Ontogeny of postural adjustments during sitting in infancy: variation, selection and modulation

Mijna Hadders-Algra*†‡, Eva Brogren* and Hans Forssberg*

*Department of Woman and Child Health and Department of Neuroscience, Karolinska Institute, Stockholm, Sweden and †Department of Medical Physiology, University of Groningen, Groningen, The Netherlands

1. The aim of the study was to find out whether the development of postural adjustments occurs via a coupling of simple muscle responses, such as stretch reflexes, or via selection from an innate repertoire of centrally generated response patterns.

2. Postural responses during sitting on a moveable platform were assessed in eleven healthy infants at 5–6, 7–8 and 9–10 months of age. Multiple surface EMGs and kinematics were recorded while the infants were exposed to slow and fast horizontal forward (Fw) and backward (Bw) displacements of the platform.

3. From the youngest testing age onwards, largely variable but direction-specific muscle activation patterns were present. Fw translations resulted predominantly in an activation of the neck flexor, the rectus abdominis and rectus femoris muscle, while the neck-, thoracal- and lumbar extensor muscles (NE, TE, LE) and the hamstrings (Ham) showed varying amounts of inhibition. During Bw translations NE, TE, LE and Ham were preferably activated. The muscle activity could not be explained by simple stretch reflex mechanisms, but is likely to reflect centrally generated motor activity maturing in a predetermined way. However, indications for a contribution of stretch reflex mechanisms were also present.

4. With increasing age the variation in muscle activation patterns decreased, resulting in a selection of the most complete patterns. The ability to modulate the amplitude of the selected, most complete patterns during Fw translations, with respect to platform velocity and initial pelvis position, emerged at 9–10 months.

Postural control is a fundamental motor function, involved in nearly every other motor task. The many degrees of freedom of the vertebrate body make postural control complex. The nervous system solves the problem imposed by the redundant motor possibilities by a functional organization into basic and direction-specific synergies, which can be adapted to specific biomechanical constraints (Horak & Nashner, 1986; Dietz, Quintern & Sillem, 1987; Diener, Horak & Nashner, 1988; Keshner, Woollacott & Debû, 1988; Moore, Rushmer, Windus & Nashner, 1988; Forssberg & Hirschfeld, 1994; Macpherson, 1994). Examples of basic, direction-specific synergies are the patterns generated by perturbations inducing a forward sway during standing or sitting, which elicit a response pattern in the muscles on the dorsal side of the body, while perturbations inducing a backward sway evoke responses in the 'ventral' muscles. Postural synergies are interpreted in various ways: some favour an explanation in which reflex mechanisms play a predominant role (e.g. Dietz, 1992), while others suggest a less prominent role of reflexes by using terms like 'strategy' (Horak & Nashner, 1986; Macpherson, 1994) or 'central pattern generator' (CPG; Forssberg & Hirschfeld, 1994). In general, CPG activity is used to describe the neural organization of rhythmical movements like locomotion, respiration and mastication. The CPGs refer to neural networks co-ordinating the activity of many muscles. The activity level in these networks is controlled by reticulospinal neurons, whereas segmental afferent input, such as from stretch receptors, results in modulation of the output pattern (Grillner *et al.* 1995). Recently, Forssberg & Hirschfeld (1994) argued that the model of CPG circuitry could also be adopted for the explanation of neural mechanisms involved in postural adjustments. In order to integrate the fixed and flexible parts of postural control mechanisms, they proposed a CPG model organized into two functional levels, which are both active prior to response execution. At the first level the system selects the robust muscle activation pattern,

‡ To whom correspondence should be addressed at the Department of Medical Physiology.

whereas at the second level the selected pattern gets fine-tuned by multisensorial input from somatosensory, visual and vestibular systems.

The issue as to how complex automatic motor functions such as postural control develop during ontogeny is still a matter of controversy. Thelen and co-workers (e.g. Thelen & Cooke, 1987), who applied the dynamic systems theory in their studies on the development of infant walking, claimed a major effect of self-organization induced by learning-by-doing. According to this view, increasing experience induces a gradual build-up of the mature motor pattern (Woollacott, 1994). In contrast to this experience-guided view, and more in line with previous opinions (McGraw, 1943; Waddington, 1962), Forssberg (1985) and Hirschfeld & Forssberg (1994) suggested that motor development is canalized by endogenous maturation of predetermined neuronal connections. The latter point of view, in which neural mechanisms play a primary role, could be in line with Edelman's 'neural group selection' theory (Edelman, 1989; Sporns & Edelman, 1993). According to this theory, the first and fundamental step in development is the generation of several genetically determined primary neuronal groups, which are not precisely wired. Next, developmental tuning of the circuitry occurs: the neuronal groups are subject to experientially driven selection mediated by synaptic modifications of the neuronal group responses.

The aim of the present study was to investigate developmental changes in postural control. Sitting offers an early ontogenetic opportunity to study postural control in an upright position. Postural adjustments during sitting on a moveable platform were assessed in a group of eleven healthy infants, who were tested three times between the ages of 5 and 10 months. Each assessment consisted of simultaneous recording of EMGs and kinematic data during series of forward and backward translations of two different speeds. The following questions were addressed: (1) Are centrally generated, basic (i.e. direction-specific), muscle activation patterns present from the first recording age onwards? (2) Does the development of postural adjustments take place via a process of selection from a repertoire of primary muscle activation patterns, or is there a gradual development from a primitive to a mature pattern? (3) At which age are postural responses adapted to initial (sitting) position and stimulus intensity (platform velocity)?

Some of the present findings have already been published in preliminary form (Hadders-Algra, Brogren, Apel & Forssberg, 1994*a*, *b*; Hadders-Algra & Forssberg, 1995).

METHODS

Subjects

A group of eleven healthy infants (6 girls and 5 boys) participated in the study. They were assessed three times with an interval of 2 months at the ages of 5–6, 7–8 and 9–10 months. All parents gave informed consent and the procedures were approved by the Medical Ethical Committee of Karolinksa Hospital. At the first experimental session none of the children could sit independently. At this occasion the parents were asked to note the day on which the infant was able to sit independently for about 10 s. Prior to each assessment on the platform, neuromotor development was evaluated by means of Touwen's examination technique (Touwen, 1976).

Protocol

The infants sat on a moveable platform that could produce horizontal forward (Fw) and backward (Bw) translations of varying velocities and amplitudes (see Forssberg & Hirschfeld, 1994). Infants not able to sit independently were supported by the experimenter. Shortly before trial onset support was withdrawn, ensuring a freely sitting infant during the perturbation. Immediately after the trial, support was re-established. Care was taken to standardize conditions before trial onset as much as possible with respect to sitting position (head in the mid-line, trunk and legs as straight as possible) and behavioural state (non-crying, awake and alert). In order to document testing conditions the whole session was recorded on video. A standard block of trials consisted of sixteen slow perturbations (Fw: 120 mm s^{-1}, 500 ms duration; Bw: 180 mm s^{-1}, 333 ms duration), followed by sixteen fast ones (Fw: 180 mm s^{-1}, 333 ms duration; Bw: 220 mm s^{-1}, 272 ms duration), stimuli that were known to be well tolerated by infants (Hirschfeld & Forssberg, 1994). A higher platform velocity was chosen for Bw than for Fw translations because of the different response threshold for the two situations (Forssberg & Hirschfeld, 1994; Hirschfeld & Forssberg, 1994). Fw and Bw trials were presented in a random order.

EMG recordings

Bipolar surface electrodes with an interelectrode distance of 15 mm and an in-built amplification (MYO 115, Liberty

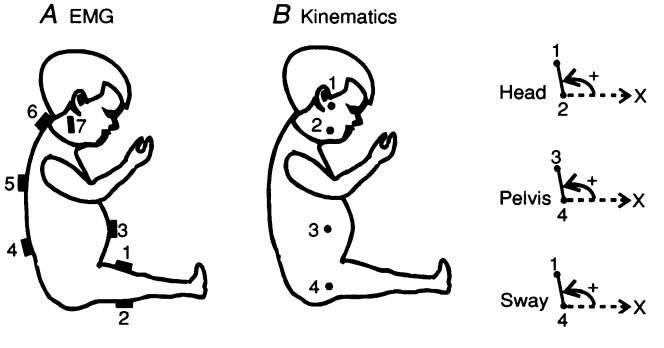

Figure 1. Schematic representation of the recorded signals

A, representation of electrode positions of recorded muscles: 1, RF (rectus femoris); 2, Ham (hamstrings); 3, RA (rectus abdominis); 4, LE (lumbar extensors); 5, TE (thoracal extensors); 6, NE (neck extensors); 7, NF (neck flexors). *B*, position of kinematic markers (1, caput mandibulae; 2, near angulus mandibulae; 3, anterior superior iliac spine; 4, trochanter major) and representation of calculated angles in the sagittal plane (head rotation, pelvis rotation and body sway in relation to the *X*-axis).

