Aberrant auditory processing and atypical planum temporale in developmental stuttering

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Abstract—*Objective:* To learn if people with persistent developmental stuttering and atypical anatomy of their auditory temporal cortex have, when compared to control subjects, changes in fluency induced with delayed auditory feedback (DAF). *Background:* DAF improves fluency in many individuals who stutter, and induces dysfluency in some normal people. The planum temporale (PT), a portion of auditory temporal cortex, is anatomically atypical in some adults who stutter and atypical anatomy might induce aberrant function. Thus, the people who demonstrate the paradoxical response to DAF might be those who have atypical anatomy. *Methods:* Experimental subjects were adults with developmental stuttering (n = 14) and control subjects (n = 14) matched for age, sex, education, and handedness. Volumetric MRI scans of all subjects were obtained and the PT was measured in the right and left hemispheres. Based on these scans, subjects were classified as typical (leftward PT asymmetry) or atypical (rightward PT asymmetry). Prose passages were read at baseline, with non-altered feedback (NAF), and with DAF, and fluency was measured in these three conditions. *Results:* At baseline the adults with developmental stuttering were significantly more dysfluent than controls ($p < 0.0005$). Controls' fluency did not significantly change with DAF, but DAF improved fluency in adults with developmental stuttering ($p < 0.0005$). In the stutter group enhanced fluency was associated with atypical (rightward) PT asymmetry, and the presence of typical (leftward) PT asymmetry was not associated with any significant change in fluency. The individuals with atypical PT asymmetry also had more severe stuttering at baseline compared to the experimental subjects with typical PT anatomy. *Conclusions:* In adults with persistent developmental stuttering and atypical PT anatomy, fluency is improved with DAF. These experimental subjects who showed improvement had more severe stuttering at baseline. Anomalous PT anatomy may be a neural risk for developmental stuttering in some individuals. Although a number of explanations are tenable, it may be that atypical rightward PT asymmetry may alter speech feedback, and treatment with DAF might allow these people to compensate.

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Developmental stuttering is a disorder of fluency characterized by involuntary repetitions, blocks, or prolongations in the utterance of speech elements, including sounds, syllables, and words. Prevalence of developmental stuttering is estimated at 4% of children and stuttering persists in 1% of adults.[1,2] Stuttering severity and the proportion of conversational speech that is dysfluent are highly variable across individuals. The dysfluencies observed in individuals who stutter may be reduced under a number of conditions including choral reading[3] and altered-auditory feedback.[4] The auditory system, at least at the level of auditory input, is involved in both of these fluency inducing conditions. Thus, there may be a defect at the level of auditory processing that is at least partially reversed with these procedures.[5]

One hypothesis is that alterations in the auditory signal under conditions of delayed auditory feedback (DAF) diminish an auditory perceptual defect in people who stutter.[6-8] This auditory perceptual defect might be related to anomalous anatomy of auditory temporal cortex. These anatomic anomalies might induce atypical activation-deactivation patterns or might perturbate the timing patterns required for the coordination of integrated neural networks. Using functional imaging, atypical activation-deactivation patterns of frontal, temporal, and cerebellar regions have been found when adults who stutter are examined during their baseline dysfluent condition, and some of these atypical activation-deactivation patterns are modified under conditions of induced fluency (e.g., choral reading).[9-11] Results from these functional imaging studies have also found that increased stuttering was related to de-

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creased activation of auditory temporal cortex, supporting the hypothesis that the auditory temporal cortex functions anomalously during dysfluent speech.[12] Further support for a central auditory processing defect comes from physiologic studies. Peripheral and brainstem auditory functions are normal in people who stutter, but alterations in the amplitude and topography of the central auditory signal have been found during stuttering. Thus, auditory perceptual defects may disrupt auditory self-monitoring. An alternative hypothesis could be that processing delays may cause instability in sound control output with stuttering behavior representing an online attempt to correct this instability.

