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Stance Posture Control in Select Groups of Children with Cerebral Palsy: Deficits in Sensory Organization and Muscular Coordination*

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Summary. This study has focused upon the automatic components of posture and movement in a group of ten cerebral palsy children carefully selected to represent a spectrum of abnormalities relatively pure by clinical standards and ten age-matched normals. Each subject stood unsupported upon a movable platform and within a movable visual surround and was then exposed to external perturbations or was asked to pull with one arm upon a movable handle. In comparing the performance of cerebral palsy children in each clinical category with the age-matched normals and with normal adults assessed in previous studies, the process of maintaining stance was subdivided into two component functions: substrates which determined the onset timing, direction and amplitude of postural actions from somatosensory, vestibular, and visual stimuli were termed "sensory organization", and those establishing temporal and spatial patterns of muscular contractions appropriate to produce effective movements were termed "muscle coordination". We found among seven of the ten cerebral palsy children a clear localization of dysfunction within either sensory organization or muscle coordination mechanisms. These results are providing some new insights into the organization of each of these processes as well as suggesting methods for developing a more systematic understanding of the abnormalities of movement control.

Key words: Posture control – Muscular coordination – Sensory organization – Cerebral palsy – Sensorimotor development

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Introduction

Cerebral palsy is a clinical syndrome which encompasses a wide variety of non-progressive sensorimotor abnormalities, the common denominator among them being a genesis of damage to the immature brain. Understanding the various postural and voluntary movement deficits associated with this syndrome has been hampered by lack of a systematic concept for explaining the coordination of posture and movement behaviors of normals. Instead, CP motor abnormalities have been categorized by combining clinical "signs"; spasticity (hyperactive stretch reflexes, clonus, increased tone), ataxia (disorders of equilibrium, decreased tone), motion disorders (athetosis, chorea, dyskinesia), and combinations thereof; with patterns of the topographic distribution; hemiplegia (involvement predominantly on one side), diplegia (both sides, more involvement in lower extremities), and quadriplegia (total body involvement). Clinical signs in conjunction with their patterns of distribution give rise to a clinical classification system of CP, the use of which has been limited to diagnostic and therapeutic purposes.

The idea that coordinated accurate movements are supported by proprioceptive feedback mechanisms which come into play to correct externally or internally induced errors in position, velocity, and force of movement has motivated a number of studies examining stretch reflex alterations in spastic patients. While there was agreement among these studies that spasticity is associated with elevated dynamic stretch reflex responses (e.g., Burke et al. 1970, 1971; Novikova 1970; Herman 1970; Hagbarth et al. 1973; Dietrichson 1971a, b), a lack of reflex suppression upon repetitive stimulation and abnormal radiation of activity to nearby muscles (Barolat-Romana and Davis 1980), stretch reflex abnormalities did not correlate well with the functional dis-

abilities of patients (Holt 1966; Milner-Brown and Penn 1979; Sahrmann and Norton 1977), nor did drug or training induced amelioration of reflex hyperactivity necessarily lead to improved voluntary function (McLellan 1977; Neilson 1982).

More functionally oriented studies have emphasized the importance in movement control of central "programs" as well as feedback mechanisms. During stepping and hopping components of postural action anticipated rather than followed external perturbations (e.g., Melvill Jones and Watt 1971); supportive postural actions anticipated rather than followed voluntary movements (Belenkii et al. 1967; Marsden et al. 1977); and the patterns of anticipatory postural action were altered following changes in the perceived postural requirements (Marsden et al. 1981; Cordo and Nashner 1982). Somewhat akin to concepts of central "programmed" control of movement has been a systematic description of cerebral palsy movement abnormalities advanced by workers within the physiotherapeutic disciplines. This school of thought attributes some cerebral palsy abnormalities to the release from central inhibition of primative (inappropriate), movement patterns termed "synergies" (e.g., Bobath and Bobath 1964). However, because of large gaps in our understanding the interactions between spinal stretch reflex mechanisms and central programs, it has not been possible to integrate functional concepts of abnormal movement with what is understood physiologically about the stretch reflex abnormalities associated with cerebral palsy. The intention of this study has been to expand our functional understanding of normal and abnormal components of movement control in cerebral palsy towards a better description of the interactions between central programs and feedback mechanisms.

Methods

Subject Selection

Approximately 100 candidates between the ages of 7–9 years with a history of infantile cerebral palsy were screened from the Crippled Children's Division, Oregon Health Sciences University. The principal criteria for the eventual selection of ten children for study were the following: (1) normal or greater intellectual capacity, (2) no history of surgical intervention, (3) impairment very mild and stable by clinical standards to allow unaided stance and ambulation, (4) no visual or inner ear impairments, and (5) no current involvement in a therapeutic program. In addition to the above criteria, we sought children whose deficits by clinical standards were judged to the extent possible to be instances of pure ataxia, spastic hemiplegia, spastic diplegia, or athetosis rather than combinations of these problems. Before confirming our final selection of three ataxics, three spastic hemiplegics, three spastic diplegics, and one athetoid (we were unable to find three who fit our rigorous criteria), each selected child was given a thorough neurologic examination, the specific results of which were withheld from the principal investigator until platform tests were completed and experimental results analyzed. A general neurologic description of the children comprising each group is summarized in the Appendix. The following code is used to identify each CP child in the text (children in each group ordered numerically beginning with the most severely impaired); spastic hemiplegics (SH1–SH3), ataxics (AX1–AX3), spastic diplegics (SD1–SD3), and the athetoid (AT1). In addition to the ten CP children, 10 age matched normal children were tested using the identical platform test protocols.

Platform Test Procedures

All procedures for testing normal adults and children, and for analyzing and interpreting results were previously documented (Nashner 1971, 1976; Nashner and Berthoz 1978; Cordo and Nashner 1982; Forssberg and Nashner 1982). In addition, protocols have been applied to adults with stance equilibrium deficits due to peripheral vestibular dysfunction (Nashner et al. 1982) and to cerebellar deficits (Nashner and Grimm 1977). All protocols utilized an instrumented platform (Fig. 1) with independently movable support surfaces, visual surrounds, and handle. The support surface was comprised of two platforms each independently movable in horizontal translation, vertical translation, and rotation about an axis colinear with the ankle joint. The visual surround was 1 m square enclosure open on back and bottom sides with a rotational axis also colinear with the ankle joints. The handle could be positioned within the child's grasp while standing upon the platform and moved forward or backward. Strain gauges within each platform measured the torsional forces and the total vertical force exerted by the foot resting upon its surface[1]. Strain gauges in the handle measured total horizontal force exerted by the child during voluntary arm movements. A potentiometer attached about the child's hips measured angular changes in the antero-posterior (AP) sway orientation of the child's center of body mass with respect to the ankle joints.

The EMG and force components of automatic postural adjustments were assessed in the children by briefly displacing both support surfaces forward or backward for 250 ms causing principally ankle centered AP sway in the direction opposite that of the surface movement (Fig. 1A) (Nashner 1977). The velocity of the displacement was scaled to height of each child to produce sway at 20°/s. Perturbations during which the orientation information derived from the support surface was incorrect for initiating the AP sway correction were produced by rotating the two support surfaces at 20°/s for 250 ms. In these instances ankle joint rotation was at the same rate as that produced by the forward or backward displacements, but now ankle joint rotation was uncorrelated with AP sway (Fig. 1B). Because CP children tend to place weight unequally upon the two legs, vertical force upon each platform was monitored (and in some instances recorded) to assure that weight distribution was approximately equal during tests. Torsional moments were also monitored prior to each trial to assure that the heel of each foot was fully in contact with the support surface.

Procedures to disrupt the orientation information derived from the forces and motions from contact of the feet and lower leg musculature with the support surface (termed "support surface"

1 Because only constant velocity perturbations were used, measurement errors due to platform and handle inertia were apparent only during the initial 20–40 ms acceleration of the structure (see Fig. 8). Forces of interest in this study, however, occurred after 100 ms

inputs) and from vision were described previously (Nashner 1971; Nashner and Berthoz 1978; Nashner et al. 1982). "Support surface stabilization" (Fig. 1C) and "visual stabilization" (Fig. 1D) were each accomplished by rotating the surface in question to precisely follow the AP sway motions of the body center of mass, thereby eliminating rotational changes in orientation of the body center of mass with respect to the "stabilized" support or visual surface[2].

During platform induced postural adjustments, EMG activity was recorded bilaterally from four leg muscles, gastrocnemius, anterior tibialis, hamstrings, and quadriceps using pediatric surface electrodes spaced approximately 2 cm apart and bandpass amplification between 50 Hz and 5,000 Hz. During the performance of free standing arm movements, EMG signals from biceps and triceps were also recorded from the moving arm. A signal proportional to the intensity of activation of a given muscle was generated by full-wave rectification and then low pass filtration (0–40 Hz) of the raw EMG signal. The latency of an EMG response in a given muscle was defined as the time when the signal first deviated more than one and one-half standard deviations from the level recorded during a 100 ms interval prior to the stimulus (see Figs. 2–5)[3].

