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Analysis of "Invariant Characteristics" in the Motor Control of Down's Syndrome and Normal Subjects

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ABSTRACT. Following Asatryan and Fel'dman (1965), two experiments were conducted to describe the so-called invariant mechanical properties underlying movement control in Down's syndrome and normal subjects. The invariant characteristic is a curve on a graph of joint torque versus joint angle. The task required subjects to maintain a steady joint angle against an external load (torque). Torque was systematically changed via partial unloading in order to obtain torque by length (joint angle) functions at three separate initial joint angles. Instructions required subjects "not to intervene" when unloading occurred in Experiment 1 and to "tense" their muscles prior to unloading in Experiment 2. Both normal and Down's syndrome groups revealed systematic torque by length functions that might be expected according to a simple mass-spring system model. Although the gross organization of movement in Down's syndrome subjects was nearly the same as normals, important differences between the two groups were found. Down's syndrome subjects revealed underdamped motions relative to normals (as shown by differences in the degree of oscillation about the final equilibrium position) and were less able to regulate stiffness (as shown by differences in slope of the torque by angle functions in Experiment 2). We promote the notion that damping and stiffness may be sensitive indices of hypotonia—the most common description of neuromuscular deficiency in Down's syndrome.

THERE IS VERY little research on the control and coordination of movement in Down's syndrome subjects. The few studies that do exist reveal that this population exhibits abnormal gait (James, 1974), slow movement responses (Berkson, 1960; Lange, 1970), and is less accurate on certain motor tasks (Frith & Frith, 1974) than their normal counter-

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parts. However, questions concerning the underlying organization of motor control in Down's syndrome subjects remain unanswered.

The most often implicated motor deficiency in Down's syndrome subjects is hypotonia, a decrease or absence of muscle tone. Indeed, nearly all Down's syndrome infants are born with hypotonia, a condition that may contribute greatly to their delay in reaching motor milestones (cf. Cowie, 1970). However, no definitive statement can be made about hypotonia in Down's syndrome individuals beyond infancy (cf. Knight, Atkinson, & Hyman, 1967; Owens, Dawson, & Losin, 1971). To complicate things, the exact relationship between hypotonia and motor control is, as yet, unclear (cf. Neilson & Lance, 1978).

One model—for which there is accumulating empirical evidence—which may allow us to understand movement organization in Down's syndrome, draws an analogy between the behavior of muscle collectives and a vibratory system (cf. Turvey, 1977). For example, in a now classic experiment, Asatryan and Fel'dman (1965) had subjects establish steady-state joint angles against an external load. On each occasion, when the load was systematically reduced and the subjects instructed not to intervene, the joint angle changed. Resulting torque by length (joint angle) functions generated from different initial positions were parallel and non-intersecting as would be expected from a mass-spring model.

Additional evidence supporting a vibratory system model comes from research demonstrating equifinality in the muscle-joint systems of human subjects (e.g., Bizzi, 1980; Fel'dman, 1966b; Kelso, 1977; Kelso & Holt, 1980; Schmidt & McGown, 1980, for reviews) and trained monkeys (Bizzi, Dev, Morasso, & Polit, 1978; Bizzi, Polit, & Morasso, 1976). Equifinality, as defined, refers to a system's ability to equilibrate based solely on its parameters, independent of initial conditions (von Bertalanffy, 1973).[1]

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[1]*Note that equifinality can be disrupted in deafferented but not normal animals when the postural relations between animal and apparatus are changed (Polit & Bizzi, 1978). When the latter are fixed however, deafferented monkeys and functionally deafferented humans (Kelso, 1977; Kelso, Holt, & Flatt, 1980) exhibit equifinality when the initial position of the limb is altered unexpectedly.*

Following Asatryan and Fel'dman's (1965) analysis, the present study sought to describe the static mechanical properties of the muscle joint system of Down's syndrome subjects using a simple mass-spring equation: $F = -K(l - l_0)$, where F is an external force, $-K$ is stiffness, l is the current length of the spring, and l_0 is the length of the spring when no forces are acting on it. Experiment 1 was designed to determine the so-called invariant characteristics (cf. Fel'dman, 1980a) of the muscle-joint system of Down's syndrome subjects. This was achieved by examining the relationship between change in joint angle under conditions of partial unloading when subjects were instructed not to intervene voluntarily against the unloading, thereby holding the parameters of stiffness and zero length relatively constant. Under these conditions, any change in joint angle should be systematic with the change in the load (torque).

A second experiment examined the extent to which Down's syndrome subjects were capable of regulating stiffness by requiring subjects to tense their muscles voluntarily at particular joint angles prior to unloading. Asatryan and Fel'dman (1965) demonstrated that normal subjects could voluntarily increase the stiffness of the muscle-joint system as reflected by increases in the slope of the torque by length function. It is not known whether stiffness can be regulated voluntarily by Down's syndrome subjects. Qualitatively speaking, the Down's syndrome population is characterized by hypotonia (flaccidity), which may relate to muscle stiffness or to damping. Damping is the internal frictional force present in the system and is indicated by the extent of overshoot or oscillation about the equilibrium point. As this study shows, both stiffness and damping appear to be sensitive indices of the impaired performance of Down's syndrome subjects.

