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ORIGINAL ARTICLE

Adaptive motor strategy for squatting in spastic diplegia

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Motor strategies, defined by kinetic, kinematic and/or muscle activation patterns, reflect neural planning of movement, which takes into account central as well as peripheral constraints. Major alteration is expected in cerebral palsy, a condition characterized by abnormal posture and movement secondary to early lesion of the brain. The objective of this study was to characterize the motor strategies involved in disruption of posture in cerebral palsy of the spastic diplegia type and compare them with normal controls. The optoelectronic ELITE system was used to record and analyse the movement of squatting from the standing position with the arms extended forward in 11 children with spastic diplegia aged between 3 and 12 years and 11 age-matched normal controls. Normal children maintained gaze and arm horizontality and trunk verticality throughout the movement. The knee followed an oblique trajectory. Its angular velocity profile showed a short, single-peaked, ascending phase. The onset of movement was preceded by deactivation of the semimembranous muscle. In diplegic children, gaze and arm horizontality and trunk verticality were lost. The ankle was rigidified, resulting in spatial fixation of the knee. The ascending phase of the knee velocity profile was prolonged and multi-peaked. There was widespread muscle co-contraction from the outset of movement. No anticipatory deactivation was evidenced, but anticipatory bursts appeared in the soleus. Patients with cerebral palsy have to organize a limited motor repertoire from a restricted neural potential. Consequent motor strategies presently demonstrated in spastic diplegia are distinct and appear as an original alternative to those of normal subjects.

Keywords: Movement. Posture. Motor control. Cerebral palsy. Spastic diplegia. Co-contraction.

Introduction

In theory, any unconstrained motor act can be realized in an almost infinite number of ways owing to the excess of biomechanical degrees of freedom.[1] This has been termed the redundancy problem. However, experimental studies show that a given subject or even population actually favours only a limited number of different ways for accomplishing a motor task. These ways are referred to as motor strategies. They can be defined by kinetic, kinematic or muscle activation patterns, or a combination of these parameters.[2–4] Motor strategies encompass not only the execution of the desired movement itself, but also eventual postural preparation.[5] Motor strategies therefore reflect neural planning of movement, which must take into account central (i.e. neural) as well as peripheral (i.e. biomechanical) constraints. The empirical notion of the existence of motor strategies is in agreement with the theory of motor control, for which the execution of movement is largely dependent on motor programmes elaborated in the central nervous system which act through commands to mononeurons of agonist and antagonist muscles.[6] In this context, the emergence of motor strategies can be regarded as a manifestation of priority management by the subject's nervous system faced with the redundancy problem. Such

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priorities depend on intrinsic and extrinsic constraints as well as on optimization factors for the task.

The study of motor patterns used by children should lead to a better understanding of the processes that govern the emergence of motor strategies. In conditions with lesions of the central nervous system leading to a limited movement repertoire, a situation which typically occurs at an early stage in cerebral palsy, the elaboration of motor programmes takes place in a young and deleted nervous system with some properties of plasticity. Studies of motor strategies in cerebral palsied patients should therefore give good insights into alternative motor control in the special setting of early non-progressive brain lesion or abnormality.

In the present work, we studied the movement of self-paced rapid flexion of the legs from a stationary standing posture in a group of normal children and in a group of children with spastic diplegia.

Subjects and methods

Patients

Eleven children aged between 3 and 12 years with cerebral palsy of the spastic diplegia type participated in the study (Table 1). All were born premature (Table 1). None had fixed contractures. Magnetic resonance imaging showed features of periventricular leucomalacia in all patients. In two, there were cystic lesions in the white matter. There was associated left temporal lobe atrophy in one and vermian atrophy in another. Motor milestones were attained late in all patients. Independent walking (Table 1) was acquired at least 17 months before participation in this study. Eight patients attend mainstream school. Two go to a special school for children with intellectual disability and one to a special school for children with motor disability. All have Bobath-type physiotherapy (one to three times per week, started in the first year of life in all but one patient).

Controls

The control group consisted of 11 age-matched children with normal development and no disabilities (Table 1). The child born at 33 weeks' gestation had a normal cerebral ultrasound in the neonatal period.

