Rhythmic coordination dynamics in children with and without a developmental coordination disorder
Volman, Michiel Joannes Maria

IMPORTANT NOTE: You are advised to consult the publisher's version (publisher's PDF) if you wish to cite from it. Please check the document version below.

Document Version
Publisher's PDF, also known as Version of record

Publication date:
1997

Link to publication in University of Groningen/UMCG research database

Citation for published version (APA):
Abstract
The study examined the stability of single and bimanual (i.e., inphase and antiphase) rhythmic finger movements in 24 children with a Developmental Coordination Disorder (DCD) and 24 matched controls. Stability was assessed by applying perturbations and measuring the time the system needed to return to its initial level of stability (i.e., the relaxation time). In addition, fluctuations in the patterns were measured. For antiphase coordination patterns, we also determined the frequency at which loss of stability occurred. It was found that children with DCD displayed less stable single and bimanual rhythmic coordination patterns than control children. Further, within the DCD group, a subgroup of 9 children was identified with a particularly poor bimanual coordination stability. According to a coupled oscillator model (Haken, Kelso, & Bunz, 1985), lower bimanual coordination stability is caused by a weaker coupling strength between the component oscillators. Individual differences suggest that the variability of individual limb oscillations might have contributed to poorer bimanual coordination stability. Findings were discussed in relation to a previous study on timing variability in DCD children in which the Wing-Kristofferson timekeeper model was applied (Williams, Woollacott, & Ivry, 1992).

5.1 Introduction

According to the ‘Diagnostic and Statistical Manual of Mental Disorders’ (DSM-IV, 1994), children with a Developmental Coordination Disorder (DCD) are characterized by poor performance of daily activities that require movement coordination that is not due to the child’s age or intellect, or to a known neurological disorder. The movement behavior of children with DCD is qualitatively different (‘clumsy’ and ‘uncoordinated’) from that of unaffected controls. Several experimental studies have been carried out to identify the underlying mechanism responsible for such clumsy behavior. One main finding is that children with DCD have a larger temporal variability (i.e., intertap-interval variability) in unimanual rhythmic tapping compared to matched controls (Geuze & Kalverboer, 1987; Lundy-Ekman et al., 1991; Williams, Woollacott, & Ivry, 1992). It has been suggested that the motor problems of children with DCD might be associated with a deficit in timing control in the sense that certain aspects of the motor programming (i.e., the sequencing and timing of motor responses) are not adequately processed (Geuze & Kalverboer, 1987; Williams, Woollacott, & Ivry, 1992). In an attempt to find the locus of such timing control difficulties, Williams et al. (1992) applied the Wing-Kristofferson (WK) timekeeper model (Wing & Kristofferson, 1973) for repetitive unimanual finger tapping, in which temporal variability is divided into a central timekeeper (or clock) component and a motor implementation component. They found that the greater temporal variability in children with DCD was mainly due to the timekeeper component, leading to the conclusion that timing problems in children with DCD are due more to central timekeeper processes (i.e., motor programming) than to motor execution processes.

The present study investigates the timing of rhythmic finger movements in children with DCD from a dynamic pattern perspective (Jeka & Kelso, 1989; Kelso, 1995). Further, since timing problems in children with DCD can also be observed in rhythmic activities which require coordination between limbs, it focuses not only on rhythmic single limb movements, but also on bimanual rhythmic coordinated movements. Research on relative timing at the level of interlimb coordination in DCD is largely lacking. Recently, some evidence for interlimb coordination problems was found: children with DCD showed a larger relative phase variability than control children during alternate tapping with both index fingers (Geuze & Kalverboer, 1993). In the present study, we investigated interlimb coordination problems in DCD more in detail.

From a dynamic pattern perspective, coordination is considered an a posteriori consequence of an evolving process of self-organization or pattern formation (Kelso & Schöner, 1988; Kelso, 1994; Beek, Peper, & Stegeman, 1995). Self-organization refers to the fact that stable patterns, and transitions from one stable pattern to another, may arise
from the cooperative coupling between components of the system without any explicit prescription (cf. Haken, 1978; Kelso, 1995). For example, phase transitions from antiphase to inphase interlimb coordination have been observed in humans when frequency is increased. Frequency is the control parameter that induces the phase transition, however, frequency does not contain itself any specific information about the relative phasing of the limbs. The dynamic perspective describes coordination patterns in terms of a relevant collective variable that captures the spatio-temporal order of the pattern and its dynamics (i.e., its evolution in time) (Kelso & Schöner, 1988).

For bimanual rhythmic coordination, the relative phase ($\phi$) between the two rhythmically moving limbs has been identified as a relevant collective variable (Kelso, 1981; 1984). A basic dynamic model for interlimb coordination is the HKB model, introduced by Haken, Kelso and Bunz (1985). Its relative phase dynamics read

$$\frac{d\phi}{dt} = -a \sin \phi - 2b \sin 2\phi + \sqrt{Q} \xi$$

(1)

where the $a$ and $b$ are constants, and $\sqrt{Q} \xi$ is a stochastic force of strength $Q$ that accounts for fluctuations of $\phi$ (Schöner, Haken, & Kelso, 1986). The ratio $b/a$ is inversely related to movement frequency. If the ratio $b/a > 0.25$, the system has two stable point attractors, namely, at $\phi = 0$ (i.e., inphase), and $\phi = \pi$ (i.e., antiphase), if the ratio $b/a < 0.25$, only the attractor at $\phi = 0$ remains (see Figure 3.1, chapter 3). The model thus predicts the presence of only two stable coordination modes (inphase and antiphase), and a loss of stability of antiphase coordination followed by a phase transition to the more stable inphase coordination mode at a critical frequency when movement frequency is increased. Signatures of the dynamics underlying coordinated movements can be found in the stability of coordination patterns. For bimanual coordination, large fluctuations of the relative phase, loss of stability at a low critical frequency, and long relaxation times (i.e., the time the system takes to restore the initial pattern stability after a perturbation) correspond to a less stable coordination pattern.

At another level of description, the rhythmic movements of a single limb can be modeled as a self-sustained nonlinear oscillator. Limb position $x$ and velocity $dx/dt$ are relevant collective variables (collective now with respect to the next lower level of description, for example, the coordination between agonist and antagonist muscles) that capture the reproducibility and stability of limb oscillations (Kelso et al, 1981; Kay et al., 1987). Such reproducible behavior can be represented by a phase plane (i.e., $dx/dt$ plotted against $x$), in which trajectories are attracted to a periodically stable orbit (i.e., a limit cycle). The stability of the limit cycle can be determined by perturbing trajectories away from the limit cycle and measuring the relaxation time, that is, the time it takes for the
system to return to the limit cycle (Kay, Saltzman, & Kelso, 1991). Long relaxation times are associated with a less stable limit cycle. By introducing nonlinear coupling functions between the component oscillators, the level of the individual limb oscillations (oscillator dynamics) has been linked to the level of interlimb coordination (relative phase dynamics) (Haken, Kelso, & Bunz, 1985; Kay et al., 1987).

