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A positron emission tomography study of short- and long-term treatment effects on functional brain activation in adults who stutter

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Abstract

Previous studies have shown that fluency-inducing techniques, such as choral speech, result in changes in neural activation as measured by functional neuroimaging. In the present study, positron emission tomography was used to investigate the effects of intensive behavioural treatment, followed by a 1-year maintenance program, on the pattern of cortical and subcortical activation in stuttering adults during silent and oral reading of single words. The results indicate changes in activation lateralisation, as well as a general reduction in overactivation, especially in the motor cortex, following treatment. The results are discussed in light of previous functional imaging studies with stuttering adults.

Educational objectives: The reader will learn about and be able to describe the: (1) use of functional neuroimaging PET in the study of stuttering; (2) differences in neural activation

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between stuttering and non-stuttering adults; and (3) effects of behavioural fluency treatment on cortical and subcortical activations in stuttering speakers.

Keywords: Stuttering; Functional neuroimaging; Treatment effects

Recent investigations of stuttering in adults using techniques of functional neuroimaging consistently have revealed differences between neural activation patterns in stuttering and non-stuttering speakers. One recurrent observation in a number of these studies was the apparent overactivation of the motor system, which also have an important role in speech and language formulation. Fox et al. (1996) reported widespread right-hemisphere biased overactivation of cerebellar and cerebral primary motor and extraprimary cortex systems during a solo reading task in stuttering speakers when compared to non-stuttering controls. Overactivation of cerebellar and cerebral systems, including the medial anterior cingulate cortex as well as frontal and temporal areas in the left and right hemispheres, also has been reported by De Nil, Kroll, Kapur, and Houle (2000) and De Nil, Kroll, and Houle (2001) during single-word reading tasks. Braun et al. (1997) similarly reported an overactivation in anterior forebrain regions of stuttering adults while they performed a series of non-linguistic and linguistic tasks. In the latter study, anterior motor cortex activation in the stuttering subjects was either bilateral or right lateralised. As shown by Wu et al. (1995), observed differences in activity may not be limited to neural activation in the cortex, but may extend to subcortical regions, such as the substantia nigra, where they documented increased FDOPA uptake in stuttering subjects during oral paragraph reading.

In addition to the observed overactivation in motor systems, stuttering subjects also showed particular patterns of reduced activation, especially in the post-rolandic and superior temporal areas of the left cerebral hemisphere (Braun et al., 1997; Fox et al., 1996). In a recent overview of functional neuroimaging studies in stuttering, De Nil (in press) has shown that the emerging pattern of activation observed during a variety of speaking tasks in these studies points to a general overactivation of the sensorimotor and higher association cortices associated with language formulation and speech production in stuttering speakers. While this overactivation appears to exist bilaterally, a right-hemisphere bias can be detected.

Several of the aforementioned studies not only investigated neural activation under normal speech conditions, but also investigated the effects of fluency-enhancing conditions on the observed patterns of activation. Wu et al. (1995), using choral speech, observed a normalisation of the activation pattern in stuttering speakers, except for decreased glucose uptake in the left caudate and increased uptake in the substantia nigra. Fox et al. (1996) also exploited the well-known effect of choral reading as a means of increasing speech fluency in their stuttering subjects. They, too, observed a relative normalisation of the neural activation pattern when compared to that seen in the non-stuttering control subjects. Nevertheless, differential activation remained present in the supplementary motor area, the right primary

motor cortex and the cerebellum. Fox et al. (2000) correlated PET derived measures of blood flow with stuttering frequency and syllable rate during solo speech and choral reading. Stuttering frequency was correlated most strongly with right cerebral and left cerebellar activation. Syllable rate, in contrast, showed strong correlations bilaterally with a bias towards left cerebral and right cerebellar activation. Strong correlations were observed with brain regions involved in speech motor production (primary motor cortex, supplementary motor area, Broca's area, the anterior insula and the cerebellum). Interestingly, stuttering rate was negatively correlated with blood flow in the right superior and middle temporal gyrus. These authors interpreted the latter observation as suggesting the presence of an auditory processing problem in persons who stutter.

Braun et al. (1997) used pacing and automatic speech to induce greater fluency in the stuttering subjects. They also observed significant changes in the overall activation pattern in the fluent versus the dysfluent conditions. In stuttering speakers, fluency-enhanced speech was characterised by decreased activation in many cortical and subcortical areas including the medial and dorsolateral prefrontal cortices, superior frontal operculum, the left lateral and bilaterally medial orbital cortices, the midline cerebellum, and the anterior cingulate cortex. In contrast, some areas showed increased activation during increased fluency. These included post-rolandic areas, supramarginal gyrus, primary and higher order auditory cortices and the posterior insular cortex. While many of these changes occurred lateralised in the non-stuttering subjects, they tended to be bilateral in the stuttering speakers.

In the above-mentioned studies, changes in fluency were effected using fluency-enhancing techniques such as choral speech, pacing or automatic speech. These techniques are well known to result often in an increase in fluency, which, while often dramatic, also is temporary in nature. That is, the fluency-enhancing effect is linked to the immediate presence of the external stimulus, or the duration of the automatic speech sequence, and has little if any carry-over in time or space once the stimulus is removed. The functional imaging studies discussed so far also have not documented any long-term effects of the fluency-enhancing condition.

Behavioural fluency treatment also often results in increased fluency, although typically not as instantaneous as other fluency-enhancing conditions. Furthermore, effective treatment provides the client with internalised strategies or speech skills, which can be applied after formal treatment has finished, and can result in longer term or permanent changes in speech fluency. Studying the changes in neural activation that occur as a result of treatment is of significant theoretical and clinical importance for our understanding of stuttering. Theoretically, understanding neural activations that are influenced by treatment and are correlated with changes in the frequency of dysfluencies, for instance, may point toward neural activations that are either innate and/or hardwired versus activations that represent acquired compensatory mechanism. Clinically, understanding the effects of treatment on cerebral and cerebellar activations, and in particular, how these activations change in those stuttering individuals that are able to maintain fluency versus those that

are not, may help us to better understand the perplexing problem why a particular treatment approach is effective with some people but not or less so with others.

The current study was designed to investigate the short- and long-term effects of a behavioural fluency treatment on the cortical and subcortical activations seen in stuttering adults. Each stuttering speaker was scanned immediately prior to starting a 3-week intensive treatment program and again immediately upon completion of the intensive program. Each stuttering participant subsequently completed a structured 1-year maintenance program and a functional brain scan was obtained again upon completion of the maintenance phase. Specifically, the study was aimed at providing answers to the following questions: (1) does behavioural treatment, as specified for the purposes of the current study, result in changes in neural activation immediately upon completion of the treatment and/or 1 year later? (2) Are these changes similar to those reported in other studies which have investigated the effects of fluency-enhancing conditions? and (3) will fluency treatment and structured maintenance result in a normalisation of activation patterns for those clients who are able to maintain their fluency.

