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POSTURAL CONTROL IN STANDING AND WALKING IN CHILDREN WITH CEREBRAL PALSY

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The inclusion of postural abnormalities in the clinical picture expressed by children with cerebral palsy (CP) is clearly underscored in a recent reappraisal, which describes this condition as ‘a group of disorders of the development of movement and posture causing activity limitation, that are attributed to non-progressive disturbances that occurred in the developing foetal or infant brain’, and recommends that ‘all body regions – including trunk, . . . – be described in terms of any impairment of movement or posture’ (Bax et al. 2005). Indeed, several lines of evidence point to a potentially relevant role of postural control in the functional performance of persons with CP. For instance, studies examining the development of motor skills in children with CP indicate that there are delays both in the emergence of behavioural motor milestones, and also in subsequent impairments in motor skills, including standing, walking and manipulation. As efficient postural control is important for the performance of voluntary skills, postural abnormalities could contribute to the delays and impairments seen in the motor skills of the child with CP (see Chapter 4). At the same time, abnormal postural attitudes might result from the need to cope with specific primary deficits such as poor balance control or muscle weakness, so that adaptive or substitutory postural changes will become part of the clinical picture of CP.

Although the above notions appear to have remarkable clinical relevance, e.g. for programming targeted therapeutic interventions, the available data on functional impairment in CP reveal a limited number of studies aimed at describing the postural conditions and the possible disturbances of the different types of postural control mechanisms. The present chapter addresses these points. In particular, the regulation of the postural attitude maintained during standing by children with CP will be dealt with in the first part, whereas in the second part analysis will focus on dynamic postural control in relation to the locomotor function.

Postural control during standing
The control of posture during stance has traditionally been divided into two categories: (1) steady-state balance control, involving postural alignment of body segments and characteristics of body sway when standing quietly, and (2) postural adjustments to externally and internally triggered perturbations. This includes both reactive responses to unexpected trips or slips and anticipatory postural adjustments before a voluntary movement. In the following sections we discuss balance control in children with CP based
on these categories of balance control. We also examine constraints on both the motor and the sensory systems contributing to balance. Postural adjustments during standing and walking in children with CP only have been assessed in children with some standing and walking capacity; thus the research applies to a limited group of children with CP.

**STEADY-STATE BALANCE CONTROL: QUIET STANCE**

This section of the chapter discusses quiet-stance postural control in children with CP, and addresses both issues of alignment of body segments and characteristics of body sway during quiet stance and the effect these may have on other aspects of balance. Alignment of the body refers to the relationship of body segments to one another, as well as to the position of the body with reference to gravity and the base of support (Shumway-Cook and Horak 1992). Alignment of body segments over the base of support determines to a great extent the effort required to support the body against gravity. In addition, alignment determines the type of movement strategies that will be effective in controlling posture. Changes in resting stance position or alignment are often characteristic of the child with CP. Abnormalities in alignment can reflect changes in the alignment of one body part to another or in alignment of the centre of mass (COM) relative to the base of support.

Children with CP often experience reduced range of motion in joints of the ankle, knee, and hip. In addition, contractures of the hip, knee, and ankle muscles may contribute to atypical postures in sitting and standing. These include excessive posterior tilt of the pelvis during sitting due to shortened hamstrings muscles, toe standing due to shortening of the gastrocnemius muscle, and flexion of the knee during stance due to hip-flexor tightness. These constraints often result in a crouched posture during stance. Atypical alignment can also be expressed as a change in the position of the body with reference to gravity and the base of support. For example, asymmetric alignment in standing is often characteristic of children with a unilateral neural lesion, such as unilateral spastic CP (spastic hemiplegia). Children with this type of lesion tend to stand with weight displaced toward the non-involved side. This asymmetric alignment is a strategy that typically develops to compensate for weakness in the hemiparetic leg (Shumway-Cook and Horak 1992).

One way of measuring postural stability during quiet stance is to ask the child to stand on a force plate and to measure their centre of pressure (COP) displacement and velocity characteristics during spontaneous sway. A number of laboratories have explored the differences in these characteristics for children with CP versus typically developing children (Ferdjallah et al. 2002, Rose et al. 2002). Rose et al. (2002) measured COP during quiet stance under eyes-open and eyes-closed conditions for a group of children with bilateral spastic CP (spastic diplegia), aged 5–18 years, and compared the results to typically developing children of the same age range. They noted that 33% showed significantly higher mean values for COP in at least two of the parameters, path length and average radial displacement of the COP. Values for frequency of sway were lower for the participants with CP than for the typically developing children. Values did not become more abnormal with eyes closed, which suggests that the children with CP had normal dependence on visual feedback for balance during quiet stance.
Postural Adjustments to Externally and Internally Triggered Perturbations

Reactive balance control: responses to slips

The typical crouched posture observed during quiet stance in children with bilateral spastic CP may contribute to the way in which muscles are recruited and coordinated for recovery of stability (reactive postural control). In a study on postural control in children with bilateral spastic CP, Burtner et al. (1998) asked children to respond to unexpected threats to balance, produced by a force plate that was moved unexpectedly forward or backwards (at 2.8–3.8 cm and 20–25 cm/sec), simulating the unexpected movement of a bus or train carriage. They found that the children with bilateral spastic CP who stood in a crouched posture showed strong coactivation of agonist and antagonist muscles of the leg when responding to balance threats, compared to typically developing children, who used limited coactivation of agonists and antagonists. This coactivation of agonists and antagonists is energetically inefficient, and may cause neuromuscular fatigue while standing.

In order to determine if this coactivation was due to the constraint of standing in a crouched posture, the authors asked typically developing children to stand in a similar crouched position, mimicking the posture of the children with bilateral spastic CP. They found that these children also used co-contraction of agonist and antagonistic muscles more often in response to platform perturbations under this stance condition. This indicates that the constraints of standing in a crouched posture may contribute substantially to the atypical muscle response patterns used in reactive balance by these children (Burtner et al. 1998).

Other studies have also examined reactive balance control in children with CP, focusing most specifically on children with unilateral spastic CP. Nashner et al. (1983) asked children with unilateral spastic CP (7–12 years of age) to stand on a platform that moved unexpectedly forward or backwards, similar to the platform described above. Postural response characteristics of typically developing children to a balance threat, for example, in the forward direction, causing backward sway include activation of the stretched muscles of the ankle joint (tibialis anterior) as well as muscles of the thigh (quadriceps) in an ascending sequence. The response onset of the ankle muscles is typically at about 100 ms followed by the thigh muscles 20 ms later (Shumway-Cook and Woollacott 1985). As mentioned above, there is little activation of antagonist muscles in typically developing children of this age. In children with unilateral spastic CP in the spastic leg the onset of the gastrocnemius response was typically delayed and the proximal thigh muscle (hamstrings) was activated early, giving a response. This was accompanied by high levels of coactivation of antagonist muscles in the spastic leg. However, the less involved leg showed normal muscle-onset characteristics (Nashner et al. 1983).

Nashner et al. (1983) also studied children with ataxia and noted that this group also showed significant delays in the onset of ankle (distal) muscles, but did not show muscle response reversals. Instead they showed approximately 30–45 ms delays between distal ankle and proximal thigh muscle response onsets. Thus, in this group of children, delays were seen for contraction of all muscles (Nashner et al. 1983). Finally, children with bilateral spastic CP were more variable, with some showing response characteristics similar to those
of children with unilateral spastic CP and others more similar to children with ataxia. The delayed contraction of the gastrocnemius in the children with unilateral spastic CP was unexpected, since, these children showed positive clinical signs of spasticity in this muscle, such as increased stiffness in response to passive stretch, clonus and equinus gait. This evidence of delayed activation of a ‘spastic’ muscle has also been seen by other researchers in adult patients with spastic hypertonia (Badke and Di Fabio 1990).

Adaptation of postural responses to changing balance threats
Research has also shown that children with bilateral spastic CP have reduced ability to adapt their postural responses to changing sizes and velocities of balance threat (Roncovesalles et al. 2002). Those researchers hypothesised that balance problems of the children with CP when recovering from large or fast platform displacements could be due to either (1) reduced ability to increase contraction amplitudes of postural muscles when faced with larger or faster compared to small or slow balance threats, (2) a delay in the onset of muscle contractions, or (3) to simultaneous contraction of agonist and antagonist muscles, reducing the efficiency of the agonist burst. They found that typically developing children showed increased muscle response amplitudes when they were given larger or faster postural perturbations, while children with cerebral palsy showed no significant increases under these conditions. There was no difference in muscle contraction onset time or agonist–antagonist co-contraction, supporting the hypothesis that problems with adaptation postural response to increased balance threat magnitudes is due to an inability to increase muscle-activation levels.