Considered movement

The studied movement was rapid squatting from the standing position with the arms extended forward, following the same paradigm as that used previously in adults.[7] Each child performed ten trials.

Movement recording and analysis

We used the optoelectronic ELITE system[8] for electromyogram (EMG)-coupled tridimensional analysis of motor strategies involved in this movement. The ELITE system consists of two infrared light-emitting cameras that detect reflective markers at a sampling rate of 100 Hz. The markers were ten adhesive plastic spheres (diameter: 15 mm) placed on the child's skin overlying the following bony landmarks: (1) the lateral aspect of the nose at the height of the infra-orbital edge, (2) the ear tragus, (3) the upper limit of the acromion, (4) the lateral epicondyle of the elbow, (5) the styloid process of the wrist, (6) the antero-superior iliac spine, (7) the great trochanter, (8) the lateral condyle of the knee, (9) the external malleolus, and

Table 1 Characteristics of patients and controls

	Spastic diplegia (n = 11)	Normal children (n = 11)
Age (years)	3–12 (mean 6.4, SD 2.6)	3–12 (mean 6.4, SD 2.6)
Gender	8 boys, 3 girls	7 boys, 4 girls
Gestational age (weeks)	27–36 (mean 30.7, SD 3.1)	33–41 (mean 38.2, SD 2.4)
Onset of independent walking (months)	21–40 (mean 30.3, SD 4.9)	10–15 (mean 12.5, SD 1.5)

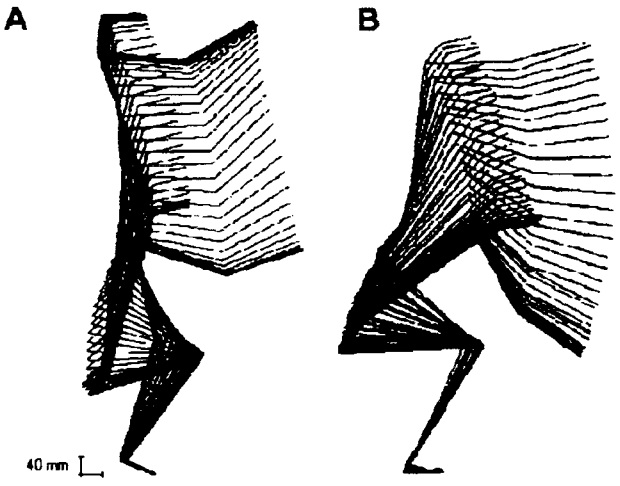

Fig. 1. Sagittal kinogram of the squatting movement, representing the superimposition of the sagittal projections of the segments defined by the markers every 80 ms from the standing posture to the squatting posture. (A) In a normal child. (B) In a child with spastic diplegia.

(10) the distal end of the fifth metatarsal. Computerized integration of the signals were then used for tridimensional reconstruction of the markers' movements.

Angles were computed as follows: *head orientation* as the angle of the (1)–(2) segment relative to the horizontal; *arm orientation* as the (3)–(5) segment relative to the horizontal; *trunk orientation* as the (3)–(7) segment relative to the vertical; *hip* as the (6)–(7)–(8) angle; *knee* as the (7)–(8)–(9) angle; and *ankle* as the (8)–(9)–(10) angle.

Surface EMG activity was recorded (sampling rate 1000 Hz) using a telemetry system and silver-silver chloride electrodes placed over the right anterior, vastus lateralis, biceps femoris, semimembranous, tibialis anterior, medial gastrocnemius, lateral gastrocnemius, and soleus muscles.

Statistical analysis

Analysis of variance between sets of data (ANOVA) was computed using the Statistica Software (Softcom).

Ethical aspects

This project has been approved by the local Ethics Committee of the University Children's Hospital Queen Fabiola (Free University of Brussels).

Results

All the children could perform the movement. Tridimensional reconstruction was complete in 198 of the 220 recorded movements, partial in 16 and invalid in six which were discarded. As illustrated in Fig. 1A, normal children kept their trunk practically erect throughout the movement. Gaze horizontality was respected and arm horizontality was well preserved, realizing a dissociated flexion of the lower limbs with maintained extension of the upper part of the body. The knee followed an oblique trajectory in the sagittal plane (back to front and top to bottom). Conversely, diplegic children accomplished the movement in an undissociated manner (Fig. 2B). Trunk verticality and gaze and arm horizontality were lost as flexion of the lower limbs was performed. The ankle tended to be rigidified, which resulted in spatial fixation of the knee. The movement was operated around the fixed knee, with backward shift of the hip.

