RESEARCH ARTICLE

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The ability to execute saccades on the basis of efference copy: impairments in double-step saccade performance in children with developmental co-ordination disorder

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Abstract The double-step saccade task (DSST) was used to test the hypothesis that children with developmental co-ordination disorder (DCD) who experience deficits in motor imagery have difficulty processing the visual spatial consequences of intended movements using efference copy signals. In order to ensure that the second saccade in the DSST was executed in the absence of visual cues and had to be programmed on the basis of extra-retinal information (efference copy), we analysed only those double-step ensembles where latency plus duration of first saccades was greater than 240 ms (total presentation time of the targets). No significant differences between DCD and control children were evident on measures of latency of first saccades, intersaccadic interval and first saccade error. As predicted, children with DCD who have impaired motor imagery demonstrated specific deficits on the DSST where efference copy had been used to program the saccade sequence. More specifically, these children were less accurate in terms of final eye position on second saccades. Our results raise the possibility that abnormalities in the processing of efference copy signals could underlie motor clumsiness in the majority of children with DCD. Furthermore, the origin of this deficit in efference copy probably exists at the level of the parietal lobe.

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J. Currie Brain Research Unit, Drug and Alcohol Services, Westmead Hospital, Westmead, Sydney, Australia Keywords Double-step saccades \cdot Efference copy \cdot Developmental co-ordination disorder (DCD) \cdot Motor imagery \cdot Parietal lobe

Introduction

Motor clumsiness in children that exists in the absence of medical conditions or low IQ is referred to as developmental co-ordination disorder (DCD; American Psychiatric Association 1994). Currently there is debate about the relative contribution of motor and cognitive impairments to the clinical presentation of DCD. In a recent meta-analysis of studies of cognitive function in DCD, we identified that the most frequent and reliable deficits in cognition, that were independent of motor function, occurred on tests that required some aspect of visuospatial processing (Wilson and McKenzie 1998). Subsequent investigations of visuospatial processes in children with DCD identified specific impairments in the endogenous control of covert attention (Wilson et al. 1997; Wilson and Maruff 1999) as well as impairments in motor imagery (Maruff et al. 1999a). On the basis of these findings, we proposed that the slow and variable motor performance of children with DCD occurred because of impairment in the processes associated with the forward modelling of efference copy (see, for example, MacKay 1966; Wolpert et al. 1995). Normally, a forward model of the efference copy allows the central nervous system to maintain the stability of motor systems despite delays in the availability of re-afferent signals. This mechanism predicts and corrects the consequences of voluntary movements before re-afferent feedback becomes available (Crammond 1997). Therefore, impairment in this operation would result in the evaluation of unfolding volitional motor acts being more reliant on slow re-afferent motor signals. This would add time and error to each step of any volitional motor sequences, a result consistent with clinical descriptions of motor clumsiness in DCD (Wilson and McKenzie 1998).

The double-step saccade task (DSST) can be used to investigate directly the forward modelling of move-