Habilitation of reactive balance control
Research on habilitation strategies for reactive posture control has shown that this type of balance control can be improved with training (Shumway-Cook et al. 2003, Woollacott and Shumway-Cook 2005, Woollacott et al. 2005). Children with cerebral palsy (bilateral and unilateral spastic CP) were given 5 days of intensive training in reactive balance control, consisting of 100 trials of variable forward and backward platform movements. Reactive balance training led to improvements in postural performance, as measured by time to recover stability after a balance threat (see Fig. 5.1). In addition, training improved the directional specificity of postural responses and other spatial–temporal muscle response characteristics, including (1) faster activation of muscle responses, associated with faster recovery of stability; (2) an increase in distal–proximal postural muscle response patterns; and (3) decreased coactivation of agonist and antagonist muscles. Training-related improvements also remained at 1 month post-training. Interestingly, each child with CP showed a unique combination of these changes that contributed to an improvement in their balance performance (as measured by total COP movement and time taken to recover stability in response to the perturbation). In addition the level of improvement was associated with the severity of involvement of the CP: children with unilateral CP showed increased improvements compared to those with bilateral CP.

In summary, reactive postural response characteristics for children with all forms of cerebral palsy show delays in the onset of contraction of distal (ankle) muscles. In addition,
children with bilateral and unilateral spastic CP showed high levels of co-contraction of agonists and antagonists at a joint. Finally, children with unilateral spastic CP and some children with bilateral spastic CP showed response reversals, with thigh muscles responding prior to ankle muscles. Some of these atypical response patterns appear to be related to atypical body alignment (crouched posture), especially in children with bilateral spastic CP. Studies indicate that intensive reactive balance training can improve these response characteristics as well as functional balance performance.

Postural adjustments during internally triggered perturbations: anticipatory responses during arm movements

Problems in initiating postural muscle activity prior to voluntary muscle activity are present in children with unilateral spastic CP. Nashner et al. (1983) performed experiments in which they asked children with unilateral spastic CP to push or pull on a handle while standing. They found that there was a lack of preparatory postural activity prior to the arm movement for the hemiparetic side, compared to the normal side in the children with cerebral palsy. Onset of contraction in postural muscles in the trunk and leg of the intact side preceded contraction onset in the prime movers of the arm. In contrast, in the hemiparetic side, muscle contraction onset in the arm preceded that of the postural muscles of the leg (Nashner et al. 1983).
SENSORY ORIENTATION FOR BALANCE

Balance control (whether it is during quiet stance, in preparation for or in reaction to a threat to balance) also involves the integration of information from multiple senses (vision, vestibular and somatosensory systems) concerning information about the position and movement of the body in space (for details see Chapter 2). For a specific task and context the neural systems of the child must determine the accuracy of a given sensory input for postural control, and then select the input that is the most appropriate for that context. For example, if a child is walking in a dimly lit environment, s/he would need to rely less on visual inputs and more on somatosensory and vestibular inputs. If s/he were walking down a ramp in a dark cinema, somatosensory cues would be ambiguous and visual cues would be almost absent, and thus vestibular inputs would be the primary inputs required to maintain balance.

Experiments by Nashner et al. (1983) have examined the ability of both typically developing children and children with CP (unilateral spastic CP and ataxia) to integrate sensory information important for maintaining balance under different conditions that changed the availability and accuracy of visual and somatosensory inputs for postural orientation. Figure 5.2 shows the amplitude of anterior–posterior sway in typically developing children, children with unilateral spastic CP and children with ataxia as they stood under six different sensory conditions. The y axis shows the sway of the child (performance ratio) with 0 indicating no sway and 1.0 indicating loss of balance. The left

Fig. 5.2 Antero–posterior postural sway (labeled performance ratio) for typically developing children (TD), children with unilateral spastic CP (UNI) and children with ataxia (ATAX) under 6 sensory conditions. Performance near 0 indicates minimal sway, while performance moving toward 1.0 indicates instability. Stars show numbers of children who lost balance in each group. Filled circles: eyes open. Open circles: eyes closed. Filled triangles: eyes open, stabilised vision. (Stabilised support surface denotes the platform moving with the child’s body sway). (Redrawn from Nashner et al. 1983.)
part of the figure shows the performance of the three groups under normal support surface conditions (platform level and not moving) with eyes open, closed, and with eyes open in a visual surround that moved with their sway. The right-hand part of the figure shows the performance of the groups when the support surface was moving with their sway (labelled stabilised support surface). You can see that the sway values for the children with ataxia were greater than those for the typically developing children and the children with unilateral spastic CP for all conditions. As shown by the stars, one of the three children with ataxia lost balance under the stabilised surface condition with eyes closed, and two of the children lost balance when standing on the stabilised surface in the visual surround that was ‘stabilised’ (moving with their sway). In addition, one child with unilateral spastic CP also lost balance in this last condition, while no typically developing children had this problem. These results suggest that these children have difficulty orienting to sensory environments in which two of the three sensory cues (vision and somatosensation) are either distorted or absent, and thus require a reliance on vestibular inputs alone.

**Changes in the Standing Pattern of Children with CP Induced by Orthoses, Postural Support and Training**

Ankle–foot orthoses (AFOS) are often prescribed by clinicians to control spasticity in children with cerebral palsy, in order to prevent excessive plantarflexion of the ankle. Two types of orthoses that have often been used include solid AFOS (allowing no movement at the ankle) and spiral or hinged AFOS (allowing limited movement at the ankle). In spite of the widespread use of orthoses, there are few studies that have examined their effect on standing balance.

One study by Burtner et al. (1999) examined the effects of using AFOS on reactive balance control, specifically documenting the effects of orthoses on characteristics of muscle response organisation underlying postural control. They compared reactive balance response characteristics both in children with bilateral spastic CP and in typically developing children when wearing no AFOS as compared to wearing solid and spiral AFOS. Postural responses to platform displacements were categorised according to whether the recovery from the perturbation was predominantly at the ankle (defined as the ankle strategy, the response seen in adults and typically developing children for this range of platform displacements) or at other joints such as the hip (the response seen in patients with neurological disorders, such as muscle weakness or neuropathy). Results showed that for both children with bilateral spastic CP and the control group of typically developing children there were significantly fewer trials in which children used the ankle strategy when recovering balance while wearing the solid AFOS versus the no AFOS and dynamic AFOS conditions (Fig. 5.3, left). In addition, there was a reduced probability of seeing a muscle response in the ankle muscles and a delay in the activation of the ankle muscles (gastrocnemius) for balance threats in the backward direction, causing forward sway (Fig. 5.3 right).

The study also showed that the frequency of activation of a normal distal–proximal muscle response organisation was lowered in both the children with bilateral spastic CP and the typically developing children in the solid AFOS condition as compared to the no AFOS and dynamic AFOS conditions. These results suggest that AFOS used to control position and motion
at the ankles can also affect the sequencing and timing of muscles used for balance recovery, and thus there may be a trade-off in the outcome of their use, with results showing improved postural alignment during stance and decreased efficiency of reactive balance control. Alternatively, the changes also might indicate an appropriate adjustment to the AFO-condition, with constrained movement of the ankle by the AFO requiring less reactive response at the ankle and increased response at other joints, such as the knee and hip.

Postural control and locomotor function
Most of the available information on locomotor control in typically developing children and children with CP is derived from studies of steady-state walking whereby lower limb movements are customarily described in terms of relative angles of hip, knee and ankle joints, mechanical joint moments and powers and timing of EMG activity in representative thigh and leg muscles. This approach has the clear advantage of dealing with the most noticeable aspects of the walking process, but fails to provide direct insight into the postural mechanisms associated with locomotor function, such as those involved in the transition between quiet stance and steady-state progression, or maintenance of whole body stability during cyclic motion of lower limb actuators. A feasible way of tackling the above issues would require (1) analysis of postural adjustments responsible for transitional walking phases and (2) assessment of the locomotor attitude of axial body segments (with specific reference to gravity-based, absolute, coordinate frames) in addition to the usual description confined to lower limbs. The above points are addressed in the following paragraphs. Due to the paucity of the available data, results will mainly refer to the most commonly encountered forms of CP, i.e. bilateral and unilateral spastic CP.

