Abnormal speech sound representation in persistent developmental stuttering

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Abstract—*Objectives:* To determine whether adults with persistent developmental stuttering (PDS) have auditory perceptual deficits. *Methods:* The authors compared the mismatch negativity (MMN) event–related brain potential elicited to simple tone (frequency and duration) and phonetic contrasts in a sample of PDS subjects with that recorded in a sample of paired fluent control subjects. *Results:* Subjects with developmental stuttering had normal MMN to simple tone contrasts but a significant supratemporal left-lateralized enhancement of this electrophysiologic response to phonetic contrasts. In addition, the enhanced MMN correlated positively with speech disfluency as self-rated by the subjects. *Conclusions:* Individuals with persistent developmental stuttering have abnormal permanent traces for speech sounds, and their abnormal speech sound representation may underlie their speech disorder. The link between abnormal speech neural traces of the auditory cortex and speech disfluency supports the relevance of speech perception mechanisms to speech production.

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Neural mechanisms underlying developmental stuttering are not fully understood despite recent progress. Individuals with persistent developmental stuttering (PDS)[1,2] have anatomic abnormalities, including an atypical planum temporale asymmetry[3] and a cortical disconnection between the frontal operculum and the ventral premotor cortex.[4] They show extensive hemodynamic hyperactivity over the motor and premotor system, with a predominant right lateralization in the primary and extraprimary motor cortices.[5] When speaking, stutterers do not properly activate the superior and posterior temporal and the inferior frontal cortices of the left hemisphere, although during induced fluency conditions, these abnormal activation patterns are reduced.[5] Moreover, they show a functional dissociation between activity in postrolandic regions involved in auditory perception and anterior forebrain regions involved in the regulation of motor function,[6] the activation imbalance between the anterior and postrolandic areas being reduced during fluency-evoking tasks.[6] Magnetoencephalography (MEG) has revealed abnormal temporal patterns of activation in PDS.[7,8] Indeed, the activation patterns after seeing a word progressed in stutterers from a frontoparietal region encompassing the left lateral central sulcus and the dorsal premotor cortex to a left inferior frontal cortex region,[7] whereas fluent speakers had a reversed activation sequence, suggesting that stutterers trigger speech motor programs before activation of the articulatory code.[7] Stuttering can be transiently alleviated by fluency-inducing techniques, including chorus reading, singing, masking, and shadowing.[1,5,6,9] These effects seem to rely on the provision of external timing patterns for speech vocalization, which in turn may diminish auditory perceptual deficits in people who stutter.[9]

In this study, we used the mismatch negativity (MMN) to test the hypothesis that stuttering adults have specific auditory perceptual deficits regarding speech sounds but not very simple sound features. Moreover, we predicted that if present, these specific speech sound perceptual deficits should relate to the stuttering behavior. The MMN is a cognitive evoked potential (ERP) elicited to unexpected auditory stimuli deviating from the preceding "standard" sounds in any of their physical or even more complex attributes.[11,12] It appears as a negative waveform peaking at 100 to 200 milliseconds with a frontocentral scalp distribution and positive voltages below the sylvian fissure, indicating generator sources located bilaterally to the supratemporal plane of the auditory cortex.[11,12-15] In fact, the electrodes positioned below the sylvian fissures, including those at the mastoid apophyses, reflect the activity of the ipsilateral supratemporal MMN generator of their side.[16,17] Critical in MMN theory is that deviant stimuli occur asynchronously with the preceding standard stimulation, so that the brain's neurophysiologic response to such rare stimuli requires a comparison trace of the preceding repetitive stimulus features, i.e., a neural representation of the standard sound.[10,18] The MMN response is therefore of a perceptual nature, the neural sound representation involved in its gen-

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eration subserving the conscious perception of sound.[10,19,20] The MMN indexes different forms of preattentive cognitive operations within the auditory cortex,[10] such as the formation of perceptual sound objects[21] or the extraction of abstract sound patterns from the auditory background.[22,23] Relevant for the current study is the MMN evidence indicating that categorical speech perception requires language-specific memory traces within the auditory cortex,[10,24,25] and the development of such permanent traces is a necessary prerequisite for the appropriate perception and subsequent production of a properly spoken language.[26,27]