In people who stutter, anatomic anomalies of the auditory cortex may be responsible for these physiologic and functional disturbances. Three studies have examined the anatomic basis of developmental stuttering,[13-15] but only one of these studies directly examined the anatomy of auditory temporal cortex. In this study volumetric MRI was used to examine the anatomy of perisylvian speech-language regions in adults with persistent developmental stuttering and in matched controls.[14] The planum temporale (PT), a portion of auditory association cortex, was found to be larger in the left and right cerebral hemispheres, and the expected leftward PT asymmetry was reduced (i.e., more symmetric) in the adults with developmental stuttering. Atypical anatomic features were also found in the brain regions that interconnect this portion of auditory cortex to frontal motor speech regions, and in the pars opercularis, a part of frontal motor speech cortex. These results provided the first evidence that adults with persistent developmental stuttering have atypical anatomy within portions of auditory and motor speech cortex and in the interconnecting brain regions.

In another recent study, diffusion tensor imaging MRI methods were used to examine white matter anatomy in adults who stutter and in fluent controls.[15] Fractional anisotropy of diffusion, an indirect measure of the coherence of diffusion, was measured in each MRI voxel and the groups were compared. A region of reduced fractional anisotropy, which may be associated with decreased fiber coherence or myelination defects, was found in the adults who stutter.[16] This region was limited to the white matter adjacent to the rolandic operculum within the left cerebral hemisphere deep to the inferior frontal gyrus and premotor cortex. Although similar white matter anomalies may be associated with atypical PT anatomy, this relationship has not been directly examined.

In our first anatomic study of adults with persistent developmental stuttering, PT size and asymmetry were found to be atypical,[14] but the relationship of atypical PT anatomy to atypical function was not examined. Central auditory processing defects have been found in some individuals with developmental stuttering, and DAF induces fluency in many people who stutter, therefore, we were interested in learning whether there was a relationship between the anatomy of the PT and fluency under altered auditory feedback conditions.

Table 1 *All subjects' scores on matching variables*

Subject pair	Hand preference		Age		Education	
	PDS	Control	PDS	Control	PDS	Control
1	−100.00	−90.63	37	32	16	20
2	−75.00	−90.63	35	19	16	12
3	−71.88	−87.50	29	44	21	18
4	−40.63	−46.88	29	21	18	15
5	42.19	43.75	23	21	16	15
6	78.13	56.25	24	43	17	15
7	87.50	59.38	22	23	17	16
8	87.50	67.19	29	23	19	16
9	90.63	75.00	29	42	20	18
10	90.63	100.00	25	40	17	14
11	90.63	100.00	27	33	19	18
12	93.75	100.00	31	41	19	16
13	100.00	100.00	47	43	12	14
14	100.00	100.00	24	47	16	16

Methods. *Subjects.* The sample included adults with persistent developmental stuttering (n = 14) and controls (n = 14) with the groups matched for age (= 33.71, SD_{PDS} = 10.34; = 29.36, $SD_{Control}$ = 6.69), education (= 15.93, SD_{PDS} = 2.06; = 17.36, $SD_{Control}$ = 2.24), and hand preference score (= 34.71, SD_{PDS} = 77.54; = 40.96, $SD_{Control}$ = 76.25). Raw scores for these variables are shown for each participant in table 1. The groups were also matched for sex and writing hand, with each group including seven right-handed men, three right-handed women, and four left-handed men. We were unable to recruit any left-handed women who stutter and therefore excluded left-handed women from the control group as well.

Our dysfluent sample was limited to adults with persistent developmental stuttering. All members of the stutter group were diagnosed before 8 years of age, and underwent treatment at some point, but continued to be dysfluent. Stuttering severity was determined using the Stuttering Severity Instrument 3rd edition (SSI-3)[17] and individuals in the stutter sample ranged from mildly to severely dysfluent. The SSI-3 provides a continuous measure of dysfluency with derived scores ranging from 0 to 56, and also provides a categorical measure of stuttering severity (mild, moderate, moderate-severe, severe). SSI-3 scores are based on the frequency of dysfluent events, length of dysfluencies, and the presence of concomitant behaviors. Speech samples were transcribed from videotaped sessions, and the number of prolongations, part-word repetitions, and blocks were computed and divided by the total number of spoken syllables to arrive at a frequency score. The length of dysfluencies was based on the average of the three longest dysfluent events. Measures of associated movements were also recorded and quantified. Although the SSI-3 is widely used for clinical assessment and research classification of stuttering severity, categorization of stuttering severity is difficult and complex, reflecting multiple dimensions of speech, language, and motor fluency measures.