Fig. 5.3 Left: Percent ankle strategy use with no AFO, dynamic AFO and solid AFO for children with CP versus control group (typically developing children). Right: Frequency of trials with gastrocnemius muscle response for children with CP versus control group.
**Transitional Walking Phases**

Transition from upright stance to steady-state walking implies destabilisation of the current antigravity postural set, whereas the opposite task, i.e. to stop walking, requires dissipation of the kinetic energy attained by the body during forward progression, in order to allow recovery of a static postural attitude. Both these tasks involve regulation of the external forces by centrally initiated actions on the relative positions of COP and COM in the anterior–posterior and mediolateral directions (Brenière et al. 1987, Crenna et al. 2001). Initiation of gait, in particular, is triggered by a backward and lateral COP shift, with a consequent fall of the body forward and toward the stance foot. The underlying motor programme, which includes inhibition of postural activity in the soleus muscle followed by activation of tibialis anterior and tensor fasciae latae, is initiated prior to any detectable displacement of trunk and limb segments. This indicates the involvement of anticipatory postural actions (APA; Crenna and Frigo 1991). In typically developing children, the ‘imbalance synergy’ associated with gait onset can be detected as early as within 1–4 months of independent walking experience, and typically consists of a lateral tilt of the pelvis and stance (trailing) leg, with consequent unloading of the contralateral swing (leading) limb (Assaiante et al. 2000). In their earlier developmental stage these postural adjustments display lower incidence, larger involvement of the upper parts of the body and more consistent medio-lateral displacements, as compared to adults (Assaiante et al. 2000, Malouin and Richards 2000). Follow-up of typically developing children from 21/2 to 8 years revealed progressively higher frequency of adult-like patterns, indicating that the developmental tuning of such a feed-forward control takes a long time (Brenière and Bril 1998, Ledebt et al. 1998). Clinical experience in children with bilateral and unilateral spastic CP does not reveal obvious difficulties in gait initiation. APA in the leg muscles associated with arm rising while standing, which have been shown to rely on the same motor programme adopted for gait onset (Crenna and Frigo 1991), have been detected in children with bilateral spastic CP by recording the COP path under the support base during reaching tasks (Jesinkey et al. 2005). This suggests that functional APA may be substantially preserved in CP (see also Stackhouse et al. 2007). Preliminary results from Malouin et al. (2003) in children with unilateral spastic CP reported a reduced magnitude of the preparatory adjustments on the paretic (swing) side, with simultaneous compensation by enhanced anteroposterior actions on the non-paretic (stance) side. It should however be understood that the standing attitude of children with CP differs from that of typically developing children. Since the initial standing posture is known to affect the gait initiation process (e.g. Couillandre and Brenière 2003), it is at present unclear to what extent the observed changes should be ascribed to a primary dysfunction or should be related to the abnormal ‘postural set’ of children with CP (and thus regarded as adequate adaptations). A further point worth considering is that the above-reported findings refer to bilateral and unilateral forms of CP, where deep (e.g. basal) brain structures are usually spared by neural lesion. In fact, electrophysiological and imaging studies in adults suggest that control systems involved in gait initiation include cortical, basal ganglia and brainstem structures and that APAs for gait initiation can be severely affected in neural disorders of basal ganglia (Crenna et al. 1990; for a review see discussion in Crenna et al. 2006). Further research, therefore, is necessary.
to ascertain whether the APA for gait initiation are specifically impaired in clinical subtypes of CP with involvement of cortico–basal ganglia circuits, such as dystonic and choreo–athetotic forms (J.F. Kerrigan et al. 1991, Cheney 1997). Analysis of more subtle changes concerning abnormal modulation of the spatio–temporal parameters of the imbalance programme warrant further evaluation as well.

As for the gait termination task, to our knowledge no quantitative studies have been carried out in typically developing children and children with CP, even though clinical experience indicates that children with bilateral spastic CP can display difficulties in stopping gait and sometimes chose to terminate walking by kneeling down, at least in the earlier phases of natural history.

**Steady-State Walking**

Analysis of postural control during steady-state ambulation will be addressed with reference to the relative weight of postural and translational roles of the individual body segments, by dealing first with the best stabilised (head–trunk), through relatively articulated (shoulder-pelvis), up to the most mobile ones (lower extremities). The impact of abnormal dynamic posture on the control of COM motion and gait energetics will be subsequently considered. A further section will be devoted to the pathophysiological mechanisms potentially involved.

**Head**

During normal walking the head and trunk segments undergo relatively small absolute angular excursions. This is true for adults (e.g. Winter 1991, Cromwell et al. 2001) and school-age children (Sutherland et al. 1988, Assaiante and Amblard 1993, Assaiante et al. 1993), which suggests that, along with propulsive and actually destabilising actions primarily aimed at forward progression, dynamic control of the position of the axial portion of the body, is effectively operational.

Three-dimensional head movements during locomotion are usually described in the sagittal (pitch), frontal (roll) and transverse (yaw) planes. In typical adults, walking at natural speed along a linear path, the average head pitch, estimated by angular position of the plane defined by horizontal semicircular canals (Frankfort plane), was shown to be approximately 10°–12° around the earth horizontal (Pozzo et al. 1990, Cromwell et al. 2001). Similar figures were demonstrated for the roll and yaw planes using various locomotor and non-locomotor tasks (Pozzo et al. 1995, Cromwell et al. 2001). Such relatively low ranges of oscillation were ascribed to a ‘head in space’ stabilisation strategy (Nashner 1983), in which vestibulo–collic and ‘long loop’ vestibulo–spinal reflexes possibly play an important function (Amblard 1996). A potential role for this strategy during walking might be to ensure minimal inflow from vestibular and visual systems during unperturbed locomotion, with consequent ‘noise’ reduction and optimisation of their sensitivity to adequate stimuli. On the same line, complementary gaze stabilisation in space was found to be provided by the vestibulo–ocular reflex (Berthoz and Pozzo 1988, Paillard 1988). Dynamic control of the head during walking shows a relatively early ontogenic evolution, as revealed by a longitudinal study performed over the first 46–80 weeks of unsupported gait, which
demonstrated that maximum amplitude of head rotations in the pitch and roll planes starts improving during the first weeks of autonomous ambulation and attains mean values close to the mature range within 6–32 weeks (Ledebt and Bril 2000).

A cross-sectional study of walking in typically developing children, in which children were studied in conditions with minimal equilibrium demands (overground walking) and in difficult balance conditions (narrow beam walking), has shown that adoption of head stabilisation in space is confined to easy locomotor tasks in 3- to 6-year-old children, to be subsequently extended to difficult tasks in 7- to 8-year-olds (Assaiante and Amblard 1993). In typically developing children aged 7–8 years, dynamic control of head displacement in the pitch and yaw planes attains near-adult features, whereas less effective performance occurs in the roll plane (Assaiante and Amblard 1993). This indicates that acquisition of the ability to ‘anchor’ the head position to gravity-linked reference frames over a functionally effective spectrum of locomotor tasks runs a protracted developmental course.

In addition to the above basic head stabilisation actions, minor patterns of head (and trunk) motion in the sagittal plane were described during ambulation. These consist of low-amplitude forward and downward head pitches, which are thought to compensate for vertical trunk translations, both by minimising peak accelerations at the head and by assisting visual fixation, when needed (Mulavara and Bloomberg 2002). Compared with the strategy of head stabilisation in space, such a fine coordination strategy has a slower developmental evolution, in that, by the end of the first year of independent walking, it has attained about 50% of the adult performance level (Ledebt and Wiener-Vacher 1996).

Quantitative evaluation of head stability during treadmill walking in 7- to 12-year-old children with bilateral or mild unilateral spastic CP was performed by Holt et al. (1999), who adopted a variability measure of the vertical displacement of the head. The study revealed that the variability of fluctuations in the amplitude of head displacement of children with CP only differed to a minor extent from that of typically developing children. At variance, the variability of fluctuation in the period of head displacement was significantly higher than that of the controls. This finding suggests that in children with CP timing rather than spatial control of head position during walking might be mainly impaired. Further studies are necessary for ascertaining the suboptimal ability to control dynamic head rotation in the three planes during ordinary overground walking in the different forms of CP. Such studies are needed in particular because it is known that children with CP, examined in a sitting position, may show significant disturbances of head–eye coordination and gaze stabilisation, which might yield possible effects on the processing of vestibular and/or visual information (van der Weel et al. 1996). Similar dysfunctions might also interfere with gait performance.

Trunk
As documented for the head, dynamic posturing of the trunk appears to be actively controlled during normal locomotion. Over the stride cycle the trunk is mildly bent forward as compared to upright standing, and during single-limb support it moves further forward and toward the weight-bearing limb, with a side-shift initiated prior to the ground contact and mainly produced during the double-support phase (Thorstensson et al. 1984, Krebs et al.
The above oscillations usually display narrow excursions in typical adults, both in the sagittal plane (2°–11°; Thorstensson et al. 1982, Pozzo et al. 1990, Cromwell et al. 2001) and in the frontal plane (2°–9°; Thorstensson et al. 1982). A detailed study of the trunk range of motion in young girls with a mean age of 12 years revealed values of 2° and 3.9° in the sagittal and frontal planes, respectively (Frigo et al. 2003). In spite of the different angular figures (which can largely be ascribed to different anthropometric landmarks selected for defining trunk axis), data indicate a great dynamic stability of the trunk, which appears to be the highest among the axial segments (Cromwell et al. 2001).

