Practice, transfer and performance enhancement of fast single-joint movements in individuals with Down syndrome

Gil Lúcio Almeida

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Practice, transfer and performance enhancement of fast single-joint movements in individuals with Down syndrome

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Iowa State University, 1993

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Practice, transfer and performance enhancement of fast single-joint movements in individuals with Down syndrome

by

Gil Lúcio Almeida

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1993
To my wife, Alba, for her love, help, and understanding.
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ABSTRACT

Eight individuals with Down syndrome practiced performing 1100 elbow movements over one distance (36°), under the instruction to move "as fast as possible." The subjects were also pretested and posttested performing elbow flexion movements over four distances (18°, 36°, 54°, and 72°) "as fast as possible", and at a "comfortable speed" over 36°. For two of these distances (18° and 36°), the movements were also performed from a second starting position. They improved their motor performance between training sessions (110 movements), as measured by the kinematic and EMG parameters. This improvement performance was described by a logarithmic function. With training the subjects increased the intensity with which they activated their motoneuron pools, decreased the antagonist onset latency, and improved the peak velocity by 67%. This remarkable improvement was obtained without an increase in variability, which was already very low at the beginning of the training.

The subjects were also able to transfer their performance improvement to the non-trained distances and to the different starting position. Subjects decreased their movement time by proportionally decreasing both the acceleration and deceleration time. This study supports the idea that subjects with Down syndrome can use patterns of muscle activation that are qualitatively indistinguishable from those employed by individuals without neurological impairment. With appropriate training, individuals with Down syndrome can achieve high levels of motor performance.
GENERAL INTRODUCTION

An explanation of the dissertation contents and organization

This dissertation describes the effects of practice and transfer on the performance of fast single-joint movements in individuals with Down syndrome. We analyze and discuss the ability of these subjects to control their movements. The discussion is based on the "dual strategy hypothesis," which is a set of rules for controlling movements (Gottlieb, Corcos, & Agarwal, 1989b). The dissertation contains two papers preceded by a general introduction where we summarize the "dual strategy hypothesis," and the studies that focus on the relationship between myoelectric and kinematic parameters as a function of practice in the framework of this hypothesis. Then, we selectively review the studies about the general characteristics of Down syndrome individuals, emphasizing those that are somehow related to their motor control system. The first paper discuss the effects of practice of fast single-joint movements during consecutive sessions of training of fast single-joint elbow movements in individuals with Down syndrome. In the second paper both the practice and transfer effects are analyzed, before and after training. This paper we have submitted for publication (Almeida, Corcos, & Latash, 1993 submitted). Following the second paper we present a general discussion and conclusions of the findings reported in the two papers, in the context of the literature review presented in the general introduction. The references cited in the general introduction follow the general discussion and conclusions. Finally, we present the appendixes which contain data and other correspondence not presented in the first paper. In the Appendix A we present the consent form we got from a human investigation committee to perform the experiments. Also, enclosed in the Appendix A is a subject information sheet. In the Appendix B we present the supplementary data, such as, confidence interval of peak velocity between sessions of training, and linear and logarithm relationships between kinematic variables and EMG variables.
The "dual strategy hypothesis"

Movements can be performed in a wide variety of ways and under diverse conditions. The time it takes for the motor control system to plan and execute a movement usually is very short, especially for fast movements. If the motor control system had to compute many parameters (i.e., trajectory, velocity) to plan and to execute a movement, this would take a lot of time. The consequence would be an inability to perform movements very fast. Because of that, certain theories of motor control assume that movements have to be controlled by generalizable rules. That is, theories in motor control try to minimize the number of parameters used by the motor control system to plan and execute movements (Bernstein, 1967; Feldman, 1986; Gottlieb et al., 1989b; Schmidt, 1975).

One theoretical approach to explain how movements are controlled is the "dual strategy hypothesis" (Gottlieb et al., 1989b). This theoretical approach is based on the notion that the parameters controlled during a movement are the descending commands sent to the motoneuron pools to activate the agonist and antagonist muscles. The agonist muscles are the prime movers accelerating the limb in the desired direction whereas the antagonist muscles brake the movement. The descending commands are assumed to have a complex wave form which can be approximated by a rectangular "excitation pulse." The basic assumption of the "dual strategy hypothesis" is that the electromyogram represents a low pass filtered version of this rectangular "excitation pulse" that can vary in height (intensity) and width (duration). By observing which of these parameters is modulated one can interpret the pattern of muscle activation associated with a movement task.

According to this approach, during the performance of single-joint movements, the motor control system uses two strategies to control this rectangular "excitation pulse." The first strategy is named "speed insensitive" (Gottlieb, Corcos, & Agarwal, 1989a), and is used when there are no explicit or implicit constraints upon movement time. Under this task condition only the duration of the "excitation pulse" affecting the agonist muscles is modified,
while the antagonist muscles are activated later for longer movements. The "speed insensitive strategy" (Gottlieb et al., 1989a) predicts coinciding EMG and kinematic traces at movement onset. The second strategy is termed "speed sensitive" (Corcos, Gottlieb, & Agarwal, 1989), and is used for movements performed under the requirement of a specific movement time. Under this task condition the pulse intensity is one of the parameters modulated, and the duration is kept constant while the antagonist muscles are activated earlier for faster movements. The "speed sensitive strategy" predicts that kinematic and EMG traces diverge shortly after movement onset.

The relationship between myoelectric and kinematic parameters as a function of practice

Motor performance enhancement with training has been well reported in the literature for neurologically normal individuals for almost one century (Woodworth, 1899). This phenomenon occurs during the practice of movements of both single mechanical degree-of-freedom (Darling & Cooke, 1987a; Darling & Cooke, 1987b; Gottlieb, Corcos, Jaric, & Agarwal, 1988) and multi degree-of-freedom (Kottke, Halpern, Easton, Ozel, & Berrill, 1978). We summarize two studies that focused on the relationship between myoelectric and kinematic parameters as a function of practice according to the "dual strategy hypothesis" (Corcos, Jaric, Agarwal, & Gottlieb, 1993; Jaric, Corcos, Agarwal, & Gottlieb, 1993).

Corcos and colleagues conducted a series of studies to determine how the intensity of the excitation pulse and the antagonist latency change over time to enhance motor performance (Corcos et al., 1993). Individuals were trained to perform fast single joint movements (1400 trials) towards a fixed target, under the instruction to move "as fast as possible." With training these subjects increased levels of muscle activation, and in most cases activated their antagonist muscles earlier and moved more quickly. As a result the subjects were able to increase peak movement velocity, and increase both peak acceleration and deceleration. However, the increase in peak deceleration was greater than peak acceleration, and the variability as measured by the standard deviation of peak velocity and final position was
decreased. These improvements in the kinematics of the movements were attributed to an increase in the intensity of motoneuron pool activation according to the rules of the speed-sensitive strategy (Corcos et al., 1989).

In a second experiment (Jaric et al., 1993), the same subjects were also asked, at the pre- and at the post-test, to perform elbow flexion movements, but over five different distances. The same changes in kinematic and EMG observed at the one trained distance were well transferred to non-trained distances. On average movement time decreased 20 ms across all distances after training. Finally, the myoelectric data showed that the intensity of the excitation pulse increased at the posttest, across all distances, beyond the level that was maximal at the pretest.

General characteristics of individuals with Down syndrome

Individuals with Down syndrome are defined as those bearing a chromosomal abnormality due to a trisomy of the 21st chromosome, translocation or mosaicism. We will always refer to the subjects as individuals with Down syndrome. By doing so we intend to avoid any pejorative connotation, such as mongolism. This term was first used by John Langdon Haydon Down (Down, 1866) to describe individuals with Down syndrome, because of some facial resemblance they shared with the Mongol people. Despite their outgoing and affectionate way of being (Silverstein, Legutki, Friedman, & Takayama, 1982), the literature about individuals with Down syndrome usually focused on their abnormal characteristics, in relation to the "normal population" or even to individuals with other mental handicaps.

Atypical and delayed milestone achievements

Gesell's theory of dependent stages states that infant's motor development occurs in a universal and invariant order of stages that unfold in the same sequence. According to Gesell (1946) the biological maturational process, which is ontogenetic in nature, internally guides the infants through a sequence of stages, despite environmental factors. Many descriptive studies
have been done to build motor development scales, based on Gesell's theory of dependent stages (Fishler, Share, & Koch, 1964), and to try to apply them to identify possible delays.

The literature in this area showed that individuals with Down syndrome reach the milestones in the same basic order as normal children. However, in comparison with neurologically normal individuals this progression is delayed (Bruininks, 1974; Carr, 1970; Carr, 1975; Carr, 1989; Cowie, 1970; Hartley, 1986; Shumway-Cook & Woollacott, 1985, Haley, 1986). For example, children with Down syndrome sit and stand unsupported around 10 months after their neurologically normal peers (Carr, 1970). On the other hand, the slower rate of motor development of young individuals with Down syndrome (from 6- to 24 months -- Carr, 1970; Dicks-Mireaux, 1966; Dicks-Mireaux, 1972) could be avoided with appropriate training (Edwards & Yuen, 1990).

However, a close analysis of the progression along these milestones showed an atypical pattern of development (Haley, 1987; Harris, 1984; Lydic & Steele, 1979; Parker, Bronks, & Snyder Jr, 1986). Parker and colleagues (1986) studied the walking pattern of 5- and 10-years-old individuals with Down syndrome. Compared with normal children, infants with Down syndrome showed wide developmental variability in their walking pattern, with a delay in some components of the walking cycle (i.e., cadence, stride and step length). Also, the posture of children with Down syndrome was more flexed at the hips and knees, with increased fluctuation of the ankles during the walking cycle. The authors associated this atypical walking pattern with the impairment of the neuromuscular mechanisms of individuals with Down syndrome.

Another example of atypical development comes from a study of Shumway-Cook and Woollacott (1985), who found that the postural synergy of young children with Down syndrome (22-months-old) was poorly organized when compared to their older peers with Down syndrome (4- to 6-years-old). However, even neurologically normal young children (3-year-old) can not appropriately modulate their postural reactions, they usually display better
organized and less variable postural responses than their older peers (4- and 5-year-old). The authors advocated that this difference in the sequence of the postural reactions could represent a difference in the evolution and development of postural control. As we will see below, this atypical sequence of development is also observed in other aspects of the motor development of individuals with Down syndrome. Several attempts have been made to unify these findings and discover a common organic cause underlying the delay in motor development, and the atypical sequence of some of their movement patterns. We will review some of these causes and how they could be related to the delay in motor development in individuals with Down syndrome.

**Hypotonia**

Hypotonia is the most common characteristic observed in individuals with Down syndrome (Cowie, 1970; Crome, Cowie, & Slater, 1966; McIntire, Menolascino, & Wiley, 1965; Morris, Vaughan, & Vaccaro, 1982), and usually is assumed to be the cause of motor deficit (for a literature review see, Harris, 1984). There is no clear definition for hypotonia, and the quantitative methods to measure muscle tone (Duggan & McLellan, 1973) present poor results, reflecting the lack of understanding about hypotonia. A decrease in the resistance to passive stretch of the limbs, floppy muscle mass, and an inability to maintain postures against gravitation are common definitions associated with hypotonia. Low cerebellar weight, poor myelinization of the descending cerebral and brain-stem neurons and a reduction of both the number and the connection of neurons in the higher nervous centers (i.e., motor cortex) have been suggested as cause of hypotonia (Cowie, 1970; Crome et al., 1966). Physiologically, Gilman, Bloedel, and Lechtenbergs (1981) defined cerebellar hypotonia as a decreased motoneuron pool excitability and pathology of the stretch reflex. Because of the low cerebellar weight of individuals with Down syndrome (Woollacott & Shumway-Cook, 1986), this definition has also been used to explain their hypotonia (Davis & Sinning, 1987). The possible inability of individuals with Down syndrome to properly activate their muscles, is corroborated, at the molecular level, by the studies showing their deficiency in the amino acid
5-hydroxytryptophan (Coleman, 1973). This amino acid is involved in the neural transmission (McCoy, Segal, & Strynadka, 1975) and in muscle contraction (Ahlman, Grillner, & Udo, 1971). However, as we will see below, several studies do not seem to support the idea that individuals with Down syndrome have a decreased motoneuron pool excitability and pathological stretch reflex.

**Primitive reflexes versus postural reactions**

With development there is a dissolution of primitive reflexes (i.e., asymmetrical tonic neck, palmar and plantar grasp reflexes), followed by the emergence of postural adjustment reactions, such as, righting, propping and tilting (Bobath, 1972). Molnar (1978) did not find any delay in the dissolution of primitive reflexes in infants mentally retarded without Down syndrome, but found a significant delay in the appearance of their postural adjustment reactions, which correlated well with the delay in motor development. However, other authors reported a predominance of primitive reflexes, controlled at the spinal level, over more centrally integrated and coordinated reflexes in individuals with Down syndrome (Cowie, 1970; Morris et al., 1982). Poor integration of primitive reflexes and a delay in the emergence of righting and equilibrium reactions could then delay the motor development of individuals with Down syndrome (Cowie, 1970). On the other hand, Shumway-Cook and Woollacott (1985) did not find differences in tonic activity and in monosynaptic reflexes between individuals with Down syndrome and control subjects in response to platform rotation.

**The tonic stretch reflex**

The equilibrium-point hypothesis (Feldman, 1966; Feldman, 1986) advocates that during the control of a single-joint movement, the motor control system centrally modulates the threshold of the tonic stretch reflex. The modulation of this reflex would define, at a certain level, the joint compliant characteristic (JCC) which is the dependence of joint angle upon joint torque. Usually JCC is recorded by asking the subjects to hold a manipulandum, against a constant load which is applied, in a certain initial joint position. Then the subject is required
"not to intervene voluntarily." In other words, not to change voluntary commands to their muscles when the load changes. After that, the torque that is driving the manipulandum is changed, moving the joint to a new equilibrium position. This process is repeated with different values of torque. By recording the final limb position produced by each torque value, a line on a torque-angle plane can be drawn. This line is assumed to represent the JCC and its slope represents the muscle stiffness.

We used this procedure to reconstruct the JCC of individuals with Down syndrome (Latash, Almeida, & Corcos, 1993). These individuals demonstrated a high linear correlation between torque and joint angle, and the slope of the JCC was not different from the neurologically normal individuals. Similar results with individuals with Down syndrome were reported by Davis and Kelso (1982). If one agrees that the JCC, as defined above, reflects the action of the tonic stretch reflex (Feldman, 1974; Feldman, 1986; Houk, 1979) then the conclusion is that the gain of this reflex is normal in individuals with Down syndrome.

Now that we have observed that at least the monosynaptic reflex and the tonic stretch reflex seem to be normal in individuals with Down syndrome, let us focus our attention on the postural reactions. The delay in the development of postural reactions in individuals with Down syndrome is assumed to be caused by their hypotonia (Haley, 1987). Bobath and Bobath (1984) went one step further and suggested that normal tone is an essential condition for the postural reaction to be developed.

**Postural reactions**

The development of postural reactions is an important achievement in child development since it allows children to align their head, trunk and limbs with postural changes. The development of these reactions allows the control of the voluntary movements (Bobath, 1967; Molnar, 1978). As a consequence, children will be able to orient themselves in tridimensional space and explore it. Young individuals with Down syndrome display a delay in postural reactions (Haley, 1986; Haley, 1987; Rast & Harris, 1985; Shumway-Cook &
Woollacott, 1985). Rast and Harris (1985) reported that infants with Down syndrome have difficulties in adjusting their heads in space when pulled against gravity. In order to compensate for this delay babies with Down syndrome develop atypical movement sequences. For example, when pulled from supine to sitting position they seem to stabilize the head by contracting the muscles in the back of the neck. The authors suggested early intervention to promote the development of postural reactions and avoid the development of these atypical strategies. However, Haley (1987) showed, in a cross-sectional study, that the delay in equilibrium reactions in individuals with Down syndrome is usually compensated by using protective responses. When compared with control subjects, these protective responses appear relatively early in the development of individuals with Down syndrome.

**Preprogrammed reactions**

Several researchers advocate that preprogrammed reactions are involved in the correction of vertical posture and locomotion during stumbling (Allum, 1983; Dietz, Quintern, & Berger, 1984; Nashner, 1980). If a movement is abruptly perturbed by an external event, a preprogrammed reaction is released to provide a crude correction, and to guarantee the execution of the primary goal (Bonnet, 1983; Houk, 1976; Marsden, Merton, & Morton, 1977; Rothwell, Day, Berardelli, & Marsden, 1986). This reaction can be observed in the electromyogram and occurs at a latency of approximately 70 ms, just after the monosynaptic reflexes and before the execution of voluntary corrections.

Unlike control groups, individuals with Down syndrome have demonstrated problems with the modulation of the preprogrammed reaction (Latash & Corcos, 1991; Shumway-Cook & Woollacott, 1985). Shumway-Cook and Woollacott (1985) studied the development of neural motor control underlying stance balance in individuals with Down syndrome and neurologically normal subjects. The onset latency of the monosynaptic reflex of individuals with Down syndrome was normal, but the onset latency of the postural reaction (preprogrammed reaction) was significantly slower than in control children. This resulted in an
increased body sway, and sometimes loss of balance. The normal short latency response, followed by a delay of long-latency reflexes is a major characteristic of patients with cerebellar lesions (Nashner, Shumway-Cook, & Marin, 1983). Because of that, Shumway-Cook and Woollacott (1985) again suggested that the poor balance of individuals with Down syndrome could be due to their low weight of the cerebellum.

Another way to test preprogrammed reactions is to require the subjects to hold a limb position against an external constant load. The limb is then perturbed and the subject is asked to either "do not react" (i.e. "let the manipulandum move your arm") or "react as fast as possible" (i.e., "do not let the manipulandum move our arm"). Under these experimental conditions, control subjects display a preprogrammed reaction when the limb is perturbed. Using this experimental procedure, Latash and Corcos (1991) found that just 1 out of 10 individuals with Down syndrome could modulate the preprogrammed reaction.

However, in a similar experiment, all eight individuals with Down syndrome tested showed EMG reactions in response to changes in instructions, typical of preprogrammed reactions (Latash et al., 1993). In order to calculate these preprogrammed reactions, we integrated the EMG activity during the interval from 75 to 125 ms, starting from the beginning of the perturbation. Then, the EMG was normalized by subtracting the integrated EMG activity that occurred 50 ms just before the perturbation. There are two reasons to believe that the interval from 75 to 125 ms represents a preprogrammed reaction, and not a voluntary correction. First, reaction time reported for individuals with Down syndrome (see reaction time below) is much longer than 125 ms, which does not allow enough time for voluntary correction. Second, previous studies recorded a similar latency for preprogrammed reactions for neurologically normal subjects (Hammond, 1954; Marsden, Merton, & Morton, 1976) and for one individual with Down syndrome (Latash & Corcos, 1991).

Our study (Latash et al., 1993) did not support the previous idea that individuals with Down syndrome have deficits in preprogrammed reactions (Latash & Corcos, 1991;
Shumway-Cook & Woollacott, 1985). One explanation for these opposite results may be due to experimental procedures. For example, in our experiment the subjects received extensive explanation and simulation of the perturbation. Before the experiment, one experimenter stood behind the subject, grabbed the proximal part of the manipulandum, and simulated the perturbation. Also, the subjects were strongly reinforced if they followed the instruction. Finally, as pointed out by Latash (1992) each trial was analyzed separately. Once the subjects used two different strategies in consecutive trials, averaging the data could hide the observation of preprogrammed reactions.

The first strategy used by individuals with Down syndrome was characterized by a reciprocal pattern of muscle activation in response to loading or unloading perturbations. In response to a loading perturbation the subjects increased agonist activation and suppressed antagonist activation. In response to an unloading perturbation, they suppressed agonist activation and increased the antagonist activation. This reciprocal pattern of muscle activation in response to a loading or unloading perturbation is usually observed in neurologically normal individuals. The second strategy involved agonist-antagonist coactivation, which was independent of the perturbation. Despite the amplitude of the perturbation the subjects coactivated their muscles to react to it. In our study, individuals with Down syndrome used both strategies, sometimes separately, sometimes in combination (Latash et al., 1993).