Parameters characterizing the temporal coordination of leg and arm muscle EMG responses were quantified by computing the relative response latencies of distal muscles and the functionally synergistic proximal leg and arm muscle EMG responses. The degree to which the contractile amplitude between pairs of proximal-distal synergists remained fixed was quantified by computing ratios of contractile amplitude. The amplitude of contraction of each muscle was quantified first by numerically integrating the processed EMG signal over a fixed 75 ms time interval beginning at the defined onset of response (see Nashner 1977; Nashner et al. 1979). EMG gains between distal-proximal synergist pairs of muscles were then normalized to give mean ratios of unity. We then quantified the trial to trial variations in the synergist ratios during each session. Parameters characterizing the degree of co-activation of distal antagonist muscles were quantified by computing for each distal leg muscle a ratio comparing its contractile amplitude under shortening conditions (i.e., backward sway for gastrocnemius) with that measured under conditions of its lengthening (i.e., forward sway for gastrocnemius).

The stability of a child standing unperturbed under altered sensory conditions was quantified by computing a "performance index" (PI). The AP sway trajectory was full-wave rectified, D.C. bias removed, and then numerically integrated over the 50 s duration of each trial. A number between 0 (no sway motion) and 1.0 (sway amplitude at limits of the feet together stance) was then determined by dividing the resulting integral by a number equivalent to AP sway oscillation at the limits of stability. PI values of 1.0 were arbitrarily assigned for those trials during which a child lost balance or was forced to step or stumble.

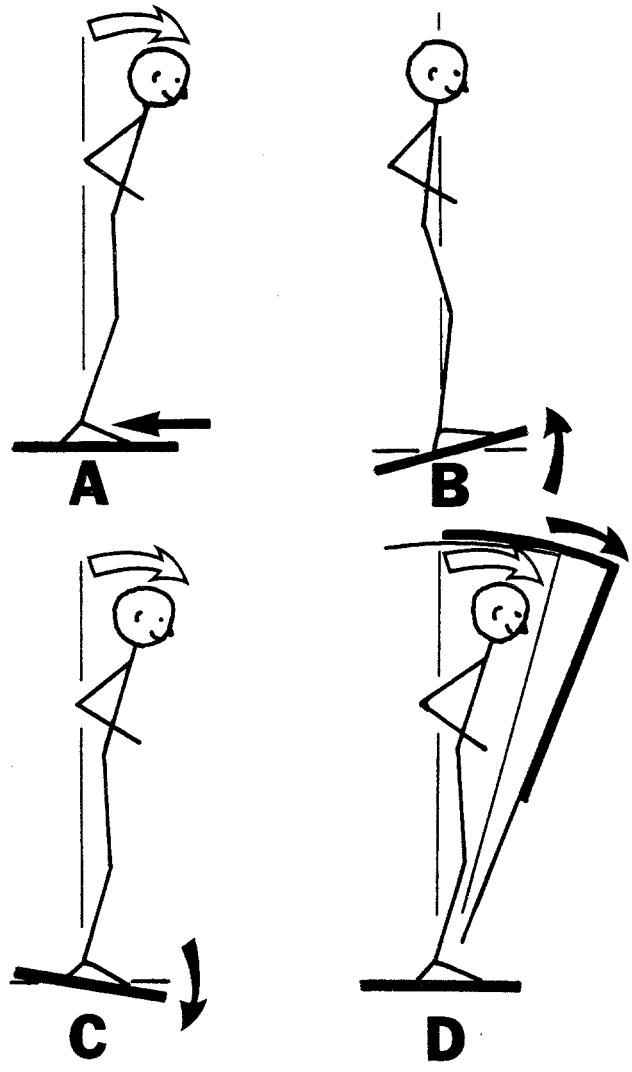

Fig. 1. The movable platform system: **A** Translating the platform support surface backward (or forward; *solid arrow*) induces AP sway centered primarily about the ankle joints and directed opposite that of the surface motion (*open arrow*). **B** Rotating the support surface toes up (or toes down; *solid arrow*) rotates the ankle joints in-place. **C** Termed "support surface stabilization". Rotating the support surface (*solid arrow*) in direct proportion to AP sway motions (*open arrow*) eliminates changes in orientation of the support surface relative to that of the center of body mass. **D** Termed "visual stabilization". Rotating the visual surround (*solid arrow*) in proportion to AP sway (*open arrow*) eliminates changes in orientation of visual surrounds relative to that of the center of body mass

Protocol

Each child was tested during at least four separate 1 h sessions, conducted during different weeks to assure repeatibility of observations over time. The first two sessions were devoted to the assessment of free-stance posture controls. The test protocol shown in Table 1 was followed in the first session and then reordered during the second to avoid anticipation by the child. Data from these two sessions were combined in presentation of

2 Stabilization of the support surface with respect to center of gravity motions would not completely eliminate rotation of the ankle joints in instances of knee-hip joint motions. However, relative motions about the knee and hip joints tend to be coordinated during stance to minimize changes in the position of the center of body mass (Gurfinkel et al. 1971). Hence that component of ankle joint rotation correlated with coordinated knee-hip motion most likely imparts little if any information about center of mass motions and therefore little information about balance

3 Using the above latency criteria, EMG responses to platform perturbations were seldom if ever observed in the children before 90 ms. Possible reasons for the absence of significant EMG components at myotatic stretch reflex latencies of 35–45 ms are addressed in the Discussion

Table 1. Test sequence for children

Type of test	Sensory conditions	Number and duration of trials
1. Performance	Normal (fixed) support and visual surfaces	2 ϱ 50 s
2. Performance	Normal support surface, eyes closed	2 ϱ 50 s
3. Performance	Normal support surface, stabilized vision	2 ϱ 50 s
4. Transient support surface translations	Normal support and visual conditions	5 forward ϱ 1 s
5. Transient support surface rotations	Normal support and visual conditions	5 "toes up" ϱ 1 s
6. Transient support surface translations	Normal support and visual conditions	5 backward ϱ 1 s
7. Transient support surface rotations	Normal support and visual conditions	5 "toes down" ϱ 1 s
8. Performance	Stabilized support surface, normal vision	2 ϱ 50 s
9. Performance	Stabilized support surface, eyes closed	2 ϱ 50 s
10. Performance	Stabilized support and visual surfaces	2 ϱ 50 s

results. The initial three tests in the Table 1 protocol quantified performance during quiet stance with a non-moving support surface under three different visual conditions (normal, eyes closed, stabilized vision). Tests 4–7 examined the structural and adaptive properties of automatic postural adjustments elicited by brief displacements of the support surfaces as the child stood with eyes open. Tests 8–10 re-examined the stance stability of each child under the same three visual conditions as used in tests 1–3, except now orientation information derived from the support surface was disrupted by "stabilizing" the support surface with respect to the sway motions of the center of body mass.

During the third and fourth 1 h sessions, the coordination of postural support with voluntary arm movements was assessed applying a protocol developed previously (Cordo and Nashner 1982). The child grasped a movable handle while freely standing and, according to prior instruction, pulled or pushed the handle as rapidly as possible upon hearing a tone. In order to remain upright while performing these arm movements, stabilization of AP sway orientation in opposition to the force exerted upon the handle was necessary. The configuration of the arm movements was chosen such that groups of leg muscles required for postural stabilization were similar to those stabilizing platform-induced AP sway disturbances.

Results

Alterations in Parameters of Muscle Coordination Associated with Spastic Hemiplegia

During both the platform-induced postural adjustments and the postural adjustments which anticipated voluntary arm maneuvers against the handle, the spastic legs of the three hemiplegic children expressed significant alterations in two parameters characterizing coordination between distal and proximal synergist pairs of muscles. Compared to the activation patterns in normal leg muscles the temporal order in which distal and proximal synergists were activated in spastic legs was reversed. Furthermore, the relative strength of contraction of distal and proximal synergists tended to be significantly more variable in muscles of the spastic legs. These same two coordination parameters were within limits established for normals in the clinically non-involved legs of the three spastic hemiplegic children. However, we did note coordination problems in the non-involved as well as the spastic legs of the three spastic hemiplegics during platform perturbations requiring reciprocal action between the two legs (one platform displaced upward, the other downward, see Nashner et al. 1979). Although these additional observations have not been reported in detail here, they were the basis for our henceforth adopting the terms "less-involved" and "spastic" to distinguish the two legs of the spastic hemiplegic children. In contrast to the coordination patterns of the spastic hemiplegic child, we found that the temporal order of synergist activation and the relative strength of synergist contractions were both within limits established for normals in the legs of the ataxic children, although this group consistently performed abnormally during the sensory organization tests.