Stabilisation of the trunk (which along with head and arms represents a large inertial load featuring about two thirds of the whole body mass; Winter 1991) is a biomechanical necessity during the equilibrium challenging conditions inherent to bipedal human gait. Moreover, a vertically aligned trunk can provide a gravity-based egocentric reference for locating external targets and effective guidance of locomotor limb movements (see Chapter 3). In typically developing children dynamic control of spine motion is initiated during the first weeks of independent walking, with relevant improvement within 6–32 weeks, at least for spontaneous gait on a flat, non-compliant terrain (Ledebt and Bril 2000). The underlying muscle synergies typically involve both anterior (rectus abdominis; obliquus, externus and internus abdominis; transversus abdominis) and posterior (multifidus deep and superficial fascicles, erectors spinae) trunk muscles, which are typically coactivated twice during the stride cycle in correspondence with the double support phases (Saunders et al. 2005).

Observational, semi-quantitative assessment of sagittal trunk attitude during gait in children with bilateral spastic CP, who had been examined longitudinally from 1–5 to 6–16 years of age, has shown two main patterns (Yokochi et al. 2001). The most common type, described in 75% of subjects, is characterised by an erect trunk posture, with possible enhancement of lumbar lordosis and anteversion of the pelvis; whereas a less common type, occurring in 25% of children, is a forward-bent-trunk walking, detected particularly in the earlier phases of autonomous walking. Based on observational kinematics, therefore, sagittal axial orientation with respect to gravity appears to be relatively maintained in the most common form of CP with preserved ambulatory function. In typical adult gait, interestingly, trunk accelerations in the progression plane were found to be attenuated by a cranio–caudal sequence of activation of paraspinal muscles (possibly controlled in a feed-forward manner) with consequent smoothing of mechanical effects on the head motion (Prince et al. 1994, Cappozzo 1981). The above reported evidence of near-normal vertical head excursion in children with CP during walking (Holt et al. 1999) would suggest that this mechanism is not consistently impaired. Further investigations however are needed to confirm these results and to assess the capability of children with CP to counteract external trunk destabilisations interfering with walking (which will require precise spatio–temporal recruitment of synergistic axial muscles; e.g. Hirschfeld and Forssberg 1991). Dynamic control of trunk sway during transitional gait phases (initiation and termination) or directional changes deserves thorough analysis as well.

As first recognised by Bobath and Bobath (1975), and subsequently outlined by several authors (see Sutherland 1984, Perry 1992), analysis of gait in children with CP can reveal relevant abnormalities of trunk motion in the frontal plane, mainly represented by enhanced
excursion, most often in phase with the lateral displacements of the pelvis and shoulder. Frontal trunk sway toward the stance limb is normally controlled by lengthening contractions of contralateral erectors spinae (multifidus and longissimus) and psoas, along with lower amplitude recruitment of the ipsilateral paraspinal groups (Thorstensson et al. 1982, Andersson et al. 1997, Penning 2001). This postural synergy is expected to be spatially up-scaled in the subgroup of children with CP exhibiting enhanced trunk sway. The reason might be either a primary deficit of dynamic postural control or an adaptation strategy. With respect to the latter possibility, the observed persistence of large frontal-plane trunk sway in children with bilateral spastic CP walking with an anterior walker (Crenna and Marzegan, unpublished data) suggests that a compensation for poor equilibrium control in the sagittal plane is not always the cause. Energy-saving mechanisms substituting for ineffective exploitation of the sagittal pendular sway (see below) might represent an additional source of adaptation.

Shoulder girdle and pelvis
The shoulder and pelvic girdles participate in the walking movements more consistently than trunk and head segments, so that their absolute locomotor excursions are relatively larger. Due to the relevant contribution of the proximal appendicular system to arm swinging, the shoulders move mainly in the horizontal plane, so that at the heel strike the ipsilateral shoulder is behind the other, with significant velocity-dependent increments of the range of motion. Data in adolescents walking at their natural speed indicate average excursions of 6.9° ± 2 in the horizontal as compared with 1.6° ± 1.1 in the frontal plane (Frigo et al. 2001). A similar trend was shown for the variability (estimated by the standard deviation) of shoulder excursion in children of 3–8 years of age, with no significant age effect (Assaiante et al. 1993). At variance with the shoulders, which move mainly in the horizontal plane, the pelvis shows consistent locomotor excursions both in the horizontal and frontal planes. It moves forward and downward at each stance phase, thereby contributing to the step length and contralateral foot clearance, respectively (Inmann et al. 1981, Kerrigan et al. 2001). According to the normative data proposed by Sutherland et al. (1988), in typically developing 7- to 8-year-old children maximal pelvic oscillations are kept on average within 20° in the transverse plane, 10° in the frontal plane and only 5° in the sagittal plane (see also Stansfield et al. 2001).

In children with CP, shoulder and pelvis kinematics during walking reveal more variable patterns as compared with axial segments. These range from mainly cranial impairment, with increased oscillation of the shoulders in the frontal and horizontal planes contributing to the frontal pendulum gait described above, up to mainly caudal involvements, characterised by either reduced or enhanced excursion of pelvic obliquity, featuring hip-adducted (frozen pelvis) gait, and pelvic oscillating (anserine, duck-like) gait, respectively (see Perry 1992). Transverse-plane gait deviations characterised by increased pelvic rotational asymmetry are common in children with unilateral spastic CP, and were correlated to femoral antetorsion and weakness of hip-flexors (Noritake et al. 1998).

Figure 5.4 shows examples of abnormal absolute angular motion and intersegmental coordination patterns of head, shoulders and trunk during walking in children with CP (data
Fig. 5.4 Abnormal absolute angular motion and intersegmental coordination patterns during walking in children with CP. Data from a typically developing child (age 10 years; column a) walking at slow, natural and fast speed (all pooled into one plot) are contrasted with data from three children with bilateral spastic CP (age 10, 8 and 14 years; columns b, c and d, respectively) walking at self-selected speed. In the upper and mid-row graphs, which refer to frontal plane angles, the Head obliquity (angle between the interorbital axis and horizontal) and the Trunk obliquity (angle made by the trunk axis, i.e. by the line joining the midpoint between posterior superior iliac spines and C7 spinous process, and the vertical) are plotted against the Shoulder obliquity (angle between the inter-acromion axis and horizontal). In the bottom row graphs, which refer to the sagittal plane, the Head tilt (angle made by the line joining the C7 spinous process and the external ocular cantus with vertical) is plotted against the trunk tilt (angle between the trunk axis and vertical). Negative values indicate inclination of the relevant segment toward the stance-limb side. R values give the linear correlation coefficients. Each loop is obtained by superimposition of 80 strides on average. See text for further explanation. Clinical involvement of children with bilateral spastic CP, as reflected by locomotion-related items of GMFM scale (dimension E, items 65–88, maximum performance level: 72) is 64/72 (column b), 34/72 (column c), and 39/72 (column d), respectively.
from Laboratory for Movement Analysis in Children, L.A.M.B., P. & L. Mariani, Milan, hereafter referred to as L.A.M.B.). Data from a typically developing control child are contrasted with data from three children with bilateral spastic CP with progressively higher impairment. Analysis of the angle-angle diagrams reveals narrow excursions with no directional trends in the non-disabled child (subject A), which indicates a good control of absolute angular motion of the axial segments. At variance, the children with bilateral spastic CP reveal either an increased loop area with no significant intersegmental coupling (subject B), or largely increased coupling of the three segments in the sole frontal plane, featuring a typical ‘frontal pendulum’ gait pattern (subject C), or else increased loop area with coupled intersegmental motion in the frontal and sagittal planes, featuring an ‘en bloc’ dynamic postural pattern (subject D).

