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Visuoperceptual and visuomotor deficits in developmental stutterers: An exploratory study

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Abstract

Although the cause of stuttering is unknown, there is strong evidence for it being a neuromotor disorder characterised by an abnormality of higher control encompassing not only speech but other motor systems. The aim of this exploratory study was to look for the presence of non-speech/language deficits – in particular, visuomotor and visuoperceptual deficits – in persons who stutter.

Twelve moderate to severe developmental stutterers were compared with a group of fluent speakers, matched for age and sex, on a range of computerized sensory-motor tasks. These tasks covered various aspects of visuomotor function – ballistic movement, dynamic steadiness, and several types of tracking – and visuoperceptual function – acuity, static perception, and dynamic perception. A novel technique was used to remove the visuospatial component from tracking performance. Stutterers had slower reaction times, less accurate random tracking, and impaired dynamic visual perception. Severity of stuttering correlated with reaction time and dynamic perception. Removal of the visuoperceptual component from tracking performance indicated that the impaired tracking in the stutterers was predominantly due to reduced dynamic perception.

This is the first study to provide preliminary evidence for the presence of non-linguistic visuoperceptual and upper-limb visuomotor tracking deficits in people with moderate to severe stuttering. These findings support a neurogenic aetiology for stuttering and are compatible with evidence of an overactive dopamine system in stutterers.

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1. Introduction

Developmental stuttering (onset during childhood) affects about one percent of the population. Despite considerable research into this disorder, its cause remains unknown (Andrews et al., 1983; Bloodstein, 1987; Fox et al., 1996; Gordon, 2002), although it is clearly multi-factorial (Foundas, Bollich, Corey, Hurley, & Heilman, 2001; Fox et al., 1996; Gordon, 2002). The neurogenic or neuromotor theory has gained particular prominence over recent years, proposing that developmental stuttering is a neuromotor disorder characterised by an abnormality of higher control encompassing not only speech but other motor systems (for review see Caruso, 1991). Other theories of developmental stuttering have suggested that sensory or sensory-motor integrative functions may also be implicated in stuttering. For instance, the cybernetic theory of stuttering contends that dysfluency is a sensory feedback disorder in which delayed auditory feedback is responsible for errors in speech (Fairbanks, 1954; Lee, 1951). While the cybernetic theory has fallen from favour (Andrews et al., 1983), the concept of stuttering as a breakdown in sensory-motor integrative processing has evolved from these origins. Neilson and Neilson (1991) have used their adaptive model theory (Neilson, Neilson, & O'Dwyer, 1992) as the basis for their proposal that stuttering results from an inadequate central processing capacity in transforming sensory input into motor output (speech).

Studies of the cerebral cortex and regional cerebral blood flow have drawn attention to certain anatomical areas in the brain (namely the supplementary motor area (SMA), anterior cingulate cortex and basal ganglia) as being important in the control of both speech and arm/hand movements (Goldberg, 1985; Kimura, 1982; Paus, Petrides, Evans, & Meyer, 1993; Webster, 1988). In a SPECT study, reduced regional cerebral blood flow in the anterior cingulate and superior middle temporal gyri was found in stutterers at rest compared with controls (Pool, Devous, Freeman, Watson, & Finitzo, 1991). Using PET, Wu et al. (1995) noted reduced regional glucose metabolism in Broca's area, Wernicke's area, frontal pole, cingulate and cerebellum during speech in developmental stutterers compared to non-stutterers. Importantly, they also found a reduction in left caudate metabolism during both stuttering and fluent speech in the stutterers when compared with controls. Similarly, Braun et al. (1997) used PET imaging to demonstrate differences in regional cerebral blood flow between persons who stutter and controls when all subjects were fluent. These findings are in contrast to a study by Ingham et al. (1996) in which no differences in resting-state cerebral blood flow were found between developmental stutterers and controls.

Anatomical abnormalities have also been demonstrated in stutterers. Using MRI scans and volumetric analysis, Foundas et al. (2001) found strong evidence of

anomylous anatomy in perisylvian speech and language areas, including significantly more gyral variants.