Our method for computing temporal and spatial parameters of muscle coordination is illustrated in Fig. 2, which compares ensemble averaged EMG records of the less-involved and the spastic legs in child SH1 during responses to forward sway perturbations (platform displaced backward). In the less-involved leg the adjustments commenced with contraction of the stretching gastrocnemius muscle at a mean latency of 97 ms (5 ms SD). Mechanically coupled motions of the hips were stabilized by contraction of the synergist hamstrings muscle beginning on the average 26 ms later (12 ms SD) than the gastrocnemius. The sequence of muscle activation beginning distally at the base of support and radiating proximally away from the support is highlighted in Fig. 2 by the rightward pointing arrow relating the relative latencies of gastrocnemius and hamstrings muscles, while the relative strengths of gastrocnemius and hamstrings contractions during the first 75 ms of response (numerical integral of EMG signals) are illustrated by the shaded areas. This

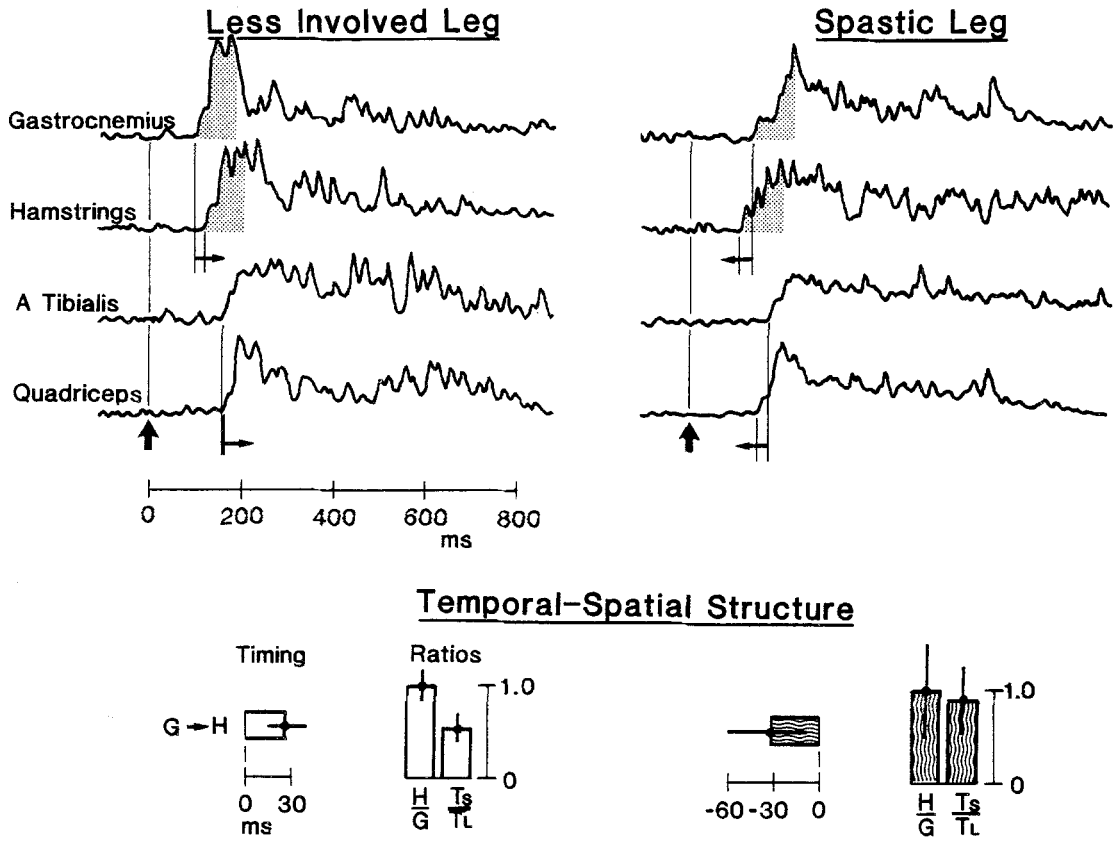

Fig. 2. EMG responses and temporal-spatial structure of muscles in less-involved and spastic legs of child SH1 (ensemble average of ten trials) in response to forward AP sway perturbations (platform surface translated backwards). *Arrows* indicate the sequence of activation of distal and proximal synergists; and *shaded portions,* the 75 ms intervals subjected to analysis of temporal-spatial structure. The timing graphs (open for less involved leg and shaded for spastic leg) indicate the relative latency (± SD) of gastrocnemius and hamstrings activation (positive values indicate gastrocnemius first). Ratio graphs indicate: (1) trial to trial variations in the relative strength (± SD) of gastrocnemius-hamstrings synergists (H/G), (2) strength (± SD) of anterior tibialis antagonist (T_S/T_L) contractions under shortening (forward sway) versus lengthening (backward sway) conditions

temporal and spatial structuring of EMG response to forward sway perturbations is the same as that observed previously in studies of normal adults (Nashner 1977) and normal juveniles aged 1½ to 10 years (Forssberg and Nashner 1982).

The pattern of contraction within muscles of the spastic leg shown in Fig. 2 was significantly different than that described above. Latency of gastrocnemius response averaged a slower 145 ms (13 ms SD), and the sequence of activity was temporally reversed having commenced in the hamstrings an average of 31 ms (25 ms SD) earlier. This reversal in the temporal order of activation is indicated by the negative timing value and by the leftward pointing arrow relating relative latencies of gastrocnemius and hamstrings muscles. Note that subsequent activation of the anterior tibialis and quadriceps muscles, antagonists which helped brake the return sway movement, were sequenced in the non-involved leg beginning at base of support and then radiating upward, while the reverse sequence of antagonist activation was again observed in muscles of the spastic leg.

The two parameters of muscular coordination used to characterize the above described patterns of EMG activity are introduced under the "Structure" headings in Fig. 2. The positive "timing" values of the less-involved leg indicate that activity commenced in the ankle joint muscles (closest to base of support) and then radiated proximally to the upper leg synergists. In contrast, the negative values of spastic leg contractions indicate that the opposite sequence of activation occurred. Under the "ratio" subheading, the standard deviation of the mean H/G ratio quantifies the degree of consistency in the relative activation strengths of distal-proximal synergists during the initial 75 ms of response. A second measure of spatial coordination, the T_S/T_L ratio, characterizes the level of co-activation of the anterior tibialis ankle muscle by comparing its response under

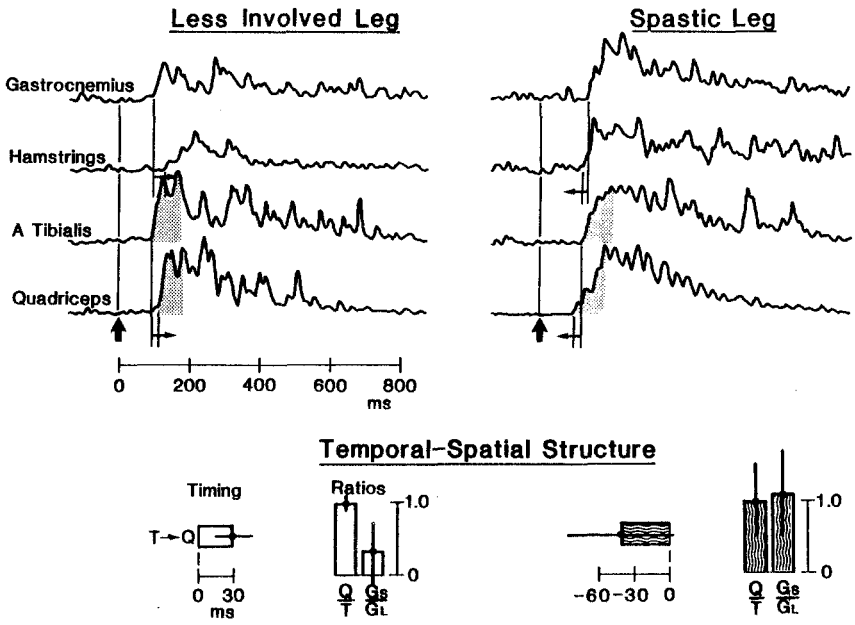

Fig. 3. EMG responses and temporal spatial structure of child SH1 (ensemble average of ten trials) to backward AP sway (platform surface translated forward). Organization of material identical to Fig. 2. Now anterior tibialis-quadriceps are synergists and the gastrocnemious is the distal antagonist

this shortening condition with that observed under conditions of its lengthening (i.e., backward sway). Compared to the less-involved leg (open bars), the linkages between synergists in the spastic leg were 3½ times more variable (larger SD of H/G ratio), and the level of co-activation of the antagonists was over twice as great (larger T_S/T_L ratio).

When parameters quantifying the temporal and spatial structure of automatic postural adjustments to backward sway perturbations (platform displaced forward) were distilled from the EMG records of child SH1, a similar distribution of normal and abnormal parameters was found (Fig. 3). Compared to the less-involved leg, the temporal order of activation in the spastic leg was reversed, the linkage between synergists was much more variable, and the level of antagonist co-activation was greater.