Methods. *Subjects.* Twelve PDS subjects (aged 22.2 ± 3.5 years, two women) and 13 fluent speakers (aged 23.3 ± 3 years, two women) gave their informed consent to participate in the study, which was approved by the Ethical Committee of the University of Barcelona. Three of the 12 PDS subjects and 1 control were left-handed according to standard laterality tests.[28] The two groups were matched for age, sex, and educational level. Sex and handedness ratios in the PDS group were similar to those reported previously (men > women, right-handed > left handed).[2,29] Subjects from the PDS group were recruited among those attending follow-up clinics at the Hospital del Mar in Barcelona. A certified speech–language pathologist diagnosed the stuttering condition according to criteria of the *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition.[30] Only nonanxious stutterers were selected, and no PDS subject had received treatment for stuttering within the preceding 5 years.

All participants were native bilingual speakers of both Catalan and Spanish languages. Eight subjects of the stuttering group reported a family history of stuttering, and none of the control group reported having a family history with speech or language disorders, this discrepancy reflecting the influence of genetic factors thought to be of significance for stuttering persistence.[31]

All study participants were given a battery of tests, including a Test of General Intelligence (TIG-1),[32] a State-Trait Anxiety Questionnaire (STAI),[33] and the Beck Hopelessness Scale (BHS).[34] No differences in these tests were found between groups. Additionally, speech fluency/disfluency was assessed by the speech pathologist using standard criteria and by means of a self-administered questionnaire, the Conduct and Attitude Scale for the Assessment of Disfluencies (CASAD), developed at the Hospital del Mar and available on request. In this questionnaire, speech fluency is rated in four different categories, which evaluate the consequences associated with the stuttering symptoms and their relationship with the patient's social behavior. Subjects had to answer 25 items using a five-point rating scale, where 0 indicated normality and 4 indicated the highest degree of severity of disfluency (maximum score = 100). According to both the speech pathologist and the results of this scale, individuals in the patient sample ranged from mild to severe (table 1).

Stimuli and procedure. Subjects sat in a comfortable armchair, in a sound-attenuated and dimly lit room. Testing lasted approximately 2:15 hours, including two recording 1-hour blocks separated by a 15-minute break. An audiometric test was administered to each subject before the experimental session, resulting in similar hearing thresholds, all below 40 dB sound pressure level (SPL), in both groups.

The MMN was obtained in three different sound-contrast conditions, two of them involving simple sound features, i.e., frequency and duration changes, and the remaining one involving phonemes. Moreover, each of the simple sound-contrast conditions included four levels of stimulus deviation with regard to the relevant standard stimulus feature. Along the recording session, subjects were presented with two stimulus blocks of each of the three different sound-contrast conditions, and the resulting six total blocks were arranged in random order. All auditory stimuli were delivered binaurally through headphones at an intensity of 85 dB SPL. Subjects were instructed to watch a silent video movie, to ignore the auditory stimulation, and to avoid blinking and extra body movements.

In the Frequency condition, each of the two stimulus blocks consisted of 1,000 pure tones, including repetitive standard tones of 1,000 Hz ($p = 0.8$) and deviant tones of four different frequency change levels, the deviant frequencies being 1,015, 1,030, 1,060, and 1,090 Hz, all of them occurring in random order and with the same deviant probability ($p = 0.05$). Both stimulus types, standard and deviant, had a duration of 50 milliseconds, including 10 milliseconds of rise/fall times.

In the Duration condition, subjects were also presented with two blocks of 1,000 stimuli each. In this condition, the standard tone duration was 200 milliseconds ($p = 0.8$), and the four deviant stimulus durations were 160, 120, 80, and 40 milliseconds, again occurring in random order with the same deviant probability ($p = 0.05$ each). All tones had a frequency of 1,000 Hz and a rise/fall period of 10 milliseconds. In both the Frequency and Duration conditions, all stimuli were delivered with a constant stimulus onset asynchrony (SOA) of 480 milliseconds.