There was no clear pattern in the evolution of the data from the first to the last of the ten trials performed by each child. No statistical differences were observed between the kinematic data of the first three trials and those of the last three trials. No clear difference appeared with increasing age across subjects or patients.

Differential angles between the onset and the end of the movement in the sagittal field are presented in Table 2 for the head, trunk and arm orientation, and hip, knee and ankle joints. All show highly significant differences between normal and diplegic children.

In normal children, the profile of the angular velocity of the knee showed a short, single-peaked ascending phase and a longer descending phase with an inconsistent number of subcomponents (Fig. 2A, top trace) reflecting unrestrained postural unloading followed by braking action. In diplegic children, the ascending phase of the angular velocity of the knee was prolonged and showed multiple peaks (Fig. 2B, top trace). The maximal amplitude of the velocity was significantly higher in normal than in cerebral palsied children (Table 1). The time interval between the onset and the end of knee flexion was significantly higher in cerebral palsied than in normal children (Table 1).

While minimal muscle activity was evidenced in normal children in the standing position (Fig. 2A, EMG traces prior to the onset of movement marked by the open headed dotted line), diplegic children demonstrated generalized tonic activity (Fig. 2B, EMG traces prior to the movement marked by the open headed dotted line). If the normal child had a

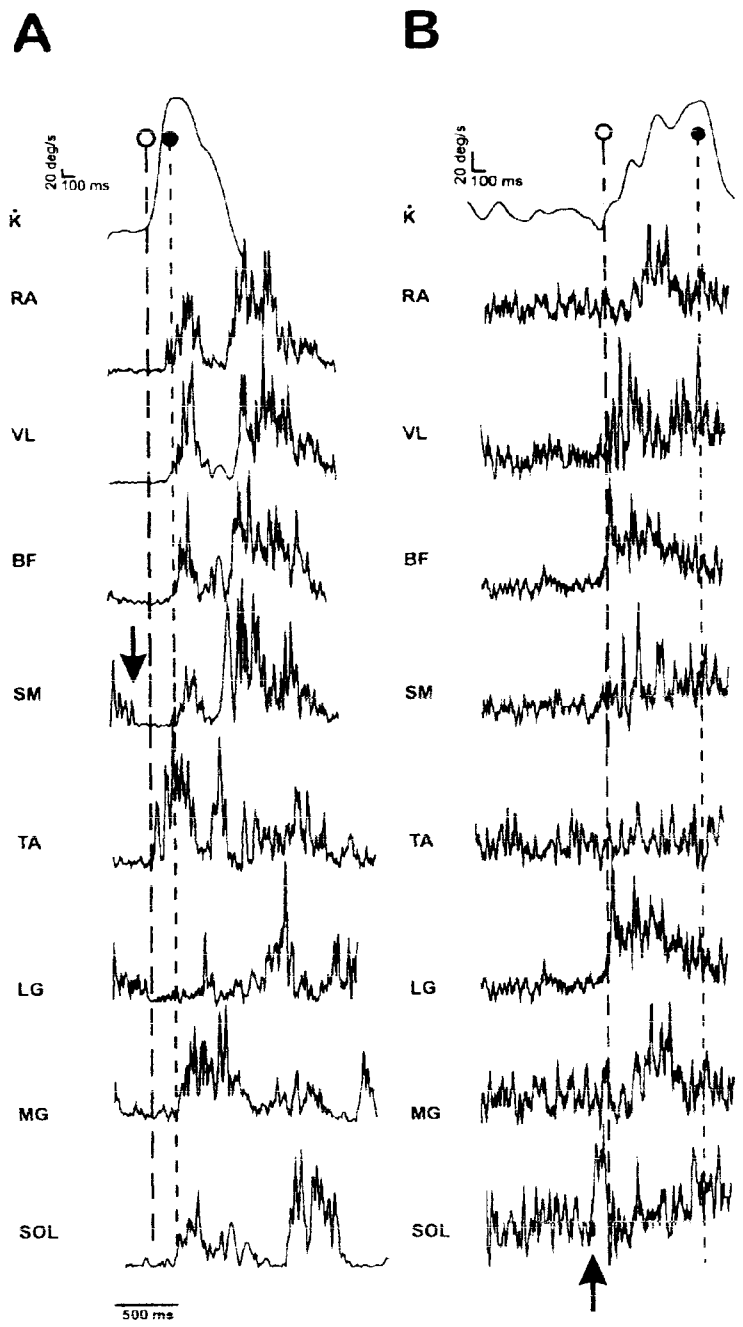

Fig. 2. Knee angular velocity and muscle activities during the squatting movement (A) in a normal child; (B) in a child with spastic diplegia. Presented EMG signals are rectified and integrated (20 ms steps). K: knee angular velocity; RA: rectus anterior muscle; VL: vastus lateralis muscle; BF: biceps femoris muscle; SM: semimembranous muscle; TA: tibialis anterior muscle; LG: lateral gastrocnemius muscle; MG: medial gastrocnemius muscle; SOL: soleus muscle; open-headed dotted line: onset of movement; black-headed dotted line: maximal peak of knee angular velocity. The downward-pointing arrow marks the onset of the anticipatory deactivation of the SM in the normal child. The upward-pointing arrow shows the anticipatory burst of SOL activity in the child with spastic diplegia.

