shoulder reversed direction for the first time (open circle and open diamond). The second and fifth landmarks are the time when both elbow and shoulder muscle torque reverse direction for the second time (open triangle and open inverted triangle). The third and the sixth landmarks are the time when both elbow and shoulder muscle torque reverse direction for the third time (open square and cross). The averaged data are presented for one NN (left panel) and one DS (right panel) for each of the three spatial orientation (0°, 90°, and 180°).

Figure 8

The first (X-axis) and the second (Y-axis) factors obtained from the Factor Analysis using the Principal Component as the extraction methods. Each symbol represents: FPLS (the first peak of the linear speed of the fingertip - closed circle), SPLV (the second peak of the linear speed of the fingertip - open square), FISH (first impulse of the shoulder - open circle), SISH (second impulse of the shoulder - cross), TISH (third impulse of the shoulder - open square with a back slash), FASH (first agonist burst of the shoulder - closed triangle), SASH (second agonist burst of shoulder - single cross), ANSH (EMG antagonist burst of the shoulder - inverted open triangle), FIEL (first elbow impulse - open square with a single cross), SIEL (second elbow impulse - closed square), TIEL (third elbow impulse - closed diamond), FAEL (first agonist burst of the elbow - open triangle), SAEL (second agonist burst of the elbow - closed inverted triangle), and ANEL (antagonist burst of the elbow - open diamond). The averaged data are presented for one NN (left panel) and one DS (right panel) for each of the three spatial orientation (0°, 90°, and 180°).

Table I

Variance Analysis for the elbow and shoulder joints. In the top of the table data for the elbow joint are presented. It shows the effects of the groups (1), spatial orientation (2), group x spatial orientation (1 x 2), distance (3), group x distance (1 x 3), spatial orientation x distance (2 x 3) and group x spatial orientation x distance (1 x 2 x 3) regarding the following variables: elbow angular excursion (E.A.E.), first impulse (F. imp.), second impulse (S. imp.), third impulse (T. imp.), agonist intensity (INTEN), first agonist burst (F. AGO), second agonist burst (S. AGO) and antagonist activity (ANT). For the shoulder joint we showed just the effect of the groups (1), distances (3) and groups x distances (1 x 3) for the three spatial orientations (180°, 90° e 0°) regarding the same variables. Finally, we presented the data for the movement error (M.E.). The plus signal showed that the data are significant ($p<0.05$) and the minus signal, that they are not significant ($p>0.05$).

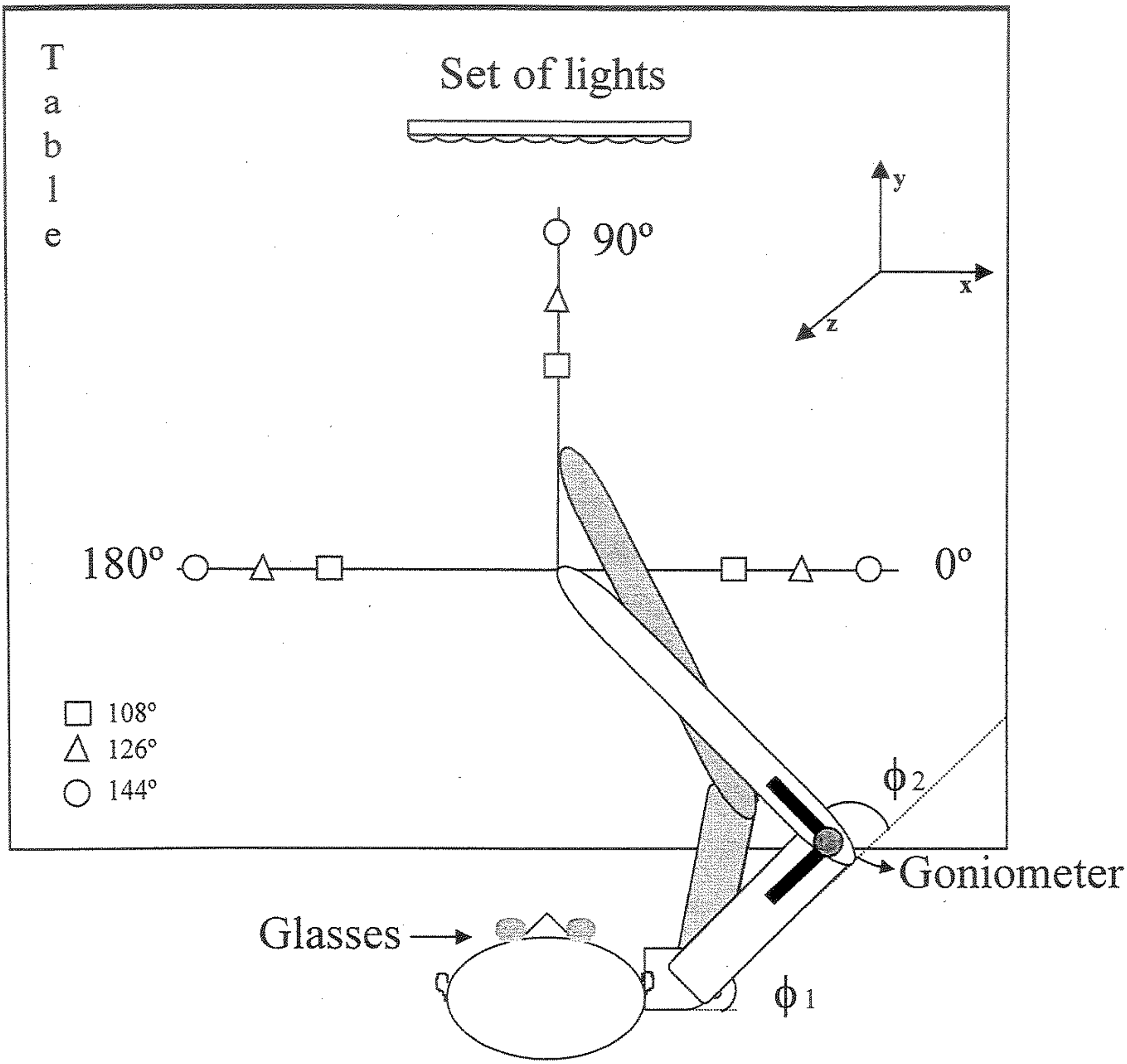


Table
Set of lights
y
x
z
90°
180°
0°
□ 108°
△ 126°
○ 144°
ϕ2
Goniometer
Glasses
ϕ1

Figure 1

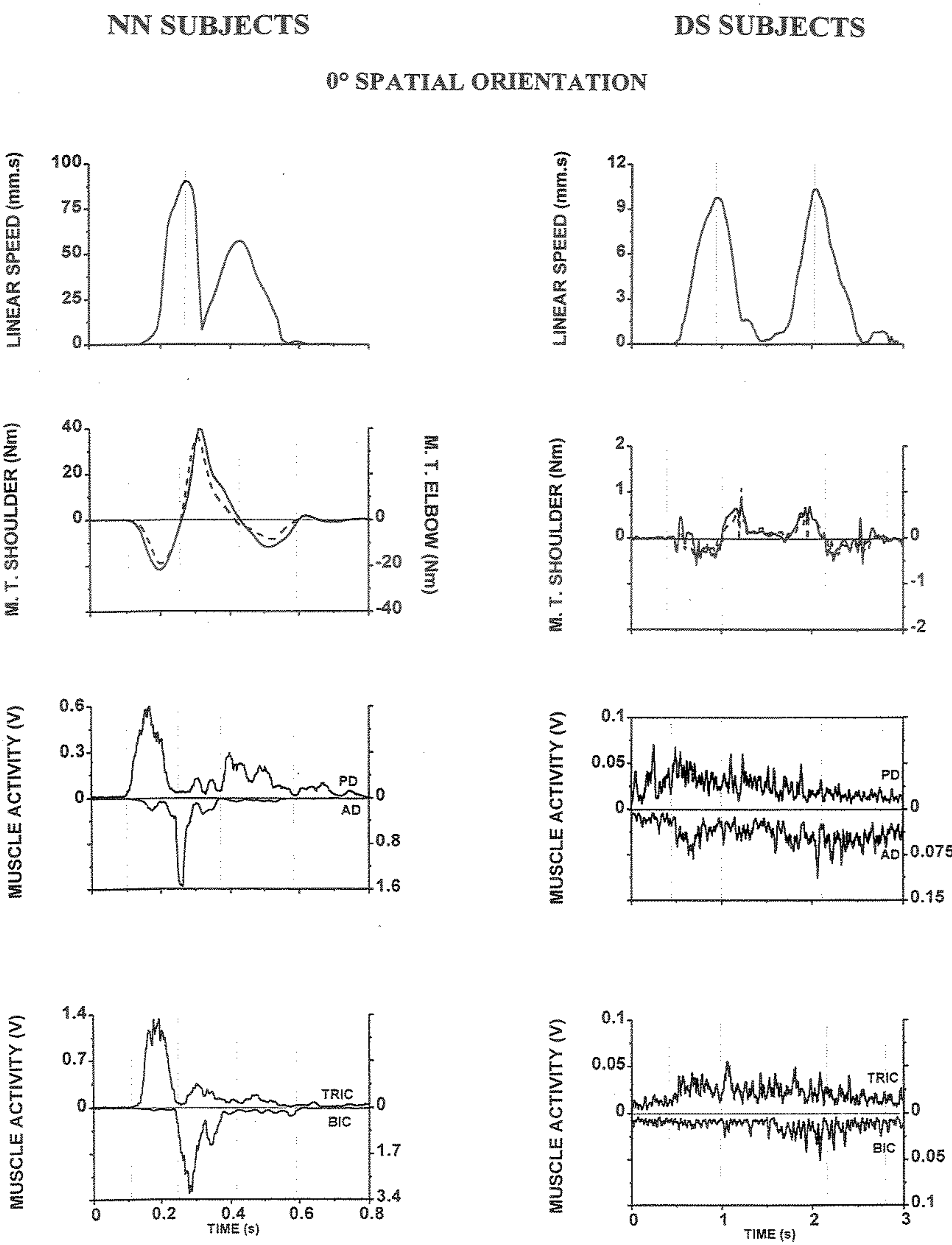
NN SUBJECTS
DS SUBJECTS
0° SPATIAL ORIENTATION
LINEAR SPEED (mm.s)
100
75
50
25
0
12
9
6
3
0
M. T. SHOULDER (Nm)
M. T. ELBOW (Nm)
40
20
0
-20
-40
2
1
0
-1
-2
MUSCLE ACTIVITY (V)
0.6
0.3
0
0.8
1.6
PD
AD
0.1
0.05
0
0.075
0.15
1.4
0.7
0
1.7
3.4
TRIC
BIC
0.1
0.05
0
0.05
0.1
0
0.2
0.4
0.6
0.8
TIME (s)
0
1
2
3
TIME (s)

Figure 2

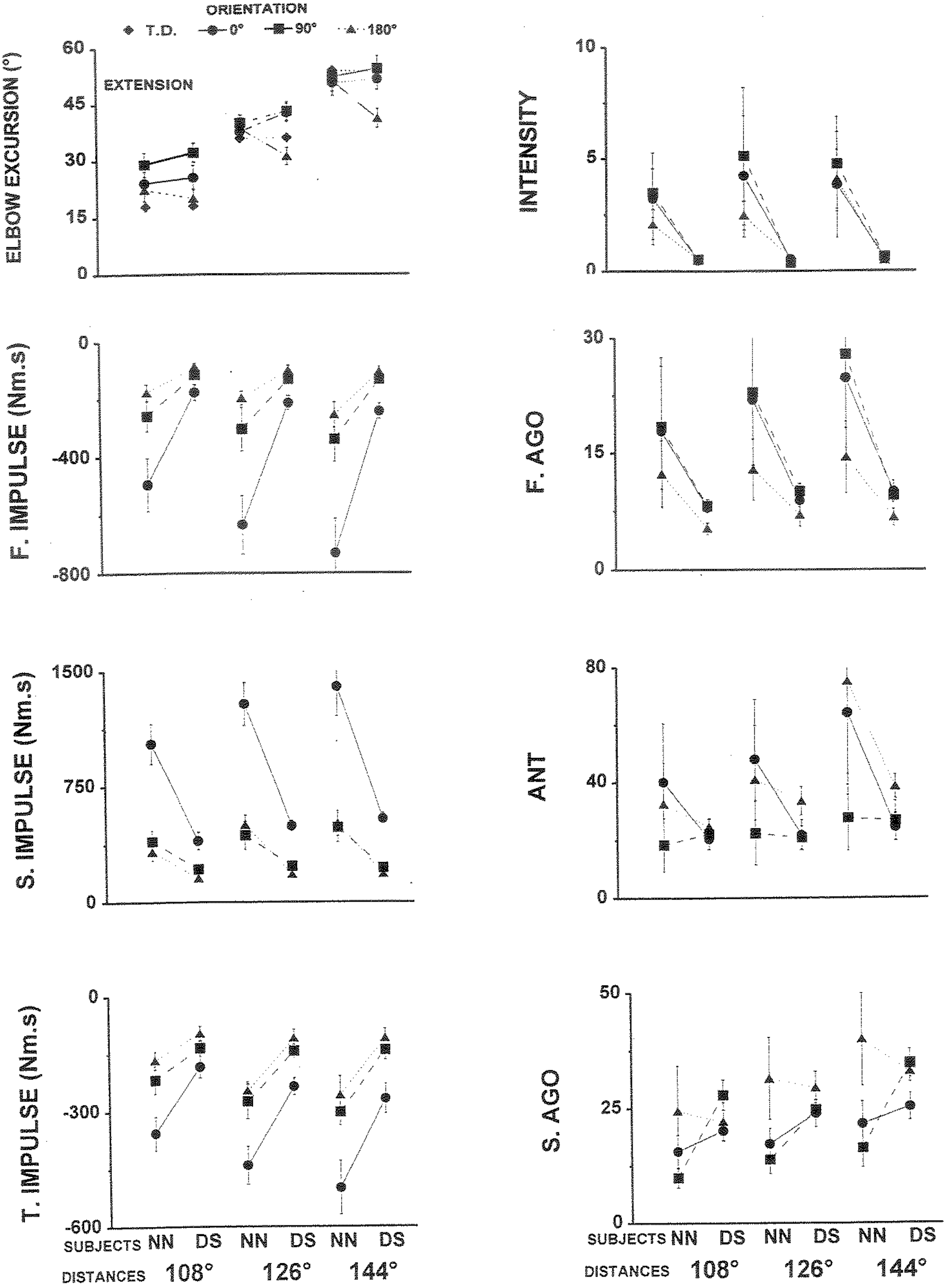

ORIENTATION
◆ T.D.
● 0°
■ 90°
▲ 180°
EXTENSION
ELBOW EXCURSION (°)
60
45
30
15
0
INTENSITY
10
5
0
F. IMPULSE (Nm.s)
0
-400
-800
F. AGO
30
15
0
S. IMPULSE (Nm.s)
1500
750
0
ANT
80
40
0
T. IMPULSE (Nm.s)
0
-300
-600
S. AGO
50
25
0
SUBJECTS NN DS NN DS NN DS
DISTANCES 108° 126° 144°
SUBJECTS NN DS NN DS NN DS
DISTANCES 108° 126° 144°