In the third condition, the auditory stimuli were semisynthetic phonemes (Phoneme condition).[25] These auditory stimuli varied in their second formant (F2) frequency, whereas the F1 (450 Hz), F3 (2,540 Hz), and F4 (3,500 Hz) frequencies, as well as the fundamental frequency (105 Hz), were kept constant across all the three phonetic stimuli used in the experiments. The standard phoneme consisted of the vowel /o/ with an F2 of 851 Hz and occurred with a probability of $p = 0.8$. The deviant phoneme was either a Spanish prototypic /e/, with a second formant of 1,940 Hz, or a nonprototypic /ö/, with an F2 of 1,533 Hz. Each of the deviant phonemes was presented in a separate block among the standard phoneme described above, with a probability of $p = 0.2$. Standard and deviant phonemes were of 400 milliseconds in duration, including 10 milliseconds of rise/fall times. Each of the blocks included a total of 400 standard and 100 deviant phonemes that were delivered in random order with a constant SOA of 900 milliseconds.

Electrophysiologic recordings. The EEG was continuously recorded and digitized (band pass 0.1 to 100 Hz, A/D rate = 500 Hz) by a SynAmps amplifier (Neuroscan, El Paso, TX) from standard 10/20 scalp locations at Fz, F3, F4, F7, F8, Cz, C3, C4, T3, T4, and two additional electrodes attached at the left (LM) and right (RM) mastoids. Blinks and horizontal ocular movements were measured by electrodes located on the infraorbital ridge and on the outer canthus of the left eye. An electrode attached to the tip of the nose served as reference for the EEG and electroculogram (EOG) recordings.

Data analysis. The ERPs were averaged offline for standard and deviant stimuli, separately for each subject and condition. Epochs with EOG or EEG exceeding ±100 μV at any channel as

Table 1 *Individual patient information for PDS subjects*

Patient/ age, y/ sex	Laterality	BHS	TiG-1	STAI		CASAD
				State	Trait	
S1/21/M	Left	15	50	99	23	29
S2/20/M	Right	0	20	99	70	51
S3/18/M	Left	0	35	95	30	58
S4/24/M	Right	8	10	90	15	41
S5/26/M	Right	8	11	95	30	56
S6/19/M	Right	8	80	99	20	38
S7/26/F	Left	2	80	95	10	53
S8/24/F	Right	2	11	99	15	32
S9/22/M	Right	0	70	97	14	39
S10/22/M	Right	0	97	99	85	34
S11/19/M	Right	2	70	99	95	11
S12/30/M	Right	0	70	99	88	36

PDS = persistent developmental stuttering; BHS = Beck Hopelessness Scale; TIG-1 = Test of General Intelligence (individual percentiles); STAI = State-Trait Anxiety Questionnaire (individual percentiles); CASAD = Conduct and Attitude Scale for the Assessment of Disfluencies.

Table 2 t *Tests of MMN mean amplitude for the Frequency, Duration, and Phoneme conditions at Fz*

	Stutterers			Controls		
	Latency window, ms	Mean amplitude, μV	t_{12}	Latency window, ms	Mean amplitude, μV	t_{12}
F: 1,015 Hz	200–240	−0.5 (0.29)	−1.872	180–220	−0.4 (0.28)	−1.638
F: 1,030 Hz	150–190	−0.5 (0.29)	−1.857	165–205	−0.5 (0.28)	−1.663
F: 1,060 Hz	155–195	−0.1 (0.30)	−3.190*	155–195	−1 (0.28)	−3.445†
F: 1,090 Hz	175–215	−1.3 (0.27)	−4.691*	177–217	−1.1 (0.30)	−3.542†
D: 160 ms	280–320	−0.5 (0.26)	−3.200*	250–290	−0.5 (0.24)	−1.932
D: 120 ms	245–285	0 (0.31)	0.006	245–285	−0.1 (0.32)	−0.434
D: 80 ms	240–280	−0.1 (0.25)	−3.889*	230–270	1.0 (0.23)	−4.227*
D: 40 ms	170–210	−1.9 (0.43)	−4.297*	190–230	−2.0 (0.40)	−4.747*
Ph : /e/ eMMN	105–125	−1.2 (0.38)	−3.092*	100–120	−1.6 (0.33)	−4.757*
Ph : /e/ lMMN	190–210	−1.8 (0.40)	−4.314*	165–185	−1.8 (0.45)	−3.856*
Ph : /ö/ eMMN	110–130	−0.9 (0.39)	−2.276*	100–120	−1.1 (0.28)	−3.799*
Ph : /ö/ lMMN	180–200	−2.1 (0.47)	−4.426*	175–195	−1.8 (0.43)	−4.218*