1. Subjects

Thirteen stuttering (mean age 27.4 years, range 20–40 years) and 10 non-stuttering (mean age 27.6 years, range 19–34 years) male adults participated in this study. All participants were native speakers of English and were screened for a negative history of relevant neurological or other medical problems and current drug use. The two groups were matched for educational level and for handedness, using the Edinburgh Handedness Inventory (Oldfield, 1971) with a cut-off laterality quotient of 90. Other than stuttering in the experimental group, none of the participants had a self-reported history of speech, language, or hearing problems. In addition, all the participants were informally screened for the presence of any speech or language problem by a qualified speech-language pathologist at the time of their initial fluency assessment.

The stuttering speakers were selected from the treatment waiting list at the Department of Speech Pathology at the Centre for Addiction and Mental Health: Clarke Division. Stuttering was diagnosed by a certified speech-language pathologist based on frequency of within-word dysfluencies (>3%) and/or the presence of significant speech-related struggle behaviour. Stuttering participants also had to qualify for the intensive behavioural treatment program (see below) and had to agree to participate in the complete program, including the 1-year maintenance component. Stuttering participant characteristics, as well as percent intra-word dysfluencies during spontaneous speech (based on a conversational speech sample of minimum 200 words), reading (based on the Amplifier Passage, a 286 word long standardised text) and the Stuttering Severity Index (SSI) score (Riley, 1994), are summarised in Table 1. Inter- and intra-rater differences for percent dysfluent word calculations, based on a random sample of three stuttering speakers,

Table 1
Age, percentage dysfluencies for reading and conversational speech, and the Stuttering Severity Score (Riley, 1994) for stuttering subjects ($n = 13$) prior to a 3-week intensive stuttering reduction therapy (pre-Tx), immediately after the therapy (post-Tx) and 1-year later (1 year)

Subject no.	Age	% Dysfluencies (reading task)			% Dysfluencies (job task)			Stuttering Severity Index Score (total)		
		Pre	Post	1 year	Pre	Post	1 year	Pre	Post	1 year
1	23	6	2	2	11	2	8	20	9	16
2	20	5	0	2	8	2	3	18	7	10
3	40	2	2	1	8	3	5	16	11	12
4	25	1	0	0	4	0	2	11	7	8
5	23	18	0	0	8	0	7	27	9	15
6	27	1	0	1	4	0	1	12	6	5
7	35	1	1	0	5	2	0	11	8	7
8	33	2	1	1	5	0	2	17	6	10
9	29	0	1	0	3	0	0	9	7	5
10	34	1	0	1	1	1	0	9	9	9
11	22	5	2	3	3	1	6	9	10	12
12	25	23	1	1	18	10	4	23	12	8
13	21	14	0	2	14	0	7	18	5	17
Mean	27.4	6.07	0.76	1.07	7.07	1.61	3.46	15.3	8.15	10.3

Table 2
Mean scores on the Perceptions of Stuttering Inventory (PSI) by the stuttering subjects, and the State-Trait Anxiety Inventory (STAI) by the stuttering and non-stuttering subjects

Subjects	% Mean PSI (range %)	Mean STAI (State)	Mean STAI (Trait)
Pre-treatment ($n = 13$)	48 (25–73)	34.8 (22–53)	43.3 (26–68)
Post-treatment ($n = 13$)	18 (3–43)	29.5 (23–47)	37.7 (25–61)
1 year later ($n = 13$)	21 (5–40)	31.7 (23–45)	36.9 (24–57)
Controls ($n = 10$)	Not applicable	31.7 (20–45)	37.0 (24–45)

ranged from 0 to 2%. In addition to the SSI, stuttering subjects also completed the Perceptions of Stuttering Inventory (PSI), a self-assessment of struggle, avoidance and expectancy behaviours (Woolf, 1967). All participants completed the State-Trait Anxiety Inventory (Spielberger, 1983), which provides measures of a person's situation specific (State) and general (Trait) anxiety. The data from these questionnaires are summarised in Table 2.

2. Treatment program

The treatment program which formed an integral part of the current study consisted of a modified Precision Fluency Shaping Program (Webster, 1974) implemented at the time of the study at the Centre for Addiction and Mental Health: Clarke Division under the direction of Dr. Kroll. The program consisted of an intensive 3-week module during which the participants received a combination of

group and individual fluency treatment for 6 h each day followed by daily activities to be completed by each participant. During the first week, emphasis was placed on the acquisition of a series of speech targets which included, among others, slowed speech, soft articulatory contacts, gentle speech onsets, proper breath support and blending. The second week primarily consisted of advanced practice and shaping of these targets towards more natural speech. During the third week, participants practiced their new fluent speech skills in everyday environments outside the clinic. In addition to the practice of these speech motor skills, the treatment program emphasised self-observation and included systematic cognitive and attitudinal intervention.

Following the 3-week intensive component of the treatment, the stuttering participants continued in a year-long maintenance program during which they returned to the clinic for 1-h treatment sessions every week for the first month, every other week for the second month, and monthly sessions for the remainder of the year. During these maintenance sessions, participants continued working on the effective use of their speech skills in daily life. Simultaneously, participants completed a systematic program of cognitive intervention (Kroll, 1986) with assistance from the clinician. All stuttering participants in this study finished the complete intervention program.

3. Baseline and language tasks

Participants were asked to perform three tasks during the experiment: a non-linguistic baseline task and two language tasks: silent and oral reading.[1] Words were selected from the MRC Psycholinguistic Database (http://www.psy.uwa.edu.au/mrcdatabase/uwa_mrc.htm). All language stimuli were single or two syllable words with a written frequency range of $\geq$80. In addition to the two reading tasks, the participants also completed a baseline task. This baseline task was always the first and last task in the experimental session. The order of the language tasks was counterbalanced across participants. The stimuli for the baseline and reading tasks were presented on a computer monitor positioned at a comfortable reading level for each participant. At the beginning of the scanning session, participants were given a short practice for each task using 10 stimuli similar to the ones used during the actual experiment.

3.1. Baseline task

During the baseline task, participants viewed individual strings of Xs. The strings varied randomly in length, similar to that of the words used in the silent and oral reading tasks. The baseline task was selected to allow measurement of

[1] In addition to the two reading tasks, subjects also completed a verb generation task. The data for the verb generation are not included in this report and will be presented in greater detail in an upcoming paper.

brain activation resulting from visual stimulation without associated phonological or semantic processes. A total of 25 individual character strings were presented for 2 s each, with an inter-stimulus interval randomly ranging from 1.5 to 2.0 s. Presentations of individual character strings were separated by a single X located in the middle of the monitor screen.

3.2. Silent reading task

In the silent reading task, 25 words were presented to the participants with the instruction to silently read each of the words while avoiding any articulatory movements. During the post- and follow-up scans (see below) subjects were instructed to mentally use their speech skills as they would during normal conversation. Similar to the baseline task, words were presented individually for 2 s each, with an inter-stimulus interval ranging randomly from 1.5 to 2.0 s for a total duration of approximately 85 s per word list.