Technology, Hopkinton, MA, USA) were placed over the surface of the hip flexor (RF, rectus femoris), hip extensor (hamstring (Ham), biceps femoris), trunk flexor (RA, rectus abdominis), lumbar and thoracal extensor (LE, 1 cm paravertebral at the L3–4 level; TE, at the T8–9 level), neck extensor (NE, at the C5–6 level) and the neck flexor (NF, sternocleidomastoideus) muscles, on the left side of the body (Fig. 1*A*). LE recording was of unacceptable quality in six infants during the second assessment. As other recordings indicated that TE and LE were usually activated in concert, TE and LE were analysed together (TE–LE).

Kinematics

Simultaneous with the EMGs, movements were recorded by an ELITE system (Bioengineering Technology and Systems, Milan, Italy) with two charge-coupled device (CCD) cameras, at a sampling rate of 100 Hz. Reflective markers were put on the left side of the body on the following landmarks: (1) on the caput mandibulae, (2) 1 cm in front of the angulus mandibulae, (3) on the anterior superior iliac spine and (4) on the trochanter major (Fig. 1*B*). Additionally, three markers were placed on the lateral side of the platform. Kinematic data were recorded for 3 s, starting 1 s prior to perturbation. During some of the recording sessions the ELITE system was out of order. Moreover, recorded ELITE data were frequently incomplete because the two camera systems often lost track of a marker due to arm movements or slight rotations of the head.

Data acquisition and analysis

Only trials fulfilling the aforementioned criteria for sitting position and behavioural state were analysed. They were selected on the basis of the video-recording.

EMG analysis. The signals from the platform and the EMGs were sampled at 800 Hz, digitized at 12-bit resolution and stored on SC/ZOOM, a dedicated signal analysis computer system (Department of Physiology, Umeå University, Sweden). The software programme converted the signals to root mean square with a 6 ms moving window-averaging technique. A graphics terminal was used to interactively define EMG events for each trial separately. The interactive assessment offered the possibility to differentiate EMG bursts from regularly occurring electrocardiac activity, which was present especially in RA. EMG baseline activity was defined as the mean activity recorded 500 ms prior to each perturbation. A perturbation-related EMG burst was considered to be present when – during the time the platform moved – a burst occurred lasting at least 30 ms and exceeding baseline activity by 2 S.D.s. Likewise EMG inhibition was defined as a drop of activity below $-1{\cdot}5$ S.D. of the baseline level.

The EMG analysis was carried out in two steps (Fig. 2). First, muscle activation patterns were documented by describing the presence of bursts and inhibition in the recorded muscles. This resulted for each infant in each condition (Fw-slow, Fw-fast, Bw-slow, Bw-fast) in rates of EMG events per muscle and rates of muscle activation patterns. The response rates were calculated for each infant and each condition by dividing the number of trials during which a response or response pattern was found by the total number of trials.

Second, for the analysis of EMG amplitudes and latencies only trials were selected which had resulted in the most complete muscle activation patterns (Fw translations, extensor inhibition and activation of NF ± RA + RF; Bw translations, activation of NE + TE–LE + Ham, see Results). The trial selection prevented confounding by pattern variation. Latencies were defined as the time interval between the onset of platform movement and the onset of an EMG response. Amplitudes were computed by calculating the mean amplitude during periods of 100 and 400 ms, respectively, starting at the onset of the first burst belonging to the postural response. The baseline activity was subtracted from these 'raw' mean amplitude values. Mean latencies and amplitudes were calculated for each infant in each condition separately.

Processed EMG data such as muscle activity rates, mean latency and mean amplitude values per infant were compared between ages and conditions with the help of the Wilcoxon and Friedman test. Pearson's correlation coefficient was used for the calculation of correlations between functional sitting behaviour and items of the neurodevelopmental examination on the one hand and muscle activation patterns on the other hand.

Kinematic analysis. Off-line data processing consisted of the calculation of spatial angles for the head (by a vector between marker 1–2), the pelvis (markers 3–4) and the body sway (markers 1–4) in relation to the horizontal axis (Fig. 1*B*). The ELITE data were converted and analysed with the help of SC/ZOOM. The