High temporal resolution of MEG has provided a means of unveiling functional abnormalities in the auditory cortices (Salmelin et al., 1998) and a neural network incorporating the left inferior cortex and the right motor/premotor cortex (Salmelin, Schnitzler, Schmitz, & Freund, 2000) of developmental stutterers. Further support for the presence of abnormalities in the speech-motor region of the right (non-dominant) hemisphere of stutterers, even during fluent speech, has been shown in a PET study by Fox et al. (2000). They also showed that the left (non-dominant) cerebellar hemisphere is implicated in stuttering.

Further strong evidence for a neurogenic basis of stuttering has come from a second PET study by Wu et al. (1997). They showed substantial increases in dopaminergic activity in moderate-to-severe stutterers at rest. The increases were in the right ventral medial prefrontal cortex, left caudate tail, limbic structures, and auditory cortex. This finding is consistent with earlier findings of decreased metabolic activity in many of the same regions in stutterers (Braun et al., 1997; Pool et al., 1991; Wu et al., 1995) due to excess dopamine having an inhibitory effect on regional cerebral glucose metabolism (London et al., 1990; Wolkin et al., 1987). Wu et al. (1997) also noted that a dominant increase in dopaminergic activity in ventral limbic cortical regions was consistent with their hypothesis of these regions being part of a neural circuit implicated in stuttering (Wu et al., 1995).

Damage to these areas of the brain have also been found to result in impairments to both speech and hand/arm movements. Kimura (1982) found that aphasic patients with left hemisphere parietal/frontal lesions were inferior to non-aphasic patients in both oral and manual-brachial movements. The clinical consequences of lesions of the SMA and anterior cingulate gyrus in the left hemisphere have been reported to include difficulty initiating speech and limb movements and sometimes complete loss of speech (Damasio, 1992; Damasio & van Hoesen, 1980; Laplane, Talairach, Meinenger, Bancaud, & Orgogozo, 1977). Luria (1966) reported a 'deautomisation' of speech and upper-limb movements after damage to the SMA, with speech characterised by a lack of spontaneity and disturbances in the smooth integrated sequences of limb movements. Non-haemorrhagic infarction in the left caudate nucleus head and internal capsule may result in mixed aphasia characterised by reduced language comprehension, oral apraxia or mutism, along with right hemiparesis (Aram, Rose, Rekate, & Whitaker, 1983; Damasio, Damasio, Rizzo, Varney, & Gersh, 1982).

Studies of acquired stuttering secondary to brain damage in adults have also highlighted the effects of basal ganglia lesions on speech and manual movements. Ludlow, Rosenberg, Salazar, Grafman, and Smutok (1987) found acquired stutterers to have significant deficits in oral/speech movements as well as in skilled rapid hand movements. Eighty percent of those studied had lesions in the caudate and lentiform nucleus and fifty percent in the cerebellum. Helm, Butler, and Benson (1978) also reported acquired stutterers as having neurobehavioural deficits associated with hand movements, including impaired ability to draw/copy three-dimensional figures or block designs, reproduce sequential motor tasks and tap rhythmic patterns.

While several studies have reported that developmental stutterers have slower finger and hand reaction times (Cross & Luper, 1983; Hand & Haynes, 1983; Rastatter & Dell, 1985; Starkweather, Franklin, & Smigo, 1984; Webster & Ryan, 1991), others have found no difference between stutterers and non-stutterers (Postma & Kolk, 1991; Prosek, Montgomery, Walden, & Schwartz, 1979; Reich, Till, & Goldsmith, 1981; Till, Reich, Dickey, & Seiber, 1983). There is, however, general agreement that, for complex finger movements requiring timing and sequencing, stutterers have slower movements and make more errors than non-stutterers (Borden, 1983; Caruso, 1991; Webster, 1986). Stutterers are slower and less accurate than non-stutterers in using the hand to track an auditory stimulus, although no differences between the same groups were found in using the hand to track a random visual target (Neilson, 1980). Zebrowski, Moon, and Robin (1997) provided preliminary evidence for impairment of lower-lip, but not jaw, visuomotor tracking of sinusoidal targets in a group of four boys who stuttered.