**Lower limbs**

The concept of dynamic control of postural set during walking, so far addressed with reference to gravity-related configuration of axial body segments, cannot be directly extended to the rhythmic rotational movements expressed by lower limbs. However, clues in favour of a ‘postural component’ possibly integrated into the locomotor pattern of legs come from the evidence, obtained by Principal Components Analysis, of a tighter covariation of the absolute locomotor angles of thigh, shank and foot as compared with the relative locomotor angles of hip, knee and ankle, which was tentatively interpreted as a possible preferential encoding of lower-limb excursions in external, gravity-related, reference frames (Borghese et al. 1996). Such a covariation was shown to exhibit consistent changes toward the mature pattern at a quite early ontogenic stage, i.e. during the first weeks of independent walking, suggesting a short-term organisation of the relevant control systems (Cheron et al. 2001). Analysis of absolute lower-limb angles in children with CP has never been performed in a systematic way, and a detailed description of the wide range of locomotor phenotypes (typically labelled after the most conspicuous changes of the relative joint angles in the sagittal plane) is beyond the scope of the present chapter. But the spectrum of these conditions in children with bilateral spastic CP spans from almost exclusive distal involvement, which includes the different forms of equinus gait (marked by increased ankle plantarflexion during the early contact phase, during the whole support phase, during the swing phase, or any combinations thereof), to progressively more proximal and severe locomotor patterns, whereby the knee or the hip joints are preferentially involved. The latter comprise the crouch knee gait, recurvatum knee gait, jump knee gait, stiff-legged gait, mild knee gait, hip hiking gait and mixed forms (D.C. Kerrigan et al. 1991, Perry 1992, Sutherland and Davids 1993, Lin et al. 2000, Steinwender et al. 2001, Wren et al. 2005). Likewise, in the asymmetric forms of CP, such as unilateral spastic CP, isolated foot drop in swing, increased plantarflexion in stance and swing, increased plantarflexion plus enhanced knee flexion, and increased plantarflexion plus enhanced knee and hip flexion have been described as progressively more proximal and severe kinematic patterns (Winters et al. 1987, Rodda and Graham 2001, Patikas et al. 2005)
FUNCTIONAL OUTCOME

The functional impact of abnormal integration between dynamic postural control and propulsive actions can be represented in a comprehensive way by assessing locomotor displacements of body COM. Using the ‘direct dynamics approach’ (see footnote), Massaad et al. (2004) demonstrated that one of the most common types of walking in individuals with CP, which as mentioned earlier implies forefoot contact with the ground, is marked by significant increase in the vertical and decrease in the forward displacement of body COM. In this study, abnormalities of COM displacement were found to be consistently correlated with a digitigrade placement of the foot, but not with the topography and severity of motor impairment, nor with the length of walking experience. Interestingly, the above COM pattern was proposed to provide some biomechanical advantage in terms of increased elastic potential storage (higher vertical COM rise) and adaptation to the lack of stability of the stance limb during disturbed foot rocker movements (shorter forward COM displacement). In this respect, abnormal COM motion might incorporate compensatory mechanisms enabling translation with lower metabolic cost and/or higher safety margins for equilibrium control. The observation that similar COM kinematics were described in the early independent walking of typically developing toddlers (Hallemans et al. 2004) may point into the same direction.

Repercussions of abnormal dynamic posture on the walking function can be further analysed in terms of effective exploitation of physiological mechanisms aimed at minimising the work done by the muscles to sustain the mechanical energy changes of the body COM, i.e. the so-called ‘external work’. Indeed, during typical walking the external muscular work is consistently reduced by the fact that the increase in forward COM kinetic energy (i.e. forward COM velocity) occurring over the stride phases marked by forward body fall (mainly the second half of single support phases) is largely obtained by a simultaneous decrease of COM potential energy (i.e. COM height) stored over the stride phases marked by upright body lift (mainly the first half of single support phases), and vice versa (see Cavagna et al. 1983). Accordingly, computing the fraction of the mechanical energy saved due to this pendular mechanism of energy transduction, (a parameter referred to as ‘recovery’), can provide a non-invasive measure of the energetic efficacy of the locomotor pattern in the individual subject (Detrembleur et al. 2000). In a study on children with bilateral spastic CP (Bennet et al. 2005), the percentage of energy ‘recovery’ was found to be 33% smaller than in typically developing controls. The loss in walking efficacy was ascribed to the following factors: (1) the increased vertical COM excursion, (2) the non-sinusoidal profile of the kinetic energy changes and abnormal phase shift between kinetic and potential energy curves, possibly related to a lack of a heel strike, and (3) the absence of coordinated ankle and knee flexion at weight-acceptance and ineffective second rocker (Bennet et al. 2005). In the L.A.M.B. data base (see above) both normal and reduced ‘recovery’ levels were observed in children with CP as compared with typically developing children walking over a same speed range. Figure 5.5 reports results from three representative children with bilateral spastic CP and reveals that ‘recovery’ values well within the control range can be observed over the whole spectrum of walking speeds, with maximums at intermediate velocities, as actually occurs in the control group (subject A).
In more severely affected children, however (subjects B and C, whose limited speed spectrum indicates a higher degree of impairment), reduced ‘recovery’ values can be detected at the higher waking speeds, which demonstrates a velocity-dependent loss in the efficacy of the physiological pendular body oscillation. Since measuring ‘recovery’ provides an estimate of the energy consumption for the sole execution of the external work, it is possible that functional impairments quantified by this parameter are underestimated in children with CP, where the contribution of increased internal work (i.e. the work which does not produce a displacement of the body COM with respect to the environment) is expected to be augmented. The study of total energy cost of walking obtained by direct measurement of oxygen consumption or estimated by heart rate changes in children with CP has shown a significant increase in children with bilateral spastic CP as compared to children with unilateral spastic CP and in children with unilateral spastic CP as compared to typically developing children walking at the same speed (Rose et al. 1989, Duffy et al. 1996, Mattsson and Andersson 1997). According to Stout and Koop (2004), the greatest energy requirements are seen in children with stiff knee gait and muscle–tendon contractures.

PATHOPHYSIOLOGICAL MECHANISMS
A number of relevant pathophysiological mechanisms, most of which amenable to objective detection and, at least in part, recognised as potential targets for treatment, can contribute to the abnormal locomotor postures in children with CP. Crenna and collaborators.
et al. 1992, Crenna and Inverno 1994) have proposed a definition of the above mechanisms (originally aimed at pathophysiological assessment of lower-limb locomotor disturbances and here extended to other body districts and processing systems) as including (a) peripheral, non-neural components related to bone and passive muscle–tendon properties, (b) central, executive factors including impaired muscle activation (paretic component), loss of selectivity in neuromuscular output (cocontraction component), abnormal EMG recruitment upon stretching (spastic component) and persistence of immature motor programmes (immature component), (c) central disturbance of sensory processing and sensorimotor integration, and (d) impaired higher level functions (Fig. 5.6). An account of the relative role of some of the above mechanisms in the gait pattern of children with CP, with specific emphasis on postural effects and related adaptation and substitution mechanisms, is presented in the following sections.

**Bone–joint deformities and abnormal passive muscle–tendon properties**

Children with CP may, especially in the later stages of their clinical history, develop bone deformities and joint abnormalities. These typically affect the foot (hindfoot varus, hindfoot valgus, metatarsus primus varus, hallux valgus and supination or pronation of the midfoot-forefoot complex), the tibio–fibular and femoral segments (rotational and torsional deformities, trochanter head anteversion), the hip joint (incongruency, dislocation, subluxation due to acetabular dysplasia), the pelvis (external rotation) and the spine (kyphosis, lordosis and scoliosis) (e.g. Renshaw et al. 1995, Chambers 2002, Duffy and Cosgrove 2002, Horstmann and Bleck 2007). Bony and joint deformities are believed to result from chronic changes in the mechanical load supported by specific body segments.
under static and dynamic conditions (Zhang et al. 2006), which in turn can be largely ascribed to changes in the stiffness (i.e. tension produced for unitary lengthening) of the muscles spanning the relevant joints. A significant contribution to the latter phenomenon, in particular, is given by increased passive stiffness, which can effect both the contractile and/or non-contractile components of the muscle–tendon units. Indeed, detailed analysis of single fibres and fibre bundles from ‘spastic muscles’ revealed abnormal morphology and passive mechanical properties, both in the myocellular and interstitial compartments. Fibre anomalies include higher variability in the average diameter with nonsignificant changes in the mean length, increased stiffness (up to twice the normal values) mainly ascribed to structural changes in cytoskeletal macromolecules, and shortening of sarcomere resting length (Shortland et al. 2001, Lieber and Fridén 2002). In the extracellular space significant changes attain the composition of the interstitial matrix, which in spite of the higher collagen content was found to exhibit increased mechanical compliance (Fridén and Lieber 2003). Other changes concern the structure of ligaments, capsular, fascial, aponeurotic, and/or tendineous tissues (Akeson et al. 1987, Booth et al. 2001, Smeuleders et al. 2005). Effects of structural morphofunctional abnormalities are likely to be enhanced by disharmonic growth of soft tissues and skeletal components during development of body proportions, as suggested by animal studies (Ziv et al. 1984) and by the clinical evidence of a frequent worsening of peripheral non-neural constraints during fast growth bouts. Changes in the passive muscle–tendon properties in children with bilateral and unilateral spastic CP are known to affect preferentially long-tended, posterior leg muscles such as triceps surae and tibialis posterior in the distal forms and medial and lateral hamstrings including gracilis, quadriceps, hip-adductors and iliopsoas in the more severe, proximal ones (Rodda and Graham 2001, Cooney et al. 2006). Increased passive stiffness prevailing in muscles acting in the sagittal plane will contribute to equinus or crouch gait postures, whilst prevalence of fixed shortening in muscles acting in the frontal plane will condition a walking pattern dominated by intrarotated, adducted limbs. Mixed forms, in which involvement of medial hamstrings and triceps surae is associated with shortening of hip-adductors, are also common.