EXPERIMENT 1

Methods

Subjects. The subjects in this study were seven Down's syndrome male students between 14 and 21 years of age who attended Celentano School for the developmentally handicapped in New Haven, Connecticut. These subjects were selected from among 30 subjects who participated in a previous study that employed limb localization movements (Davis & Kelso, Note 1). The subjects were selected on the basis of their ability to complete the task. The Celentano School administration reported an IQ range of 25–60 for the subjects involved in this study. A control group consisted of six normal male subjects selected from the subject pool listed at Haskins Laboratories in New Haven who were paid for their services.

Apparatus. The apparatus consisted of a 593C finger positioning device along with an associated electronics control package (see Kelso & Holt, 1980, for description). The main parts of the apparatus were two movable arms each attached to a separate metal shaft mounted vertically on the top of an open box frame. Only the right hand was used in these experiments. The frame was mounted on a table 78.5 cm high.

The movements allowed by the positioning device were flexion and extension of the index finger about the metacarpophalangeal joint. The distal end of the moving finger was fitted with a plastic collar that slipped into an open-ended cylindrical support attached to the movable arm. The movable arm consisted of two parallel bars fitted perpendicularly into the metal shaft. A pointer was attached to the end of the movable arm and moved along a protractor calibrated in degrees. The apparatus was also equipped with padded adjustable braces with which to secure the subject's wrist, hand, and remaining fingers and thumb during the movements.

The gear arrangement driven by a torque motor provided resistance (load) to the movable arm (and hence to the finger when placed in the cylinder attached to the arm) when current was supplied to the motor. The electronics control box allowed for regulation of the current supply and could be set in either of two modes. While in the first mode, which shall be referred to as "servo-torque control," a resistance (torque) acted on the finger whenever the finger deviated from the set servo-position. When the finger was in the servo-position, no resistance acted upon it. When in the second mode, referred to as "torque control," a constant resistance could be applied to the finger (settable in either direction). The amount of resistance (torque) was adjustable and could be set anywhere from 0 to 100% of the maximum torque available from the motor (81.6 ounce-inches).

Procedure. Procedures for the normal and Down's syndrome subjects were identical. The normal subjects were tested at Haskins Laboratories and the Down's syndrome subjects were tested at Celentano School over a four-day period. Each subject was scheduled for a 15-min session each day. As a consequence of the difference in venue, Down's syndrome subjects' movements were analyzed via visicorder tracings (Honeywell, 1508C) while normal subjects' movements were fed directly into a PDP 11/45 for later computer analysis. For both types of recordings, a set number of degrees of movement corresponded to a calibrated voltage.

Each subject sat comfortably facing the apparatus with his right finger placed securely into the cylinder attached to the arm. Upon a signal from the experimenter, the subject moved to a specified steady-state joint angle (target) acting against a load supplied by the torque motor. The target angles for Experiment 1 were as follows: S1 = 155°, S2 = 165°, and S3 = 175°. When the subject's finger pointed straight ahead, the position was equal to 180°. The subject reached the target angle with a flexion movement from a starting position of 192°. The target angle was set by the subject by matching a line on the oscilloscope. When the angle was set, the subject closed his eyes and the oscilloscope was turned away. Within 0.5 to 2.0 sec after the target angle was achieved, a proportion of the load was released. The subject was instructed to maintain a steady resistance against the load—as indexed by the stable achievement of initial angle—and not to interfere voluntarily with the movement of the finger if the load was released.

For this study, partial unloading was achieved in the following manner. The control was set in servo-torque mode at the specified servo-position corresponding to the starting position as noted above. After the subject moved his finger to a specified joint angle, counteracting the resistance (100% torque) exactly, part of the load was released by switching from servo-torque control to torque control. This was achieved by the experimenter moving the manual switch on the control box. The amount of load released was regulated by setting the percentage of torque load. To obtain negative load release, the torque load was set at 5 and 10%, but the direction of the resistance was reversed to act in the direction of flexion (i.e., in the direction that the finger was moving).

The measurements taken were changes in joint angle and changes in the amount of resistance acting on the finger. Measurements of joint angle change were taken by hand from visicorder tracings of finger displacement and measured to the nearest .5 cm or obtained directly following A-to-D conversion (200 Hz) on the PDP 11/45. The criterion for determining the new steady-state joint angle was the point at which movement ceased (as shown by the movement tracings) for at least 500 msec (.5 cm on the tracing) following unloading.

A series of partial unloadings was conducted with each subject at each of three initial joint angles, S1, S2, and S3. For each series, seven separate unloadings were conducted (60%, 40%, 25%, 10%, 0%, −5%, and −10%), representing a percentage of maximum torque. The seven separate unloadings provided a sufficient amount of data to describe the torque by joint angle functions.

For each of the unloadings, at least four trials were carried out. In all, more than 184 trials were conducted with each subject. The order in which the trials were given within each series, for the different unloadings, was randomly assigned. Series 1, 2, and 3 were presented to each subject in a predetermined balanced order.

