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DYSFLUENCY AND INVOLUNTARY MOVEMENTS: A NEW LOOK AT DEVELOPMENTAL STUTTERING*

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Studies using modern imaging techniques suggest that, in developmental stuttering, there is dysfunction within the cortical and subcortical areas of the motor control system wider than that pertaining to speech motor control alone. If this is the case, one might expect motor deficits extending beyond and unrelated to the production of speech in people who stutter. This study explored this proposal by investigating the presence and characteristics of involuntary movements accompanying stuttering. Sixteen adults with developmental stuttering and 16 controls matched for age and sex were audio-video-taped during 5 minutes of conversational speech and reading a passage of 350 words. Audio-data were examined for dysfluencies. Movements of the face, head and upper body considered involuntary and not part of normal facial expression or gesture and not part of the mechanics of speech were identified and described from muted video-data.

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Subjects who stuttered had a higher proportion of classic (within-word) dysfluencies accompanied by involuntary movements (IMs) than controls during speech (24.4% *vs.* 4.5%, $p = .054$) and reading (28.6% *vs.* 4.9%, $p = .033$). There was no difference in proportion of classic dysfluencies accompanied by IMs between speech and reading for either group. IMs were also seen in both groups during fluent speech, with a similar incidence during free speech (3.9% *vs.* 3.0%, NS) but a greater incidence in the subjects who stuttered during reading (2.4% *vs.* 0.8%, $p = .03$). In contrast, there was no difference between the two groups for IMs accompanying normal (between-word) dysfluencies. This suggests that classic and normal dysfluency and their accompanying IMs have different etiologies. The notion that stuttering and IMs are due to altered function in a motor control system wider than that of speech motor control alone is supported by a higher incidence of IMs in people who stutter during both classic dysfluencies *and* fluent speech.

Keywords: Stuttering; Involuntary movements; Motor control

INVOLUNTARY MOVEMENTS IN STUTTERING

Evidence from modern imaging techniques of brain function indicates that the cause of developmental stuttering may lie in dysfunction of either or both of the cortical and subcortical motor systems of the central nervous system. Abnormal movements have long been described as accompanying developmental stuttering as a secondary feature (Bloodstein, 1993). They have traditionally been thought to emerge and intensify as stuttering becomes more severe and chronic (Bloodstein, 1960). If stuttering is caused by subcortical dysfunction, because of the influence of the subcortical areas on the control of movement, it could be hypothesized that the abnormal movements are also caused by the same dysfunction and as such are a primary feature of the stuttering phenomenon. This study characterizes these movements in developmental stuttering and investigates their association with speech in order to explore their potential relationship with the systems causing stuttering.

Whilst many theories on the cause of stuttering have been advanced, there has been considerably less emphasis on the cause and significance of abnormal movements of the face, head and body which accompany stuttering and which do not appear to be part of facial expression or related to speech gestures. They have been variously termed ancillary body movements (Wingate, 1964), associated symptoms (Bloodstein, 1960), accessory features (Van Riper, 1971), physical concomitants (Riley, 1972), nonverbal accessory behaviors (Kraaimaat and Janssen,

1985), secondary symptoms (Bloodstein, 1993), and nonspeech behavior (Conture and Kelly, 1991). To date, the movements have been described clinically as being most often associated with stuttering blocks and consisting of various kinds of movements such as jerking of the head, shutting of eyes, sticking out the tongue, clenching the fists, gasping, and sudden expiratory thrusts of air (Bloodstein, 1993). They have been investigated by counting as individual movement units (Conture and Kelly, 1991; Prins and Lohr, 1972; Schwartz, Zebrowski, and Conture, 1990; Schwartz and Conture, 1988). These movements have been traditionally classified as a conditioned or learned response to stuttering (Prins and Lohr, 1972), being thought to emerge and increase as stuttering progresses towards adulthood (Bloodstein, 1960; Conture and Kelly, 1991; Van Riper, 1971).

Observations of very young children who stutter show the movements to be present at an early stage (Yiari, Ambrose, and Niermann, 1993; Zebrowski, 1995). For example, Schwartz and Conture (1988) found that all 43 children (aged 3–10 years) in their study produced these movements to a greater or lesser degree, regardless of how developed their stuttering problem appeared to be. In addition, early stuttering often takes on a moderate to severe form but has a strong tendency for a reduction to occur in both the stuttering dysfluency and the number of head and face movements over time, in many cases with complete recovery (Yiari and Ambrose, 1992a). These findings support a primary association of these movements with stuttering itself and contradicts the influential and traditional views of stuttering onset as a gradual process (*e.g.*, Bloodstein, 1960) in which the movements are considered to appear after the stuttering has become more entrenched. They lend support to the hypothesis that these movements are an inherent part of the stuttering disorder itself and therefore less likely to be learned.

Caruso (1991) reviewed a growing body of data suggesting that stuttering may be a neuromotor deficit of temporal control of both speech and nonspeech movements. He suggests that, because abnormalities in the timing and coordination of speech, eye and finger movements have been demonstrated in those who stutter, there may be deficient functioning in areas such as the supplementary motor areas (SMA) and/or the basal ganglia in the condition.

This hypothesis may be further supported by findings of Kraaimaat and Janssen (1985) who studied what they called nonverbal accessory facial behavior in 25 male adults who stuttered. Most (75%) of the movements occurred during stuttering but some of the movements, notably eye blinks, were unrelated to stuttering, being just as likely to occur with fluent speech or with what the authors termed normal dysfluencies. Normal dysfluencies are interjections and repetition of words or phrases uttered by normally fluent people and do not distinguish those who are regarded as stuttering from those who are not. The authors concluded that the movements function in different ways, some being conditioned responses to speech-associated anxiety, with others the result of deficits in fine motor control of speech-muscle systems.