ment-related efference copy in children with DCD. On the DSST, subjects are instructed to make saccades to the locations indicated by two brief target appearances, as quickly and as accurately as possible and in the same order as that indicated by the targets. The total duration of targets is very brief so both targets are usually extinguished before the first saccade begins. The critical feature of the DSST is that while the first saccade can be programmed within a retino-topic reference frame, the second saccade does not start at the spatial location from which the second visual target was observed. Thus, the DSST introduces a retino-spatial dissonance between the retinal co-ordinates of the second target and the motor co-ordinates of the second saccade (see, for example, Heide et al. 1995). When this retino-spatial dissonance occurs, it is generally agreed that second saccades are programmed on the basis of a forward model of the efference copy of the first saccade (Duhamel et al. 1992a; Heide et al. 1995; Colby and Goldberg 1999). Single unit studies of the lateral intraparietal sulcus of alert primates performing the DSST show that the accurate programming of second saccades requires a dynamic reorganisation of visual and motor neurons so that their receptive fields predict the sensory consequences of the first saccade, before it is executed (Goldberg and Colby 1992; Duhamel et al. 1992a; see Anderson et al. 1997; Colby and Goldberg 1999 for a review). Studies of human adults performing the DSST indicate that it is possible to disrupt specifically this forward modelling process. For example, focal lesions of the posterior parietal cortex (PPC) interfere specifically with the accuracy of second saccades but do not effect the metrics of the first saccade. Interestingly, focal lesions of the frontal eye field, dorsolateral prefrontal cortex or supplementary motor area do not disrupt the metrics of second saccades on the DSST (Heide and Kompf 1998). Taken together, these data suggest that the accuracy of second saccades on the DSST provides a behavioural index of forward modelling processes when the metrics of first saccades are normal. On some DSST trials individuals can acquire the first target while it is still visible. If this occurs second saccades may be visually guided removing the condition of retino-spatial dissonance and the requirement for any forward modelling of the efference copy. Thus for DSST trials, retino-spatial dissonance occurs only when the latency and duration of the first saccade is greater than the total presentation time of both targets. The aim of the current study was to examine the forward modelling of efference copy in children with DCD who showed impairments in motor imagery, using the DSST. We hypothesised that these children would show a decrease in accuracy for second saccades in double-step saccade ensembles planned on the basis of efference copy.

Materials and methods

Subjects

The DCD group originally consisted of 14 children between the ages of 7 and 11 years (mean age=9.5 years, SD=1.1 years) who met the DSM-IV criteria for the disorder scoring at or below the 15th percentile for their age group on the movement assessment battery for children (MABC; Henderson et al. 1992). Four children from this group were excluded on the basis that they showed normal motor imagery whereas the remaining 10 showed impaired motor imagery (see methods and results for details). The control group consisted of 10 children between the ages of 8 and 11 years (mean age=10.0 years, SD=1.3 years) who scored above the 15th percentile on the MABC and showed no impairment in motor imagery (see methods and results for details). Both DCD and control children were recruited from local schools in Melbourne and were matched according to verbal IQ (estimated using the short form of the Wechsler intelligence scales for children, WISC III; Wechsler 1992). Exclusion criteria for both the DCD and control groups included a history of psychiatric disorder, gross neurological impairment or a WISC III-estimated verbal IQ of 80 or below. Prior to participating in this experiment, written consent was received from children and parents.

Assessment of motor imagery

The visually guided pointing task (VGPT) was used to measure movement durations for real and imagined movements. The task and procedure used was modified from that of Sirigu and colleagues (1996). On each trial, subjects were presented with a piece of clear plastic upon which was drawn an 80 mm vertical line. A black target box was also drawn on the plastic with its closest edge 30 mm from the vertical line. A set of ten different box stimuli was used each with a different target width. The target widths used were 1.9, 3.0, 4.4, 5.3, 7.5, 10.6, 14.9, 18.9, 23.4 and 28.0 mm. One hand movement was defined as the subjects' hand moved from the far side of the vertical line, to touch the inside of the target box, and return to the far side of the vertical line using the tip of a felt tip pen (see Fig. 1). For each trial of a single target width, subjects made five of these back and forth movements. A stopwatch was used to record the total duration of subjects' hand movements. For real movement trials, timing began when the experimenter said "go" and stopped when the subject completed the fifth hand movement. For imagined movement trials, the subject was required to imagine, without actually performing, a motor movement. The timing for these trials began when the experimenter said "go" and stopped when the subject said "stop" upon completing the fifth imagined hand movement. All subjects performed two sets of the ten trials under the real and imagined conditions using their dominant hand. The order of condition (real or imagined) and the order of target widths within each condition was randomised across subjects.