postural tonic activity in the semimembranous muscle, this was deactivated 120–200 ms before the onset of the movement (Fig. 2A, black arrow). This deactivation was not found in cerebral palsied children. On the contrary, in 40–70% of trials in six of the diplegic children, an anticipatory burst of activity was recorded in the soleus muscle (Fig. 2B). The latter contributed, together with the tibialis anterior muscle, to locking the ankle as a postural preparation for the movement. During the

Table 2 Kinematic data for normal children and children with spastic diplegia.

	Normal children	Spastic diplegia		
Head orientation	−2.61° (SD 8.92)	−25.30° (SD 15.77)	$F(1,218) = 87.68$	$p \ll 0.001$
Trunk orientation	−35.00° (SD 8.80)	−54.20° (SD 11.90)	$F(1,206) = 72.77$	$p \ll 0.001$
Arm orientation	−28.38° (SD 18.24)	−68.84° (SD 28.49)	$F(1,204) = 93.86$	$p \ll 0.001$
Hip angle	88.48° (SD 16.74)	68.36° (SD 27.10)	$F(1,200) = 25.68$	$p \ll 0.001$
Knee angle	103.43° (SD 18.59)	58.31° (SD 24.54)	$F(1,208) = 173.51$	$p \ll 0.001$
Ankle angle	12.66° (SD 8.60)	0.24° (SD 4.01)	$F(1,198) = 29.40$	$p \ll 0.001$
Maximal knee angular velocity	251.85°/s (SD 47.30)	112.46°/s (SD 59.61)	$F(1,204) = 128.23$	$p \ll 0.001$
Duration of movement	823.35 ms (SD 264.18)	1388.70 ms (SD 409.66)	$F(1,205) = 86.43$	$p \ll 0.001$

Differential angles between the onset and the end of the movement in the sagittal field are shown for all considered segments.

movement, no braking activities were observed in the normal children up until the maximal peak of knee angular velocity (Fig. 2A, black headed dotted line). Thereafter, braking activities consisted of bursts in most recorded muscles. In contrast, diplegic children showed phasic co-contraction activities in the vastus lateralis, biceps femoris, semimembranous, lateral gastrocnemius and medial gastrocnemius muscles from the outset of the movement, as well as eventual bursts in the right anterior muscle before the maximal peak of knee angular velocity (Fig. 2B, black headed dotted line).