One study of the frequency and type of abnormal movements in children who stutter used the Facial Action Coding System to count individual movement units of the face or neck (Conture and Kelly, 1991). Although the authors found a higher incidence of movements in the children who stuttered compared to their fluent peers, there was considerable overlap in the type of movements found between the two groups.

In a more recent study, Kiziltan and Akalin (1996) claimed a striking similarity between the involuntary movements (IMs) in 23 adults who stuttered to the IMs in patients with dystonic syndromes. This is in keeping with the finding that stuttering is more prevalent in the families of patients with idiopathic torsion dystonia, a genetically determined movement disorder considered to be due to a basal ganglia abnormality (Fletcher, Harding, and Marsden, 1991). Similarly, stuttering has a genetic predisposition suggesting an inherited deficit in brain structure or function (Ambrose, Yairi, and Cox, 1993; Kidd, 1980; Mellon, Umar, and Hanson, 1993), with the risk of stuttering in first degree relatives being more than three times the population risk (Andrews *et al.*, 1983). In addition, monozygomatic twins have a higher concordance (63% *vs.* 19%) for stuttering than do same-sexed dizygomatic twins (Howie, 1981). Spontaneous recovery or, alternatively, chronicity of stuttering is also influenced by genetic factors (Yairi, Ambrose, and Cox, 1996).

A number of studies using modern imaging techniques have investigated both cortical and subcortical areas in the brain in an attempt to

shed light on the cause and mechanisms of developmental stuttering. Two studies, using single-photon emission computed tomography (SPECT), found global cerebral blood flow (CBF) reduction in people who stutter compared with their fluent peers in regions of the cerebrum responsible for speech-motor control (Pool, Devous, Freeman, Watson, and Finitzo, 1991; Watson *et al.*, 1994). In a study of four men with developmental stuttering and four fluent controls, using the more sensitive positron emission tomography (PET) technique, decreased metabolism was seen in the regions of the cerebral cortex associated with speech and language control (*i.e.*, Broca's area, Wernicke's area and frontal pole) and in the left caudate nucleus during stuttering (Wu *et al.*, 1995). Importantly, reduced metabolism was also seen in the left caudate during fluent speech of the subjects who stuttered.

Four further studies have used PET to focus on the suggestion that people with developmental stuttering have physiological differences in regions of the brain when compared to fluent peers. Ingham *et al.* (1996) found no significant differences in regional cerebral blood flow (rCBF) between 10 subjects who stuttered and 19 controls during the resting state and considered that this provided evidence against the presence of focal functional lesions in persons who stutter when at rest. In addition, there was no evidence to support the theory that stuttering may be associated with an absence of the normal asymmetry between cerebral hemispheres. In contrast, in a subsequent study which included reading conditions which induced stuttering (solo reading) or fluency (chorus reading), stuttering induced widespread overactivation in both cerebrum and cerebellum, particularly on the right side (Fox *et al.*, 1996). In addition, the auditory system, which is thought to support the self-monitoring of speech, lacked the normal left-side activation. Induced fluency largely reversed these abnormal activation patterns. They concluded that stuttering is a physiological disorder affecting the multiple neural systems used for speaking.

A study by Braun *et al.* (1997) had similar findings, particularly with respect to overactive right-sided brain activity during stuttered speech that did not, however, normalize during fluent speech. In addition the anterior forebrain regions (which play a role in the regulation of motor function) were found to be disproportionately active in developmental stuttering, while post-rolandic regions responsible for perception and decoding of sensory information were relatively silent. These results

suggest that there are underlying differences in sensory-motor function in those who stutter. The authors proposed that the anterior regions cannot efficiently coordinate speech because the posterior regions fail to provide the integrated sensory feedback necessary for this co-ordination. The basal ganglia and cerebellum – areas involved in the initiation and regulation of motor activity – were also found to have raised rCBF during stuttering.

A recent study by Fox *et al.* (2000) used stuttering measures from rest, fluent and dysfluent speech tasks in 10 men who stuttered and compared these to 10 controls matched for age and sex. They found positive correlates for stuttering in the regions of the primary motor cortex, supplementary motor cortex, Broca's area, the anterior insula on the right and the cerebellum on the left with decreased activation in the primary auditory and associated areas in the right hemisphere. This suggests that the causes of stuttering lie in the speech motor regions of the right cerebral hemisphere and left cerebellum and that feedback of one's own speech is suppressed with stuttering.

With the finding of involvement of the subcortical areas in developmental stuttering, Wu *et al.* (1997) used PET imaging to measure presynaptic dopamine activity to investigate the hypothesis that stuttering may result from excessive dopamine activity. Their preliminary study of three men with moderate stuttering used 6-FDOPA to demonstrate a significant increase in dopamine metabolism in the cortex and subcortical regions associated with speech even when the men were at rest. This included a three-fold increase in the left caudate tail and the right medial prefrontal cortex and a two-fold increase in limbic structures of the subjects who stuttered compared to six fluent controls. This observation of an increase in dopamine activity in persons who stutter is compatible with the finding that stuttering is improved by the dopamine blocker haloperidol (Brady, 1991).

In summary, several studies indicate a functional dissociation between areas that control motor output and those that affect this output by auditory feedback and language processing. Furthermore, this dissociation may be due to an overactive presynaptic dopamine system that leads to altered metabolism in regions that control speech.

The present study was undertaken to investigate the hypothesis that the abnormal movements seen in people who stutter reflect a motor control disorder related to the cause of the stuttering itself. If the

movements are the result of subcortical dysfunction, including basal ganglia and cerebellar dysfunction, they may be viewed as an epiphenomenon of stuttering instead of appearing as a result of stuttering. They may, therefore, be present even when speech is fluent. We compared the abnormal movements in people with developmental stuttering to those in matched fluent peers to characterize the movements. We then investigated relationships between the movements and different types of dysfluency and fluent speech.