The imposition of support surface rotations and the voluntary exertion of force against the hand-held manipulandum were two additional paradigms designed to assess parameters of muscle coordination. Postural adjustments elicited by in-place rotation of the ankle joints commenced at similar latencies but in this instance occurred in the absence of congruent visual and vestibular inputs. In the case of voluntary pulls upon the handle, postural responses in the leg musculature anticipated the disturbance associated with the pull. Figure 4 compares the less-involved and the spastic leg EMG records of child SH1 to "toes up" and "toes down" support surface rotations, stimuli which at the ankle joint corresponded to those produced by forward sway and backward sway perturbations, respectively. Figure 5 compares the leg and arm EMG records of the same spastic hemiplegic child (SH1) during tone-triggered voluntary pulls and pushes upon the handle, movements which displaced the body center of mass forward and backward, respectively, but accompanied by a very different configuration of sensory inputs. Despite gross differences in the sensory events which preceded the postural adjustments in these two instances, the coordination parameters illustrated in Figs. 4 and 5 correspond to those observed during the automatic postural adjustments to the equivalent forward and backward sway perturbations. In contrast to the less-involved side of the body, activation sequences of spastic leg and arm muscles were in all instances temporally reversed. On the spastic side during platform surface rotations, the proximal muscles were activated in advance of their distal synergists, even though the stimuli in these instances consisted of isolated stretch of the distal ankle joint muscle. Similarly, leg muscles on the spastic side did not respond in anticipation of postural disturbances caused by voluntary arm pulls and pushes.

The computational procedures applied to the EMG records of the first hemiplegic child (Figs. 2–5), were reapplied to determine the group mean values of the temporal and spatial parameters of coordination for the ten normal children (data from two sides lumped), the three spastic hemiplegics (less-involved and spastic sides shown separately) and the three ataxic children (data from two sides lumped). As shown in Fig. 6, there were significant differences among normals and the two clinical categories of CP

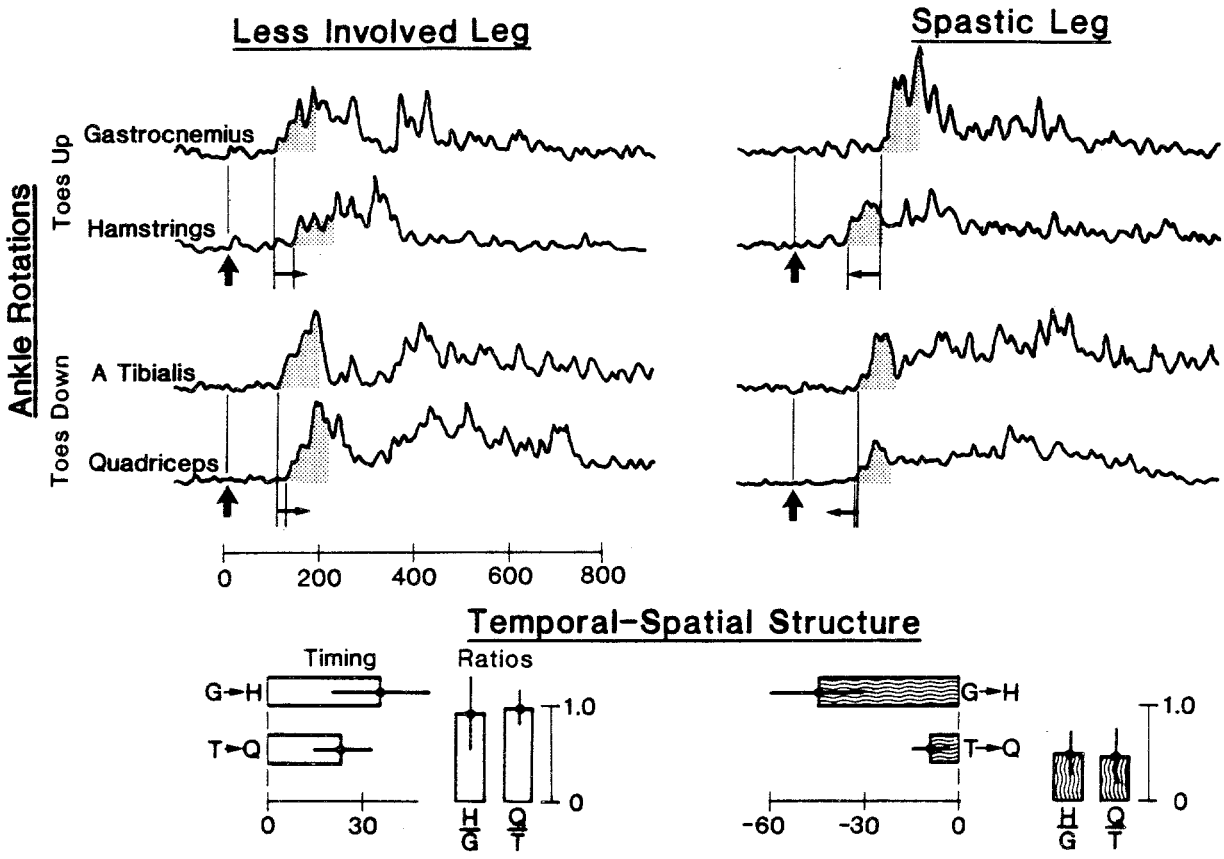

Fig. 4. EMG responses and temporal-spatial structure of child SH1 (ensemble average of ten trials) to rotations of the platform surface. Only the distal-proximal synergists muscles are presented in each case. Organization of material is otherwise identical to Figs. 2 and 3

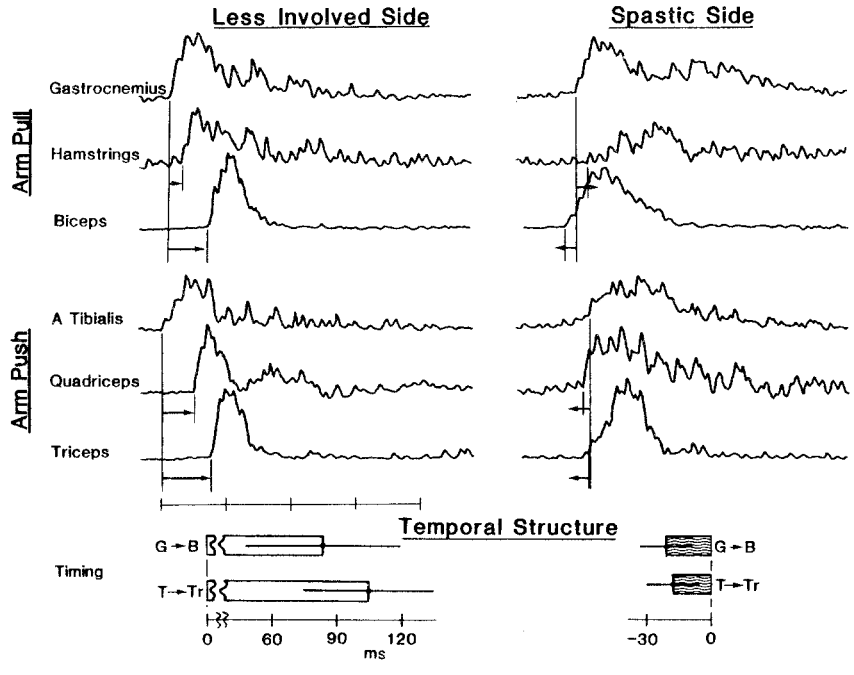

Fig. 5. EMG responses and temporal structure of child SH1 during voluntary pushes and pulls upon the handle during free stance

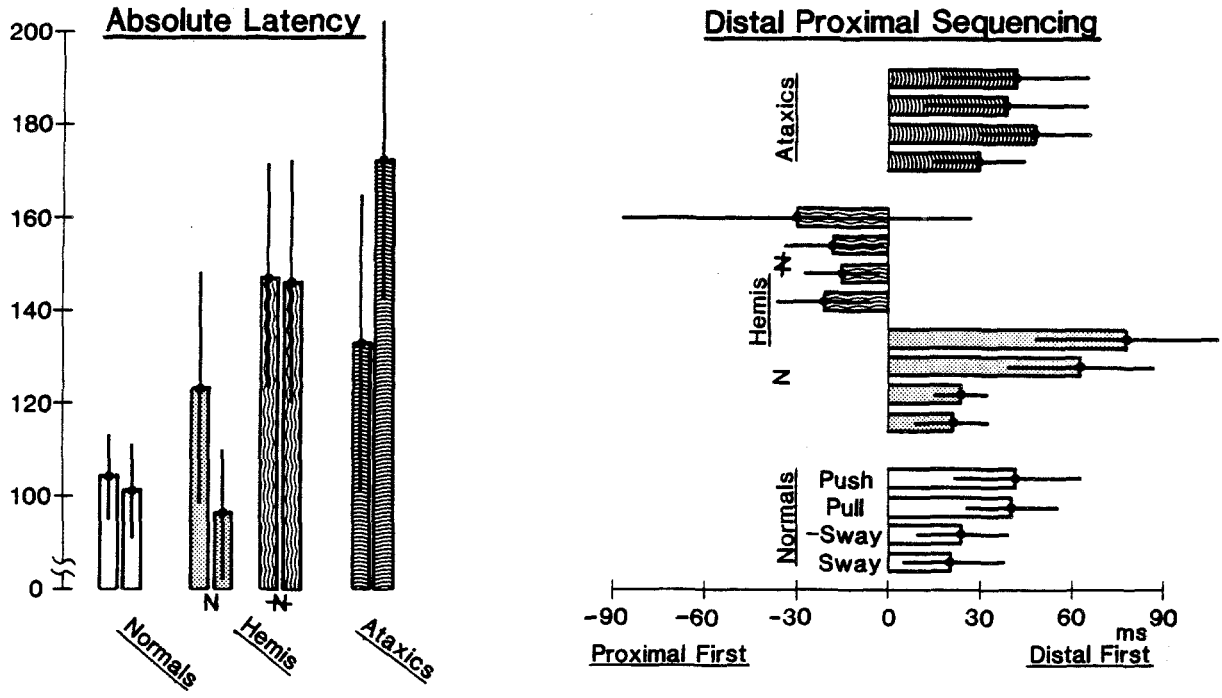

Fig. 6. Temporal structure of postural responses of the ten normals, the three spastic hemiplegics (less involved [N] and spastic [N] sides shown separately), and the three ataxics (average ± SD for each group). Absolute latencies characterize earliest EMG responses of ankle joint muscles to forward AP sway perturbations (left-hand bar in each group) and to backward AP sway (right-hand bar). Distal to proximal sequencing of synergists for each group are shown separately for forward (plus) sway, backward (minus) sway, arm pulls and arm pushes