Design. The data obtained from both Down's syndrome and normal subjects were graphed to determine if the series of torque by joint angle functions were parallel and non-intersecting as might be expected from the static equation, $F = -K(l - l_0)$. The algebraic error data were analyzed using a 2 (groups) × 3 (series) × 7 (unloadings) analysis of variance with repeated measures on the last two factors. A similar analysis was performed on variable error. In addition, tests of linearity (Wine, 1964) were also performed on the curves.

Results

Three sets of torque by joint angle functions were obtained as a result of partial unloading. The joint angle change in this case represents a deviation from the initial angle and conforms to standard measures of mean algebraic (constant) error. The results for the normal and Down's syndrome subjects are shown in Figure 1. The curves obtained for both normal and Down's syndrome subjects are indeed parallel and non-intersecting. It is clear from the figure that the two groups are nearly

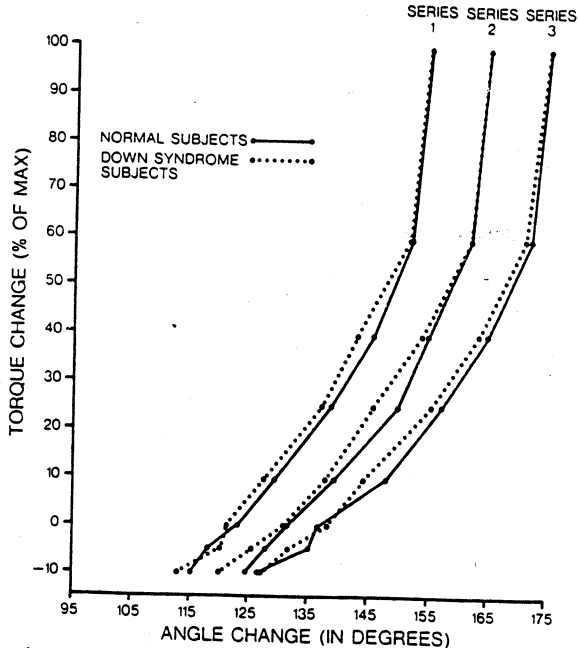

Fig. 1—Angle change as a function of torque change under the instructions of Experiment 1—"do not intervene." The large dots represent the mean angle change for the group on each of the seven unloadings.

identical, and this was borne out by statistical analysis, $F(1,11) = .20$, $p > .05$. As expected, there was a significant torque change effect, $F(6,132) = 173.8$, $p < .001$, as well as a significant series effect, $F(2,132) = 6.23$, $p < .01$. There was also a significant series by torque interaction, $F(12,132) = 2.65$, $p < .01$. Analysis of this interaction revealed that for the three greatest unloadings (0%, −5%, and −10%) the joint angle change was larger between series 3 and 2 as well as series 3 and 1. Series 2 also revealed larger joint angle changes than series 1 at these unloading values.

Analysis of variable error, like constant error, reflected a high degree of similarity between Down's syndrome and normal groups. Only the torque effect was significant, $F(6,132) = 25.67$, $p < .001$. For the seven different unloadings (from 60% to −10%), variability in angle change appeared to increase systematically (1.0°, 1.7°, 2.4°, 3.2°, 3.8°, 4.4°, and 4.1°, respectively). No other main effect or interaction was significant for variable error.

A trend analysis of the F by I function obtained from each of the three series of unloadings for Down's syndrome and normal subjects was performed. The results revealed that the functions for both groups were essentially linear. For example, for the Down's syndrome group, the proportion of variance accounted for by linearity in series 1, 2, and 3 was 94%, 93%, and 92%, respectively.

Examination of the individual movement tracings, however, revealed some interesting differences. Representative movement tracings of individual subjects are presented in Figure 2A (normal subject SK) and 2B (Down's syndrome subject BV). The three movement tracings for each subject were recorded during unloadings of 60%, 10%, and −10% in Experiment 1. Qualitatively speaking, movements of normal subjects from the initial position to the target angle were, on the whole, more direct than the Down's syndrome subjects whose movements were more step-like or discontinuous (e.g., Brooks, 1974) and less stable. Although movement speed was not specified to the subjects beforehand, a rather interesting finding is that Down's syndrome subjects took significantly longer to reach the target angle. A random sample of 136 trials from Down's syndrome subjects was compared to the same number of trials for normal subjects. The mean movement time for the Down's syndrome group was 2.0 sec and the standard deviation was .4 sec compared to the mean and standard deviation of .8 sec and .1 sec for normal subjects. These movement times were significantly different from each other ($p < .001$).