Visuospatial perception in stutterers has been little studied. Hamilton (1940) reported that stutterers have normal visual acuity and binocular perception. Kelly (1932), however, found that stutterers were deficient on a digit visuoperception task. Hemispheric processing of visual information in stutterers has also been investigated via tachistoscopic procedures using linguistic stimuli. These studies indicated that stutterers have a left visual field preference and slower responses when stimuli are presented to the right visual field/left hemisphere (Hand & Haynes, 1983; Moore, 1976). Similarly, Forster and Webster (2001) used divided visual field tasks – lexicon decision and dot enumeration – to show different asymmetries between the cerebral hemispheres in stutterers than those in non-stutterers; however, both tasks possess, at most, only a simple visuoperceptual component. Thus, except for binocular perception, there have been no studies of visuospatial perception in stutterers using non-linguistic stimuli.

The aims of this study were to (i) confirm and extend previous investigations of upper-limb sensory-motor function in developmental stutterers by measuring their performance on a range of visuomotor tasks comprising ballistic movement, dynamic steadiness, and several visuomotor tracking tasks (Jones, Donaldson, & Parkin, 1989; Jones, Sharman, Watson, & Muir, 1993), (ii) examine visuoperceptual function in stutterers using tests of acuity and of static and dynamic perception (Jones & Donaldson, 1995), and (iii), in the event of deficits being found in both areas, attempt to determine the extent to which visuoperceptual deficits might be responsible for the visuomotor deficits.

2. Method

2.1. Participants

The participants consisted of 12 right-handed developmental stutterers ranked as moderate to severe on the Riley stuttering severity instrument (SSI) (Riley, 1972) and a control group matched for age (±2 years), sex, and handedness. Nine of the stut-

terers had received speech and language therapy for their dysfluency at some stage. There were ten male and two female stutterers with ages ranging from 21 to 55 years (mean 34 years). All participants had visual acuity of 6/9 or better in at least one eye as measured on the Snellen chart. No participants had neurological, musculoskeletal or joint problems affecting limb movement. No participants were taking psychoactive medication at the time of testing.

2.2. Apparatus and tests

The apparatus included a PC and two colour monitors: one displaying test stimuli for the subject and the other used by the assessor for task generation and analysis. All tests were run and analysed by the program SMTests® which is menu-driven and includes features such as a range of sinusoidal and random targets, standard test instructions, storage/retrieval of raw data, performance analyses and graphical displays of results (Jones et al., 1993). Each test lasted 1–2 min. Participants were seated in front of their monitor (312 × 234 mm) with an eye-to-screen distance of 132 cm. All of the visuomotor tests were one-dimensional and had a steering wheel (395 mm diameter) as subjects' output sensor. Rotation of the wheel moved an arrow horizontally on the screen.

2.3. Visuoperceptual tests

- *Visual acuity:* Best eye on the Snellen chart at 6 m.
- *Visual resolution:* Ability to identify the position of a dot with respect to a vertical line on screen (Jones & Donaldson, 1995; Jones, Donaldson, & Timmings, 1992) (Fig. 1). Dot-line separations were in multiples of 0.27 mm. Visual resolution was defined as the minimum separation at which a participant was always able to correctly identify the dot as being off the centre of the line.
- *Static perception:* Perception of position of an arrow point with respect to a static vertical line in four trials and a static sinusoidal-wave in 16 trials (Jones & Donaldson, 1995). The test score of number of incorrect responses was converted to static perception resolution using a technique described by Jones and Donaldson (1995).

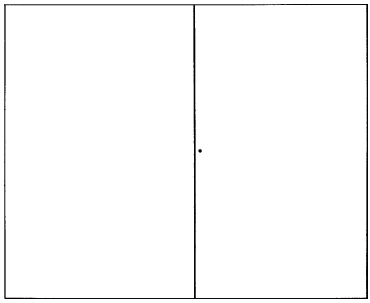

Fig. 1. Visual display for the visual resolution test.