Figure 3

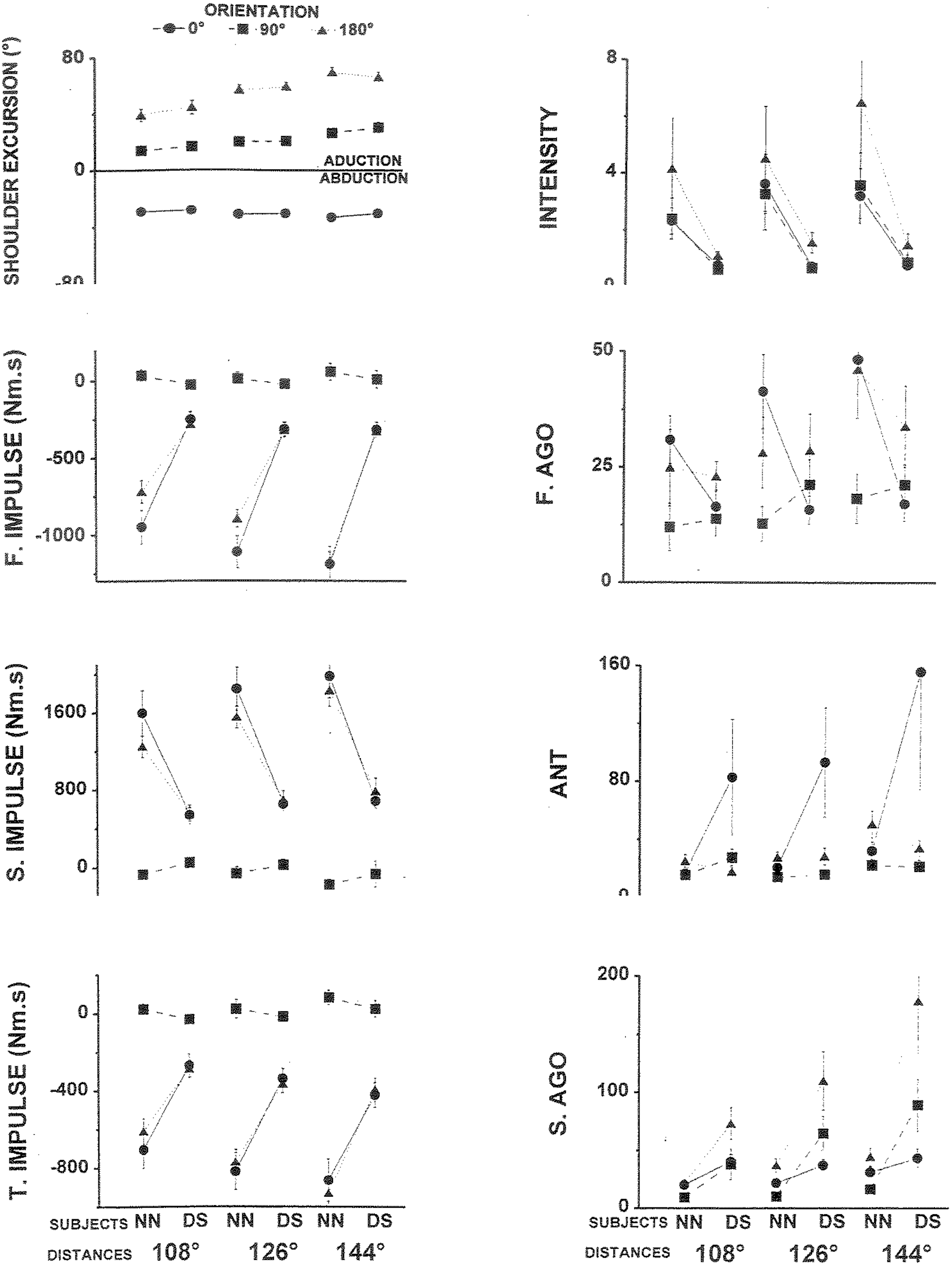

ORIENTATION
0°
90°
180°
SHOULDER EXCURSION (°)
80
0
-80
ADUCTION
ABDUCTION
INTENSITY
8
4
0
F. IMPULSE (Nm.s)
0
-500
-1000
F. AGO
50
25
0
S. IMPULSE (Nm.s)
1600
800
0
ANT
160
80
0
T. IMPULSE (Nm.s)
0
-400
-800
S. AGO
200
100
0
SUBJECTS NN DS NN DS NN DS
DISTANCES 108° 126° 144°
SUBJECTS NN DS NN DS NN DS
DISTANCES 108° 126° 144°

Figure 4

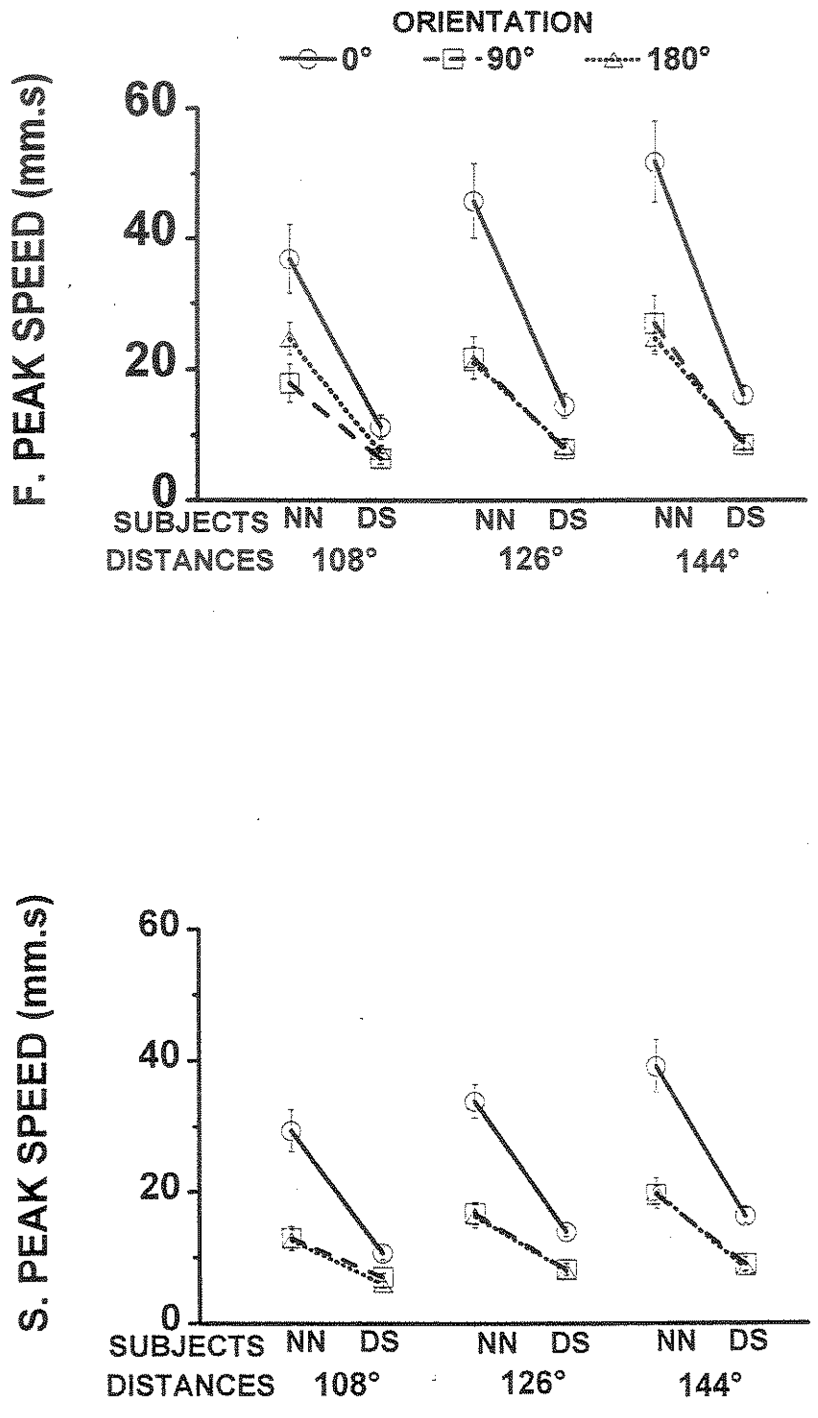
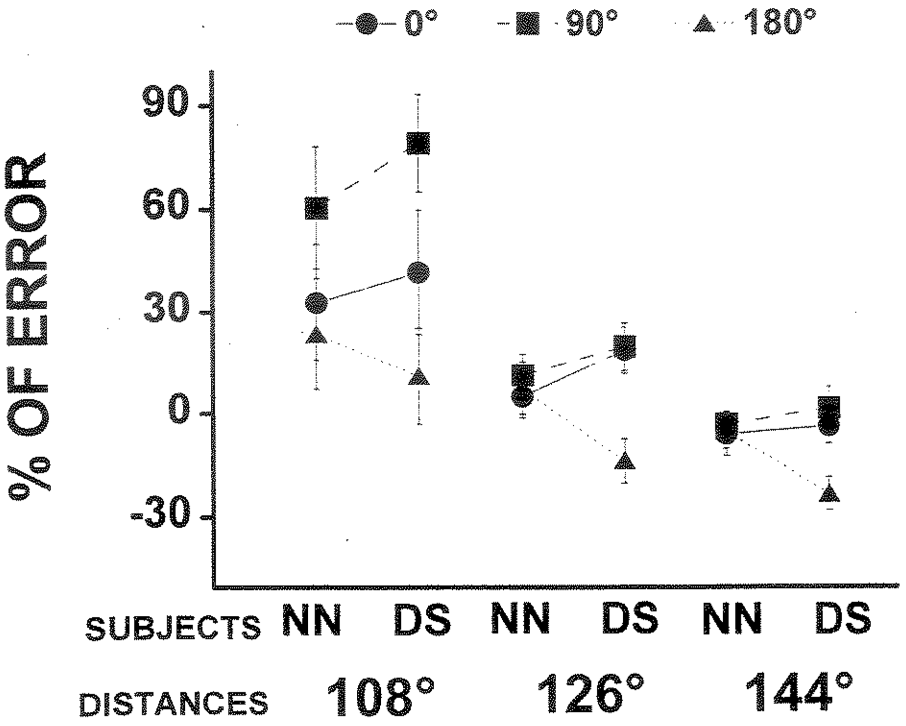

ORIENTATION
0°
-90°
180°
F. PEAK SPEED (mm.s)
60
40
20
0
SUBJECTS NN DS NN DS NN DS
DISTANCES 108° 126° 144°
S. PEAK SPEED (mm.s)
60
40
20
0
SUBJECTS NN DS NN DS NN DS
DISTANCES 108° 126° 144°