The purpose of the present study was to examine differences in the stability of rhythmic movements between a group of children with DCD and a group of control children. Stability is tested at the level of interlimb coordination (i.e., relative phase stability), and at the level of individual limbs (i.e., limit cycle stability). We hypothesized that, compared to controls, children with DCD would show less stability of (i) interlimb coordination (i.e., a larger relative phase variability, longer relaxation times, and loss of stability at lower frequencies), and (ii) individual limb oscillations (i.e., larger limit cycle fluctuations, and a longer relaxation time). We also expected a smaller difference in temporal stability between preferred and non-preferred hands in children with DCD. In adults (Peters & Durding, 1979; Truman & Hammond, 1990), and in normal children (Peters & Durding, 1978; Fagard, 1987), the non-preferred hand has been found to be temporally more variable than the preferred hand. This might, however, be different for children with DCD. It has been suggested that coordination problems may be due to a poorly established hand preference (i.e., ambidexterity), which has a higher incidence in DCD than in normal children (Armitage & Larkin, 1993). To test these hypotheses, we conducted an experiment in which we perturbed the stability of single limb and bimanual rhythmic movements, and measured the time the system took to return to initial stability (i.e., the relaxation time). We also determined the stability boundary (critical frequency) for antiphase coordination by increasing the frequency to the point where loss of stability occurred. Finally, we measured the temporal and spatial variability of single limb oscillations, and the relative phase variability during unperturbed steady state performance.

5.2 Method

Subjects
We selected 24 children with DCD (age-range 7-12 years) and 24 matched controls from 10 primary schools in the town of Groningen and its surroundings. The selection procedure was as follows. Classroom teachers of grades 4 to 8, together with the physical education teacher, selected a total of 57 children with signs of clumsiness. Teachers were given a short list with relevant characteristics of clumsiness as a guide (see appendix). For each ‘clumsy’ child, the next child on the classlist of the same gender and age (range ± 3
months) was selected as a control child. The parents of the 45 children with signs of clumsiness gave their consent for their child to participate in the study. These children and their matched controls were tested on the ‘Movement Assessment Battery for Children’ (M-ABC; Henderson & Sugden, 1992), a standardized test for children from 4 to 12 years of age. The 24 children that scored below the 15th per centile were selected to participate in the experiment. Twenty-one of the 24 controls scored above the 15th per centile. The remaining 3 scored between the 5th and 15th per centile. Characteristics of the DCD and control group are summarized in Table 5.1.

<table>
<thead>
<tr>
<th>Table 5.1 Subject characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
<tr>
<td>DCD</td>
</tr>
<tr>
<td>Control</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>N</td>
</tr>
<tr>
<td>Age (years; months)</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Gender (boys / girls)</td>
</tr>
<tr>
<td>Handedness (R / L)</td>
</tr>
<tr>
<td>M-ABC per centile (%)</td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>

Apparatus
A custom-built apparatus was used to register flexion-extension movements of the index fingers. The apparatus consisted of two light metal braces that were attached to a left and right vertical rotation axis. The index fingers were fixed to these braces, while the hands were positioned by holding a grip. The metacarpophalangeal joint of the index finger was placed co-axially under the vertical rotation axis. Almost frictionless flexion-extension finger movements could be made in the horizontal plane. Potentiometers (angular precision .044°) mounted on top of the axes registered angular rotations of the fingers. Signals were A/D converted (DAT16, 12 bits) and recorded on a PC with a sampling rate of 100 Hz. On one of the rotation axes a brake device was mounted. Computer-controlled perturbations of rhythmic flexion-extension movements were applied by activating the brake (i.e., mechanical block; duration 120 ms) when the finger started to move in the opposite direction (criterion: velocity > 1.1 deg/sec). The effect of the perturbation was an almost immediate stop of the finger at brake onset and an acceleration after brake offset.
Procedure

Steady state frequency trials
Subjects were instructed to perform self-paced rhythmic flexion-extension movements of the index fingers. Tasks were demonstrated by the experimenter at a frequency of about 1.25 Hz, a frequency which is comfortable for children in the age range of 7 to 12 years. Children were asked to choose a similar tempo, that could easily be maintained for a longer time. The selection and maintenance of a steady pace, even when the finger movements were subjected to perturbations, were emphasized to the children. Movement amplitude was not constrained. Subjects were instructed to move their fingers in three different task modes: (i) bimanual inphase coordination; (ii) bimanual antiphase coordination; (iii) unimanual (single) with the index finger of the preferred/non-preferred hand. Hand preference was determined during the M-ABC test. Each mode was demonstrated by the experimenter. Subjects performed 1 practice trial without and 1 trial with a perturbation in each mode, after which 30 experimental trials (6 blocks of 5 trials) were run. After 3 blocks of trials subjects were given a 5 minute break. In each perturbation trial, two perturbations were applied (10th and 20th movement cycle), either at maximum finger flexion (F) or maximum finger extension (E). Four combinations of perturbation positions (EE, EF, FE, or FF) were applied in random order to avoid that subjects could anticipate the perturbation. Trial duration was limited to 30 seconds to avoid muscular and/or attentional fatigue effects. We employed a 2 Group (DCD, Control) x 3 Mode (inphase, antiphase, single) x 2 Hand (preferred, non-preferred) design. The 6 blocks of trials corresponded to a particular Mode (3) x Hand (2) combination. Each block started with 1 trial without perturbation, followed by 4 perturbation trials, yielding 8 perturbations per block. The order of presentation of mode and hand was balanced across subjects. DCD subjects and their individually matched controls were given the same order of presentation. In the steady state trials we measured the relaxation time of perturbed single limb and bimanual coordination patterns, as well as fluctuations of unperturbed patterns.

Scaled frequency trials
In the scaled frequency trials we determined the frequency at which loss of stability of antiphase coordination occurred. Subjects were asked to perform rhythmic finger movements in an antiphase coordination mode. They were instructed to synchronize their movements to computer beeps (duration 50 ms, pitch 600 Hz) so that one full cycle of oscillation was produced for each beep. The frequency of the beeps started at 1.0 Hz (8 cycles) and was linearly increased by 0.1 Hz per two beeps to 3.0 Hz. We did not use a step-wise increase of frequency as used by Kelso and others (cf. Kelso, 1984), because in a
pilot we found that synchronizing on a ‘step-wise’ increased pacing was more difficult for the children in this age-range than synchronizing on a ‘linearly’ increased pacing. Subjects were instructed to maintain the antiphase pattern, but not to resist if the pattern changed spontaneously. One practice trial and four experimental trials were performed.