Down's syndrome subjects also differed from their normal peers in terms of oscillatory behavior at the newly established equilibrium position (i.e., following unloading, see Figure 2). Overshoot was measured to the nearest .5° for each of the trials and was analyzed using a group by series by torque analysis of variance with repeated measures on the last two factors. As shown in Table 1, a significantly greater amount of overshoot was found for Down's syndrome than for normal subjects, *F*

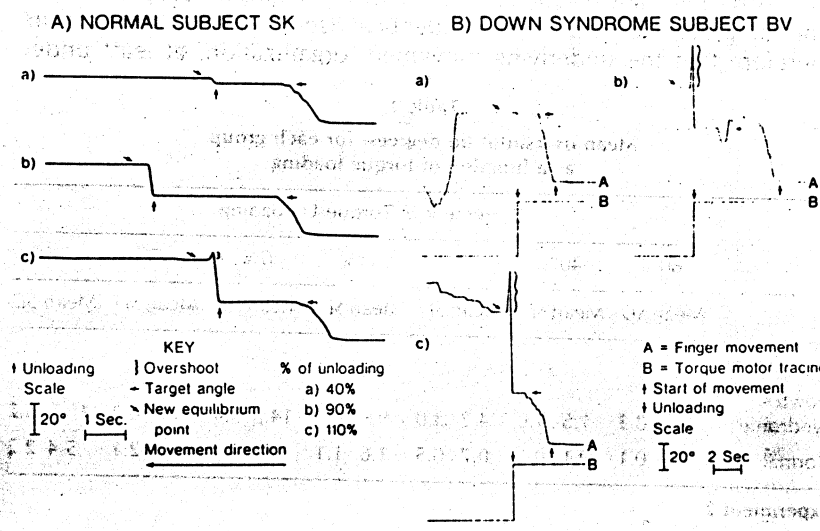

Fig. 2—Tracings of finger movements of individual subjects in Experiment 1. Tracings for normal subjects were taken directly from the computer and tracings from Down's syndrome subjects were taken from the visicorder. Tracings a, b, and c occurred at unloadings of 40% (60%), 90% (10%) and 110% (−10%), respectively.

(1,11) = 21.38, $p < .001$. There was also a significant series main effect, F (2,132) = 6.32, $p < .01$, and torque main effect, F (6,132) = 58.67, $p < .001$. The mean overshoot for each series was 4.87°, 5.48°, and 6.91°, respectively. As the amount of unloading increased (and thus angle change increased), the amount of overshoot increased. This finding holds for both groups, but is magnified in the Down's syndrome group as evident in a group by torque interaction, F (1,132) = 17.27, $p < .001$ (see Table 1).

Discussion

The results of Experiment 1 support the notion that when muscles are constrained to act as a unit in controlling movement about a joint, that unit behaves qualitatively like a mass-spring system. The three sets of torque by joint angle functions obtained for both normal and Down's syndrome subjects were parallel and non-intersecting, and concur with the findings of Asatryan and Fel'dman (1965). It may be reasoned that for the subjects in this study, the $-K$ and l_0 parameters were established in counterbalancing the external force to maintain the specified joint angle. A systematic angle change accompanied the systematic torque change. Thus, it appears that the parameters of $-K$ and l_0 remained relatively constant during unloading. Furthermore, when the subjects were asked, on different occasions, to reach new joint angles, new zero angles were established. The change in zero angle resulted in parallel and non-intersecting functions.

Of course the significant result of the present experiment is that the torque by joint angle functions for Down's syndrome subjects and a normal group were practically identical (see Figure 1). It appears therefore that the underlying movement organization, at least under

Table 1

Mean overshoot (in degrees) for each group as a function of torque loading

	Percent of Torque Unloading													
	60%		40%		25%		10%		0%		5%		10%	
	Mean	*SD*	Mean	*SD*	Mean	*SD*	Mean	*SD*	Mean	*SD*	Mean	*SD*	Mean	*SD*
Experiment 1														
Down's Syndrome	0.2	0.3	1.6	1.6	4.2	3.0	9.9	4.3	14.3	5.4	17.1	6.2	18.0	5.2
Normal	0.1	0.1	0.1	0.1	0.7	0.5	1.6	1.1	3.3	1.6	4.2	2.1	5.4	2.2
Experiment 2														
Down's Syndrome	0.1	0.1	—		3.5	2.3	—		9.4	4.9	—		12.5	5.5
Normal	0.2	0.3	—		0.9	0.5	—		1.3	1.1	—		1.1	1.9

static conditions, is basically similar in both populations. From a mass-spring perspective, Down's syndrome subjects, like normals, appear capable of specifying system parameters— stiffness and equilibrium length—that may determine movement at the joint.

Although the curves appear graphically non-linear, the torque by joint angle functions were characterized by a statistically linear trend. Thus, the non-linear component in this study is somewhat less than might be expected based on the findings and qualitative analysis of Asatryan and Fel'dman (1965). However, these authors did not subject their obtained functions to any statistical analysis as we have done here.

One way of interpreting the present findings is that the nervous system (in both Down's syndrome and normal populations) is able to "tune" the muscle-joint system by adjusting the length-tension relationships of the muscles involved. In the simple case, agonist-antagonist pairs can be represented by parallel length-tension curves whose slopes correspond to muscle stiffness. A change in innervation rate to one muscle or the other will shift the equilibrium point of the system (cf. Bizzi, 1980; Cooke, 1980; Fel'dman, 1966b, 1980a, 1980b; Kelso & Holt, 1980). In this view, supported by the ability of deafferented monkeys (Bizzi, Dev, Morasso, & Polit, 1978; Bizzi, Polit, & Morasso, 1976) and humans without intact joint and cutaneous reception (Kelso, 1977; Kelso & Holt, 1980) to accurately localize limbs, stiffness is set prior to movement and is a *control* parameter. On the other hand, Houk (1978) has presented evidence in favor of a view in which a combination of muscle spindle and tendon proprioceptors provides feedback about muscle stiffness. In this view, stiffness is a *regulated* variable of the system. Regardless of which view one adopts, *both* are consonant with the perspective offered here (but see General Discussion, herein, for possible qualifications on this view and also Cooke, 1980, for a model of how mechanical and reflex variables may interact). It is the specification of *dynamic* variables (e.g., stiffness, damping) rather than kinematic variables (e.g., displacement, velocity) that appropriately characterize the neuromuscular organization of the muscle-joint system.