METHOD

Subjects

The experimental group comprised subjects aged 14 years or older with developmental stuttering and without diagnosed neurological or psychiatric condition. English was their preferred language. Subjects were included in the study if they produced three or more stuttering dysfluencies per 100 words of conversational speech or reading. Stuttering dysfluencies were defined as prolongation and repetition of sound or syllable as defined in Riley's Stuttering Severity Instrument (Riley, 1972). Of the 27 possible recruits for the study, 16 met the inclusion criteria.

Each of these 16 study subjects (mean age = 41.7, $SD = 14.6$, range = 15 – 67 years) were matched by age (± 5 years if adult and ± 1 year if under 20 years), and sex with a fluent peer to form the control group. Control subjects (mean age = 41.3, $SD = 15.8$, range = 16 – 70 years) were recruited from friends of the experimental subjects or from acquaintances of the principal investigator. There were 11 males and five females in each group of subjects. Thirteen were right- and three left-handed. Of the subjects who stuttered, three were classified as very mild, seven as mild, five as moderate, none as severe and one as very severe on the Riley's Stuttering Severity Instrument (Riley, 1972).

Apparatus

Subjects were seated comfortably in a quiet room in front of a plain background. A video camera (Sony Hi8 CCD-TR91OE) was placed

on a tripod 3 m away from the subject. A lapel microphone (Sony Electret ECM-T110) was attached to the subject's clothing 15–20 cm away from the mouth. A reading passage of 350 words taken from a popular magazine, enlarged and mounted on a stand was placed at a distance of 1.0–1.5 m in front of the subject depending on his/her preferred reading distance.

Procedure

Subjects were asked to read the passage aloud in their normal voice and speed whilst being audio-videotaped. This was followed by 5 minutes of free conversational speech with the principal investigator. The subject was asked to speak about his/her work or school, family or interests.

Analysis of Data

The reading task consisted of the middle 300 words of the reading passage. The speech task consisted of 300 words of free speech, taken by transcription from the middle of the five minutes of conversational speech. The reading and free speech tasks were analyzed in the same manner for both subject groups.

Dysfluent words were identified and marked on the reading script and the transcript of the free speech by one researcher (MJW) to maximize reliability. Each was classified by the same researcher as either a prolongation, sound repetition, syllable repetition, interjection, word repetition, phrase repetition, revision, incomplete phrase or broken word. There is controversy and discussion in the literature regarding the classification of dysfluencies as stuttering dysfluencies or what might be called normal dysfluencies. The latter would be those dysfluencies uttered by normally fluent speakers that do not distinguish a person who stutters from a person who is normally fluent. It was decided for the purposes of this study, therefore, to include both within-word and between-word dysfluencies as suggested by Cordes and Ingham (1995) and the World Health Organisation (1992). Consequently, dysfluencies were grouped into classical dysfluencies, comprising prolongation and

part-word repetition of sound or syllable, and normal dysfluencies, being whole-word or between-word dysfluencies.

Using the time locked on to the video data, each individual movement of the face, head and upper body considered involuntary was identified and described according to location and duration by a neurologist (TJA) with a specialist interest in movement disorders. Subject data samples were presented randomly for analysis and with the audio turned off, so that the neurologist was not explicitly aware whether the sample was from an experimental or a control subject. Movements were identified only if they were not considered to be part of normal facial expression (*e.g.*, smiling) or movement (*e.g.*, eye blinks) or gesture (*e.g.*, head movements in agreement or disagreement) and not part of the mechanics of speech. Individual involuntary movements (IMs) were grouped into those of the upper face (prolonged eye closure, repetitive eye blinking, brief eyebrow arching, and movements of the eyes to each side or upwards), lower face (facial grimace, mouth grimace, repetitive lip movement, lip smacking, lip pursing, tongue protrusion, forceful swallowing and jaw shuddering), movements of the head and neck (head jerking and sustained movements of the neck to either side) or movements of the upper limbs (arm jerking or hand scratching). Movements identified in this way were matched to type of word-fluent, classically dysfluent or normally dysfluent-using the time code on the video recording. Each word was deemed either to have an IM accompanying it or to be free of IMs. Where more than one IM occurred with a particular word, only one movement was counted.

Statistical Analysis

Although data were interval, it was considered to be nonparametric due to data being highly skewed, particularly in the stuttering group, with substantial differences in variances between the two groups. The Wilcoxon matched pairs test (one-tailed for comparison between experimental subjects and controls and two-tailed for within-group comparisons) was used for inferential statistics. The Spearman correlation coefficient was used for correlational analyses relating to severity of stuttering.

RESULTS

Dysfluency

As expected, the stuttering group had substantially more classic dysfluencies than the control group during free speech ($p = .0002$) and reading ($p = .0002$, Tab. I). The stuttering group also had more normal dysfluencies during speech ($p = .002$) and reading ($p = .003$, Tab. I). The number of classic dysfluencies within each group was similar for both reading and free speech (median for stuttering group during reading 25.2 *vs.* speech 23.7, $p = .354$ and median for controls during reading 0.8 *vs.* speech 1.6, $p = .345$) in contrast to normal dysfluencies which were considerably greater during free speech in both groups (stuttering group during speech 26.9 *vs.* reading 3.0, $p = .001$ and controls during speech 13.1 *vs.* reading 1.8, $p = .001$).