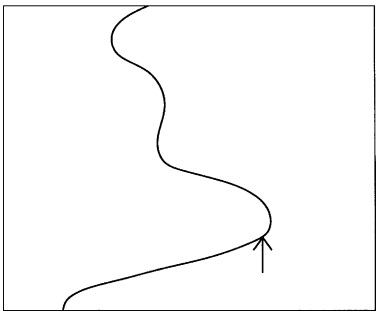

Fig. 2. Visual display for the dynamic perception test and the random tracking (preview) task.

- *Dynamic perception:* Determination of whether an arrow point stayed perfectly on a random input descending the screen with an 8.0 s preview time (Fig. 2). The duration of 20 trials decreased from 10 to 2 s and various error offsets were simulated. A dynamic perception resolution was defined as the minimum spacing between the point of the arrow and the target over the 20 trials at which a subject was always able to perceive the arrow as being off the target at some stage during its descent (Jones & Donaldson, 1995).

2.4. Visuomotor tests

- *Ballistic movement:* Fastest possible arm movement in response to a non-target stimulus (no accuracy required). This required moving the arrow out of a box and across a pass-line equivalent to 90° of movement on the steering wheel in response to a random 3–7 s latency stimulus. The best reaction time and speed of movement over eight attempts were recorded.
- *Steady movement:* Steadiness of attempted constant-speed non-pursuit movement on the steering wheel over a range of 116°. The best of eight attempts within speed range of 17.7–34.7°/s was recorded.
- *Sine tracking (non-preview):* Ability to keep arrow point on a sinusoidal target (0.15 Hz) moving horizontally on the screen. Mean absolute error and mean delay between response and target were recorded for all four tracking tasks.
- *Random tracking (non-preview):* Ability to keep arrow point on a random target (0.21 Hz) moving horizontally on the screen. The task required smooth movements over a 175° range of steering wheel.
- *Random tracking (preview):* Ability to keep arrow point on target signal as in non-preview random task but with an 8.0 s preview as it descended from top of screen before reaching arrow point (Jones, 2000; Jones & Donaldson, 1986; Jones et al., 1989) (Fig. 2).
- *Step tracking (non-preview):* Ability to keep arrow point on a vertical line as it moved abruptly over 32 steps alternating between displacement from and return to centre screen (Jones, 2000; Jones & Donaldson, 1986). Spatial unpredictability was incorporated via four randomly distributed amplitude/direction movements:

large steps (90° on wheel); small steps (22°), both to right and left of centre. Four randomly distributed durations between steps (2.8, 3.4, 4.0, 4.6 s) and lack of preview ensured temporal unpredictability. Mean absolute error and lag were recorded.

2.5. Procedure

Severity of stuttering was assessed using the Riley SSI to give measures of dysfluency during reading and monologue. Percentage dysfluency during conversation was also calculated for a measure of more 'natural' context speech. All dysfluency evaluation sessions were videotaped. Videos were reviewed and rated by two observers training in speech-language therapy. After the three visuoperceptual tests, participants completed the six visuomotor tests using their dominant arm.

2.6. Analysis

Comparisons of performance between the two groups were analysed using the non-parametric Wilcoxon matched-pairs test (two-tailed values) due to its greater robustness over the parametric paired *t*-test equivalent. Due to the exploratory nature of this study and so as to achieve a reasonable balance between Type I and Type II errors, Bonferroni-type correction for multiple comparisons was considered inappropriate.

The contribution of visuoperceptual function to tracking performance was removed using the concept of an invisible visuoperceptual buffer-zone (Jones, Donaldson, & Sharman, 1996). This zone extended either side of the target and was the equivalent of sweeping a circle of radius equal to the dynamic perception resolution along the target trajectory. If participants could hypothetically track the target perfectly except for visuoperceptual limitations, they would be within or, at worst, on the boundary of the visuoperceptual zone at all times. Consequently, the contribution of visuoperceptual limitations to a participant's tracking trajectory could be removed by moving each sample of the trajectory towards the target by the width of the visuoperceptual zone at the level of the arrow. The modified tracking output data could then be reanalysed to give a reasonable estimate of performance equivalent to that which the participant would have achieved with perfect visual acuity and perception.