Objective detection of changes in the passive properties of synergic muscle-tendon units can be obtained in vivo under quasi-static conditions by means of noninvasive biomechanical techniques (e.g. Hoang et al. 2005), but assessment of their interference during functional (e.g. locomotor) movements is extremely difficult, mainly due to the almost continuous contraction of several muscle groups, with frequent presence of motor units recruitment in the examined muscle, its agonist and/or antagonists. Analysis of the relationship between external joint moments and relative joint angles over specific stride phases marked by EMG silence in the tested muscles can provide an indirect estimate of the impact of increased passive stiffness during walking (Frigo et al. 1996a). For instance, an increased slope of the knee moment–knee angle relationship in late swing, prior to hamstring activation, was found to occur in children with bilateral spastic CP, with a possible contribution to the abnormal locomotor attitude of the ipsilateral lower limb at touchdown (Crenna 1999). Interference of passive components from other muscle groups typically involved in CP (e.g. calf muscles), however, cannot be reliably obtained by this method, due to the frequent presence of consistent motor output over the relevant time window.
Postural adaptations for non-neural deficits
When peripheral non-neural components dominate the pathophysiological gait profile, they can produce changes in the dynamic postural attitude, but can also prime the adoption of adaptive solutions aimed at coping with the primary deficits. Clues about these actions can be obtained by comparing the abnormal locomotor pattern with either the gait of non-disabled subjects in whom walking was constrained as to reproduce typical CP figures, or with the gait of children with primary osteo-articular or tendineous impairments. With reference to the former condition, a number of studies, whereby mechanical restrictions limiting joint range of motion (e.g. taping or casting) were applied at the ankle and/or the knee joint in neurologically intact individuals, have shown the emergence of abnormal gait kinematic and kinetic patterns reminiscent of those classically described in children with CP (Papariello et al. 1985, Abdulhady et al. 1996, Nahorniak et al. 1999). Using the same paradigm, EMG features recalling abnormal activity profiles (including prolonged quadriceps recruitment in stance, co-contraction of tibialis and gastrocnemius in stance and premature activation of hamstrings and gastrocnemius in swing), as well as unfavourable energetic indexes of gait efficiency frequently observed in CP walking (see above), have been reproduced by typically developing individuals when asked to mimic a crouch (increased knee flexion) and crouch-equinus gait (increased knee and ankle plantarflexion) (Sienko Thomas et al. 1996). Similar information can be obtained by the study of primary orthopaedic impairments. For instance, congenital or early-onset shortening of Achilles tendon (which is also known as ‘short tendo calcaneus’ and is typically associated with a forefoot contact walking) might help identifying postural compensations associated with changes in passive muscle–tendon properties contributing to the equinus form of CP gait (Hall et al. 1967). The above examples underscore the relevant notion that several aspects of the gait pattern observed in children with CP can be ascribed to anomalous joint positions conditioned by passive non-neural factors.

Paresis
Fixed muscle–tendon shortening in children with CP can be ascribed at least in part to a relative imbalance between muscle activities across the spanned joints. The underlying mechanisms can be evaluated noninvasively by means of integrated kinematic, kinetic and EMG locomotor measures. Defective ability to produce and modulate motor unit recruitment during walking (paretic component) is one of these mechanisms (see Crenna and Inverno 1994, Rose and McGill 2005 Stackhouse et al. 2005). In children with bilateral or unilateral spastic CP capable of unaided walking, defective muscle activation in the distal muscles can be frequently observed in the pre Tibial group, most often close to the ground contact phase and, to a lesser extent, at toe-off. This condition is typically detected in the gait pattern of the most affected limb in children with unilateral spastic CP, where – along with increased stiffness of plantarflexors – it contributes to the foot drop during the swing phase (Frigo and Crenna 2008). Involvement of plantarflexors during single support is also common in forms of CP which particularly affect distal muscles, even though it is not clear to what extent peripheral non-neural constraints related to passive properties of the calf group might contribute to shape the EMG profile of these muscles. In more severe forms of CP, with a
higher incidence of an abnormal locomotor posture, defective locomotor output is extended to the proximal muscles, including hip-flexors and abductors (e.g. gluteus medius).

**Postural adaptations and substitutions for paresis**

Children with CP can use adaptive strategies against the interference of paretic components into the walking process. Information about the type of strategies which might be used may be obtained from gait analysis in children with peripheral motor impairment (myopathies in different stages of the disease, systemic or focal motor neuropathies), where descending commands from normally functioning central structures responsible for locomotor control are expected to be modified in order to cope with non-responsive or hyporesponsive peripheral muscle systems, with consequent emergence of compensatory dynamic postures and reorganisation of the whole gait pattern. The enhancement of lumbar lordosis with possible backward lean of the upper trunk, often observed during ambulation in children with CP, is one such example, in that a similar dynamic attitude can be found in children with Duchenne dystrophy in the late stage, as a compensation for weak hip-extensors obtained by reducing the lever arm of gravity (and thus external flexor moment) relative to the hip (e.g. Boccardi et al. 1997, Armand et al. 2005). In this pathology, however, the same figure can be related to fixed shortening of hip-flexors displacing the lumbosacral joint anterior to the hip axis, so that both paretic and non-neural components should be considered to be among the possible sources of compensation (Perry 1992). With reference to postural abnormalities in the frontal plane, the increased shoulder sway described earlier in walking children with CP may function as a compensation for paretic hip abductors (e.g. gluteus medius), as a similar movement is observed in children with Duchenne muscular dystrophy (Sutherland 1984).

Hints about compensations for defective locomotor output can be obtained also by producing short-term, reversible corrections of the relevant mechanical effects, e.g. by external wearable supports. Correction of paresis by orthotic aids can be employed in children with unilateral spastic CP, especially in case of localised signs, such as major involvement of the pretibial group. In this condition, a number of abnormal segmental patterns expressed on the affected side during the swing phase (e.g. lower-limb circumduction, increased knee and hip flexion, increased pelvic obliquity, enhanced trunk and shoulder sway toward the contralateral side, and even outstretched upper limb motion) can be significantly ameliorated when defective foot clearance is improved by AFOs (Bichon et al. 2004, Radtka et al. 2005). This indicates that the abnormal dynamic posture is partially dependent on compensatory actions. The same paradigm can be applied to the non-affected side, where vaulting the supporting foot, premature heel-off and enhanced knee and hip extension are usually ascribed to compensatory mechanisms aimed at assisting foot clearance on the paretic side. It should be mentioned, however, that in a study of stroke patients where non-affected side kinematics were assessed during stair negotiation before and after correction of the contralateral foot drop by AFO, minimal differences were found between basal and AFO-on conditions, suggesting that the modified kinematic pattern is not always primarily related to the assistance of contralateral foot clearance (Lam et al. 2005).
Co-contraction
Abnormal co-contraction of antagonist muscles is another mechanism which can be associated with anomalous postural attitudes during walking. Analysis under static conditions (Stackhouse et al. 2005) has shown that antagonist muscle coactivation (e.g. semitendinosus with respect to quadriceps femoris and tibialis anterior with respect to triceps surae) is markedly augmented during maximal voluntary contractions of single lower-limb muscles in children with CP. Similar conclusions, albeit with lower severity values, were provided by Ikeda and colleagues (1998), and the quantitative discrepancy was ascribed to different postural attitudes of the children (supine versus prone) during measurements, which suggests a relevant role of the static postural set into the interference of the co-contraction component. A more recent study by Chen and collaborators (2003) used multichannel EMG to evaluate the selectivity of motor output in supine position in children with bilateral and unilateral spastic CP, and described four distinct patterns ranging from partial reciprocal to complete synchrony among functionally antagonist muscles of both sides. Interestingly, the amount of co-contraction as assessed under static conditions was found to be positively correlated to the level of functional impairment displayed during walking assessed by the Gross Motor Function Measure (GMFM) scale. A further sign of the functional significance of the above effect is the finding that increased co-contraction among antagonist thigh and lower leg muscles was shown to significantly account for the greater O₂ cost of walking in children with CP (Unnithan et al. 1996).