There were, however, qualitative differences among the movement patterns of Down's syndrome and normal subjects. There were clear differences between the graphs in the trajectories toward the target angle (see Figure 2). Moreover, Down's syndrome subjects were less able, after reaching the target, to maintain a steady position. One possible explanation for the latter finding is that once the target was reached, visual guidance was removed. In a previous study (Davis & Kelso, Note 1), it was found that Down's syndrome subjects were less able than normal subjects to reproduce movements accurately without visual guidance. However, the movements of Down's syndrome subjects in the present study were also less smooth and accurate when visual guidance was available. In the present study, movements were made by matching a cursor to a fixed line target on an oscilloscope screen. Visual guidance from an oscilloscope may not be the same as direct visual guidance of the finger. Nevertheless, under the present conditions of visual

guidance to a target as well as maintaining a set joint angle with visual information absent, Down's syndrome subjects were not as accurate as their normal peers.

Second, significantly greater overshoot or fluctuation about the equilibrium point was found in Down's syndrome subjects following unloading. Overshoot may be taken as an index of the damping parameter. For example, an underdamped system will fluctuate about the equilibrium position and, for most purposes, is unstable. On the other hand, an overdamped system exhibits slowed movement speed and no oscillation about the equilibrium point. A critically damped system is one in which the movement will reach the equilibrium position in the fastest possible time. Most human muscle joint systems appear to be damped just under critical (cf. Kelso & Holt, 1980; Neilson & Neilson, 1978). Neilson and Neilson (1978), for example, found their normal subjects did not exceed five percent of the movement arc in overshoot during a rapid voluntary movement.

The results of our Experiment 1 conform to the above figure; overshoot was found to be 8.4% of the total movement arc for all normal subjects' movements. Different amounts of overshoot may be due to the differences in the movement conditions between the present study and that of Neilson and Neilson (1978). Overshoot was measured in the Neilson and Neilson study following a rapid voluntary movement. In our study however, overshoot was measured following unloading during which an active halting of the limb was assumed not to have occurred. It seems reasonable therefore to expect some difference in overshoot between the normal subjects in our study and those in the Neilson and Neilson (1978) study. Perhaps the more important finding is that Down's syndrome subjects, in sharp contrast to normal subjects, appear to behave in an underdamped manner, as suggested by the 27.4% of overshoot found for this group.

Although the results of Experiment 1 revealed that the torque by joint angle functions were nearly identical for Down's syndrome and normal subjects, a question remains as to whether both populations can alter the slope of the functions (specify stiffness) to a similar degree. Following Asatryan and Fel'dman (1965), one way to examine this question is to require subjects to voluntarily tense the muscles prior to unloading. "Stiffening" the muscle-joint system in this manner should reduce the amount of absolute joint-angle change, thus increasing the slope of the torque-joint angle functions. In Experiment 2, the foregoing strategy was employed to determine whether Down's syndrome individuals could control stiffness as effectively as normal subjects.

EXPERIMENT 2

Methods

The subjects, methods, and procedures in Experiment 2 were the same as in Experiment 1 except that subjects were instructed to tense (co-contract) their muscles in an effort to maintain the joint angle against

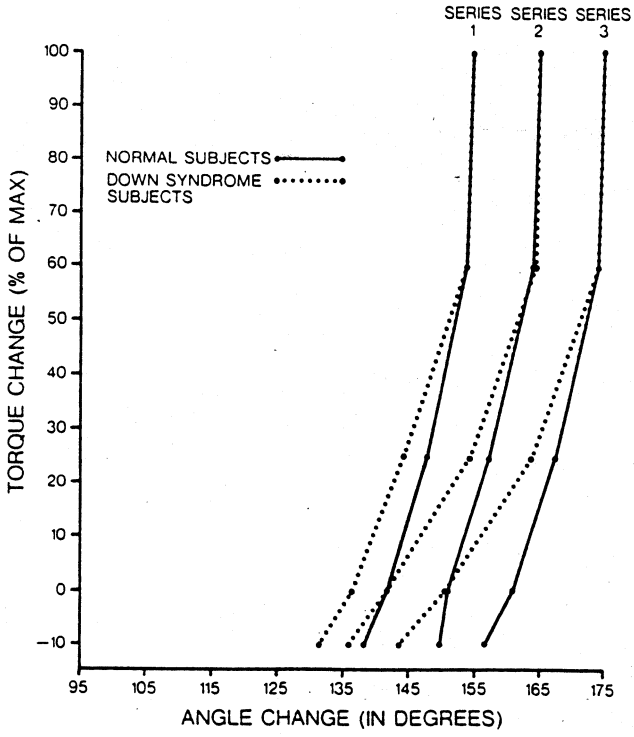

Fig. 3—Angle change as a function of torque change under the instructions of Experiment 2—"stiffen your muscles." The large dots represent the mean angle change for the group on each of the four unloadings.

the perturbation. Each subject was given some practice prior to the experiment proper to ensure that the instructions were understood. In the experimental trials, on reaching the target angle, subjects were asked to "stiffen their muscles" to maintain the joint angle.