3.3. Oral reading task

During the oral reading task, a list of 25 words, equivalent to those used for silent reading, was presented to the participants in a manner similar to the silent reading task. Participants were instructed to read the words out loud at a normal loudness level. During the post- and follow-up experimental sessions, participants again were instructed to use their speech skills as they would during normal conversation. To avoid the introduction of time pressure during reading, especially for the stuttering speakers, all participants were instructed to complete each word prior to attempting to say the next one. If the production of a word overlapped with the presentation of the next one, participants were instructed to ignore the new word, finish saying the previous word, and wait for the presentation of the next word. Each participant's oral production was recorded on audiotape using a lapel microphone positioned approximately 20 cm from the mouth.

4. Positron emission tomography scanning

Positron emission tomography scans were obtained using a GEMS-Scanditronix PC 204815B head scanner at the PET Centre of the Centre for Addiction and Mental Health: Clarke Division. Scans were obtained at three time intervals: immediately preceding the start of the 3-week intensive treatment component (pre-treatment $\leq$1 week), immediately following the termination of the intensive component and prior to the start of the maintenance program (post-treatment $\leq$2 weeks), and finally at the 1-year interval following the conclusion of the maintenance program (follow-up 51–58 weeks). Participants were scanned in a supine position with a custom-fitted thermoplastic mask for head stabilisation, which does not interfere with speech movements. Emission scans were done using 1110 MBq (30 mCi) of $[^{15}O]H_2O$ injected as a bolus into a forearm vein at the beginning of each task. Scans were

acquired for 60 s, with individual scans separated by an 11-min interval. Word stimulus presentation for each of the control or reading tasks was started at the time of the injection, and 60-s scans were acquired during each task starting approximately 20 s following injection. Each control or reading task ended within 10 s after completion of the l-min scan. PET scans were reconstructed using a Hanning filter and attenuation correction. To avoid arterial blood sampling during the scanning to determine absolute blood flow, which would make the experiment more invasive, integrated regional counts were used as an index of regional cerebral blood flow.

5. Data analysis

PET scans were analysed using Statistical Parametric Mapping (SPM 99; Wellcome Department of Cognitive Neurology, 1999). Between- and within-subject variations in global blood flow across the scans were removed using analysis of covariance with global activity as covariate on a pixel-by-pixel basis (Friston, Frith, Liddle, & Frackowiak, 1991). Statistical Parametric Mapping analysis involved the following steps: (1) three-dimensional stereotactic reorientation of the images along the Anterior Commissure-Posterior Commissure (AC/PC) line, (2) plastic transformation of these images using a non-linear resampling technique to minimise for anatomical variance across participants, and (3) spatial filtering to enhance the signal in the presence of the noise introduced by anatomical and functional heterogeneity across participants (Frackowiak, Friston, Frith, Dolan, & Mazziotta, 1997). All data were smoothed using a 10 mm filter in the *x*, *y*, and *z* directions. The scans of different tasks were compared with each other on a pixel-by-pixel basis. The statistical significance of each difference was assessed by comparing the magnitude of the difference at a pixel with the error variance at that particular pixel. The resulting map of the *Z* statistic at each pixel constitutes the SPM (Friston et al., 1991). Type I errors were minimised by evaluating only those pixels where the *Z* statistic exceeds a threshold that had been adjusted for the number of resolvable elements in the image. This is effectively the same as controlling for multiple comparisons, but uses the inherent spatial resolution of the processed PET images to make the correction (Frackowiak et al., 1997). For the within-group comparison, pixels were considered statistically significant if they reached a minimum *Z* value of 3.09 (Friston et al., 1991). Regions of activation were referenced relative to Talaraich and Tournoux brain coordinates (Talairach & Tournoux, 1988).

6. Results

6.1. Behavioural results

The results are summarised in Table 1. Mean percentage disfluency during reading for the stuttering subjects decreased from 6.07% pre-treatment to 0.76%

immediately post-treatment. Percentage dysfluent speech during monologue decreased from 7.07% pre-treatment to 1.61% post-treatment. This decrease was statistically significant for both measures (reading: $q = 3.90$; monologue: $q = 5.43$; $P < 0.05$). For reading as well as monologue, there was an increase in disfluency at the 1-year follow-up to 1.07 and 3.46%, respectively, but this increase was not statistically significant (reading: $q = 0.94$; monologue: $q = 1.83$; $P > 0.05$). This pattern of fluency change also was confirmed on the SSI score, which yielded a statistically significant decrease from pre- to post-treatment ($q = 5.07$; $P < 0.05$), while the increase from post-treatment to 1-year follow-up did not reach significance ($q = 1.86$; $P > 0.05$).

Fluency on the single-word oral reading tasks during the PET scans was high for all subjects.[2] During the pre-treatment scans, only two subjects showed dysfluencies (6/20 and 2/20, respectively). No dysfluencies were observed at the post-treatment or the 1-year follow-up scans for any of the subjects.

Table 2 shows the results for the PSI and the STAI. On the PSI, stuttering subjects showed a significant decrease pre- to post-treatment from 48 to 18%, with a slight increase to 21% at the 1-year follow-up. In contrast, no changes were observed in the scores on the STAI for both stuttering and non-stuttering subjects. Pre-treatment, the STAI scores for the stuttering subjects were 34.8 and 43.3 for State and Trait, respectively. The non-stuttering subjects scored 31.7 and 37.0, respectively, for each of these two measures. Post-treatment, both scores for the stuttering subjects decreased somewhat to 29.5 and 37.7, and stayed essentially the same at the 1-year follow-up (31.7 and 36.9 for State and Trait, respectively). None of these changes were statistically significant at $P < 0.05$.

6.2. Neuroimaging results

6.2.1. Silent reading

The cortical and subcortical activation observed during silent reading was compared against the activation observed during the non-linguistic baseline task, effectively subtracting the neural activation that was related solely to the processing of visual characters in the baseline and silent reading task. Results from this analysis are shown in Fig. 1. The three-dimensional localisations of peak activations and the size of these activations are listed in Table 3.

For the non-stuttering participants, the analysis revealed that neural activation during silent reading was primarily left hemisphere localised in the anterior cingulate gyrus, the inferior and middle frontal gyrus, and the middle temporal gyrus. These areas are typically involved in attentional, motor and phonological/semantic processing of language stimuli. The activation observed during the pre-treatment scan in the stuttering subjects was much elevated compared to the non-stuttering participants. Activation was observed in the inferior frontal gyrus, precentral gyrus, and cerebellum, areas known to be involved in motor control processes in speech.

[2] Audio recordings for three subjects were unavailable due to technical difficulties during the scans.