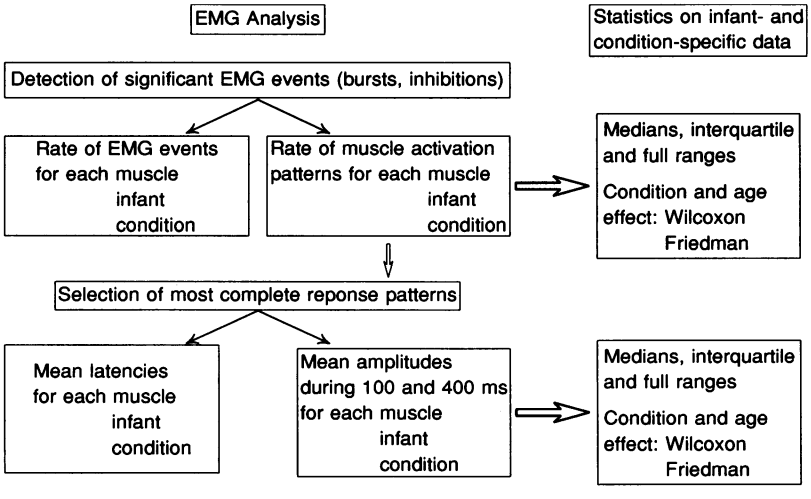

Figure 2. Diagram of EMG analysis

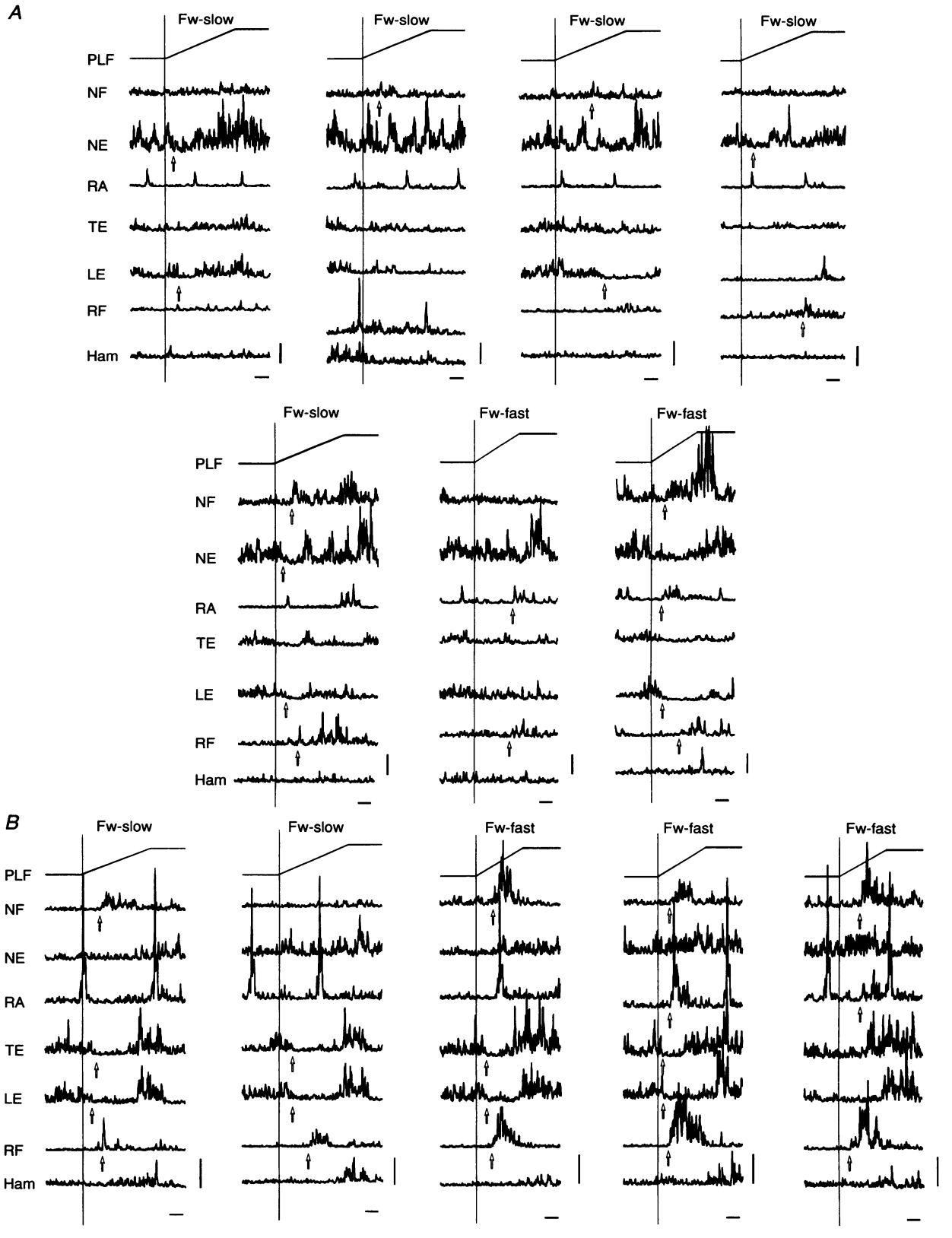

Figure 3. Response variation during Fw translations

Different response patterns exhibited by infant B at 5 months (*A*) and 9 months (*B*) during single Fw translations. *A*, 5 slow and 2 fast trials; *B*, 2 slow and 3 fast trials. Time calibration (horizontal bar), 100 ms; amplitude calibration (vertical bars) 0·01 and 0·05 mV for *A* and *B*, respectively. The arrows indicate the onset of a direction-specific muscle response (excitation or inhibition). PLF, platform signal. Here and in subsequent figures see Fig. 1 for the abbreviations of the muscles.

analysis focused on: (1) angular values at perturbation onset (ANGON), (2) angular values at the peak of the induced movement, (3) angular displacements, defined as the difference between ANGON and angular values at specific moments during the perturbation (at 60 ms (Bw translations) and 100 ms (Fw translations) after platform onset and at movement peak) and (4) latencies to the onset of the elicited movements. Pelvis rotation analysis was carried out in nine, ten and four infants at 5–6, 7–8 and 9–10 months, respectively, whereas the analysis of head rotation and body sway at each age was performed on the two infants with the most complete kinematic recordings. For the majority of kinematic data only descriptive statistics were used (see Table 1). Pearson's correlation coefficient was applied for the calculation of correlations between ANGON data and muscle activation patterns and EMG amplitude.

Throughout the analyses, differences and correlations with a P value $< 0{\cdot}05$ were considered to be statistically significant.

Additional experiments

The infant study showed that Fw translations consistently evoked an inhibition of the extensor muscles (see Results). Normally, in adults such an inhibition is absent (Forssberg & Hirschfeld, 1994). In order to see whether the inhibition was due to a high level of background activity in the extensor muscles, three healthy adults were tested. Two consecutive standard series were run, the first one with the subject sitting with the arms folded at the waist, the other with the subject holding an object (size, $20 \times 30 \times 5$ cm^3, weight, 2·4 kg) above the head. The choice for the latter procedure was based on empirical findings. We selected a relatively easy task which induced a high baseline activity in the dorsal trunk muscles. No kinematic data were recorded.

RESULTS

The number of successful trials per infant for the various conditions (Fw-slow, Fw-fast, Bw-slow, Bw-fast) at the different test ages ranged between one and eighteen, with a median value of eight at 5–6 and 7–8 months of age, and five at 9–10 months. The lesser number of successful trials during the last examination was due to the fact that the older infants often started to cry after a limited number of trials.

From the youngest testing age onwards Fw translations resulted in a predominant activation of the 'ventral' muscles (the flexors NF, RA and RF), while the 'dorsal' muscles (the extensors NE, TE, LE and Ham) showed varying amounts of inhibition (Fig. 3). During Bw translations the extensor muscles were mainly activated (Fig. 4). Still, response organization changed considerably with increasing age.

Muscle activation patterns

At 5–6 months both Fw and Bw translations elicited a large variation in muscle activation patterns in subsequent trials consisting of any combination of the direction-specific 'ventral' or 'dorsal' muscles (For Fw translations see Fig. 5). At this age, patterning was independent of platform velocity. With increasing age the number of patterns decreased, resulting in a few preference patterns. During Bw translations the preference pattern at 9–10 months

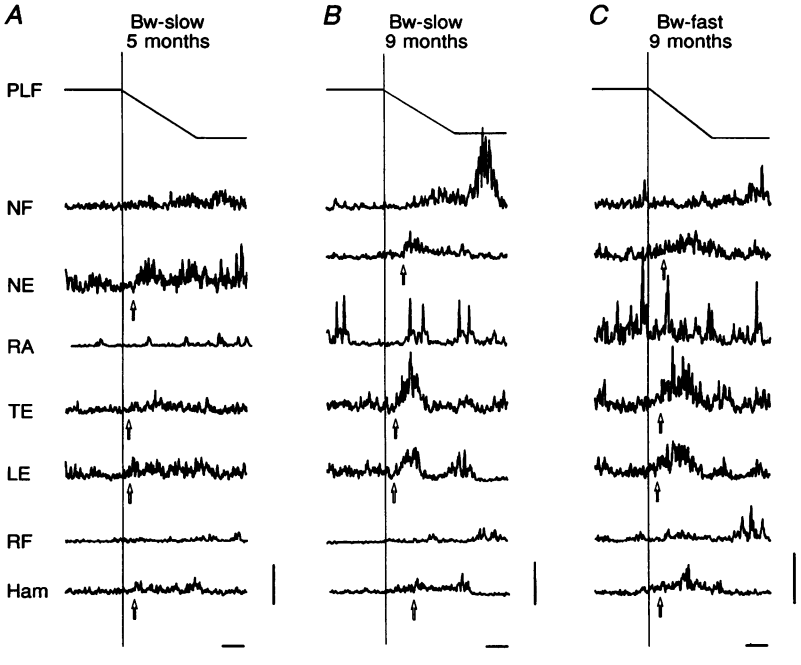

Figure 4. Responses during Bw translations

Response activity of infant B in average EMGs of all Bw trials during which all 'dorsal' muscles were activated. Average activity during 2 slow trials at 5 months (*A*), 4 slow (*B*) and 3 fast trials (*C*) at 9 months. Time calibration (horizontal bar), 100 ms; amplitude calibration (vertical bars) 0·01 and 0·05 mV at 5 and 9 months, respectively. The arrows indicate the onset of a direction-specific muscle response.

consisted of the activation of all extensor muscles. During Fw translations two patterns became predominant: E-Inh + NF + RA + RF and E-Inh + NF + RF, i.e. extensor inhibition (E-Inh) with NF and RF activation, with or without RA participation (Fig. 5).

During slow and fast Bw translations the frequency of the total extensor pattern (NE + TE–LE + Ham) increased significantly from about 25% (median value) at 5–6 months to about 80% at 9–10 months. The complete Fw pattern (the 'red' pattern in Fig. 5: E-Inh + NF + RA + RF) was