children in the absolute latency of at which EMG responses first occurred in ankle joint muscles following support surface displacements. However, these absolute EMG latency measures did not clearly distinguish among the categories of CP children. With the exception of anterior tibialis responses in the less-involves legs of the three spastic hemiplegics, absolute latencies of automatic postural adjustments were significantly longer than normal (0.01 level; one-way analysis of variance used in this and all succeeding tests) in all groups of CP children, although response delays and trial to trial variations in latency were greatest in the three ataxics and in the spastic legs of the three hemiplegics.

Another observation apparent from assessment of absolute latencies was the almost complete lack of EMG responses observed at myotatic stretch reflex latencies (i.e., 35–50 ms latencies). For example, the reader might note a very small increase at 40 ms in the averaged gastrocnemius EMG record of the less involved leg in Fig. 2. However, this increase as well as those seen occasionally in the stretching ankle joint muscle of other children were extremely small in proportion to the later vigorous contractions and always insignificant according to the latency criteria outlined in Methods.

In contrast to absolute latency measurements, the sequencing of muscle activity was a highly significant parameter in distinguishing patterns of activity in limbs judged spastic by clinical standards (Fig. 6). Reversals in the normal distal to proximal sequence of leg and arm muscle activation were found statistically significant (0.001 level), but only on the spastic muscles of the three hemiplegic children. In contrast, there was normal distal to proximal sequencing of leg and arm muscles in the ten normals, the three ataxics, and in the less-involved legs of the three spastic hemiplegic children.

The spatial (ratio) parameters of coordination shown in Fig. 7 were also useful in isolating abnormal patterns of muscle activity. Variations in the amplitudes of contraction between distal and proximal synergists were greatest and the co-activation of ankle joint antagonists most prominent in the spastic leg muscles of the three hemiplegic children. The standard deviations of the H/G and Q/T ratios for the spastic leg muscles were significantly larger (0.05 level) compared to the ratio variations of normals, ataxics, and of the less-involved muscles of hemiplegics. Levels of antagonist co-activation of spastic leg muscles (T_S/T_L and G_S/G_L ratios) were also higher compared to normals, ataxics, and the non-involved

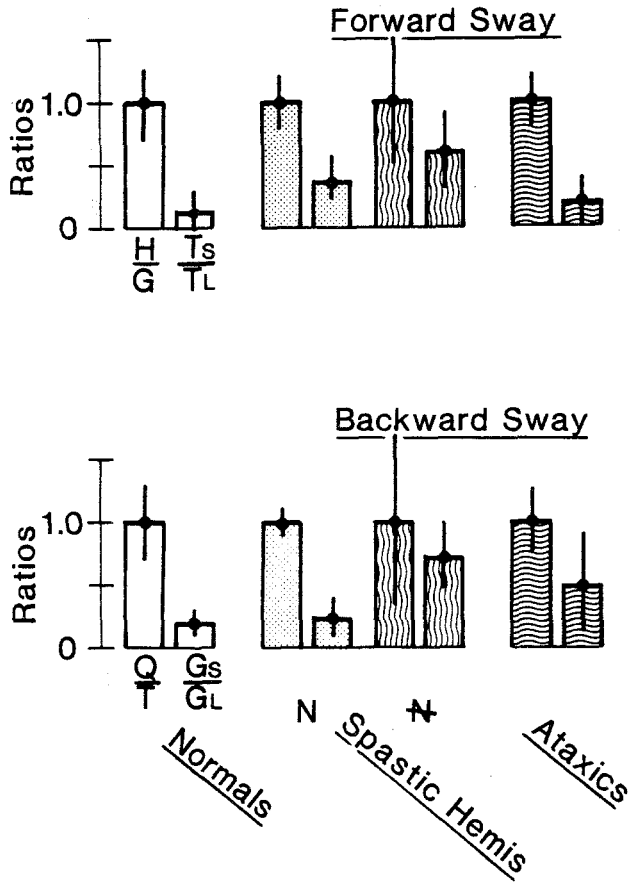

Fig. 7. Spatial structure of postural responses to forward and backward AP sway perturbations in normals, spastic hemiplegics (less involved (N) and spastic sides (N) shown separately), and ataxics. During forward AP sway, the gastrocnemius-hamstrings ratio (± SD) shows the degree of fixation of activity in synergists, while the anterior tibialis ratio of strengths under shortening (forward) versus lengthening (backward) conditions shows the degree of antagonist co-activation. During backward sway, the anterior tibialis-quadriceps ratio (± SD) measures fixation of synergist activation, while the gastrocnemius ratio of strengths under shortening (backward) versus lengthening (forward) condition quantifies the degree of antagonist coactivation. Relative gains of synergist muscle pairs were normalized to give mean ratios of unity. Thus, the standard deviation of a ratio characterizes the extent to which it remains constant over the series of trials

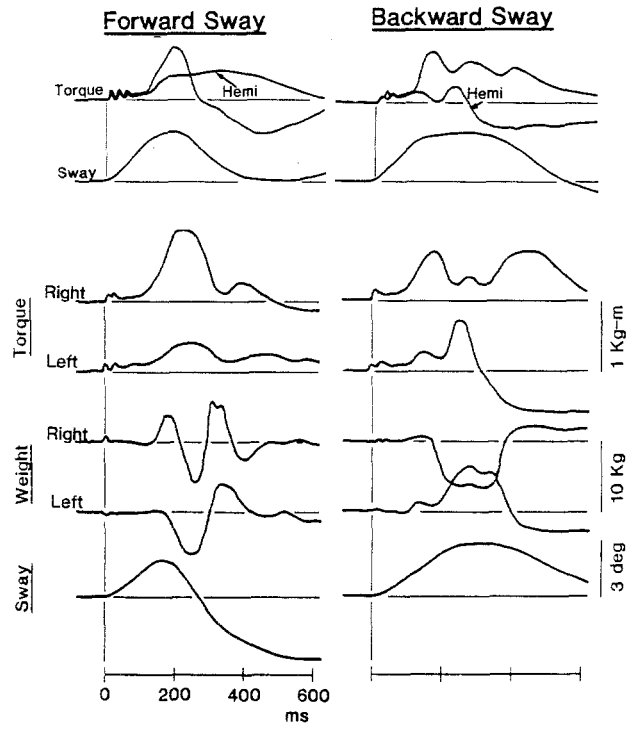

Fig. 8. Top traces: The mechanics of AP sway adjustments in child SH1 (EMG's shown in Figs. 2–5) to forward and backward AP sway perturbations. Separate ankle torque records for less involved and spastic hemiplegic legs and the AP sway record are ensemble averages of ten trials. Bottom traces: Mechanics of AP sway adjustments in child SH2 (left spastic hemiplegic) to forward and backward AP sway perturbations. In addition to torque and sway records, weight carried by left and right legs is shown (ensembles average of ten trials)

side of hemiplegics, although the level of significance of this effect was low in the case of backward sway. The level of antagonist co-activation in response to backward sway perturbations was abnormally high not only in spastic limbs but also in both limbs of the three ataxics. However, increased co-activation of antagonists during backward sway was also noted in younger normal children (Forssberg and Nashner 1982) and in adult vestibular patients with sensory rather than motor related balance difficulties (Nashner et al. 1982).