3. Results

Conflicting results were obtained on the two measures of visual acuity used in the study (Table 1). The stutterer group had poorer visual acuity than the control group on the Snellen chart (6/5.7 vs. 6/4.5, 27%, $p = 0.028$) whereas no significant difference between the groups was found on the computerized visual resolution test (0.42 vs. 0.52 mm, 19%, NS). Irrespective, the stutterers were found to be impaired on dynamic visual perception (1.74 vs. 1.21 mm, 44%, $p = 0.028$).

On the visuomotor tasks (Table 1), the stutterers had slower reaction times (290 vs. 261 ms, 11%, $p = 0.028$) and were less accurate on the non-preview random

Table 1
Performance of stutterer and control groups on sensory-motor tests

Performance measure	Stutterers (mean ± SD)	Controls (mean ± SD)	Difference (mean)	Difference (%)	p
Visual acuity (Snellen) (m)	5.7 ± 1.4	4.5 ± 0.9	1.2	27	*
Visual resolution (mm)	0.42 ± 0.11	0.52 ± 0.19	−0.1	−19	NS
Static perception (mm)	1.03 ± 0.07	1.00 ± 0.02	0.03	3	NS
Dynamic perception (mm)	1.74 ± 0.71	1.21 ± 0.16	0.5	44	*
Reaction time (ms)	290 ± 22	261 ± 34	29	11	*
Peak velocity (mm/s)	976 ± 283	1117 ± 189	141	13	NS
Unsteadiness (mm/s)	2.17 ± 0.91	2.03 ± 0.86	0.14	7	NS
Sine (npv)					
Error (mm)	7.35 ± 2.20	6.97 ± 1.90	0.38	6	NS
Lag (ms)	93.2 ± 39.2	97.4 ± 36.6	−4.2	−4	NS
Random (pv)					
Error (mm)	6.25 ± 1.99	5.38 ± 1.51	0.87	16	NS
Lag (ms)	65.3 ± 76.9	63.2 ± 54.7	2.1	3	NS
Random (npv)					
Error (mm)	6.14 ± 1.31	5.30 ± 0.87	0.84	16	∼
Lag (ms)	117.0 ± 37.1	102.3 ± 29.1	14.7	14	NS
Step (npv)					
Error (mm)	10.57 ± 1.17	10.13 ± 1.46	0.44	4	NS
Lag (ms)	588.6 ± 79.8	574.4 ± 98.0	14.2	2	NS

pv: preview; npv: non-preview; NS: not significant; $\sim p < 0.10$, $^{*}p < 0.05$.

tracking task although this difference did not reach significance (6.14 vs. 5.30 mm, 16%, $p = 0.06$). Stutterers were also less accurate than the controls on the preview random tracking task but this result was also non-significant (6.25 vs. 5.38 mm, 16%, $p = 0.14$). No significant differences were found on steady movement or on sine or step tracking.

Severity of stuttering (Riley SSI) correlated with dynamic perception $r(10) = 0.79$, $p = 0.002$ and reaction time $r(10) = 0.58$, $p = 0.047$.

To estimate the contribution of the visuoperceptual deficits to the impaired tracking performance of the stutterer group, the raw tracking data for each subject in the stutterer and control groups were reanalysed following removal of the visuoperceptual resolution. Differences between the subsequent visuoperception-removed tracking error scores were substantially smaller than for the raw tracking errors (Table 2).

Table 2
Tracking errors after removal of dynamic perception component

Task	Stutterers (mean ± SD)	Controls (mean ± SD)	Difference (mean)	Difference (%)	p
Sine (npv) (mm)	5.74 ± 1.77	5.81 ± 1.81	−0.07	−1.1	NS
Random (pv) (mm)	3.39 ± 1.68	3.33 ± 1.38	0.06	1.8	NS
Random (npv) (mm)	4.58 ± 1.12	4.16 ± 0.77	0.42	10.0	NS
Step (npv) (mm)	9.72 ± 1.00	9.51 ± 1.42	0.21	2.1	NS

pv: preview; npv: non-preview; NS: not significant; $\sim p < 0.10$, $^{*}p < 0.05$.

In particular, the difference on the preview random task reduced from 16% to 2%, indicating that the mildly inferior performance of stutterers on the preview random tracking task can be mostly attributed to impaired visuoperceptual function.