Data reduction
The position signal was smoothed with a triangular (three samples) filter. Velocity time series were derived by differentiating the position time series and smoothed with the same triangular filter. Position and velocity time series were used to determine the position-velocity plots of left and right finger. A peak picking algorithm was applied to determine the maxima and minima in the position and velocity time series. The continuous relative phase $\phi$ between the fingers was estimated following

$$
\phi = \varphi_r - \varphi_l = \arctan \left( \frac{dX_r}{dt} / X_r \right) - \arctan \left( \frac{dX_L}{dt} / X_L \right) + C
$$

where $\varphi_r$ and $\varphi_l$ are the phase angles of left and right index finger for each sample, $X_L$ and $X_R$ are the angular positions of the left and right index finger rescaled to the interval [-1,1] for each cycle of oscillation, and $dX_L/dt$ and $dX_R/dt$ are its normalized instantaneous velocity (Scholz & Kelso, 1989). $C$ is a constant, which equals 0, -$\pi$ or $\pi$, depending on the sign of the quotients $(dX_r/dt)/X_r$ and $(dX_l/dt)/X_l$ (Fuchs et al., 1995).

We used a cursor control from interactive graphic displays of the position, velocity, and continuous $\phi$ time series to estimate the relative phase relaxation time ($\tau_\phi$) (cf. Scholz & Kelso, 1989). The 95% confidence interval of $\phi$ was calculated over a 3.5 second pre-perturbation time interval, and displayed as a $\phi$-stability threshold bandwidth. The cursor was scrolled (precision 10 ms) through the time series from the offset of the perturbation onwards. The relative phase relaxation time was defined as the time from the perturbation offset to the beginning of two consecutive movement cycles that $\phi$ stayed within the stability threshold bandwidth, with the exception of peaks in the $\phi$ time series that exceeded the threshold for only a short time (< 120 ms) (Figure 5.1). We were conservative in our estimate of $\tau_\phi$ in the sense that instabilities occurring more than two cycles after the pattern stability was regained were not taken into account, because we could not be certain that such delayed instabilities resulted from the previous perturbation (cf. Scholz & Kelso, 1989).

To estimate the limit cycle relaxation time ($\tau_{lc}$), we used an interactive graphic display of the index finger’s position-velocity plot (Figure 5.2). Ten movement cycles (5 pre- and 5 post-perturbation cycles) were displayed, while the cursor was scrolled through the limit cycle trajectories from the offset of the perturbation onwards. The limit cycle relaxation
time was defined as the time from the perturbation offset to the moment the perturbed trajectory reentered the limit cycle band, and stayed within the band for at least one cycle of oscillation. The limit cycle band was defined as the phase space between the inner and outer boundaries of the displayed trajectories.

Both relative phase and limit cycle relaxation times were pooled within each Mode x Hand block. Because the frequency distributions of the relaxation times were found to be

**Figure 5.1.** Position time series (panel A) and $\phi$ time series (panel B) of a DCD child in an antiphase perturbation trial. Dashed lines represent the relative phase stability threshold bandwidth. The left vertical line indicates the perturbation offset, while the right vertical line indicates the point where relative phase stability is regained.
FIGURE 5.2. Position-velocity plot of finger oscillations of the preferred hand of a DCD child (upper panel) and its matched control (lower panel) in a single hand trial with perturbation. The thick line represents the limit cycle trajectory between the perturbation offset and the point where the trajectory returns to the limit cycle. Note that the limit cycle trajectories of the DCD child are more variable than that of the control child.
positively skewed, we used the logarithmic transform of the relaxation times for further statistical analysis.

From the steady state trials without perturbation we determined the mean and variability of cycle frequency, amplitude and peak velocity for movement cycles 5 to 21. Coefficients of Variation (CV) were used as the variability measure to remove the effect of frequency differences between conditions and subjects. We also determined the variability at maximum flexion and extension. These movement reversal points represent ‘anchor points’ within the limit cycle of component oscillators (Byblow, Carson, & Goodman, 1994), and provide information about the band of variability in the limit cycle trajectories at four discrete points of the limit cycle (i.e., maximum flexion and extension, and flexion and extension peak velocity). We also calculated a performance asymmetry index (AI), i.e., the absolute value of the difference between the preferred (P) and non-preferred (N) hand normalized by the summed score of both hands (Armitage & Larkin, 1993) following

\[ AI = \left| \frac{(P - N)}{(P + N)} \right| \times 100 \]

For inphase and antiphase coordination trials, we determined the average relative phase (\( \phi \)), the absolute deviation of the relative phase from the intended phase at \( \phi = 0^\circ \) and \( \phi = 180^\circ \) (\( AE \phi \)), and the variability of the relative phase (SD\( \phi \)). A discrete estimate of the relative phase was calculated following

\[ \phi_i = \frac{t_i^N - t_i^P}{t_{i+1}^P - t_i^P} \times 360 \]

where \( t_i^N \) and \( t_i^P \) are the times of maximum finger extension of the preferred (P) and non-preferred (N) hand for the \( i \)th cycle in the position time series. The position time series of the preferred hand was used as reference.

In the scaled frequency trials we used a cursor control from interactive graphic displays of the position and \( \phi \) time series to determine the critical frequency. Transitions from antiphase to inphase coordination were detected in the position time series. Critical frequency was defined as the mean frequency of the four cycles of the preferred hand before a transition occurred. Antiphase trials in which no distinct transition was observed were checked for the presence of large instabilities in the \( \phi \) time series (i.e., \( \phi < 90^\circ \), or \( \phi > 270^\circ \)). If a large instability was present, then the critical frequency was defined as the average frequency of the four cycles of the preferred hand just before the instability occurred. The mean critical frequency across trials was used for statistical analysis.
Table 5.2. Average relative phase (ϕ) and relative phase variability (SDϕ) in steady state trials without perturbation (in degrees).

<table>
<thead>
<tr>
<th></th>
<th>DCD</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ϕ</td>
<td>SDϕ</td>
</tr>
<tr>
<td>Inphase</td>
<td>-3.0 (7.7)</td>
<td>19.5 (3.3)</td>
</tr>
<tr>
<td>Antiphase</td>
<td>178.4 (11.0)</td>
<td>23.7 (6.7)</td>
</tr>
</tbody>
</table>

Note. Within-group standard deviation between brackets.

5.3 Results

Steady state trials without perturbation

Frequency. In unimanual trials, the mean self-paced movement frequency was 1.28 Hz (range 0.67 - 2.10 Hz) for the DCD group and 1.24 Hz (range 0.72 - 1.94 Hz) for the control group. In bimanual trials, the mean self-paced frequency was 1.15 Hz (range 0.74 - 1.45 Hz) for the DCD group and 1.23 Hz (range 0.72 - 1.94 Hz) for the control group. These differences were not significant (see statistics section limit cycles).