In summary, individuals with Down syndrome have a normal monosynaptic (Shumway-Cook & Woollacott, 1985) and tonic stretch reflex (Davis & Kelso, 1982; Latash et al., 1992), and display normal latency of the preprogrammed reactions (Latash et al., 1993). Also, individuals with Down syndrome can normally activate their muscles as suggested by the studies in response to platform rotation (Shumway-Cook & Woollacott, 1985). These authors did not find differences in tonic activity between individuals with Down syndrome and control subjects. Taken together, these findings do not support the idea that hypotonia, in individuals
with Down syndrome, results from a pathology of the stretch reflex mechanism in individuals with Down syndrome, and a decrease in motoneuron excitability.

**Hyperflexibility**

Hypotonia and the hyporeflexia are associated with hyperflexibility of the joints in individuals with Down syndrome. Parker and James (1985) compared the joint flexibility (shoulder, elbow, wrist, hip, knee, and ankle) in individuals with Down syndrome and in control subjects, from ages 5, 10 and 15 years-old. The authors reported a decline in flexibility with age, for both groups. Although the individuals with Down syndrome were more flexible than the control group, they had substantial reduction in mobility between ages 10 and 15 years. The decrease in hyperflexibility during this age was also followed by an increase in the gain of height (Rarick, Rapaport, & Seefeldt, 1964) and by increase in skeletal maturation (Rarick & Seefeldt, 1974).

It is also important to know, when working with or testing individuals with Down syndrome, that the congenital laxity of ligaments can provoke major complications, such as, atlanto-axial instability, which is associated with occipitoatlantal instability, and is common among individuals with Down syndrome (Hreidarsson, Magram, & Singer, 1982). Atlanto-axial subluxation can provoke spinal compression that may require surgical treatment. Gait disturbance, progressive clumsiness, head tilt and spasticity in the legs are common manifestations of spinal compression. The detection of abnormal space between the odontoid and the anterior arch of the atlas is a good indicator of spinal compression. In this case large amplitude movements of the neck and head should be avoided. The same care should be taken before asking individuals with Down syndrome to produce excessive force. Usually they have cardiovascular anomalies (Coleman, 1978) and the use of excessive force in this case can be very dangerous.
Grasping and strength

Morris and colleagues (1982) reported that Down syndrome subjects have a diminished muscular grip strength as compared with neurologically normal individuals. However, Hogg and Moss (1983) did not find differences in the sophistication of prehensile grip between preschoolers with Down syndrome and their neurologically normal peers. Cole, Abbs and Turner (1988) studied the ability of individuals with Down syndrome and neurologically normal individuals to adapt grip forces to changes in the properties of lifted objects. Unlike neurologically normal individuals, individuals with Down syndrome were unable to modulate grip force with changes in the frictional properties of the objects. The individuals with Down syndrome grasped the object with the production of excessive force. By producing excessive grip force the individuals with Down syndrome could ensure adequate grip force for a variety of tasks. Studies with tracking tasks reported similar results. When encouraged to go fast, individuals with Down syndrome merely pressed harder on a tap-pad or tracing surface (Frith & Frith, 1974; Henderson, Morris, & Frith, 1981a).

Besides diminished muscular grip strength, individuals with Down syndrome display an overall muscular weakness when compared with neurologically normal individuals (Pitetti, Climstein, Mays, & Barrett, 1992). Pitetti and colleagues compared isokinetic arm (elbow flexion and extension) and leg (knee flexion and extension) strength of individuals with Down syndrome, with individuals mentally retarded without Down syndrome (MR), and sedentary young adults without mental retardation (SYA). The arm and leg strength were significantly higher for SYA individuals than for individuals with Down syndrome and MR individuals. Also, individuals with Down syndrome demonstrated inferior leg strength when compared to their peers with mental retardation. Brown (1977) showed that this muscular weakness of individuals with Down syndrome was reduced with training. However, Davis and Sinning (1987) did not find a significant increase in muscular strength of individuals with Down syndrome submitted to 8-weeks of weight-training using free weights. Even after training,
individuals with Down syndrome displayed a smaller magnitude of maximum torque and EMG than non-Down syndrome mental retarded, and control subjects.

**Kinematic and myoelectric parameters**

When neurologically normal subjects are asked to move "as fast as possible" their movements are fast, which correlates well with force if the moment of inertia is kept constant. However, when asked to perform elbow flexion movements in the horizontal plane "as fast as possible," naive individuals with Down syndrome moved very slowly (Latash & Corcos, 1991). One implication of this study could be the inability of individuals with Down syndrome to generate appropriate levels of force in an isotonic conditions. Also, a kinematic analysis of the movements of individuals with Down syndrome displayed a bell-shaped velocity curve, but with high trial to trial variability (Latash & Corcos, 1991). For some trials the movement trace was relatively smooth, whereas in others, it was wobbly. This variability was also observed in the electromyogram (EMG). For some trials the subjects produced EMG patterns typical of slow movements of neurologically normal individuals (Bouisset & Lestienne, 1974; Corcos, Gottlieb, Jaric, Cromwell, & Agarwal, 1990; Freund & Budingen, 1978). The activation of the agonist muscles generate elbow flexion. This agonist activation was then followed by a delayed phasic burst of antagonistic activity which helped to brake the movement. However, in other trials the subjects demonstrated poorly modulated EMGs. Atypical EMGs of individuals with Down syndrome was also reported by Anson (1989a) who showed that these individuals have an atypical inverted sequence of muscle activation order (from distal to proximal) during multi-joint movements.

**Reaction time**

The slowness of individuals with Down syndrome is also reflected in their reaction time. Reaction time (RT) is defined as a measure of the "real time" that someone takes to display one action (response) after receiving a command (stimulus or signal) to do so (Berkson, 1960). The stimulus can be an electric shock, a word, light, etc., directed to any
sensory system (i.e., visual or auditory). The action can vary from a simple to a complex movement, involving part or the whole body. The speed in making a decision is interpreted as an organic process that measures the biological efficiency of the brain to perform mental processes. As the task complexity increases so does the RT, indicating that more time is necessary to process the information (Berkson, 1960). A fast RT could indicate an ability to process information rapidly to produce an appropriate response to the demands of the environment, which is interpreted as an adapted advantage for living. Reaction time is fractionated in two parts, the first is the premotor time, which extends from the time the stimulus is presented to the beginning of the muscle contraction. The second is the electromechanical delay or motor time that is defined as the time between the first discernible electrical activity in a muscle and the first detectable mechanical response. We have showed that the electromechanical delay is so sensitive to the way in which the data are collected and processed that it provides no useful physiological or psychological information (Corcos, Gottlieb, Latash, Almeida, & Agarwal, 1992a).

Because of these limitations we should be very careful when interpreting the data about RT. Nevertheless, the literature shows longer reaction times for individuals with Down syndrome when compared with other mentally retarded subjects or with control subjects (Anson, 1989a; Anson, 1989b; Berkson, 1960; Cowie, 1970; Frith & Frith, 1974; Lincoln, Courchesne, Kilman, & Galambos, 1985; Seyfort & Spree, 1979). Berkson (1960) compared four groups of handicapped subjects with one control group. All subjects were men, from 15- to 30-years-old. In the simple response task, the mean of RT was 250 ms (SD=30) for control subjects, 360 ms (SD=120) for subnormal subjects, 480 ms (SD=150) for severely subnormal, and 830 ms (SD=600) for individuals with Down syndrome. For a complex response these values were 500 ms (SD=70), 810 ms (SD=290), 1310 ms (SD=330), and 2370 ms (SD=920) respectively for control, subnormal, severely subnormal and individuals with Down syndrome. However, more recent studies showed similar reaction time for
individuals with Down syndrome and subjects non-Down syndrome with mental retardation subjects (Mack & Mackay, 1989; Mackay & Bankhead, 1983). In addition to reacting very slowly, individuals with Down syndrome seem to perform poorly in tasks which a time criteria are applied.

A problem of timing

Henderson, Morris, and Ray (1981b) administrated the Cratty Test of Gross-Motor Performance to 18 children with Down syndrome and 18 other children mentally retarded, between the ages of 7 and 14 years. The authors showed that individuals with Down syndrome suffer from specific deficits in motor coordination, performing the tasks much slower and displaying poorer balance than did their mentally retarded peers. The individuals with Down syndrome did particularly badly in tasks in which the time criteria were applied or when they had to plan a sequence of movements to coincide with one external event. In another study Henderson and colleagues (1981a) had individuals with Down syndrome and neurologically normal individuals, matched on mental and chronological age, performing continuous drawing tasks. There were no group differences related to the spatial component of the tasks. The subjects could perceive the regularity of the sinusoidal track while moving along it, and also draw it from memory on stationary paper. However, individuals with Down syndrome did very poorly when a time constraint was imposed on the task. For example, on the acceleration track, individuals with Down syndrome were unable to increase the speed to correctly follow up the track. These studies (Henderson et al., 1981a; Henderson et al., 1981b) supported the hypothesis that individuals with Down syndrome are impaired in using predictability in time to control their movements (Frith & Frith, 1974). In other words, individuals with Down syndrome may not have the ability to plan strategies to perform the task accurately and efficiently. A second explanation is that this poorer level of motor performance may be attributed to a greater emphasis that children with Down syndrome put on accuracy rather than speed (Kerr & Blais, 1985).
Lack of perceptual integration

In addition to impaired predictability in timing, individuals with Down syndrome also seem to lack perceptual integration. Anwar and Hermelin (1979) found that individuals with Down syndrome could effectively use kinesthetic feedback information in the absence of vision to perform a straight-ahead pointing movement to guide a subsequent one. Despite good spatial orientation, when compared with groups of neurologically normal individuals and other severely subnormal children, individuals with Down syndrome had their performance disrupted by asymmetrical pointing. Anwar (1981) advanced the hypothesis that individuals with Down syndrome have problems in integrating the perceptual information, and as a consequence they display a motor delay. The lack of adaptation of individuals with Down syndrome to changes in sensory information is supported by studies with different tasks (Cole et al., 1988; Nativ & Abbs, 1989; Shumway-Cook & Woollacott, 1985; Woollacott & Shumway-Cook, 1986). Cole and colleagues (1988) argued that the inability of individuals with Down syndrome to modulate grip force could reflect a general deficit in sensorimotor integration. As a consequence they could have more problems in postural regulation and hand control which is dependent on somatosensory information.

Other neurological findings

Several studies have focused attention on cerebral specialization in individuals with Down syndrome. The ability of the cerebral hemispheres to process certain types of information is related to hand differences in the performance of various motor tasks (Todor & Kyprie, 1980). Right-hand advantage in motor tasks (i.e., finger-tapping and complex finger sequences) is attributed to left cerebral hemisphere superiority for sequential processing (Todor, Kyprie, & Price, 1982). When someone learns a novel task with one hand, to some degree, transfer of training will occur to another hand. When the task involves finger sequencing in the absence of visual information, this transfer of training is greater for right-handers than for left-handers. In other words, the transfer is asymmetric (Todor & Kyprie,
Several investigators have examined if these patterns of manual asymmetry also occur in individuals with Down syndrome (Edwards, Elliott, & Lee, 1986; Elliott, 1985; Elliott, Weeks, & Jones, 1986). They provide substantial evidence indicating that individuals with Down syndrome are left hemisphere dominant for movement sequencing. However, on manual spatial tasks they fail to show typical left-hand advantages, suggesting they are less lateralized for tasks requiring that type of processing than neurologically normal individuals.

Also, electroencephalographic (EEG) and evoked potential studies with individuals with Down syndrome showed some neurological abnormalities (for review see -- Lott, 1986). The electroencephalographic studies reported that individuals with Down syndrome have an underlying cerebral dysrythmia and a disturbed sleep pattern, with longer total sleep time, more awakening and movement episodes than control subjects (Clausen, Sersen, & Lidsky, 1977). Evoked potential studies showed that individuals with Down syndrome have a deficiency in their inhibitory capacity and event related potentials, implicating a hippocampus disorder. Schafer and Peeke (1982) reported that individuals with Down syndrome failed to habituate to cortical evoked potentials. As advocated by Luria (1963) the lack of central inhibitory capacity is associated with the inability of the brain to adapt to changing environment demands. This lack of plasticity could be one characteristic of brain dysfunction in individuals with Down syndrome.

**Summary of general characteristics of the motor control of the individuals with Down syndrome**

**The negative view**

So far we have observed that several studies have advocated that there are abnormalities in the motor control system. According to this view, an organic dysfunction, such as low cerebellar weight, is assumed to be the cause of a behavioral deficit, for example, poor balance (Shumway-Cook & Woollacott, 1985). The first problem with this point of view is that the correlation between organic dysfunction and behavioral deficit cannot be taken for granted as a
causal relationship. Since we cannot manipulate the causal factor (i.e., produce an organic dysfunction), there is always a possibility of another explanation. The second problem refers to the basic assumption underlying this view, which assumes that an intact neuromuscular mechanism is a necessary and sufficient condition for the movement control. However, a subject can have an intact neuromuscular mechanism and still be unable to perform a motor task. A good example showing that the intact neuromuscular mechanism is not a sufficient condition for movement control comes from studies of alternating stepping movements in normal neonates (Thelen, 1986; Thelen & Niles, 1987) and with Down syndrome babies (Ulrich, Ulrich, & Collier, 1992). Ulrich and colleagues (1992) showed that 11-months-old babies with Down syndrome responded to the treadmill stimulus by producing alternating steps. As non-handicapped young children, the infants with Down syndrome displayed the intact neural substrate necessary for upright locomotion before they were able to walk independently. This study does not support the idea that atypical walking patterns are associated with the impairment of the neuromuscular mechanisms of individuals with Down syndrome (Parker et al., 1986).

The positive view

The studies about alternating stepping drive us to the hypothesis that the motor control mechanism of individuals with Down syndrome may in fact be intact. If this is the case, a good candidate for their delay in motor performance could be the lack of opportunity for practicing the movements during daily life. This view is supported by several studies showing enhancement of motor performance with practice in individuals with Down syndrome (Edwards & Yuen, 1990; Kanode & Payne, 1989; Kerr & Blais, 1987; Kerr & Blais, 1988). Kerr and Blais tested individuals with Down syndrome and other subjects on a discrete pursuit tracking task. During this task the presentation of each new target position simultaneously provided information about movement extent (2 to 4 alternatives) and movement direction (1 or 2 alternatives). Kerr and Blais (1987) found that extensive training (2400 responses)
enhanced the performance of individuals with Down syndrome. This improvement was due primarily to a gain in reaction time with movement time remaining relatively unchanged. The increase in speed in which the movements were performed was followed by a decrease in the number of errors. The subjects also were able to retain their performance almost one year later. Despite this enhancement in performance their reaction time increased as the distance to be moved increased. This finding led the authors to suggest that movement extent appeared to influence performance of individuals with Down syndrome independently of the probable movement direction. Also, it supports the idea that individuals with Down syndrome can automate the spatial component of the task (Henderson et al., 1981a).

In another experiment Kerr and Blais (1988) gave additional specific training to the same group of individuals with Down syndrome reported in the experiment above (Kerr & Blais, 1987) and to another group of individuals with Down syndrome that had low training (800 responses). This additional training was about the directional probability of the task (choice of the direction), and it caused an improvement primarily in terms of movement time, rather than in terms of reaction time. These findings led the authors to advocate that individuals with Down syndrome may process the information about movement direction and movement extent independently and successively (Kerr & Blais, 1988; Kerr & Blais, 1987).

**Specific practice versus high-variability practice**

It is also interesting to note that the variation in the complexity of the training does not seem to affect the performance of individuals with Down syndrome. Among the important components of "schema theory" of motor learning (Schmidt, 1975) is the prediction that an increase in performance variability leads to schema strength. By increasing the variability of practice, recall and recognition are enhanced which allows the performer to select the response more accurately. Because of the strength of the recall schema, the subjects are able to transfer what they have learned to a new task, and better select an appropriate response given the initial conditions and the desired outcome. The variability of practice could also lead to better
retention of the task than specific practice. The acquisition of the learning schema is completed during childhood, and adult motor learning might involve a recombination of old habits rather than learning new ones (Kelso & Norman, 1978). However, study with Down syndrome children did not support the practice hypothesis (Kanode & Payne, 1989). Kanode and Payne (1989) tested 23 Down syndrome children in two throwing tasks. The authors did not find significant differences in performance between the subjects that had specific practice and the subjects that had high-variability practice. This may suggest that practice can be varied for Down syndrome without a decrease in skill development.
PAPER 1. PERFORMANCE ENHANCEMENT OF FAST SINGLE-JOINT MOVEMENTS DURING TRAINING IN INDIVIDUALS WITH DOWN SYNDROME
INTRODUCTION

Individuals with Down syndrome do have some residual problems in their motor control system (Bruininks, 1974; Carr, 1970; Carr, 1975; Carr, 1989; Cowie, 1970; Hartley, 1986; Shumway-Cook & Woollacott, 1985, Haley, 1986). This can be observed by the delay of their motor performance at a very young age. They in fact display some atypical sequences of motor development (Haley, 1987; Harris, 1984; Lydic & Steele, 1979; Parker, Bronks, & Snyder Jr, 1986), and sometimes use different strategies to control their movements (Shumway-Cook & Woollacott, 1985; Latash et al., 1993).

From the literature about the motor control system of individuals with Down syndrome a major observation could be their inability to properly activate their muscles. For example, they did very poorly when a time constraint was imposed on different tasks (Henderson et al., 1981a). When encouraged to go fast they merely pressed harder on a tap-pad or tracing surface (Frith & Frith, 1974; Henderson et al., 1981a). They displayed a smaller magnitude of maximum torque and EMG than individuals with other mental retardation, and neurologically normal individuals (Davis & Sinning, 1987). Individuals with Down syndrome also could not modulate the rate of change of their grip force. When asked to lift objects with different frictional surfaces, they compensated for this problem by prolonging the duration of the grip force (Cole et al., 1988). Finally, even though some individuals with Down syndrome could generate movements with kinematic and EMG traces according to the speed insensitive strategy, their movements were at lower speeds when compared with subjects neurologically normal (Latash & Corcos, 1991).

However, based on the optimistic view about the motor control system of individuals with Down syndrome we can expect that appropriate training would increase their motor performance. The enhancement in motor performance with practice could show that individuals with Down syndrome can properly activate their muscles and produce kinematic and myoelectric changes similar to those observed in neurologically normal individuals (Corcos et
al., 1993; Jaric et al., 1993). Also, similar to neurologically normal individuals, individuals with Down syndrome should be able, with training, to increase the intensity in which they activate their motoneuron pool, generating more force, and, as a consequence, moving faster. As predicted by the speed sensitive strategy, this increment in the level of activation of the motoneuron pool should be followed by early activation of the antagonist muscle (Corcos et al., 1989). In order to address this hypothesis, individuals with Down syndrome were trained to perform fast single-joint movements over one distance.

Unlike several of the studies pointed out above, we focused our attention on changes that occur in the performance of individuals with Down syndrome without comparing these changes with data from normative population. In this study the following questions were addressed: To what extent training can lead to enhanced performance within and between sessions of practice, and what is the mechanism by which this is accomplished? Does the pattern of changes observed within sessions differ from that observed between sessions? Can improvement observed with practice lead to an increase in performance variability? Are the observed averaged changes representative of the individual changes? Is the pattern of muscle activation related to movement? Is the performance improvement due to an increase in the maximum values of the variables recorded or caused by a reduction in the frequency in which small response occur?
METHODS

Subjects

Four male and four female subjects with Down syndrome took part in the experiments. The chronological age and sex of each subject are presented in the Table 1. The subjects and their parents gave informed consent according to protocols approved by The Human Investigation Committee of Rush Medical Center (see Appendix A). We also got the authorization from Human Subjects Review Committee of Iowa State University to perform this study. It is important to point out that the kind of training and the tests we submitted these subjects to did not expose them to any risk of atlanto-axial subluxation or cardiovascular anomalies (see the discussion about hyperflexibility in the General Introduction).