Biomechanical Consequences of Activation from Base of Support

The torques exerted by each leg of child SH1 during forward and backward sway perturbations and his AP sway trajectories are illustrated in Fig. 8 (top traces). Although EMG responses were approximately as strong in both legs (identical electrode configurations and amplifier gains) and sway displacements were quickly and accurately compensated by this child, torque traces show that nearly all of the muscular effort to achieve the correction was exerted by the less-involved leg. In contrast, even though the spastic foot was placed flatly upon the support surface and at the outset of each trial carried an approximately equal share of the vertical load (torsional and vertical forces monitored prior to each trial), the torque trajectory of the spastic leg showed little if any correlation with the AP sway motion. Figure 8 (bottom traces) shows ensemble averaged mechanical components of automatic postural adjust-

ments to forward and backward AP sway in spastic hemiplegic child (SH2) for whom vertical forces as well as torsional movements and AP sway trajectories were recorded. In addition to showing lack of torque generation in the spastic leg, the vertical force traces of this child recorded large, oscillatory changes in distribution of weight bearing between the two legs during automatic postural adjustments. Because these oscillatory weight shifts commenced with the onset of the postural adjustment and were much too rapid (completed within approximately 200 ms) to be attributed to lateral shifts in body center of mass (natural frequency of lateral body sway approximately 0.3 Hz), these rapid shifts were most probably a direct consequence of the abnormally coordinated muscular contractions. We observed rapid oscillatory weight shifts in two of the three hemiplegic children SH1 and SH2. In contrast, there were no significant weight shifts (less than ± 0.75 kg variation in vertical force throughout trials) among five normals and two ataxic children for whom the vertical forces measures were also recorded. To be introduced in the Discussion is the suggestion that the lack of torsional resistance and the emergence of oscillatory shifts in weight are both consequences of reversals in the normal distal-to-proximal sequencing of muscle activation.

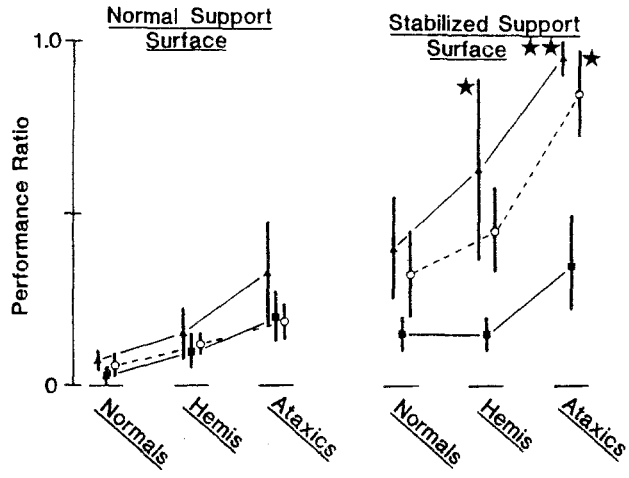

Fig. 9. Performance ratios for quiet stance under various sensory conditions for the ten normals, the three spastic hemiplegics, and the three ataxics (average ± SD for each group). Ratios nearer to unity indicate increasingly unstable stance. *Stars* indicate numbers of children in each group who lost balance. Eyes open within a fixed visual surround (*filled squares*), with eyes closed (*open circles*) and with eyes open within a "stabilized" visual surround (*filled triangles*)

Alterations in Parameters of Sensory Organization Associated with Ataxia

Although the three ataxic children exhibited normal distal to proximal sequencing, a high degree of consistency in the relative strengths of distal-proximal contractions, and with one exception a low level of antagonist coactivation (see Fig. 7), their ability to accurately maintain upright equilibrium was significantly poorer than either the normal or the spastic hemiplegic groups, especially when these three groups are compared under altered sensory conditions. Figure 9 compares the amplitude of AP sway in the normal and two clinical subgroups of CP children as they stood unperturbed under six different sensory conditions. The left-hand axis of the figure shows mean PI values (± SD) achieved under three different visual conditions while standing upon a fixed support surface. The right-hand traces show the PI values achieved for the same three visual conditions while standing upon a "stabilized" support surface (eyes open, closed, or open within a visual surround also "stabilized" by rotating it to precisely follow the AP sway motions of the child's center of mass). Compared to values achieved by normals and spastic hemiplegics, the mean PI values for the three ataxics were greater under all conditions. However, when provided with a fixed support surface, the mean PI values achieved by all groups were well below the limits of stability for feet-together stance.

When "stabilization" of the support surface with respect to the AP sway rotations of the body center of mass disrupted the sway-related inputs derived from the support surface and therefore, to some extent, rapid automatic postural adjustments mediated by these inputs, the three ataxics on the average were much less stable than either the ten normals or the three spastic hemiplegics (right-side traces). Differences in the mean PI values between the normals and the hemiplegics performing with support surface "stabilized" and eyes either open or closed were not statistically significant. In contrast, the mean PI values achieved by the three ataxics (2 × normal with eyes open and 3 × normal with eyes closed) were significantly greater than those achieved by either normals or spastic hemiplegics (0.01 level, both instances). As indicated by the "star", one of the three ataxic children AX2 lost balance when attempting to perform upon the stabilized support surface with eyes closed.

When standing upon the "stabilized" support surface and reopening their eyes within a visual surround also "stabilized" with respect to the AP sway motions of the body center of mass, children in all three groups were less stable than when performing the same task with eyes closed. However,

Table 2. Spastic diplegics: Parameters of coordination

Child	Absolute latencies		Temporal structure			
			G→H		T→Q	
	G(+sway)	T(−sway)	Left	Right	Left	Right
SD1	121 ± 12	110 ± 15	25 ± 11	16 ± 8	(1)(4)	(1)
SD2	109 ± 5	101 ± 5	19 ± 9	22 ± 21	3 ± 17	−4 ± 22
SD3	165 ± 28	150 ± 16	35 ± 23	39 ± 19	28 ± 16	44 ± 30

Spastic diplegics: Sensory Organization

Child	Performance ratio					
	S_NV_N(5)	S_NV_C	S_NV_S	S_SV_N	S_SV_C	S_SV_S
SD1	0.14	0.09	(2)	0.28	1.00(3)	1.00(3)
SD2	0.11	0.04	(2)	0.15	0.38	0.33
SD3	0.12	0.05	0.19	0.36	0.46	1.00(3)

(1) Loss of directional specificity; not possible to quantify
(2) These measurements were not made
(3) Lost balance on these tests
(4) Underlines indicate abnormal parameters
(5) "S" is the support surface and "V" the visual condition. "N" is normal, "c" is eyes closed, "s" is stabilized

increases in sway amplitudes associated with the introduction of an orientationally incorrect visual input were statistically insignificant in normals. The 40% increase in mean PI of the three spastic hemiplegics was due entirely to the fact that one of the three children in this group SH2 lost balance upon his first attempt. Spastic hemiplegics SH1 and SH3 performed equally well as had the normals with the support surface stabilized and eyes either closed or open within a stabilized visual surround. Thus, the spastic hemiplegic child SH2 demonstrated a mixed configuration of abnormalities, including not only marked alterations in muscle coordination but also alterations in sensory organization less severe but qualitatively similar to those expressed by the three ataxics. PI values of ataxics exposed to concurrently "stabilized" support surface and visual surround were consistently the largest of all children; ataxics AX1 and AX2 repeatedly lost balance under this condition while AX3 remained precariously close to doing so.

Different Combinations of Muscle Coordination and of Sensory Organization Abnormalities in Spastic Diplegics

Results of the spastic diplegic and athetoid children are summarized in Table 2. Among the three spastic diplegic and one athetoid child, several different configurations of abnormal parameters were noted. In spastic diplegic SD2, abnormality was limited to parameters of muscular coordination. Thus, this child resembled the three spastic hemiplegics with the exception that abnormalities were distributed more bilaterally. In child SD2, the temporal sequence of activation leg synergists was highly variable and frequently reversed in both legs, but only in muscles compensating backward sway. However, this child performed within normal limits in sensory organization tests when exposed to stabilization of the support surface and visual surrounds. In spastic diplegic SD3, abnormal parameters were more apparent in sensory organization; the performance of this child most closely resembled that of the three ataxics. In child SD3, absolute latencies of automatic adjustments were delayed and highly variable; the child lost balance when exposed to simultaneously "stabilized" support surface and visual inputs; and yet, the sequencing of activity in leg synergists was distal to proximal as in normals, although ratio data showed variability in the synergic coupling between proximal and distal muscles.

In the remaining two children, SD1 and the single athetoid AT1, abnormalities included both coordination and sensory organization. In addition to the forms of muscular coordination and sensory organization abnormalities already described, a parameter of muscular coordination not found altered in any of the preceding groups was abnormal in one leg in each of these two children. The pattern of muscular contraction was virtually identical in response to platform displacements of different waveforms and directions. In other words, there was a loss of directional specificity. To illustrate loss of directional

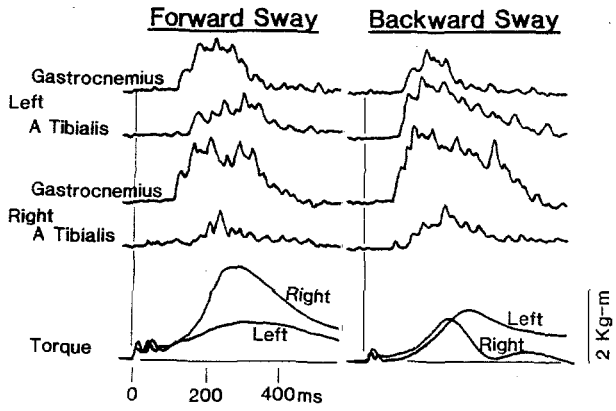

Fig. 10. The loss of directional specificity in the right leg of spastic diplegic child SD1 in response to forward and backward AP sway perturbations. The child stood with eyes open with normal visual inputs. Upper traces show EMG records of ankle joint muscles and the resulting torque exerted by each leg. Both measures are the ensemble average of ten trials

specificity, Fig. 10 compares ensemble averaged EMG responses of the distal ankle musculature of child SD1 during forward and backward AP sway. In left leg muscles, the relative amplitude of gastrocnemius and anterior tibialis is appropriate to effectively resist rotation of the ankle joints during both forward and backward sway perturbations. In contrast, the relative amplitudes of right leg gastrocnemius and anterior tibialis contractions are almost identical during both forward and backward perturbations. Loss of directional specificity in the right leg is further indicated by the torque response records; resistance to sway provided by this leg was exceptionally strong during forward sway but was ineffective following onset of the automatic postural response during backward sway. In contrast, the left leg exerted approximately equal and functionally useful levels of torsional resistance in both sway directions.