Table 3
Three-dimensional (*x*, *y*, *z*) peak activation coordinates, and associated *Z* scores, for silent reading minus baseline (voxel cluster size >5) for the non-stuttering subjects and the stuttering subjects pre- and post-treatment and at 1-year follow-up

Region	Laterality	Coordinate			*Z* score
		x	*y*	*z*	
Control subjects					
Frontal lobe	R	32	37	2	3.92
Cingulate gyrus	L	−4	30	26	3.84
Middle frontal gyrus	L	−34	37	−4	3.63
Middle temporal gyrus	L	−50	−36	−12	3.50
Inferior frontal gyrus	R	26	13	−14	3.31
Inferior frontal gyrus	L	−38	29	−10	3.30
Stuttering subjects (pre-Tx)					
Inferior frontal gyrus	L	−53	29	−3	4.49
Middle frontal gyrus	R	36	45	11	4.40
Anterior cingulate	L	−8	38	22	4.23
Cuneus	R	8	−82	30	4.17
Precentral gyrus	R	36	9	35	4.02
Middle/inferior frontal gyrus	R	46	24	21	3.95
Inferior frontal gyrus	R	51	29	−8	3.79
Cerebellum	R	46	−62	−30	3.71
Lentiform nucleus	R	16	15	−7	3.59
Superior temporal gyrus	R	51	−10	2	3.55
Anterior cingulate	R	2	51	−1	3.42
Frontal lobe	R	42	23	−15	3.24
Stuttering subjects (post-Tx)					
Precentral gyrus	L	−59	10	11	4.84
Inferior frontal gyrus	L	−44	37	7	4.05
Superior temporal gyrus	L	−40	−44	17	3.77
Claustrum	L	−24	18	10	3.62
Parahippocampal gyrus	R	22	−42	6	3.55
Lentiform nucleus	R	22	−2	−8	3.52
Precentral gyrus	R	65	−10	37	3.41
Precentral gyrus	R	59	−7	11	3.23
Parahippocampal gyrus	R	18	−16	−11	3.14
Stuttering subjects (1 year)					
Insula	L	−28	−30	18	4.45
Temporal lobe	L	−36	−16	−13	4.36
Precentral gyrus	L	−61	−7	11	4.04
Middle temporal gyrus	R	51	−33	3	4.03
Frontal lobe	R	38	39	5	3.93
Middle frontal gyrus	R	50	40	20	3.89
Lentiform nucleus (globus pallidus)	R	24	−8	2	3.88
Superior temporal gyrus	L	51	6	−2	3.70
Middle frontal gyrus	L	−42	45	9	3.58
Superior frontal gyrus	R	22	56	−4	3.35
Precentral gyrus	L	−44	−22	34	3.30
Insula	L	−42	−15	19	3.28
Supramarginal gyrus	L	−59	−41	35	3.26

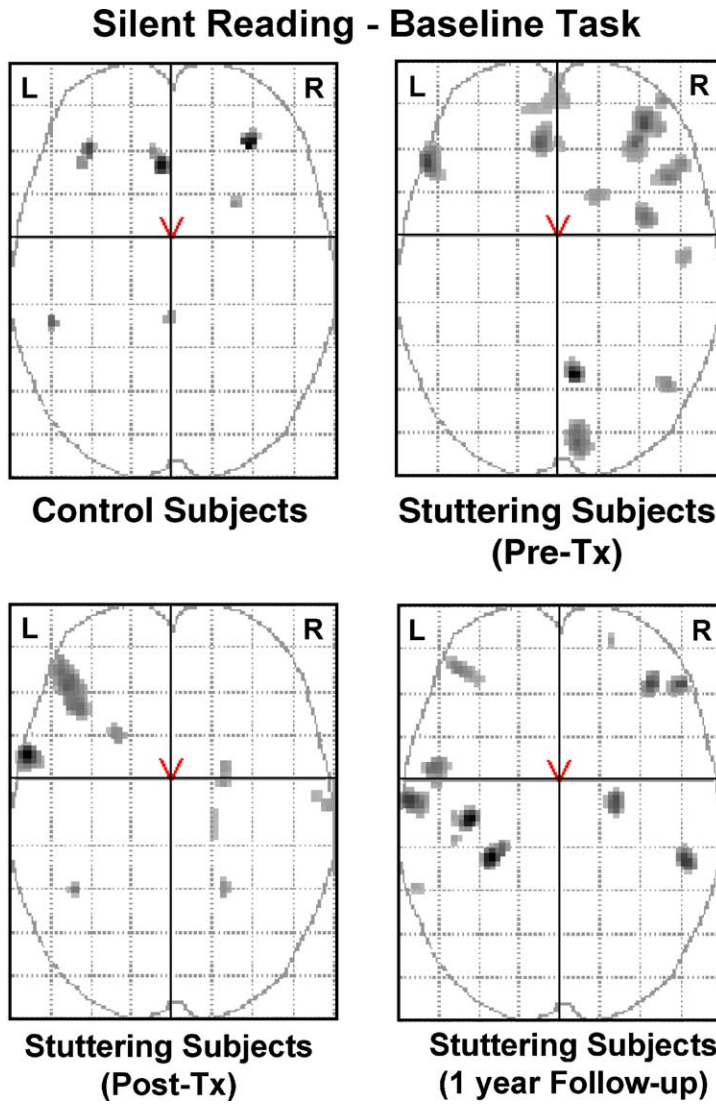

Fig. 1. Axially projected Statistical Parametric Maps (SPM) for silent reading minus baseline in non-stuttering ($n = 10$) and stuttering subjects ($n = 13$) prior to a 3-week intensive stuttering reduction therapy (pre-Tx), immediately following the therapy (post-Tx) and 1 year later (1 year).

With the exception of the inferior frontal gyrus, which showed bilateral activation, activation in these regions was right lateralised. In addition, significant activation was observed in the anterior cingulate (left $>$ right), the right superior temporal gyrus, and right cuneus. Activation in these latter regions may be related to attentional processes, auditory processing and visual imagery, respectively.

At the post-treatment scan, the overall level of activation in the stuttering subjects was decreased compared to the pre-treatment scan. Areas of activation were increasingly left localised in the inferior frontal gyrus and the precentral gyrus. The latter area showed some bilateral activation although this too was substantially left biased. In addition, activation was observed in the left superior temporal gyrus, typically involved in auditory processing, and the right parahippocampal gyrus, an area often activated in memory tasks.

For the stuttering participants at the 1-year follow-up, neural activation was observed primarily in regions of the brain involved in motor control of speech, including the precentral gyrus, globus pallidus, middle frontal gyrus and insula. In addition, activation was observed in the superior and middle temporal gyrus. Most of the activation was left lateralised with the exception of the middle temporal gyrus which was right lateralised and the middle frontal gyrus which showed bilateral activation.