Figure 5



0°
90°
180°
% OF ERROR
90
60
30
0
-30
SUBJECTS
NN
DS
NN
DS
NN
DS
DISTANCES
108°
126°
144°

Figure 6

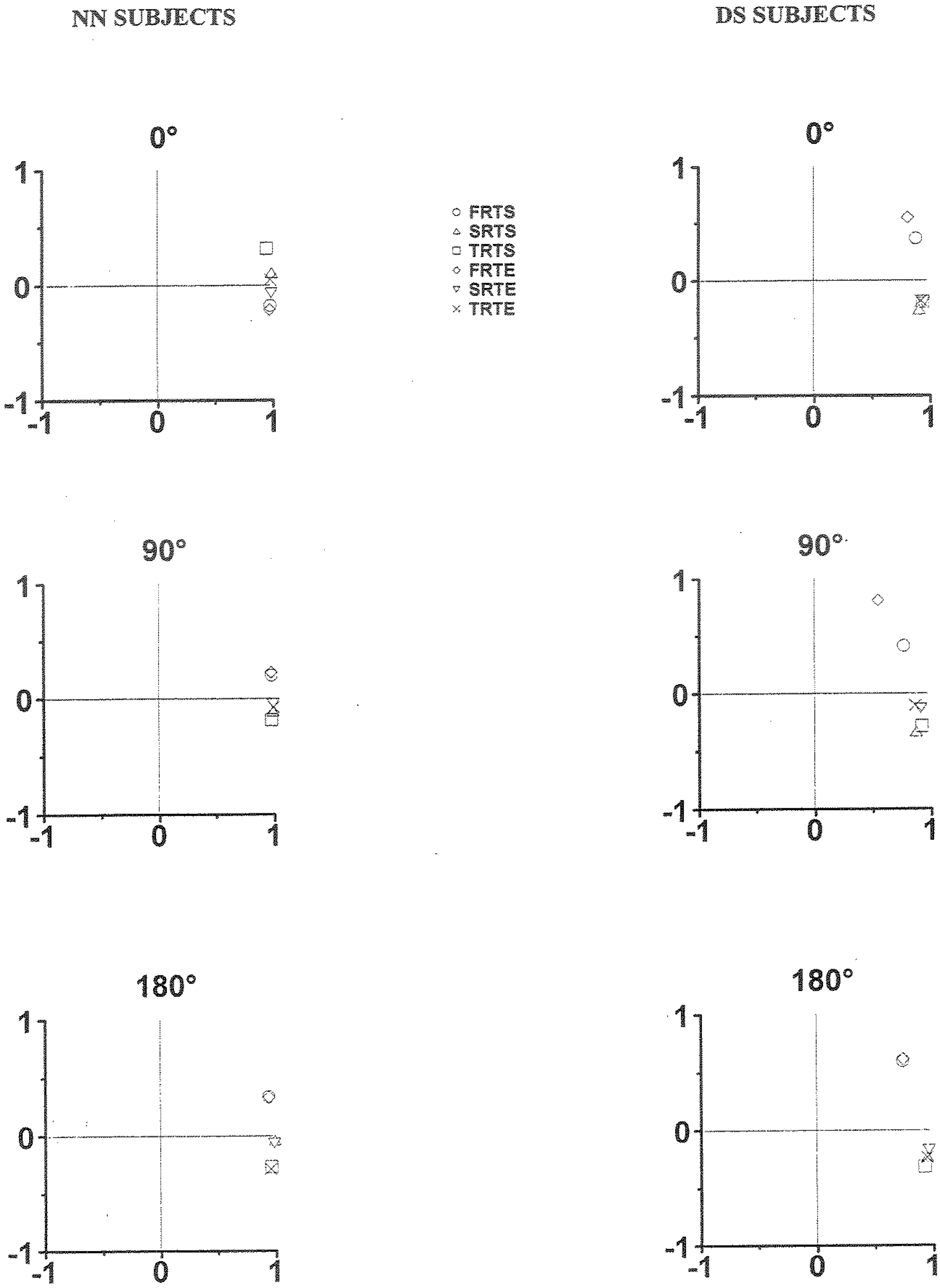

NN SUBJECTS
DS SUBJECTS
0°
90°
180°
○ FRTS
△ SRTS
□ TRTS
◇ FRTE
▽ SRTE
× TRTE
1
0
-1
-1
0
1

Figure 7

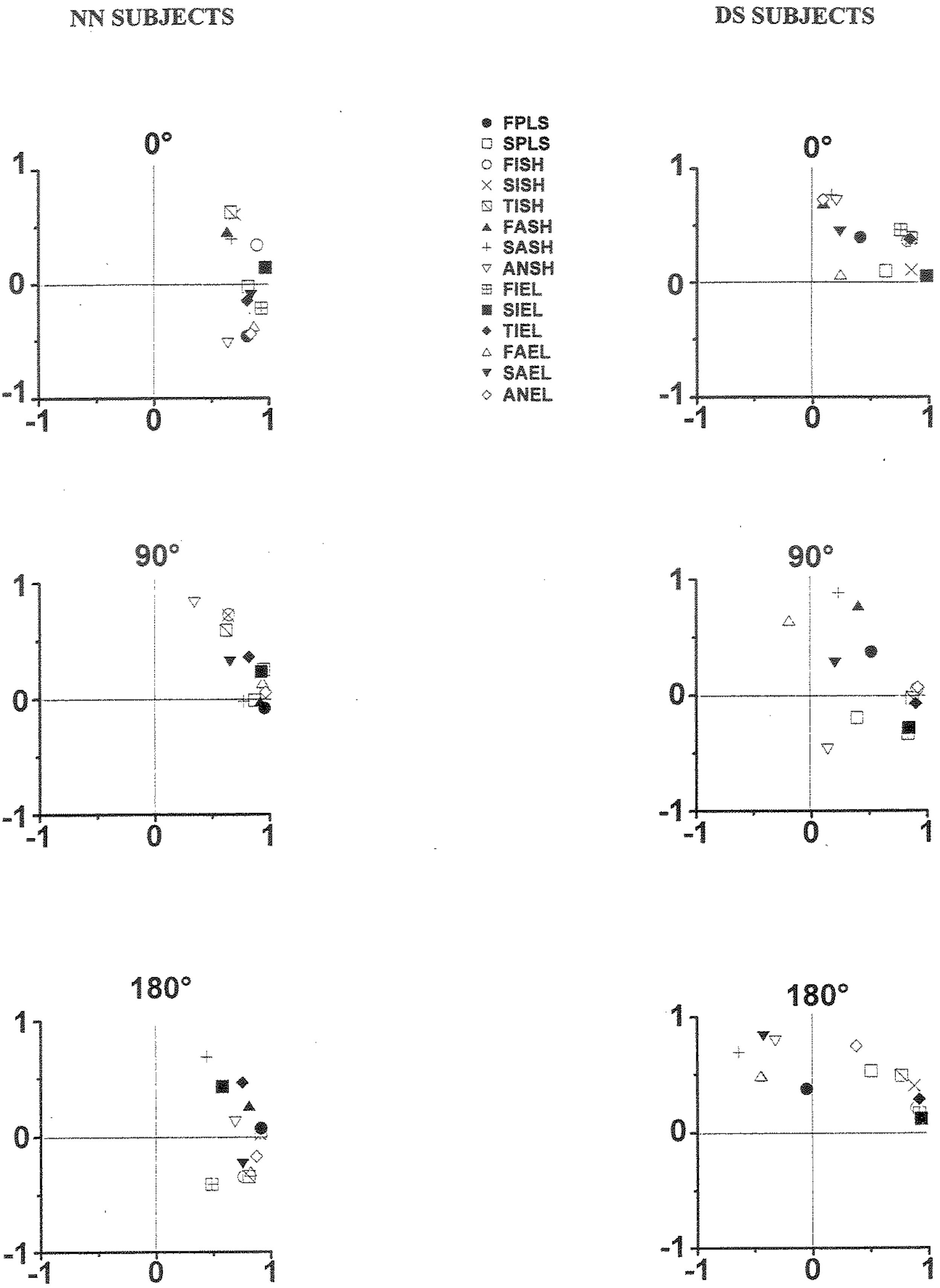
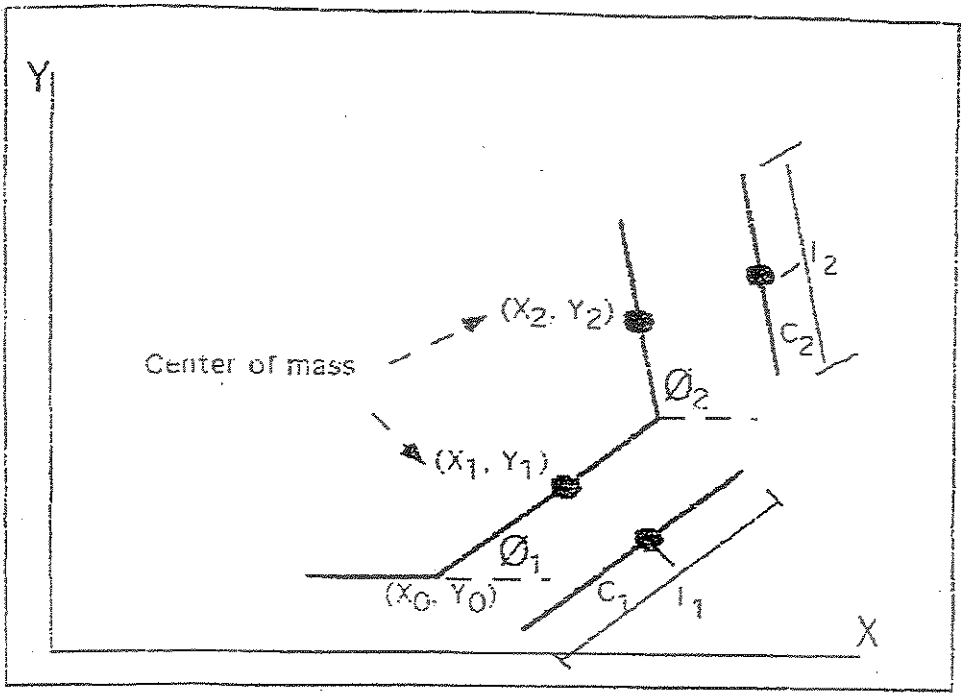

NN SUBJECTS
DS SUBJECTS
0°
90°
180°
● FPLS
□ SPLS
○ FISH
× SISH
⊠ TISH
▲ FASH
+ SASH
▽ ANSH
⊞ FIEL
■ SIEL
◆ TIEL
△ FAEL
▼ SAEL
◇ ANEL
1
0
-1
-1
0
1

Figure 8

			1	2	1 x 2	3	1 x 3	2 x 3	1 x 2 x 3
ELBOW	E. A. E.		-	+	+	+	-	-	-
	F. IMP.		+	+	+	+	+	+	-
	S. IMP.		+	+	+	+	+	+	-
	T. IMP.		+	+	+	+	+	-	-
	INTEN		+	-	-	-	-	-	-
	F. AGO		+	+	-	+	+	-	+
	S. AGO		-	+	+	+	-	-	-
	ANT		-	+	+	+	+	+	-
	M.E.		-	+	+	+	+	+	-
SHOULDER	S. A. E.	180°	-			+	-		
		90°	-			+	-		
		0°	-			+	-		
	F. IMP.	180°	+			+	+		
		90°	-			-	-		
		0°	+			+	+		
	S. IMP.	180°	+			+	+		
		90°	-			-	-		
		0°	+			+	+		
	T. IMP.	180°	+			+	+		
		90°	-			-	-		
		0°	+			+	+		
	INTEN	180°	+			+	+		
		90°	+			-	-		
		0°	+			-	-		
	F. AGO	180°	-			+	-		
		90°	-			+	-		
		0°	+			+	+		
	S. AGO	180°	+			+	+		
		90°	+			-	-		
		0°	+			+	-		
	ANT	180°	-			+	-		
		90°	-			-	-		
		0°	-			+	-		

Table I



$$\mu_2 = m_2c_2\,[-\ddot{x}_1\sin\phi_2 + \ddot{Y}_2\cos\phi_2] + I_2\ddot{\phi}_2$$

$$\mu_1 = \mu_2 + m_1c_1\,[-\ddot{X}_1\sin\phi_1 + \ddot{Y}_1\cos\phi_1] + m_2l_1\,[-\ddot{x}_2\sin\phi_1 + \ddot{Y}_2\cos\phi_1]$$

$$\mathrm{elb}_{\mathrm{INT}} = I\ddot{\phi}_2 - \mu_2$$

$$\mathrm{Sho}_{\mathrm{INI}} = \mu_1 - [I_2 + m_2c_2^2 + I_1 + m_1c_1^2 + m_2l_1^2 + 2m_2l_1c_2\cos\phi_2]\ddot{\phi}_1$$

Where:

1 = shoulder
2 = elbow
I = moment of inertia in relation to the center of mass.
¨ acceleration
μ_2 = elbow muscle torque
μ_1 = shoulder muscle torque
$\mathrm{Elb}_{\mathrm{INT}}$ – elbow interaction torque
$\mathrm{Sho}_{\mathrm{INT}}$= shoulder interaction torque

Equation of Motion

III. Discussão

Os estudos do comportamento da precisão dos movimentos de reversão executados com feedback intrínseco (Almeida *et al*, 2000b) e extrínseco (Almeida *et al*, 2000a) favorecem a idéia de que os movimentos sejam planejados baseado em informações extrínsecas. Esta idéia é suportada por vários outros estudos.

Do ponto de vista da execução dos movimentos, o (SN) parece utilizar uma estratégia intrínseca. A "Building Block Strategy" (Almeida *et al*, 2000b; Marconi *et al*, 2000) demonstra que existe uma co-variação linear entre EMG agonista e antagonista com os torques musculares gerados nas articulações. Este acoplamento entre atividade muscular e torque muscular, facilitaria o controle dos movimentos via a redução do número de variáveis a serem independentemente computadas (Bernstein 1967). Em outras palavras, este modelo seria uma sinergia utilizada pelo SN para reduzir os graus de liberdade disponíveis durante a execução dos movimentos.

Neste modelo, o movimento seria controlado ao nível da execução como um todo, isto é, os dois "bursts" de atividade EMG agonista e o "burst" de atividade antagonista, assim como os três impulsos musculares, estariam co-variando linearmente em duas articulações interligadas em cadeia. Desta forma, bastaria ao SN definir os parâmetros que caracterizam a fase inicial do movimento; por exemplo, o primeiro "burst" de atividade EMG agonista e/ou o primeiro impulso. Isto ocorreria porque as outras duas fases do movimento estariam linearmente correlacionadas com a primeira. Para que o primeiro "burst" de atividade agonista gere o primeiro impulso, bastaria que o SN o modulasse de acordo com as regras estabelecidas pela "Speed Insensitive Hyphotesis" (Gottlieb *et al*, 1989). Como demonstramos, os padrões de atividade EMG e os impulsos foram modulados com o aumento da distância do alvo, para os indivíduos normais (Almeida *et al*, 2000b).

Como a geração da atividade EMG antecede o início da geração do impulso muscular e este por sua vez, antecede o início da velocidade linear do dedo indicador, poder-se-ia argumentar que o SN estaria controlando a atividade EMG. No entanto, antes do comando central (potencial de ação) chegar ao neurônio motor alfa, é necessário que o SN já tenha tomado várias decisões no que diz respeito à escolha e ao planejamento do movimento a ser executado.

A co-variação linear entre os parâmetros intrínsecos (atividade muscular e torque muscular) e extrínsecos (velocidade linear do dedo indicador) não determina de forma causal qual seria a variável controlada pelo SN. O SN poderia escolher qualquer uma delas ou mesmo codificá-las a partir de um comando central. Esta questão não pode ser respondida a partir dos dados obtidos neste estudo.

Em suma, a "Building Block Strategy" simplificaria a tarefa do SN em controlar movimentos ao nível articular, ao nível da coordenação entre as articulações e ao nível da transformação intrínseca para extrínseca. A correlação linear entre estas variáveis é compatível com a sugestão de que em algum nível superior, as propriedades cinemáticas dos movimentos poderiam ser planejadas em termos experimentais, tais como aquelas fornecidas por observação visual das trajetórias. A execução destes planos cinemáticos entretanto, é em termos das forças esperadas para produzi-las. Essas forças podem ser estimadas de um modelo interno da dinâmica dos segmentos e sua carga externa.

A aplicação desta estratégia no entendimento de como os movimentos de indivíduos portadores da Síndrome de Down (SD) são controlados nos permite algumas conclusões. A

"Building Block Strategy" (Almeida *et al*, 2000b; Marconi *et al*, 2000) não é uma estratégia fruto de um artefato metodológico que seria observada em movimentos normais e descoordenados. Como vimos, os indivíduos portadores da SD, falharam em acoplar os parâmetros extrínsecos e intrínsecos na forma observada para os indivíduos neurologicamente normais (NN). Segundo, estes indivíduos utilizam um padrão de co-ativação generalizada da musculatura agonista e antagonista (Almeida 1993; Almeida *et al*, 1991; Almeida *et al*, 1994; Latash *et al*, 1993). Eles falharam em modular as atividades EMGs e os torques musculares nos padrões definidos pela "Speed Insensitive Hyphotesis" (Gottlieb *et al*, 1989). Esta incapacidade em modular as atividades EMGs, poderia explicar a falta de acoplamento entre esta variável e os impulsos musculares.

Terceiro, o primeiro e o segundo picos da velocidade linear do dedo indicador não se correlacionaram linearmente entre si para os indivíduos SD. Estes indivíduos também falharam em acoplar os parâmetros intrínsecos (atividade muscular e torque muscular) e extrínsecos (velocidade do dedo indicador). Estes dados mostram que os indivíduos SD falharam em planejar o movimento de reversão como um todo. A origem da dificuldade destes indivíduos em acoplar os parâmetros extrínsecos e intrínsecos pode ser central ou periférica. No entanto, fica difícil atribuir a qualquer uma destas estruturas a incapacidade de acoplamento.

Como vimos, os indivíduos portadores da SD têm problemas em modular as atividades EMGs e os impulsos musculares com o aumento da distância do alvo e com a mudança das orientações espaciais. No entanto, eles foram capazes de realizar os movimentos de reversão com feedback intrínseco tão precisos quanto os indivíduos NN (Almeida *et al*, 2000b; Ferreira

2000). Eles também foram capazes de realizar os movimentos nas mesmas orientações espaciais que os indivíduos NN.

Estes dados favorecem a idéia de que o problema destes indivíduos não está na capacidade de perceber a mudança no deslocamento angular ou na posição do membro no espaço. Outros estudos têm demonstrado uma boa percepção espacial nesta população (Henderson 1986). O problema nestes indivíduos poderia estar na percepção da geração da força muscular. Neste caso, teríamos um problema periférico. Uma limitação nas informações do Órgão Neurotendinoso de Golgi, por exemplo, poderia dificultar o desenvolvimento de um modelo interno (Sainburg *et al*, 1995) das forças musculares necessárias à execução de uma tarefa motora. Uma outra alternativa seria um déficit nas estruturas corticais que dificultaria o envio de estímulos para o neurônio motor alfa (potencial de ação).