Involuntary Movements During Free Speech

IMs were found in both groups of subjects in each area of the face (*i.e.*, upper and lower face) and in movements of the head and upper limbs. Subjects who stuttered had a greater number of IMs than controls (overall total of 354 *vs.* 187 with a mean number per subject of 22.1 *vs.* 11.7, $p = .013$). Five types of IMs were not found in the controls, *i.e.*, eyes up, repetitive lip movement, lip smacking, forceful swallowing and jaw shuddering. Although these movements were found in subjects who stuttered, they were both infrequent and found only in a

TABLE I Median number of dysfluent words during the free speech and reading tasks

	Stutterers			*Controls*			
Dysfluency type	*S*	*Median*	*Range*	*S*	*Median*	*Range*	*p*
Free speech							
Classic dysfluencies	16	23.7	4–132	10	1.6	0–7	***
Normal dysfluencies	16	26.9	7–96	16	13.1	2–28	**
Reading							
Classic dysfluencies	16	25.2	4–128	9	0.8	0–5	***
Normal dysfluencies	14	3.0	0–50	10	1.8	0–7	**

S = number of subjects with the dysfluency type.
** $p < .01$.
*** $p < .001$.

small number (up to three) of subjects. A difference between the stuttering and control groups was found in the number of prolonged eye closures (41 *vs.* 1, $p = .009$) and head jerks (46 *vs.* 8, $p = .026$). Marginal differences were found in repetitive eye blinking (46 *vs.* 19, $p = .071$) and mouth grimacing (13 *vs.* 5, $p = .078$).

Involuntary Movements During Reading

The experimental group, as in the speech task, had a greater number of IMs (overall total of 297 *vs.* 47, mean number per subject of 18.6 *vs.* 2.9, $p = .001$). Twelve types of IMs (prolonged eye closure, repetitive eye blinking, eye movement upwards, facial and mouth grimacing, repetitive lip movement, lip pursing, forceful swallowing, jaw shuddering, sustained movement of the head to the left or right and scratching of the right hand) were found only in the group that stuttered although some were quite infrequent. Four IMs were found to occur with a greater incidence in the stuttering group. These were repetitive eye blinking (overall total of 10 *vs.* 0, $p = .034$), mouth grimacing (12 *vs.* 0, $p = .034$), head jerks (55 *vs.* 7, $p = .043$) and right arm jerks (17 *vs.* 1, $p = .014$). Brief eyebrow arching was marginally different (60 *vs.* 18, $p = .063$).

Involuntary Movements and Type of Word

The group who stuttered exhibited a higher proportion of words accompanied by IMs than controls during both speech (mean 6.4% *vs.* 3.8%, $p = .028$, Tab. II) and reading (mean number 5.5% *vs.* 1.0%, $p = .0005$). This group had a higher proportion of their classic dysfluencies accompanied by IMs than controls both during free speech (mean 24.5% *vs.* 4.5%, $p = .054$) and reading (28.6% *vs.* 4.9%, $p = .033$). The relative proportion of classic dysfluencies accompanied by IMs was, however, very similar within each subject group during speech and reading (experimental group mean 24.4% *vs.* 28.6% respectively, NS and controls 4.5% *vs.* 4.9%, respectively, NS). Only one of the 10 control subjects who had classic dysfluency during speech had IMs accompanying the dysfluency; this accounted for the group mean of 4.5%, with the proportion of dysfluencies accompanied by IMs in that subject being 45%. A similar situation was found

TABLE II Proportion of words (%) with involuntary movements according to dysfluency type during free speech and reading

	Stutterers				*Controls*				
Dysfluency type	*S*	*mean*	*median*	*range*	*S*	*mean*	*median*	*range*	*p*
Free speech									
Classic dysfluencies	16	24.4	18.9	0–100	10	4.5	0	0–45	~
Normal dysfluencies	16	18.5	17.1	0–69	16	31.5	21.6	0–95	
Fluent	16	3.9	2.5	0.4–15	16	3.0	3.0	0–6	
Total	16	6.4	6.0	0.3–14	16	3.8	3.8	0–7	*
Reading									
Classic dysfluencies	16	28.6	22.0	0–75	9	4.9	0	0–44	*
Normal dysfluencies	14	10.7	0	0–49	10	22.2	0	0–100	
Fluent	16	2.4	1.5	0–11	16	0.8	0.3	0–5	*
Total	16	5.5	2.8	0–15	16	1.0	0.5	0–5	***

S = number of subjects with the dysfluency type.
Total refers to the total number of words uttered including fluent and dysfluent.
~ $p < .10$.
* $p < .05$.
*** $p < .001$.

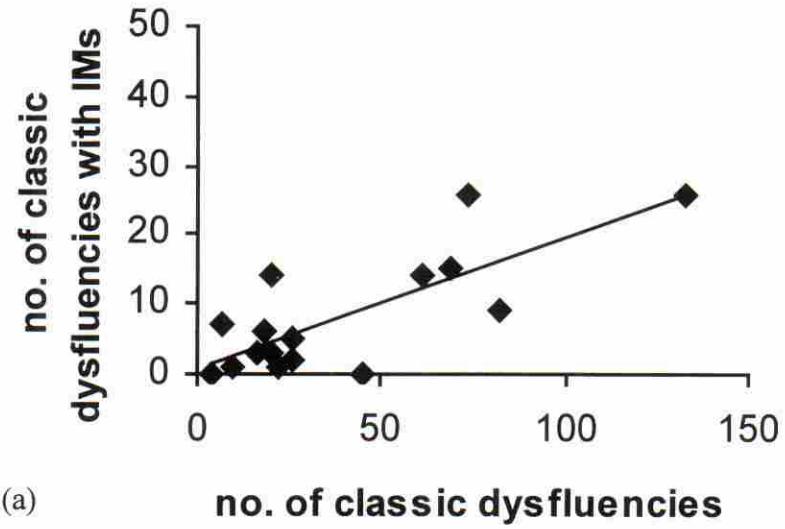
during reading with only one subject of the nine controls with classic dysfluencies exhibiting IMs accounting for the group mean of 4.9%, with the proportion of dysfluencies accompanied by IMs in that subject being 44%. This was not the same subject who exhibited the IMs in free speech.