All participants were native English speakers without history of dyslexia, specific language impairment, attention deficit disorder, personal or family history of tic disorder, traumatic brain injury, substance abuse, or other neuropsychiatric conditions. Subjects were recruited by word-of-mouth and by advertisement. All participants gave informed consent before participating.

Behavioral procedures. All testing was conducted in a sound insulated suite, and consisted of an audiologic screening test and

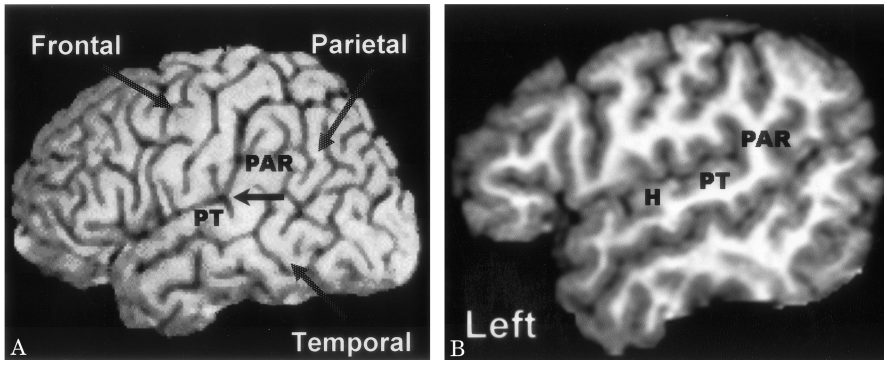

Figure 1. Anatomy of the planum temporale (PT). The PT is located along the superior surface of the superior temporal gyrus with the anterior boundary defined by the first Heschl's sulcus and the posterior boundary defined as the termination of the horizontal Sylvian fissure as this segment angles upward to form the posterior ascending ramus (PAR). The posterior boundary of the PT is identified by the arrow on the lateral surface of the three-dimensional surface rendering of the volumetric MRI in A. Heschl's gyrus (H), the PT, and the PAR are shown in the volumetric MRI sagittal image in B.

the experimental procedures. The audiologic screening test was performed to evaluate high frequency hearing loss and ear asymmetry. Hearing thresholds for each ear were assessed at frequencies of 500, 1,000, 2,000, and 3,000 Hz (Grason-Stadler GSI 61 Clinical Audiometer). Subjects with a hearing loss or threshold differences between the ears of greater than 10 dB on any of the frequencies tested were excluded from participation.

The experimental procedures consisted of having subjects read prose passages under three conditions: baseline, non-altered feedback (NAF), and DAF. In the baseline condition, the subject read the prose passage without earphones. A PhonicEar miniDAF unit (model PM505) was used for the NAF and DAF conditions. During NAF conditions, the participant heard his or her own voice amplified through headphones. During DAF conditions, the participant heard his or her own voice, again via headphones. However, under DAF the participant heard the amplified voice 120 msec after he or she emitted the speech sound. Thus, whereas DAF produces what sounds like an echo of the speaker's own voice, NAF involves no delay. Each participant spoke into a microphone clipped to his or her clothing approximately 15 cm from his or her mouth. Auditory feedback was provided via insert earphones. Output to the insert earphones was calibrated in an attempt to provide a speech level output that was consistent with auditory self-monitoring during normal conversation.[4] For all three conditions, subjects were instructed to read at "as normal a rate as possible while attempting to maintain maximum fluency."[18] These speaking rate instructions were given to all subjects across all conditions.

Each subject read the same three passages,[19] but the order of passages and reading conditions were randomized across subjects and between groups. The prose passages were matched for number of syllables (average 250), and were similar in thematic and syntactic complexity. Performance on the three tasks was video recorded with a camera and cassette recorder for transcription and scoring. Speech samples were transcribed, coded, and validated for accuracy. Two judges scored all of the speech samples using the video with the cassette recording as back-up. Intrajudge reliability for total dysfluencies was tested in a randomly selected subset of subjects (intraclass correlations > 0.95).