Existem vários candidatos que poderiam explicar a falta de modulação das atividades EMGs com a mudança da distância do alvo e da orientação espacial nestes indivíduos. Por exemplo, a redução do número de sinapses, uma mielinização pobre, a redução no peso do cerebelo (Aylward *et al*, 1997; Crome *et al*, 1966). Vale lembrar que, com o treinamento, estes indivíduos foram capazes de aprenderem a modular as atividades EMGs da forma observada para os indivíduos NN (Almeida, 1994).

IV. Anexos

IV. I. Trabalhos Publicados

PERCEPTUAL-MOTOR BEHAVIOR IN DOWN SYNDROME

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Simon Fraser University

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Digby Elliott
McMaster University

Human Kinetics

7

CHAPTER

Sensorimotor Deficits in Down Syndrome: Implications for Facilitating Motor Performance

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Key words

sensorimotor ◆ clumsiness ◆ practice ◆ muscle activation ◆
synergy ◆ hypotonia ◆ reaction time ◆ movement sequences ◆
somatosensory information ◆ intervention ◆ decision-making

Casual observation of the everyday movements of individuals with Down syndrome (DS) gives the impression of "clumsiness" or even "extreme motor clumsiness" (Frith & Frith, 1974). Several studies have shown that the movements of individuals with DS are slower, less smooth, and more variable from trial to trial when compared with the movements of the overall population. Evidence of slowness and lack of smoothness has been observed even in simple elbow-flexion movements (Almeida, Corcos, & Latash, 1994; Latash & Corcos, 1991), as well as in multijoint pointing tasks (Aruin & Almeida, 1997). The difference in motor performance between neurologically normal individuals and individuals with DS suggests that individuals with DS might have specific, experimentally identifiable sensorimotor deficits. Examples include deficits in timing (Henderson, Morris, & Frith, 1981), deficits in motor programming (Frith & Frith, 1974), and deficits in the ability to adjust the rate at which grip force is generated (Cole, Abbs, & Turner, 1988).

On the other hand, it has also been argued that there are either no specific motor deficits associated with DS or that they are not really significant in comparison with the other deficits observed. For example, Wang (1996) has suggested that verbal short-term memory skills are diminished in individuals with DS compared to other individuals with mental retardation. However, visual-motor skills are comparatively well preserved. Based on this and other observations, including impaired verbal communication skills, he has suggested that individuals with DS should be taught sign language. Also, Latash and Anson (1996) have argued that the way in which the movements of individuals with DS are performed is a reflection of an impaired decision-making process and not necessarily a consequence of a primary motor deficit. Specifically they have suggested, "Our basic assumption implies that the CNS of a person whose decision-making component of the system for movement production is somewhat impaired, may 'prefer' to facilitate clumsy movements rather than risk total failure during motor performance" (p. 66). Their argument suggests that although movement patterns and muscle activation patterns may differ from those observed in neurologically normal individuals, they are optimal adaptations given a primary impairment of decision-making.

In this chapter, we will review the evidence that relates to whether individuals with DS have specific sensorimotor deficits. We shall first discuss similarities in the control of movement between neurologically normal individuals and those with DS, and then consider differences. We will use this analysis to draw conclusions about the extent to which intervention should be used to facilitate motor performance. Whenever possible we will refer to studies that have used electromyograph
Electromyography enables one to determine whether the patterns of motor co

mands are the same or different between groups of individuals. Studies of movement kinematics alone do not allow one to distinguish between problems of how movements are represented and how they are implemented (Anwar, 1986).

Similarities to Individuals Without Down Syndrome

In the following review, we look at many studies that show consistent similarities between control subjects and individuals with DS in the control of movement distance and speed, in the improvement of motor performance with practice, and in linear synergy. In our research, we also have not found any evidence of a causal relation between hypotonia and motor impairment in individuals with DS.

Control of Movement Distance

Numerous studies have investigated the muscle activation patterns associated with movements of different extents in neurologically normal individuals (Benecke, Meinck, & Conrad, 1985; Gottlieb, Chen, & Corcos, 1996a; Wachholder & Altenburger, 1926). Typically, a biphasic or triphasic pattern of muscle activation is observed in which an agonist burst of muscle activation is followed by an antagonist burst, and then sometimes a second agonist burst. For movements of different distances, the first agonist and antagonist bursts scale in a very consistent way. For movements that exceed 10° to 15°, the angle, velocity, acceleration, and net joint torque all rise at similar rates at the beginning of the movement. The same is true for the agonist EMG (Pfann, Hoffman, Gottlieb, Strick, & Corcos, 1998). For short movements and very strong experimental subjects, the initial EMG activation and movement kinematics diverge at the onset (Pfann et al., 1998). The agonist EMG also increases in duration for longer movements. The antagonist EMG is delayed in time. This is true for movements that are constrained by a manipulandum (Gottlieb, Corcos, & Agarwal, 1989) and also movements that are not constrained (Almeida, Hong, Corcos, & Gottlieb, 1995).

Movements of different extents have been studied in individuals with DS by Latash and Corcos (1991). The angle, velocity, and acceleration traces rose at the same rate in several of the subjects in their study. The agonist muscles (biceps and brachioradialis) also rose at the same rate, and the EMG duration increased with distance. In a follow-up study (Almeida, Corcos, & Gottlieb, manuscript in preparation), individuals with DS were asked to perform movements over three different distances using the same paradigm as Almeida et al. (1995). The data in figure 7.1 illustrate the performance of an unconstrained single-joint elbow movement performed by an individual with DS. The agonist muscle (biceps) was activated generating a flexor torque that accelerated the limb toward the target. The initial rate of rise of the agonist EMG over the first 50 ms was independent of movement distance. The agonist EMG increased in both duration and area. After about 100 ms, the antagonist muscle was activated to decelerate the limb.

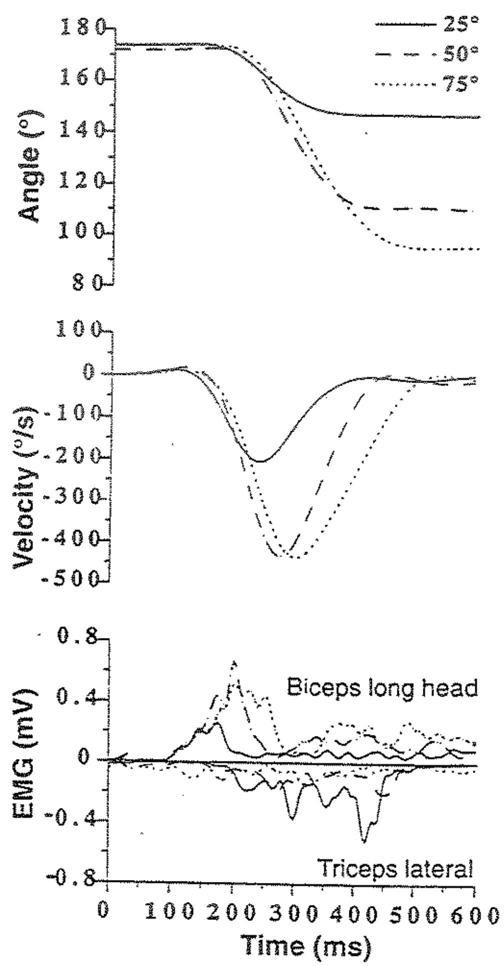

Figure 7.1 Elbow angle, velocity, and electromyographic activity (long head of biceps, agonist muscle, and lateral head of triceps, antagonist muscle) during unconstrained elbow-flexion movements performed in the vertical plane over three angular excursions (25°, 50°, and 75°). The time-series data are from an individual with DS, averaged across 11 trials and aligned according to the agonist (biceps) onset (100 ms). The EMG signals were smoothed by a 10 ms rectangular window. The subject was asked to move "as fast as possible."

Data taken from G.L. Almeida, D.M. Corcos, and G.L. Gottlieb. (manuscript in preparation). *Unconstrained single-joint movements of different distances in individuals with DS.*

Even though movement speed was slower for individuals with DS, the velocity increased for both groups of individuals with increasing angular excursion, as was also true in a previous study (Almeida et al., 1994). From three sets of studies, we can conclude that the capability of individuals with DS to modulate the agonist and antagonist EMG bursts is not impaired.

Control of Movement Speed

Several studies have shown that increases in movement speed are caused by increasing the intensity of excitation to the motoneuron pool of both agonist and antagonist muscles (Corcos, Gottlieb, & Agarwal, 1989; Mustard & Lee, 1987). This increase in neural excitation produces agonist and antagonist EMGs that have different slopes. The steeper the slope of the agonist EMG, the faster the rise of torque and, consequently, the faster the movement. Almeida et al. (1994) were interested in whether this ability to modulate the input to the motoneuronal pool is preserved in individuals with DS given that Cole et al. (1988) had previously reported that individuals with DS did not adjust the rate at which grip force was produced. The data in figure 7.2 depict angle, velocity, acceleration, and agonist and antagonist EMGs for one set of movements performed "as fast as possible" and another set of movements performed at a "comfortable speed" before and after extensive practice. Even prior to practice (see pretest), the pattern of muscle activation was very similar to that observed in individuals who are neurologically normal. Again, there was no obvious movement deficit in the neural activation of muscle.

Improvement of Motor Performance With Practice

It is well known that many individuals with DS have been institutionalized and that this can influence intellectual function (Wisniewski, Miezejeski, & Hill, 1988). As such, they may not have received the same environmental stimulation and opportunity to take part in physical activity as noninstitutionalized individuals. In addition, societal expectations of the capacity of individuals with mental retardation to take part in physical activity is often considerably less than the expectations for neurologically normal individuals. Therefore, part (maybe a large part) of the reduced performance that is observed in individuals with DS or other groups of individuals with mental retardation may be caused by their lack of experience: the so-called experiential hypothesis (Newell, 1989). Therefore, it is important to know the extent to which individuals with DS can improve their motor performance when given the opportunity for extensive practice.

Hulme and Mackenzie (1992) have suggested that individuals with DS have a selective deficit in acquiring motor programs because they showed less improvement across trials than other groups of children. This statement was based on a study by Frith and Frith (1974), who compared the performance of a group of normal individuals, autistic individuals, and individuals with DS on a pursuit-tracking task. Although it is true that the individuals with DS did not improve on the tracking task

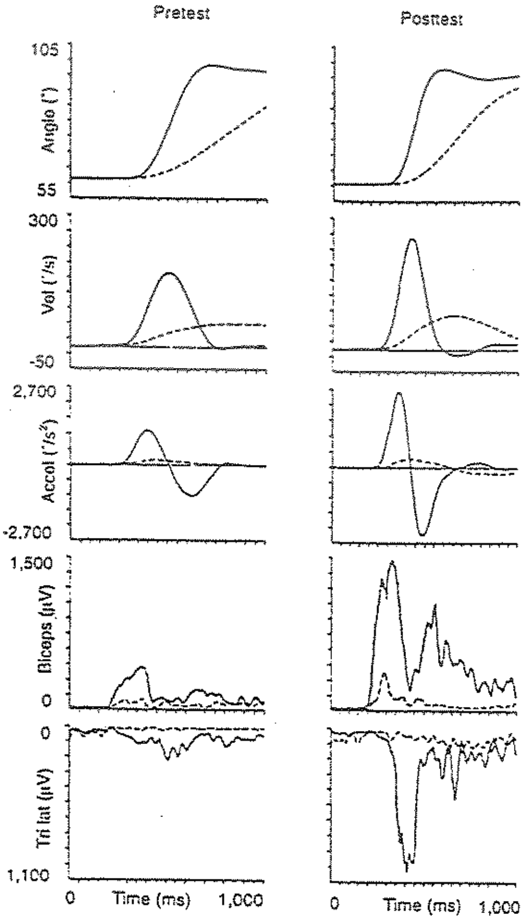

Figure 7.2 Angle, velocity (vel), acceleration (accel), biceps brachii muscle (biceps), and lateral head of triceps brachii muscle (tri lat) electromyographic (EMG) signals for elbow-flexion movements for the pretest and the posttest for subject S2. The EMG data have been filtered with a 25 ms moving average. The lateral head of triceps brachii muscle has been inverted. The subject was asked to move as fast as possible (solid line) and at a comfortable speed (broken line) over the same distance (36°). The data are averages of four and five trials, respectively, for the pretest and the posttest aligned at the onset of the agonist EMG activity (200 ms).

Reprinted from G.L. Almeida, D.M. Corcos and M.L. Latash, *Physical Therapy*, Alexandria, VA, American Physical Therapy Association, 1994, 1000-1012, with permission of the American Physical Therapy Association.

only two 3-minute sessions of practice with a 5-minute break were used. This is hardly sufficient time or practice to draw such a strong conclusion about impaired motor learning.

In contrast to this somewhat negative view of motor learning, Kerr and Blais (1985) had three groups of individuals (control, individuals with mental retardation, and individuals with DS) practice for several hundred trials on a discrete-tracking task involving movements over different distances and in different directions. The individuals with DS were slower than the other two groups of individuals. Also, as can be seen in figure 7.3, the individuals with DS were still improving their performance after 800 trials and showed no signs of reaching a plateau.

Improved performance as a result of practicing movements is not limited just to the limb practicing the task. Elliott (1985) had individuals practice tapping with one hand and showed there was a considerable increase in tapping speed in the nonpracticed hand for individuals with DS. Other studies have also shown that improved performance can result from practice (Kanode & Payne, 1989; Kerr & Blais, 1987).

Because individuals with DS can improve motor performance in a variety of motor tasks, the question arises as to whether they do so using normal patterns of

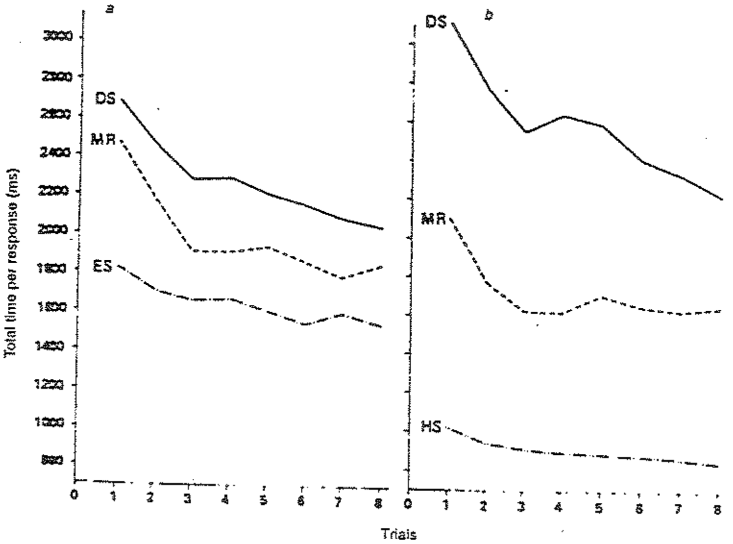

Figure 7.3 Total response time: *(a)* functional age match and *(b)* CA match. DS = Down syndrome, MR = mentally retarded, HS = high school, and ES = elementary school.