Discussion

Our intent in carefully analyzing the postural controls of a small but highly selected group of cerebral palsy children and an equal number of age-matched normals was to begin the development of a systematic framework for understanding the genesis of abnormal movement control in children. Specifically, studies focused upon the ability of cerebral palsy children to establish, in advance of the execution of discrete postural actions, strategies for organizing sensory inputs and for coordinating activities of leg and arm muscles which were appropriate for the given environmental context. In seven of the ten cerebral palsy children abnormalities were focused primarily within either muscle coordinating or sensory organizing processes, while three of the children showed abnormalities which reflected problems in both processes. Platform experiments and clinical assessments tended to separate the cerebral palsy children into similar subgroups: Children described clinically as pure spastic hemiplegics expressed abnormalities in muscle coordination but were not destabilized by sensory conflicts, while those clinically described as pure ataxic responded inappropriately to sensory inputs under conflict conditions and yet always coordinated muscular actions appropriately. Furthermore, clinical and experimental descriptions of the cerebral palsy children tended to agree in their assessments of the relative severity of deficit (often for different reasons). The least symptomatic spastic hemiplegic and ataxic children were also those whose parameters of performance were closest to age-matched normals, while the two cerebral palsy children judged clinically the most symptomatic were the ones expressing abnormalities in both muscular coordination and sensory organization.

Pathophysiology of Sensory Organization Disorders in Cerebral Palsy

When normal adults were exposed to platform surface motions which unexpectedly altered the orientation information derived from the support surface and from vision, the strategy for resolving the resulting conflicts was a fixed hierarchical one in which inputs discongruent with the inertial-gravitational reference provided by the vestibular system were suppressed within three to five trials during a sequence of transient perturbations (Nashner 1976; Nashner and Berthoz 1978). In contrast, normal children under 6 to 7 years of age and adults with subtle peripheral vestibular losses lost their balance under sensory conflict conditions because they responded frequently to perceptually correct but orientationally incorrect visual and support surface inputs (Nashner et al. 1982; Forssberg and Nashner 1982). Nevertheless, when normal children under 6 years of age and adults with peripheral vestibular losses did respond to perturbations, the temporal-spatial structure of muscular actions was not distinguishable from that of normal adults. In their inability to balance under conflict conditions the three CP children diagnosed as primarily ataxic and one of the three spastic diplegics, SD3, closely resembled the adults with peripheral vestibular disorders and normal children under 6–7 years.

Although there is reason to believe that standard clinical tests for assessing peripheral vestibular function in posture control are inadequate (Black et al., in press), there was no direct clinical evidence suggesting that normal children under 6 years or the older ataxic cerebral palsy children included in this study suffered peripheral vestibular abnormalities; the clinical summaries of all three ataxics clearly stated the absence of inner ear or visual abnormalities. However, the two children not diagnosed as purely ataxic who nevertheless showed significant sensory organization deficits (SH2 and SD3) also presented significant sensory and perceptual deficits in the clinical examination. Hence, there may be at least two different physiological substrates for the sensory organization disorders among cerebral palsy children; one being delay or disruption of the normal ontogenetic development of central sensory organizing mechanisms and the other evolving disruption of peripheral sensory inputs.

Pathophysiology of Muscular Coordination Disorders in Cerebral Palsy

The temporal and spatial structure of the postural actions of adults was fixed with respect to the configuration of external support independent of the way the postural actions were initiated; either following a variety of external perturbations or anticipating the disturbances caused by self-initiated arm movements (Nashner 1977; Nashner et al. 1979; Cordo and Nashner 1982). Specifically, patterns of action were characterized temporally by the sequencing of activation beginning in muscles perceived closest to the base of support and spatially by the proportionate activation of ankle and hip synergists. In children categorized clinically as pure spastic hemiplegics, we have observed in this study a breakdown in those processes which according to the configuration of external support fix the temporal and spatial structure in advance of discrete postural actions. In the spastic legs of the three hemiplegic children as well as in both legs of two of the three spastic diplegics (SD1 and SD2) and the athetoid (AT1), muscular actions were initiated in either proximal leg or in arm muscles first and the proportional relations among muscle synergists were significantly more variable. While these observed changes in muscle coordination seem consistent with the observed functional abnormalities of the spastic children, current physiological assumptions about the role of stretch reflex mechanisms in support of controlled movement and about the pathophysiology of reflexes in spasticity are not consistent with our observations.

Implicit in many of the studies of reflex pathology cited in the Introduction was the assumption that elevated stretch reflexes increased resistance to and therefore interfered with the execution of controlled movements. Based upon this assumption, one might have predicted that the spastic ankle joint muscles in our cerebral palsy subjects would have been unusually responsive to perturbation-induced stretch during stance. However, despite the fact that "deep tendon reflexes" of gastrocnemius muscles in the spastic legs of the three hemiplegic children were judged elevated in clinical examinations, responses of gastrocnemius to stretch during forward AP sway perturbations were nevertheless sufficiently delayed (150 ms on the average compared to 95–110 ms in the normals) to allow the synergist hamstrings to contract first, thereby reversing the normal distal to proximal sequence of contraction. Furthermore, these sequence reversals could not be attributed to elevated hamstrings stretch responses, since response latencies of hamstrings if anything were also slightly delayed (0–10 ms on the average) compared to the normals (Figs. 2 and 6). The finding that postural responses in distal ankle muscles of spastic limbs were abnormally delayed relative to their proximal synergists was particularly surprising during responses of standing spastic hemiplegic children to in-place ankle joint rotations (Fig. 4), in as much as the spastic gastrocnemius muscles received a direct, isolated stretch input during these trials.

Several previous experimental observations on reflex posture control support our contention that pathological alterations in the excitability in the myotatic stretch reflex pathways of leg muscles would play a relatively smaller role in the overall postural problems of freely standing spastic patients when compared to the role played by alterations in the longer latency automatic postural components. Observations of stretch reflex function in freely standing subjects suggested that myotatic reflex excitability levels are significantly reduced compared to the levels typical during ankle movement tasks performed while seated (Gurfinkel et al. 1976; Nashner 1976). However, the above two studies used only slow rates of ankle joint rotation (less than 50 degrees/s), and the possibility remained that myotatic stretch reflexes could be elicited in the freely standing subject by imposing higher rates of stretch. Recently, Dietz and Berger (1982) showed that in freely standing subjects the earliest EMG responses in the tibialis anterior muscles subjected to stretch at 400–600 deg/s (exact rates unspecified but said to be 20–30 times faster than our 20 deg/s rate) were 50–60 ms. Furthermore, they argued that these earliest responses were not myotatic because they occurred

bilaterally depending upon the distribution of weight between the legs when only one anke joint was rotated. In contrast, several groups reported vigorous myotatic stretch reflex responses in the ankle joint musculature of seated individuals subjected to disturbances producing approximately 200 deg/s rates of ankle joint rotation (e.g., Gottlieb and Agarwal 1979; Kearney and Chan 1982). Unfortunately, however, Dietz and Berger (1982) did not report the effects of gastrocnemius stretch at 400–600 deg/s rates in freely standing subjects, since the possibility remains that, due to asymmetries in myotatic reflex excitability levels, a myotatic stretch reflex as well as the later bilaterally organized component would have been observed. What Dietz and Berger's (1982) observations do clearly point out is the fact that latencies of the more centrally organized automatic components of posture control are probably not fixed but dependent upon stimulus parameters. Woollacott et al. (1980) showed that postural set can also affect latencies of the automatic postural components, reporting that automatic response latencies to platform horizontal displacements in some subjects decreased from 95–110 ms to 60–70 ms when the identical waveform was repeated 20 times.

Although quantitative information about the distribution of tonus in the spastic hemiplegic children during stance was limited, we could not attribute the delayed activation of distal leg muscles to abnormally depressed levels of background activity. For one example, in Figs. 2 and 3, gastrocnemius and anterior tibialis responses were significantly slower in the spastic legs of SH1, even though there was measurable background EMG activity in both of these muscles prior to the imposition of AP sway. As a second example, spastic diplegic SD2 was clinically described as having "generalized" extensor hypertonus while standing; nevertheless, we observed delays in gastrocnemius and hamstrings responses and sequence reversals in the activation patterns of this child. In several other unpublished observations of adults with more dramatically elevated extensor tone in spastic legs secondary to multiple sclerosis, we also observed reversals in the distal to proximal activation of leg muscles during automatic postural adjustments.