Three fluency measures were scored: frequency of stuttering (% Syllables), stuttering severity (SSI score), and reading time (Read time). The frequency of stuttering events included the number of prolongations, part-word repetitions, and blocks during passage reading and this event measure was computed as a percentage of the total number of syllables in the passage. Stuttering severity scores, based on the frequency of dysfluent events, length of dysfluencies (average of the three longest dysfluent events), and the presence of inaudible postural fixations, were computed following standard procedures,[17] yielding a continuous measure of dysfluency with scores ranging from 0 (no dysfluency) to 56 (most severe). Reading time was computed in seconds, and was derived from the total length of time it took to read each passage. Dysfluent speech characteristics were measured using standard procedures with high inter- and intrarater reliability. It is important to note that there are problems with dysfluency measurement. Stuttering behaviors and associated movements are assessed, and these measures are not necessarily correlated and may be associated with different biologic variables. In addition, the behavioral repertoires of people who stutter show individual differences, and behavioral patterns of a single stutterer are not entirely predictable.[20]

Neuroimaging procedures. Volumetric MRI scans were acquired on a General Electric 1.5 Tesla Signa Scanner with a T1-weighted spoiled GRASS sequence, as a gapless series of 124 contiguous sagittal images with the following technical factors: 1.5 mm slice thickness, field of view = 240 mm, 10 degree flip angle, 1 excitation, 256×256 pixel matrix. Measurements were performed on a Silicon Graphics computer using NIH image[21] software and macros written for NIH image. All MRI datasets were assigned a number, and were aligned along the anterior commissure–posterior commissure (AC-PC) line in the sagittal plane to correct for head rotation and then examined and corrected for rotation in the axial and coronal planes. One-half of the MRI studies were randomly selected and hemispheres were flipped (so that right and left were reversed). These formatting procedures were performed to assure that measurements were performed blind to group, sex, writing hand, and hemisphere. Inter- and intrarater reliability were established on the measurements described below in a randomly selected subsample of subjects ($n = 8$) before the formal experimental measurements commenced (intraclass correlations >0.90). All anatomic measures were done in the sagittal plane in real space with no warping of the images; orthogonal views were used to assist in the determination of landmarks and anterior-posterior boundaries.[22,23]

The PT is a flat triangular plane, comprised of auditory association cortex (Brodmann's area [BA] 22), located along the temporal bank of the Sylvian fissure on the surface of the superior temporal gyrus. The PT extends from the first Heschl's sulcus to the end of the horizontal Sylvian fissure (figure 1, A and B). The posterior extent is demarcated by the bifurcation of the posterior horizontal ramus into an ascending, and descending ramus. When these rami were not clearly demarcated or in cases with an absent posterior ascending or descending ramus, the posterior endpoint of the PT was determined by following the plane of the posterior horizontal ramus to its intersection within the parietal bank. This approach is called a "knife-cut" method, and has been used in many other in vivo MRI studies of the PT,[22-24] and in studies of postmortem brains.[25]

Measurements were made by using a computer-guided cursor to trace the cortical surface in the sagittal plane, conforming to the topography of the gyrus, including the depth of the sulcus, except when adjacent sulci were closely opposed. These methods have proven to be more reliable than tracing the gray-white border, or than by following every surface irregularity. For each hemisphere mean volumes were calculated by summing surface areas in successive sections and multiplying by slice thickness. Our PT measures have been reported elsewhere, correlate with volumetric measures,[22] are similar to methods used by Geschwind and Levitsky[25] in their landmark postmortem study, and were used in our study that found a significant relationship between planar asymmetries and language laterality identified by Wada (intracarotid barbiturate injection) testing.[23]

Analyses. Data were analyzed using three-way mixed analyses of variance, with group (control, developmental stuttering) and PT asymmetry (leftward, rightward) entered as grouping factors and with auditory feedback condition (baseline, NAF, DAF) en-

tered as a repeated measure. PT asymmetry quotients (AQs) were computed using the following formula:

$$AQ = \frac{Left - Right}{1/2(Left + Right)}$$

Subjects were categorized as having leftward PT asymmetry (left>right) when the AQ was positive, and were categorized as having rightward PT asymmetry (right>left) when the AQ was negative. For effects involving auditory feedback conditions, violations of the sphericity assumption were corrected via adjustments in degrees of freedom using the Huynh-Feldt method.[26] Pairwise comparisons subsequent to significant interactions were made using the estimated marginal means method.[27]