Reprinted, by permission, from R. Kerr and C. Blais, 1985. "Motor skill acquisition by individuals with Down Syndrome." *American Journal on Mental Deficiency* 90(3): 313-318.

muscle activation or whether they use different patterns of muscle activation. Almeida (1993) attempted to determine if movement slowness observed in individuals with DS is due to lack of experience or due to a specific inability to increase the intensity of neural activation to the motoneuron pool. He trained individuals with DS to perform a specific, 36° single-joint elbow-flexion movement. Even without training, individuals with DS can make movements at different speeds using "normal" patterns of muscle activation, as was shown in figure 7.2. However, before training, the total amount of EMG activity was small, which was reflected in a movement speed that was half of that observed for neurologically normal individuals (Corcos, Jaric, Agarwal, & Gottlieb, 1993). With extensive practice, all individuals increased movement speed substantially. They did so by learning how to increase the intensity with which the agonist and antagonist muscles were activated and by decreasing the antagonist latency as shown in figure 7.4. This was the same muscle activation pattern used by neurologically normal individuals who followed a similar training protocol (Corcos et al., 1993). More importantly, the movement speed of each of the eight individuals with DS improved, as shown in figure 7.5.

The movement accuracy of the individuals with DS was very high at the beginning of training and did not deteriorate with increasing speed. In other words, they did not trade off speed for accuracy with training (cf. Kerr & Blais, 1987). Five of the eight individuals did not display muscle coactivation. Three out of eight individuals who exhibited a pattern of muscle coactivation before training shifted to a reciprocal pattern of muscle activation after training. We can therefore conclude that individuals with DS do not have a neurological deficit in their ability to exhibit "normal" patterns of muscle activity. The remarkable improvement with training supports the idea that they can change from a pattern of muscle coactivation to a more universal reciprocal pattern of muscle activation (the posttest in figure 7.2). What was most impressive was that not only did performance improve at the practiced distance, it also improved at other distances that had not been practiced (18°, 54°, and 72°). This dramatic transfer in motor performance can be seen most clearly in figure 3 of Latash (1992), in which the performance of individuals with DS was compared to that of neurologically normal individuals.

The issue has been raised as to whether such dramatic performance improvement can occur only in artificial laboratory tasks (Connolly, 1994). In this context, a study by Perán, Gil, Ruiz, and Fernandez-Pastor (1997) is most revealing. They enrolled a group of 20 adolescents with DS in an extensive physical training program. The results were impressive. For example, the mean time to perform the 50 m dash dropped from 15.39 seconds to 10.69 seconds. Other measures of physical performance also improved dramatically. This study suggests that performance can dramatically improve in whole-body tasks that require balance and coordination.

Linear Synergy

The motor control studies that we have reviewed so far have all been restricted to single degree-of-freedom elbow joint movements. This raises the possibility that

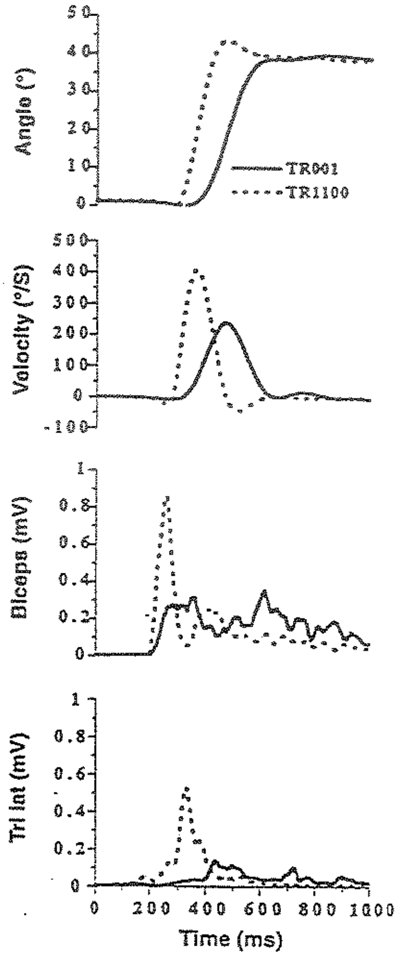

Figure 7.4 Elbow angle, velocity, and electromyographic activity (biceps long head, agonist muscle, and triceps lateral head, antagonist muscle) during constrained elbow-flexion movements performed over 36° "as fast as possible." The time-series data are from one individual with Down syndrome for the 1st and 1,100th trial. The data were aligned according to the agonist onset (200 ms). The EMG signals were smoothed by a 10 ms rectangular window.

Data taken from G.L. Almeida. 1993. Practice, transfer and performance enhancement of fast single-joint movements in individuals with Down syndrome. Unpublished. Ph.D. dissertation, Iowa State University.

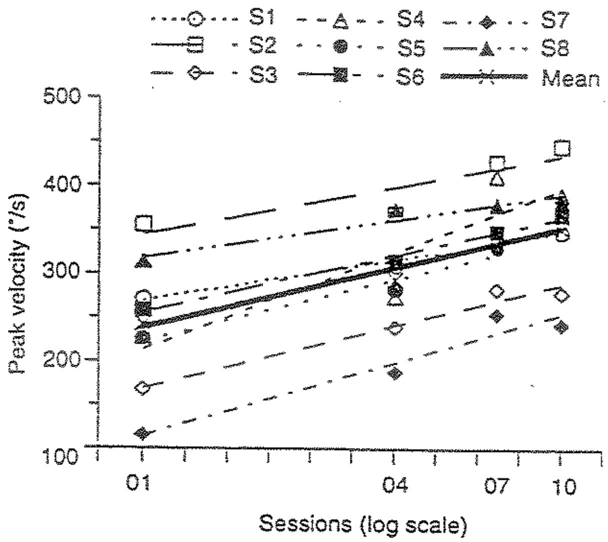

Figure 7.5 Increases in movement speed over the 1st, 4th, 7th, and 10th practice sessions. The data are for each of the eight individuals with Down syndrome and the group average (bold line) during the performance of the 36° movements. The data are plotted on a log scale.

Data taken from G.L. Almeida. 1993. Practice, transfer and performance enhancement of fast single-joint movements in individuals with Down syndrome. Unpublished Ph.D. dissertation, Iowa State University.

individuals with DS might have greater difficulty making movements that require the coordination of multiple joints since these are the types of movements that are generally performed in daily activities of living, such as buttoning one's clothes or drinking a glass of juice. It has been argued that in order to generate a multijoint movement, an internal model of the movement is necessary and that proprioception is necessary to continuously update this model (Ghez & Sainburg, 1995). Sainburg, Ghilardi, Poizner, and Ghez (1995) hypothesized that if proprioception was not available or was impaired, individuals would have difficulty performing reversal movements to targets. They hypothesized that the extent of the movement impairment would be related to the magnitude of the interaction torque generated during the movement. They had individuals who were neurologically normal and patients with large fiber neuropathy make reversal movements to six targets and found the greatest deviations from a straight-line path for movements at which the interaction torque was the greatest.

Given that there is very little evidence about the role of the proprioceptive system during movement in individuals with DS, Almeida, Corcos, and Hasan (manuscript in preparation) conducted a study similar to that of Sainburg and colleagues, in which subjects made reversal movements to a target in four different directions. Four target locations were employed for which the required elbow angular excursion was comparable, but the required shoulder excursion varied from 10° abduction to 75° abduction. The idea was that if individuals with DS have a proprioceptive deficit, they would not make the movements using a straight path. Instead, the path would be very erratic and similar to that observed by Bastian, Martin, Keating, and Thach (1996) in cerebellar patients and Sainburg et al. (1995) in patients with a proprioceptive deficit. In contrast to these studies, Almeida, Corcos, and Hasan (manuscript in preparation) found that the movement path of individuals with Down syndrome was not erratic. Shoulder and elbow torques were calculated and plotted against one another. Although the correlation of the joint torques was higher in individuals who are neurologically normal than in individuals with DS, the correlation was still high in individuals with DS. Also, the ratio of the joint torques changed systematically for movements of different directions. The fact that there was a high correlation between shoulder and elbow torque and that the ratio of joint torques changed with movement direction is consistent with a series of studies on pointing movements performed by Gottlieb and colleagues. They have shown that there is a very high correlation between shoulder torque and elbow torque for these movements and have referred to this relation as "linear synergy" (Gottlieb, Song, Hong, Almeida, & Corcos, 1996; Gottlieb, Song, Hong, & Corcos, 1996). They have also shown that the ratio of joint torques changes very systematically for movements of different directions (Gottlieb, Song, Almeida, Hong, & Corcos, 1997). As such, the findings by Almeida, Corcos, and Hasan (manuscript accepted pending revision) on reversal movements suggest that there is no clear movement impairment in individuals with DS for reversal movements that involve the coordination of two joints.

Torque, Angle Characteristic, and Hypotonia

Nearly every article or book chapter on DS refers to the fact that individuals with DS have reduced muscle tone, or hypotonia, especially during infancy (Coleman, 1978; McIntire, Menolascino, & Wiley, 1965). Whereas few would dispute that babies and young infants have clinically identifiable hypotonia, it is by no means clear that this is true of young adults. Part of the problem with investigating hypotonia is establishing an operational definition. If a muscle is pressed or lightly pinched, a certain amount of tissue deformation will be produced. This deformation is larger for individuals with DS, especially when young (Morris, Vaughan, & Vaccaro, 1982). However, the body type of individuals with DS is also different, with a much greater tendency to endomorphy (Perán et al., 1997), and this may partially account for the observation that there is greater deformation in their muscles when pinched. Although hypotonia has often been measured by palpation, we are unaware of any study that has explicitly related hypotonia measured by this method to impaired motor control in adolescents or adults with DS.

Other groups of investigators have investigated "muscle tone" in terms of segmental motoneuron pool excitability. Shumway-Cook and Woollacott (1985) investigated the postural control of individuals with DS by means of applying perturbations to individuals standing on a movable force platform. They reported the presence of monosynaptic reflex latencies in young children with DS and concluded that any balance problems are related to defects in higher level postural mechanisms and not to decreased segmental motoneuron pool excitability. Latash, Almeida, and Corcos (1993) measured the joint compliant characteristic of individuals with DS. They applied stretches to the biceps muscle by applying different torques at the elbow joint. The individuals were instructed "not to react" voluntarily. Then the amount of joint displacement was measured. The slope of the relation between angle and torque was used as a measure of "joint stiffness," and the values calculated for individuals with DS were similar to those calculated for neurologically normal individuals (Latash & Gottlieb, 1990). Unlike muscle palpation, stretching a muscle takes into account the integrity and functionality of the neural network acting on the muscle-joint complex in addition to the visco-elastic properties of the muscles. Given that measuring "muscle tone" through palpation has not been directly related to impaired motor control and that the studies of Shumway-Cook and Woollacott and Latash and colleagues suggest that the spinal neural networks are intact, we concur with Anson (1992): "In sum, the role of hypotonia in accounting for movement disorders in DS individuals can no longer be considered a default explanation when all alternatives fail" (p. 392).

Differences in Comparison to Individuals Without Down Syndrome

In the previous section, we reviewed the results from numerous studies that have shown no striking evidence for any motor abnormality in the control of movement. We have also found no evidence of a causal relation between hypotonia and motor impairment in individuals with DS. In this section, we highlight those differences that have been observed.

Slower Reaction Time

One frequently observed performance difference between individuals with DS and people with other forms of mental retardation is the slowed reaction time in the individuals with DS. Several studies have reported that the simple reaction time and choice reaction time for individuals with DS is longer than that of mentally retarded individuals (Berkson, 1960; Blais & Kerr, 1986; Henderson, Illingworth, & Allen, 1991; Kerr & Blais, 1987). As pointed out by Anson (1992) in a review of DS and reaction time, differences in simple reaction time have varied from 25% to greater than 300%. This slowness in reaction time can have two consequences. First, in all movements made in response to an external stimulus, the initiation of the movement is likely to be delayed and can therefore give the impression of movement slowness even if the actual movement itself is reasonably quick. For example, in the study by Blais and Kerr (1986), individuals with DS took 600 ms to react to a stimulus, whereas control subjects took about 300 ms. Second, when individuals with DS are asked to perform sequences of movements, if each movement in the sequence is treated as a separate movement the sequence will be performed extremely slowly because of the increased reaction time to program each component movement.

Higher Incidence of Muscle Coactivation

One finding that we have observed in our studies of individuals with DS that is different from what is seen in neurologically normal individuals is the extent to which muscles are coactivated. Instead of the biphasic pattern of muscle activity, subjects in several studies have shown a simultaneous pattern of activation (coactivation) of the agonist and antagonist muscles during constrained and unconstrained movements when the subject is seated (Almeida et al., 1994; Aruin, Almeida, & Latash, 1996; Latash & Corcos, 1991) and during unconstrained single-joint movements performed while standing (Aruin & Almeida, 1997). Further studies are required to determine whether muscle coactivation is task dependent or whether it is a preferred default strategy (Latash & Anson, 1996) used to perform unpracticed movements that have an element of uncertainty.

Slowness Becomes Accentuated at Longer Distances

Although we have shown that individuals with DS can make movements over different angular excursions using essentially normal patterns of muscle activation, there are clearly differences in performance that might be linked to differences in how muscles are activated. In three separate studies, we observed that the absolute difference in movement speed of individuals with DS and neurologically normal individuals is greater as angular excursion increases (Almeida, Corcos, & Gottlieb, manuscript in preparation; Almeida et al., 1994; Latash & Corcos, 1991). An example of this observation is shown in figure 7.6, in which we have plotted movement velocity, biceps long head, antagonist latency, and muscle torque for movements over three distances.

One implication of this observation is that the ability of individuals with DS to generate muscle force does not increase to the same extent for longer distances as it does in neurologically normal individuals. This relative decrease in muscle force could be a consequence of differences in modulating the intensity and/or duration of the agonist and antagonist EMG burst and/or the antagonist latency. As can be

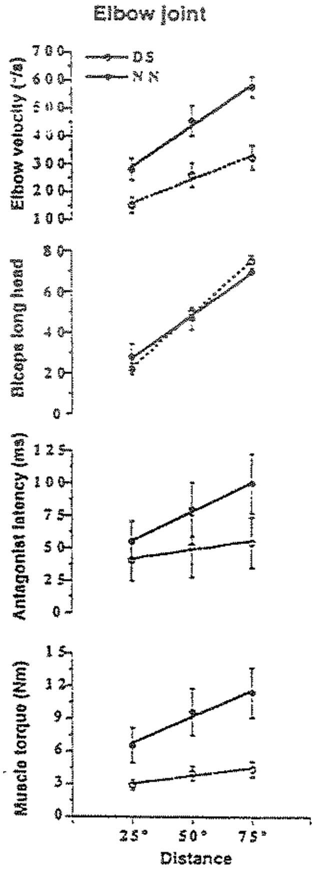

Figure 7.6 Changes in movement velocity, the total amount of agonist muscle activity, antagonist latency, and muscle torque for three different movement distances. The data were averaged across eight individuals with Down syndrome (broken line) and eight neurologically normal individuals (solid line) during the performance of unconstrained elbow movements over three different distances. Muscle torque is given in Newton meters, antagonist latency in milliseconds, and velocity in degrees per second. The amount of biceps long head muscle activity was normalized to the background EMG activity.