Relative phase. In Table 5.2, discrete estimates of ϕ and SDϕ are presented for DCD and control group. Both groups were quite accurate in producing the intended relative phase at ϕ = 0, and ϕ = 180°. In the control group, a small phase lag for the non-preferred hand was found, while in the DCD group a small phase lag for the preferred hand was found, indicating that the asymmetry at the level of the relative phase was small. A 2 Group x 2 Mode (inphase, antiphase) ANOVA revealed that the absolute deviation of ϕ from the intended phase did not differ significantly between the groups. SDϕ was significantly larger for the DCD group, F(1,92) = 17.79, p < .001. Furthermore, SDϕ was larger for antiphase coordination than for inphase coordination, F(1,92) = 13.84, p < .001. No interaction effects were found.

Limit cycles. In Table 5.3, the averages and Coefficients of Variation (CV) of cycle frequency, amplitude, and flexion and extension peak velocity of the preferred and non-preferred hand are summarized for DCD and control group. A 2 Group x 3 Mode (single, inphase, antiphase) x 2 Hand ANOVA was applied. A significant difference between the two groups was found on CV of frequency, F(1,276) = 43.34, p < .001 (Figure 5.3),
**Table 5.3.** Means and Coefficients of Variation (CV) of Frequency, Amplitude and Peak Velocity in steady state trials without perturbation for preferred and non-preferred hand. Manual asymmetry is indicated by the Asymmetry Index (%).

<table>
<thead>
<tr>
<th></th>
<th>DCD</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Freq. (Hz)</td>
<td>Ampl. (°)</td>
</tr>
<tr>
<td></td>
<td>Mean</td>
<td>CV</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P</td>
<td>1.31</td>
<td>8.7</td>
</tr>
<tr>
<td>N</td>
<td>1.24</td>
<td>9.9</td>
</tr>
<tr>
<td>AI</td>
<td>7.7</td>
<td>9.6</td>
</tr>
<tr>
<td>Inphase</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P</td>
<td>1.19</td>
<td>9.2</td>
</tr>
<tr>
<td>N</td>
<td>1.19</td>
<td>9.8</td>
</tr>
<tr>
<td>AI</td>
<td>0.2</td>
<td>7.3</td>
</tr>
<tr>
<td>Antiphase</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P</td>
<td>1.11</td>
<td>10.5</td>
</tr>
<tr>
<td>N</td>
<td>1.11</td>
<td>11.0</td>
</tr>
<tr>
<td>AI</td>
<td>0.4</td>
<td>8.0</td>
</tr>
</tbody>
</table>

Note. P = preferred, N = non-preferred hand, AI = Asymmetry Index. Scores represent averages across trials.

CV of amplitude, F(1,276) = 16.73, p < .001, CV of flexion and extension peak velocity, F(1,276) = 11.89, p < .001, and F(1,276) = 9.72, p < .01, and on SD of maximum flexion and extension F(1,276) = 13.56, p < .001, and F(1,276) = 12.88, p < .001 (Figure 5.4), indicating that the finger oscillations of DCD children were temporally and spatially more variable than those of controls. Cycle frequency did not significantly differ between the two groups. Significance for mode was found on frequency, F(2,276) = 3.68, p < .05, and CV of frequency, F(2,276) = 3.09, p < .05 (Figure 5.3). Tukey HSD comparisons revealed that movement frequency was slower in the antiphase than in the single mode. Tukey HSD comparisons did not reach significance for CV of frequency. A significant effect of hand was found on CV of frequency, F(1,276) = 3.85, p < .05, CV of amplitude, F(1,276) = 9.66, p < .01, CV of extension peak velocity, F(1,276) = 10.22, p < .01, and on SD of maximum flexion F(1,276) = 9.87, p < .01, revealing that
non-preferred hand oscillations were temporally and spatially more variable than preferred hand oscillations. No significant interactions were found.

Table 5.3 also shows the asymmetry index of the average and CV of frequency, amplitude and peak velocity. A 2 Group x 3 Mode ANOVA was applied. DCD and control group did not differ significantly in manual performance asymmetry. A significant effect of mode was found on the asymmetry index of frequency, amplitude, flexion and extension peak velocity, CV of flexion and extension peak velocity, SD of maximum flexion (all, \( p < .001 \)), and CV of amplitude (\( p < .05 \)). Tukey HSD comparisons revealed that the asymmetry was reduced in the inphase and antiphase mode compared to the single mode, indicating that coupling synchronized the oscillating index fingers in space and time. A significant interaction between group and mode on the CV of amplitude, \( F(2,136) = 3.63, p < .05 \), showed that, in DCD children, the reduction in asymmetry was larger in the inphase mode than in the antiphase mode. No further interaction effects were found.

Steady state perturbation trials
In 9.2% of the 1536 perturbations instabilities of \( \phi \) were observed just before the perturbation onset, or the perturbation did not clearly disturb the ongoing coordination pattern, e.g., when the onset of perturbation was applied before the beginning of the finger
movement, because the brake was activated due to noise or finger tremor in the period of movement reversal. These perturbation trials were not included in the analysis. For one 8-year-old DCD child (S4), more than 50% of the perturbations in the antiphase coordination mode failed, mainly because instabilities occurred before perturbation onset. This child was excluded from the antiphase relaxation time analysis.

Relative phase stability. A 2 Group x 2 Mode (inphase, antiphase) x 2 Hand ANOVA was applied on $\tau_\phi$. A significant effect for group, $F(1,182) = 29.69, p < .001$, revealed that inphase and antiphase coordination patterns of DCD children were less stable (i.e., longer relaxation times) than those of controls (Figure 5.5). Further, antiphase patterns were less stable than inphase patterns, $F(1,182) = 124.77, p < .001$. A lack of significance for hand showed that perturbing the preferred or non-preferred hand had no differential effect on $\tau_\phi$. The interaction between group and mode was close to significance, $F(1,182) = 3.48, p = .064$, indicating that coordination patterns of DCD children were particularly less stable in the antiphase mode.

Limit cycle stability. A 2 Group x 3 Mode x 2 Hand ANOVA was applied on $\tau_{lc}$. A significant effect for group, $F(1,264) = 12.29, p < .001$, revealed that finger oscillations of DCD children were less stable than those of controls. Significance for hand, $F(1,264) = 12.74, p < .001$, showed that finger oscillations of the non-preferred hand were less stable than those of the preferred hand. A significant effect for mode, $F(2,264) = 3.43, p < .05$,
indicated that limit cycles were most stable in the inphase, and least stable in the antiphase mode (Figure 5.6). Tukey HSD comparisons, however, did not reach significance. No significant interactions were found. A 2 Group x 3 Mode ANOVA was applied on the asymmetry index of $\tau_{lc}$ of the preferred and non-preferred hand. No significant effects were found.