Results. *Planum temporale anatomy.* In the entire sample, the majority of adults had a leftward PT asymmetry. In each group (developmental stuttering, control), there was a leftward PT asymmetry in 9 of 14 (64%) subjects, and a rightward asymmetry in 5 of 14 (36%) subjects. Thus, PT asymmetry patterns did not differ significantly between the groups, and these distributions are similar to figures reported in large sample studies with the same handedness mix.[28-31]

Structure–function relationships. The hypothesis that PT asymmetry would be related to changes in fluency induced by DAF was supported by significant group × PT asymmetry × auditory feedback condition interactions for each fluency measure (stuttering events, $p = 0.005$; stuttering severity, $p = 0.045$; reading time, $p = 0.008$). The developmental stuttering subgroup with atypical rightward PT asymmetry was significantly more dysfluent at baseline than the stuttering subgroup with typical leftward PT asymmetry ($ps < 0.020$). Fluency was not induced with DAF in the stuttering subgroup with typical leftward PT asymmetry ($ps > 0.206$). In contrast, fluency was induced with DAF in the stuttering subgroup with atypical rightward PT asymmetry (all three fluency measures $ps < 0.0005$). Overall, the control group was unaffected by DAF, although the subgroup of control subjects with typical leftward PT asymmetry became significantly more dysfluent with DAF compared to the baseline condition (stuttering severity, $p = 0.029$). No other significant effects of auditory feedback on speech behavior were observed in the controls ($ps > 0.056$). In summary, controls and the developmental stuttering group with typical PT asymmetry were relatively unaffected by DAF, whereas the stuttering group with atypical PT asymmetry exhibited significant enhanced fluency associated with DAF. The fluency performance of PDS subjects with atypical anatomy improved to the level of the performance of PDS subjects with typical anatomy. A summary of means is shown in table 2 and a summary of between-fluency-group significance tests is shown for all conditions in table 3. Specific effects for each fluency measure are described in the sections below, and are graphically represented in figure 2, A through C.

Frequency of stuttering events. There was a significant group effect ($F[1,24] = 18.66$, $p < 0.0005$), with the developmental stuttering group (mean = 4.26, SE = 0.624) more dysfluent than the control group (mean = 0.44, SE = 0.624). There was a significant DAF condition × Group ($F[1.89,45.34] = 6.24$, $p = 0.005$) and DAF condition × PT asymmetry interaction ($F[1.89,45.34] = 7.04$, $p = 0.003$) with both modified by a significant Group × DAF condition × PT asymmetry interaction, $F[1.89,45.34] = 6.18$, $p = 0.005$.

Table 2 *Means (SD) for fluency measures at baseline and under non-altered feedback (NAF) and delayed auditory feedback (DAF) conditions*

	Fluency measures		
Condition/group	% Syllables	SSI score	Read time, s
Baseline			
Stutter	5.15 (5.57)	12.00 (7.30)	96.51 (38.38)
Control	0.30 (0.40)	1.08 (1.32)	47.72 (6.29)
NAF			
Stutter	4.33 (4.08)	10.64 (6.63)	93.64 (40.27)
Control	0.33 (0.47)	1.23 (1.74)	49.37 (8.29)
DAF			
Stutter	2.60 (3.22)	8.00 (6.21)	84.66 (25.94)
Control	0.83 (1.24)	3.31 (3.74)	52.92 (9.36)

Stuttering severity. There was a significant group effect ($F[1,24] = 24.80$, $p < 0.0005$). The developmental stuttering group (mean = 10.61, SE = 1.25) had significantly higher SSI-3 scores than controls (mean = 1.82, SE = 1.25). There was a significant DAF condition × Group interaction ($F[1.94,46.47] = 12.10$, $p < 0.0005$) and DAF condition × PT asymmetry interaction ($F[1.94,46.47] = 7.67$, $p = 0.001$), and both were modified by a significant Group × DAF condition × PT asymmetry interaction, $F[1.94,46.47] = 3.35$, $p = 0.045$.

Reading time. There was a significant group effect ($F[1,24] = 22.10$, $p < 0.0005$); the developmental stuttering group (mean = 92.16 seconds, SE = 6.30) took significantly longer to read the prose passage than did the control group (mean = 50.28 seconds, SE = 6.30). There was a significant DAF condition × Group interaction ($F[2,48] = 3.75$, $p = 0.031$) and DAF condition × PT asymmetry interaction ($F[2,48] = 4.93$, $p = 0.011$), and both were modified by a significant Group × DAF condition × PT asymmetry interaction, $F[2,48] = 5.42$, $p = 0.008$.