Only four separate unloadings (60%, 25%, 0%, and −10%) were used for Experiment 2. These four unloadings provided a sufficient description of the torque by joint angle function without inducing undue fatigue. For each of the unloadings, at least four trials were carried out and more than 48 trials were conducted for each subject.

Data were graphed as in Experiment 1 and analyzed as before using a 2 (group) × 3 (series) × 4 (unloadings) analysis of variance with repeated measures on the last two factors.

Results

Changes in joint angle under "muscle stiffening" conditions are shown in Figure 3 for normal and Down's syndrome subjects. Visual inspection of the figure suggests some potential difference in the torque-joint angle functions of normal and Down's syndrome subjects.

Although overall differences between the groups just failed to reach significance, F (1,11) = 4.35, $p > .05$, there was a significant torque effect, F (3,66) = 69.86, $p < .001$, and group by torque interaction, F (3,66) = 4.61, $p < .01$. Inspection of the means shows that joint angle increases as a function of the magnitude of unloading, and that this effect is greater for Down's syndrome subjects (see Table 1). The mean angle changes for each unloading for Down's syndrome subjects were 1.42°, 12.40°, 23.49°, and 29.52°, and 1.29°, 8.08°, 14.66°, and 17.84° for normal subjects.

A series by torque interaction was also found, F (3,66) = 3.13, $p < .01$. As in Experiment 1, at the higher levels of unloading (the last two in Experiment 2), the magnitude of angle change increased between series 1 and 2 and between 2 and 3.

There were no significant differences between the groups in the standard deviation of mean angle change, F (1,11) = 3.18, $p > .05$. However, there was a significant torque effect, F (3,66) = 20.62, $p < .001$, and a significant torque by series interaction, F (6,66) = 4.76, $p < .001$. Variability increased as the magnitude of the unloading increased, but was significant only in series 1 and 3.

Representative movement tracings from Down's syndrome and normal subjects in Experiment 2 are shown in Figure 4. Qualitatively, the movements of the subjects are similar to those in Experiment 1. Down's syndrome subjects appeared less capable of reaching and maintaining the target angle (compare Figures 2 and 4).

Likewise, overshoot or oscillation about the equilibrium point following unloading was amplified in Down's syndrome subjects (see Table 1)

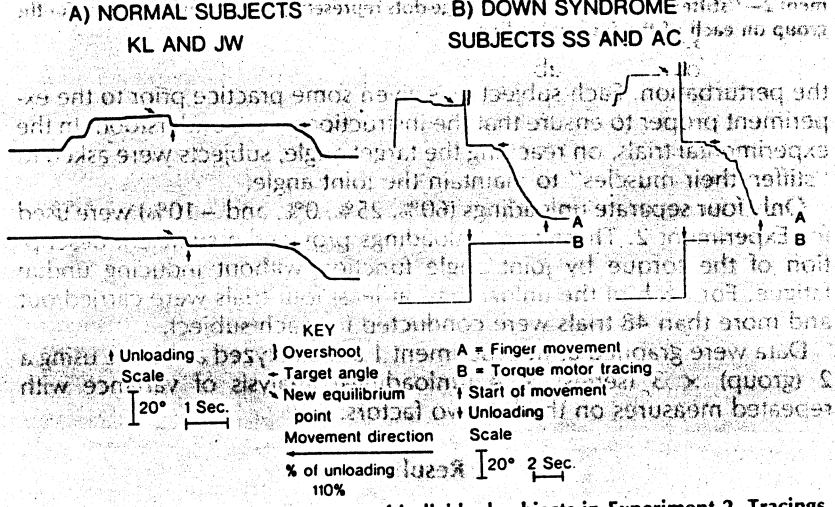

Fig. 4—Tracings of finger movements of individual subjects in Experiment 2. Tracings for normal subjects were taken directly from the computer and tracings for Down's syndrome subjects were taken from the visicorder. The tracings occurred during an unloading of 110%, i.e., - 10%.

and was revealed in a group main effect, $F(1,11) = 8.88$, $p < .05$. The main effects of series, $F(2,66) = 5.96$, $p < .01$, and torque, $F(3,66) = 1.82$, $p < .001$, were also significant. Increases in overshoot occurred from series 1 to 2 and from 2 to 3, and were also evident as the magnitude of unloading increased. Increases in overshoot held for each series in Down's syndrome subjects, but not normals, and resulted in a significant group × series interaction, $F(2,66) = 4.39$, $p < .05$. Significant group × torque, $F(3,66) = 8.3$, $p < .001$, and series by torque, $F(6,66) = 3.95$, $p < .01$, interactions were also found. As the unloading increased, the degree of overshoot increased; this effect was magnified for Down's syndrome subjects and for series 2 and 3 (see Table 1, Experiment 2).