Double-step saccade task and recording

Eye movements were recorded using high-resolution infrared scleral reflectance oculography (IRIS; Skalar), with the head stabilised on a chin rest. The room was completely dark and subjects were seated 1 m from the visual display that consisted of five light emitting diodes (LEDs). A green LED (16.93 cd/m^2) in the centre of the display was used as a central fixation point. Red LEDs (8.3 cd/m^2) positioned at 5° and 10° to the left and right of the central fixation point were used as targets for saccades. A computer software algorithm controlled illumination of the LEDs with an accuracy of 1 ms and the rise time of the LEDs was 3 ms. Eye and target position signals were recorded at 1 kHz and stored for off-line analysis. Eye position was differentiated using a computer-based algorithm to obtain eye velocity. The recording and scoring



Fig. 1 Schematic representation of the real condition of the visually guided pointing task (VGPT) showing amplitude and target width

of eye movements have been described in detail elsewhere (Currie et al. 1991; Shafiq et al. 1998).

The timing of targets for the DSST was based on the optimum target durations identified by Heide et al. (1995). Subjects were required to maintain fixation on the central green LED. Following a delay randomised between 900 and 1,300 ms, the fixation point disappeared and the first target appeared in one of the four possible peripheral target locations for 140 ms. Immediately following, a second target appeared at a different location in either the left or right visual field for 100 ms (see Fig. 2). Subjects were instructed to generate two sequential saccades in response to the targets in the same order that they had appeared. The importance of acquiring each target position sequentially and accurately was emphasised. Each subject performed 80 double-step saccade ensembles. Measurements were made of first saccade latencies and the duration between the end of the first saccade and onset of the second saccade referred to as the intersaccadic interval (ISI). The measure of first and second saccade accuracy was represented as the degree of error between target position and final eye position.

Data analysis

Motor imagery

For each subject, the duration of real and imagined movements at each target width was determined. As amplitude (A) was constant, target width (W) was expressed as item difficulty (ID) using the following equation:

$ID = Log_2[2 \times A/W]$

For each subject, the strength of the relationship between movement duration and ID was determined by calculating the correlation (Pearson's r) between movement duration and ID from the ten data points in both the real and imagined movement conditions. A normal range for the correlation between movement duration and ID for real and imagined movements using the dominant hand was established from a previous study of motor imagery in a large group of adults (group mean correlation plus/minus two times the group SD; Maruff et al. 1999b). For real movements, the lower limit of this normal range of correlations was *r*=0.38 (for example, group mean dominant hand, r=0.48, SD=0.05). For imagined movements, the lower limit of this normal range of correlations was r=0.26 (for example, group mean dominant hand, r=0.37, SD=0.07). Performance on the VGPT by control children was within the adult range and therefore it was considered appropriate to use the norms developed for adults. Subjects were classified as having impaired motor imagery if Pearson's correlation between ID and target width was less than 0.20 on imagined trials of the VGPT. Conversely, subjects were considered to have no impairment in motor imagery if Pearson's correlation between ID and target width was greater than 0.30 on the VGPT.



Fig. 2 Schematic representation of the sequence of events in a single trial of the double-step saccade task showing target position, eye position, first saccade latency and intersaccadic interval (*ISI*)

Double-step saccade task

Trials were rejected from the analysis if any of the following were observed: no saccade in response to one or both targets, saccade disrupted by a blink, a head movement or other artefact, initial eye movement in the direction opposite to that of the target, an initial saccade made to the second target location or first saccade initiated before presentation of the first target or with a latency between 0 and 80 ms (anticipatory saccades; Smit and Van Gisbergen 1989). In addition, we rejected saccade ensembles in which both targets were not obtained using a saccade that fell within 15% accuracy and, thus, trials in which end position was reached using multiple saccades were rejected. According to these criteria, 35.25% of all double-step saccade ensembles executed by the control group and 42.25% of double-step saccades made by the DCD group were excluded. From the remaining valid DSST trials, first saccade latency plus duration were calculated to determine the double-step saccade ensembles that met the criterion for retinospatial dissonance. Of the valid DSST trials, the number of saccades that met the criterion for retino-spatial dissonance was not different between the groups (control=55%, DCD=57%, $\chi^2 > 1$). Mean first saccade latency, ISI and degree of error on final eye position for both first and second saccades were then calculated for each subject and submitted to between-groups ANOVAs.