Discussion. It is well established that altered-auditory feedback enhances fluency in some individuals with persistent developmental stuttering, but there is controversy regarding the factors that con-

Table 3 *Between-fluency-group comparisons for all conditions*

		Condition		
PT asymmetry group	Measure	Baseline	NAF	DAF
L>R	% Syllables	0.200	0.077	0.087
	SSI score	0.006	0.011	0.141
	Read time	0.020	0.032	0.001
R>L	% Syllables	0.000	0.002	0.461
	SSI score	0.000	0.000	0.075
	Read time	0.000	0.003	0.080

The table's cells contain p values for control vs PDS comparisons under each condition for both PT asymmetry groups.

PT = planum temporale; NAF = non-altered feedback; DAF = delayed auditory feedback.

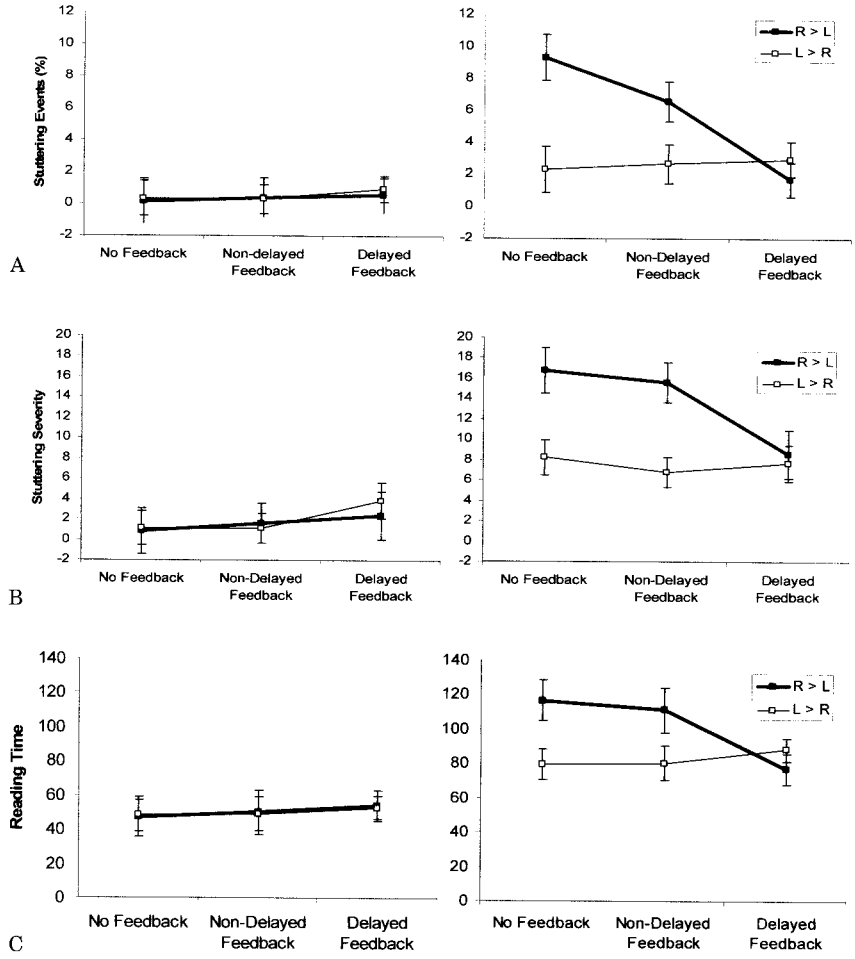

Figure 2. Structure–function relationships in developmental stuttering. Relationships and means associated with the significant Group × delayed auditory feedback (DAF) condition × planum temporale (PT) asymmetry interactions are shown for each fluency measure. The frequency of stuttering events variable is depicted in A, the stuttering severity variable is depicted in B, and the reading time variable is depicted in C. Error bars represent standard error (SE) of the mean.