Discussion

This study has focused on the squatting movement, a multi-joint whole-body movement which involves voluntary rupture of posture, an equilibrium challenge and dissociated movement schemes in the upper and lower parts of the body. We found that the motor strategy of normal children is similar to that of adults.[7] This strategy combines movement dissociation (maintained extension of the trunk and upper limbs throughout lower limb flexion), nearly vertical translation of the body segments with an oblique trajectory of the knee; short, monophasic ascending phase of the knee angular velocity; and anticipatory deactivation of the semimembranous muscle. Prior to the movement, muscle activity patterns were similar to those recently described in standing children.[9] Anticipatory deactivation of the hamstring muscle before the squatting movement is evidence for a feed-forward mode of postural control.[7]

As regards children with spastic diplegia, we also found a common and consistent motor strategy. However, this strategy is different from that of normal controls, and is not a simplified or truncated form of it. It combines undissociated execution of the movement (global flexion), complete loss of trunk verticality, backward shift of the hip, locking of the ankle; prolonged, multiphasic ascending phase of the knee angular velocity; and anticipatory activation of the soleus muscle. Prior to the movement, tonic activity was high in all muscle groups. Phasic agonist–antagonist co-activation patterns appeared from the onset of the movement, reflecting global, non-variable and poorly organized coordination of muscle activation as shown for other motor tasks in spastic diplegia.[9,10]

Evidence of feed-forward postural control was present, represented by additional muscle activation over an already high background tonic activity, occurring before the onset of the movement arguing against a compensatory response to the movement. In contrast to normal children, who organize the movement with a specific postural inhibition pattern, diplegic children organize it with non-specific additional activations, realizing postural shifts between so-called primitive motor patterns (antigravity extension, fetal symmetric flexion). This finding is a reflection or a consequence of impaired reciprocal inhibition in children with cerebral palsy both before and during voluntary movement, which has been shown to be related to sustained hyperexcitability of alpha motoneurons.[11]

The latter results in exaggerated monosynaptic stretch reflexes whose effects add to those of reduced facilitation of polysynaptic reflexes which are also dependent on supraspinal control.[12] Consequently, movements involving reciprocal antagonist inhibition are difficult to organize. Experimental findings in a monkey suggest that the early antagonist inhibition is mediated by the primary motor cortex (M1).[13] A similar situation is encountered in unimpaired young children, when supraspinal control of these mono- and polysynaptic reflexes is physiologically not functional yet because of insufficient myelination.[12]

Locking the ankle may be viewed as taking advantage of lower limb distal hypertonia (rather than opposing it) in order to ensure greater stability in a context where selective movements cannot be realized. This dynamic, task-dependent stiffness of the ankle joint is achieved through co-contraction of agonist and antagonist muscles, which may also compensate for distal muscle weakness in the lower limbs.[14] A degree of biomechanical stiffness is also likely to be present,[15] but has not been measured. The trunk is bent forward, respecting its basal state of hypotonia. Backward rotation of the hip around the knee (which is fixed in space as a result of the locking of the ankle) results in preventing excessive forward projection of the centre of gravity and consequent falls. The relatively low maximal angular velocity of the knee and total slowness of the movement reflect low mechanical efficiency previously reported in spastic diplegia.[16]

This motor strategy can therefore be regarded as an adaptive one taking into account poor motor tuning possibilities. In other words, as reviewed by Latash and Anson,[17] the central nervous system may solve Bernstein's redundancy problem[1] differently for different states of the system for movement production, elaborating new optimal patterns according to reconsidered priorities. Or more simply, the motor patterns developed by children with spastic diplegia represent their solution for the underlying cerebral problem.

We speculate that this view may have implications for the management of children with spastic diplegia which might indicate controlled evaluation studies. First, the adaptive character of the motor patterns could imply that the goal of management should not be to transform abnormal strategies into normal ones. However, abnormal patterns may appear maladaptive in some functional contexts, and in the long term some of them may be associated with severe orthopaedic complications. The pathophysiology of these complications is not clear. It may involve a direct effect of abnormal biomechanical forces and restricted mobility,[18] but also neurogenic changes in the muscles. Some or all of these factors may be related to the selection of priorities by the central nervous system for motor control.

According to the optimization theory,[17] a local minimum of optimized function can be reached and kept because the slightest deviation from it would lead to a deterioration of the optimized function. However, there may be a further but deeper minimum corresponding to a much better value of the optimized function. In this view, therapeutic interventions could primarily aim at reordering the priorities of the central nervous system towards optimized function. Second, given the possibility of cerebral plasticity, controlled studies would be needed to evaluate whether early intervention taking advantage of the adaptive abilities of the central nervous system and learning processes, could lead to rearrangement or setting of new priorities, and therefore to improved strategies for optimal function.

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