Insert Table 1 here

Experimental protocols

The subjects sat in a chair with their right forearm positioned on a low friction horizontal manipulandum (moment of inertia = 0.086 NmS²/rad). The axis of rotation of the manipulandum was aligned with the elbow joint (90° elbow flexion was defined as zero degree). In front of the subject, a monitor continually displayed a cursor showing the limb position (see Figure 1 in Almeida et al., 1993). Both the target size and the distance were specified with two sets of narrow bars displayed on the computer monitor. The target size was 6° across all experiments.

Practice effects

Between the pretest and the posttest (see paper 2) subjects had ten training sessions of ten blocks of isotonic movements, which consisted of 11 trials each block, at the 36° target distance from the -35° initial position. The total number of practice trials was 1100. During the first day subjects performed the pretest followed by the first training session. Then subjects had two more training sessions per day for four days on the second, eighth, ninth and fifteenth days after the pretest. Finally, on the 16th day after the pretest, subjects performed
the last training session which was followed by the posttest (see Table II). Here we analyze all
data from the first, fourth, seventh, and tenth training sessions, and the first and the last block
of all 10 training sessions.

**Insert Table II here**

The use of any technical words in the instructions was avoided. The experimenter tried
to use expressions familiar to each subject by first asking what she/he called different parts of
the apparatus. Some examples of the instructions used are: "Pull the arm rest (manipulandum)
towards you (flexion direction) as strongly as you can" for isometric flexion; or, "Move as
fast as possible but do not overshoot or undershoot the red line (target distance) too much" for
isotonic movements. A strong and concomitant verbal reinforcement was given if the subject
followed the instruction. For the set of isotonic movements, knowledge of results was based
on the peak velocity of the movement performed by the subject. If the subject increased speed,
the feedback took the form of encouragement: "Now you moved faster than before. This is
fantastic, but you can do better!". If the subject started to play during the trials, verbal
disapproval was used. For example, "Do not do that!" or "I do not like this!". Before the
beginning of each training session the experimenter held the hand of the subject, and they
performed together one trial of elbow flexion (36°).

At the beginning of each trial the subject was asked to relax her/his muscles and to
move after hearing a computer-generated sound together with the experimenter's verbal
command "GO!". Neither reaction time nor accuracy was stressed in these experiments.

**Mechanical measurements**

The elbow angle was measured by a capacitative transducer mounted on the axis of
rotation of the manipulandum. Elbow acceleration was measured by a piezoresistive
accelerometer which was mounted 46.7 cm from the center of rotation at the distal end of the
manipulandum. The accelerometer axis of maximal sensitivity was oriented to measure
tangential acceleration. Acceleration and angle were digitized with 12 bit resolution at a rate of
1000/s. Velocity was derived from the angle signal and low pass filtering at 25 Hz. The torque was measured by a strain-gauge transducer and filtered at 25 Hz.

Movement time was defined as the interval from the first acceleration deflection, which was calculated as 1% of the acceleration peak (line A in Figure 1), to the time in which velocity fell to 5% of its peak (line C in Figure 1). Movement time was divided in acceleration and deceleration time. Acceleration time was defined as the interval of time from the onset of acceleration until it first crosses zero (from line A to line B in Figure 1). Deceleration time was defined as the interval from the end of acceleration time to the time in which velocity fell to 5% of its peak (from line B to line C in Figure 1). This procedure decreased deceleration time around 55% with respect to the time when deceleration first crossed zero (J in Figure 1).

The identification of the onset of the movement is better defined, but the identification of the end of the movement is always arbitrary. For this reason we calculated movement symmetry from the velocity profile. We defined symmetry as the time in which velocity achieves 5% of its peak to the time of its maximum value (see D in Figure 1), divided by the time from its maximum peak to the time in which peak velocity falls to 5% of its peak (see E in Figure 1). Peak acceleration and peak deceleration, as well as peak velocity were defined as the maximum value of each of these parameters. Finally, we defined overshoot as the maximum position achieved during the movement subtracted from the movement target (36°), and final position as the position achieved at 100 ms after the end of the movement time.

**Insert Figure 1 here**

**EMG measurements**

ECG disposable pediatric electrodes (self-adhesive) were placed over the bellies of two agonists (biceps brachii and brachioradialis) and two antagonists (lateral and long head of the triceps). Since the electrodes left their trace on the subject's skin, even after five days, we tried to place the electrodes at the same anatomical site from session to session. These EMGs were amplified (1600X) and band-pass filtered (60-500 Hz). Each signal was digitized at the rate of
1000/s with 12 bit resolution. After that it was full-wave rectified and filtered with a 10 ms moving average window. Then, this signal was displayed at high gain on a computer monitor where the onsets of the agonist and the antagonist muscle were visually estimated for each trial. After that, the data were processed and analyzed. In this study only the data for the biceps brachii and lateral head of triceps are presented. The data for the brachioradialis were qualitatively similar to the biceps and those of the long head of triceps were similar to the lateral head of triceps.

The onset of the agonist muscle was defined as the first time the EMG rose above the baseline. All the trials were aligned for averaging according to this time. The onset of the antagonist EMG was defined at the first sustained rise above baseline. Antagonist latency was defined as the time from the onset of agonist muscle to the onset of the antagonist muscle (see H in Figure 1).

The following procedure was used to help identify the onset of the late antagonist component in each individual trial. First the average of the antagonist EMG for a set of trials was plotted from 100 ms before the antagonist onset to 400 ms after it. Then, the beginning of the late component of the antagonist onset was determined for this averaged record. This value was used to help to estimate visually the late component of the antagonist burst in each individual trial. The trials in which the subject over or undershot the target by more than 10°, and/or in which the agonist or antagonist onset was ambiguous to identify were rejected from further analysis. Also, the first trial of each condition was always rejected for purposes of analysis. Finally, we rejected the trials in which the subjects moved to different directions.

For further quantification, we first tried to normalize the EMGs with respect to the EMG of the maximal voluntary isometric contraction. The isotonic EMGs (see agonist, antagonist, and Q30 below) were normalized by dividing them by the value of the EMG from the maximum voluntary contraction (MVC). The EMG of the MVC was calculated from the trial in which the torque value was the largest by integrating it from the interval between 500
ms and 1000 ms after the agonist onset. Both the isometric and isotonic EMGs used in the normalization procedure came from the same session. However, the subjects did not perform a consistent MVC from session to session. As a consequence, the values of EMG from the maximum voluntary contraction presented considerable variability. By plotting the normalized isotonic EMG values for each subject against peak velocity we observed that the EMG values in some sessions were shifted up or down approximately by a constant value. Second, we tried to normalize the isotonic EMG values by dividing them by the maximum values of the isotonic EMG values into each session. Again the result was a great scatter of the normalized EMG values from session to session. Then we decided to plot the isotonic EMG values against peak velocity without normalizing them. The result was a good correlation between isotonic EMG values and peak velocity, and we decided to use the non-normalized EMGs in our analysis.¹

The agonist EMGs of the isotonic contractions were integrated over two time intervals, the first 30 ms after the agonist EMG onset (Q30 - see G on Figure 1), and from the onset of the agonist EMG to the first zero crossing of the acceleration (Agonist Activity - see G+F in

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¹ Basmajian and DeLuca (1985) pointed out several factors that could influence the amount of EMG activity recorded from a muscle, within and across experimental sessions. Among them are "i) The motor unit recruitment and firing rate properties, ii) the relative location of fast twitch fibers within a muscle and with respect to the detection electrodes; iii) cross-talk from signals originating in adjacent muscles (page, 195), iv) agonist-antagonist muscle interaction; v) the modulation of the EMG signal induced by a relative movement of the electrodes with the active fibers; vi) the force-length relationship of muscles; vii) the possible presence of reflex activity; and viii) the change in the instantaneous center of rotation of a joint which will effect the moment (force X distance) of the tendon insertion" (page, 200). However, even if we assume that we could record standardized isometric EMGs, from session to session, to normalize the isotonic EMGs, this procedure does not account for the variability that can be produced by several of the factors pointed out above, since they may not be equally distributed across trials. Among these factors the replacement of the electrodes is the one that can introduce more variability between sessions. Because of that we should be cautious, mainly when comparing the EMG from different sessions when there were replacement of electrodes. Since we tried to place the EMG electrodes at the same anatomical position for each subject, and we have a good theoretical reason to believe that EMG values correlate well with peak velocity (see discussion), we decided to use in our analysis the non-normalized EMG values.
The time at which the acceleration first crossed zero (line B on Figure 1) is also the time of maximum peak velocity. The antagonist EMGs were integrated over the time interval from the agonist onset to the movement time (see I in Figure 1). Integration over a fixed interval (Q30) was chosen to determine whether the slopes of the initial component of the EMG records were similar across experimental conditions. This method is analyzed in the Appendix 1 of Gottlieb and colleagues (1989a).

Statistical analysis and the presentation of the data

In the introduction we pointed out several questions that we planned to address with this study. The first is to what extent can practice lead to enhanced performance in one experimental session of 110 trials and between sessions, and what is the mechanism through which this is accomplished. The second question concerns whether the pattern of changes observed within sessions differs from that observed between sessions. To answer the first two questions we present the changes observed from the data averaged across subjects, during the four sessions (1st, 4th, 7th, and 10th) and in each session for the ten blocks. Then we present the changes for the first and the last block observed during ten sessions. Repeated measures analyses of variance were performed to determine whether the observed changes, across sessions and blocks, were statistically significant. Because this test can violate the basic assumptions of homogeneity of variance and normality in our repeated design, we then adjusted the degrees of freedom by using the procedure advocated by Huynh and Feldt (1970). Also, to determine the degree of similarity across blocks of different sessions we calculated some intervals of confidence using a modified Bonferroni procedure.²

Our third question refers to the possibility that the changes observed with practice could lead to an increase in performance variability. To answer this question we presented the

² The overall error rate was set at .1 (see -- Shott, 1990).
standard deviation of peak velocity and final position across sessions and blocks, and we ran repeated measures analyses of variance to determine the level of significance of their changes.

The fourth question relates to the degree in which the observed changes averaged across subjects are representative of the individual changes. To answer this question we showed the individual changes observed between sessions and across blocks of practice. We fit the data for individual subjects with a logarithmic relationship and tested them for statistical significance.

The fifth question of this study refers to whether or not the pattern of muscle activation is related to the movement. To answer this question we performed correlation analyses between myoelectric variables and peak velocity for individual subjects, and for data averaged across subjects, over training sessions and over blocks of sessions. Also, we performed correlation analyses between acceleration time and antagonist latency. These data are showed following the presentation of kinematic and electromyographic changes.

Finally, since our analysis is based on averaged data across ten trials for each subject we first present the number of trials of each block of practice that we analyzed. Then we explored the possibility that the improvement in performance could be due to a reduction in the frequency in which small responses occur rather than an increase in the maximum value of the variables recorded. To do so we plotted the frequency distribution of peak velocity for each trial of the 4 sessions of 10 blocks.
RESULTS

We start this result session by presenting a time series of electromyographic and kinematic data to illustrate the kind of kinematic and electromyographic changes we analyzed. Second, we present the quantification of the kinematic changes (peak velocity, the symmetry of velocity profile, movement time, acceleration and deceleration time, and acceleration and deceleration peak). Third, we present the quantification of the changes of the myoelectric activity (the slope of the agonist activity, the quantity of the agonist activity and antagonist activity). Fourth, we present the relationship between myoelectric activity and kinematic parameters (agonist latency versus acceleration time, and peak velocity versus the myoelectric parameters). Fifth, we present the data related to the variability and overshoot (standard deviation of peak velocity, standard deviation of final position, and overshoot). Finally, we present the number of trials for peak velocity and their response distribution. For each kinematic and electromyographic data we started by showing the changes between the 1st, 4th, 7th, and 10th sessions of practice. Then we presented these changes between the first and the last block for each of the 10 sessions.

*Time series*

In Figure 2 we present a time series of kinematic variables (angle, velocity, and acceleration), and the agonist (biceps) and antagonist (triceps lateral) myoelectric activity for subject S2. The data are averaged across the first block of trials of sessions 1, 4, 7, and 10. The slopes of the kinematic and EMG profiles rose more sharply during the late sessions, showing an increase in the intensity of activation of the motoneuron pools. Also, the EMG quantities were higher with training, with a concomitant decrease in movement time and in antagonist latency. The myoelectric changes were followed by an increase in peak velocity, peak acceleration and peak deceleration.

*Insert Figure 2 about here*
The changes observed across blocks 1, 3, 5, and 7 of the first session for subject S2 is showed in Figure 3, for the same kinematic and myoelectric variables presented in Figure 2. Similarly to what we observed across sessions, the kinematic and myoelectric activity were higher with an increase of the number of blocks of training. As we will see later these general EMG and kinematic patterns of improvement across blocks presented more individual variability. To quantify these changes we analyzed the kinematic and EMG parameters of the movement performed by the subjects, between and within sessions of training.

Insert Figure 3 about here

Peak velocity

Changes between the 1st, 4th, 7th, and 10th session of practice.

Figure 4A depicts peak velocity averaged across all eight subjects for movements over 36° during four sessions. Each session is plotted against the ten blocks of practice. A two-way repeated measures analysis of variance with sessions (1st, 4th, 7th, and 10th) and blocks (1st, 2nd, ..., 10th) showed a statistically significant main effect due to sessions ($F(3,21) = 40.39, p < .0001$) and blocks ($F(9,63) = 2.47, p < .0522$). The interaction between sessions and blocks was not significant ($F(27,189) = 1.75, p < .1143$). To see if the same blocks from different sessions were similar, we calculated the confidence intervals using a modified Bonferroni procedure between each of 10 blocks from the 10th session, with their respective blocks from the other three sessions. The range from the small to large values of each confidence interval is presented in Table B1 in the Appendix B. The results showed that in general each block of the 1st and the 4th sessions was significantly different from their respective blocks of session 10. The only exception was for blocks 8, 9, and 10 of session 4, which were not significantly different from the equivalent blocks of the 10th session. Table B1 in the Appendix B shows that the confidence interval for these blocks was not significantly different from zero. Also, except for the 4th block, the other blocks of the 7th and 10th sessions of training were not significantly different.
In Figure 4B we showed data of peak velocity which were fitted with a logarithmic function of sessions. The data were averaged for each of the eight individual subjects. The slopes of each individual subject were pooled together in a group and their significance were tested, with a paired t-test, against the hypothesis of zero slope. The result showed that the individual data for peak velocity were fit by a logarithmic function \( t(7) = 8.73, p < .0001 \).

**Insert Figure 4 about here**

To further understand the enhancement in peak velocity during blocks of training, we also fit the data of individual subjects, within each of the 4 sessions, with a logarithmic function. The result is shown in Figure 5 and displayed considerable variability from session to session. The subjects increased peak velocity with blocks of training in some sessions and decreased in others. Only the slopes of the individual curves of session 4 were significant \( (t(7) = 2.73, p < .0292) \), showing that, for this session, the subjects tended to improve peak velocity with block of training in a logarithmic function. The data from sessions 1, 7, and 10 also were fit by a logarithmic relationship but did not achieve statistical significance.

**Insert Figure 5 about here**

**Changes between the first and the last block for each of the 10 sessions.**

Figure 6A shows that with successive sessions there was an increase in peak velocity for both the first (broken line) and the last block (solid line), and overall the subjects did better at the last block of each session. A two-way repeated measures analysis of variance showed a main effect due to sessions \( (F(9,63) = 15.23, p < .0001) \) and blocks \( (F(1,7) = 5.98, p < .0444) \). The interaction between sessions and blocks was not significant \( (F(9,63) = 2.16, p < .1040) \). However, as we can better observe in Figure 6B the effect due to block seems to achieve a plateau at session 5 (SE5). We calculated the interval of confidence between the first and the last block across each session using the individual data. The results showed that the first and the last block of training were significantly different in the 1st, 3th, and 4th sessions, and were not significantly different in the other sessions.
Averaged peak velocity for each individual subject, from the first (A) and the last (B) block of trials, was plotted in Figure 7 against the logarithm of the ten sessions. The subjects improved their peak velocity over sessions for the first block and the last block of practice. Paired t-tests for the slopes were $t(7) = 8.73, p < .0001$ and $t(7) = 5.70, p < .0007$, respectively for the first and the last block.

Movement time

Changes between the 1st, 4th, 7th, and 10th session of practice

The data in Figure 8A depict the averaged movement time for all eight Down syndrome subjects, which decreases with sessions. For the purpose of analysis, movement time was divided into both acceleration time (Figure 8B) and deceleration time (Figure 8C). A two-way repeated measures analysis of variance was performed to assess the effect of session and block, for both acceleration and deceleration time. The results showed a decrease in acceleration time with sessions ($F(3,21) = 24.08, p < .0001$) and with block of practice ($F(9,63) = 5.68, p < .0063$), and the interaction between both was not statistically significant ($F(27,189) = 2.255, p < .0622$). Similarly, the deceleration time decreased with sessions of practice ($F(3,21) = 13.36, p < .0023$), but did not decrease with blocks of practice ($F(9,63) = 1.31, p < .2487$). Also, there was not interaction between blocks and sessions ($F(27,189) = 1.31, p < .2487$). The data of individual subjects for acceleration time and deceleration time were for the 1st, 4th, 7th, and 10th sessions and plotted against the logarithm of the ten blocks of practice. Similar to what we observed in Figure 5 for peak velocity, just the data of the acceleration time of session 4 were statistically significant $t(7) = 2.74, p < .0291$. 

Insert Figure 6 about here

Insert Figure 7 about here

Insert Figure 8 about here
Changes between the first and the last block of each 10 sessions.

The movement time data were averaged for all eight subjects, for the first and the last block, of each of the ten sessions of practice and are presented in Figure 9A. These data were divided into acceleration and deceleration time, and are presented in Figure 9B. A two-way repeated measures analysis of variance showed that acceleration time decreased with sessions ($F(9,63) = 7.974, p < .0001$) and with blocks of practice ($F(1,7) = 5.143, p < .0577$). The interaction between both was not significant ($F(9,63) = 1.11, p < .3693$). However, for deceleration time there was a main effect due to sessions ($F(9,63) = 3.477, p < .0134$) with no main effect due to blocks ($F(1,7) = 1.817, p < .2197$) and no interaction between blocks and sessions ($F(9,63) = 1.60, p < .1853$).

Insert Figure 9 about here

Symmetry

Between the 1st, 4th, 7th, and 10th session of practice

Figure 10A depicts movement symmetry as measured by the velocity profile, during the 1st, 4th, 7th, and 10th sessions of training. A two-way repeated measures analysis of variance showed that the velocity profile presented a typical bell shape with the time it takes to achieve its peak being equal to the time it takes to return to its initial level (see methods). This symmetry was observed across sessions ($F(3,21) = .43, p < .7135$) and blocks of practice ($F(9,63) = 1.50, p < .2315$). The interaction between sessions and blocks was not significant ($F(27,189) = 1.03, p < .4092$).

Between the first and the last block of each 10 session.