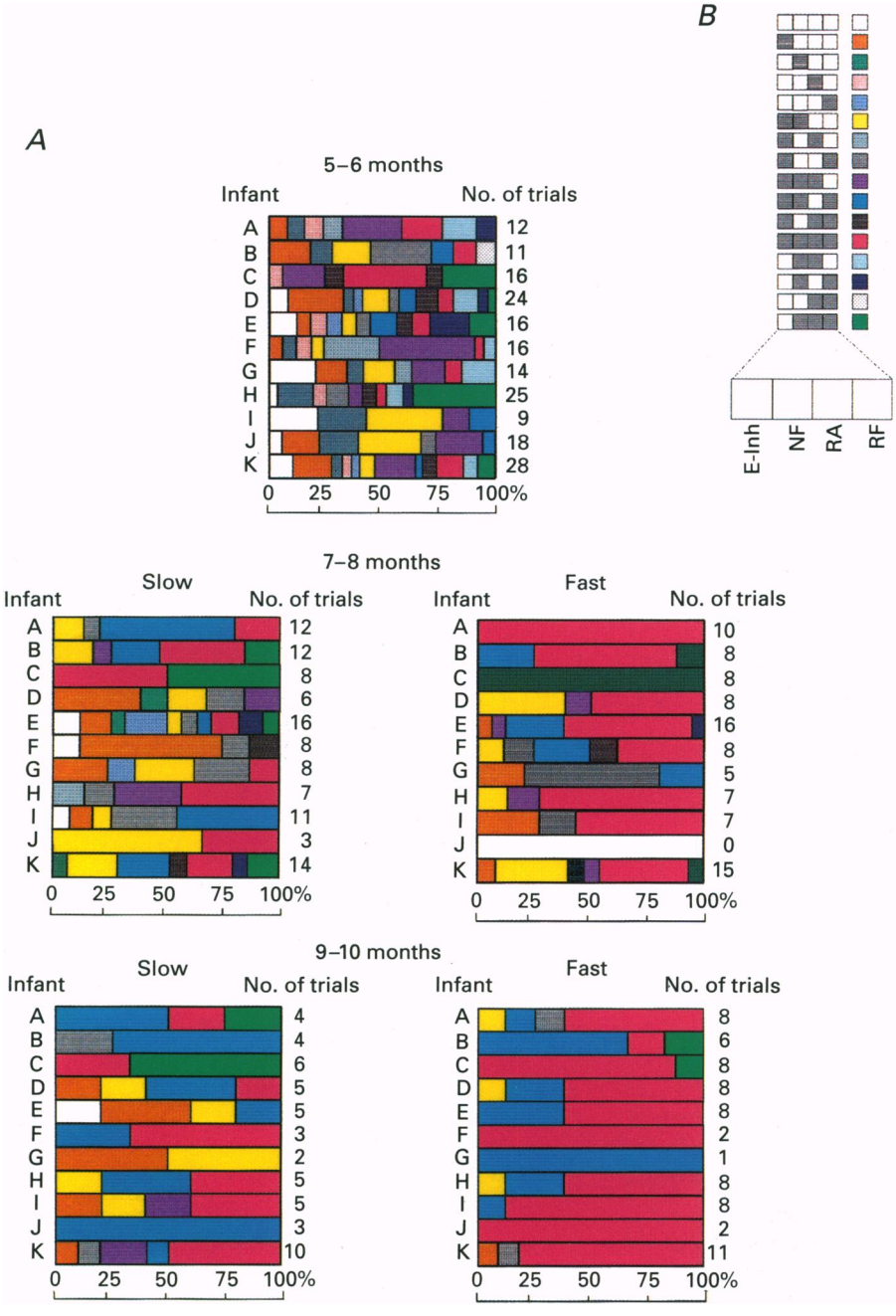

Figure 5. Developmental changes in response pattern during Fw translations

A, developmental changes in response pattern during slow and fast Fw translations. The data of the slow and fast trials at 5–6 months were pooled as a velocity effect on patterning was absent at this age. Each horizontal bar represents the distribution of response patterns for one subject. At 5–6 months none of the infants could sit independently, at 7–8 months all infants except infant E and H could sit alone and at 9–10 months all infants could sit without help. The explanation of the colour codes for the response patterns is given in *B*. Hatching of a square indicates participation of a muscle in a particular pattern. E-Inh, extensor inhibition, i.e. inhibition of (any combination of) NE, TE–LE or Ham.

rarely present at 5–6 months, whereas it was observed in about 25% of the slow trials and in about 60% of the fast trials at 9–10 months. In general, the frequency of the complete pattern was higher during Bw translations than during Fw translations. The difference in the rate of occurrence of the complete pattern between Fw and Bw translations was statistically significant for slow and fast perturbations at 5–6 months ($P < 0{\cdot}01$) and for the slow translations at 9–10 months ($P < 0{\cdot}01$). This difference could be due to the different velocities used for the perturbations in the two directions. However, comparison of translations with the same velocity (i.e. Bw-slow and Fw-fast), revealed that the above mentioned differences remained significant (5–6 months: $P < 0{\cdot}01$; 9–10 months: $P < 0{\cdot}05$).

The time interval between the first assessment and the age at which the child could sit independently varied from 7 to 98 days. The amount of complete pattern observed during Bw or Fw translations during the first examination was not related to this time interval. Neither was the period of time the infant had been sitting independently related to the amount of complete pattern found during Bw and Fw translations during the second examination. Also, no relation could be established between the amount of complete pattern during Bw and Fw translations and the developmental performance on items of the neurological examination such as the lateral supporting reaction, rotation during sitting, head and arm posture during pull-to-sit, grasping and crawling behaviour.

Direction specificity

At 5–6 months NE, TE–LE and Ham showed an EMG burst significantly more often during Bw than during Fw translations, whereas NF and RA bursts occurred mainly during Fw translations. In contrast, RF was activated equally often during Bw and Fw translations. Only from the second assessment onwards all muscles showed direction-specific responses (Fig. 6).

During Fw translations an inhibition of the extensor muscles was frequently found. This inhibition, usually

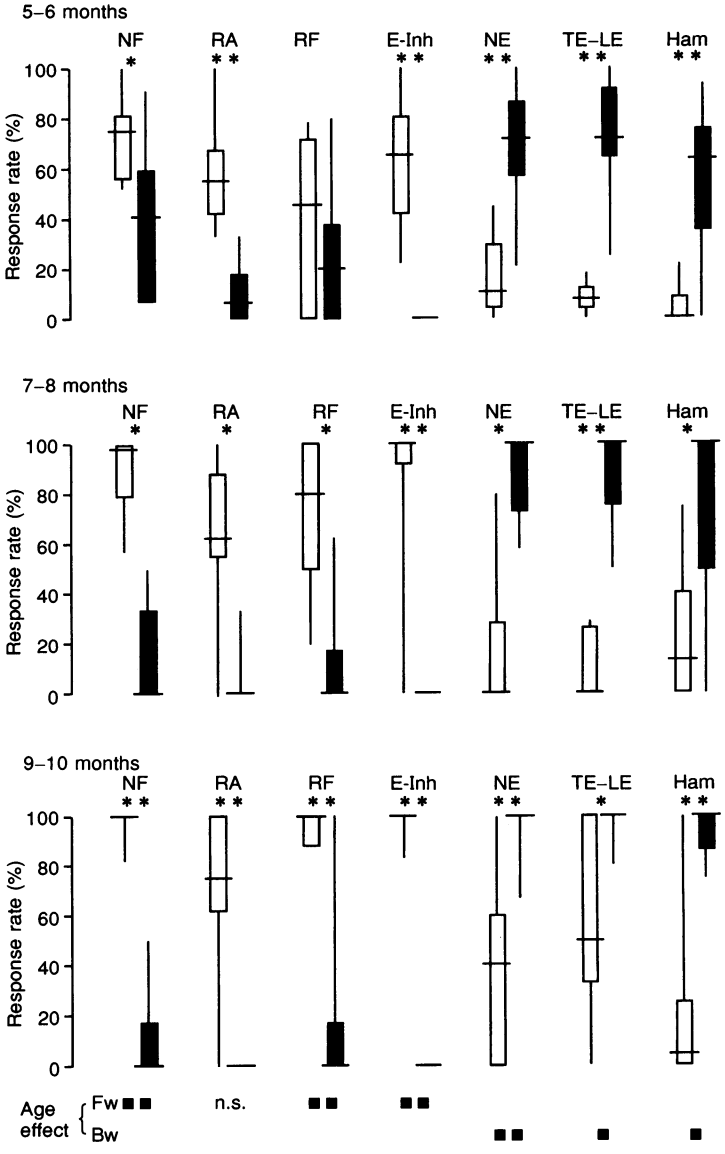

Figure 6. Response rates during fast Fw and Bw translations

Group data on response rates of specific muscles during fast Fw and Bw translations at the 3 different test ages (see Methods). A response consists of a burst unless otherwise specified. The data are presented by ranges (vertical bars), interquartile ranges (boxes) and median values (horizontal bars). The absence of the interquartile boxes in some of the graphs of the oldest infants symbolizes identical median and interquartile values which, in turn, points to a small variation in the observed data. □, data on Fw translations; ■, data on Bw translations. Asterisks indicate statistically significant differences in the distribution of the response rates between Fw and Bw translations (Wilcoxon): * $P < 0{\cdot}05$, ** $P < 0{\cdot}01$; black squares denote significant changes in the distribution of the response rates with age in the muscles preferred in a specific condition (Friedman): ■, $P < 0{\cdot}05$, ■ ■, $P < 0{\cdot}01$. E-Inh, extensor inhibition, i.e. inhibition of (any combination of) NE, TE–LE or Ham.