4. Discussion

This is the first study to provide preliminary evidence for the presence of *non-linguistic visuoperceptual deficits* and *upper-limb visuomotor tracking deficits* in people with moderate to severe stuttering.

A deficit was found in the stutterers on dynamic visual perception (44%) but only to a minimal degree, at most, on static visual perception (3%, $p = 0.11$). This suggests a possible subtle deficit in the perception of static visuospatial relationships that can, for the most part, be accommodated for, given sufficient stimulus exposure time. Conversely, when the stimulus time is limited and/or the stimulus is moving, as is the case in the dynamic perception task, the visuospatial deficit may be considerably exacerbated. This may reflect a subtle reduction in central processing *capacity* for visual stimuli, similar to that proposed by Neilson and Neilson (1991) for auditory stimuli in stutterers. Further investigation using static, tachistoscopic and dynamic non-linguistic visuospatial stimuli would be needed to determine whether the visuoperceptual deficit was related more to the brevity or to the dynamic nature of the stimulus.

It is of interest that similar subtle visuospatial deficits have been found in patients with Parkinson's disease (PD) using the same tests (Jones & Donaldson, 1995). As with stutterers, the mechanism between PD and visuospatial deficits is unclear (Jones & Donaldson, 1995; Jones et al., 1992). Mohr, Litvan, Williams, Fedio, and Chase (1990) suggest that mild visuospatial deficits in PD may implicate dorsolateral striatal–frontal connections, which have been tentatively linked to spatial memory (Alexander, DeLong, & Strick, 1986). Alternatively, Blonder, Gur, Gur, Saykin, and Hurtig (1989) suggest that visuospatial deficits in PD may reflect thalamic dysfunction. It is also of interest that visuospatial processing is markedly affected in Huntington's disease (Brouwers, Cox, Martin, Chase, & Fedio, 1984; Mohr et al., 1991). This disorder is characterised by marked degeneration in the caudate and putamen and by concomitant striatal hypometabolism (Kuhl, Metter, Riege, & Markham, 1984; Young et al., 1986) – the latter having also been demonstrated in stutterers, specifically in the left caudate (Wu et al., 1995). Furthermore, Mohr et al. (1991) provide preliminary evidence for the impaired spatial manipulation (e.g., spatial rotation) in Huntington's disease being a consequence of neostriatal degeneration, as opposed to gross reduction in overall visuospatial ability due to global atrophy.

A small significant difference in visual acuity was found between the two groups using the Snellen chart but not the visual resolution task; in fact, the trend on the latter task was for better performance in stutterers. This discrepancy may be related to the fact that only a participant's best eye score was used for visual acuity on the Snellen chart while visual resolution allowed use of both eyes. These results, along

with a finding elsewhere of normal visual acuity (Hamilton, 1940), suggest that the primary sensory processes subserving vision are most likely normal in stutterers. This notwithstanding, the presence of a subtle deficit in high-contrast visual acuity would be in keeping with that in PD (Jones et al., 1992) and be a further example of a number of deficits in common between persons who stutter and those with PD (as indicated elsewhere in this section).

The stutterers were found to have slower reaction times than non-stutterers on the ballistic movement task. This parallels prior research which has shown slower finger/hand reaction times in stutterers (Cross & Luper, 1983; Hand & Haynes, 1983; Rastatter & Dell, 1985; Starkweather et al., 1984; Webster & Ryan, 1991) and lends support to the view that stuttering reflects a more generalized motor disorder than simply that of speech. This abnormality may reflect a more widespread difficulty with initiation of movement involving the SMA, basal ganglia and anterior cingulate cortex which is considered to be associated with stuttering (Caruso, 1991; Webster, 1988).

Stutterers were also less accurate at tracking a smooth-changing random visual target irrespective of presence or lack of preview. While there are differences between the tasks in Neilson's (1980) and our studies with respect to sensor, target signal and sensor-display compatibility, it is difficult to see how these might account for the different findings.