Adaptive use of co-contraction
The degree of co-contraction in antagonist muscles can be increased as a function of the walking speed or show relatively constant values, suggesting two distinct modulatory actions. In addition, it can be susceptible to manipulation of sensory input. For instance, a mild change in somatosensory tactile information, obtained during walking by application of a light touch, was shown to induce significant improvement of the non-selective recruitment pattern in subjects with CP (Crenna and Inverno, 1994). This suggests that co-contraction can be employed as an adaptive strategy, possibly aimed at stiffening lower-limb joints against poor equilibrium control and fear of falling (see also Lamontagne et al. 2000, Jeka and Lackner 1994, Dickstein and Laufer 2004). Providing tactile stimulation, e.g. by the simple contact of the hand of an accompanying person, therefore, might help distinguishing a sensory-modulated loss of selectivity in the motor output. In this respect, adaptive mechanisms appear to be prone to manipulation of the operative context (including conditions of task execution), by showing flexibility to changed affordances. A study by van Roon et al. (2005), dealing with trunk muscles during reaching tasks with variable accuracy demand, indicates that the increased cocontraction levels typically adopted by non-disabled children when high accuracy is requested can be observed also in children with CP, suggesting that the latter are able to exploit a physiological strategy for modulation of agonist–antagonist coactivation.
Spasticity

Although the term spasticity is frequently used with a broad meaning (encompassing diverse neural mechanisms and phenomena such as increased tone, tendon hyperreflexia, co-contraction and even increased cutaneous flexor responses), it is felt that a narrow definition, which incorporates the substantial features originally identified by the ad hoc task force guided by James Lance (1980) would be less confusing. Accordingly, spasticity will here be equated to increased coupling between levels of muscle activation and estimated muscle–tendon lengthening velocity, associated with enhanced mechanical resistance to rotation (dynamic stiffness) of the relevant joint(s) (Crenna 1998). This notion implies assessment of abnormally enhanced EMG recruitment during stride periods marked by peak stretching rates of the tested muscle, so that objective detection of the interference of a spastic component (which can be the expression of increased excitability of the stretch reflex system) will require simultaneous measurement of EMG output, including amplitude, duration, degree of synchronisation, and estimated muscle kinematics. The latter is now feasible using geometrical models of the muscle–tendon complex, based on anthropometric parameters and relative joints angles, which can provide sufficiently reliable measures of the length and velocity of length changes of the muscle–tendon complex during functional movements (see Frigo et al. 1996b, Crenna and Marzegan 2005). The mechanical counterpart of the abnormal stretch-dependent EMG recruitment can be also evaluated in terms of dynamic joint stiffness, i.e. as the slope of the moment-angle relation computed over the movement periods in which (1) the two variables exhibit the same versus and (2) the motor output is limited to only one group in a pair of antagonistic muscles. Application of this approach to the analysis of gait of children with bilateral spastic CP, with a focus on the thigh and leg muscles mainly acting on the sagittal plane (Crenna 1998, 1999, 2003), has shown that, among the distal groups, abnormally increased activation upon stretching typically affects foot plantarflexors, with a preference for the lengthening phase which occurs in the early stance. Among the proximal muscles, involvement was found to be more frequent in the hamstring group, during the lengthening phase which occurs in late swing. The prominence of mechanically effective spastic components in the posterior thigh muscles may contribute to walking patterns dominated by knee flexion (e.g. the crouch gait pattern), whereas the occurrence of mechanically effective spastic components in the posterior leg muscles may contribute to walking styles dominated by plantarflexion (e.g. the equinus gait pattern). In the presence of comparable involvement of both posterior groups, intersegmental effects produced by the proximal muscles in late swing will create mechanical conditions for abnormal kinematics and kinetics of ankle and knee joints over the stance phase of the next stride. Indeed, limitation of hip flexion and knee extension induced by spastic hamstrings activity in late swing will bring about verticalisation of the tibia and consequent touch down by foot-flat or forefoot contact. The resulting calf muscle stretch and spastic reflex response during the subsequent weight loading, will brake the forward rotation of the shank, with possible occurrence of recurvatum knee pattern and, possibly, enhanced lumbar lordosis (see Knuttson and Richards 1979). The above figure indicates that spastic phenomena can produce intersegmental influences between distant muscles, thereby yielding complex changes in the overall locomotor pattern.
Adaptive use of spasticity
The availability of pharmacological agents such as botulinum toxins, capable of reversibly titrating the motor output of one or several muscles by selective chemodenervation, allows medium-term correction of motor abnormalities related to hyperactivity of specific muscle groups (including spastic muscles), thereby enabling identification of their pathophysiological role, as well as their possible ‘compensatory or substitutional’ function into the abnormal locomotor profile. In fact, the clinical observation and instrumental detection of worsened pelvic sway in the frontal plane after botulinum injection in the adductor groups (Crenna and Marzegan, unpublished data) indicates that hyperactivity of these muscles can play a stabilising role on the pelvis and lumbar spine. Similar conclusions can be obtained by comparison of the gait pattern before and after surgical correction of fixed shortening of hip adductors. In this respect, abnormally augmented activity and/or increased passive stiffness in the medial thigh muscles can be regarded as a form of protection (compensation) against poor pelvic control.

Sensorimotor integration and higher level impairments
Although the presence of non-motor disturbances in CP has been long recognised by clinical observation, their formal inclusion in the definition of this condition is relatively recent. In a recent review, Bax et al. (2005) concluded that ‘the motor disorders of CP are often accompanied by disturbances of sensation, cognition, communication, perception and/or behaviour’. In fact, as far as sensory modalities typically related to postural control are concerned (such as visual, vestibular and proprioceptive channels), quantitative assessments of sensory impairment and correlation thereof with functional (loco)motor performance have been rarely attempted in individuals with CP.

Visual function. Vision, and specifically peripheral vision along with dynamic changes in the visual scene (including optic flow), are known to be quite effective in determining postural reactions, particularly in childhood, although their relative contribution with respect to vestibular and proprioceptive inflow appears to be ‘re-weighted’, depending on the task or environmental context (Shumway-Cook and Woollacott 1985, Stoffregen et al. 1987, Peterka 2002, Sparto et al. 2006; see also Chapter 3). In children with CP, particularly in children with bilateral spastic CP, dyskinetic CP and CP related to preterm birth, ophthalmological deficits (e.g. strabismus, refraction errors, cataract, retinopathy), cerebral visual impairment (e.g. lower visual acuity, reduced visual field, optokinetic nystagmus asymmetries, oculomotor disorders) and visual-perception abnormalities (e.g. oculomotor apraxia, anomalous saccades, reduced selective visual attention, abnormal scanning order) have been documented (Fedrizzi et al. 1998, Jacobson and Dutton 2000). The visual-perception abnormalities have been ascribed to the involvement of optic radiations and deep parietal white matter (Cioni et al. 1997, Pennefather and Tin 2000, Da Costa et al. 2004). Among the central visual pathways, a specific vulnerability was found to affect the ‘dorsal stream’, i.e. the pathways projecting to parietal cortex, which contribute to the processing of spatial (location and arrangement) and movement related aspects of visual inflow (Koeda et al. 1990, Gunn et al. 2002, Fazzi et al. 2004).
In children with relatively severe forms of CP, retrochiasmatic damage of the visual pathways yielding ocular and ocularmotor disorders was shown to be correlated with anomalous head–trunk control, consisting of torticollis and misalignment of the upper part of the body (Porro et al. 2005). Interestingly, the disturbed head–trunk posturing was interpreted as an adaptation strategy aimed at compensating for visual defects and optimising residual visual functions. It is unknown whether this condition has an impact on locomotor postural set, whereby lateral body sway potentially elicited by changes in the rate of optic flow associated with head and eye rotations, are expected to be normally controlled or gated (Warren et al. 2001, Dumbar 2004).

**Vestibular function.** Recent studies of vestibular function employing transmastoid galvanic stimulation during walking in typical adult persons (Bent et al. 2004) have shown phase-dependent effects on lower limb positioning in the frontal plane (with tendency to foot displacement toward the anode side), with maximal effectiveness around double-support phases (in line with phase-dependent activation of lateral vestibulo–spinal projections on lumbar motoneurons described in animals by Matsuyama and Drew 2000). Interestingly, the same galvanic stimulation was found to yield homogeneous, tonic effects on the trunk motion throughout the stride cycle. This phenomenon was ascribed to a dual mode control involving assistance of antigravitary support and forward propulsion by phasic facilitation of extensor muscles of hip and lower extremities, with simultaneous maintenance of dynamic stability of the trunk. To our knowledge, no studies are available on vestibular deficits under static conditions or vestibular control during walking in persons with CP.