In Figure 10B we showed the symmetry from the velocity profile for the first (BL1) and the last block of practice (BL10) for each of the ten sessions of practice. A two-way repeated measures analysis of variance also showed symmetry between sessions ($F(9,63) = 1.27, p < .3120$) and blocks of practice ($F(1,7) = 4.10, p < .0827$). The interaction between sessions and blocks was not significant ($F(9,63) = 1.74, p < .2071$).
Insert Figure 10 about here

Peak acceleration and peak deceleration

Changes between the 1st, 4th, 7th, and 10th session of practice

The data in Figure 11 depict the average for all eight Down syndrome subjects for both peak acceleration (A) and peak deceleration (B), for the 1st, 4th, 7th, and 10th sessions plotted against each of the ten blocks of practice. A three-way repeated measures analysis of variance was performed to assess the effect of sessions and blocks of practice of both peak acceleration and peak deceleration, and the symmetry between peak acceleration and peak deceleration. As a result of sessions of practice, the subjects performed their movements at higher peak acceleration and deceleration \( (F(3,2i) = 28.42, p < .0001) \). However, the main effect due to blocks of practice did not achieve significance \( (F(9,63) = 2.18, p < .0860) \). The acceleration and deceleration peaks were symmetrical \( (F(1,7) = .60, p < .4650) \). The interactions between acceleration symmetry (peak acceleration versus peak deceleration) and sessions \( (F(3,2i) = .40, p < .6776) \), between acceleration symmetry and block of practice \( (F(9,63) = .32, p < .8552) \), between acceleration symmetry, sessions and blocks of practice \( (F(27,189) = 1.25, p < .2962) \), and between sessions and blocks \( (F(27,189) = .1.80, p < .0706) \) were not significant.

Insert Figure 11 about here

Changes between the first and the last block of each 10 session.

In Figure 12A the averaged data of peak acceleration (solid line) and peak deceleration (broken line) for the first and the last block is plotted against the 10 sessions of practice. A three-way repeated measure analysis of variance was performed to assess the effects of sessions and blocks of practice of both peak acceleration and peak deceleration, and the symmetry between peak acceleration and peak deceleration. The results showed an increase of both acceleration and deceleration peak with sessions of practice \( (F(9,63) = 17.88, p < .0001) \). Also, both acceleration and deceleration were higher at the last block of practice as compared with the first one \( (F(1,7) = 6.61, p < .0369) \). The acceleration and deceleration peaks were
symmetrical \( (F_{(1,7)} = .305, p < .5982) \), this also can be observed in Figure 12B where we plotted this ratio. Neither of the interactions were significant. The interaction between acceleration symmetry (peak acceleration versus peak deceleration) and sessions \( (F_{(9,63)} = .25, p < .8751) \), between acceleration symmetry and block of practice \( (F_{(1,7)} = 1.31, p < .2893) \), between acceleration symmetry, sessions and blocks of practice \( (F_{(9,63)} = 1.72, p < .1413) \), and between sessions and blocks \( (F_{(9,63)} = 1.21, p < .3279) \).

Insert Figure 12 about here

Myoelectric activities

a) The slope of the agonist activity

Changes between the 1st, 4th, 7th, and 10th session of practice

The data in Figure 13A are the averaged EMG quantities, of all eight Down syndrome subjects, for the first 30 ms of the agonist activity \( (Q_{30}) \) of the ten blocks of the 1st, 4th, 7th, and 10th sessions of practice. This interval \( (Q_{30}) \) describes the slope of the initial component of the agonist activity. With sessions of practice the subjects activate the agonist with great intensity. A two way repeated measure analysis of variance showed a main effect due to session \( (F_{(3,21)} = 10.13, p < .0031) \), no main effect due to blocks \( (F_{(9,63)} = 1.08, p < .3862) \), and no significant interaction between sessions and blocks of practice \( (F_{(27,189)} = .969, p < .4291) \).

Changes between the first and the last block of each 10 session

In Figure 13B we presented \( Q_{30} \), for the first and the last block of the 10 sessions of practice. Again, a two way repeated measure analysis of variance performed for these data showed a main effect due to session \( (F_{(9,63)} = 4.96, p < .0013) \), no main effect due to blocks \( (F_{(1,7)} = 3.23, p < .1154) \), and no significant interaction between sessions and blocks of practice \( (F_{(9,63)} = .776, p < .5147) \).

Insert Figure 13 about here
b) Quantity of the agonist activity

Changes within and between the 1st, 4th, 7th, and 10th session of practice

The data in Figure 14A are the averaged EMG quantities of all eight Down syndrome subjects for the agonist muscle. With sessions of practice the subjects increased significantly the EMG quantities of the agonist muscles. A two way repeated measure analysis of variance showed significant main effect due to sessions \( (F(3,21) =14.37, p < .0001) \), no main effect due to block \( (F(9,63) = 2.14, p < .0888) \), and no interaction between sessions and blocks \( (F(27,189) = .989, p < .4686) \). As was observed for peak velocity (see Figure 4B), the individual data for the agonist activity over sessions was well fit by a logarithmic function \( (t(7) = -4.91, p < .0017) \). We also analyzed the individual data of the agonist activity from each of the four training sessions against the ten blocks. As for peak velocity (Figure 5), only the data from the 4th session was fitted by a logarithmic function \( (t(7) = -4.72, p < .0022) \).

Changes between the first and the last block of each 10 session

In Figure 14B we showed the agonist activity for the first and the last block of each of the ten sessions of practice. A two way repeated measure analysis of variance showed that the agonist activity increased with sessions \( (F(9,63) =7.97, p < .0001) \) and with blocks of practice \( (F(1,7) = 5.14, p < .0507) \). The interaction between session and block was not significant \( (F(9,63) = 1.11, p < .3693) \). Also, the individual subject data of agonist activity of the first block and the last block of practice were fitted by a logarithmic function. The paired t-tests were \( (t(7) = 6.14, p < .0005) \) and \( (t(7) = 4.23, p < .0039) \) respectively for block 1 and block 10.

Insert Figure 14 about here

c) Quantity of the antagonist activity

Changes between the 1st, 4th, 7th, and 10th session of practice

The antagonist activity for the 1st, 4th, 7th, and 10th sessions is plotted in Figure 15A against each of their ten blocks of practice. A two way repeated measures analysis of variance
showed increase in the antagonist activity with sessions of practice ($F(3,21) = 15.45$, $p < .0001$). Also, there was no main effect due to block ($F(9,63) = .711$, $p < .6540$), and the interaction between sessions and blocks was significant ($F(27,189) = 1.81$, $p < .0454$).

Changes between the first and the last block of each 10 session

In Figure 15B we showed the antagonist activity for the first and the last block of each of the ten sessions of practice. A two way repeated measure analysis of variance showed that the amount of antagonist activity increased with sessions ($F(9,63) = 3.48$, $p < .0134$), but did not increase across blocks of practice ($F(1,7) = 1.82$, $p < .2197$), and the interaction between session and block was not significant ($F(9,63) = 1.60$, $p < .1853$).

Insert Figure 15 about here

Relationship between myoelectric activity and kinematic parameters

a) Antagonist latency versus acceleration time

The antagonist latency averaged for all eight subjects for the 1st, 4th, 7th, and 10th sessions of practice is plotted against acceleration time$^3$ (Figure 16A). Over sessions both antagonist activity and acceleration time decreased linearly. A two way repeated measure analysis of variance showed a decrease in antagonist latency with sessions ($F(3,21) = 6.92$, $p < .0162$) and with blocks of practice ($F(9,63) = 2.88$, $p < .0246$). The interaction between sessions and blocks was not significant ($F(27,189) = 2.06$, $p < .0635$).

Insert Figure 16 about here

We plotted the individual subject data of antagonist latency against acceleration time, for the four sessions of practice, and fit them with linear relationship. This procedure allowed us to check whether a linear decrease between both antagonist latency and the acceleration time

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$^3$ We also plotted the antagonist latency against movement time. However, the correlation observed between both was slightly smaller than the correlation between antagonist latency and acceleration time. For this reason we chose to plot antagonist latency against acceleration time.
could be observed for each individual subject. The intercept, slope and the correlation coefficient for these data are presented in Table B2 in the Appendix B. When the data between sessions of practice (1st, 4th, 7th, and 10th session) were plotted together, the linear correlation between antagonist latency and acceleration time was between .71 and .97. The only exception was for subject (S7) who displayed a negative slope with a coefficient of correlation of .08.

We also calculated the linear correlation between antagonist latency and acceleration time within sessions of practice, for each of the eight subjects. The intercept, slope and the correlation coefficient for these data are also presented in Table B2 in the Appendix B. Then we calculated the confidence interval to determine whether the correlation coefficients for each sessions differed from the correlation coefficients calculated between sessions. The results showed that the correlation coefficients of the 4th and the 10th session were similar to those correlation coefficients calculated across the four practicing sessions. Because acceleration time remained approximately constant at the 10th sessions of practice, we did not expect to see a high linear correlation between antagonist activity and acceleration time within this session.

In Figure 16B we showed the antagonist latency for the first and the last block of the ten sessions of practice plotted against acceleration time. The antagonist latency decreased with acceleration time. A two way repeated measures analysis of variance showed that the antagonist activity decreased with sessions ($F(9,63) = 4.38, p < .0088$) and blocks of practice ($F(1,7) = 10.41, p < .0145$), and the interaction between session and block was not significant ($F(9,63) = 1.04, p < .4038$).

b) Peak velocity versus $Q_{30}$

Figure 17A depicts peak velocity plotted against $Q_{30}$. The data were averaged for all subjects for the 1st, 4th, 7th, and 10th session of practice, and were well fit by a logarithmic relationship ($r = .92$). We also plotted the individual subject data of peak velocity against $Q_{30}$, and fit them with a logarithmic relationship. We did that for the data within and between the
four practicing sessions (see Table B3 in the Appendix B). Overall the coefficients of correlation between sessions was high. To compare the coefficients of correlation between and within sessions we calculated the confidence interval. The result showed that only the coefficients of correlation of the first practice session were similar to those coefficients of correlation between sessions.

In the Figure 17B we plotted peak velocity against $Q_{30}$ for the first and the last block of the ten sessions of practice. The correlation between peak velocity and $Q_{30}$ between sessions was also high ($r= .92$).

**Insert Figure 17 about here**

c) Peak velocity versus agonist activity

Figure 18A depicts data of peak velocity plotted against the data of agonist activity, for the 1st, 4th, 7th, and 10th sessions of practice. The data were averaged across all eight subjects, and fit by a logarithmic function ($r= .97$). We also plotted the individual subject data of peak velocity against agonist activity, and fit them with a logarithmic relationship. We did that for the data within and between the four practicing sessions (see Table B4 in the Appendix B). Overall the coefficients of correlation between sessions was high, ranging from .57 to .91. We then calculated the confidence interval between the coefficients of correlation within sessions with coefficients of correlation across sessions. The result showed that the coefficients of correlation of peak velocity with agonist activity of the first and the fourth practice session were similar to those coefficients of correlation between sessions.

In Figure 18B we averaged the individual data of peak velocity, and plotted it against the averaged data of agonist activity, for the first (Block 1) and the last block of practice (Block 10). Again, the correlation between peak velocity and the logarithm of the agonist activity was very high ($r= .94$).

**Insert Figure 18 about here**
d) Peak velocity versus antagonist activity

Figure 19A depicts averaged data of peak velocity plotted against the equivalent averaged data of antagonist activity for the 1st, 4th, 7th, and 10th sessions of practice. These averaged data were fitted well by a logarithm function ($r = .98$), and so it was the individual subject data which presented a coefficient of correlation ranging from .57 to .91 (see Table B5 in the Appendix B). We also calculated the correlation between peak velocity and antagonist activity, for each individual subject, within each of the four practice sessions. We then calculated the confidence interval between the coefficients of correlation within sessions with coefficients of correlation across sessions. The result showed that only the coefficients of correlation of peak velocity and antagonist activity of the first practice session were similar to those coefficients of correlation between sessions.

In Figure 19B we averaged the individual data of the peak velocity and plotted it against the averaged data of agonist activity, for the first (Block 1) and the last block of practice (Block 10). The results showed a good correlation between peak velocity and the logarithm of the antagonist activity ($r = .90$).

Variability and overshoot

a) Standard deviation of peak velocity

Figure 20A depicts the standard deviation of peak velocity of the four sessions of practice plotted against the ten blocks. A two way repeated measure analysis of variance showed that the standard deviation did not increase with sessions ($F(3,21) = .22, p < .7077$) nor with blocks of practice ($F(9,60) = .729, p < .5437$), and the interaction between sessions and blocks was not significant ($F(27,180) = .976, p < .4677$). In Figure 20B we showed the standard deviation of peak velocity plotted against the four practice sessions. All subjects, with exception of subject (S4), did not increase the standard deviation of peak velocity with sessions of practice.
We also plotted the standard deviation of peak velocity for individual subjects for the first (Figure 21A) and the last block (Figure 21B), for each of the ten sessions of practice. A two way repeated measure analysis of variance showed that the standard deviation did not increase with sessions of practice ($F(9,63) = .52, p < .7326$). Also, on average, the subjects displayed less variability at the last block than at the first block of practice ($F(1,7) = 6.38, p < .0395$). The interaction between sessions and blocks was not significant ($F(9,63) = 1.13, p < .3604$).

**b) Standard deviation of final position**

Figure 22A depicts the angle at the final position for the 1st, 4th, 7th, and 10th sessions, and Figure 22B shows its standard deviation. A two way repeated measure analysis of variance showed that the variability of final position did not increase with sessions ($F(3,21) = .1.96, p < .1905$) nor with blocks of practice ($F(9,63) = 1.73, p < .1937$). Also, the interaction between sessions and blocks was not significant ($F(27,189) = .534, p < .7202$).

**c) Overshoot**

Figure 24 depicts the degree with which each individual subject overshot the target during the ten blocks of the 1st, 4th, 7th, and 10th sessions of practice. Overall the subjects
tended to overshoot the target by 4.7, 4.8, 5.8, and 5.7°, respectively for the 1st, 4th, 7th, and 10th sessions of practice. However, a two way repeated measure analysis of variance showed that the difference in overshooting was not significant during the sessions (F(3,21) = .66, p < .4819) and blocks of practice (F(9,63) = .50, p < .8706). The interaction between sessions and blocks also was not significant (F(27,189) = .521, p < .9155).

Insert Figure 24 about here

We obtained similar results with the analysis of the degree of overshooting during the first (BLOCK 1) and the last (BLOCK 10) of each of the ten sessions of practice (Figure 25). A two way repeated measure analysis of variance showed that the standard deviation of final position did not increase with sessions (F(9,63) = 1.08, p < .3862), nor with block of practice (F(1,7) = .103, p < .7574). The interaction between sessions and blocks was not significant (F(9,63) = 1.18, p < .3264).

Insert Figure 25 about here

Number of trials and response distribution

On average the total number of trials analyzed was 6.6, 7.5, 7.6, and 8.1 respectively for sessions 1, 4, 7, and 10. The number of trials analyzed in each session, for each of the ten blocks and for each subject are presented in Figure 26. A two way repeated measure analysis of variance did not show a significant difference between sessions (F(3,21) = 3.31, p < .0645) and across blocks (F(9,63) = 1.09, p < .3846). The interaction between sessions and blocks also was not significant (F(27,189) = 1.17, p < .3064).

Insert Figure 26

Similar results were obtained when we analyzed the number of trials for blocks one and ten, for each of the ten sessions. These data are presented in Figure 27.

Insert Figure 27

All the analyses we performed so far were based on the data averaged across these trials (see Figure 26, Figure 27) which were categorized as blocks of trials. Even though the
number of trials in one block was small (7.5 on average) there was the possibility that the kinematic and myoelectric improvement was due to a decrease in the frequency of the small responses, rather than an increase in the number of the maximum values of the variables recorded. A visual inspection of the individual data of peak velocity showed an increase in the number of the maximum values of the trials with a session of practice. In Figure 28A we presented the values of peak velocity recorded for each trial performed by the subject S2. The data are for each of the ten blocks of the 1st, 4th, 7th, and 10th session of practice. In Figure 28B we plotted the frequency distribution of the trials for each of the four sessions. As we can observe the frequency of the maximum values shifted to the right with sessions of practice. For example, at the 10th session 92% of the all trials of peak velocity were above 410 °/s whereas during the first session this number was just 15%. All eight individuals with Down syndrome displayed this shift to the right of the maximum value of peak velocity and agonist activity.

Insert Figure 28 about here
DISCUSSION

*Enhancement of motor performance between sessions of training*

The improvement in motor performance of individuals with Down syndrome between sessions of practice, as measured by kinematic and myoelectric activity, was well described by a logarithmic function. This finding was observed for all eight subjects analyzed, and is in accordance with the literature in motor learning, which reports a curvilinear improvement in motor performance as a function of training. This curvilinear pattern of improvement is well fit by a logarithmic relationship, and is independent of the motor task (Welford, 1987).

A similar finding was reported for neurologically normal individuals, also performing elbow flexion movements (Corcos et al., 1993), whose peak movement velocity changed as a function of the logarithm of the amount of training. Nevertheless, the improvement in peak velocity of the neurologically normal individuals differed in two ways from individuals with Down syndrome. First, the averaged absolute amount of improvement in peak velocity was larger for individuals with Down syndrome (150 °/s) than for neurologically normal individuals (95°/s). Second, as measured by the slope of the logarithmic relationship between peak velocity and the amount of training, the rate of gain in peak velocity was larger for individuals with Down syndrome (Figure 6) than for neurologically normal individuals. The slopes of peak velocity for the first block of practice were 152 and 97, respectively for individuals with Down syndrome and for neurologically normal individuals.

The implication of the changes observed between sessions being described by a logarithmic function is that the greatest improvement occurs in the early phase of training. After 37% of the training, individuals with Down syndrome were already able to move at the same speed as they did at the end of the training, and at 70% of training, their peak velocity across blocks of practice was not distinguishable from that at the end of the training (Figure 4A). This fast rate of improvement in motor performance of individuals with Down syndrome does not support the common belief that they learn in a slow pace.
Enhancement of motor performance within sessions of training

So far we have showed that the pattern of changes observed between training sessions is described by a logarithmic relationship. We would also argue, based on the anova tests, that individuals with Down syndrome increased their peak velocity within training sessions. However, a different picture emerged when we analyzed the pattern of these changes within experimental sessions. First, only the individual enhancement in performance observed in the 4th session of training was fit by a logarithmic relationship (Figure 5). Second, there was individual variability in the gain in performance within each session. For example, within the first session, four out of eight subjects did not improve their average peak velocity among the ten blocks of practice, and within the fourth session of practice, two out of eight subjects did not increase their peak velocity. This finding reveals that within training sessions the improvement in peak velocity is subject dependent. Also, this finding shows us that we have to do additional analyses with the individual data before making any generalization from averaged data.

Symmetry

As we discussed above, the subjects were able to perform their movements faster. This increment in peak velocity was also observed for peak acceleration and peak deceleration (Figure 11 and Figure 12). As a result, the subjects spent less time completing the task, as measured by a decrease in both the acceleration and the deceleration time (Figure 8 and Figure 9). Because of the way we calculated the end of movement time (see methods), the deceleration time was shorter than the acceleration time (Figure 1). Since the end of the movement is arbitrarily defined, the result will be a function of the method used to calculate it. Because of that, we determined symmetry on the velocity profile using the identical algorithm to calculate the beginning and the end of the velocity. The velocity profiles of the movements of individuals with Down syndrome were symmetrical (Figure 10), and so were the increments in the acceleration and deceleration peaks (Figure 12B). These results agree with what we have
reported in Paper 2, where we show that individuals with Down syndrome decreased movement time by proportionally shortening both the acceleration and deceleration time.\textsuperscript{4} Taken together, these results are different from those that have been reported for neurologically normal individuals, who presented a proportionally greater reduction in the deceleration phase than at the acceleration phase, and larger increment in peak deceleration than in peak acceleration (Corcos et al., 1993). It remains to be determined if, with specific training, individuals with Down syndrome can reduce the deceleration phase more than the acceleration phase, and if this can lead to enhancement in performance beyond the level reported here.