Discussion

In the second experiment, it was demonstrated that Down's syndrome subjects were able to increase the stiffness parameter voluntarily when asked to tense their muscles against the load change. This capability is shown by the increase in slope of the torque by joint angle functions from Experiment 1 to Experiment 2 (compare Figures 1 and 3). It is well known that an increase in activation of motoneurons increases the stiffness property of the muscle (Agarwal & Gottlieb, 1977; Andreeva & Shafranova, 1975; Barmack, 1976; Houk, Singer, & Goldman, 1970; Rack, 1969; Safronov, 1970). An increase in stiffness can occur without an increase in force or without a change in joint angle.

Normal subjects were also capable of increasing stiffness and apparently to a somewhat greater extent than Down's syndrome subjects. The mean slopes of the functions generated in both experiments were 1.77 and 3.56, respectively, for Down's syndrome subjects, and 1.83 and 4.67 for normal subjects. There was no statistically significant difference between groups on overall angle change in Experiment 2; however, at higher levels of torque change, significant differences between groups did exist as revealed in the significant group by torque interaction.

There were also some noteworthy differences among individual subjects that warrant some discussion. For example, Down's syndrome subject LM's stiffness characteristics were closer to that found for normal subjects (see Figure 5) and, in fact, exceeded some of the normal subjects. Likewise, one normal subject, GA, displayed muscle stiffness more aligned to the Down's syndrome group than to the normal group (see figure 5). If these extremes are excluded from the groups, the difference between Down's syndrome and normal subjects is magnified. That wide individual differences among subjects exist within both groups is expected. It is known that stiffness varies between and within normal individuals (Safronov, 1970). Also, extreme individual differences among Down's syndrome subjects on a number of psychological and physical variables have been found (James, 1974; LaVeck & Brehm, 1978). It can be reasoned that stiffness as with many

other variables, operates on a continuum rather than strictly dichotomizing the populations observed.

Finally, there is some indirect evidence that suggests Down's syndrome subjects have reduced capacity of muscle activation that might be associated with the ability to regulate muscle stiffness. Most Down's syndrome infants are deficient in the amino acid 5-hydroxytryptophan (Coleman, 1973; Koch & de la Cruz, 1975), which is thought to play an important role in neural transmission (McCoy, Segal, & Strynadka, 1975) and muscle contraction (Ahlman, Grillner, & Udo, 1971). The finding that Down's syndrome subjects have slower movement response times (Lange, 1970) may support the idea that Down's syndrome subjects are less able to activate their muscles. If, as some have claimed (cf. Lestienne, Polit, & Bizzi, 1980), integrated electromyography is an accurate reflection of active stiffness in muscles, then it seems worthwhile to subject the foregoing speculations regarding stiffness to further experimental test.

GENERAL DISCUSSION

A major finding in this study is that the gross underlying organization of motor control in Down's syndrome subjects is qualitatively similar to normal subjects and can be described, to a first approximation, in terms of a vibratory system. We have found that the torque by joint angle curves, described as invariant characteristics of the system by Asatryan and Fel'dman (1965; see also Fel'dman, 1980a, 1980b), are obtainable from Down's syndrome subjects under two conditions of maintaining a

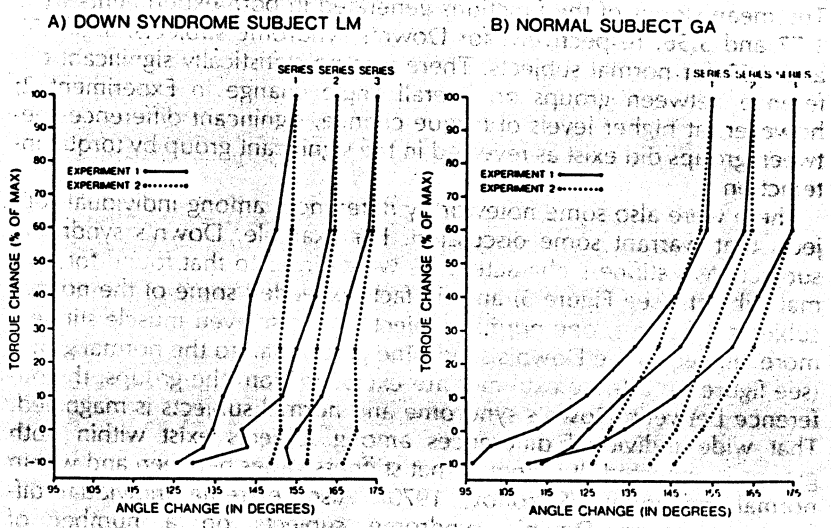

Fig. 5—Angle change as a function of torque change for individual subjects during Experiments 1 and 2. The large dots represent mean angle change for the subject at each of the unloadings.