Results

Motor imagery

Performance on the real movement trials of the VGPT was within the normal adult limits for all subjects. Of the 14 subjects who met the clinical criteria for DCD, 4 were excluded because their performance on the imagery trials of the VGPT was also within adult normal limits. In the remaining 10 children in the DCD group, motor imagery was abnormal. Motor imagery was within normal adult limits for all of the control children (Fig. 3).

Double-step saccade task

To examine whether performance or accuracy on the second saccade differed between the groups, a 2(group: con**Fig. 3** Correlation (Pearson's *r*) between item difficulty (*ID*) and movement duration for both control and developmental co-ordination disorder (*DCD*) children on the real and imagined trials of the VGPT



 Table 1
 Means (SD) of first saccade latency, intersaccadic interval, and first and second saccade error. (*DCD* Developmental co-ordination disorder)

	Control (n=10)	DCD (<i>n</i> =10)
First saccade latency	360.317 (80.299)	397.797 (159.390)
Intersaccadic interval	467.221 (144.289)	479.453 (149.806)
First saccade error	3.898 (0.708)	4.363 (1.234)
Second saccade error	4.126 (0.732)	5.887* (1.861)

*Result is statistically significant, P<0.05

trol, DCD)×2(error of final eye position: first saccade, second saccade) ANOVA was conducted. A main effect for saccade order was found [F(1,9)=36.744, P<0.000)with the second saccade being less accurate than the first (see Table 1). A significant interaction between saccade order and group was also found [F(1,9)=7.545, P=0.023]and represented in Fig. 4. Further post hoc analysis revealed that the DCD group was significantly less accurate on the second saccade in double-step ensembles. That is, the amount of error from final eye position after the second saccade in relation to the second target was significantly greater in the DCD group. Finally, when examining the time to execute saccades (i.e. first saccade latency and ISI) no significant effects were found for group [F(1,9)=0.295, P=0.600], saccade [F(1,9)=4.582,P=0.061] or an interaction [F(1,9)=0.072, P=0.795].



Fig. 4 Saccade accuracy represented by degree of error (mean \pm standard error) for first and second saccades by normal control (*NC*) and DCD children

Discussion

Children with DCD who have a deficit in motor imagery demonstrate an impairment in their ability to program saccades on the basis of efference copy. When doublestep saccades were executed under conditions of retinospatial dissonance, the latency of first saccades and ISI were equal in the control and DCD groups. In addition, there was no difference between groups on error of final eye position after first saccades. Importantly, under conditions of retino-spatial dissonance, targets for first sac-

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cades have been extinguished before these saccades begin. The normal accuracy and latency for first saccades in the DCD group therefore indicates that these children do not have a general impairment in the ability to generate saccades to remembered targets. Children in the control group were able to generate accurate second saccades even though these saccades did not begin at the same location as that from which the second target had been observed. In the DCD group, the final eye position after second saccades was considerably different from position of the second target and the magnitude of this error was much greater than that observed in the control group. This specific impairment in the accuracy of second saccades support the hypothesis that a significant proportion of children with DCD have a deficit in assembling movements on the basis of forward models of efference copy (see, for example, Maruff et al. 1999a).

First saccade accuracy on the DSST of the current DCD group indicates that these children do not have an absolute deficit in the representation of space for intended saccades. Instead, when the preparation of a saccade must compensate for a previous eye movement that has changed the retino-topic representation of the target, these children have difficulties in executing the saccade accurately. A qualitatively similar but quantitatively more severe abnormality has been observed in the DSST performance of patients with unilateral lesions of the PPC (Duhamel et al. 1992b; Heide et al. 1995). When first saccades were directed to the ipsilesional field, the accuracy of second saccades, directed further into the ipsilesional field or back into the contralesional field, was normal. However, when first saccades were directed into the contralesional field, second saccades directed further into the contralesional field or back into the ipsilesional field were either grossly dysmetric or were not generated at all. This abnormality was observed in patients with lesions of both the right and left PPC although its magnitude was greater in the right PPC group. Importantly, the accuracy of both first and second saccades of the DSST was normal in patients with focal lesions of the right and left prefrontal cortex, right frontal eye field or left supplementary motor area. Thus, inaccuracy of second saccades occurred only with PPC lesions and did not reflect a retino-topic or directional deficit in the preparation of second saccades. These data indicate that patients with focal lesions of the PPC had difficulty using extra-retinal information about the amplitude and direction of the motor vector of the first saccade, in order to update the spatial representation of the second target. Thus, like the children with DCD, they were unable to use the efference copy of the first saccade to construct a spatial representation of the second target location in order to generate an accurate saccade towards it.