\textit{Improving motor performance while keeping variability at a low level}

The subjects in our experiment were allowed to overshoot or undershoot the target size in order to move "as fast as possible." It is remarkable that under this instruction they were very accurate at the beginning of the training and kept this accuracy throughout the training sessions. This could be observed by the standard deviation of both final position (Figure 22 and Figure 23) and peak velocity (Figure 20 and Figure 21), and by the degree of overshooting (Figure 24 and Figure 25), which did not change between sessions of training. The only exception was subject S4 who increased the standard deviation of final position and peak velocity (Figure 20B) with training. The subjects were also more accurate at the last block of practice of each session than at the first one (Figure 21 and Figure 23). The increase in movement speed without a concomitant increase in movement variability is a violation of the impulse variability model (Schmidt & Quinine, 1979). This model predicts that movement time (MT) increases with movement distance (D), and decreases with the standard deviation of final position (SDfp):

\[ MT = k \cdot (D / SDfp) \]  
(eq. 1)

\textsuperscript{4} We used a different algorithm to calculate the end of the movement (see methods on Part II).
Since the distance was kept constant during the training, and the standard deviation of final position did not change between sessions of training, the movement time should be constant. However, with training, individuals with Down syndrome were able to decrease movement time by almost 50%. It was only after an extensive practice (700 trials) that movement time became constant (Figure 8A). After this extensive practice, movement time data could be predicted by the eq.1. The impulse variability model also predicts that because faster movements are generated by the production of greater forces they will produce greater variability. This variability presents a curvilinear shape, increasing until certain level of force and decreasing a little after that (Sherwood & Schmidt, 1980). However, with training, individuals with Down syndrome generated force at a higher level, as measured by the increase of peak velocity, without increasing variability. This finding supports the idea that the trade-off between force and force variability, as advocated by the impulse variability model, does not apply to the situation in which the motor skills are changed as a function of training (Corcos et al., 1993). The implication from our findings is that the trade-off between force and force variability may occur just when the performer is on one of the two practice extremes, either naive or high skilled.

However, unlike neurologically normal individuals, individuals with Down syndrome did not decrease the variability of peak velocity and final position, between sessions of training (Corcos et al., 1993). This does not mean that individuals with Down syndrome were less accurate than neurologically normal individuals. To show this comparison, we plotted the data of standard deviation of peak velocity and final position of individuals with Down syndrome and neurologically normal individuals (Figure 29). When compared with neurologically

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5 The mechanical impulse can be defined as a function of moment of inertia multiplied by peak velocity. Once the moment of inertia was constant for each subject we can assume that the impulse will be proportional to peak velocity. See Gottlieb, Corcos, and Agarwal (1992) for a good review about the methods to measure torque during isometric movements.
normal individuals, the peak velocity variability of individuals with Down syndrome was already very low at the beginning of the training (32°/s, against 50°/s for neurologically normal individuals, see Figure 29A). An explanation for this consistency is that the optimal level of accuracy at the beginning of the training did not leave room for more improvement in variability. However, we have to consider that the neurologically normal individuals moved a larger distance (54°), and to a smaller target (3°). Under this condition, there is a possibility that the variability of the performance of individuals with Down syndrome could be higher than the one reported here.

**Insert Figure 29 about here**

When compared with neurologically normal individuals, individuals with Down syndrome were on average 2° less accurate at the final position (Figure 29B), and their accuracy did not improve with training. However, we would argue that the accuracy with which individuals with Down syndrome hit the target was quite remarkable. First, and most important, the final position for the neurologically normal individuals was calculated more than 1.6 seconds after the end of the movement time (see methods in Corcos et al., 1993). This time was long enough to allow for the correction of the movements after its end. As a consequence this method could artificially decrease the standard deviation of final position. In our experiment, we measured the final position a hundred milliseconds after the end of the movement time, which was much shorter time to allow for corrections. Also, the algorithm we used to calculate movement time reduced deceleration time, making the time from which we calculated the final position even smaller (see methods). Under these conditions, construct validity of the measure of final position was higher in our experiment than in the other experiment with neurologically normal individuals. Second, in our experiment the target size was twice as larger than in the experiment with neurologically normal individuals, and our instructions allowed both overshooting and undershooting of the target. Taking these two
considerations together, it is fair to say that individuals with Down syndrome were very accurate.

*The mechanisms underlying motor performance enhancement*

So far we saw that with training individuals with Down syndrome were able to improve their motor performance without increasing variability. What are the mechanisms underlying this enhancement in motor performance? With training the individuals with Down syndrome increase the level of the intensity of motoneuron pool excitation beyond which was initially maximum. This could be observed by a significant increase in the slope of the agonist activity with sessions of practice (Figure 13). Also, they increase the total amount of the agonist (Figure 14) and the antagonist activity (Figure 15), and seven out of eight subjects activated their antagonist muscles earlier with training (Figure 16). The only exception was subject S7, who kept antagonist latency constant during the training (see Table B1 in the Appendix B).

The earlier activated of the antagonist muscles with training is in contradiction with the data reported for neurologically normal individuals showing an increase in the antagonist latency with training (Normand, Lagasse, Rouillard, & Tremblay, 1982). The increase in the intensity of the motoneuron pool activation and a decrease of the antagonist latency are both predictions of the speed sensitive strategy, when the subject is required to move at different speeds (Corcos et al., 1989). The change in speed can be achieved by explicitly instructing the subject to do so or by imposing different accuracy requirements. However, in our experiment the changes in speed was induced by training. Similar results were reported for neurologically normal individuals (Corcos et al., 1993).

With increases in the intensity of motoneuron pool excitation the subjects were able to fire a larger number of motor units producing more force, and, as a consequence, they were able to move faster. To break the movement in time, the antagonist muscle was activated earlier. This strategy avoids excessive overshoots and allows the reduction of the movement time. The early activation of the antagonist muscles also produces co-contraction, which is a
simultaneous activation of the agonist and antagonist muscle during the early phase of the movement. This co-contraction increases the energy expenditure. However, the same co-contraction produces joint stability helping the performance of fast and accurate movements, as we observed in our experiment. It is interesting to observe that subject S7, who kept antagonist latency constant during the training, had the worst performance. This finding indicates that the ability to activate the antagonist muscle earlier could represent a better adaptative response when the task requirement is speed.

The increase in the intensity of the motoneuron pool activation was followed by an increase in the total amount of the agonist activity (Figure 14). This finding is also consistent with another rule for the speed sensitive strategy that the duration of the excitation pulse remains constant, generating constant agonist burst duration (Corcos et al., 1989). We did not calculate this duration, but visual inspection of Figure 2 and Figure 3 suggested they were approximately constant. A constant agonist burst duration, and an increase in intensity of the agonist activation would naturally lead to an increase in the total amount of the agonist activity.

**Relationship between the pattern of muscle activation and the kinematic of the movements**

Our findings are consistent with several studies that show a scaling of the agonist activity with movement velocity (Gottlieb et al., 1989a; Wadman, Denier van der Gon, Geuze, & Mol, 1979; Cheron & Godaux, 1986; Mustard & Lee, 1987; Finley, Wirta, & Cody, 1968). Several other studies showed a decrease in myoelectric activity of the biceps brachii muscle with training (Engelhorn, 1983; Engelhorn, 1988). However, in these studies the subjects were instructed to keep movement time constant during the training, whereas in our experiment the subjects were encouraged to decrease movement time. The additional finding from our experiment is that the increase of peak velocity, between sessions of practice, correlated very well with the logarithm of the agonist activity (Figure 18). This means that, proportionally, agonist activity increased more than the increase in peak velocity. Indeed, a
similar relationship between force and agonist activity, during an isometric contraction of the elbow flexion, has been shown for neurologically normal individuals (Lawrence & DeLuca, 1983; Davis & Sinning, 1987), for individual with Down syndrome and for individuals with other kinds of mental retardation (Davis & Sinning, 1987). The authors reported that the amount of biceps myoelectric activity increased more than the increase in force, as a consequence the increment in force correlated very highly with the logarithm of the biceps myoelectric activity.

Also, the increment in peak velocity between sessions of training was well correlated with the logarithm of the antagonist activity (Figure 19) and $Q_{30}$ (Figure 17). The bigger increment in myoelectric activity (agonist and antagonist activity, and $Q_{30}$) than in peak velocity was observed for all eight individuals with Down syndrome (see Tables B3, B4, and B5 in the Appendix B). These findings differ in two ways from the one reported for neurologically normal individuals (Corcos et al., 1993b). First, the authors reported that with training, neurologically normal individuals proportionally increased more peak velocity than myoelectric activity. Second, for neurologically normal individuals, the relationship between myoelectric activity and the logarithm of peak velocity was dependent on the subject, and usually it was low. We observed exactly the opposite with the performance of individuals with Down syndrome. The increment in peak velocity between practice session correlated well with logarithm of myoelectric activity, and this correlation was subject independent.

The explanation for this discrepancy in the correlation of peak velocity and the myoelectric activity, between the performance of the neurologically normal individuals and the individuals with Down syndrome, could be attributed to the method used to analyze the EMGs. In the study with neurologically normal individuals (Corcos et al., 1993b) the EMGs were normalized in relation to the EMG of the maximum isometric contraction. We first tried this procedure in our experiment, but it introduced considerable variability in the normalized EMG values (see methods). For this reason, and because we tried to keep the electrodes at the
same anatomical position, from session to session, we analyzed the raw EMGs. However, we have to be careful in drawing any physiological meaning from the EMG data. As pointed out by (Basniajian & DeLuca, 1985): "Even among well-executed studies it is difficult to compare EMG data because the detected signal is a function of the detection procedure as well as the physiological events (page 193)." Nevertheless, the study of the kinematic, as well as the myoelectric activity associated with the movement performance, revealed a pattern of muscle activation, between sessions of training, which could be related to the movement performance.

However, an individual analysis of the correlation between kinematic and myoelectric parameters within sessions showed more individual variability. We did expect to observe a poorer correlation within the 7th and 10th session, since the kinematic parameters of these sessions displayed a plateau. For the opposite reason, we expected the correlation to be high at least for the 4th session of practice, where the individual data of peak velocity were well fitted by a logarithmic relationship (Figure 5). This prediction was partially fulfilled. First, contrary to our expectation, we observed a good correlation between antagonist latency and deceleration time within the 10th session. Second, just within the 1st session peak velocity correlated well with the logarithm of $Q_{30}$ and with the logarithm of antagonist latency. Third, peak velocity did not correlate well with the logarithm of the amount of antagonist activity within the 4th session of practice. This poor correlation between peak velocity and the antagonist activity observed within session 4 is also described for neurologically normal individuals (Gottlieb et al., 1989a; Gottlieb, Latash, & Corcos, 1993). Finally, as we showed between sessions, peak velocity also correlated well with the logarithm of the agonist activity within the 1st and 4th sessions of training.

*The motor control system of individuals with Down syndrome is functionally normal*

The findings we presented in this experiment were very robust. All eight individuals with Down syndrome demonstrated remarkable change in performance as a result of practice of simple elbow flexion task, under reproducible conditions, and with knowledge of result...
based on movement speed. Also, we observed that this improvement in performance was due to an increase in the maximum values of the variables recorded (Figure 28). On average, with training individuals with Down syndrome we able to improve the peak velocity of their movements by 67%, from 210°/s after the first block of training to 350°/s at the end of the training. This improvement in peak velocity was 34% below what was reported for the neurologically normal individuals, trained in a similar experimental protocol (Corcos et al., 1993). However, we should first consider that the neurologically normal individuals were graduate students who had 300 more practice trials than the individuals with Down syndrome. Second, and more important, the neurologically normal individuals were trained to move at a longer distance (54°). Therefore we can conclude that the improvement of the individuals with Down syndrome was similar to individuals neurologically unimpaired.

Besides of being able to perform at a high level, the individuals with Down syndrome also were able to transfer what they learned, moving at one distance, to different distances (see Paper 2). Because of this high level of motor performance with training we can say that the motor control mechanism of individuals with Down syndrome is functionally normal, as could be observed from the kinematic and myoelectric changes reported above. These changes do not support the idea that the motor control system of individuals with Down syndrome cannot properly activate their muscles (Henderson et al., 1981a; Frith & Frith, 1974; Henderson et al., 1981b; Davis & Sinning, 1987; Cole et al., 1988; Latash & Corcos, 1991). This does not mean that individuals with Down syndrome do not have any of the organic or mental problems we reviewed in the introduction. What emerges from this study is that we should be very careful in assuming that an organic dysfunction is the cause of a behavioral deficit. We did not control any of the possible organic dysfunctions which could be somehow associated with motor performance (i.e., hypotonia, low cerebellar weight, etc.). However, despite of any handicap that could affect their motor performance they were able to overcome their own limitations with simple training.
Learning or understanding?

So far we showed that, when trained to move faster, individuals with Down syndrome increased intensity of the motoneuron pool excitation and decreased the antagonist latency, as predicted by the speed sensitive strategy (Corcos et al., 1989). Also, we showed that under this condition the movement velocity increased with the logarithm of the amount of the myoelectric activity. However, one could argue that these kinematic and myoelectric changes are just a function of better task comprehension. In other words, had the individuals with Down syndrome understood the task well at the beginning of the training session they would have performed at a higher level. Let us discuss this point by defining what is motor learning.

Several attempts have been made to define what is motor learning without any definitive conclusion. Nevertheless, we would argue that to be considered learned, the motor task has to satisfy four criteria: i) It has to improve with practice; ii) It will not become more variable with practice; iii) It will be retained over a long period of time, even if the task is not performed; and iv) The performance learned during the training of one motor task will be transferred, or generalized, to different variations of this task. The first three criteria are advocated by several authors (Ito, 1976; Ito, 1984; Brooks & Watts, 1988) and the last one by Schmidt (1988).

The findings presented so far strongly satisfy the first two criteria. The third criterion will depend on what we define as "long" time. In our experiment, the interval between sessions varied from two hours to seven days (see method). Overall the individuals with Down syndrome retained what they learned from one session to another, as measured by the improvement in peak velocity of the first block of each session of training (Figure 7). We did not re-test the individuals with Down syndrome for retention after training, but Kerr and Blais (1987) showed that individuals with Down syndrome were able to retain their performance one year later after intensive training. Finally, we reported on Paper 2, that individuals with Down syndrome were also able to transfer what they learned with the training at 36° to three other
different distances, and to a different initial position. This fulfills the final criterion of generalization.

Also, let us consider what could be the effect of understanding the task on motor performance. If the limiting factor is task comprehension, we would expect an abrupt change in motor performance when the subject finally grasped the meaning of the task. In fact, we observe a great change in peak velocity from the pretest (160°/s -- see Figure 4 in Paper 2) to the first block of practice (210°/s -- Figure 4). Since the subjects had just a few trials of practice and the performance jumped 31%, this change could be attributed to a better understanding of the task. This is also in accordance with the complexity of the task which was very simple and did not require a complex reasoning. Under these conditions, few trials are enough for the subjects to grasp and to adapt to the requirement of the task. We also have to consider that 110 fast movements, represented by one session of training, could "warm up" the muscles, rising the temperature and the blood flow, increasing the work capacity (Astrand & Rodahl, 1977), and the speed of the nerve transmission (Hill, 1927). However, these physiological adaptations could account for the changes observed within a single session, but not the retention in performance between sessions. Finally, the changes in performance reported during the training session were very smooth and gradual. Taking these considerations together and the fact that the motor performance of the Individuals with Down syndrome satisfied the four criteria pointed above, we can conclude that individuals with Down syndrome learned how to improve their motor performance with training.
REFERENCES


### Table I

**Subjects**

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F = FEMALE
M = MALE
Table II

Experimental Protocol

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<td>Posttest</td>
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Figure Captions

Fig. 1. Illustration of one trial of elbow flexion movement showing how kinematic and myoelectric variables were quantified.

Fig. 2. Averaged data of angle, velocity (vel), acceleration (accel), biceps and lateral head of triceps (Tri Lat) for the first block (BL1) of sessions (SE) 1, 4, 7, and 10 for subject S2. This subject was asked to move "as fast as possible" over the same distance (36°). The data are aligned at the onset of the agonist EMG (200 ms). The EMG unit is in μV.

Fig. 3. Kinematic and myoelectric variables are shown for the 1st, 3th, 5th, and 7th block of the first session for subject S2. Figure captions are the same as in Fig. 2.

Fig. 4. A. Averaged peak velocity (n = 8) for sessions 1, 4, 7, and 10 is plotted for each of the ten blocks that encompass each session. B. Peak velocity is averaged across ten blocks for each session and plotted against a logarithmic scale of sessions. The data are for all eight individual subjects, as well as, the averaged data across all subjects (thick line).

Fig. 5. Peak velocity for individual subjects for sessions 1, 4, 7, and 10 is plotted against the logarithm of blocks.

Fig. 6. A. Averaged peak velocity across eight subjects for the first (broken line) and the last (solid line) block of each session. These data are fitted by a logarithmic relationship. B. The same data presented in A are plotted against the first (B1) and the last block (B10) for each session (SE).

Fig. 7. Individual data for peak velocity for block 1 (A) and the last block 10 (B) is plotted against the ten sessions using a logarithmic scale.

Fig. 8. Movement time (A), acceleration (B) and deceleration time (C) for sessions 1, 4, 7, and 10. The data are plotted against blocks of practice, and were averaged across all eight subjects. Time is in milliseconds.

Fig. 9. A. Movement time for each of the ten session of practice was plotted against the first (B1) and the last block (B10) of practice. B. Acceleration (solid line) and deceleration
time (broken line) for the first block (square) and the last block of practice (circle). The data are averaged data across all subjects.

Fig. 10. A. Symmetry from the velocity profile, for the 1st, 4th, 7th, and 10th sessions, plotted against each the ten blocks of practice. B. Symmetry from the velocity profile for the first (BL1) and the last block of practice (BL10) for each of the ten sessions of practice. The data are averaged across all eight subjects.

Fig. 11. Averaged peak acceleration (A) and peak deceleration (B) across all eight subjects, for the 1st, 4th, 7th, and 10th sessions. The data are plotted against each of the ten blocks of training.

Fig. 12. A. depict the average for both peak acceleration (solid line) and peak deceleration (broken line) for the first (triangle) and the last block of practice (square). The data are plotted against each of the ten sessions of practice. B. The ratio of peak acceleration divided by peak deceleration is plotted for the first (solid line) and the last block of practice (broken line). The data are averaged for all eight Down syndrome subjects and plotted against the 10 practicing sessions.

Fig. 13. A. The first 30 ms of agonist activity ($Q_{30}$) is plotted for the 1st, 4th, 7th, and 10th sessions against each of the ten blocks of practice. B. $Q_{30}$ is plotted for the first and the last block of each of the ten sessions of practice. The data represents the average for all eight subjects. $Q_{30}$ is given in µV.

Fig. 14. A. The agonist activity for the 1st, 4th, 7th, and 10th sessions plotted against each of the 10 blocks of practice. B. Agonist activity for the first (circle) and the last (square) block is plotted for each of the ten sessions of practice. These data are averaged over all eight subjects. The agonist activity scale is in µV.

Fig. 15. A. Antagonist activity for the 1st, 4th, 7th, and 10th sessions plotted against each of their 10 blocks of practice. B. The antagonist activity for the first (broken line) and the
last (solid line) block is plotted for each of the ten sessions of practice. These data are averaged over all eight subjects. The antagonist activity scale is in µv.

Fig. 16. A. Antagonist latency is plotted against acceleration time (Tac). The data are averaged for all eight subjects for the 1st (diamond), 4th (triangle), 7th (circle), and 10th (square) sessions of practice, and are fitted with a linear relationship. B. Same as in A. Averaged data are for the first (open square) and the last (closed square) block of the ten sessions of practice.

Fig. 17. A. Peak velocity is plotted against Q30. The data are averaged for all eight subjects for the 1st (closed circle), 4th (square), 7th (triangle), and 10th (open circle) sessions of practice and are fitted with a logarithmic relationship. B. Same as in A. Averaged data are for the first (open square) and the last (closed square) block of ten sessions of practice. Q30 scale is in µv.

Fig. 18. A. Averaged data of the peak velocity are plotted against the averaged data of the agonist activity, for the 1st, 4th, 7th, and 10th sessions of practice. B. The averaged data of peak velocity over all eight subjects, are plotted against the agonist activity for the first (open square) and the last block of practice (closed circle). The agonist activity scale is in µv.