In contrast, there was no difference between groups in the proportion of normal dysfluencies which were accompanied by IMs (experimental group mean 18.5% *vs.* controls 31.5%, $p = .802$ for speech and 10.7% *vs.* 22.2%, $p = .124$ for reading). There was also no difference between the proportion of normal dysfluencies accompanied by IMs during speech and reading in the group who stuttered (speech 18.5% *vs.* reading 10.7%, $p = .345$), or the controls (31.5% *vs.* 22.2%, $p = .859$. Four controls exhibited IMs with normal dysfluencies. One of these had 100% of normal dysfluencies accompanied by IMs.

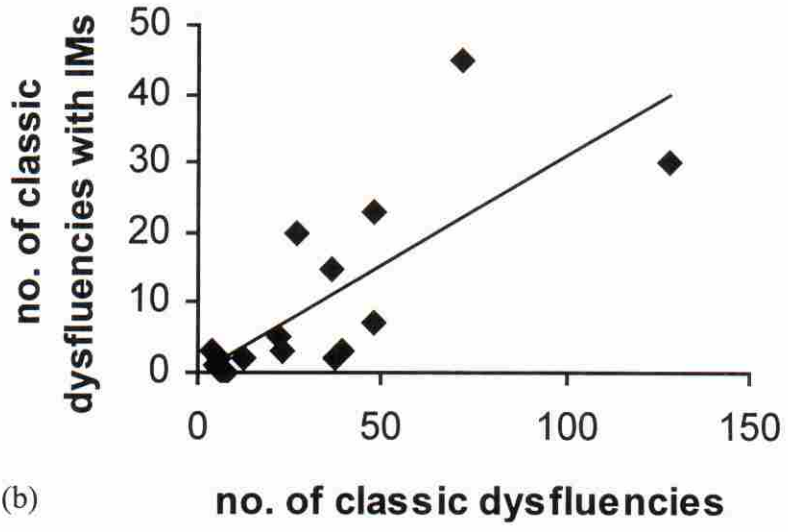
The proportion of fluent words accompanied by IMs was higher in those who stuttered than in controls during reading (2.4% *vs.* 0.8%, $p = .03$) but not during free speech (3.9% *vs.* 3.0%, $p = .204$).

Involuntary Movements in Relation to Stuttering Severity

For the experimental group of subjects who stuttered, a direct relationship was found between number of IMs and number of classic dysfluencies during free speech ($r_s = 0.573$, $p = .019$, Fig. 1a) and



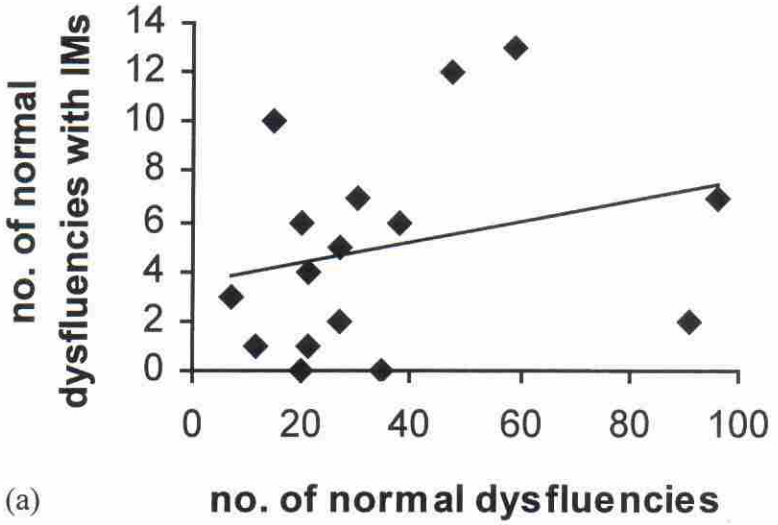
(a)



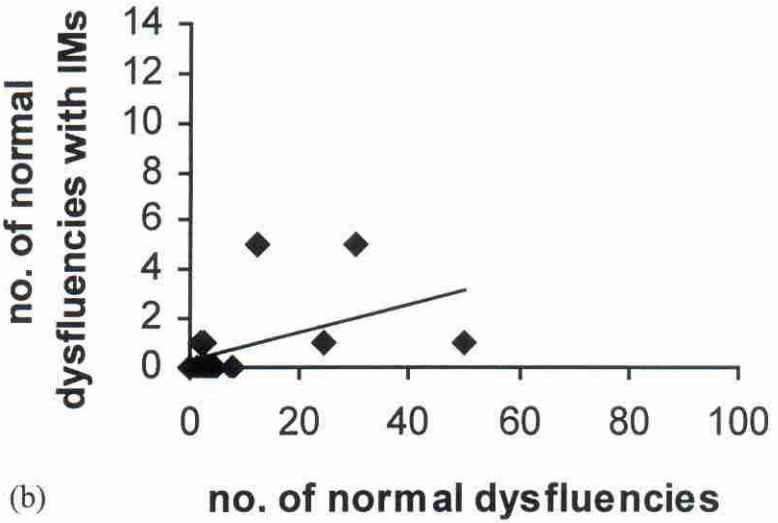
(b)

FIGURE 1 Relationship between number of words with IMs and stuttering severity in classic dysfluency for (a) free speech ($r_s = 0.573$, $p = .019$) and (b) reading ($r_s = 0.762$, $p = .0006$).

reading ($r_s = 0.762$, $p = .0005$, Fig. 1b). There was a similar though less significant relationship between number of IMs and normal dysfluencies during free speech ($r_s = 0.342$, $p = .194$, Fig. 2a) and reading ($r_s = 0.543$, $p = .029$, Fig. 2b).