tribute to this enhanced fluency effect.[5] One explanation is that alterations in the auditory signal might correct an auditory perceptual defect.[3-8] We hypothesized that this auditory perceptual defect would be related to an anatomic anomaly of the auditory temporal cortex, such as atypical PT asymmetry. Atypical structure can induce atypical function, thus abnormal function of the auditory association cortex might disrupt the normal transmission of auditory information to frontal motor regions, and this disruption might perturb the coordination of integrated neural networks yielding dysfluent speech output. Our finding that developmental stuttering paired with atypical PT asymmetry yielded more dysfluency at baseline than did persistent developmental stuttering paired with typical PT anatomy supports this hypothesis. Further support of this hypothesis comes from the observation that with DAF, persistent developmental stuttering subjects with anomalous anatomy of auditory cortex (i.e., rightward PT asymmetry) improved more than those subjects with typical leftward PT asymmetry.

The PT, which includes cytoarchitectonic areas TA and TB (BA 22), plays an important role in higher order processing of auditory stimuli. The left PT is part of Wernicke's area, which, when lesioned, produces a fluent aphasia with impaired auditory comprehension, repetition, and naming.[29,32] Lesion and functional imaging studies suggest that this area performs phonologic and lexical analyses or decoding.[33-35] A leftward PT asymmetry is well documented in the majority of adults,[24,28,29] and one study found a direct relationship between PT asymmetry and language laterality.[23] The finding that planar asymmetries are present in the fetus (29 weeks gestational age) before language acquisition suggests that this region may represent a biologically determined anatomic substrate that is preprogrammed for the asymmetric representation of speech–language functions.[36] In addition, leftward asymmetries of the intrinsic circuitry[37] and of cytoarchitectonic areas[38] within auditory association cortex have been reported. Although investigators have advised caution in attributing functional significance to these anatomic asymmetries, there has been a tendency to regard the distinctive morphologic characteristics of the PT as probably representative of a neuroanatomic substrate for language.[24,25] Thus, the importance of the PT in auditory language functions is well-established, and our results, which demonstrate that auditory processing defects are directly related to atypical PT anatomy, suggest that neural defects of the PT may be partly responsible for stuttering in some individuals.

Based on our subjects' responses to DAF, PT asymmetry, and stuttering severity, our results also

suggest that there are at least two biologic subgroups of people who stutter. Thus, the influence of behavioral and phonologic therapies of stuttering may be different depending on group membership. In one group, stuttering might relate to a defective speech monitoring system.[6-8] The basic theory is that ongoing sequential and fluent speech output is dependent on auditory sensory feedback, which monitors and corrects errors online. In normally fluent individuals, a dysfluency would be detected as an "error," and this error would be automatically corrected online. Stuttering behaviors, such as involuntary repetitions, blocks, or prolongations in the utterance of speech elements, may be induced by auditory perceptual defects that disrupt auditory self-monitoring within a closed-loop system.

Stuttering can be modeled as the result of disturbed auditory feedback. The subgroup that we identified with atypical PT anatomy may represent a distinct biologic subgroup with this self-monitoring or feedback perceptual disorder. However, neither atypical PT anatomy nor DAF induced fluency was observed uniformly throughout the developmental stuttering group. Thus, deficits in auditory processing cannot account for stuttering in all people with persistent developmental stuttering. Rather, there may be a developmental stuttering subgroup for which other neural systems are the main cause of stuttering.

An anatomic model, based on a two-loop timing theory of speech output, is presented in figure 3. According to this model, there are two main neural networks that work together to coordinate speech production.[14,39] These neural circuits have been characterized as an outer "linguistic" and an inner "phonatory" loop or circuit. The outer linguistic circuit is involved with phonologic, lexical, syntactic, and semantic language functions, and the more elemental processing of auditory verbal information, such as selecting and monitoring speech sounds. It is proposed that this neural circuit plays an important role in auditory self-monitoring. In contrast, the inner circuit is involved with the motor programs of the vocal apparatus, and may be more important in the motor control of speech output. Stuttering can be modeled as a momentary instability in these systems when the timing between or activation of these two circuits is interrupted.