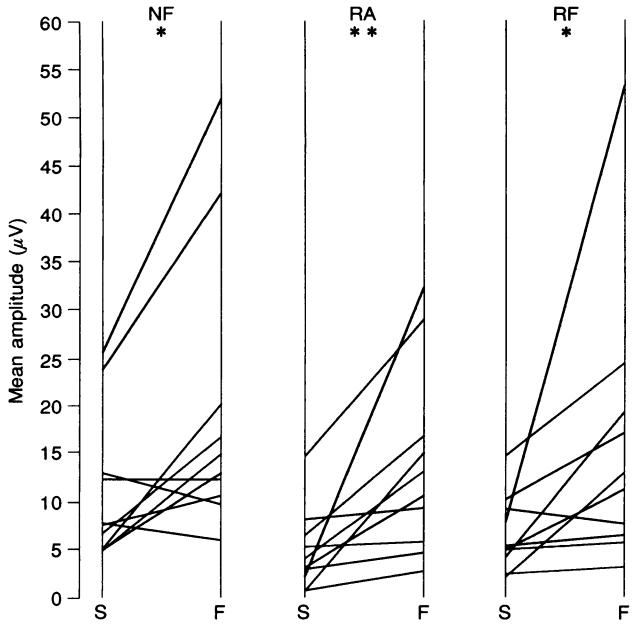

Figure 7. Effect of platform velocity on mean burst amplitude of flexor muscles during Fw translations

Mean amplitude of NF, RA and RF during the first 400 ms after response onset plotted for slow (S) and fast (F) translations, respectively. Each line connects the mean of the amplitude values per infant. Data on infant G are missing, because she had no E-Inh + NF ± RA + RF pattern during slow Fw translations. The group effect of platform velocity is indicated with asterisks (Wilcoxon): * $P < 0{\cdot}05$, ** $P < 0{\cdot}01$.

present in TE–LE, frequently in NE and only rarely in Ham, occurred in about 65% (median value) of the Fw trials at 5–6 months and in nearly all Fw trials at 9–10 months (Fig. 6).

Spatial distribution of muscle responses

During Fw translations at 5–6 months, NF was activated significantly more often than RA and RF ($P < 0{\cdot}01$). The frequency of responses in NF and RF increased with increasing age. NF activation rose from 70% of the slow and 75% of the fast trials at 5–6 months, to a response rate of about 100% at 9–10 months. RF response rate, which was quite low initially (median value: slow, 27%; fast, 46%), increased to nearly 100% at 9–10 months. In contrast, RA did not show a significant developmental change in response frequency (Fig. 6).

The various extensors were already activated in about 50–70% of the Bw trials at 5–6 months. The response rate was not significantly different for NE, TE–LE or Ham. At the last examination the extensors virtually always responded (Fig. 6).

Effect of platform velocity

The variation in muscle activation pattern was not affected by platform velocity during the first examination. However, from the second assessment onwards an increase in platform velocity was associated with a reduction in the

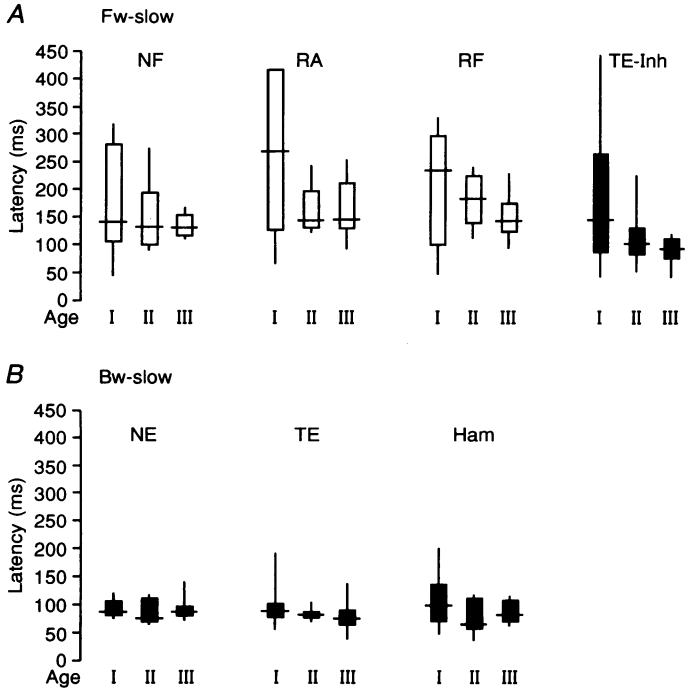

Figure 8. Latencies to response onset during slow perturbations

Group data on latencies to response onset during slow Fw (*A*) and Bw (*B*) translations. The data are presented by ranges (vertical bars), interquartile ranges (boxes) and median values (horizontal bars). □, data on flexor muscles; ■, data on extensor muscles. Age I, 5–6 months; age II, 7–8 months; age III, 9–10 months.

number of muscle activation patterns, resulting in a more frequent use of the most complete patterns (Fig. 5). This held true for Fw and Bw translations.

During Bw translations platform velocity never affected EMG burst amplitude. During Fw translations such an effect was absent at 5–6 and 7–8 months, but it did emerge at 9–10 months of age. Mean flexor response amplitude, calculated during the first 400 ms after response onset was significantly higher during fast than during slow Fw translations (Fig. 7). No such velocity effect was found on mean flexor amplitude calculated during the first 100 ms of the response.

Our protocol consisted of sixteen slow, randomly presented Bw and Fw perturbations, followed by sixteen fast, random Bw and Fw translations. This trial sequence could have induced certain expectancies and, consequently, a central presetting of response amplitude (Horak, Diener & Nashner, 1989). However, such an effect was never observed.

Latencies and sequential order

No statistically significant developmental changes were found in the latencies to the various EMG responses. Yet two ontogenetic trends could be noted, especially in the flexor muscles during Fw translations (Fig. 8). First, at the youngest ages the latency values of the flexor muscles showed a substantial variation. This variation decreased with increasing age. Second, the latency to RF onset tended to decrease (median value at 5–6 months: 232 ms; at 9–10 months: 142 ms), whereas the median value to the onset of NF remained nearly constant (130–142 ms).

The latencies to the onset of extensor inhibition during Fw translations were shorter than those to the flexor bursts, significantly so at the third examination (slow trials: $P < 0{\cdot}05$; fast trials: $P < 0{\cdot}01$). Extensor activity during Bw translations started throughout the age period studied some 70–100 ms after platform onset (Fig. 8). Platform velocity had no effect on latency values.

The order in which the muscles were activated showed a large variation. At 5–6 months responses during which the neck muscle was activated prevailed first, both during Bw and Fw translations. At 9–10 months the responses started less often in the neck muscle, in favour of a start in the leg. The developmental changes in temporal ordering did not reach statistical significance.

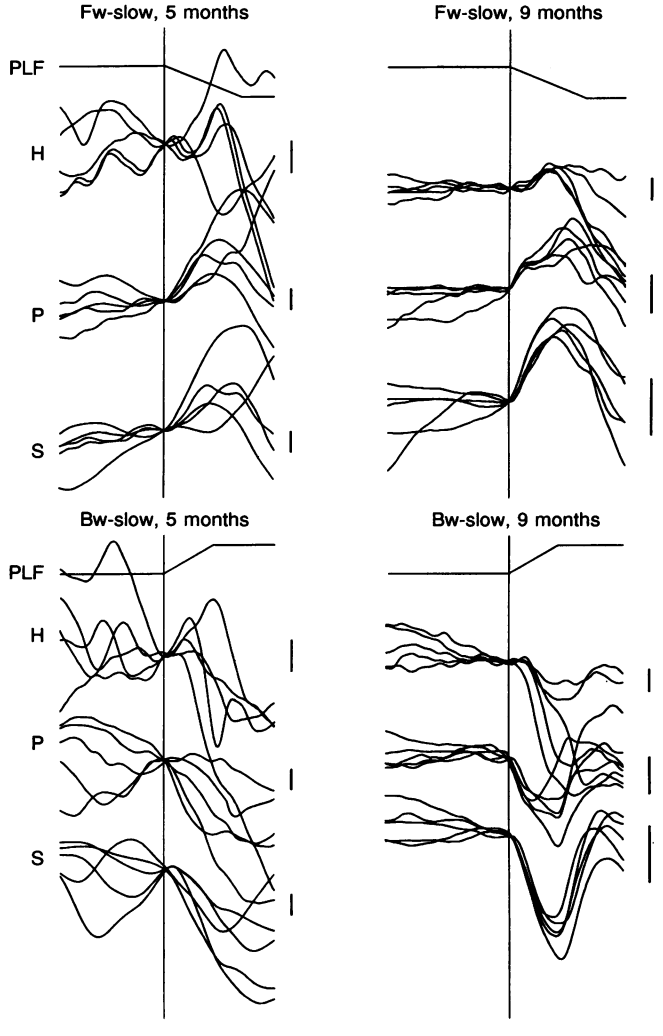

Figure 9. Kinematic recordings at 5 and 9 months
Kinematic recordings of spatial angles in the sagittal plane of head, pelvis and body sway during slow Fw and Bw translations of infant E at 5 and 9 months. The traces were aligned with respect to (largely varying) starting position at platform onset. The recordings of the first 5 trials in a specific condition are presented. PLF, platform signal; H, head rotation; P, pelvis rotation; S, body sway (upward deflexions denote backward rotation (H, P) and backward fall (S). Time calibration (horizontal bar), 100 ms; angle calibration (vertical bars), 5 deg.