Scaled frequency trials
In 76% of the trials a transition from antiphase to inphase coordination was observed. On most of the trials (32%) the inphase pattern remained stable after the transition, but in a number of trials a loss of 1:1 entrainment (i.e., phase drift) between the oscillating fingers was observed (22%), or children decreased their cycle frequency and switched back to the antiphase pattern (22%). In 16% of the trials, no transition to inphase coordination occurred, but an instability in the antiphase pattern was detected according to our criterion (see method section). In 8% of the trials the critical frequency could not be determined because no transitions or instabilities were observed. A one-way ANOVA was applied on critical frequency. A significant effect for group, $F(1,46) = 8.02, p < .01$, revealed that the antiphase patterns of DCD children became unstable at a lower frequency ($f_{\text{critical}} = 1.94 \text{ Hz}; \text{Sd} = .33$) than those of controls ($f_{\text{critical}} = 2.24 \text{ Hz}; \text{Sd} = .32$).

Relation between stability and kinematic variability measures
In Table 5.4 Pearson’s correlations (between subjects; $N = 48$) between the relative phase stability, the limit cycle stability (single mode), and kinematic measures (single mode) are presented. Most of the different relative phase stability measures ($\tau_{\phi}$, $f_{\text{critical}}$, and $\text{SD}_{\phi}$) were significantly correlated with each other. Long relaxation times corresponded with a low critical frequency, and a large relative phase variability. This finding is in accord with predictions from the HKB model. Significant correlations between $\tau_{\phi}$ and kinematic variability measures (i.e., CV’s of frequency, amplitude, and peak velocity, and SD’s of maximum extension and flexion) indicate that children with less stable inphase and antiphase coordination patterns also displayed a larger temporal and spatial variability in the limit cycles of their finger oscillations. The limit cycle relaxation time $\tau_{lc}$ did not significantly correlate with any of the relative phase stability or kinematic variability measures. However, $\tau_{lc}$ did correlate significantly with cycle frequency, indicating that children with a higher cycle frequency had a smaller limit cycle relaxation time. It should be noted, however, that the limit cycle stability differences found between the DCD and control group are not due to a difference in frequency, since the two groups did not significantly differ in cycle frequency.
Table 5.4 Pearson’s correlations (between subjects; N = 48) between (i) relative phase stability measures (relaxation time [τφ], critical frequency [fcrit], relative phase variability [SDφ]), and limit cycle relaxation time [τlc]), and (ii) between relative phase and limit cycle stability measures, and kinematic measures (average and CV of frequency [f], amplitude [A], peak velocity [pv], and maximum extension and flexion variability [SDext, SDfl]).

<table>
<thead>
<tr>
<th></th>
<th>τφ</th>
<th>τφA</th>
<th>fcrit</th>
<th>SDφ</th>
<th>SDφA</th>
<th>τlc</th>
<th>f</th>
<th>A</th>
<th>v</th>
<th>CVf</th>
<th>CVA</th>
<th>CVpv</th>
<th>SDext</th>
<th>SDfl</th>
</tr>
</thead>
<tbody>
<tr>
<td>τφ</td>
<td></td>
<td>.75*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>τφA</td>
<td>-.66*</td>
<td>-.74*</td>
<td></td>
<td>.36</td>
<td>.45*</td>
<td>-.44</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>fcrit</td>
<td>.43</td>
<td>.46*</td>
<td>-.48*</td>
<td>.41</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SDφ</td>
<td>.24</td>
<td>.27</td>
<td>-.32</td>
<td>.25</td>
<td>.23</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SDφA</td>
<td>.05</td>
<td>.01</td>
<td>.25</td>
<td>.10</td>
<td>-.04</td>
<td>-.50*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>f</td>
<td>.09</td>
<td>.10</td>
<td>-.01</td>
<td>-.16</td>
<td>-.13</td>
<td>.11</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>.06</td>
<td>.03</td>
<td>-.24</td>
<td>.18</td>
<td>.20</td>
<td>.12</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>v</td>
<td>.50*</td>
<td>.52*</td>
<td>-.37</td>
<td>.27</td>
<td>.18</td>
<td>.12</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CVf</td>
<td>.47*</td>
<td>.49*</td>
<td>-.43</td>
<td>.37</td>
<td>.35</td>
<td>.07</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CVA</td>
<td>.48*</td>
<td>.54*</td>
<td>-.54*</td>
<td>.43</td>
<td>.36</td>
<td>.35</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CVpv</td>
<td>.50*</td>
<td>.47*</td>
<td>-.40</td>
<td>.29</td>
<td>.25</td>
<td>.04</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SDext</td>
<td>.45*</td>
<td>.39</td>
<td>-.38</td>
<td>.17</td>
<td>.19</td>
<td>.04</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. Correlations are tested for $p = .001$; *$p < .001$. Index: I = inphase; A = antiphase; S = single hand.

Individual differences

Individual differences within the DCD group on different relative phase stability measures suggest that there is a subgroup of DCD children with particularly poor relative phase stability (see Figure 5.7). To identify such a subgroup we applied a statistical procedure in which the individual scores of DCD children were compared with the mean score of the control group, which was regressed for age. We did not directly compare matched subjects, because such a procedure suffers from the between-subject variability which enhances the detection of false positives and false negatives (e.g., if a DCD child is matched with a particularly skilled control child). We calculated the standard normalized residuals of the stability scores for individual DCD children with reference to the age-regression line of the control group. We then determined which DCD children were deviant using an age-dependent cut-off point of 1.65 standard deviation. DCD children with a residual above this cut-off point were considered to have particularly poor stability. We added the criterion
that children had to be deviant on two or more of the different relative phase stability measures. Nine DCD children were found to have a particularly poor relative phase stability, whereas 4 children showed a particularly poor limit cycle stability (Table 5.5; Figure 5.7). One child (S 3) was particularly less stable on both stability measures. Thus, three subgroups could be distinguished within the DCD group: (A) a subgroup (N = 9) with particularly poor relative phase stability; (B) a subgroup (N = 4) with particularly poor limit cycle stability; and (C) a subgroup (N = 12) without particularly poor relative phase or limit cycle stability. We applied one-way ANOVA's with Tukey HSD comparisons with Subgroup (A, B, C) as a factor to test whether the subgroups differed with regard to the Movement-ABC and kinematic variability scores. Subgroup A scored significantly lower on the Movement-ABC (4th per centile) in comparison to subgroup B (9th per centile) and C (8th per centile). In addition, subgroup A had significantly larger CV's of frequency and amplitude, and a larger SD of maximum extension than subgroup C. These findings provide further evidence that subgroup A is a functionally relevant subgroup. Subgroup B did not differ significantly from subgroup C with regard to the Movement-ABC, or kinematic variability scores. Note that the DCD children in subgroup B also deviated on one of the SD measures in the bimanual tasks. Because \( J_{lc} \) was found to be highly correlated with frequency, we applied a one-way ANOVA to test whether the subgroups differed in cycle frequency. A significant effect was found, \( F(2, 70) = 3.59, \ p < .05 \), revealing a lower cycle frequency for subgroup B (f = 1.03 Hz) compared to subgroup A (f = 1.22 Hz), and C (f = 1.22 Hz). Although Tukey HSD comparisons did not reach significance, it indicates that the \( J_{lc} \) in subgroup B might be overestimated due to a lower frequency. The status of subgroup B, therefore, remains unclear.