Data for the individuals with Down syndrome taken from G.L. Almeida, D.M. Corcos, and G.L. Gottlieb (manuscript in preparation). *Unconstrained single-joint movements of different distances in individuals with Down syndrome.* Data for the neurologically normal individuals taken from G.L. Almeida, D.A. Hong, D.M. Corcos, and G.L. Gottlieb. 1995. Organizing principles for voluntary movement: Extending single-joint rules. *Journal of Neurophysiology* 74:1374-1381.

observed in figure 7.6, the problem seems to be related to the modulation of the antagonist latency. Observe the similar slopes for elbow velocity and antagonist latency. In other words, even though both groups of individuals were able to modulate the duration of the agonist EMG burst, the individuals with DS turned on the antagonist earlier for longer distances compared with control subjects (figure 7.6). The result of this difference in EMG activity is a decreased gain in the net muscle torque with increasing movement angular excursion. Note the similarity between the slope of muscle torque and movement speed. These differences in the way individuals with DS activate their muscles might explain the relative decrease in the gain of movement speed for longer joint excursions, but not movement clumsiness itself.

Movement Sequences

Perhaps the simplest possible repetitive movement sequence, and easiest to perform, is finger-tapping. Frith and Frith (1974) showed that individuals with DS tap slowly, inconsistently, and sometimes just stop tapping. They also showed that a group of individuals with DS tapped more slowly than a group of individuals with autism. Seyfort and Spreen (1979) demonstrated that, when asked to tap between two different targets, not only were individuals with DS slow but they sometimes tapped the same target twice in succession. The individuals with DS in the Seyfort and Spreen (1979) study were tapping only about two times a second. Elliott (1985) replicated the study of Frith and Frith (1974), confirming that individuals with DS tap slowly. However, he also showed that there was no difference in tapping speed between individuals with DS and participants in a mentally handicapped control group, and that individuals with DS do not tap more quickly with their right hand than their left hand as do nonhandicapped individuals. In a follow-up study, Elliott, Weeks, and Jones (1986) showed that individuals with DS were slower to lift up their fingers than to press down. These four studies suggest that it would be most informative to investigate how individuals with DS learn and perform sequences of movement. All of the studies reviewed in the first section in this chapter, in which no deficits were postulated, used pointing movements to a target or reversal movements (movements with one change of movement direction). None of the movement tasks required continuous movements. The heightened variability in the tapping movements, coupled with the fact that releasing a key press takes longer in individuals with DS than in neurologically normal individuals, suggests that individuals with DS have greater difficulty performing sequences of movements than individual movements. They may find switching from one movement to another difficult, just as they find changing movement direction difficult when tracking a moving target (Henderson et al., 1981).

Movement sequences do not have to be long to show that individuals with DS find them very difficult to perform. Chiarenza (1993) had individuals with DS perform a task in which pressing a button with the left hand started the sweep of an oscilloscope and pressing a button with the right hand stopped the sweep. The button to stop the oscilloscope trace had to be pressed in the time interval of 40-60 ms following the start of the sweep. This task requires that individuals centrally program two separate movements with a time delay of no more than 40-60 ms. Initially, individuals with DS pressed the second button much too late. Although practice enabled them to learn to press the second button earlier, the majority of their performances had a time interval of about 200 ms. Analysis of the EMG activation of the hand that started the stimulus sweeping and the hand that stopped the stimulus sweeping showed only minor differences in comparison to the chronologically and maturationally matched control subjects. The rate of rise of the EMG of the stopping hand was reduced. What is most striking is that, "From a neurophysiological point of view, these behavioral patterns express themselves in reduced preparation of the movement, absent or very low Bereitschaftspotential, a lack of elaboration of the reafferent somatosensory information, absence of motor cortex potential, impairment of the processes involved during the evaluation of the outcome of the performance, and the presence of low skilled positivity" (Chiarenza, 1993, p. 464; see also chapter 14).

One possibility is that these difficulties in sequencing are simply due to an inability to sustain concentration on a task. These difficulties are also entirely consistent with the notion of an impaired central timekeeper (Ivry & Keele, 1989). In order to tap consistently with one limb or to generate one tap followed by another tap with a different limb, it has been argued that a clocklike mechanism is responsible for sending out periodic commands to muscles. The tapping data imply that in individuals with DS this central timer is slower, more variable, and cannot send commands to different sets of muscles with only a short time delay. One prediction is that if the tapping performance of individuals with DS was decomposed into a "motor component" and a "clock" component (Wing, Keele, & Margolin, 1984), it would be the clock component that would be impaired. This idea of a timekeeping problem is consistent with a study by Henderson and colleagues (1981). They had subjects perform four tasks. The first task involved subjects tracking a moving sinusoidal target. The second task was to continue tracking when the target was no longer present. The third task was to draw the sinusoidal track from memory. The fourth task was to track a sinusoidal target that increased in speed and decreased in amplitude. Individuals with DS could draw the sinusoidal track from memory, suggesting that they do not have a spatial or perceptual problem. The individuals with DS showed more undershooting at corners and had difficulty keeping up with the moving pattern in both sinusoidal tracking tasks. They could not continue to draw the sinusoid when it was no longer visually presented. This inability to keep drawing a sinusoid when the stimulus was no longer present suggests that individuals with DS have difficulty timing an internally generated sequence of movements. Given that Wang (1996) has argued in favor of teaching sign language to individuals with DS, further research is needed to determine how well individuals with DS can perform different internally generated sequences of movements.

Somatosensory Information

Almeida, Corcos, and Hasan (manuscript accepted pending revision) provide evidence with reversal movements that argues against a proprioceptive deficit influencing the control of movement. However, two studies suggest that tactile perception might be impaired in persons with DS. Cole, Abbs, and Turner (1988) showed that when individuals are asked to grip an object whose gripping surface and vertical load force were systematically manipulated, they did not change the rate at which grip force was generated, as was the case with normal control subjects. They also generated much more force than necessary to perform the task. One possibility is that there is an abnormality in the detection and processing of cutaneous afferent information. Brandt (1996) has shown that individuals with DS do not perform well in tactual perception tests. For example, the data in figure 7.7 show performance on a stereognosis test. Children were asked to feel a wooden object in a cloth bag. A formboard with holes of various shapes was then placed in front of the child, and the child was asked to indicate into which hole the object in the bag fit. As shown in figure 7.7, the children with DS did very poorly on this test. Clearly, further neurophysiological studies are required to discern which mechanisms are responsible for the impaired tactual perception and how this relates to the control of movement.

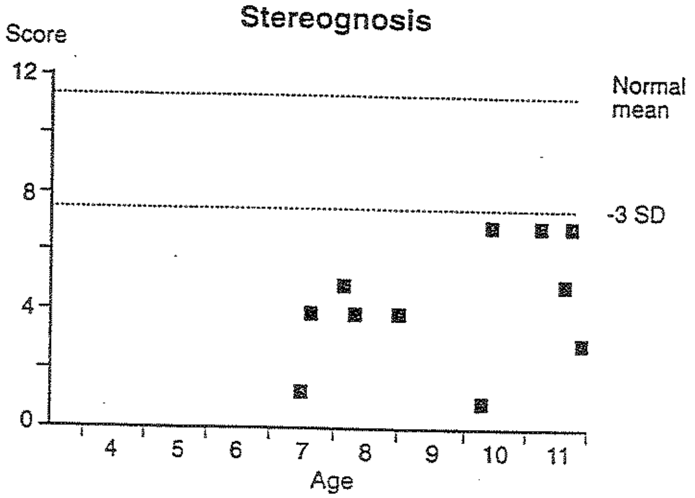

Figure 7.7 Results of the stereognosis test for 11 children with DS. Age and score are indicated for each individual (filled squares). Mean (upper broken line) and -3 SD (lower broken line) for normal children aged 7 years are shown for comparison.

Reprinted, by permission, from B.R. Brandt, 1996, "Impaired tactual perception in children with Down's syndrome," *Scandinavian Journal of Psychology* 32(4): 312-316.

Can Movement Clumsiness Be Attributed to Impaired Decision-Making in Down Syndrome?

Are the motor problems observed in individuals with DS specific to abnormal sensorimotor mechanisms or are they consequences of cognitive limitations that are associated with mental retardation? At one end of the spectrum is the argument that individuals with DS have severe motor problems. At the other end lies the view that their sensorimotor systems are intact, but they have decision-making problems that lead them to use conservative movement strategies adequate for most movement tasks but perhaps not optimal for any specific task. This raises the question of what is meant by "poor decision-making." If poor decision-making refers to task comprehension, this explanation does not always hold true. The reason for this can most easily be seen in the study by Almeida et al. (1994) in which subjects were asked to make movements over different speeds and also different distances (see sections on speed and distance earlier in this chapter, pages 153-155). Both types of movements were made with relatively normal patterns of muscle activation, suggesting that individuals with DS can decide between two tasks and can use relatively normal patterns of muscle activation to perform the tasks. It should be pointed out that these tasks were presented in a block of trials so the subjects knew exactly which movement they had to make. It would be interesting to replicate these studies in a choice reaction-time paradigm in which greater decision-making is required.

However, if impaired decision-making refers to the inability to rapidly distinguish between different movement possibilities, there is evidence to support the idea that appropriately timed patterns of muscle activation are not always used. Instead, muscle groups are often coactivated. In addition, there is considerable anecdotal evidence that individuals with DS may not interpret experimental instructions in the manner expected by the experimenter. For example, the instruction to move faster can result in subjects pressing down harder. Henderson and colleagues (1981) pointed out that when subjects were encouraged to go faster, subjects merely pressed harder on the paper they were tracking on. This difference in how instructions are interpreted is clearly a decision-making problem that can dramatically influence how some movement tasks are performed. However, just as Anson (1992) noted that hypotonia should not be considered the default explanation for impaired motor performance, neither should impaired decision-making be considered the default explanation for impaired motor performance. Careful experiments are required that manipulate both the complexity of the movement and the complexity of the decision-making to determine how impaired decision-making impacts the control of movement.

Conclusions

One therapeutic strategy that has been suggested for improving motor function in individuals with DS is to increase muscle tone. The logic behind this approach is the assumption that "normal" tone is a necessary condition for normal movement control. The cause of hypotonia has been linked to the reduced weight of the cerebellum of individuals with DS (Cowie, 1970; Crome, Cowie, & Slater, 1966). Also, Gilman, Bloedel, and Lechtenbers (1981) have shown in experiments on primates that a lesion in the cerebellum can produce cerebellar hypotonia. Therapeutic interventions for altering muscle tone include isometric exercise, training of muscle force, and tactile stimulation (Linkous & Stutts, 1990). The idea is that these interventions improve muscle activation and therefore muscle tone. As we have argued earlier in this chapter, individuals with DS can adjust patterns of muscle activation, and, in this sense, their motor control systems are functionally intact. At least adolescents and young adults with DS do not seem to have muscle hypotonia. This may explain the lack of notable success for drugs that are designed to increase muscle tone in DS (Coleman, 1978). It may also explain only the small gain in the acquisition of functional abilities with training designed to improve muscle tone (Shumway-Cook & Woollacott, 1985). In sum, we see no compelling reason to recommend any training based on increasing muscle tone to improve the control of voluntary movement (Blanche, Botticelli, & Hallway, 1995).

Despite the clear observation of movement slowness and increased variability, we have shown that the differences in motor performance between individuals with DS and neurologically normal individuals are very subtle for a variety of pointing movements and movements with a single reversal. We do not know at this point if there is a general impairment in proprioception that could account for the subtle differences in performance reported. Whether training to enhance proprioceptive acuity will lead to a decrease in movement clumsiness is an open question. We are not optimistic about this kind of treatment. Indeed, it would be difficult to explain the accuracy in movement experiments demonstrated by individuals with DS without assuming an intact proprioceptive system (Almeida, 1993).

The idea of a universal and invariant sequence of development, as advanced by Gesell (1925), had and still has a great impact on therapeutic intervention. The mapping of "normal" movement sequences and stages was argued to be the best framework for therapeutic approaches. Learning and practicing movements that obey this universal and invariant sequence would cause the client's movements to become as close as possible to "normal." Latash and Anson (1996) have criticized approaches that routinely try to normalize the control of movement. Because the mechanisms and priorities of normal motor control are generally unknown, they have argued for a "hands-off" approach and that "adaptive changes in motor patterns should be considered normal and, as such, should not be corrected" (p. 67). Given that we do not know the priorities of the CNS, it is only supposition that the CNS changes its priorities in certain situations. As such, the idea that the central nervous system of individuals with DS may "prefer" to use safer motor strategies that prevent failure might be as detrimental as the idea of imposing a "normal" pattern.

At this point we would like to offer our recommendations regarding therapeutic intervention. When we trained individuals with DS to move quickly, neither did we tell them which pattern of muscle activation to adopt, nor did we impose any kinematic pattern of normality (Almeida et al., 1994). Latash and Anson (1996) have argued that the considerable improvement we observed in individuals with DS could be attributed to the friendly and reinforcing environment offered during training. While we agree that a friendly environment is conducive to performance, individuals with DS may need considerably more practice to improve their ability to control everyday movements. They need more practice for two reasons. The first is that individuals with DS have less life experience, so that even if the experimental protocol gives equal formal practice, this is not enough to equalize the real experience of the two groups. The second is that they learn at a reduced rate and therefore need more practice. In our opinion, the biggest problem with the design of the studies investigating the motor performance of individuals with mental retardation is that they do not include practice sessions or refer to two 3-minute practice sessions as "learning" (Frith & Frith, 1974). Society does not treat its handicapped and nonhandicapped populations equally, and we should not assume that research participants have had the same opportunity for practice prior to arriving at the laboratory. Training can help to decrease the differences that might erroneously be attributed to structural or functional changes within the CNS, making comparisons more realistic. This effect of lack of prior experience, coupled with the benefits of practice shown in numerous studies that we have reviewed, suggests caution in implementing a hands-off approach.