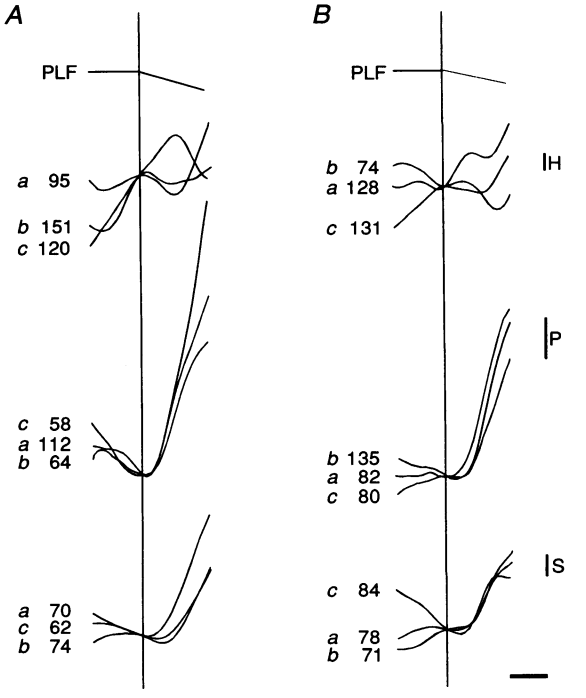

Figure 10. Pre-trial kinematics

Kinematic recordings of spatial angles in the sagittal plane of head, pelvis and body sway during fast (*A*) and slow (*B*) Fw translations. The traces were aligned with respect to starting position; the numbers on the left side indicate the angular values at platform onset. *A*, 3 trials with more or less similar pre-trial dynamics of infant D at 6 months resulting in different EMG response patterns; *a*, E-Inh + RA + RF; *b*, NF + RF; *c*, E-Inh + NF + RF. *B*, 3 trials with different pre-trial dynamics of infant I at 9 months resulting in the most complete ('red') EMG pattern. PLF, platform signal; H, head rotation; P, pelvis rotation; S, body-sway (upward deflexions denote backward rotation (H,P) and backward fall (S). Time calibration (horizontal bar), 100 ms; angle calibration (vertical bars), 1 deg.

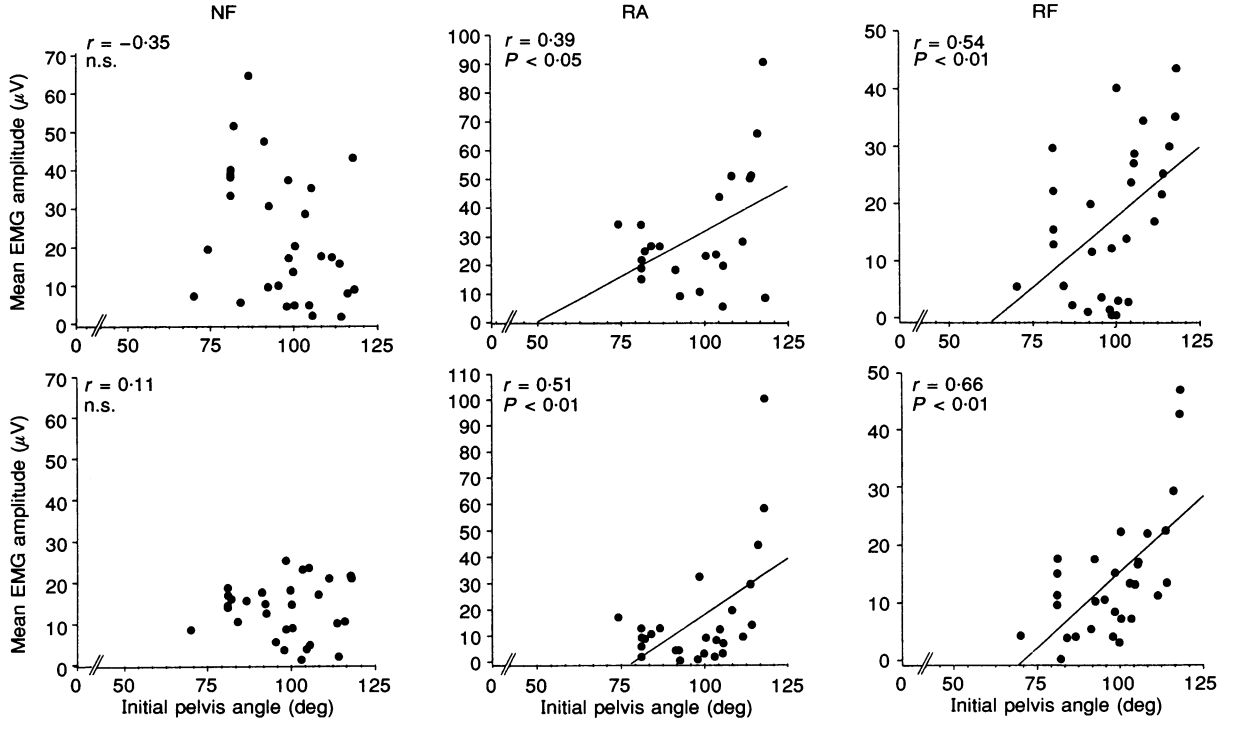

Figure 11. Correlation between initial pelvis angle and EMG amplitudes during fast Fw translations at 9–10 months

Correlations between EMG amplitudes of NF, RA and RF during the first 100 ms (upper panels) and 400 ms (lower panels) after response onset and initial pelvis angle during fast-Fw translations at 9–10 months. Data on 31 trials of four infants.

Table 1. Latencies (mean ± S.D. in ms) to onset of movements, calculated for a varying number of trials (numbers in parentheses) of two infants

Age (months)	Bw-slow	Bw-fast	Fw-slow	Fw-fast
	Pelvis rotation			
5–6	22 ± 30 (21)	14 ± 15 (17)	12 ± 16 (23)	14 ± 21 (20)
7–8	5 ± 9 (18)	5 ± 7 (16)	12 ± 24 (19)	6 ± 11 (18)
9–10	10 ± 11 (8)	10 ± 16 (10)	10 ± 14 (20)	6 ± 10 (15)
Age (months)	Head rotation			
5–6	113 ± 75 (20)	95 ± 51 (16)	132 ± 85 (23)	113 ± 93 (20)
7–8	86 ± 40 (19)	113 ± 39 (16)	106 ± 35 (19)	107 ± 52 (18)
9–10	110 ± 43 (8)	127 ± 55 (10)	111 ± 36 (20)	132 ± 31 (15)

Biomechanical parameters: a reflection of afferent input and motor output

Afferent input

Trial selection was carried out according to strict, video-based criteria (see Methods). Nonetheless, the children showed a large variation in the position of the various body parts at platform onset. For example, in a particular 6-month-old child the initial spatial angles in the sagittal plane for the pelvis and the head varied between 50–86 and 72–151 deg, respectively. Notwithstanding this variation in starting position, the platform translations evoked a body sway and pelvis rotation in a consistent direction in all infants, including the youngest ones, who were not able to sit independently. The rotational movements of the head followed a less predictable course, especially so at 5–6 months (Fig. 9).

To see whether the elicited responses at the youngest age could be explained by stretch reflex activity, the amount of stretch of a muscle as reflected by the kinematic data, was related to the presence or absence of a burst in the specific muscle. Neck muscle activity occurred already often at the youngest ages, but it was not related to the direction of head rotation during the first 60 ms (Bw translations) or 100 ms (Fw translations). For example, 56% of slow Fw translations resulting in a backward rotation of the head (and consequently stretching of NF) elicited a NF burst, but also 80% of the slow-Fw trials with a forward rotation of the head (resulting in a NF lengthening) showed NF activation. Leg muscle activity occurred relatively seldomly at the youngest age, notwithstanding a consistent stretch of the leg muscles through pelvis rotation. No significant relation could be established between the amount of pelvis displacement at 60 ms (Bw translations) or 100 ms (Fw translations) and the presence of leg muscle activity. We wondered whether the large variation in muscle response patterns at the youngest ages could be explained by the variation in conditions at perturbation onset, such as the static position or the on-going movement of the various body segments. However, no distinct relations between the afferent and efferent variation could be found. For example, the few trials with similar pre-trial dynamics at 5–6 months elicited different EMG response patterns, whereas identical EMG patterns at 9–10 months were associated with different pre-trial dynamics (Fig. 10).