**Table 5.5** DCD children with particularly poor pattern stability.

<table>
<thead>
<tr>
<th>Antiphase</th>
<th>( J_{\phi} )</th>
<th>3</th>
<th>4</th>
<th>11</th>
<th>12</th>
<th>13</th>
<th>18</th>
<th>20</th>
<th>22</th>
</tr>
</thead>
<tbody>
<tr>
<td>f critical</td>
<td></td>
<td>3</td>
<td>4</td>
<td>12</td>
<td>13</td>
<td>20</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SD( \phi )</td>
<td></td>
<td>2</td>
<td>4</td>
<td>6</td>
<td>7</td>
<td>11</td>
<td>13</td>
<td>19</td>
<td>20</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Inphase</th>
<th>( J_{\phi} )</th>
<th>3</th>
<th>4</th>
<th>11</th>
<th>12</th>
<th>13</th>
<th>22</th>
<th>23</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>SD( \phi )</td>
<td></td>
<td>3</td>
<td>4</td>
<td>8</td>
<td>11</td>
<td>12</td>
<td>15</td>
<td>18</td>
<td>22</td>
</tr>
</tbody>
</table>

| Single      | \( J_{lc} \)   | 3 | 7 | 15 | 19 |    |    |    |    |

*Note.* Bold numbers: subjects that deviate at two or more relative phase stability measures.
5.4 Discussion

The present study examined the stability of single and bimanual rhythmic finger movements in children with a Developmental Coordination Disorder and controls from a dynamic pattern perspective. Such a perspective assumes that stable and reproducible rhythmic single limb patterns result from nonlinear self-sustained oscillatory processes, and that stable interlimb coordination patterns may arise in a self-organized fashion from cooperative coupling of the component oscillators (Kelso et al., 1981; Haken, Kelso, & Bunz, 1985; Kelso & Schöner, 1988; Kay et al., 1987; Kelso, 1995). We examined the intrinsic dynamics of single and bimanual rhythmic movement patterns in a quantitative way by testing the stability of these patterns. We hypothesized that movement patterns of children with DCD would be less stable than those of controls. Our main hypotheses were confirmed. Children with DCD displayed less stable inphase and antiphase coordination patterns, and less stable single limb movement patterns than controls.
Limit cycle stability

In accord with other studies (Geuze & Kalverboer, 1987; Lundy-Ekman et al., 1991; Williams, Woollacott, & Ivry, 1992), rhythmic movements of children with DCD were temporally more variable than those of controls. In addition, their finger movements were also spatially more variable, as indicated by larger fluctuations of the limit cycle trajectories at maximum extension and flexion, and flexion and extension peak velocities. Further, the limit cycle relaxation time was longer for the DCD group. This latter finding is not due to the reciprocal relation found between frequency and limit cycle relaxation time, since the DCD and control group did not differ in cycle frequency. Altogether, these findings show that the intrinsic spatio-temporal patterns of children with DCD are less stable and more variable than those of controls, which indicates that the attractor strength of the oscillator dynamics underlying the finger oscillations of children with DCD is less strong than that of controls (Kay et al., 1987; Kay et al, 1991). It should be noted here, that the observed fluctuations in the limit cycle trajectories of finger oscillations are probably deterministic in nature (i.e., stemming from specific neuromotor processes such as neuromotor tremor), and that we do not know whether these processes are different for DCD children and controls (cf. Van Galen et al., 1993). These questions go beyond the scope of the present study, but they may be addressed in future research by applying spectral analysis (Kay, Saltzman, & Kelso, 1991; Van Galen et al., 1993) or dimensionality analysis (Kay, Saltzman, & Kelso, 1991).

Since limit cycle relaxation time and limit cycle variability are both reflections of the stability of the underlying limit cycle dynamics, we expected to find high correlations between them. However, the correlations were rather low (Table 5.4, last column). This might be caused by the fact that the limit cycle relaxation time was significantly influenced by movement frequency, and the limit cycle variability was measured by the Coefficient of Variation, which is insensitive for differences in frequency. The significant correlations between limit cycle variability and relative phase stability measures indicate that children with large fluctuations in the limit cycle trajectories also had less stable bimanual coordination patterns. We will discuss this further in the next section.

With regard to manual asymmetry, single limb oscillations of the non-preferred hand were less stable (i.e., longer relaxation time), and both spatially and temporally more variable than those of the preferred hand, which is in line with other studies (e.g., Peters & Durding, 1978; Byblow, Carson, & Goodman, 1994). In contrast to studies with adults (e.g., Kelso et al., 1981; Helmuth & Ivry, 1996), coupling of the hands (inphase and antiphase mode) did not result in a reduction of temporal or spatial variability of finger oscillations as such (Table 5.3). However, the decrease in the asymmetry index of
frequency, amplitude, and peak velocity suggests that the limit cycles of the preferred and non-preferred hand become entrained temporally and spatially in the coupled condition. Since the differences in manual asymmetry between the DCD and control group were rather small, it seems that asymmetries at the level of the individual oscillators do not play a major role in explaining differences in stability between DCD and controls.