Fig. 19. A. Averaged data of peak velocity is plotted against the averaged antagonist activity, for the 1st, 4th, 7th, and 10th sessions of practice. B. The averaged data of the peak velocity, is plotted against the averaged antagonist activity, for the first (closed circle) and the last block of practice (open square). These data are averaged over for all eight subjects.

Fig. 20. A. Standard deviation of peak velocity of the 1st, 4th, 7th, and 10th sessions plotted against ten blocks of practice. The data are the average of all eight subjects. B. Individual data of the standard deviation of peak velocity averaged across blocks for each of the four sessions of practice.

Fig. 21. Standard deviation of peak velocity of the first block (A) and the last block of practice (B) plotted against the ten sessions. The data are averaged for each individual subject.
Fig. 22. Final position (A) and its standard deviation (B) of the 1st, 4th, 7th, and 10th sessions of practice plotted against each of the ten blocks. The data are averaged for all eight subjects.

Fig. 23. Final position (A) and its standard deviation (B) of the first (BL1) and the last (BL10) block of the ten sessions of practice. The data were averaged for all eight subjects.

Fig. 24. The degree of overshooting for each of the eight subjects, during each of the ten blocks of the 1st, 4th, 7th, and 10th sessions of practice.

Fig. 25. The degree of overshooting during the first (A) and the last block (B) of the 1st, 4th, 7th, and 10th sessions of practice.

Fig. 26. Number of trials plotted for each of the eight subjects for each of the blocks of practice of the 1st, 4th, 7th, and 10th practice sessions.

Fig. 27. Number of trials plotted for each of the eight subjects for the first (A) and the last (B) block of each of the ten practice sessions.

Fig. 28. A. Values of peak velocity of each individual trial recorded for one subject (S2). The data were from each of the ten blocks of the 1st, 4th, 7th, and 10th sessions of practice. The mean for each session is showed with a arrow. B. The frequency distribution of the trials of each session presented in A.

Fig. 29. Standard deviation of peak velocity (A) and final position (B) plotted against the number of trials of practice. The data are the average of eight Individuals with Down syndrome (close circle), and five neurologically normal individuals (open circle). The data for the neurologically normal individuals are from the studies of (Corcos et al., 1993).
Peak Velocity

Final Position

Peak Acceleration

$\frac{D}{E} = \text{SYMMETRY}$

$A \rightarrow B = \text{Acceleration Time}$

$B \rightarrow C = \text{Deceleration Time}$

$A \rightarrow C = \text{Movement Time}$

$G = Q_{30}$

$G + F = \text{Agonist Activity}$

$H = \text{Antagonist Latency}$

$I = \text{Antagonist Activity}$

Fig. 1
Fig. 2
Fig. 3
Fig. 4
SESSION 1

SESSION 4

SESSION 7

SESSION 10

Fig. 5
Fig. 6
Fig. 7
Fig. 10
Fig. 11
Fig. 12
Fig. 13
Fig. 14
Fig. 15
**Fig. 16**
\[ P_{vel} = 472.16 + 218.54 \times \log(Q30) \quad R = 0.92 \]

\[ P_{vel} = 499.27 + 247.05 \times \log(Q30) \quad R = 0.86 \]

Fig. 17
\[ PVEL = 480.33 + 269.19 \times \log(A.Ac) \quad R = 0.97 \]

\[ PVEL = 498.17 + 306.05 \times \log(A.Ac) \quad R = 0.94 \]

**Fig. 18**
Fig. 20
Fig. 21
Fig. 22
SESSION 1

OVERSHOOT (°)

Session 4

OVERSHOOT (°)

Session 7

OVERSHOOT (°)

Session 10

OVERSHOOT (°)

Fig. 24
Fig. 26
Fig. 27
Fig. 28
NEUROLOGICALLY NORMAL INDIVIDUALS

INDIVIDUALS WITH DOWN SYNDROME

Fig. 29
PAPER 2. PRACTICE AND TRANSFER EFFECTS DURING FAST SINGLE-JOINT ELBOW MOVEMENTS IN INDIVIDUALS WITH DOWN SYNDROME
Practice and transfer effects during fast single-joint elbow movements in individuals with Down syndrome.

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Running title:
Motor learning in Down syndrome

Key words:  Motor Control, Motor Learning, Down syndrome, Motor Strategies, Electromyography, Kinematics, Transfer.

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ABSTRACT

Eight subjects with Down syndrome performed elbow flexion movements "as fast as possible" over four distances (18°, 36°, 54°, and 72°), and at a "comfortable speed" over 36°. For two of these distances (18° and 36°), the movements were also performed from a second starting position. The subjects were tested before and after extensive practice on one task (1100 movements "as fast as possible" over 10 sessions at 36°). After training over a 2 week period, all subjects improved their performance on all tasks as reflected by both kinematic and EMG parameters. In particular, they increased the quantity of the agonist activity, decreased the antagonist onset latency, and doubled peak velocity. They were able to transfer the improvement in their performance to the non-trained distances and to the different starting position. Subjects decreased their movement time by proportionally decreasing both the acceleration and deceleration time. This study supports the idea that subjects with Down syndrome can use patterns of muscle activation that are qualitatively indistinguishable from those employed by individuals without neurological impairment. With appropriate training, individuals with Down syndrome can achieve high levels of motor performance.
INTRODUCTION

Because humans have the ability to perform a wide variety of movements under diverse conditions, certain theories of motor control assume that movements are controlled by generalizable rules. The basic idea of these theories is that the motor control system minimizes the number of parameters used to plan and execute movements. One theoretical approach to explain how movements are controlled is the "dual strategy hypothesis," which is predicated on the notion that the complex waveform of descending commands to muscle can be approximated by a rectangular "excitation pulse." This approach suggests that during the performance of single-joint movements, the motor control system uses two strategies to control the "excitation pulse" to motoneuron pools of agonist and antagonist muscles. Movements result from either the modulation of the duration of the excitation pulse and/or its intensity. The agonist muscle is the prime mover accelerating the limb in the desired direction whereas the antagonist muscle brakes the movement.

For movements performed without explicit or implicit constraints upon movement time, only the duration of the "excitation pulse" affecting the prime mover muscles is modified and the antagonist muscles are activated later for longer movements. This strategy was named "speed insensitive" and predicts coinciding EMG and kinematic traces at movement onset. For movements performed under the requirement of a specific movement time, pulse intensity is one of the parameters modulated. This second strategy was termed "speed sensitive," and it generates kinematic and EMG traces that diverge shortly after movement onset.

Corcos and colleagues conducted a series of studies to determine how the intensity and duration of the excitation pulse change over time in order to enhance motor performance. Subjects were trained to perform fast single joint movements towards a fixed target. With training these subjects increased levels of muscle activation, activated their antagonist muscles earlier in most cases and moved more quickly. These improved kinematics were attributed to...
an increase in the intensity of motoneuron pool activation in accordance with the rules of the speed-sensitive strategy.

Cole, Abbs and Turner reported that, unlike subjects without neurological impairment, individuals with Down syndrome could not modulate the rate of change of their grip force when asked to lift objects with different frictional surfaces. To compensate for this problem, individuals with Down syndrome achieved the appropriate force level by prolonging the duration of the grip force. Latash and Corcos observed that some subjects with Down syndrome could generate movements with kinematic and EMG traces according to the speed insensitive strategy, but their movements were at lower speeds when compared with control subjects.

Based on the findings reported in these two studies, the following question can be posed. Can individuals with Down syndrome modulate the intensity of motoneuron pool activation? If this is the case, can subjects under training increase the intensity of motoneuron pool activation beyond their unpracticed maximal levels and produce qualitatively similar changes in myoelectric and kinematic parameters to those reported for neurologically unimpaired subjects? To address this issue, individuals with Down syndrome were trained to perform fast single-joint movements over one distance. A transfer of learning paradigm was used in which the subjects were pre- and posttested on movements over different distances and from two initial positions. Training led to a significant improvement in the myoelectric and kinematic profiles. Like neurologically unimpaired subjects, subjects with Down syndrome could both modulate the duration and the intensity of the excitation pulse.
METHODS

Subjects

Four male and four female subjects with Down syndrome took part in the experiments. The chronological age and sex of each subject are presented in the Table 1 (Paper 1). The subjects and their parents gave informed consent according to protocols approved by The Human Investigation Committee of Rush Medical Center.

Experimental protocols

The subjects sat in a chair with their right forearm positioned on a low friction horizontal manipulandum (moment of inertia 0.086 Nm s² rad). The axis of rotation of the manipulandum was aligned with the elbow joint (90° elbow flexion was defined as zero degrees). In front of the subject, a monitor continually displayed a cursor showing the limb position as depicted in Figure 1. Both the target size and the distance were specified with two sets of narrow bars displayed on the computer monitor. The target size was 6° across all experiments.

Insert Figure 1 about here

The subjects were pre and posttested performing one set of isometric contractions and three sets of isotonic movements. During the isometric set, the subjects performed three maximum voluntary contractions in both flexion and extension under the instruction to push (or to pull) "as strongly as possible". The elbow joint position for these isometric tests was zero degrees. Between the isometric trials the interval was approximately 30s. During the first isotonic set of experiments, subjects moved over 18°, 36°, 54°, and 72° from an initial position of -35°. During the second isotonic set of experiments the initial position was -17° and the target distances were 18° and 36°. In these two sets of tests subjects were asked to move "as fast as possible". Finally, in the third set, subjects moved 36° from an initial position of -35° at a "comfortable speed". Six trials were recorded for each distance in each set of isotonic
movements. The interval between consecutive trials was eight seconds with one minute between each set of trials for both test and training sessions.

Between the pretest and the posttest subjects had ten training sessions of ten blocks of isotonic movements, which consisted of 11 trials each, at the 36° target distance from the -35° initial position. The total number of practice trials was 1100. During the first day subjects performed the pretest followed by the first training session. Then subjects had two more training sessions per day for four days on the 2nd, 8th, 9th and 15th days after the pretest. Finally, on the 16th day after the pretest, subjects performed the last training session which was followed by the posttest.

The use of any technical words in the instructions was avoided. The experimenter tried to use expressions familiar to each subject by first asking what she/he called different parts of the apparatus. Some examples of the instructions used are: "Pull the arm rest (manipulandum) towards me (flexion direction) as strongly as you can" for isometric flexion; or, "Move as fast as possible but do not overshoot the red line (target distance) too much" for isotonic movements. A strong and concomitant verbal reinforcement was given if the subject followed the instruction. For the set of isotonic movements, knowledge of results was based on the peak velocity of the movement performed by the subject. If the subject increased speed, the feedback took the form of encouragement: "Now you moved faster than before. This is fantastic!". But if the subject started to play during the trials, verbal disapproval was used. For example, "Do not do that!" or "I do not like this!".

At the beginning of each trial the subject was asked to relax her/his muscles and to move after hearing a computer-generated sound together with the experimenter’s verbal command "GO!". Neither reaction time nor accuracy was stressed in these experiments.

**EMG measurements**

ECG disposable pediatric electrodes (self-adhesive) were placed over the bellies of two agonists (biceps brachii and brachioradialis) and two antagonists (lateral and long head of the
triceps). These EMGs were amplified (1600X) and band-pass filtered (60-500Hz). Each signal was digitized at the rate of 1000/s with 12 bit resolution. After that it was full-wave rectified and filtered with a 10 ms moving average window. Then, this signal was displayed at high gain on a computer monitor where the onsets of the agonist and the antagonist muscle were visually estimated for each trial. After that, the data were processed using a filter with a 25 ms moving average window and analyzed. In this study only the data for the biceps brachii and lateral head of triceps are presented. The data for the brachioradialis were qualitatively similar to the biceps and those of the long head of triceps were similar to the lateral head of triceps.

The onset of the agonist muscle was defined as the first time the EMG rose above the baseline. All the trials were aligned for averaging according to this time. The value for the onset was verified by checking that the acceleration signal also changed within the next 40 ms. Two onsets for the antagonist were identified. The first component was defined as the first detectable rise above baseline and normally occurred only a few milliseconds following the onset of the agonist. The onset of the second component of the antagonist EMG was defined as the first sustained rise above baseline. These two components correspond to the early and late components (see Gottlieb and colleagues\(^5\)). The following procedure was used to help identify the onset of the late antagonist component in each individual trial. First the average of the antagonist EMG for a set of trials was plotted from 100 ms before the antagonist onset to 400 ms after it. Then, the beginning of the late component of the antagonist onset was determined for this averaged record. This value was used to help to visually estimate the late component of the antagonist burst in each individual trial. The trials in which the subject over or undershot the target by more than 10°, or in which the agonist or antagonist onset was ambiguous to identify were rejected from further analysis. Also, the first trial of each condition was always rejected for purposes of analysis. The total number of trials analyzed for each condition was about four for the pretest and five for the posttest.
For further quantification, the EMGs were normalized with respect to the EMG of the maximal voluntary isometric contraction. First, the agonist EMGs of the isotonic contractions were integrated over two time intervals, the first 30 ms after the agonist EMG onset ($Q_{30}$) and from the onset of the agonist EMG to the first zero crossing of the acceleration ($Q_{acc}$). The antagonist EMGs were integrated over the time interval from the agonist onset to the projected end of deceleration ($Q_{dec}$) (see movement time ahead). Integration over a fixed interval ($Q_{30}$) was chosen to determine whether the slopes of the initial component of the EMG records were similar across experimental conditions. This method is analyzed in the Appendix 1 of Gottlieb and colleagues. $Q_{acc}$ corresponds approximately to the first agonist burst.

Second, $Q_{30}$, $Q_{acc}$ and $Q_{dec}$ were normalized by dividing them by the value of the EMG from the maximum voluntary contraction (MVC). The EMG of the MVC was calculated from the trial in which the torque value was the largest by integrating it from the interval between 500 ms and 1000 ms after the agonist onset. Both the isometric and isotonic EMGs used in the normalization procedure came from the same session. The normalized quantities $Q_{30}$, $Q_{acc}$ and $Q_{dec}$ will be referred to in this paper as $Q^*_{30}$, $Q^*_{acc}$ and $Q^*_{dec}$. Because it was not possible to have a consistent measurement of torque from two subjects (S3 and S7) during the pretest their data could not be normalized. For this reason, their EMGs were discarded from quantitative analysis.

The antagonist latency was defined as the interval from the agonist onset to the late component of the antagonist burst. Three out of eight subjects (S3, S7 and S8) presented a pattern of muscle activation in which an early and a late component could not be identified.

**Mechanical measurements**

The elbow angle was measured by a capacitative transducer mounted on the axis of rotation of the manipulandum. Elbow acceleration was measured by a piezoresistive accelerometer which was mounted 46.7 cm from the center of rotation at the distal end of the manipulandum. The accelerometer axis of maximal sensitivity was oriented to measure
tangential acceleration. Acceleration and angle were digitized with 12 bit resolution at a rate of 1000/s. Velocity was derived by integration of the acceleration signal after low pass filtering at 25 Hz. The torque was measured by a strain-gauge transducer and filtered at 25 Hz.

Movement time was defined as the interval from the first acceleration deflection, which was visually determined, to the projected end of deceleration. The projected end of deceleration was determined by linearly extrapolating the deceleration to 0 from the point at which it had fallen to 50% of its negative peak (see Gottlieb and colleagues 5).

Statistical analysis

Repeated measures analyses of variance were used to analyze changes in peak velocity, peak acceleration and deceleration, acceleration and deceleration time, Q°30, Q°acc, Q°dec and the antagonist latency. Paired, two-tailed t-tests were also used in the analysis of peak velocity.
RESULTS

Voluntary changes in movement speed

The data in Figure 2 depict angle, velocity, acceleration and both the agonist (biceps) and antagonist (lateral head of the triceps) EMGs for the elbow flexion movements of one subject performed over 36° "as fast as possible" (solid line) and "at a comfortable speed" (broken line). For the pretest, the EMG and kinematic traces rose more steeply under the instruction to move "as fast as possible" as opposed to "at a comfortable speed" in four out of eight subjects. For the post-test, the slopes of the EMG and kinematic traces rose more steeply under the instruction to move "as fast as possible" in all eight subjects.

Insert Figure 2 about here

The effect of practice on movement performance

Practice had a significant effect on performance over all the movement distances as can be seen in Figure 3. The subject (S.7) was asked to move "as fast as possible" over four different distances.

Insert Figure 3 about here

This subject had the worst kinematic performance of any subject in both pre and posttest. The slopes of the kinematic and EMG profiles rose more sharply during the posttest than during the pretest. In another words, there was an increase in these slopes with training both at the practiced distance (36°) and the other distances. After training, peak velocity, peak acceleration and peak deceleration, and the EMG quantities were higher during the posttest, with a concomitant decrease in movement time. These general EMG and kinematic patterns of improvement were observed for all eight subjects.

Peak Velocity

Figure 4A depicts peak velocity averaged across subjects for movements over four different distances. A two-way repeated measures analysis of variance with practice (pre-practice versus post-practice) and movement distance (18°, 36°, 54° and 72°) showed a
The data are presented in Figure 4B. The difference between the pretest and posttest means is significantly different from zero for all four distances but the difference is greater for the 36°, 54° and 72° movements than for the 18° movements. The relative change from the pretest to the posttest was also calculated (i.e. the difference between pretest and posttest was divided by the pretest value for all distances). These data are presented in Figure 4C and, as one can observe, this ratio remained almost constant demonstrating that, after training, the subjects on average doubled their peak velocity for each movement distance.

**Insert Figure 4 about here**

**Movement time**

The data in Figure 5 depict the averaged movement time for all eight Down syndrome subjects.

**Insert Figure 5 about here**

For purposes of analysis, movement time was divided into both acceleration time and deceleration time. Figure 6 depicts the averaged data for both acceleration time and deceleration time for the pretest and posttest. A three-way repeated measures analysis of variance was performed to assess the effect of practice (pre practice versus post practice), acceleration profile symmetry (acceleration time versus deceleration time) and distance. There was a decrease in movement time due to practice \( F(1,7) = 19.96, p < .0029 \), acceleration and deceleration time were symmetrical \( F(1,7) = .152, p < .7078 \) and there was a significant main effect due to distance \( F(3,21) = 18.91, p < .0001 \). Finally, none of the interactions was significant (practice versus acceleration symmetry \( F(1,7) = .51, p < .4968 \); practice versus
distance \( F(3,21) = 1.62, p < .2156 \); acceleration symmetry versus distance \( F(3,21) = 2.36, p < .1010 \); and practice versus acceleration symmetry versus distance \( F(3,21) = .49, p < .6927 \).

**Insert Figure 6 about here**

*Peak acceleration and peak deceleration*

The data in Figure 7 depict the average for both peak acceleration and peak deceleration at pretest and at posttest, for all eight Down syndrome subjects, across four distances. A three-way repeated measure analysis of variance was performed to assess the effect of practice on both peak acceleration and peak deceleration. The subjects performed their movements at higher peak acceleration and deceleration as a result of practice \( F(1,7) = 27.53, p < .0012 \). The acceleration and deceleration peaks were symmetrical \( F(1,7) = .20, p < .6661 \). There was a main effect due to distance \( F(3,21) = 12.99, p < .0001 \). There was also significant interaction between acceleration symmetry (peak acceleration versus peak deceleration) and distance \( F(3,21) = 4.37, p < .0153 \) as well as between practice (pretest versus posttest) versus distance \( F(3,21) = 7.63, p < .0012 \). There was no significant interaction between acceleration symmetry versus practice \( F(1,7) = 2.27, p < .1757 \) and acceleration symmetry versus practice versus distance \( F(3,21) = .90, p < .4520 \).