The variation in starting position of the head was not related to the variation in EMG response pattern at any age. Neither were the postural responses during the first examination influenced by the initial pelvis position. During slow-Fw translations at 7–8 months, a weak but statistically significant correlation was found between the initial pelvis angle and the amount of the most complete response patterns present (E-Inh + NF ± RA + RF, $r = 0{\cdot}33$, $P < 0{\cdot}01$). During none of the other conditions and ages could an effect of initial pelvis position on muscle response pattern variation be established.

Initial head position did not affect EMG burst amplitude at any age. However, whilst initial pelvis position did not influence amplitude at 5–6 and 7–8 months of age, at 9–10 months, it showed a significant correlation with the mean amplitude of RA and RF during fast Fw translations. This correlation was stronger for the mean values over 400 ms than those calculated for the first 100 ms period (Fig. 11). During slow Fw translations only mean RF amplitude was positively correlated to pelvis angle (mean over 100 ms (RF_{100}): $r = 0{\cdot}52$, $P < 0{\cdot}05$; mean over 400 ms (RF_{400}): $r = 0{\cdot}61$, $P < 0{\cdot}01$). During Bw translations similar relationships between pelvis position and mean amplitude values over 400 ms were absent at any of the ages studied.

Motor output

The onset of pelvis rotation occurred soon after the onset of platform movement (mean: 5–22 ms, Table 1). The latencies to the onset of head rotation varied largely and were considerably longer than those to the onset of pelvis rotation (mean: 86–132 ms, Table 1).

The perturbations elicited varying amounts of body sway and rotation of the head and pelvis, especially at the youngest ages. To see whether the selection of response patterns could be guided by the afferent input produced by

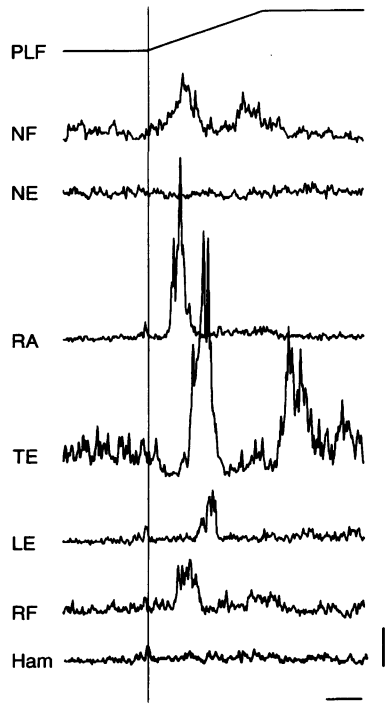

Figure 12. Extensor inhibition in TE during Fw translations in an adult.

Average EMGs of 8 trials of an adult holding a heavy object above the head during fast Fw translations. PLF, platform signal. Time calibration (horizontal bar), 100 ms; amplitude calibration (vertical bars), 0·1 mV.

the EMG pattern, we tested relationships between EMG patterns and maximal angular displacement of the various body parts at 7–8 months, i.e. the only age at which considerable pattern variation and a substantial quantity of the complete pattern are seen. If such a motor output guidance would be present, a complete pattern should cause a smaller displacement of the body parts than a non-complete pattern. Indeed, in the data of the two infants with the best kinematic data such a relationship could be established for head rotations during fast (Fw and Bw) translations. Both infants demonstrated that a complete EMG pattern resulted significantly more often in a small head rotation ($\leqslant$ 20 deg) than non-complete EMG patterns (Fisher exact test for each infant: Fw translations $P < 0{\cdot}01$, Bw translations $P < 0{\cdot}05$). Similar associations were absent during slow perturbations, probably because slow trials failed to elicit large head rotations. EMG patterning was not related to the angular displacement of the pelvis and of body sway.

Extensor inhibition during Fw translations in adults

During standard Fw translations the three adults showed consistent postural responses in NF, RA and RF with onset latencies compatible with those reported by Forssberg & Hirschfeld in 1994 (RF, 67 $\pm$ 8 ms; RA, 80 $\pm$ 15 ms; NF, 94 $\pm$ 16 ms). In the condition with the heavy object held above the head, which induced a high level of background activity in NE and TE, an inhibition of NE, and especially of TE, was added to the flexor muscle activation pattern (Fig. 12). The latencies to TE inhibition were significantly shorter than those to flexor activation (paired t test, $P < 0{\cdot}0001$; TE inhibition, 41 $\pm$ 11 ms; RF, RA and NF activation: 75 $\pm$ 17, 68 $\pm$ 13 and 58 $\pm$ 12 ms, respectively).

DISCUSSION

The present study revealed that infants of 5–6 months, just on the verge of learning to sit independently, exhibited a large variation of direction-specific muscle activation patterns when they were perturbed during sitting. With increasing age the variation in muscle activation patterns decreased, resulting in selection of the most complete patterns. At 9–10 months the selected patterns could be modified by platform velocity and initial sitting position.

Direction specificity

To allow assessment of postural adjustments in infants not able to sit independently, the 5- to 6-month-old infants were provided with postural support until 0·5–1·0 s before trial onset. Withdrawal of postural support in young infants is known to elicit a Fw sway of the body and a preferential activation of the dorsal muscles (Harbourne, Guiliani & MacNeela, 1993). Such withdrawal-related responses could have interfered with responses induced by the platform movement, but the kinematic recordings and the EMG responses indicated that this was not the case. The platform movements consistently elicited a sway of the body in the opposite direction (Fig. 9), which consequently evoked direction-specific EMG responses (Bw translations, activation of extensor muscles; Fw translations, activation of flexor muscles combined with extensor inhibition).

Similar direction-specific responses have been demonstrated in standing adults (Horak & Nashner, 1986; Keshner *et al.* 1988), standing children (Forssberg & Nashner, 1982; Woollacott, Debû & Mowatt, 1987), sitting adults (Forssberg & Hirschfeld, 1994) and independently sitting children (Hirschfeld & Forssberg, 1994). Two findings indicated that such direction-specific muscle responses cannot solely be explained by stretch reflex mechanisms. First, Forssberg & Hirschfeld (1994), who performed Bw sway-inducing perturbations in adults, reported that Fw translations, which cause a stretch of RF, and legs-up rotations, which induce a RF relaxation, both result in a response of RF. Second, in the present study, the youngest infants showed significant EMG bursts in the neck muscles whether or not a trial had resulted in stretch of the particular muscle. It therefore can be surmised that the afferent input generated by the postural destabilization triggers a centrally generated response pattern. The fact that the muscle activation patterns are not composed of stretch reflexes does not exclude a contribution of muscle stretch reflexes to the final motor output.

The present study showed, in contrast to previous studies (Forssberg & Hirschfeld, 1994; Hirschfeld & Forssberg, 1994), that extensor inhibition is an intrinsic part of the basic postural response pattern during Fw translations. Not only was extensor inhibition consistently present during infant responses, but also in adult responses when baseline activity in the extensor muscles was sufficiently high to disclose the phenomenon of 'automatic gain compensation' (Matthews, 1986).

The finding of direction-specific postural adjustments in infants not able to sit independently contrasts with the report of Woollacott *et al.* (1987), that 3- to 5-month-old 'non-sitters' lack direction-specific responses. The absence of direction-specific postural adjustments in the latter study might be explained by the support of body and head provided during the perturbations, which is known to inhibit or reduce the gain of postural responses (cf. Cordo & Nashner, 1982). This corresponds to the large attenuation of EMG responses we observed in the few trials omitted from the analysis because of delayed withdrawal of postural support. The study of Harbourne *et al.* (1993) on postural responses after removal of trunk support in sitting infants indicated that a direction-specific activation of the dorsal muscles was present in 4- to 5-month-old infants, but absent in those aged 2–4 months. The above findings indicate that, even before the milestone age of sitting independently, the nervous system develops the ability to detect and store relevant afferent information on postural stability, such as the orientation of the vertical axis and the relation of the centre of mass to the margins of the support surface, in a so-called internal representation of the body (Gurfinkel, Lipshits, Mori & Popov, 1981; Mittelstaedt, 1983). From their very beginning onwards, postural responses appear to be guided by information on direction-specific stability limits stored in this internal body representation.