Relative phase stability

Inphase and antiphase coordination patterns appeared to be intrinsically stable for children with DCD and controls (i.e., the patterns could be more or less stably performed without extensive learning), but the patterns of children with DCD were clearly less stable than those of controls. This was expressed by a longer relative phase relaxation time after perturbation, by a lower critical frequency, and by a larger relative phase variability in steady state trials without perturbation. The implications are that rhythmic coordinated actions of children with DCD will be more easily disturbed by small environmental perturbations, and that coordination stability will be more easily lost when performing tasks at a high frequency. Moreover, less stable intrinsic coordination patterns might constrain these children in learning and performing more complex interlimb coordination patterns. Theoretical interpretation of the differences in stability between the groups leads to the following considerations. According to the HKB model, the reduced stability of DCD children’s interlimb coordination can be explained by a weaker coupling strength between the component oscillators given the assumption that the stochastic force strength $Q$ (see equation 1) is equal for both groups (Haken, Kelso, & Bunz, 1985). However, factors other than coupling strength per se may have contributed to the reduction in relative phase stability. It has been suggested that interlimb coordination is better described by phase entrainment than by phase-locking, and that phase entrainment is determined by characteristics of the individual oscillators. Such as, asymmetry in the oscillator dynamics of coupled limbs and asymmetry in the coupling. (Schmidt, Shaw, & Turvey, 1993). Such symmetry-breaking characteristics may lead to an increase in relative phase fluctuations. Schmidt et al. (1993) therefore suggested modifying the HKB model, in which the coupled oscillators are identical, to include dynamically different oscillators. Are there indications that such oscillator characteristics may have contributed to the differences in relative phase stability between DCD and controls? There was no evidence that the two groups differed in coupling asymmetry, since no differences in relative phase asymmetry (i.e., a phase shift, indicating that one of the hands systematically leads the other) were found (Table 5.2). The kinematics of finger oscillations of the preferred and the non-preferred hands differed, but no difference in manual asymmetry between the groups was found (Table 5.3). However,
since we did not investigate the dynamic characteristics of the individual oscillators underlying the limb oscillations in DCD children and controls, the possibility that differences in the asymmetry of the individual oscillators contributed to differences in relative phase stability remains. This should be be examined further. Another possibility which seems relevant in the present context is that enhanced fluctuations in the limit cycle trajectories of finger oscillations might have contributed to the less stable relative phase patterns in children with DCD. Our data provide some support for this idea. DCD children showed both a larger variability in individual finger oscillations and less stability of bimanual coordination patterns than controls. Furthermore, the subgroup of nine DCD children with particularly poor relative phase stability showed a significantly larger variability in finger oscillations in comparison with the remaining DCD children. These findings are in line with the significant correlations between limit cycle variability and relative phase stability measures. In terms of the HKB model, such enhanced variability at the level of the individual oscillators can be associated with an increase in the stochastic force strength \( Q \) at the level of the relative phase (see equation 1). Thus, the reduced relative phase stability in children with DCD seems to be due both to a weaker coupling between the individual oscillators and to a larger stochastic force strength \( Q \).

Antiphase patterns were less stable than inphase patterns, which is in agreement with the HKB model and with other empirical findings on interlimb coordination (e.g., Kelso, 1984; Kay et al., 1987; Kelso & Jeka, 1992). Interestingly, in the perturbation task, children with DCD tended to have a particularly long relaxation time in the antiphase coordination mode. It is unlikely that this is due to a critical slowing down effect, i.e., an increase in the relaxation time of antiphase coordination at frequencies close to the critical frequency (Scholz, Kelso, & Schöner, 1987). Critical slowing down has been observed at frequencies of 0.2 Hz or 0.4 Hz below the critical frequency (Scholz & Kelso, 1989). The difference between the ‘steady state’ frequency in the antiphase perturbation trials and the critical frequency were probably too large (DCD children: mean difference 0.8 Hz; Sd 0.3 Hz) to result in critical slowing down. Closer inspection of the position time series of the antiphase perturbation trials revealed that, in several trials, within the first cycle after the perturbation, a short transient inphase tendency was observed, lasting no longer than half a cycle of oscillation. In controls this was observed only occasionally, whereas in seven DCD children (Ss 3, 11, 12, 13, 18, 20, 22) this was observed in more than 25% of the perturbations. These short inphase tendencies might have contributed to a prolonged relaxation time. A similar interaction for mode of coordination has been reported in patients with neurological disorders. For instance, the interlimb coordination patterns of Parkinsonian (Swinnen, Verschueren, & Dounskaia, 1996) and hemiplegic patients (i.e., coordination between unaffected limbs) (Baldissera, Cavallari, & Tesio, 1994) have been
found to be particularly less stable (i.e., SD\(\phi\)) in the antiphase coordination mode than those of unaffected subjects. Swinnen et al. (1996) suggested that the antiphase patterns of such patients are constrained by a strong intrinsic inphase tendency that is caused by the pathological condition of the central nervous system (CNS). Such an explanation seems not to apply to the findings of the present study. Although children with DCD often display so-called neurological ‘soft’ signs (e.g., Rasmussen et al., 1983; Lundy-Ekman et al., 1991) which are an indication of a minor dysfunction of the CNS (Touwen, 1987), they do not have a known neurological disorder (DSM-IV, 1994). Furthermore, DCD children’s inphase patterns were not more, but less stable than that of controls. Hence, we would like to conclude that the longer relaxation times after perturbation are mainly due to the reduced stability of the antiphase pattern itself.

When we compare the different measures of relative phase stability, the differences between DCD children and controls are more pronounced in the antiphase perturbation and scaled frequency task than in the steady state task without perturbation. Apparently, tasks in which the system is perturbed or driven towards loss of stability are more sensitive. They are therefore more useful in the diagnosis and in the investigation of interlimb coordination problems in DCD than tasks in which the system operates in a ‘comfortable’ steady state mode. In addition, antiphase coordination tasks seem to be more sensitive for this purpose than inphase coordination tasks.

Timing control in DCD: a central timekeeper or dynamic control deficit?

What can we conclude with regard to the mechanism underlying the poor timing abilities of children with DCD? Applying the Wing-Kristofferson model, Williams et al. (1992) concluded that problems in the timing of rhythmic single limb movements of children with DCD are caused by a deficit in central timekeeping processes (i.e., motor programming), rather than by a deficit in motor execution processes. This conclusion should be viewed with some caution first of all, because in 22% of the trials a basic assumption of the WK model was violated. Further, the present study provides evidence that problems children with DCD have in the (relative) timing of rhythmic movements may be explained by a deficit in the self-organizing capacity of the motor system that accounts for the production of stable and reproducible spatio-temporal movement patterns, rather than by a deficit in motor programming. The differences in stability found between children with DCD and controls are difficult to explain using hierarchical timekeeper models (e.g., Wing & Kristofferson, 1973; Wing, 1982), because such models do not account for inherent stability properties such as resistance to perturbations, or loss of stability and phase transitions when frequency is increased. We conclude that the problems in timing control of
rhythmic movements in DCD are due to a lack of intrinsic pattern stability, and can therefore be characterized as a dynamic control deficit.

Can we interpret such a dynamic control deficit in terms of ‘timekeeper’ or ‘motor’ processes? This distinction was introduced in the WK model but has not yet been fully understood and worked out within the dynamic approach. For interlimb coordination, a model consisting of three levels each with its own dynamics has been proposed (Kugler & Turvey, 1987; Turvey et al., 1986; Kelso, 1995). The three levels are the lower level of the component oscillators, the intermediate interlimb coordination level, and the upper intentional level. It was suggested that the ‘timekeeping’ processes may be linked to the subject’s intention to maintain a specified pattern, such as an inphase ($\phi = 0$) or antiphase ($\phi = \pi$) coordination pattern, whereas the ‘motor’ processes relate to the dynamics of the component oscillator. These dynamics reflect the underlying muscular activation pattern (i.e., the energy dissipation) of the agonist and antagonist muscles of the limb (Turvey, Schmidt, & Rosenblum, 1989). The coordination level represents the average $\phi$, and interacts with both the upper intentional and lower component oscillator level. In such a view, the reduced relative phase stability might be caused by a deficit in ‘central timekeeping’ or ‘motor’ processes, or in the interaction between them, while a reduced limit cycle stability might be caused by a deficit in peripheral ‘motor’ processes rather than a deficit in ‘timekeeping’ processes.