Poorer random tracking performance in stutterers could reflect a motor deficit (such as difficulty initiating corrective movements), a visuoperceptual deficit, or a deficit in motor planning or sensory-motor integration. However, our ability to remove the visuoperceptual component from tracking performance data indicates that the poorer preview random tracking performance can be almost entirely attributed to impaired dynamic visuoperceptual function. A possible residual deficit on the non-preview random task is at most marginal (10%, $p = 0.18$) and could be explained by slightly prolonged reaction times, these having more impact on a non-preview task for which there is less scope for predictive motor planning. A lack of difference between the two groups on the step tracking task, with its much greater inherent unpredictability, adds weight to our contention that slower reactions are not the primary factor leading to impaired random tracking. The similarity of the level of deficits (both 16%) on the preview and non-preview modes of random tracking indicates that the stutterers were not impaired in their ability to improve performance when given a preview of the target. In contrast, PD patients have been found impaired in their ability to utilise preview of the target (Jones & Donaldson, 1989). In addition, the fact that stutterers were impaired on non-preview tracking with a random but not a sinusoidal target indicates that they are able to utilise the repetitive nature (i.e., periodicity) of sine waveform to improve performance but are deficient in their ability to maximally utilise the more limited predictability of the random target to improve tracking performance. This deficiency could simply be a consequence of the deficits in dynamic perception and reaction time seen in the stutterers, although one cannot discount the presence of an independent abnormality in stochastic prediction itself. Interestingly, in their preliminary study of visuomotor tracking in older boys, Zebrowski et al. (1997) found the converse to our study, with their stutterers impaired on lower-lip tracking of sinusoidal, but not random, targets.

Several of our findings are similar to those observed in PD patients. They, like the stutterers in the present study, have slower reaction times and arm speed in ballistic tasks (Bloxham, Mindel, & Frith, 1984; Evarts, Teräväinen, & Calne, 1981; Jones & Donaldson, 1989; Sheridan, Flowers, & Hurrell, 1987). Both stuttering and PD are motor disorders characterised by overt difficulties with the initiation and maintenance of movement. PD is associated with degeneration of substantia nigra pars compacta (SNpc). The projection from SNpc to posterior putamen is dominantly affected but, later, nigral projections to anterior putamen and head of caudate may also become involved with disease progression (Brooks et al., 1990; Kish, Shannak, & Hornykiewicz, 1988). There are several reasons for believing that stuttering, like PD, may have an origin in defective functioning of the basal ganglia. Firstly, cerebral blood flow within the left caudate is reduced in developmental stutterers during both fluent and dysfluent speech (Wu et al., 1995). Secondly, 80% of patients with acquired stuttering have lesions of the caudate, putamen, or pallidum (Ludlow et al., 1987). Thirdly, there is a greater incidence of involuntary movements in stutterers during both fluent and dysfluent speech (Mulligan, Anderson, Jones, Williams, & Donaldson, 2001).

Although the precise role of the basal ganglia in motor control remains uncertain (Marsden & Obeso, 1994), primate studies have provided important indicators. From single unit recordings and pharmacological lesions in the globus pallidus, Brotchie, Iansek, and Horne (1991a,b) concluded that the basal ganglia generate phasic internal motor cues for predictable movement sequences which switch off sustained premovement activity in the SMA for the impending movement and to turn on the preparatory phase of sustained activity for the next, allowing each movement in the sequence to be executed. Observations of increased activation of SMA during voluntary limb movement (Colebatch, Deiber, Passingham, Friston, & Frackowiak, 1991) and eye movement (Anderson, Jenkins, Brooks, Frackowiak, & Kennard, 1994) are consistent with this proposal. Similarly, the failure to activate SMA during voluntary movements in PD patients is consistent with disruption of the phasic discharges in the pallido–thalamic–SMA projections (Jenkins et al., 1994; Playford et al., 1992). Stuttering is characterised by speech blocking and repetitions. Thus, it is tempting to speculate that these abnormalities, representing a failure to switch from the current speech movement to the next, emanate from similar disruption of phasic discharges in basal ganglia projections to speech cortex (i.e., SMA, cingulate, Broca's area and Wernicke's area).