6.2.2. Oral reading

For oral reading, analogous to silent reading, baseline activation was subtracted out from the activation observed during oral reading. In addition, a second analysis was done by subtracting silent reading from oral reading activation. This double comparison allowed us to obtain a more complete picture of the activation patterns observed across the various scans. Figs. 2 and 3 show the three-dimensional localisations of peak activations for oral reading minus baseline, and oral reading minus silent reading, respectively. The three-dimensional localisations of peak activations and size of activated areas for these two subtractions are shown in Tables 4 and 5, respectively.

6.2.3. Oral reading minus baseline

For the non-stuttering participants, oral reading minus baseline activation was located primarily in regions directly involved in motor execution of the oral reading task. In the left hemisphere, activation was observed in the post-central gyrus, the temporal lobe and the cerebellum. Areas activated in the right hemisphere included the superior temporal gyrus, the cerebellum and subcortically, the subthalamic nucleus.

In the stuttering participants, pre-treatment, oral reading was characterised by bilateral activations in the pre- and post-central gyrus (left > right), superior temporal gyrus (left > right), insula (left > right) and cerebellum (right > left). In the right-hemisphere, activations occurred in the putamen and in the medial frontal gyrus and anterior cingulate. Post-treatment, the stuttering participants again showed widespread activation throughout the brain. The largest activation was observed bilaterally in the precentral gyrus (left > right), the cerebellum (right > left), the insula (right > left) and the left parietal lobule. In addition, activation was observed in the right superior temporal gyrus, and a number of subcortical regions such as the thalamus and the area of the red nucleus. At the 1-year follow-up, activation was observed almost exclusively in motor execution

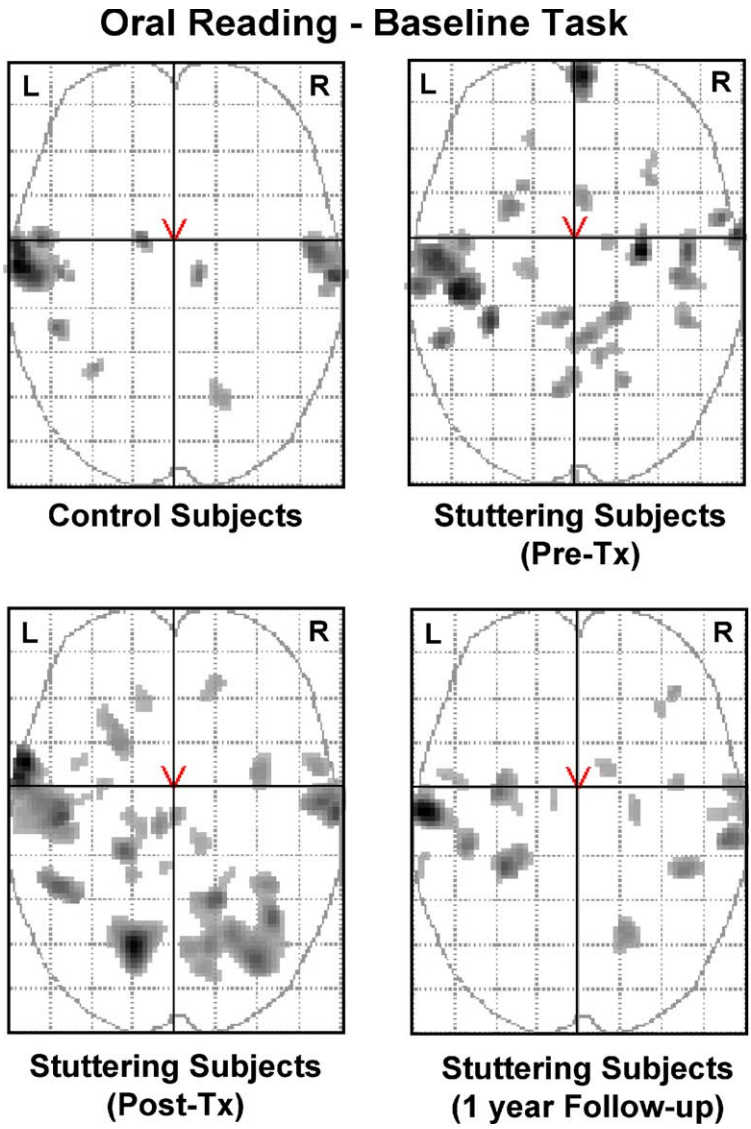

Fig. 2. Axially projected Statistical Parametric Maps (SPM) for oral reading minus baseline in non-stuttering ($n = 10$) and stuttering subjects ($n = 13$) prior to a 3-week intensive stuttering reduction therapy (pre-Tx), immediately following the therapy (post-Tx) and 1 year later (1 year).

areas bilaterally. These included pre-central (right > left) and post-central gyrus (left > right), insula (left > right) and right cerebellum. Additional activation could be observed in the superior temporal gyrus (right > left) and the left cingulate gyrus.

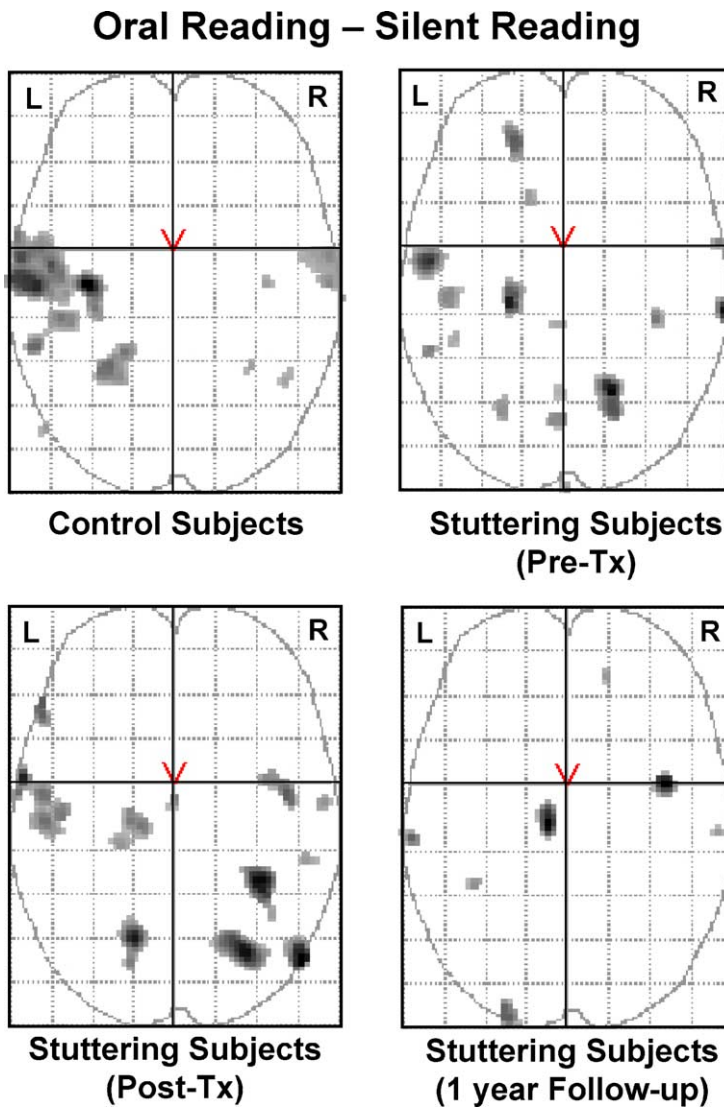

Fig. 3. Axially projected Statistical Parametric Maps (SPM) for oral reading minus silent reading in non-stuttering ($n = 10$) and stuttering subjects ($n = 13$) prior to a 3-week intensive stuttering reduction therapy (pre-Tx), immediately following the therapy (post-Tx) and 1 year later (1 year).