The data from studies of adult humans is consistent with single unit studies in the lateral intraparietal sulcus of alert non-human primates performing the DSST. These indicate that the use of efference copy from the first saccade to anticipate the retinal consequence of the new fixation begins before the first saccade is executed. Goldberg and colleagues (1990) demonstrated that this dynamic remapping is calculated on the basis of the efference copy for intended movements rather than the neuronal signals generated after an actual movement has been made. For example, when an error trial occurred in which the sequence of the two saccades were reversed, the neuronal activation measured reflected the sequence that was planned initially (i.e. double-step sequences in the correct order) rather than the erroneous sequence that actually occurred (Goldberg et al. 1990). Goldberg and colleagues (1990) have speculated that this remapping updates the representation of space in conjunction with eye movements so that it remains constant and matches the current eye position. The use of internal forward dynamic models to predict the consequences of action has emerged as an important theoretical concept in motor control (MacKay 1966; Wolpert et al. 1995; see Wolpert 1997 for review). In addition to anticipating and cancelling out the sensory consequences of a given movement, these forward models also maintain the stability of motor systems in the presence of sensorimotor feedback delays including slow parasympathetic nerve transmission times and psychological refractory periods. The predictive nature of forward modelling provides a mechanism that can modify and update actions before sensory feedback is available. They can be used to calculate potential error in the outcome of a movement and use this to alter the motor command, thus facilitating motor learning. Furthermore, by anticipating the sensory outcome of a movement before it is performed, forward models can be used in motor imagery training to select optimal action sequences. Finally, forward models can be integrated with motor commands and sensory inputs to estimate and maintain the current state of the motor system (Wolpert 1997). Given these points, an abnormality in the generation or integration of internal forward dynamic models may provide a parsimonious explanation for the variety of cognitive and motor deficits found in children with DCD. Certainly, it provides a useful model within which the deficits in motor imagery and visual attentional shifts that we have observed in these children can be accommodated. Furthermore, these findings do suggest strongly that there is some abnormality in parietal lobe function in children with DCD.

Previously we reported that in DCD imagined movements were not constrained by conventional speed-accuracy trade-offs (for example, Fitts' Law; Maruff et al. 1999a). This impairment was also found in the majority (71%) of children with DCD recruited for the current study. Jeannerod (1997) proposed that motor imagery is the efference copy of real movements that can reach awareness because the real movements are inhibited. In tasks such as the VGPT, children must use this efference copy to predict the time necessary to make accurate movements to target locations of different sizes. However, the imagined movements of most children with DCD are not constrained by the same biomechanical and environmental factors as their real movements. This pattern of performance is consistent with the hypothesis that these children are unable to imagine movements by forward modelling an efference copy of a motor command whilst actually suppressing the movement. Interestingly, the dissociation between intact real motor sequences and abnormalities in imagined motor sequences is found only in adult patients with lesions of the right parietal lobe, but not in patients with lesions of the motor cortex (Sirigu et al. 1995), basal ganglia (Dominey et al. 1995) or cerebellum (Kagerer et al. 1998). In these last three groups, impairments in imagery are equivalent to impairments in actual motor movements. Importantly, we found a small group of children who met the clinical criteria for DCD whose ability to imagine movements was normal. Unfortunately, this group yielded too few correct double-step saccades so we were unable to determine whether DSST performance was normal in this group as our hypothesis would predict. We are currently pursuing more children like this. However, the association between abnormalities of motor imagery and impaired DSST performance in the majority of our DCD group suggests strongly that parietal lobe dysfunction underlies impairments in the forward modelling of efference copy in a significant proportion of children with DCD.

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