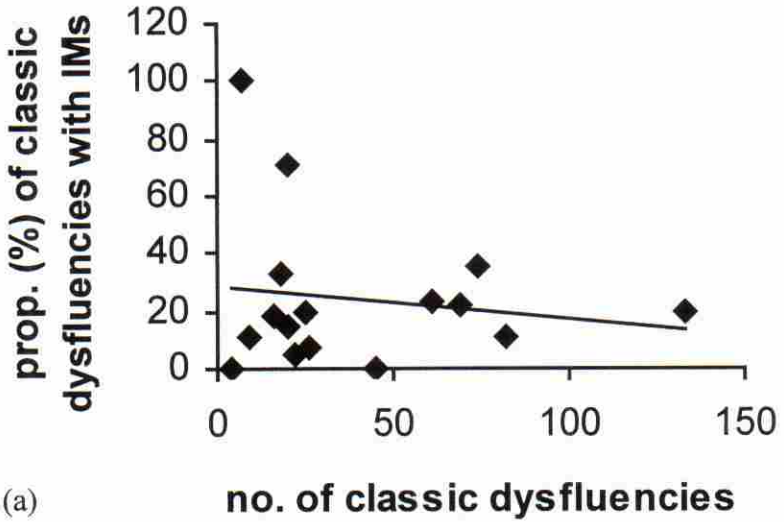
(a)



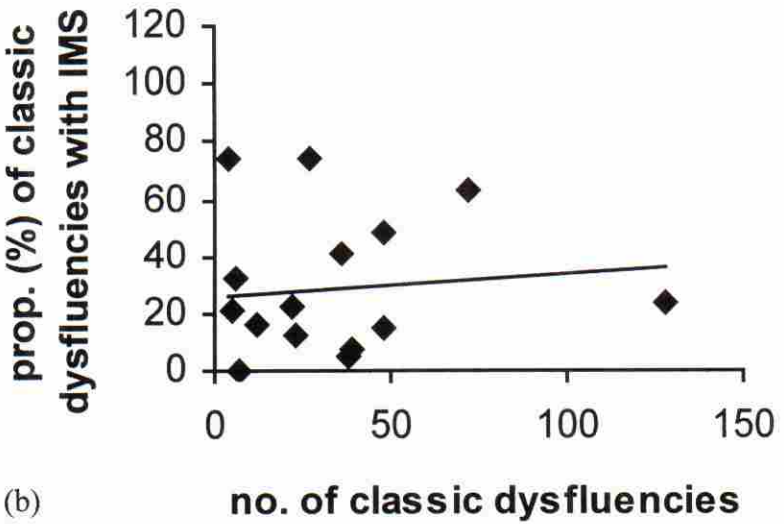
(b)

FIGURE 2 Relationship between number of words and stuttering severity in normal dysfluency for (a) free speech ($r_s = 0.342$, $p = .194$) and (b) reading ($r_s = 0.543$, $p = .029$).

In contrast, the proportion of classic dysfluencies with IMs was relatively constant irrespective of severity of stuttering in both free speech ($r_s = 0.047$, $p = .862$, Fig. 3a) and reading ($r_s = 0.098$, $p = .716$,



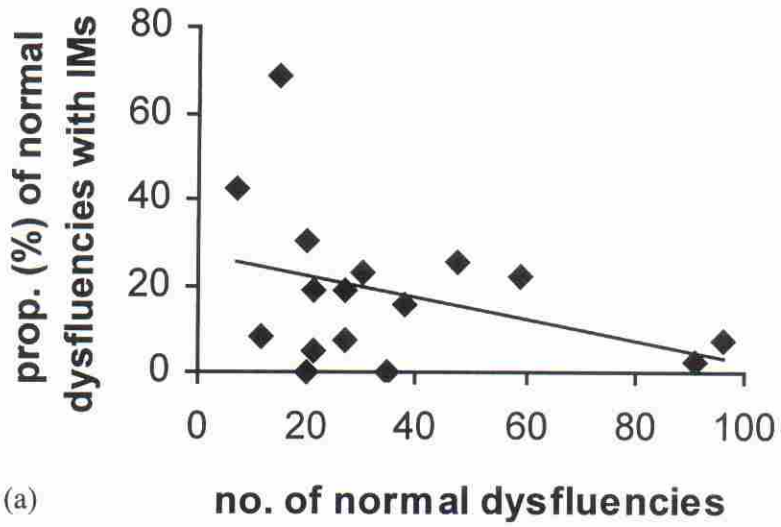
(a)



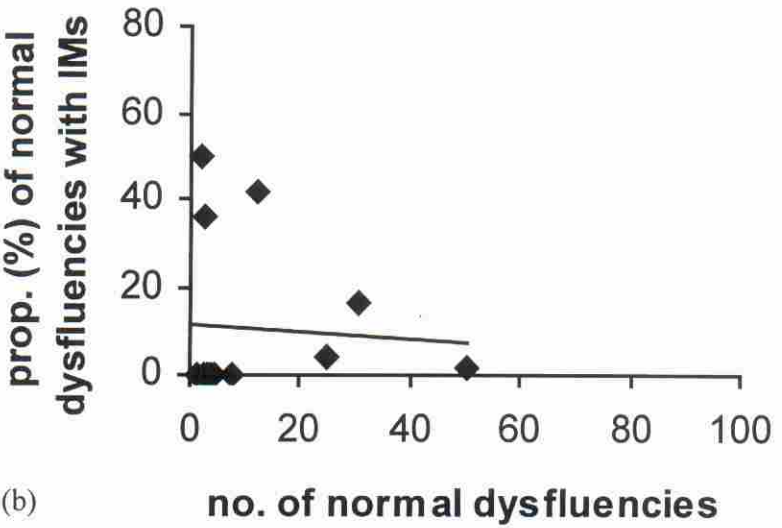
(b)

FIGURE 3 Relationship between proportion of words with IMs and stuttering severity in classic dysfluency for (a) free speech ($r_s = 0.047$, $p = .862$) and (b) reading ($r_s = 0.098$, $p = .716$).