6.2.4. Oral minus silent reading

In the non-stuttering subjects, oral reading showed greater activation than silent reading in the left insula, bilateral superior temporal gyrus, and left cerebellum. Pre-treatment, in the stuttering participants, bilateral activation was observed in

Table 4
Three-dimensional (*x*, *y*, *z*) peak activation coordinates, and associated *Z* scores, for oral reading minus baseline (voxel cluster size >5) for the non-stuttering subjects and the stuttering subjects pre- and post-treatment and at 1-year follow-up

Region	Laterality	Coordinate			*Z* score
		x	*y*	*z*	
Control subjects					
Post-central gyrus	L	−63	−9	17	4.84
Superior temporal gyrus	R	65	−15	6	4.36
Globus pallidus	L	−12	0	−8	3.99
Temporal lobe	L	−48	−36	−12	3.94
Subthalamic nucleus	R	10	−16	−4	3.86
Cerebellum	L	−32	−52	−31	3.61
Cerebellum	R	18	−63	−19	3.46
Temporal lobe	L	−50	−8	−10	3.30
Stuttering subjects (pre-Tx)					
Putamen	R	28	−5	11	4.68
Medial frontal gyrus	R	4	64	−10	4.56
Post-central gyrus	L	−48	−20	32	4.53
Transverse temporal gyrus	L	−34	−32	13	4.25
Precentral gyrus	L	−55	−7	13	4.24
Precentral gyrus	R	65	3	16	4.18
Insula	R	46	−5	17	4.02
Superior temporal gyrus	L	−53	−40	13	3.94
Superior temporal gyrus	R	59	7	−5	3.89
Cerebellum	R	16	−32	−20	3.79
Precentral gyrus	R	42	−14	34	3.73
Claustrum	L	−26	12	12	3.69
Interhemispheric	L	−4	−31	5	3.67
Cerebellum	L	−6	−61	−9	3.63
Insula	R	46	−32	20	3.56
Anterior cingulate	R	4	17	25	3.52
Cerebellum	R	20	−57	−22	3.52
Cerebellum	R	10	−48	−25	3.50
Frontal lobe	L	−22	19	−9	3.49
Frontal lobe	R	30	24	23	3.41
Lentiform nucleus	L	−18	−13	3	3.33
Lentiform nucleus	R	12	−4	−7	3.27
Medial frontal gyrus	L	−18	40	24	3.22
Stuttering subjects (post-Tx)					
Precentral gyrus	L	−59	10	11	5.75
Cerebellum	L	−16	−63	−15	5.64
Precentral gyrus	R	64	−8	35	4.89
Cerebellum	R	34	−69	−18	4.67
Inferior parietal lobule	L	−46	−40	22	4.60
Thalamus	L	−20	−24	16	4.59
Red nucleus	L	−6	−16	−3	3.99
Claustrum	L	−22	16	10	3.93
Insula	R	36	6	7	3.88
Pons	L	0	−13	−26	3.81

Table 4 (*Continued*)

Region	Laterality	Coordinate			*Z* score
		x	*y*	*z*	
Superior temporal gyrus	R	57	7	−7	3.80
Pulvinar	L	−18	−35	9	3.53
Thalamus	R	12	−21	16	3.45
Insula	L	−30	−1	17	3.34
Pons	R	20	−40	−27	3.31
Orbital gyrus	L	−10	32	−28	3.30
Pons	L	−4	−32	−20	3.26
Insula	L	−38	10	12	3.20
Stuttering subjects (1 year)					
Post-central gyrus	L	−61	−9	15	5.27
Insula	L	−30	−30	18	4.39
Precentral gyrus	R	61	−19	3	4.26
Superior temporal gyrus	R	61	−19	2	4.19
Insula	L	−32	−1	11	4.12
Post-central gyrus	L	−46	−22	32	4.01
Superior temporal gyrus	R	44	−32	11	3.90
Cerebellum	R	20	−59	−22	3.78
Precentral gyrus	R	63	1	13	3.65
Frontal lobe	R	40	39	7	3.58
Insula	R	40	2	11	3.47
Putamen	R	24	−6	4	3.44
Superior temporal gyrus	L	−53	4	−2	3.35
Frontal lobe	R	32	32	13	3.31
Cingulate gyrus	L	−10	−4	33	3.28
Superior temporal gyrus	L	−63	−29	11	3.27

the precentral gyrus (left > right), the cerebellum (right > left) and the superior temporal gyrus (right > left). In addition, left hemisphere unilateral activation was seen in the superior frontal gyrus, the post-central gyrus, and inferior parietal lobe. Unilateral right activation was observed in the insula and the occipital lobe. Post-treatment, the stuttering subjects showed significantly increased activation in the cerebellum (right > left), the precentral gyrus (left > right) and the superior temporal gyrus (left > right). Activation also was seen in the inferior frontal gyrus and the insula, both in the left hemisphere. After 1 year, the overall level of activation was greatly reduced in the stuttering adults, with activation localised in the right insula, the superior temporal gyrus (left > right), the left middle occipital gyrus and parietal lobe, and subcortically in the thalamus.

7. Discussion

The aim of the current study was three-fold. First, we wanted to investigate whether behavioural treatment resulted in immediate and long-term changes in

Table 5
Three-dimensional peak activation coordinates, and associated *Z* scores, for oral reading minus silent reading (cluster size >5) for the non-stuttering subjects and the stuttering subjects pre- and post-treatment and at 1-year follow-up