steady joint angle against a load. In one condition, the subjects did not voluntarily intervene during partial unloading. In the other condition, the subjects voluntarily tensed their muscles which resulted in an increase in the active stiffness of the system. The importance of obtaining the invariant characteristics (static muscle torque versus angle) in this special Down's syndrome population is magnified by the recent findings of Fel'dman (1980a, 1980b). With normal subjects, Fel'dman has shown that the Invariant Characteristic (IC) may characterize the behavior of muscle-joint systems not only during the maintenance of a steady posture (Asatryan & Fel'dman, 1965) but also during rhythmic (Fel'dman, 1980a) and discrete movements (Fel'dman, 1980b; see also Kelso et al., 1980). For example, in order to maintain a steady angle against changing loads, the system need only shift from one IC to another. Referring to Figure 1, it can be seen that in order to maintain a 150° joint angle against a 60% load, the IC of series 1 is used. In order to maintain the 150° angle when the load changes to 25%, a shift from the IC of series 1 to the IC of series 2 is required. This transition from one IC to another appears to be effected through a change of the threshold angle at which motor units are recruited (Fel'dman, 1966a, 1980b; see also Crago, Houk, & Hasan, 1976).

By the same token, movements may be accomplished by shifts along the form of the invariant characteristics, that is, by shifts of the equilibrium point of the muscle load system and by changes in the form of the IC (Fel'dman, 1974a, 1974b). The latter is shown by the set of ICs obtained in Experiment 1 (Figure 1) and the set obtained in Experiment 2 (Figure 3). Through co-contraction of the antagonist muscles, the stiffness of the muscle load system may be increased and this is associated with increases in slope of the IC. It may be assumed that, during movement, transformation from one set of curves to another is possible (Fel'dman, 1980a, 1980b). Thus, movements may be achieved through simple changes in the parameters of the muscle-load system. According to this view, amplitude of movement (position) may be regulated through changes in zero length, and velocity and acceleration through changes in stiffness (cf. Kelso & Holt, 1980).

A cautionary point worth emphasizing here is that the majority of experiments (including ours) and their consequent interpretation, deal with movements in a very restrained environment (e.g., sitting down with shoulder or wrist position fixed). New data reveal that the pattern of stiffness changes at a joint (say, the arm) is mutable, depending in a significant way on the postural status of the subject (Nashner, Note 2). Some modification (or even rejection) of the type of model proposed here for rather fixed actions at a joint, may well be in order when more real-life situations are examined (e.g., a standing subject supported to varying degrees).

Although there appear to be overall similarities in the gross organization of motor control of Down's syndrome and normal subjects, there are notable differences in the *precision* with which Down's syndrome subjects attain target positions (see Figures 2 and 4). As shown by in-

dividual movement tracings and subsequent analysis, the movement patterns were qualitatively different between Down's syndrome and normal subjects in both Experiments 1 and 2. The findings here are consistent with other studies that show Down's syndrome subjects to be less accurate in controlling movements than their normal peers (e.g., Davis & Kelso, Note 1).

Relatedly, and perhaps most important, Down's syndrome subjects differed from their normal peers in oscillatory behavior about the newly established equilibrium position (i.e., following unloading, see Figures 2 and 4). Oscillatory behavior is taken as an index of the damping parameter. As previously noted, the finding that underdamping characterizes the muscle-joint system of Down's syndrome subjects is consistent with the finding that these subjects are less accurate in movement than normal subjects. It is the underdamping characteristic, in addition to stiffness, that may distinguish Down's syndrome from normal subjects. In this regard, it is important to note that although individual subjects GA (normal) and LM (Down's syndrome) were unlike their respective groups with respect to stiffness, they did not deviate from group performance on measures of damping. Mean overshoot for GA was 2.56° compared to the overall mean of 2.20° for the normal group. Likewise the means for LM and the Down's syndrome group were 9.35 and 9.31, respectively. It is not known whether Down's syndrome subjects are able to modify the damping parameter through training. Other studies have suggested that normal subjects can be trained to regulate damping during voluntary movement (e.g., Neilson & Lance, 1978). But further investigations are needed to determine to what extent Down's syndrome subjects have this capacity.

Overall, a number of findings concerning the motor control of Down's syndrome subjects are provided in this study and several avenues of research are suggested. We found that the Down's syndrome subjects in this study are not readily distinguishable from normal subjects in terms of gross movement organization; systematic torque by joint angle functions were obtained for both groups. Apparently, muscles are constrained to act as a unit in both normal and Down's syndrome subjects and this unit exhibits behavior—to a first approximation—qualitatively similar to a mass-spring system (cf. Kelso, Holt, Kugler, & Turvey, 1980, for review). Where the groups differ, however, is in the specification of stiffness, especially at high values of torque unloading and in the damping characteristic. It is interesting in this regard that a recent analog model—similar to the one under consideration here—characterizes hypotonia in terms of decreases in resting stiffness (Cooke, 1980). If our interpretation is reasonable, we may advance the hypothesis that it is a deficiency in setting damping and stiffness parameters that best characterizes the motor behavior of people with Down's syndrome—at least in simple, discrete movements. This view promotes a trend away from more descriptive terms, like hypotonia, that have been used up to now.

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