Our results support the contention that pathological changes in stretch reflex mechanisms, rather than being an inherent cause of the ineffective movements of the spastic leg during stance, are instead secondary to the primary abnormality, alterations in central and spinal programs which in advance of a motor action normally impose the appropriate temporal and spatial structure upon motor activities in synergistic groups of muscles. Studies of the treadmill-walking cat preparations indicate that the neural circuitry within the spinal cord (of the cat at least) is sufficient to generate stereotyped, coordinated limb movements and reflex postural corrections which are at least as complex as those described here (e.g., Grillner 1975). Due to delay in the development of descending pathways, stepping movements of newborn humans presumably were also generated primarily by spinal circuits (Forssberg and Wallberg 1980). However, when the coordinated spinal actions of cats and humans were isolated from central influences the resulting stereotyped behaviors were not sufficient to render the animal functional within its environment. Spinal cats could support their own weight but not balance themselves, and the footfall pattern for newborn stepping was inappropriate for bipedal locomotion. These results emphasize the importance of central influences as well as the spinal motor programs in generating the appropriate context-specific patterns of muscular action characteristic of normal postural adjustments.

Despite the fact that our results suggest a degree of independence between sensory organizing and motor coordinating processes within the motor control system, we are not concluding that there is a lack of interactive linkages. The independence between sensory and motor processes observed here may have been influenced by our highly specific selection criteria and by our limiting analysis to myotatic and automatic components of postural action. One phenomenon which may represent an interactive linkage was the loss of directional specificity in the most severe spastic diplegic, SD1, and the single athetoid, AT1, both of whom expressed most dramatically mixed sensory and motor abnormalities. Whether loss of directional specificity characterizes a predominantly sensory problem, something akin to the inappropriately directed responses of ataxics under conflict conditions, or a motor coordination problem is a distinction which cannot be drawn at this time given the present limits of our experimental-conceptual framework.

Biomechanical Correlates of Abnormal Muscular Coordination in Cerebral Palsy

In all three of the hemiplegic children, the spastic leg did not effectively exert force upon the support surface, and in two of these children the knee joint was also destabilized causing rapid oscillatory shifts in weight bearing between the less-involved and the spastic legs. Although muscle weakness and alterations in muscle tone could have contributed to the ineffective movements of the spastic leg, a biomechanical model of leg motion currently under

development predicts that reversals in the normal distal to proximal activation of muscles alone would be sufficient to cause ineffective force production and proximal joint instability (McCollum and Nashner 1982). Specifically, during forward sway perturbations the activation of gastrocnemius followed by hamstrings thrusts the subject backward about the ankle joints and stabilizes mechanically coupled antiphase motion about the hips while not disrupting the metastable equilibrium of the knee joint (Nashner 1981). However, because both gastrocnemius and hamstrings are two joint muscles, a hamstrings contraction in advance of gastrocnemius will create a flexor force which disrupts the equilibrium of the knee joint, causing the involved leg to collapse under the weight of the body. In contrast, activation of gastrocnemius first rotates the lower leg backward about the ankle, a motion which indirectly extends the knee joint with sufficient force to counterbalance the flexor forces generated directly by gastrocnemius and hamstrings insertions at the knee. A logically similar argument explains why contraction of quadriceps in advance of anterior tibialis during response to backward sway perturbations hyperextends the knee, creating a "peg leg" stance. In fact, both of the above predictions correlate with the biochemical records shown in Fig. 8; the spastic leg was transiently unloaded during adjustments to forward sway adjustments perturbations, while it transiently carried extra weight during backward sway adjustments.

In the three ataxic children, the relations between muscle contractile patterns and the resulting waveforms of force exerted by each leg upon the support surface were similar to the normals. Nevertheless, their overall performance was poorer with respect to minimizing the amplitude of sway (Fig. 9). However, poorer performance can be attributed to abnormally long and variable latencies in the automatic postural adjustments of the ataxics. Because the inverted pendulum dynamic characteristics of the body in free-stance dictate that the rate of AP sway will increase exponentially prior to a postural correction, the relation between the latency of correction and the amplitude of AP sway excursion at the onset of a correction will be a geometric one.

Impact of Results Upon Concepts for Normal and Abnormal Movement Control

The finding that cerebral palsy movement disorders are in some patients clearly focused within specific substrates supports the contention that within the system for posture and movement control gross sensorimotor functions are broken down into functional elements executed by specialized subsystems. However, the most useful aspect of these observations has been in further defining principles underlying elements of sensory organization and muscle coordination, and in understanding the relations between gross motor behavior and the normal and pathological interactions among specific sensory and motor substrates of posture. Finally, this experimental-conceptual approach may prove quite useful in bridging the large gaps between what is presently understood about gross movement abnormalities and their pathophysiological substrates. Specifically, the primary substrates of postural disorders in ataxia appeared to be breakdowns in the central sensory feedback mechanisms which perceive motions of the body with respect to the inertial gravitational vertical and surrounding objects and surfaces and from this matrix of information determine the timing, direction, and amplitude of postural actions necessary to maintain the center of body mass over the base of support. In contrast, the problems of the spastic hemiplegics involved breakdowns in the mechanisms which in advance of the execution of a postural action appropriately structure the temporal-spatial characteristics of muscular contraction necessary to shift the center of body mass in a coordinated way. Thus, according to our findings alterations in gross motor behaviors could in some patients arise from rather different substrates than previously espoused in the clinical literature. For example, proximal instability due to incorrectly sequenced actions might be confused with weakness or inactivity in proximal muscles. Hypertonus, which in some instances might be a compensatory mechanism helping to stabilize proximal limb segments against inappropriately structured contractions, is usually considered a primary cause of functional abnormality. Finally, the intention tremor of the ataxic patient is in the clinical literature usually attributed to coordination abnormalities whereas projection of our postural results to voluntary systems would suggest that inappropriate or ineffective use of feedback information and not dyscoordination is the genesis of the tremor. However, if one clinical implication is to be drawn from this work, it is in demonstrating the active, proactive nature of the sensorimotor system: It may be misleading to extend observations upon isolated component functions derived from a passive system in developing a systematic understanding of active movement states and their disorders.

Appendix

Spastic Hemiplegics

Among the three spastic hemiplegic children, SH1 was a female aged 10 years and 6 months, SH2 a male aged 8 years and 1 month,

and SH3 a male aged 9 years. All had a history of brain hypoxia at birth. SH1 and SH3 were right and SH2 a left hemiplegic. All presented significantly elevated deep tendon reflexes (3+ on the clinical scale) and extensor plantar responses on the involved side. On the non-involved sides, SH1 was normal, while SH2 and SH3 had very mildly elevated deep tendon reflexes (1+ on the clinical scale). Peripheral losses in SH1 were the only sensory abnormalities among the three. All were independent ambulators but placed the toe rather than the heel first. All had normal mental status.

Ataxics

AX1 through AX3 were all relatively pure examples of ataxia. AX1 was a 6 year 1 month old female, AX2 an 8 year old female, and AX3 also an 8 year old female. AX1 and AX2 had a history of hypoxia at birth, while AX3 was normal until a severe flu-like illness with severe dehydration at 14 months. All were hypotonic with depressed deep tendon reflexes bilaterally. Stance and gait were ataxic. All exhibited trunkal instability and tended to fixate proximal segments rigidly when attempting to move. AX1 had severe and AX2 mild dysarthria, while AX3 had ataxic ocular movements. AX2 and AX3 exhibited some tremor. None had sensory deficits and all were normal mentally.

Spastic Diplegics

Among the three spastic diplegics SD1, a 10 year old male and SD2 a 10 year old female, had a history of hypoxia at birth (SD2 was a premature twin birth). A history of brain hypoxia at birth was equivocal in SD3, a 7 year old male. While diagnosed diplegics, the degree of involvement was asymmetrical in all three. Involvement was greater on the right in SD1, who presented no sensory, visual-perceptual, or mental abormalities. SD2 was a more symmetrical case with deep tendon reflexes significantly elevated and extensor plantar responses bilaterally. In SD3, spasticity was greater on the right side and in the lower limbs, but with elevated deep tendon reflexes and extensor plantar responses bilaterally. Fine motor skills varied among the group from good in SD1 (left hand) to bilateral and marked clumsiness in SD2 and SD3. SD2 was of borderline low intelligence, while SD3 had obvious sensory and visual-perceptual deficits. All were independent ambulators but with flexion at hip and knee and toe rather than heel placement.

Athetoid

AT1 was a 6 year old male with a history of toxemia gravidorum during pregnancy, premature induced birth, and delayed motor development. Deep tendon reflexes and plantar responses were normal bilaterally. He presented bilateral choreo-athetotic movements with increased trunkal and proximal joint fixing bilaterally during voluntary movements. Sensation, visual-perceptual skills, and intellect were all within normal limits considering the severity of the motor deficits.

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