**Proprio- and exteroceptive sensation.** Disturbances of proprioceptive and exteroceptive perception have been reported in children with CP (Lee et al. 1990, Cooper et al. 1995, Odding et al. 2006), with lower scores in children born preterm than in those born at term (Bumin et al. 2005). Experiments using muscle vibration during locomotion in normal adults have shown that proprioceptive input from the paraspinal muscles can influence the direction of foot positioning during walking, suggesting that trunk proprioception plays a major role in the definition of the locomotor trajectory (Schmid et al. 2005). In this respect, the question might be forwarded as to whether the sensory disturbances reported in children with CP might contribute to abnormal dynamic alignment of the trunk, particularly when changing direction or during transitional phases.

**Cognitive functions.** In addition to the evidence of sensory integration deficits, children with CP may have cognitive impairments (e.g. Mulas et al. 2000, Kolk and Talvik 2002). They may also display limitations in the executive components and – at least in children with ataxic CP – in the alerting and orienting components of the attentional networks (Saavedra et al. 2005). Thus high-level control systems may also play a role in the expression of atypical locomotor posturo–kinetic profiles. Indeed, it is a common experience that, when walking in the artificial gait laboratory set, both typically developing children and children with gait disturbances most often reveal a walking style which, based on parents reports, is different from the usual pattern expressed in the familiar environment. This suggests that context-dependent, high-level components can strongly affect the (loco)motor control systems (Crenna et al. 2005a). The possibility that the gait pattern of children with CP is influenced by cognitive data processing is also prompted by the finding that application of
a dual task paradigm, consisting of counting backwards while walking, produced significant changes in the gait pattern (Crenna et al. 2005b), suggesting a possible interference between cognitive and motor networks.

**INDIVIDUAL PathophysiologicaL Profiles**

A final point might be mentioned when discussing putative mechanisms underlying abnormal dynamic postural control in children with CP. Pathophysiological profiles, performed in individual subjects with CP were shown to be marked by (1) different incidence of paresis, spasticity, cocontraction and passive component, (2) different levels of severity of individual factors, and (3) different topographical distribution within the tested muscles. Subject-specific profiles, however, can be obtained not only in children belonging to the same clinical form (e.g. bilateral or congenital unilateral spastic CP), but also in subjects exhibiting the same class of kinematic pattern of walking (e.g. equinus or crouch knee gait). This means that in children with neuromotor disturbances, the clinical and instrumental descriptions of abnormal gait pattern could be conveniently integrated by pathophysiological oriented assessments, enabling acquisition of additional, yet not available otherwise, clues about functionally relevant underlying mechanisms. The value of a pathophysiologically oriented assessment may be illustrated by the preliminary results of a follow-up study performed at the L.A.M.B in a group of children with bilateral spastic CP. The children had been assessed by clinical examination, standard gait analysis and pathophysiological gait profile. Thereafter, they were treated with botulinum toxin on the sole basis of clinical examination and instrumental kinematic description of the gait pattern. Indeed, subjects within this group revealing poor or no locomotor improvement after treatment were those whose pathophysiological gait profiles were dominated by paretic components or characterised by a very low incidence of spastic components.

**Conclusions**

The analysis of experimental findings on postural control during standing has shown that, in the most common forms of CP (bilateral and unilateral spastic CP) a normal balance regulation with normal dependence on visual feedback characterises the quiet stance in the majority of children, whereas increased COP sway and/or abnormal speed of COG displacement occur in a subgroup of subjects, particularly in those with ataxic forms. Results demonstrate also that abnormal standing postures can be conditioned either by reduced range of motion of relevant joints (bilateral spastic CP) or by compensation for weakness (unilateral spastic CP), which indicates that postural disturbances do not necessarily result from primary impairment of postural control systems, but can be consequent to specific pathophysiological factors, such as peripheral non-neural components or paresis. Analysis of reactive postural control to external destabilisation disclosed anomalies in timing (e.g. increased delay of distal muscles activity or reversed activation order of distal versus proximal groups) and spatial control (e.g. increased cocontraction of antagonistic muscles and defective spatial scaling of responses as a function of destabilising stimulus intensity). Once again, however, conditioning by standing postural constraints seems to play a role, at least in some of the observed deficits.
Studies on dynamic postural regulation during transitional walking phases and during steady-state walking reviewed in the second part of this chapter indicate that data on sufficiently numerous cohorts of children with homogeneous forms of CP are limited. Moreover, the general adoption of the sole kinematic measurements with large inter-author differences in the experimental protocols, as well as the rare use of axial muscles EMG recording, further hinder the possibility to evaluate dynamic postural attitude in depth and possibly uncover abnormal control mechanisms. With the obvious caveat derived from the above limitations, however, a number of points can be considered. Upon transition between upright standing and steady state walking, functional APA have been shown to be relatively preserved or not largely affected in children with bilateral and unilateral CP who are able to walk with or without aids. During unperturbed linear walking, moreover, abnormalities in the absolute position and excursion of the axial segments (head and trunk) show moderate intensity in the sagittal plane, whereas higher degrees of disturbance characterise the frontal plane sway.

In all cases, we have shown that when descriptive assessment of abnormal gait and related functional outcome are considered, several lines of evidence indicate that dynamic postural profile of CP children embodies, albeit at different degrees, adaptive strategies, i.e. functional solutions emerged in the individual subject from the interaction between primary deficits and spared functions. As proposed for the standing posture, compensatory strategies are possibly aimed at coping with pathophysiological factors, such as peripheral constraints (e.g. bony/joint deformities and/or passive non-neural components) and central motor deficits (e.g. paresis). The enhanced backward trunk lean and lumbar lordosis frequently marking the gait pattern of children with bilateral spastic CP is an example of this condition. Compensations can be uncovered by comparing the gait pattern of the individual child with CP with the locomotor patterns expressed by children with purely peripheral motor impairment. Alternatively, significant changes in the gait pattern of the child with CP can be searched for by contrasting the unperturbed self-paced walking with walking produced upon reversible manipulation of motor, sensory, cognitive or environmental parameters. The important notion underlying the use of the latter protocols is that primary deficits are expected to be more robust and resistant to externally induced changes, whereas adaptations become more flexible and amenable to larger variations when specific variables are controlled.

Pathophysiological mechanisms potentially involved in disturbed gait in CP include peripheral, executive and possibly sensorimotor and higher level components, with different relative contributions to the individual walking phenotype. If on the one hand these mechanisms appear to play a functionally relevant role in the disturbed kinematic pattern of lower limbs, on the other hand they do not seem to be always directly involved in the anomalous motion of the axial segments. Rather, the very components of the pathophysiological profile can be interpreted, in several instances, as adaptive mechanisms (e.g. co-contraction), or turn out to produce compensatory or substitutary actions (e.g. spasticity). Localised deficits, moreover, such as increased passive stiffness of a single muscle-tendon group, can bring about a cascade of actions which will spread toward distant body segments including the axial chain, thereby resulting in disturbed postural alignment of pelvis, trunk, upper limbs and head.
Of interest in this connection, it was shown that, when freely adopted and externally imposed walking patterns (e.g. preferred versus non-preferred step frequencies) are compared in a same child with CP, a set of cost variables (including metabolic energy expenditure, asymmetry in lower limb movements and variability of intra- and inter-limb coordination) appear to attain minimal values in the former condition (Jeng et al. 1996). This indicates that individual solutions for walking in children with CP can satisfy optimality criteria, and suggests that prudence should be adopted in using normalcy indexes based on typical kinematic templates for ranking gait impairment in children with CP (see also Darrah 2003).

The evidence of a possibly relevant contribution of compensatory mechanisms to dynamic postural abnormalities of CP gait would be in keeping with the above posited moderate severity of primary dynamic postural deficits. Indeed, as mentioned above, the subpopulation of children usually considered for the analysis of gait disturbance mechanisms is characterised by relatively low functional impairment (e.g. bilateral spastic CP with preservation of independent walking function). Imaging studies in these forms indicate main involvement of executive components of the motor system (e.g. paraventricular portions of the corticospinal tracts; see Volpe 2001 for a review), which are not expected to be principal actors in the postural regulation. The possible dysfunction of second level (tuning) postural mechanisms (see Hadders-Algra et al. 1999), along with additional impairments of sensorimotor integration (e.g. central and perceptual visual deficits) and higher-level components, prompts the use of ad hoc designed paradigms for dissecting out more subtle primary dynamic postural deficits from compensatory or substitutory actions. Such a distinction is obviously crucial for conceiving rational guidelines for planning treatment interventions, so that research on this new field is expected to become a challenge both for the clinician and pathophysiologist over the next few years.

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