Region	Laterality	Coordinate			*Z* Score
		x	*y*	*z*	
Control subjects					
Insula	L	−36	−13	8	4.93
Superior temporal gyrus	L	−55	−39	6	4.00
Cerebellum	L	−26	−48	−30	3.96
Superior temporal gyrus	R	63	−13	6	3.70
Middle occipital gyrus	L	−51	−72	5	3.41
Cerebellum	R	−32	−48	−25	3.39
Insula	R	38	−13	19	3.39
Temporal lobe	R	46	−52	−1	3.38
Stuttering subjects (pre-Tx)					
Superior temporal gyrus	R	67	−25	12	4.58
Cerebellum	R	20	−59	−22	4.48
Thalamus	L	−22	−21	12	4.36
Precentral gyrus	L	−53	−5	11	4.21
Superior frontal gyrus	L	−20	44	25	3.90
Insula	R	38	−28	14	3.65
Superior temporal gyrus	L	−53	−40	9	3.59
Precentral gyrus	R	63	3	27	3.56
Post-central gyrus	L	−48	−18	30	3.54
Posterior cingulate	–	0	−67	16	3.53
Cerebellum	L	−26	−67	−19	3.47
Occipital lobe	R	2	−97	−5	3.43
Caudate	L	−14	20	3	3.35
Inferior parietal lobe	L	−44	−36	24	3.27
Cerebellum	L	−2	−59	−19	3.17
Stuttering subjects (post-Tx)					
Cerebellum	R	51	−73	−25	4.43
Cerebellum	R	36	−40	−28	4.34
Cerebellum	R	30	−69	−29	4.29
Cerebellum	L	−16	−63	−15	4.24
Precentral gyrus	L	−61	3	27	4.21
Inferior frontal gyrus	L	−53	31	8	3.82
Superior temporal gyrus	L	−63	−4	−1	3.67
Thalamus	L	−14	−17	10	3.60
Insula	L	−46	−13	15	3.58
Precentral gyrus	R	61	−7	24	3.39
Superior temporal gyrus	R	53	−30	13	3.33
Stuttering subjects (1 year)					
Insula	R	40	1	13	4.22
Thalamus	L	−8	−15	10	4.19
Middle occipital gyrus	L	−26	−93	6	3.66
Superior temporal gyrus	L	−63	−21	1	3.64
Parietal lobe	L	−38	−40	22	3.36
Orbital gyrus	R	16	42	−24	3.31
Superior temporal gyrus	R	61	−19	5	3.28

patterns of neural activation in adults who stutter. Secondly, we wanted to analyse to what extent these changes are similar to those reported in other studies which have used fluency-enhancing conditions. Lastly, we wanted to investigate the extent to which fluency treatment resulted in a partial or complete normalisation of cortical and subcortical activation patterns in stuttering adults.

The behavioural fluency treatment used in the current study clearly resulted in changes in the brain activations observed in the stuttering subjects. Silent reading in the non-stuttering subjects was characterised primarily by left hemisphere activation in speech- and language-related areas in the frontal and temporal cortex. No activation of premotor or motor cortex was observed in these subjects, which is congruent with the nature of the silent reading task and the fact that the subjects were instructed to avoid any overt articulatory movements. Three main observations characterised the activations observed in the stuttering subjects pre-treatment. First of all, the overall level of activation in these subjects was significantly elevated compared to that of the non-stuttering speakers. This observation is consistent with our hypothesis that stuttering subjects tend to recruit more neural resources for accomplishing even relatively simple speech-related tasks (De Nil, in press, 1999; De Nil et al., 2000). A second observation emerging from our pre-treatment data is that stuttering subjects show activation in motor cortex and cerebellum even during a silent reading task. This latter observation is consistent with our previous suggestion that stuttering subjects tend to approach even silent reading tasks with a strong emphasis on the articulatory processes rather than, or in addition to, the cognitive linguistic processing of the written stimuli (De Nil et al., 2000, 2001). A third observation relates to the fact that the activation in the stuttering subjects is particularly right lateralised, with the exception of the inferior frontal gyrus (Broca's area). The right biased activation in the stuttering subjects pre-treatment is congruent with early electrophysiological observations by Moore (1984) and Boberg, Yeudall, Schopflocher, and Bo Lassen (1983).

Following treatment, activation in the stuttering subjects shifted towards a more left-lateralised activation pattern. The bilateral activation in the inferior frontal cortex was replaced by a unilateral left activation. Activation in the precentral gyrus, which was right lateralised pre-treatment, became bilateral (left $>$ right) immediately post-treatment and unilateral left lateralised after 1 year. The shift to more left-lateralised activation immediately following treatment may reflect the strong behavioural approach used in treatment during which the participants were trained intensively to monitor their speech movements and use the learned speech skills to sequence their articulatory movements into fluent speech. The monitoring, sequencing and timing of sequential movements is a task well-suited to the particular strengths of the left hemisphere (Bradshaw & Nettleton, 1981). Some increased bilateral or right-hemisphere activation could be observed at the 1-year follow-up scan, primarily in the middle frontal and temporal gyri and the insula, which plays an important role in the control and execution of articulatory movements (Dronkers, 1996).

During oral reading, the non-stuttering subjects showed activation primarily in cortical and subcortical sensorimotor areas. This was the case regardless of whether the subtraction was done using the silent reading or the non-linguistic baseline task. In both cases, sensorimotor cortical activations, such as in the insula and post-central gyrus, were lateralised primarily to the left, as would be expected in our right-handed subjects. Further activation was observed primarily in the cerebellum, which also is involved in motor control, and the superior temporal gyrus. The latter activation most likely was related to the auditory processing of the overt speech as it was not observed in the silent reading task. Again, as with silent reading, the stuttering subjects showed significantly more widespread activation compared to the non-stuttering subjects. This overall overactivation showed a gradual reduction from pre- to post-treatment to follow-up. Similar to the non-stuttering subjects, this overactivation in the stuttering speakers involved primarily the motor cortex, both in frontal and post-central cortex and cerebellum, and this overactivation was present whether silent reading was subtracted or not. While the motor activation in the non-stuttering speakers was primarily left-lateralised, activation in the stuttering subjects was clearly bilateral with a bias toward either left or right-hemisphere activation. Interestingly, this lateralised bias for some areas shifted as subjects first completed intensive therapy and subsequently the maintenance phase. Activation in the insula, which showed a strong tendency to be right lateralised pre-treatment, shifted to stronger left-lateralised activation post-treatment and at follow-up. Cerebellum activation, in turn, became increasingly more right lateralised following treatment congruent with its crossed connections to the motor cortex. These shifts in activation can be understood within the behavioural paradigm used in the treatment program, during which the stuttering subjects are taught to exert increased control over the execution and sequencing of their articulatory movements. As discussed before, such voluntary control over the sequential coordination of speech movements fits with the strengths of the left hemisphere in such time-dependent processes (Bradshaw & Nettleton, 1981). The emphasis on voluntary control of speech movements and, consequently, the greater need for sensory monitoring, also may explain the increased activation in the post-central gyrus observed in the stuttering subjects.

Similar to Fox et al. (1996), we observed significant activation in the right but not left superior temporal gyrus pre-treatment in our stuttering subjects. When activation during silent reading is subtracted from oral reading, a clear shift can be seen in the superior temporal gyrus activation from right lateralised pre-treatment to left-lateralised post-treatment and at the 1-year follow-up. The absence of activation in the left superior temporal gyrus in stuttering adults has been interpreted as suggesting a deficiency in the verbal fluency system in stuttering speakers (Fox et al., 1996, 2000). However, an absence of activation in this region during speech has been observed in non-stuttering speakers in other studies (Hirano et al., 1996). Interestingly, when the normal speech patterns was altered in their subjects, activation in the STG became stronger, suggesting that activation in this area may be absent or minimal during normal speech, but increases as the need for speech

monitoring grows (Hirano et al., 1997; Houde, Nagarajan, Sekihara, & Merzenich, 2002). The increased activation in the left STG observed in our study, thus, may not necessarily reflect a "shift" in activation from right to left hemisphere, but may have resulted from an increased activation in left STG activation when stuttering subjects are actively controlling their speech for effective use of the acquired speech targets.