**Insert Figure 7 about here**

*Agonist and antagonist EMGs*

The data in Figure 8A are the averaged, normalized EMG quantities of six Down syndrome subjects for the agonist \( Q_{\text{acc}}^* \) and antagonist \( Q_{\text{dec}}^* \) muscles. After training, the subjects increased significantly the EMG quantities of both agonist and antagonist muscles. A two way repeated measure analysis of variance was performed for both agonist \( Q_{\text{acc}}^* \) and antagonist \( Q_{\text{dec}}^* \) activity. For \( Q_{\text{acc}}^* \) there was a significant main effect due to practice \( F(1,5) = 13.22, p < .0150 \). The main effect was also significant for distance \( F(3, 15) = 9.34, p < \)
and there was no significant interaction between practice (pretest versus posttest) and distance ($F(3,15) = 2.37, p < .1388$).

For antagonist activity ($Q^*$) there was a significant main effect due to practice ($F(1,7) = 7.94, p < .0372$), but no significant main effect due to distance ($F(3,15) = 1.17, p < .3532$). The interaction between practice and distance was also not significant ($F(3,15) = .93, p < .4505$).

**The agonist EMG slope**

The data in Figure 8B depict the average of the normalized EMG quantity $Q^*_{30}$ for six Down syndrome subjects at the pretest and at the posttest. The quantity $Q^*_{30}$ describes the slope of the initial component of the agonist EMG. A two-way repeated measures analysis of variance revealed that with practice there was a significant increase in the slope of the agonist EMG ($F(1,5) = 16.62, p < .0096$). The main effect due to distance was not significant ($F(3,15) = .78, p < .4900$) and the interaction between practice (pre-practice versus post-practice) and distance was also not significant ($F(3,15) = .57, p < .6422$).

**Insert Figure 8 about here**

**Antagonist latency**

Three out of eight subjects displayed patterns of antagonist activity in which early and late components could not be separately identified. The latency of the antagonist burst for those three subjects was not calculated. For the other five subjects, a two-way repeated measures analysis of variance revealed a significant main effect due to both practice ($F(1,4) = 37.62, p < .0036$) and distance ($F(3,12) = 20.05, p < .0001$), and no significant interaction ($F(3,12) = 3.112, p < .0667$). Because there is a strong relationship between movement time and antagonist latency in neurologically unimpaired subjects, we have plotted antagonist latency versus movement time at the pretest (broken line) and at the posttest (solid line) for the five Down syndrome subjects in Figure 9. With practice there was a decrease in both the antagonist latency and in movement time at all four distances.
The Effect of different initial positions on movement performance

To quantify the effect of different initial positions on the performance of practiced movements over different distances, a three-way repeated measures analysis of variance was performed on peak velocity. The three factors were different initial position (-17° versus -35°), practice (pre-practice versus post-practice) and movement distance (18° and 36°). These data are depicted in Figure 10A and represent the averages of peak velocity for the eight Down syndrome subjects.

There was no main effect due to initial position ($F_{(1,7)} = .631, p < .4530$). But there was a significant main effect due to both practice ($F_{(1,7)} = 50.414, p < .0002$) and distance ($F_{(1,7)} = 30.525, p < .0009$), and there was a significant interaction between practice and movement distance ($F_{(1,7)} = 22.68, p < .0021$). The interaction between initial position and practice was not significant ($F_{(1,7)} = 1.351, p < .2832$), but the interaction between initial position and distance was ($F_{(1,7)} = 7.087, p < .0324$). The interaction between initial position and distance is presented in Figure 10B. Note that, on average, the subjects performed better over 18° from -17° initial position than over 18° from -35°. On the other hand, over 36° the peak velocity was almost the same for both initial positions. Finally, there was no significant interaction between practice, initial position and distance ($F_{(1, 7)} = 1.579, p < .2493$).

To understand the interaction between initial position and distance a two-way repeated measures analysis of variance was performed for the pretest and for the posttest separately. At the pretest, the interaction between initial position and distance was not significant ($F_{(1, 7)} = .08, p < .7806$). However, at the posttest this interaction between initial position and distance was close to being significant ($F_{(1,7)} = 3.917, p < .0883$).
DISCUSSION

*Speed-sensitive and speed-insensitive strategies*

Individuals with Down syndrome can use the speed-sensitive strategy when asked to move at two different speeds over the same target distance as can be seen in Figure 2. The kinematic and EMG slopes that accompany their movements diverged soon after movement onset for both the pretest (four out of eight individuals) and the posttest (all eight individuals). This ability to modulate the rate of acceleration and rate of torque production even before training is contrary to what was initially hypothesized from the study of Cole, Abbs and Turner⁹ who showed that six out of eight Down syndrome subjects did not increase their rate of grip force to achieve the greater force required to lift more slippery objects. There are two possible reasons why some individuals modulate rate and others do not. The first reason is simply that some individuals with Down syndrome have this ability and others do not. This line of reasoning argues that some individuals with Down syndrome have a deficit in their capacity for activating motoneuron pools with different levels of intensity. The second reason is that individuals with Down syndrome may display this ability under certain experimental conditions but not under other conditions. For example, it may be the case that there are certain subtle deficits in sensory functioning¹³ that limit the types of motor control strategies that can be used by certain individuals with Down syndrome to perform tasks requiring perceptual recognition of object characteristics such as the task used by Cole, Abbs and Turner⁹. The view that individuals with Down syndrome perform the same movement task in a variety of ways ties in with findings from our laboratory¹² in which we have demonstrated that individuals with Down syndrome respond differently to a given motor task. The same individuals as in the present study were asked to react or not to react to different limb perturbations. Some subjects coactivated their muscles in response to the perturbation while others displayed a reciprocal pattern. Finally, it may be the case that all Down syndrome individuals can perform the task employed by Cole, Abbs and Turner⁹ in the same way as
neurologically unimpaired individuals if they are provided with knowledge of results that induces them to modulate the rate of force generation. We will discuss the effects of KR and practice in the section on "The mechanisms underlying motor performance enhancement".

The capability of some individuals with Down syndrome to perform movements according to the "speed insensitive strategy" has been previously reported. The additional finding in this paper is that individuals with Down syndrome activate their antagonist muscles later for longer movements. This can be seen for the pretest in Figure 9 and is very similar to previously reported findings for neurologically unimpaired individuals.

Training enhances motor performance in individuals with Down syndrome

All eight individuals with Down syndrome demonstrated substantial changes in performance as a result of practicing the simple elbow flexion task under reproducible conditions with knowledge of results based on movement speed. This was reflected by a significant improvement in all kinematic parameters associated with the movements. After practice, on average subjects doubled peak movement speed (Figure 4A) and decreased movement time (Figure 5). The averaged peak velocity of the movements after practice was 185.9°/s, 320°/s, 400°/s, and 430°/s respectively for 18°, 36°, 54°, and 72° distance. These peak velocities are approximately 30% below the ones previously reported for neurologically unimpaired individuals who were trained using a similar experimental protocol. If one considers the fact that those subjects were all male university graduate students, each of whom had had 300 more practice trials than the subjects with Down syndrome in this study, it can be assumed that with appropriate training some individuals with Down syndrome could perform single-joint movements at performance levels very similar to the overall population.

In our experiments, practice resulted in proportional changes in peak velocity for all movement distances (Figure 3C). This means that $\frac{V_{\text{post}}}{V_{\text{pre}}} = \text{const}$. Peak velocity can be defined
by the duration and the level of acceleration. If we assume that the acceleration profile can be modeled as an inverted parabolic function:

\[ A(t) = -at^2 + bt \quad \text{Eq. (1)} \]

where \( a \) and \( b \) are positive constants, \( A \) is acceleration, \( t \) is time, \( A_p \) is peak acceleration and \( t_a \) acceleration time, then, after simple calculations based on Eq. (1):

\[ \frac{t_{a2}A_{p2}}{t_{a1}A_{p1}} = \text{const} \quad \text{Eq. (2)} \]

where subscripts 1 and 2 refer to pretest and posttest correspondingly. In other words, the proportional increase in velocity means that a relative increase in peak acceleration was accompanied by a proportional decrease in acceleration time for all the distances (cf Figures 6 and 7). Note that \( t_aA_p \) is proportional to accelerating impulse. This is of interest because Gottlieb et al.\textsuperscript{14} have suggested that impulse might be a particularly appropriate variable for describing the rules underlying the control of a variety of single-joint movements. Our findings suggest that relative changes in impulse with practice may be an invariant preserved over different movement distances. Searching for parameters that display invariance is one way of solving the "redundancy problem" in voluntary motor control\textsuperscript{1}.

Increases in peak velocity are highly correlated with decreases in movement time. Decreases in movement time with practice have been reported in other studies of individuals with developmental disabilities \textsuperscript{15, 16} and specifically for those with Down syndrome \textsuperscript{17}. However, the performance improvement is much higher in the present study (50% and more, Figure 5) than that of Kerr and Blais \textsuperscript{17} in which improvements in movement time ranged from 8% to 25% depending on the degree of practice and the amount of overshoot allowed in performing the movements. Unlike control subjects who decreased their movement time as a function of practice by proportionately shortening the deceleration time \textsuperscript{8}, subjects with Down syndrome did so by decreasing both acceleration and deceleration time in the same proportion. Approximately equal acceleration times and deceleration times were used by subjects with
Down syndrome for the pretest and the posttest (Figure 6) and are used by naive subjects before training. The symmetrical way in which Down syndrome subjects performed their movements, even after training, was the only qualitative difference observed between Down syndrome subjects and control subjects. It remains to be determined whether this symmetry can be changed with specific training and whether changes in symmetry can enhance motor performance of individuals with Down syndrome beyond the level reported in this study.

Schmidt has pointed out that what is learned for one task should be well generalized or transferred to variations on this task. In this sense, individuals with Down syndrome can improve not just the particular movement distance that they practiced but they can transfer enhancements in performance from one context to another. This ability to transfer performance was observed in two task situations. First, the improvement in motor performance of all subjects with Down syndrome observed at the trained distance (36°), as measured by the kinematic and EMG parameters, was well transferred to the non-trained distances (18°, 54° and 72°). After training, for example, the subjects were able to double the initial peak velocity of their movements across all different distances (Figure 4C). Second, when asked to move from a different initial position (-17°) subjects with Down syndrome performed as well as they had done at the trained initial position (Figure 10). This means that they could transfer what they had learned at one initial position to another initial position.

The mechanisms underlying motor performance enhancement

Motor performance enhancement with training is well reported in the literature for normal subjects and more recently for subjects with Down syndrome. The results of the present study suggest that the effect of extensive training of fast single-joint movements is to enable subjects to increase the level of the intensity of motoneuron pool excitation beyond that which was initially maximum. The increase in intensity of motoneuron pool excitation with practice was observed when the slopes of the EMG traces between pretest and the posttest were compared (Figure 8B). The values of $Q^*_{30}$ approximately doubled.
Many studies have reported a scaling of agonist activity with movement distance for normal subjects 20, 21, 22, 5. For the antagonist, however, there is often less or the same antagonist EMG for longer movements than for shorter movements in neurologically unimpaired individuals 23, 5, 14. The data in the present study are consistent with these findings. Movement speed can be increased not only by increasing EMG quantity but also by decreasing antagonist latency (Figure 9) that can have two potentially different consequences. On the one hand, a decrease in antagonist latency can lead to an overlap in the agonist and antagonist activity which is not the most energy efficient mechanism for generating a movement. Minimizing energy expenditure or any other variable, however, is clearly not part of the task demands of performing the most rapid movements possible. On the other hand, muscle co-activation can help to increase joint stability which clearly is important for the performance of rapid, accurate movements. Also, decreased latency of antagonist activation allows the motor control system to start to brake the movement early, thus reducing the time necessary to perform the movement.

Until now, we have discussed the findings using the framework of the dual-strategy hypothesis and the notion of an excitation pulse. This approach is based on the control of the net input to the a-MNs (excitation pulse) and either implies a lack of reflex-mediated changes in a-MN activity or relies upon the predictive abilities of the hypothetical central controller. This central controller is assumed to predict the reflex contribution in reproducible conditions of movement execution and modulate the descending input so that the net input can be modeled as an excitation pulse 24. An alternative approach considers the descending control of movements as based upon the regulation of the reflex loops from the peripheral receptors. In particular, the equilibrium point hypothesis 3, 25 considers voluntary motor commands as time functions of the thresholds of the tonic stretch reflex for the participating muscles. The equilibrium point hypothesis explicitly generates predictions for experimental manipulations in which individuals have to reproduce movement distance as opposed to final position 26.
The equilibrium point hypothesis suggests that practicing a standard movement "as fast as possible" under standardized conditions may be associated with learning how to shift a control variable (associated with joint angle in isotonic conditions) at a maximal rate to a certain final value. Our subjects practiced a 36° movement from -35° to +1°. That is, they learned how to quickly shift the hypothetical control variable so that the limb would stop at +1°. The EP-hypothesis suggests that moving from a different initial position to the same final position is "easier" than moving the same movement distance but to a different final position since two variables are required to reproduce movement distance (rate of change and final value of the hypothetical control variable).

Our experiments with moving from a different initial position (-17°) over 18° (i.e. to the same final position of +1° as for the practiced movement) and over 36° to a different final position than that practiced were designed to test this prediction. We observed an interaction in the post test between initial position and distance that approached statistical significance with the subjects demonstrating higher peak velocities during movements from -17° to +1° than during movements from -35° to -17°. This finding is consistent with the predictions from the EP-hypothesis. Therefore, one may conclude that two variables have been learned during practice, a higher rate of change of the controlled variable associated with generally higher movement velocities and final equilibrium position of the joint.

Similarities and differences with neurologically unimpaired individuals

The subjects in our study were asked to perform four different motor control tasks. The first task was to move at different subject selected movement speeds. The second task was to move four different distances. The third task was to move two distances from two initial starting positions. The fourth task was to perform 1100 movements at one movement distance. The first three tasks were performed before and after the practiced movements. As expected, the subjects all moved slowly before practice. While it is true that the dramatic improvement in kinematic and EMG traces reported here for all Down syndrome subjects was
partly possible because they performed poorly in the pretest, their final levels of performance and myoelectric patterns were comparable to those of neurologically unimpaired individuals. This suggests that the motor control system of individuals with Down syndrome is functionally intact. This is because individuals with Down syndrome are not only capable of successfully performing a wide range of movement tasks but also generalizing performance improvement over unpracticed movement distances and from unpracticed initial positions.

However, many studies do show levels of performance below those of neurologically unimpaired individuals. One possible reason for why some studies have shown deficits in the performance of individuals with Down syndrome is that these studies have placed a higher emphasis on cognition and, as such, the task is more demanding and the requirements of the task are less clear to the subject. This can be seen in a comparison of two studies by Kerr and Blais 28,17 in which subjects did not improve movement time in the first study but did in the second. A second possibility is that insufficient attention is paid to the degree to which the subjects fully comprehend the experimental instructions. A third possibility relates to the observation that some subjects perform a task in the same way as normals and others do not.

Finally, the results presented have two implications for the study of Down syndrome. First, they strongly support the idea that there is considerable room for improvement in the motor performance of Down syndrome individuals with training 17. Second, they corroborate the idea that any kind of comparison between special and normal populations should be avoided unless the special group has had the opportunity to perform at an optimal level 29, 30.
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REFERENCES


Figure Captions

Fig. 1. The components of the experimental apparatus illustrating the position of the subject with respect to the elbow device and computer monitor. The display shows the initial starting position of the limb and the target.

Fig. 2. Angle, velocity (vel), acceleration (accel), biceps and lateral head of triceps (tri lat) for the pretest and the posttest for subject S5. The lateral head of triceps 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 the averages of four and five trials respectively for the pretest and for the posttest aligned at the onset of the agonist EMG (200 ms).

Fig. 3. Movements over four distances (18°, 36°, 54°, and 72°) as fast as possible during the pretest and posttest for subject S7. Figure captions are the same as in Fig. 1.

Fig. 4. A. Averaged peak velocity (n = 8) ± standard error is plotted versus distance during the pretest (broken line) and the posttest (solid line). B. Averaged increase and confidence intervals are plotted for peak velocity from the pretest to the posttest for all four distances. C. Averaged increase in peak velocity divided by pretest peak velocity is plotted versus distance. The vertical bars represent the standard error.

Fig. 5. Averaged (n = 8) movement time ± standard error is plotted versus distance during the pretest (broken line) and the posttest (solid line).

Fig. 6. Averaged (n=8) acceleration time (open circle pretest, closed circle posttest) and deceleration time (open rectangle pretest, closed rectangle posttest) ± standard error is plotted versus distance for the pretest (broken line) and the posttest (solid line).

Fig. 7. Averaged (n = 8) peak acceleration (open circle pretest, closed circle posttest) and peak deceleration (open rectangle pretest, closed rectangle posttest) ± standard error is plotted versus distance for the pretest (broken line) and the posttest (solid line).
Fig. 8. A. Averaged (n=6) agonist EMG $Q^{*}_{\text{acc}}$ (open circle pretest, closed circle posttest) and antagonist EMG $Q^{*}_{\text{dec}}$ (open rectangle pretest, closed rectangle posttest) ± standard error is plotted versus distance. B. Averaged EMG $Q^{*}_{30}$ (n=6) is plotted versus distance. The data are for the pretest (broken line) and for the posttest (solid line).

Fig. 9. Averaged (n=5) antagonist latency is plotted versus movement time for the pretest (broken line) and posttest (solid line) for four distances. The data are from five individuals with Down syndrome for whom an early and late EMG component could be identified.

Fig. 10. A. Averaged peak velocity (n=8) ± standard error versus distance during the pretest (broken line) and the posttest (solid line). The subjects were asked to move "as fast as possible" from two initial positions (-17° - circle and -35° - rectangle) over two different distances. B. The data in this figure depict the interaction between initial position (-17° and -35°) and distance (18° and 36°).
Fig. 1
Fig. 2
Fig. 3
Fig. 4
Fig. 5
Fig. 6
Fig. 7
Fig. 9
GENERAL DISCUSSION AND CONCLUSIONS

*Individuals with Down syndrome, great motor learners and fantastic performers*

We showed that with appropriate training, in very standardized conditions, individuals with Down syndrome are able to improve significantly their motor performance, and to transfer what they learned at one distance to different distances and different initial positions. The enhancement in motor performance, with training, showed that individuals with Down syndrome can properly activate their muscles and produce similar kinematic and myoelectric changes to those observed in neurologically normal individuals (Corcos et al., 1993; Jaric et al., 1993). With training they were able to increase the intensity in which they activate their motoneuron pools, generating more force, and moving faster. As predicted by the speed sensitive strategy, this increase in the level of activation of the motoneuron pool was followed by an early activation of the antagonist muscle (Corcos et al., 1989). These findings do not support the idea that individuals with Down syndrome necessarily have decreased motoneuron pool excitability (Davis & Sinning, 1987) or that they cannot generate appropriate levels of force (Frith & Frith, 1974; Henderson et al., 1981a; Latash & Corcos, 1991; Davis & Sinning, 1987; Cole et al., 1988). On the contrary, our findings support the optimistic view about the motor control of individuals with Down syndrome, which shows that they can improve their motor performance with appropriate training (Edwards & Yuen, 1990; Kanode & Payne, 1989; Kerr & Blais, 1987; Kerr & Blais, 1988).

In addition to being able to move very fast, individuals with Down syndrome improved their motor performance at faster rate than neurologically normal individuals (Corcos et al., 1993; Jaric et al., 1993). This finding is in contradiction with studies showing that individuals with Down syndrome improve their motor performance at a slow pace (Kerr & Blais, 1988). With training the individuals with Down syndrome doubled the speed of their movement, and decreased in a half their movement time. The decrease in movement time with training has been
reported for other individuals with handicapped other then Down syndrome (Hoover, Wade, & Newell, 1981; Wade, Hoover, & Newell, 1984) and for those with individuals with Down syndrome (Kerr & Blais, 1988). However, the decrease in movement time of individuals with Down syndrome was higher in our experiment than in the study of (Kerr & Blais, 1988) in which the decrease in movement time ranged from 8% to 25%, depending on the degree of practice and the amount of overshooting allowed during the training.