Variation and selection

At 5–6 months of age, response patterns were extremely variable, consisting of the activation of any combination of the direction-specific muscles. A fundamental question is whether this variation is a primary characteristic of the function of the young nervous system or can be attributed to variations in afferent input. It is well known that differences in starting conditions can modify reflexes to a large extent (Prochazka, 1989). However, in the present study we were not able to demonstrate a clear relationship between the variation in starting conditions, such as the direction and velocity of head rotation and body sway and the sitting position before trial onset, and the variation in EMG response patterns. This does not, of course, preclude a contribution of a variable afferent input to the variation in pattern output, but our results suggest that the EMG response variation at the youngest ages is a genuine functional expression of the young nervous system due to immaturity of afferent, central and efferent parts of the neural circuitry controlling postural adjustments. Earlier EMG studies on young subjects also reported variation as a striking finding (Thelen & Cooke, 1987; Bradley & Bekoff, 1990; Cazalets, Menard, Crémieux & Clarac, 1990; Hadders-Algra, Van Eykern, Klip-Van den Nieuwendijk & Prechtl, 1992; Harbourne *et al.* 1993). In the present study we found, like Cazalets *et al.* (1990) and Hadders-Algra *et al.* (1992), that variation decreased with increasing age. The decrease in response variation during Fw and Bw translations probably can be explained by two complementary mechanisms: (1) selection from a repertoire of primary response patterns and (2) a maturation-induced increasing efficiency of signal transport in the circuitry of the complete response patterns, in analogy to developmental processes occurring during the development of human locomotion (Berger, Altenmüller & Dietz, 1984; Forssberg, 1985). An indication of the second mechanism is the (hazardous to interpret) finding that the surface EMG response amplitudes tended to increase with increasing age (see Figs 3 and 4). However, analysis revealed that baseline activity and response amplitudes in general did not show significant age-dependent changes (exception RF_{400} during slow Fw translations, which just reached the 5% significance level). The presence, however, of clear-cut complete response patterns at the youngest testing age suggests that selection also plays a substantial role in the development of postural adjustments. Such a developmentally induced response selection is in perfect agreement with Edelman's 'neuronal group selection' theory. This theory proposes that variation reduction is due to experientially driven selection of circuits ('neuronal groups') resulting in the most successful output (Edelman, 1989; Sporns & Edelman, 1993). More recently, Georgopoulos (1995) also suggested that selection from an innate repertoire of specific networks might be a central

mechanism in the development of directional motor control. In this respect it is interesting to note that the selection of a complete EMG response pattern was associated with a smaller angular displacement of the head. Stabilization of the head in space is an important aspect of postural control: it provides a stable gravitational reference for the vestibular system and it facilitates the processing of visual information (Pozzo, Berthoz & Lefort, 1990). Possibly the afferent systems in the head, in particular the vestibular system, guide the selection of the most appropriate postural responses. Thus, in our point of view, the early ontogeny of postural adjustments during sitting is primarily guided by two distinct processes. The first and fundamental one implies access to an epigenetically determined variable repertoire of motor programmes with direction-specific responses; the second consists of a mainly experientially driven selection, a process which is enhanced by concurring improvement of synaptic efficiency within the complete pattern's circuitry.

The presence of the most complete patterns at the youngest testing age refutes the concept of a gradual development, in which motor components are added in a cephalo-caudal fashion (McGraw, 1943; Touwen, 1976) or a caudal-to-cranial order (Woollacott, 1994). Still, it is noteworthy that within the patterns at the youngest age the neck muscles were more frequently activated than the leg and trunk muscles, especially during Fw translations, which again points to the priority of head stabilization in postural control. With increasing age, the increase in response rate in the extensors (during Bw translations) followed a cephalo-caudal ordering, but in the flexors (during Fw translations) it was RF that increased response rate secondly, with RA coming third or not at all. Remarkably, the developmental changes in the postural response patterns were never related to the performance on the items of the neurological examination or to the emergence of the ability to sit independently.

Within the selected, most complete patterns, temporal coordination developed also via a process of selection. At 5–6 months of age the latencies to the onset of muscle responses were characterized by a large variation, especially so in the flexor muscles. A decreasing variation with increasing age in temporal aspects of EMG events of young subjects has been reported before (Bradley & Bekoff, 1990; Cazalets *et al.* 1990; Hadders-Algra *et al.* 1992). The large variation in temporal organization at the youngest ages could be attributed either to imprecise tuning characteristics of the newly selected motor circuitry itself, to an inaccurate coupling to afferent inputs, or to a weakness in supraspinal control (see also Cazalets *et al.* 1990). Remarkably, the youngest infants preferred to activate their neck muscles first, which differs from the bottom-up recruitment normally present in standing and sitting adults (Horak & Nashner, 1986; Forssberg & Hirschfeld, 1994) and sitting children (Brogren, Hadders-Algra & Forssberg, 1996). It reflects the primacy of head control during the first phases of the development of postural adjustments (Assaiante & Amblard, 1995).

Modulation of responses

At the age of 9–10 months the platform velocities we used during the present study started to have a modulatory effect on the EMG amplitude of the selected muscle activation patterns. The velocity ratio of fast *versus* slow trials in the present study (fast : slow ratio; Bw, 1·2; Fw, 1·5) was considerably smaller than the ones used in other studies (fast : slow ratio $\geqslant 3$; Allum & Pfaltz, 1985; Diener *et al.* 1988; Dietz, Horstmann & Berger, 1989). Our findings indicate that at 9–10 months, the nervous system develops the capacity to adapt postural response amplitude to mildly varying intensities of the perturbing stimulus. Platform velocity is known to affect the amplitude of EMG responses in standing adults both during Bw and Fw body sway-inducing perturbations (Allum & Pfaltz, 1985; Dietz *et al.* 1987, 1989; Diener *et al.* 1988). In our study, a positive effect of a higher platform velocity on EMG burst amplitude was only present in the flexor muscles during Fw translations, but not in the extensor muscles during Bw translations. For this difference between Fw and Bw translations two explanations can be offered. First, the velocity ratio of fast *versus* slow trials in our study was smaller during Bw than during Fw trials. Second, the difference could be due to a difference in control mechanisms. It could be, as Dietz and coworkers suggested, that flexor control is more dynamic in nature than extensor control (Dietz *et al.* 1989; Dietz, 1992).

At 9–10 months of age initial sitting position started to have a modulatory effect on the EMG amplitude of the selected patterns. Previous studies showed that postural responses can be affected by the stability of the initial (standing) position, either by decreasing the support surface (Horak & Nashner, 1986) or by changing the initial position (Macpherson, 1994). In our study the starting angle of the pelvis showed a distinct relationship with the EMG amplitude of the flexor muscles during Fw translations. Remarkably, the modulatory effect was only present in the muscles adjacent to the pelvis (RF and RA), which suggests that the effect is mediated by segmental circuitry.

Refinement of the CPG model

Our data fit well into the two-level CPG model of postural control of Forssberg & Hirschfeld (1994). On the basis of the present findings we suggest that the circuitry for postural responses during sitting at the first level – the level in charge of the selection of the robust muscle activation pattern – consists of at least two separate networks, one involved in postural responses evoked by a Bw sway of the body, another elicited by Fw sway-producing perturbations. Bw body sway triggers an inhibition of the extensors and an excitation of the flexors, whereas Fw body sway elicits the reverse response.

Whether or not an inhibition can be observed depends on the level of background activity of the muscles. Both networks are probably triggered by afferents involved in the monitoring of the centre of mass, such as the somatosensory signals derived from the early occurring pelvis rotation (see Forssberg & Hirschfeld (1994) and Table 1). Possibly, proprioceptors in the lumbar vertebral column mediate a major part of the signalling effect (Gurfinkel *et al.* 1981). Once the basic pattern has been generated, visual, vestibular and somatosensory inputs are used to tune the final output to task specific constraints.

Various findings of the present study suggest that the flexor parts of both networks are controlled in a different way from the extensor parts. First, extensor muscles responded more consistently than flexor muscles. Second, flexor activity could be modulated by platform velocity and initial pelvis position, whereas extensor activity could not. Third, the latencies to the flexor responses were considerably longer than those to the extensor events, thus leaving considerable time for supraspinal effects on flexor bursts. Possibly, flexor muscles can be affected more by supraspinal mechanisms than the antigravity extensor muscles, which are probably more dependent on spinal mechanisms, such as stretch reflex mechanisms (Dietz *et al.* 1989; Dietz, 1992).

Developmental changes occur at both levels of the hypothesized model. Functional expression of activity of the first level emerges before the infant is able to sit independently. At the beginning, CPG activity results in a large repertoire of direction-specific responses from which the most appropriate, i.e. the most complete muscle activation patterns, are selected. Ontogenetic changes at this level might be attributed to a maturation of components within the CPG itself, an improved tuning to the afferent trigger and/or an increase of supraspinal influences. Modulating activity originating from the second level emerges at about 9 months of age, when stimulus intensity and initial pelvis position start to modify the amplitude of the responses.

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