Important differences were found between the changes observed in our treatment study and those observed in other studies using various other fluency-enhancing techniques (Braun et al., 1997; Fox et al., 1996). In our study, fluent speech post-treatment was associated with increased activation in cerebellar and cortical sensorimotor regions in frontal and temporal cortex, especially in the left hemisphere. The reduction in overactivation reported by Fox et al. (1996) when speech became fluent also was seen in the overt speech in our subjects, but only after the 1-year follow-up. Differences in activation patterns between this and other studies are not unexpected given the differences in how increased fluent speech was accomplished in the stuttering speakers. The question remains of course whether activation changes observed following behavioural treatment are more stable than those observed with fluency-enhancing techniques. In addition, it remains to be seen if these changes are directly related to increased fluency or rather reflect variations in motor planning and execution regardless of the level of fluency. This is true particularly given the high level of fluency of our subjects on the single-word reading task, even pre-treatment. While the SSI and percent disfluency scores indicate a significant increase in fluency from pre- to post-treatment and at 1-year follow-up, the same change in fluency was not reflected in the subjects' single-word fluency scores during the scans. In the present study, this was not a crucial issue given that the research questions were focused on the influence of behavioural fluency treatment, rather than on speech fluency, on brain activation. The influence of the behavioural treatment on fluency was reflected in the significant changes observed on the reading and monologue tasks. However, the high level of fluency during the scans did prevent us from correlating levels of fluency with our measures of neural activation (see Fox et al., 2000 for such a correlation). Nevertheless, activation in our subjects showed a tendency to become more left lateralised immediately post-treatment, with some reappearance of increased right activation at the 1-year scan. This observation, together with observation that subjects tended to show somewhat higher levels of dysfluency at the 1-year follow-up visit (see Table 1), indirectly may suggest that changes are to some extent fluency related, but further investigation is needed.

Although the patterns of brain activation at the 1-year follow-up scan do not indicate a normalisation of activation, it is obvious that observed cortical and subcortical overactivation is significantly reduced after 1 year, especially during oral speech. At the 1-year scan, the stuttering subjects in the present study had completed a structured maintenance program during which great emphasis is placed on continued practice to promote greater automatisation of the speech

skills acquired during the intensive treatment phase. Greater automatisation of skill performance has been shown to result in reduced overall brain activation (Raichle et al., 1994). Follow-up studies are needed to determine if reduction in overactivation, or the absence thereof, is linked to a subject's ability to successfully maintain treatment-induced fluency. Such investigations could help shed light on the question why some clients are successful in maintaining increased fluency following treatment while others show partial or complete relapse (Boberg, 1981; De Nil & Kroll, 1995).

The results from this study clearly have demonstrated that there are significant differences in neural activation patterns between stuttering and non-stuttering adults during speech, even when articulatory movements are not present. The use of single-word reading in the present task does pose some limitations on the generalisability of the findings to spontaneous conversational speech, which inherently is more complex (Cordes & Ingham, 1995). However, the similarities of our findings to other functional imaging studies where the investigators analysed connected speech, support the validity of our results. An important characteristic of the neural pattern seen in stuttering speakers is the overactivation relative to the level of activation seen in non-stuttering speakers. Such overactivation, and greater involvement of cerebellar functions (De Nil et al., 2001), supports the hypothesis that speech control in stuttering speakers lacks automatisation normally observed in non-stuttering speakers. Importantly, this overactivation can be reduced significantly as a result of intervention, although the current data did not support the notion that the activation in stuttering speakers became completely normalised post-treatment. Research is underway in our lab to investigate in greater detail the link between stuttering and deficiencies in the acquisition of skill automatisation. Another question that remains unanswered is whether brain activation patterns, and changes in these patterns resulting from treatment, differentiate between those who successfully maintain their fluency and those that do not. Measures of treatment outcome in this study were limited to speech fluency and did not include other measures such as naturalness or fluency in various speech tasks and situations. Given the presence of relapse following treatment in a number of clients, such investigations may shed light on mechanisms underlying maintained fluency or the lack thereof.

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CONTINUING EDUCATION

A positron emission tomography study of short- and long-term treatment effects on functional brain activation in adults who stutter

QUESTIONS

1. Previous research using functional neuroimaging investigating neural activation in stuttering and non-stuttering speakers during natural (stuttered) speech has demonstrated:
   a. activation in motor-related areas of the brain did not differ between fluent and disfluent speakers
   b. while the activation in association cortex differed significantly between stuttering and non-stuttering speakers, no differences were observed in motor-related areas
   c. stuttering speakers systematically showed overactivation in the motor systems of the brain
   d. differences between stuttering and non-stuttering adults were limited to subcortical regions, especially the basal ganglia
2. Using functional imaging techniques such as positron emission tomography, researchers can:
   a. investigate structural abnormalities in the brains of persons who stutter
   b. investigate changes in blood flow that are associated with the performance of various speech tasks
   c. investigate changes in neuronal electrical activity resulting from increased or decreased intercellular activity
   d. none of the above

3. Immediately following behavioural treatment:
   a. stuttering subjects showed increased fluency but no change in functional brain activation. Such changes in brain activation are seen only after 1-year maintenance intervention
   b. stuttering subjects showed significant changes in functional brain activation during oral reading
   c. functional brain activation in the stuttering subjects shifted towards a more left-lateralised pattern of activation
   d. stuttering subjects showed significant changes in functional brain activation during silent reading but not during oral reading
4. In the stuttering speakers, a comparison between silent and oral reading pre-treatment showed that:
   a. both tasks significantly activated cortical and cerebellar motor and pre-motor cortex
   b. cortical and cerebellar motor and pre-motor cortex was activated only during the oral reading task
   c. while activation during silent reading was exclusively left-lateralised, activation during the oral reading task was bilaterally distributed
   d. both b and c
5. The results from this investigation show that:
   a. behavioural fluency treatment had no effect on patterns of activation observed cortically or subcortically
   b. the acquisition of new speech motor patterns during behavioural fluency treatment was associated with significant changes in patterns of neural activation in stuttering but not non-stuttering subjects
   c. neural activation changes observed as a result of treatment were short lived and the activation reverted to its pre-treatment pattern at the 1-year follow-up
   d. effects of behavioural fluency treatment on patterns of brain activation, as measured by PET, are completely different from those observed in studies that have used fluency-enhancing techniques such as choral speech
   e. none of the above