Can we say more about the neurophysiological underpinnings of the observed reduction in limit cycle stability and relative phase stability in children with DCD? Generally speaking, a dynamic pattern perspective does not seek causal explanations of movement coordination in terms of structural properties of the system. Because biological systems are complex systems (i.e., composed of a large number of interacting elements), it tries to identify elementary principles of coordination using the language of dynamics. These dynamics are abstract and independent of the system’s material substrate (Kelso, 1995). The dynamic pattern approach is, therefore, not a structural, but a phenomenological approach (Beek, Peper, & Stegeman, 1995). With regard to movement disorders, this implies that such disorders can be described and understood in terms of the dynamics of a relevant collective variable that underlies the observed -deviant- spatio-temporal movement patterns, without knowing the locus of dysfunction at the neural level (cf. Wagenaar & Van Emmerik, 1996). However, since the dynamic pattern approach may be applied at different levels of analysis, phenomena observed at the behavioral level may be linked to similar phenomena observed at the neural level (Kelso, 1995). In this respect, the introduction of a coupled oscillatory neural network that generates phase transitions from antiphase to inphase, resulting from a change in the coupling coefficients between excitatory and inhibitory neurons in the network is relevant (Nagashino & Kelso, 1992). Such change in
the coupling coefficients is probably induced by a tonic external input that is capable of
modulating the synaptic characteristics of the neurons (Kelso, 1995). Thus, the functional
coupling between the component oscillators in the HKB model may be associated with the
synaptic coupling strength among neurons in a coupled oscillatory neural network.
Physiologically, rhythmic limb movements may be produced by lower level circuits that
include the cerebellum, the brainstem and propriospinal networks (also known as ‘spinal
generators’), which receive tonic input from higher brain centers. Intraspinal processes are
the main source of the rhythmic activity of the spinal generator. However, the activity may
be modulated by the spino cerebellum, which receives input from the spinal cord about the
rhythmic activity of the spinal generator and about the current state of the peripheral motor
system (Archavsky, 1983). Phase entrainment of rhythmically moving limbs is also likely
to depend on such lower level circuits (Wiesendanger, 1994). It was found, for example,
that inphase coordination patterns are relatively well preserved both in the presence of
cortical lesions outside the primary motor cortex, and in split-brain patients (i.e., with a
dissection of the corpus callosum), who showed an even greater attraction to inphase and
antiphase coordination patterns (Tuller & Kelso, 1989).

It is generally agreed upon that the cerebellum plays an essential role in the temporal
organization of coordinated movements (Brooks & Thach, 1981; Ivry & Keele, 1989). A
specific hypothesis about the role of the cerebellum in the problems of children with DCD
in the timing of rhythmic movements was put forward by Williams and others (Williams,
Woollacott, & Ivry, 1992). They suggested that the large timekeeper variance in DCD
children might be specifically related to a dysfunction of the lateral cerebellum. Their
conclusion was based on a clinical study in cerebellar patients in which the Wing-
Kristofferson model was applied. This study found that a dysfunction of the lateral
cerebellum related more to central timekeeping processes, and a dysfunction of the
intermediate cerebellum more to motor execution processes (Ivry, Keele, & Diener, 1988). Do our findings give support to this cerebellar hypothesis? There is considerable
neurophysiological evidence that two functionally different parts of the cerebellum
contribute differently to the control of coordinated movements. The lateral cerebellum
(i.e., the cerebrocerebellum) participates in the initiation, planning and programming of
movements, whereas the intermediate part (i.e., the spino cerebellum) plays a major role in
controlling the ongoing execution of limb movements, specifically in the on-line
modification of the kinematic and dynamic characteristics of the movements (see for a
review Bloedel, 1992; Kandel, Schwartz, & Jessell, 1991). The spino cerebellum is, for
example, involved in regaining initial stability after perturbations of the limb (Schwartz,
1987). Thus, there is converging neurophysiological and clinical evidence indicating that
the spino cerebellum plays an essential role in the stability and reproducibility of rhythmic
limb movements (Ivry, Keele, & Diener, 1988; Kandel, Schwartz, & Jessell, 1991; Bloedel, 1992). Our findings therefore support the idea that the cerebellum might be the critical site with regard to the problems in the timing of rhythmic movements in children with DCD (Williams, Woollacott, & Ivry, 1992; Lundy-Ekman et al., 1993). In contrast to the study of Williams et al. (1992), who pointed specifically to a dysfunction of the cerebrocerebellum, the findings of our study suggest that problems in the timing of rhythmic movements are due rather to a dysfunction of the spinocerebellum. This is in line with a study on congenital hypothyroidism (CHT), an endocrine disorder in children causing problems in movement coordination. This study suggested that problems in the timing of rhythmic movements in CHT children is due to a spinocerebellar rather than to a cerebrocerebellar dysfunction (Kooistra et al., 1997). The spinocerebellar hypothesis in DCD might be empirically falsified by conducting similar perturbation experiments with cerebellar patients and comparing the findings with those of the present study.

Dynamic control deficit: a subtype in DCD?

The heterogeneous nature of motor difficulties in DCD makes it difficult to identify mechanisms that might be responsible for the coordination disorders of children with DCD (Henderson, 1987). Larkin and Hoare (1992) therefore suggested that researchers attempt to identify subtypes of DCD based on inter-individual differences in movement dysfunction. With respect to the stability of rhythmic movements, we were able to identify a subtype of DCD children with a particularly poor relative phase stability. The fact that these DCD children also showed enhanced limit cycle fluctuations and lower scores on the Movement ABC test provides further evidence that this ‘interlimb coordination’ subtype is indeed functionally relevant. No conclusive evidence was found for a second DCD subtype based on our results on limit cycle stability measure. A more qualitative approach might be appropriate for identifying subtypes of DCD, if any, at the level of individual limb movements by examining the type of oscillator that fits the behavior of limb movements in individual DCD children. This may be done by comparing the topology of limit cycle trajectories of limb oscillations with trajectories produced by a known dynamical system, or by modeling observed amplitude-frequency relations and peak velocity-frequency relations (i.e., the functional form of the oscillator) (cf. Kay et al., 1987; Beek, Rikkert & Van Wieringen, 1996).
Acknowledgements
This research was supported by the Netherlands Organization for Scientific Research (NWO), grant number 575-59-051, and -partly- grant number 400-10-020. We would like to thank Peter Beek, Pascal van Lieshout, Lieke Peper, Piet van Wieringen, and two anonymous reviewers for helpful comments on an earlier draft of the manuscript.