One major contribution of the systematic study of motor control is to offer insight into when and how to intervene. We suggest a hands-on approach without imposing any "normal" pattern, just as we did with the single-joint flexion movements. One can then observe if normality comes naturally. The next step would be to determine if the subtle qualitative differences in the way individuals with DS control their muscles disappear with simple repetition of the motor task. The elbow-flexion training study suggested that all anatomical and neurophysiological differences that have been well documented in this population of individuals may not be detrimental to movement performance if appropriate training is provided (Almeida et al., 1994; Perán et al., 1997). However, care should be taken in drawing such a conclusion. We may discover that during more complex tasks, individuals with DS may be unable to couple the muscle forces across different joints even after extensive training. It may turn out that the coactivation strategy for postural muscles (Aruin & Almeida, 1997) does not change with practice. In such instances, it is preferable to structure therapeutic sessions in ways that allow the individual to master the task in his or her own preferred way as opposed to trying to teach one preferred way.

Summary

In summary, we suggest that a balanced approach is required when considering different therapeutic interventions for facilitating sensorimotor performance in individuals with DS. Although there are clear verbal, linguistic, and auditory-processing deficits (Wang, 1996; Wisniewski et al., 1988), individuals with DS do not seem to have any striking deficits in activating muscle. There is also such incredible diversity between different individuals with DS that physicians, therapists, and parental associations need to be very careful when making prognostications and giving advice. A message that is too negative can lead to reduced expectations, with the result that individuals with DS do not achieve their full potential. Similarly, an overly optimistic message can cause severe parental emotional stress if the individual is profoundly retarded and not capable of reaching expected functional levels (Gath, 1994).

However, the training and practice studies that we have reviewed show that large improvements in motor performance are possible. This fact, coupled with the fact that no clear muscle activation deficits have been identified, strongly suggests that active intervention may be very important and very beneficial. We have identified no study that has shown a decrease in performance after extensive practice. We concur that practice should not be directed at normalizing movement patterns (Latash & Anson, 1996), and clearly any vigorous hands-on approach should take into account potential problems (e.g., orthopedic, cardiac) of individuals with DS (Block, 1991). However, practice and training should be directed at improving performance, consistency, and generalizability of movement skills. In very rare cases, sustained practice at physical activity can lead to such high skill levels that an individual with DS has competed in mainstream open competition in gymnastics (Jones, 1994).

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Effects of Displacement and Trajectory Length on the Variability Pattern of Reaching Movements

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ABSTRACT. The design of the present study enabled the authors to distinguish between the possible effects of movement displacement and trajectory length on the pattern of final positions of planar reaching movements. With their eyes closed, 9 subjects performed series of fast and accurate movements from different initial positions to the same target. For some series, the movements were unconstrained and were therefore performed along an approximately straight vertical line. For other series, an obstacle was positioned so that trajectory length was increased because of an increase in movement curvature. Ellipses of variability obtained by means of principal component analysis applied to the scatter of movement final positions enabled the authors to assess the pattern of movement variable errors. The results showed that the orientation of the ellipses was not affected by movement displacement or by trajectory length, whereas variable errors increased with movement displacement. An increase in trajectory length as a consequence of increased curvature caused no change in variable error. From the perspective of current motor control theory, that finding was quite unexpected. Further studies are required so that one can distinguish among the possible effects of various kinematics, kinetics, and other variables that could affect the pattern of variable errors of reaching movements.

Key words: displacement, error, human, reaching movements, trajectory length

For one-dimensional tasks (e.g., single-joint movements or movements of the working point performed along an approximately straight line), the pattern of the final position variability is usually described by two parameters: the constant error (the distance between the averaged recorded final position and the target position), and the variable error (standard deviation of the final positions). For multidimensional tasks, however, researchers have often calculated ellipses or ellipsoids of variability (for a two- or three-dimensional [2D, 3D] movement task, respectively) by using principal component analysis applied to the scatter of movement final positions (Gordon, Ghilardi, & Ghez, 1994; McIntyre, Stratta, & Lacquaniti, 1997; Messier & Kalaska, 1997). In that research, the orientation of the ellipses or ellipsoids has been assumed to correspond to the direction of the main principal component. The area or volume of the ellipses and ellipsoids has also been calculated from the eigenvalues so that variable errors of the tested movements could be assessed (Gordon et al., 1994; Messier & Kalaska, 1997).

Although orientation of the ellipses of variability has played an important role in some recent models and hypotheses concerning the frame of reference for movement planning and execution (cf. Gordon et al., 1994; McIntyre et al., 1997; Soechting & Flanders, 1989), its possible relations with movement distance have not as yet been studied. In several studies, however, an increase in variable error with movement distance, as assessed by the areas and volumes of the ellipses and ellipsoids, respectively, has been demonstrated (Gordon et al., 1994; McIntyre et al., 1997; Messier & Kalaska, 1997). Those results provide support for behavioral laws suggesting that the maintenance of a required accuracy becomes increasingly difficult when movement distance increases (see Schmidt, 1988, for a review).

In a recent study of 2D reaching movements, Jaric, Tor-

Correspondence address: Slobodan Jaric, Department for Musculoskeletal Research, National Institute for Working Life, Box 7654, S-907 13 Umeå, Sweden. E-mail address: juric@niwl.se toza, Fatarelli, and Almeida (in press) failed to demonstrate an increase in variable errors, as assessed by the area of the ellipses of variability, despite an approximately two-fold increase in the trajectory length resulting from increased curvature. A literature review revealed that the relationship between the movement distance and variability of the attained final position had been almost exclusively found in studies in which the trajectory length (equal to the total distance moved along movement path) was assumed to be equal to the movement displacement (the distance between the initial and final position). That approach has been inevitable in studies of single-joint movements because only one trajectory is available for reaching the final position from the initial one. Multidimensional movements, however, are inevitably curved (see Boessenkool, Nijhof, & Erkelens, 1998, for a review); in those movements, therefore, the trajectory is longer than the displacement. Nevertheless, from the classical work of Fitts (1954) until the most recent studies of the effects of movement distance on the scatter of movement final positions (McIntyre et al., 1997; Messier & Kalaska, 1997), the possible distinction between the effects of movement displacement and trajectory length has not been studied.

Because Jaric and his colleages had not designed their previous study (Jaric et al., in press) to distinguish between the discussed effects, we performed another experiment in which we independently manipulated movement displacement and trajectory length. We expected that the results would show whether movement displacement or trajectory length represent a major factor affecting the pattern of the final position variability of reaching movements.

Method

Subjects

Nine neurologically healthy volunteers (5 men and 4 women, age range = 19–46 years) participated in the study. All were right-handed and used their right hand. They signed an institutionally approved informed consent form.

Procedure

During testing, each subject sat comfortably in a chair. A vertical metal surface was positioned lateral to the right shoulder and oriented parallel to the sagittal plane of the subject. The subject was able to see markers of the initial position, the target, and the obstacle. All markers were made of plastic and fixed on the surface with magnets; therefore, they were easy to rearrange. The target was made of a light, flexible piece of plastic representing a rectangle with a semicircular hole (diameter 6 = cm, distance from the frame = 12 cm) oriented at a 45° angle with respect to the horizontal direction (see Figure 1A). Even the slightest touch by the subject's index finger caused the target to slide along the metal surface. The markers of the initial position and target as well as the subject's right shoulder were positioned in a vertical plane that was parallel with both the subject's sagittal plane and the metal surface. We refer herein to that plane as the *movement plane* because it was the one

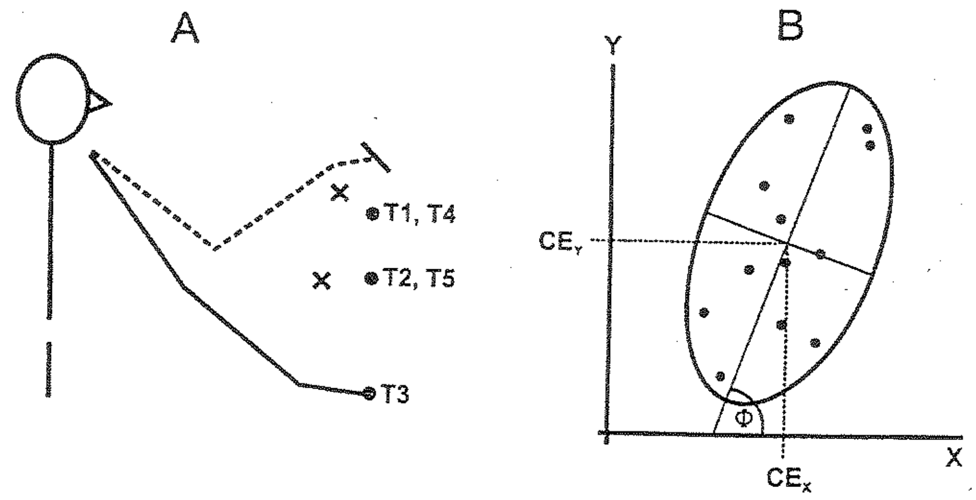

FIGURE 1. A. An illustration of the experimental conditions depicted in the movement plane. Arm postures in one of the initial positions (unbroken line) and final position (dashed line) are shown, together with markers for three initial positions for five movement tasks (T1–T5 [filled circles]), target (sloped thick line), and obstacles (crosses). B. An example of the recorded scatter of the final positions and the corresponding ellipse of variability. *CEx* and *CEy* correspond to the constant errors along the *x*- and *y*-axes, respectively, whereas ϕ corresponds to the orientation of the main principal component.

in which fingertip movements as well as the movements of the wrist and elbow mostly occurred.

The single target position applied in all movement tasks was adjusted for each subject so that the following two requirements were fulfilled: (a) The target height was at the level of the subject's eyes, and (b) the subject's elbow was at approximately 120° of flexion when he or she placed the extended fingertip inside the target by using a comfortable, slow movement. The starting and the obstacle positions were adjusted thereafter to the target position.

The subjects were asked to perform five different movement tasks. Three tasks were approximately vertical, straight-line movements from three different initial positions in to the same target (see Figures 1 and 2A for illustrations). Therefore, the length of the trajectories approximately corresponded to the displacement of the initial position from the target: about 15 cm in Task T1, 30 cm in Task T2, and 60 cm in Task T3. An additional two tasks were performed from the same initial positions as T1 and T2, but with a prominent curvature as a result of the obstacle avoidance. Specifically, they were performed over displacements of 15 cm (Task T4) and 30 cm (Task T5), whereas the trajectory lengths were approximately 30 and 60 cm, respectively (see Table 1 for details and Figure 2A for an illustration). We had performed a pilot experiment before the reported one in order to assess positions of the obstacle that we expected would provide the required trajectory lengths in the two curved tasks.

The subjects were instructed to keep the tip of the extended index finger in the initial position, with muscles relaxed as much as possible. After adjusting the fingertip and observing the target, subjects closed their eyes. Thereafter, a computer-generated tone sounded, and the subjects were instructed to perform fast and accurate movement into the target. They were also asked not to hesitate to hit the target nor to correct the final movement position. Two seconds after the movement termination, the subjects were allowed to open their eyes and observe the actual final position. Then the experimenter moved the marker into the starting position for the next movement, corrected the target position (if it was shifted during the preceding movement), and placed or removed the obstacle, if necessary. Finally, the subjects were asked to return the fingertip to the next initial position and to get prepared for the next movement. The time interval between two consecutive movements was about 15 s.

On the 1st day, subjects participated in a practice session identical to the experimental one, although the data were not collected. The experimental session was performed on the following day. A single block of 65 movements was performed consisting of 13 trials for each of the five tasks. The sequence of trials was partially randomized so that blocks of 5 consecutive trials (e.g., Movements 1–5, or 6–10) consisted of all five different tasks. The first 5 trials were rejected, and, thereafter, 12 trials of each task remained for further analysis.

A light-emitting diode marker was placed on the tip of the index finger. Three Optotrak cameras (Northern Digital, Waterloo, Ontario) tracked movement of the marker in the Cartesian coordinates at a rate of 200 frames per second, where the x-axis corresponded to the horizontal and the y-axis to the vertical axis of the movement plane (see Figure 1B), whereas the z-axis was orthogonal to that plane. We smoothed (35-ms moving average window) and differentiated the x- and y-coordinates in order to calculate the tangential velocity of the fingertip. Movement time was calculated as a time interval from the moment when velocity of the fingertip first exceeded 10% of its peak value to the first moment when it fell again below that value in a vicinity of the target. The length of the trajectory was measured along the fingertip path from the initial position to the point reached at the end of the movement time. The final movement position was measured 0.3 s after the end of the movement time.

Constant errors, as well as the pattern of variable errors of each task, were calculated from the scatter of the movement final positions. Two components of the constant error corresponded to the horizontal and vertical displacements of the averaged movement final position from the target position (see Figure 1B). Variable errors were calculated as the mean distance between individual final positions and the averaged one. We assessed the pattern of variable error by using ellipses of variability calculated by means of principal component analyses (Figure 1B). The eccentricity of the ellipses was calculated as the ratio between the main principal component and the orthogonal component. The orientation of the ellipses corresponded to the orientation of the main principal component. Because the direction of the component was not defined (i.e., the component's orientation has two possible directions that a priori differ by 180°), the orientation averaged across the subject in each particular task was calculated in various 180° intervals of the full circle. The result that provided the lowest variability was taken for further analysis.

Results

We assessed movement deviations from the movement plane by calculating the peak-to-peak motion interval of the fingertip along the z-axis. For all subjects and in all tasks, the interval was smaller than movement distance by an order of magnitude. Therefore, we refer in the results only to those movement kinematic parameters measured in the movement plane.

Trajectories of the movements performed by 1 subject are shown in Figure 2A. As depicted, Tasks T1, T2, and T3 were approximately straight-line tasks, whereas Tasks T4 and T5 were performed along highly curved trajectories. Straight-line tasks were also characterized by bell-shaped velocity curves typical for rapid discrete human movements, whereas the velocity pattern of the curved tasks usually showed several peaks (Figure 2B). As depicted, movement time also varied considerably across the tasks. Similar results in terms

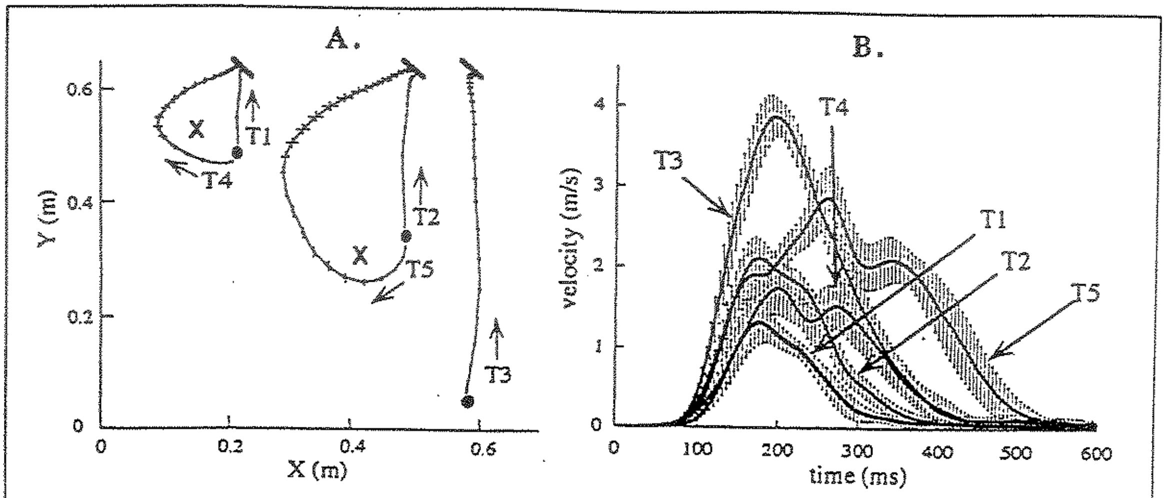

FIGURE 2. Movement kinematics of all five tasks recorded in 1 subject. Each set of data represents the average of 12 trials aligned with respect to the beginning of the movement time, while the error bars depict standard errors. A. Movement trajectories depicted with starting positions (filled circles), positions of the obstacle (crosses), and target position (thick sloped lines). Note that although all the movements were performed in to the same target, we plotted groups of tasks separately in order to avoid overlapping of the trajectories, particularly in tasks T1, T2, and T3. As a consequence, the same target had to be shown in three different positions. B. Velocity profiles of the same movements as are depicted in Figure 1A. In addition to the differences in the velocity profiles among the tasks, note the differences in movement time.

of the shape of trajectory, velocity profile, and movement time of a particular task were observed in all subjects.