This two-loop timing hypothesis was not originally proposed in relation to brain anatomy, but it has been suggested that the outer loop includes perisylvian brain regions that control speech–language functions, and the inner loop includes cortical and subcortical motor regions (cortical-striatal-cortical circuits).[14,39,40] Theoretically, a defect at any point within either of these distributed neural networks could induce stuttering by disrupting the flow of information, which in turn would induce asynchronous activation of the paired muscles that mediate speech production. A number of defects within these neural networks may induce stuttering. It may be that specific anatomic anomalies within the outer loop disrupt the flow of information within this pathway relative to the inner loop. In contrast perhaps in some people with developmental stuttering asynchrony is induced by slowing of the inner loop. Support for this postulate comes from the observation that many patients with PD may exhibit stuttering,[41,42] and there is some evidence that the dopaminergic neurotransmitter systems are disrupted in some people who stutter.[43] Thus, the reduction of dopamine with slowing of the inner loop by Parkinson disease (PD)[40,44] or by the administration of neuroleptics[45] might alleviate developmental stuttering because it allows activation of these networks to be synchronized. The finding that delayed auditory feedback, which slows the speech rate, improved fluency in some subjects would suggest that these subjects have slowing of their inner loop. Delaying auditory feedback slowed their outer loop, which helped re-establish synchrony. It is, however, possible that the subjects who improved did have slowing of their outer loop, and further slowing of speech allowed synchrony to take place at another harmonic.

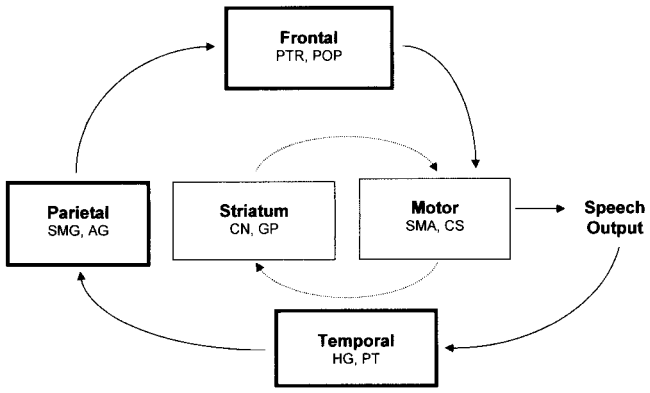

Figure 3. Anatomic model of speech–language output. The outer linguistic loop is comprised of perisylvian speech–language areas and interconnecting white matter pathways. Specific regions include the PT, the inferior parietal lobe (PAR), and frontal language areas: pars triangularis (PTR) and pars opercularis (POP). The inner phonatory loop is comprised of cortical and subcortical motor areas including portions of the pyramidal and extrapyramidal motor systems, and the cerebellum. Portions of the pyramidal system include primary (motor cortex along the length of the central sulcus [CS]) and premotor cortex (supplementary motor area [SMA]). Extrapyramidal or subcortical motor areas include the striatum (caudate nucleus [CN], globus pallidus [GP]), substantia nigra, and subthalamic nucleus. The inner and outer loops depicted in figure 3 are interconnected with the final common pathway at the level of speech output. Speech output in turns feeds back to the auditory temporal areas.

We noted previously that within the PDS group those with atypical planar asymmetry (right > left) exhibited significantly greater dysfluency (at baseline) than did those with typical planar asymmetry (left > right). We also noted that only in those individuals with atypical anatomy did fluency improve with the presence of DAF. As discussed above, these

observations are consistent with the theory that PT asymmetry and associated auditory processing abilities influence the effects of DAF. This is theoretically consistent with the notion that leftward asymmetry in this area is adaptive and that absence of this asymmetry is a risk factor for PDS that is correctable by DAF. However, it is important to acknowledge an alternative explanation for this pattern of results. Specifically, it is possible that the difference between PT asymmetry groups is evidence of a ceiling effect in the ability of DAF to induce fluency; e.g., that DAF simply can improve severe stuttering but cannot improve mild stuttering. If this is the case, the asymmetry group difference in DAF effect may have been caused by the concomitant group differences in stuttering severity rather than by PT asymmetry itself.

The results of the present study demonstrate that anomalous anatomy of the PT, a brain region that comprises a portion of the outer linguistic loop, may be associated with aberrant auditory processing. Other portions of these networks have been implicated in developmental stuttering, but have not been examined at the anatomic level. For example, it is important to determine whether anatomic anomalies in cortical and subcortical motor systems are present in some individuals who stutter. Based on anatomic, physiologic, and pharmacologic studies, different biologic subgroups may be identified that in turn may be responsive to different types of behavioral and pharmacologic treatments.

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