Mink and Thach (1991) found that inhibition of monkey pallidal neurons with the GABA agonist muscimol resulted in involuntary muscle co-contraction during voluntary movement, leading them to conclude that the normally functioning basal ganglia acts to inhibit other motor centres and thereby suppress inappropriate muscular activity during the running of desired motor programmes. Failure of this suppression of inappropriate muscular activity may underlie the rigidity and bradykinesia of PD (Miller & DeLong, 1987). Likewise, it might be argued that dysfunction of the basal ganglia in stutterers may also result in failure of such suppression and produce the stereotypic facial movements (Bloodstein, 1987; Mulligan et al., 2001; Riley, 1972) and increased laryngeal muscle tension (Freeman & Ushijima, 1978;

Starkweather, 1982) during dysfluent speech and increased incidence of involuntary movements seen during fluent speech (Mulligan et al., 2001).

Overall, there is a definite similarity between the distribution, but striking differences in the characteristics, of secondary deficits in stuttering and PD. We believe that the common factor is an abnormal dopaminergic system which is paradoxically overactive in stutterers (Wu et al., 1997) and underactive in PD. This helps explain why both have deficits of certain functional areas, such as visuomotor and visuoperceptual, but indicates a different mechanism to account for the deficits. In PD, dysfunctional neurotransmission leads to impaired function. In contrast, in stuttering an abnormally high dopaminergic activity appears to inhibit cortical processing, as evidenced by reduced cerebral glucose metabolism (Wu et al., 1995) and blood flow (Braun et al., 1997; Pool et al., 1991); it should be noted, however, that other studies have seen no evidence of reduced blood flow (Fox et al., 2000; Ingham et al., 1996). While no abnormalities of glucose metabolism, blood flow, or presynaptic dopaminergic activity have been seen in the visuoperceptual areas of the brain, in particular the right parietal cortex, abnormally high levels of dopamine in certain areas of the brain are a likely cause of slight reductions in visuospatial processing capacity, as most evident on visual perception. This could be mediated by the influence of an underactive caudate nucleus (Wu et al., 1995) projecting to the parietal cortex via the globus pallidus and thalamus (Afifi, 1994; Leichnetz, 2001).

Further evidence for the pathogenesis of stuttering being associated with excessive dopaminergic activity has been shown by substantial reductions in severity of stuttering seen in stutterers injected with the dopamine blocker haloperidol (Burns, Brady, & Kuruvilla, 1978). It is also supported by the observation that patients with PD and speech dysfluency have their dysfluency exacerbated when treated with levodopa (Anderson, Hughes, Rothi, Crucian, & Heilman, 1999; Louis, Winfield, Fahn, & Ford, 2001).

Several issues have arisen from this study that warrant consideration in future research in this area. Firstly, in selecting participants for the dysfluent group it may be necessary to use a more comprehensive stuttering measure than the Riley SSI alone. The Riley SSI is an adequate screening tool that considers dysfluency during monologue and reading aloud but does not consider dysfluency during conversation. Even though the participants in our study were selected on the basis of a moderate to severe Riley score, we found that the speech samples recorded during conversation tended to be more representative of the participants' natural speech, containing more dysfluencies than in the other two contexts. The Riley SSI was found to correlate with the conversation score (percentage dysfluency during conversation) but not as strongly as might have been expected ($r = 0.68$, $p < 0.05$). Secondly, it would be of interest to study a sample of left-handed stutterers and non-stutterers on the same tests. Only right-handers were included in the current study and only right arm function tested. However, as stutterers appear to have atypical cerebral lateralisation (Forster & Webster, 2001; Moore, 1984; Yeudall, 1985) it would be interesting to determine the extent to which the findings of this study apply to left-handed stutterers. Similarly, it would be of interest to compare right and left arm function in right-handed stutterers. Importantly, a prospective study of a larger number of stutterers

and matched controls would be of considerable value in helping confirm the preliminary and, in some cases, marginal findings of visuoperceptual and visuomotor deficits from this study.

While the precise cause of developmental stuttering remains unclear, recent research points strongly to a neurogenic cause and, more specifically, to stuttering being a neuromotor disorder characterised by an abnormality of higher control encompassing not only speech but other motor systems. This study supports this view, having found both visuomotor and visuoperceptual deficits in persons who stutter.

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