In Table 1 basic variables (excluding variable errors) obtained in five tasks and averaged across the subjects are shown. The differences in trajectory lengths between T2 and T4, as well as between T3 and T5, were insignificant. Movement times of both curved tasks (T4 and T5) were longer than movement times of each of the straight-line tasks (T1, T2, and T3, averaged across both the subjects and trials; $p < .01$, one way analysis of variance). For all tasks, the constant errors along each axis were usually small, within the 2-cm margin; they were also scattered randomly around the target position. No systematic constant errors across subjects were observed along either axis of the movement plane.

Only 7 out of 45 ellipses of variability (i.e., 9 subjects × five tasks) demonstrated significant eccentricity, meaning that the variable error was higher along the main principal component than along the orthogonal component ($p < .05$; Wilcoxon signed-rank test). Those 7 ellipses were obtained mainly in the tasks with short movement displacements (3 in T1, 3 in T4, and 1 in T5). It should be noted that this result could partly be a consequence of the relatively small number of trials performed within each movement task (n = 12). Eccentricity of the ellipses of variability changed in a narrow margin, whereas their orientation remained approximately vertical despite changes in movement displacement and trajectory length. Changes in both the eccentricity and orientation of the ellipses of variability were tested according to the predicted displacement (i.e., between T2 and T4, as well as between T3 and T5) and trajectory (i.e., between T1 and T4, as well as between T2 and T5) effects. No significant changes were observed.

Variable errors obtained in all five tasks and presented according to the predicted displacement and trajectory effects are shown in Table 2. In line with the displacement effect, there was a difference between T4 and T2 (p = .01; paired t test) and between T5 and T3 (p <.05; paired t test). No significant differences were observed in line with the trajectory effect (i.e., T1 vs. T4 and T2 vs. T5). It should be mentioned, however, that the difference between T4 and T1 was close to the level of significance (p < .1; paired t test) but in a direction opposite to the predicted one. Note that the diagonal changes (i.e., the differences in variable errors among T1, T2, and T3, as well as between T4 and T5) were not tested because those were predicted by both effects.

Discussion

The increase in movement displacement we tested was equivalent to the increase in movement distance tested in a number of previous studies performed on multijoint movements (cf. Gordon et al., 1994; Messier & Kalaska, 1997). Therefore, the increase in variable errors with movement displacement we observed does provide support for the findings of the aforementioned studies. As a consequence, the main problem remaining for further discussion is a paradoxical finding that a two-fold increase in trajectory length caused by increased movement curvature did not

TABLE 1
Summary of Basic Variables Measured in Five Movement Tasks

Task	L (cm)	MT (ms)	CE_x (cm)	CE_y (cm)	ECC	ϕ (deg)
T1	16.9 ± 1.9	265 ± 52	0.3 ± 0.7	–0.2 ± 1.0	2.09 ± 0.79	117 ± 18
T2	30.9 ± 2.1	264 ± 61	–0.8 ± 1.2	0.2 ± 0.6	1.59 ± 0.50	87 ± 41
T3	60.9 ± 2.6	289 ± 74	1.0 ± 1.7	0.7 ± 0.9	2.19 ± 0.48	113 ± 13
T4	33.0 ± 3.5	379 ± 98	0.0 ± 0.7	–0.2 ± 1.0	2.35 ± 0.74	102 ± 41
T5	59.6 ± 3.7	428 ± 97	–0.9 ± 1.6	0.1 ± 1.0	1.89 ± 0.66	90 ± 32

Note. The data represent trajectory length (L) and movement time (MT), averaged across both subjects and trials, as well as the constant errors measured along the horizontal and vertical directions (CE_x and CE_y); eccentricity (ECC); and orientation of the ellipses of variability (ϕ, measured with respect to the anterior axis, see Figure 1B), averaged across subjects.

TABLE 2
Variable Errors

		Displacement Effect →		
Trajectory Effect ↓		15	30	60
	15	(Task 1) 1.28 ± 0.25	–	–
	30	(Task 4) → 1.15 ± 0.27	(Task 2) 1.35 ± 0.25*	–
	60	–	(Task 5) → 0.39 ± 0.25	(Task 3) 1.74 ± 0.38**

Note. Variable errors ($M \pm SD$ in centimeters, averaged across subjects) were calculated as the mean distance between individual final positions and the averaged one. They are depicted according to the expected increase according to the displacement and the trajectory effects. The arrows denote the direction of an increase.
$^*p < .05$, $^{**}p < .01$; paired t test.

increase movement variable error. Although that finding is supported by recent study (Jaric et al., in press), we consider it paradoxical because only with difficulty can one explain it by using models and hypotheses of the current motor control theory.

In general, the curvature of the tested movements (i.e., T4 and T5) was far beyond a subtle curvature usually observed in nonconstrained, discrete reaching tasks (see, for an illustration, Boessenkol et al., 1998, and Gordon et al., 1994). Whatever mechanisms are involved in planning and execution of reaching tasks (cf. Bernstein, 1967; Gottlieb, Corcos, & Agarwal, 1989; Latash & Turvey, 1996; see also McIntyre et al., 1997, and Soechting & Flanders, 1989), it seems reasonable to assume that the applied obstacle per se imposed changes in those mechanisms with respect to their spontaneous functioning under the regular, unconstrained conditions. Therefore, one would expect that an artificial factor (such as the applied obstacle) that adds to the complexity of a tested task would be associated with an increase in the variable errors and, consequently, with a reduced accuracy of reaching movements. Nevertheless, we failed to observe that effect in some of the tasks, whereas in others (i.e., T1 vs. T4) the difference was close to the level of significance but in the opposite direction than expected.

The finding that the approximately vertical orientation of the ellipses of variability is unaffected by the applied manipulations of movement displacement and trajectory length (i.e., curvature) is in line with some previous results. Several authors have suggested that reaching movements are planned in a hand-held system of coordinates because scatter of final position variability was stretched along the main movement direction defined by the initial and final position (Georgopoulos, Kalaska, Caminiti, & Massey, 1982; Gordon et al., 1994). The absence of a curvature effect on the orientation of ellipses of variability is also supported by the results of a previous study by Jaric et al. (in press).

In conclusion, we found that (a) an increase in variable errors of reaching movements was associated with an increase in movement displacement rather than an increase in trajectory length as a consequence of increased curvature, whereas (b) the orientation of the ellipses of variability remained unaffected by the applied experimental manipulation. The first finding could be of exceptional importance for further development of motor control theory, particularly in the formulation of behavioral laws of human complex movements. Although the movement displacement effect on variable errors could be considered expected, the absence of a trajectory-length effect deserves further studies so that some potentially confounding effects of the applied experimental approach can be discerned. For example, although we manipulated the obstacle position in order to affect the trajectory length, one should note that a number of other potentially important kinematic and kinetic variables (such as movement time, velocity pattern, or torque profiles) were also affected. In addition, one should consider that manipulations of movement distance because of changes in the initial position and changes in the target position might have different effects on variable errors of reaching movements (Jaric, Corcos, Gottlieb, Ilic, & Latash, 1994). Therefore, it remains possible that a test of the movement displacement and trajectory-length effects based on manipulation of the target position instead of the initial one (as we did) would provide different results.

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IV.II. Apresentações em Congressos Científicos

Poster 03.71

EFFECTS OF DISPLACEMENT AND TRAJECTORY LENGTH ON THE FINAL POSITION VARIABILITY OF REACHING MOVEMENTS

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The present study has been designed to test separately the effects of displacement (i.e., the distance between the initial and final position) and trajectory length on 2-D variable errors of multi-joint movements. Nine subjects with their eyes closed performed series of fast and accurate reaching movements from the initial positions displaced vertically 15, 30, and 60 cm below the target. In some series the movements were unconstrained and, therefore, approximately straight. In others, an obstacle was positioned in order to increase trajectory length due to increased movement curvature. The movement variable errors were assessed by surface of the ellipses of variability calculated by means of principal component analysis applied to the scatter of the movement final positions. The lengths of the ellipses' long and short axes were scaled with respect to the final position variability in direction of the principal component main axes and in the orthogonal direction, respectively. The results demonstrated that variable errors increased with an increase in movement displacement. However, with the exception of the task performed with an excessive curvature (i.e., the trajectory resembled more to a reversal movement), even an insignificant increase in variable errors was not associated with an increase in trajectory length. Therefore, we concluded that movement displacement, rather then trajectory length, could be considered as an important factor affecting variable errors and, consequently, the accuracy of multi-joint movements. It was concluded that the lack of trajectory length effects on the movement variable error could not be in agreement with some contemporary motor control models and theories. We suggest that the single target position used in the present experiment could be an important methodological factor affecting the results obtained. We also conclude that the results obtained could generally emphasize importance of the final position representation in motor programs of multi-joint reaching movements.

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Poster 03.72

MUSCLE TORQUE COVARIANCE IN NEUROLOGICALLY NORMAL AND DOWN SYNDROME INDIVIDUALS: THE EFFECT OF ANGULAR, LINEAR DISPLACEMENT AND SPATIAL ORIENTATION.

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We have identified a principal of linear co-variance in which the muscle torque at the non-focal joint was very close to linearly proportional to that at the focal-joint throughout the movement. Elbow and shoulder torques differed by a linear scaling constant and went through extreme and zero crossings almost simultaneously. These findings were also observed during planar human movements. Compared with neurologically normal subjects, Down syndrome individuals use proportionally more distal than proximal muscle torque while performing reversal movements. This difference in strategy in the coordination of muscle torques may contribute to the movement clumsiness observed in the movements of these individuals. Here we studied the effect of angular, linear displacement, and spatial orientation on the linear co-variance between elbow and shoulder torques of DS and NN individuals. These individuals performed a planar reversal movements into four spatial orientation. In each spatial orientation they performed tasks into three different angular excursion (18, 36 and 54°), and into three different linear displacement (30, 45 and 60% of the forearm length). Subjects did not have any visual feedback of the elbow joint for the angular excursion tasks, but they have visual feedback of the fingertip for the linear displacement task. They were instructed to move "as fast as possible", from the initial to the target position, reversal the movement and return to the initial position. A set of light were turned off, in front of the subjects, signalling the angular displacement required during the angular excursion task. For the linear displacement task the target was fixed at the position with a mark. The results showed that there is no group difference in the linear co-variance between angular excursion and linear displacement tasks. However, the preliminary results indicate that there was a change in the linear co-variance with change in spatial orientation. These findings were the same across the group of subjects. However, the individuals with Down syndrome used more elbow muscle torque than shoulder muscle torque. Also, the coupling between these two torques was less efficient for the Down syndrome group as compared with the NN individuals. Our findings support the idea that there is a subtle differences in the way DS individuals control their movement that could be related with their movement clumsiness.

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XIV CONGRESSO BRASILEIRO DE FISIOTERAPIA

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

CERTIFICADO

Conferido a

NADIA FERNANDA MARCONI

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: CONTROLE MOTOR DOS MOVIMENTOS DE REVERSÃO NA PRESENÇA DE FEEDBACK ANGULAR EM INDIVIDUOS CONTROLE E PORTADORES DA SÍNDROME DE DOWN.

Nadia Fernanda Marconi (Universidade Estadual de Campinas), Sandra Maria Sbeghen Ferreira (Universidade Estadual Paulista - Rio Claro), Charli Tortoza (Universidade Estadual Paulista - Rio Claro) e Gil Lucio Almeida FT, MS, PhD. (Universidade Estadual de Campinas e Universidade de Ribeirão Preto/SP)

Claudia Maria Bahia Pinheiro
Presidente

Dayse Dantas Oliveira
Vice-Presidente

A VARIAÇÃO DA LATÊNCIA ANTAGONISTA COM A DISTÂNCIA LINEAR EM MOVIMENTOS PLANARES DE INDIVÍDUOS NORMAIS E DE PORTADORES DA SÍNDROME DE DOWN[3]

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Existem evidências de que o tempo entre o início da ativação da musculatura agonista e o da antagonista (latência antagonista – LA) aumente com a amplitude angular. Este fenômeno foi observado para movimentos uniarticulares restritos e não restritos, em indivíduos neurologicamente normais (NN) e em portadores da Síndrome de Down (SD), embora a LA fosse menor para os indivíduos SD. Nos movimentos uniarticulares, os fatores intrínsecos (amplitude angular) e extrínsecos (distância linear) co-variam linearmente, o que não acontece, necessariamente, para os movimentos multiarticulares. Aqui verificamos se a LA aumenta com a mudança de fatores extrínsecos (distância linear do dedo indicador) durante a execução de movimentos planares de reversão, envolvendo a extensão do cotovelo e abdução ou adução do ombro. Seis sujeitos NN e 6 com SD executaram movimentos, o mais rápido possível, em 3 distâncias lineares (30%, 45% e 60% do comprimento do antebraço) e em 3 orientações espaciais (à direita, ao centro e à esquerda do sujeito). Os movimentos foram reconstruídos no espaço utilizando um sistema óptico-tridimensional. Simultaneamente foram registradas as atividades eletromiográficas de 4 músculos (deltóide posterior, deltóide anterior, tríceps e bíceps). Os sujeitos de ambos os grupos aumentaram a LA da musculatura antagonista do cotovelo com o aumento da distância linear, independente da orientação espacial do movimento. No entanto, esta LA foi menor para os indivíduos SD. Primeiro, estes dados indicam que o SN pode utilizar informações extrínsecas (distância linear) para definir a LA do tríceps. Segundo, a diminuição da LA para os indivíduos SD favorece o aumento da estabilidade articular desta população. No entanto, a LA da musculatura do ombro foi próxima de zero para ambos os grupos. Este dado pode indicar uma incapacidade do SN de modular a LA para musculatura proximal.

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