Fig. 3b), with a similar finding for the proportion of normal dysfluencies with IMs during speech ($r_s = 0.347$, $p = .187$, Fig. 4a) and reading ($r_s = 0.236$, $p = .416$, Fig. 4b). That is, the probability of



(a)



(b)

FIGURE 4 Relationship between proportion of words with IMs and stuttering severity in normal dysfluency for (a) free speech ($r_s = 0.347$, $p = .187$) and (b) reading ($r_s = 0.236$, $p = .416$).

having IMs associated with either type of dysfluency (*i.e.*, classic or normal) is not related to the severity of stuttering during either free speech or reading.

DISCUSSION

This is the first study to compare involuntary movements (IMs) during free speech with IMs during reading and one of only two studies to compare IMs in persons with developmental stuttering to those in a matched group of normally fluent controls. Key findings of this study are:

(1) Persons who stuttered exhibited more IMs than controls during both reading and free speech.
(2) The number of classic dysfluencies, and the proportion of these accompanied by IMs, remained almost constant within each subject group irrespective of whether subjects were reading or speaking.
(3) The number of normal dysfluencies was considerably greater during free speech than during reading in both groups, although the proportion of these dysfluencies accompanied by IMs was the same for each condition in both groups.
(4) IMs accompanied a small proportion of fluent words in both subject groups during reading and speech.
(5) The proportion of IMs during fluent speech was greater in those who stuttered than in controls during the reading task but not during the free speech task.

This study on adults confirms the finding in children by Conture and Kelly (1991) of a higher prevalence of IMs in those who stutter than in fluent controls during free speech. It also confirms Conture and Kelly's finding that the frequency of IMs overall and the frequency of certain types of IMs, rather than an absolute difference in the type of IM, distinguishes the two subject groups.

Prins and Lohr (1972) conducted a study on 23 young adult males who stuttered to investigate IMs accompanying stuttered words during free speech. They found eyelid movement and suspension of jaw movements and lip activity occurred in the majority of the stuttered words evaluated. They did not, in contrast to the present study, identify eyebrow movements or any facial grimacing in their subjects despite having a category of "excessive movements of structures not directly involved in the utterance of speech" (Prins and Lohr, 1972, p. 64). It is possible that these movements may have been identified too

rarely to be included in their factor analysis accounting for 83% of the variance obtained. Dysfluencies were not separated into classic or normal and only ten words from each subject were investigated for the presence or absence of IMs.

The study by Kraaimaat and Janssen (1985) of accessory movements accompanying stuttering, like the present study, found forehead, head, jaw and mouth movements and a greater total number of movements accompanying classically stuttered words than normal dysfluencies and fluent words. The study by Kraaimaat and Janssen did not have a fluent control group so that between group findings cannot be compared with the present study.

The study by Kiziltan and Akalin (1996) did not count IMs, nor did it have a control group with which to compare IMs, so that no comparison can be made with the present study regarding the number of any of the IMs or of the total number of IMs.

Classic and Normal Dysfluencies

Traditionally, it has been within-word, as opposed to between-word, dysfluencies that have been regarded as stuttering (Riley, 1972). As expected, subjects who stuttered in our study had more dysfluent words than controls during speech and reading. Interestingly, when the dysfluent words were separated into within-word (called classic) and between-word (called normal) dysfluencies, it was found that the number of classic dysfluencies was the same within each subject group during free speech and reading. In contrast, the number of normal dysfluencies was substantially higher during free speech than reading in each subject group. This finding suggests that classic and normal dysfluencies could have different etiologies and supports the suggestion by Cordes and Ingham (1995) that both types of dysfluency be included in studies into the cause and symptoms of stuttering.

Normal dysfluency might be more prevalent in free speech than reading because of the extra demands created by the need to generate language for speech, which are not present during reading. Alternatively, the cause might be the interaction that occurs with a listener (*e.g.*, eye contact) placing additional demands on the speaker. Reading, on the other hand, requires vision, visual perception, and intelligence for word recognition and word comprehension; qualities

not required for free speech. It may be that classic dysfluency, whatever the cause, is influenced both by the need to formulate language *and* the skills required for reading, and hence does not change from reading to speech because each task presents the speaker with a challenge, different but equal in consequence. A more likely explanation might be that classic dysfluencies are part of the make-up of the person's underlying (speech) motor control system and independent of skills required for language formulation and reading. Normal dysfluency, on the other hand, does appear to be related to, or at least be affected by, the need to formulate language and interaction with a listener, but not by skills required for reading.

Involuntary Movements and Dysfluencies

Subjects who stuttered had more IMs than controls during both speech and reading. All control subjects had more IMs during free speech than during reading. Conversely, this was not the case for the stuttering group, with five of the sixteen subjects having more IMs during reading than speech.

Closer examination of subjects who stuttered showed classic stuttering and its concomitant IMs were in the same proportion in both reading and free speech, suggesting that neither classic stuttering nor its concomitant IMs are influenced by the requirement to formulate language. In contrast, the proportion of normal dysfluencies accompanied by IMs was higher in free speech than in reading in both subject groups, suggesting that this type of dysfluency and its IMs may be caused by a mechanism different from that causing classic stuttering.