The first explanation for the difference in the rate of gain in improvement related to the nature of the task. In our experiment, the task (single-joint elbow flexion) was very simple, and did not require a lot of cognition, whereas the task in the study of Kerr and Blais (1988) was very complex (discrete pursuit tracking), and placed great emphasis on cognition. With different cognitive requirements, it is natural that individuals with a mental handicap like individuals with Down syndrome, will perform better on the simplest task. The second explanation for the different rates of improvement relates to different levels of motivation offered to the subjects in both experiments. In our experiment, knowledge of results, combined with strong reinforcement to move faster, may have played an important role in the enhancement of motor performance. In the experiment of Kerr and Blais (1988) the reinforcement was not very strong ("good"). The literature in Special Education is full of reports that mentally handicapped individuals usually receive sub-optimal stimulation, have a history of failure, and are poorly reinforced to overcome their own limitations. We simply tried to make the individuals believe that they could exceed their own limits and move faster. We encouraged them to compete with themselves. When we said, "Fantastic, you are doing a great job, but you can do better!" They often answered in a loud voice with a big smile on their face, "Yes, I'm gonna do it." The friendly and encouraging environment we tried to create is hard to describe in terms of experimental methods, even they produce a great impact on the results.
Comparative studies between the performance of individuals with Down syndrome and control subjects usually assumes that they come to the test with the same prior opportunities. This is not true. We learned two important lessons from our experiment. First, we should not deprive individuals with Down syndrome from strong motivation. Society usually offers motivation to the control groups since they achieve success in much of what they do. Second, any kind of comparison between special and normal populations should be avoided, unless the special group has had the opportunity to perform at an optimal level and with sufficient practice (Worringham, 1989; Newell, 1989).

Besides performing at a high level, and improving very fast, individuals with Down syndrome also moved with a remarkable accuracy. Kerr and Blais (1987) reported that individuals with Down syndrome put more emphasis on accuracy rather than speed, in the same way Latash (1992) advocated that individuals with Down syndrome prefer safety over efficacy. From our experiment, we would rather argue that individuals with Down syndrome can move very accurately, and with appropriate training they can keep this accuracy and speed up their movements. They did not trade-off speed for accuracy or safety for efficacy. Similar to any naive performer, individuals with Down syndrome just learned how to improve their motor performance when they were allowed to do so. Because of this ability to improve their motor performance very quickly and at a high level, without losing initial accuracy, individuals with Down syndrome might be good candidates for several jobs that require these abilities. Since they were able to keep this high level of motor performance over time, individuals with Down syndrome might qualify for assembly line jobs in that require accuracy, speed, and repetition.

The motor control system of individuals with Down syndrome is functionally intact, their education may be handicapped.

We advocated that, because of the high level of performance with training, the motor control system of individuals with Down syndrome is functionally intact. However, we still
need to address some residual problems in their motor control system, reported in several studies. For example, they did very poorly when a time constraint was imposed on different tasks (Henderson et al., 1981a). When encouraged to go fast they merely pressed harder on a tap-pad or tracing surface (Frith & Frith, 1974; Henderson et al., 1981a). They displayed a smaller magnitude of maximum torque and EMG than individuals mentally retarded without Down syndrome, and control subjects (Davis & Sinning, 1987). They could not modulate the rate of change of their grip force. When asked to lift objects with different frictional surfaces, instead of modulating the rate of grip force they prolonged the duration of the grip force (Cole et al., 1988).

First, it was clear from our experiment that individuals with Down syndrome can generate force at a higher levels with training. Since the maximum inertial torque is proportional to peak acceleration we can say that during training individuals with Down syndrome increased their level of force by 267% (Figure 11). Second, we have no reason to believe that mentally retarded can generate high levels of force under isotonic conditions, but not under isometric conditions as reported in the training study of Davis and Sinning (1987). Third, we showed that individuals with Down syndrome can properly modulate the rate of acceleration and the rate of torque even before training, which is in contradiction with the finding that they could not modulate the rate of change of their grip force (Cole et al., 1988).

In our opinion one strong candidate to explain these residual problems in the motor control of individuals with Down syndrome is a lack of appropriate training. By appropriate training we mean what we usually offer to naive performers. We can teach them to overcome their own limits with motivation and increases in self-stem by using appropriate reinforcement. The literature in Child Development is teaching us that the expectation a teacher creates about the capability of a child to learn will largely influence his or her academic achievement. Motor behavior is no different from any other academic behavior. To learn a motor skill the child has to be exposed to and to practice it. This seems to be obvious and
trivial, but unfortunately it is not always the case. The literature in Language Development, for example, has shown that mothers adapt their speech to the level of understanding of their babies. We also observed that mothers of individuals with Down syndrome adapt their communication to the level that they thought it is understandable to their children (Prorok & Almeida, 1985). Basically, we observed two kinds of mothers. The first type wants to prove that her child can learn, and her wish to help the child was so great that she ended up by providing over stimulation. Unlike the first mother, the second mother believed that her child was mentally retarded and because of that could not learn. This mother was passive and usually did not make any effort to communicate with her child. After instruction, the first mother gave more time to her child to answer questions, and the second mother become more talkative to her child. After this training, we observed improvement in the verbal behavior of both children with Down syndrome. Similar to verbal behavior, we believe that delays and handicaps in the motor behavior of individuals with Down syndrome could be largely associated with a deficient motor training or motor stimulation. That is, we may underestimate the ability of individuals with Down syndrome to learn a motor task, and as a consequence, we may end up by depriving them of adequate motor training.

**Practical and theoretical implications**

The implication that training could eliminate some residual problems in the motor control mechanisms of individuals with Down syndrome is still waiting to be proved. We are not ruling out other possibilities, such as organic or psychological problems, as the cause of some of the residual problems in the motor control mechanisms of individuals with Down syndrome reported in the literature. Our sample was not randomly selected, and there is a possibility that the individuals with Down syndrome in other studies were considerably more handicapped in some aspects of their development (i.e., mentally) than the subjects in our experiment.
Nevertheless, two important message emerges from our experiment. First, we should believe in the ability of individuals with Down syndrome to improve their motor performance, even if after a session of practice (110 trials) they did not increase their performance. Four out of eight subjects did not improve their movement velocity within the first practice session (see Figure 5 in Paper 1), but they moved faster between practice sessions (see Figure 4B in Paper 1). Second, we should avoid creating any kind of expectation about the capability of individuals with Down syndrome to learn how to perform a motor task, unless we give them the same opportunity to practice and to learn that we usually gave to a "naive learner." The expectation could create stereotypes and produce biases in the way we teach motor task.

One example of a stereotype is the belief that an organic dysfunction (i.e., low cerebellar weight) could be the cause of a behavioral deficit (i.e., poor balance). The problem with this point of view is that the correlation between organic dysfunction and behavioral deficit cannot be taken for granted as causal. The basic idea behind these studies is that an intact neuromuscular mechanism is a necessary and sufficient condition for the movement control. However (Ulrich et al., 1992) showed that 11-months-old babies with Down syndrome responded to the treadmill stimulus by producing alternating steps. Like non-handicapped young children, the infants with Down syndrome displayed the intact neural substrate necessary for upright locomotion before they were able to walk independently. This study is showing that an intact neuromuscular mechanism is not sufficient condition for the movement control.

More important, some of the associations between organic dysfunction and behavioral deficit do not hold when we give subjects the opportunity to practice. Let us make this point clearer by discussing the overall idea that low cerebellar weight of individuals with Down syndrome may underline the residual deficit in their motor mechanism. An altered movement acceleration profile has been associated with an abnormality in cerebellar dysfunction (Hallett, Berardelli, Matherson, Rothwell, & Marsden, 1991). The authors reported that when patients
with cerebellar deficits performed elbow flexion movements, "as fast as possible," they prolonged the acceleration time and the duration of agonist activity. The natural consequence of this prolonged acceleration is hypermetria.

In our experiment, the movements of individuals with Down syndrome did not present any of these possible signs of cerebellar dysfunction. First, individuals with Down syndrome did not prolong the acceleration time. Their movements were characterized by symmetry between acceleration and deceleration time. Second, the individuals with Down syndrome did not seem to increase the duration of their agonist (see Figure 2 at Paper 1). Unlike patients with cerebellar dysfunction (Hallett et al., 1991), individuals with Down syndrome were able to generate high levels of inertial torque, as measured by the increase in peak acceleration. Also, they increased the intensity with which they activated their motoneuron pools, producing considerable agonist activity. Third, given our experimental condition, the degree of overshoot of the movements of individuals with Down syndrome was in the normal range, and did not present any characteristics of hypermetria. The only exception could be subject S4, but this subject did not prolong his acceleration time. This subject was the only one who did not cooperate completely with the experiment. In many practice trials he did not follow the instructions, and ended up by having the largest number of trials rejected (see Figure 26 and Figure 27 in Paper 1).

Another major characteristic of patients with cerebellar lesions is a normal short latency response, followed by a delay of long-latency reflexes (Nashner et al., 1983). Because of that, (Shumway-Cook & Woollacott, 1985) suggested that the poor balance of individuals with Down syndrome could be due to their low weight of the cerebellum. However, all eight individuals with Down syndrome that we tested showed EMG reactions in response to changes in instructions typical of preprogrammed reactions (Latash et al., 1992). That is, their long-latency reflexes were at normal onset.
Since we did not have any control about the real characteristics of the cerebellum of the individuals with Down syndrome we tested, one could argue that their cerebellum may be intact. Nevertheless, if the finding that the low cerebellar weight of individuals with Down syndrome could be generalized for the population with Down syndrome, it is amazing that all eight individuals with Down syndrome did not present signs of cerebellar dysfunction reported by (Hallett et al., 1991) and (Nashner et al., 1983). One explanation is that the overall loss in cerebellar weight of individuals with Down syndrome was not enough to compromise its role as a clock that controls the timing of movements (Eccles, 1977; Gilman et al., 1981). A concurrent explanation is that the role the cerebellum plays during movement production, as a timing controller, may be overestimated. The exact explanation is still waiting to be tested. The important point is that despite the cerebellum's role during the movement performance, and the characteristics of the cerebellum of individuals with Down syndrome, these individuals were able to improve their motor performance at high level. Also, the studies of the movements of individuals with Down syndrome may present a good model from which we could better understand the role of the cerebellum during the movement execution.

The danger of these stereotypes is not just the misconceptions they reproduce at a theoretical level, but above all, their influence at a practical level. If therapists, parents or child development teachers believe that because of an organic dysfunction (i.e., low cerebellar weight) the individuals with Down syndrome will be unable to improve their motor performance, then there is no room for teaching. This vision reflects a predeterministic view about child development, which is heavily influenced by Gesell's theory of dependent stages. According to (Gesell, 1946) the biological maturational process, which is ontogenetic in nature, internally guides infants through a sequence of stages, despite environmental factors. Today, this predeterministic view about development does not find many adherents in several areas, such as language, cognition, social behavior. However, the area of motor behavior is still under the influence of this predeterministic view. The reason for this may be associated
with our lack of understanding of how environmental factors enhance motor performance. Research in this area could lead to a better understanding of the mechanisms behind motor acquisition. The research about the effects of environmental factors on motor performance acquisition in different populations is imperative.
LITERATURE CITED


APPENDIX A - CONSENT FORM AND SUBJECTS INFORMATION SHEET
RUSH-PRESBYTERIAN-ST. LUKE'S MEDICAL CENTER
HUMAN INVESTIGATION COMMITTEE

CONSENT FORM

(Please type all information.)

I, ____________________________________________, an adult (or legal guardian of ____________________________________________, a minor), have been invited to participate in a study of _______________ under the direction of Drs. Danieli Corcos and Mark Latash

universally. (312) 636-5616 in which I voluntarily consent to participate.

The implications of my voluntary participation in this medical study, its nature, duration and purpose, the methods and means by which it is to be conducted, and the inconvenience and hazards which may be expected have been thoroughly explained to me by ________________________________

Danieli Corcos, Mark Latash, or Gil Almada

I have read and understand all written materials which have been provided to me, further describing the study and its potential risks and benefits to me.

I have been given an opportunity to ask any questions I wish concerning this study and all such questions have been answered to my complete satisfaction. I understand that I may terminate my participation in this study at any time without affecting the level of my medical care. I also understand that my participation in this study may be terminated at any time if in the opinion of my personal physician or the director of the study this is in my best interests. If I have any further questions, problems or questions about my rights as a research subject, I should contact the above named director of the study.

(If not applicable, check X). I certify that, to the best of my knowledge, I am not pregnant at this time. I agree that if I become pregnant during the course of this study I will notify the above named director of the study.

I understand that the information gathered in this study (including medical records) may be reviewed by the sponsor, and appropriate government agencies, including the U.S. Food and Drug Administration (21 CFR Part 50.25 (a) (5)), when authorized by statute and regulation. I further understand that my identity will be kept confidential and no identifying information will be released or published.

I understand that in the event of injury resulting from this study, there is no compensation available from the Medical Center for such injury and that I will obtain any necessary medical care for such injury in the same manner in which I obtain any other medical care. (This notice is printed here pursuant to Federal regulations 21 CFR Part 50.25 (a) (6) and (7) and 45 CFR Part 46.116 (a) (6) and (7)). I understand that in the case of injury resulting from this study, I should contact the Office of Risk Management at (312) 942-7625.

I understand that the director of the study will inform me of significant new findings developed during the course of the study which may affect my willingness to continue to participate in the study.

I understand that any drugs or devices provided to me for use outside of the Medical Center should be safely stored, kept away from children and used only as directed by my physician.

If subject is a minor:

C Verbal assent has been obtained.

C Verbal assent has not been obtained because of a waiver of this requirement (check only if granted by the Human Investigation Committee).

Signature of Parent or Guardian (when applicable)

I was present during the explanation referred to above, as well as the subject's opportunity for questions, and hereby witness his or her consent to participate in the study

Witness signature and date

NOTE: Please type or print name below signature line. This consent form cannot be signed by a minor. Signatures on this form must be as returned on the Principal Investigator, as witnessed by the subject's principal investigator, and given to the subject.

Attachment information shown on this form

REAPPROVED

06/7/99

HIC

WRZ-07-30-99

WILL EXPIRE 30 YEARS FROM ABOVE DATE
SUBJECT INFORMATION SHEET

The present research is directed towards understanding movement disorders in Down syndrome. This is an experimental procedure, and we cannot assure you of any personal benefit from participating in the study. We shall immediately discontinue the testing session if you or your guardian request.

During the testing, you will sit in a chair, the forearm lying on a plank. When the plank is fixed, you will be asked to rapidly increase the force of your muscles; when the plank is free, you will be asked to perform fast or slow movements of the elbow. You can see a green “home”, a white moving spot, and a pair of red horizontal bars on the screen. The white spot always shows position of your elbow. You should start all the movements from the green “home”. When you hear a “beep”, move your arm so that the white spot gets to the red bars and stop them. When the beep is over, return to the green “home”. The recording self-adhesive skin electrodes will be placed over the main elbow muscles. Before placing the electrodes, we will rub the skin with alcohol solution. If you feel any discomfort from the electrodes, tell us, and we shall try to find a comfortable placing. We ask you to participate in eleven sessions, one hour each one.

The apparatus you see is designed to record signals from your muscles, muscle forces, and movements in your elbow joint. This procedure is quite safe. If you have any questions, please ask them, and we shall try to answer them for you.

“I have read and understood the information in this Subject Information Sheet and have received a copy. I have volunteered to participate based on this information.”

________________________________________
Signature of Patient or Legal Guardian
Date

________________________________________
Name of Patient or Legal Guardian
and Social Security Number

________________________________________
Home address

________________________________________
Signature of Witness

________________________________________
Name of Witness

REAPPROVED
OCT 8 1996
BY H.I.C.
YOU USE YEAR FROM ABOVE DATE
APPENDIX B - SUPPLEMENTARY DATA
Table B1

Confidence interval of peak velocity between each of the 10 blocks of the 10th session and the respective blocks of the 1st, 4th, 7th session of training.

<table>
<thead>
<tr>
<th>BLOCKS</th>
<th>SE 10 - SE 1</th>
<th>SE 10 - SE 4</th>
<th>SE 10 - SE 7</th>
</tr>
</thead>
<tbody>
<tr>
<td>B1</td>
<td>205.76; 72.34</td>
<td>164.98; 18.57</td>
<td>-.81; 38.04</td>
</tr>
<tr>
<td>B2</td>
<td>144.51; 83.85</td>
<td>86.71; 42.05</td>
<td>-21.81; 31.81</td>
</tr>
<tr>
<td>B3</td>
<td>143.07; 69.70</td>
<td>100.44; 27.76</td>
<td>-39.51; 33.35</td>
</tr>
<tr>
<td>B4</td>
<td>155.50; 70.32</td>
<td>88.74; 47.75</td>
<td>5.97; 36.18</td>
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<tr>
<td>B5</td>
<td>164.22; 82.22</td>
<td>94.21; 24.37</td>
<td>-9.85; 24.84</td>
</tr>
<tr>
<td>B6</td>
<td>161.76; 33.88</td>
<td>81.24; 4.95</td>
<td>-34.14; 38.19</td>
</tr>
<tr>
<td>B7</td>
<td>127.06; 81.49</td>
<td>75.38; 14.77</td>
<td>-27.06; 29.38</td>
</tr>
<tr>
<td>B8</td>
<td>149.58; 58.97</td>
<td>84.78; -1.86</td>
<td>-34.12; 22.78</td>
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<td>B9</td>
<td>124.87; 53.47</td>
<td>81.08; -19.15</td>
<td>-22.86; 32.88</td>
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<tr>
<td>B10</td>
<td>116.98; 36.73</td>
<td>87.03; -14.78</td>
<td>-43.91; 39.91</td>
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Table B2

a) Linear correlation between antagonist latency and acceleration time for the average of the 1st, 4th, 7th, and 10th session of practice.

<table>
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<tr>
<td>S3</td>
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<td>.95</td>
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<td>S4</td>
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<td>S5</td>
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<td>-.05</td>
<td>.08</td>
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<tr>
<td>S8</td>
<td>11</td>
<td>.73</td>
<td>.71</td>
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</table>

b) Linear correlation between antagonist latency and acceleration time for the average of the 1st session of practice.

<table>
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<td>13.22</td>
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<td>S3</td>
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<td>S4</td>
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<tr>
<td>S8</td>
<td>50</td>
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<td>.69</td>
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c) Linear correlation between antagonist latency and acceleration time for the average of the 4th session of practice.

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</tr>
<tr>
<td>S4</td>
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<tr>
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d) Linear correlation between antagonist latency and acceleration time for the average of the 7th session of practice.

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e) Linear correlation between antagonist latency and acceleration time for the average of the 10th session of practice.

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<td>.95</td>
<td>.49</td>
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Table B3

a) Logarithm relationship between the average of peak velocity and $Q_{30}$ of the 1st, 4th, 7th, and 10th session of practice.

<table>
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<th>SLOPE</th>
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b) Logarithm relationship between the average of peak velocity and $Q_{30}$ of the first session of practice.

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c) Logarithm relationship between the average of peak velocity and $Q_{30}$ of the 4th session of practice.

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e) Logarithm relationship between the average of peak velocity and $Q_{30}$ of the 10th session of practice.

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Table B4

a) Logarithm relationship between the average of peak velocity and agonist activity of the 1st, 4th, 7th, and 10th session of practice.

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b) Logarithm relationship between the average of peak velocity and agonist activity of the first session of practice.

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c) Logarithm relationship between the average of peak velocity and agonist activity of the 4th session of practice.

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e) Logarithm relationship between the average of peak velocity and antagonist activity of the 10th session of practice.

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Table B5

a) Logarithm relationship between the average of peak velocity and antagonist activity of the 1st, 4th, 7th, and 10th session of practice.

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b) Logarithm relationship between the average of peak velocity and antagonist activity of the first session of practice.

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c) Logarithm relationship between the average of peak velocity and antagonist activity of the 4th session of practice.

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e) Logarithm relationship between the average of peak velocity and the antagonist activity of the 10th session of practice.

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