Involuntary Movements and Fluency

This study is the first to investigate IMs during fluent speech in persons who stutter and controls. IMs accompanied a small number of fluent words in both subject groups during reading and free speech. This suggests that IMs seen in those who stutter and to a lesser extent in normal subjects may be part of a more generalized motor control difficulty or dysfunction than one confined to the area concerned with the formulation and control of speech and language. The same

dysfunction might be responsible for both the stuttering and the abnormal movements.

Subjects who stuttered had a higher proportion of fluent words with IMs than controls during reading but not during free speech. Stress of reading under experimental conditions is said to increase dysfluency (see Andrews, 1983 for review). Of the 27 people recruited as potential experimental subjects for our study, two had so much difficulty reading the required passage that it was not possible to include their videotaped data for analysis. In contrast, none of the control subjects had any difficulty reading the passage. It is not possible to discount a difference in intelligence, language ability, or academic background between the two groups that may have led to the subjects who stuttered having a lesser ability to read than their fluent peers. If IMs are part of the same dysfunction as that causing stuttering, stress of reading in an experimental situation, as in this study, could therefore be an explanation for the higher proportion of fluent words with IMs during reading than during free speech in the experimental group.

This study is the first unequivocally to demonstrate an abnormally high presence of nonspeech related IMs during fluent speech in subjects who stutter. Kraaimaat and Janssen (1985) also reported accessory movements of the face accompanying fluent free speech in their group of 25 adults who stuttered. However, because they did not have a control group, they were unable to determine whether such movements also occur during fluent speech of fluent control subjects. Furthermore, they defined accessory movements as *any* observable movement of the orofacial structure that was not an integral part of the ongoing process of speech. This included eyelid movements, such as complete or partial closing of the eyes, and eye blinks defined as any fast closure of an eye or eyes. Over 50% of fluent syllables in their study were accompanied by eye blinks with eye blinks accounting for 90% of the accessory movements accompanying fluent syllables. In contrast, eyelid movement was noted considerably less (0.3%) during fluent speech. Our study regarded eye blinks as a normal phenomenon and distinguished between these and repetitive eye blinking, which was considered abnormal and included as IM. Therefore, although our study supports the finding of Kraaimaat and Janssen in the presence of abnormal or nonspeech related movements accompanying fluent

speech in subjects who stutter, the two studies differ markedly in definition of movement considered to be abnormal.

The Cause of Stuttering and Associated Involuntary Movements

Recent PET studies that support Caruso (1991) in his review on the theories of the cause of developmental stuttering have shown abnormal activation of the subcortical areas and SMA (in addition to cortical auditory and speech and language regions) (Braun *et al.*, 1997; Fox *et al.*, 1996; Wu *et al.*, 1997). These PET findings, allied to studies of the effect of antidopaminergic drugs on movement disorders and stuttering (Brady, 1991) and the finding that the movements accompanying stuttering resemble those seen in some movement disorders (Kiziltan and Akalin, 1996), suggest that both stuttering and the IMs seen in these people may be due to subcortical dysfunction or a more widespread abnormality of the dopaminergic system. This complex system which includes the cerebellum, the basal ganglia nuclei of caudate, lentiform and subthalamus, the claustrum and substantia nigra and the dopaminergic neurons of the ventral tegmental area is not well understood (Alexander and Crutcher, 1990; Brown and Marsden, 1998; Marsden and Obeso, 1994; McGeer and McGeer, 1987). It is proposed that the system includes several structurally and functionally distinct circuits that link cortex, basal ganglia and thalamus, with each circuit focused on a different part of the frontal lobe (Alexander and Crutcher, 1990) so that they serve or influence motor, oculomotor and cognitive functions (Brown and Marsden, 1998).

Our findings support the growing body of evidence for classic dysfluencies and associated IMs being a consequence of an organic abnormality in the brain more widespread than that associated with speech motor control only. The presence of IMs during both dysfluent and fluent speech indicates that IMs are caused by an abnormality that is concomitant with but different from the abnormality that causes classic dysfluencies. There is growing evidence from this and other studies that the abnormality responsible for IMs is *not* part of the primary language and speech production process.

On the other hand, normal dysfluencies may be 'triggered' by the need to formulate language, as the number of IMs accompanying

normal dysfluencies was found to be considerably higher during free speech than during reading. The difference in findings between the two types of dysfluencies and their accompanying IMs during reading and free speech emphasizes that the two types have different underlying mechanisms.

This and other studies indicate that there is a large range of IMs seen both in people who stutter and in controls. Mouth and jaw movements not identified as part of normal speech production and seen only in those who stutter could be part of the production of stuttered words. Other IMs seen more in those who stutter are jerks of the head and arms, grimaces of the mouth, eye blinks, and prolonged eye closure. The function of these movements is unclear. With the exception of Kiziltan and Akalin (1996), the origin of these movements has been explained in terms of being a conditioned or learned consequence of speech anxiety or a result of difficulty with correct performance of the lips and jaw for speech motor control (Conture and Kelly, 1991; Prins and Lohr, 1972). However, many of the studies have hinted that there may be other reasons or causes for the movements accompanying stuttering (Conture and Kelly, 1991; Janssen and Kraaimaat, 1986; Kraaimaat and Janssen, 1985; Prins and Lohr, 1972). A number of authors have suggested that people who stutter may not be a homogenous group, so that dysfluencies and their accompanying movements may manifest differently and even have different causes (Andrews *et al.*, 1983; Janssen and Kraaimaat, 1986; Kraaimaat and Janssen, 1985; Yiari and Ambrose, 1992b).

Our study indicates that IMs may have different causes, some being an epiphenomenon arising from a cerebral abnormality closely related to the abnormality causing classical stuttering, while others arise as a consequence of language formulation or interaction with a listener. Whatever their cause, the frequency of IMs and, to some extent, the types of IMs can distinguish between persons who stutter from those who are normally fluent.

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