The latency window in which the mismatch negativity (MMN) was measured and the mean amplitude in this latency window (SEM in parentheses) for each condition and group are given. The frequency values given for the Frequency (F) condition and the duration values given for the Duration (D) condition correspond to the deviant values in these conditions for a standard tone of 1,000 Hz in the F condition, and for a standard tone of 200 ms in the D condition. The early MMN (eMMN) and late MMN (lMMN) in the Phoneme (Ph) condition correspond to the early and late portions of MMN measured in this condition.

* $p < 0.01$.
† $p < 0.001$.

well as the first five periods of each block were automatically excluded from averaging. The epoch of the Frequency and Duration conditions was of 580 milliseconds, including a prestimulus baseline of 100 milliseconds. The epoch of the Phoneme condition was of 1,000 milliseconds, including 100 milliseconds of baseline as well. Standard sound epochs immediately after deviant sound epochs were also excluded from the averages. Individual ERPs were digitally band-pass filtered between 0.1 and 30 Hz.

Mismatch negativity was measured in the difference waves obtained by subtracting the standard ERPs from those elicited to deviant sounds, as the mean amplitude in a 40-millisecond latency window around the maximum peak identified in the grand-average difference wave, for each group and condition separately. The latency windows yielded in each condition and group are given in table 2. Furthermore, in the difference waves obtained in the Phoneme condition, two consecutive peaks could be identified in the MMN latency range. Consequently, an early MMN was identified as the largest response in the 100 to 140 latency window, and a late MMN was identified as the largest response within the 160 to 200 latency window, and the mean amplitude in a 20-millisecond latency window around these peaks was measured.

To determine whether a significant MMN was elicited in each condition and group, one-tailed t tests were used to compare the MMN mean amplitude at Fz against zero. MMN comparisons were performed by means of analyses of variance (ANOVAs) for repeated measures, including group (PDS and control) as the between-subjects factor, and the electrode, including the F3, Fz, F4, C3, Cz, and C4 leads, as a within-subjects factor. These ANOVAs were performed for all deviant stimuli in the three conditions. In addition, a four-way ANOVA was performed in the Phoneme condition using group (PDS and control) as a between-subjects factor and three within-subjects factors, which examined laterality of MMN (right—F4, RM vs left—F3, LM, hemispheres), phoneme type (prototypic /e/ vs nonprototypic /ö/), and MMN generator, with supratemporal (RM, LM) and frontal (F4, F3) levels. In all of the ANOVAs, the Greenhouse–Geisser correction was applied when appropriate, and the corrected p values are reported.

Results. *Mismatch negativity elicited in the Frequency and Duration conditions.* Figure 1 shows MMN grand-average difference waveforms in the Frequency and Duration conditions for the PDS and control groups. As expected, MMM amplitude increased with the magnitude of the stimulus deviance in both the Frequency and Duration conditions. Statistical analyses revealed significant

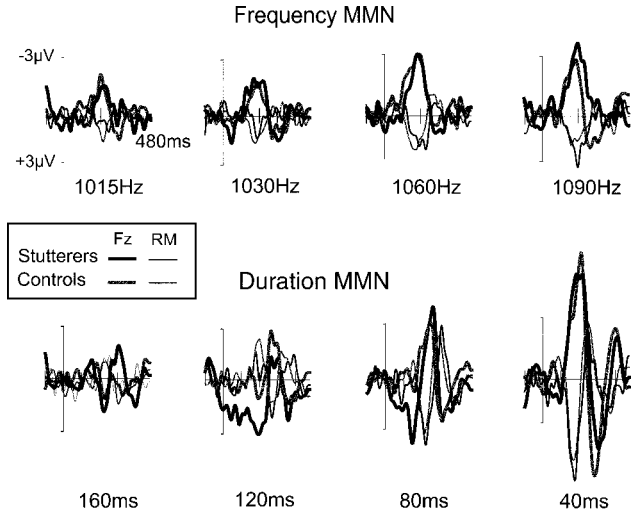

Figure 1. Difference waves obtained by subtracting the event-related brain potentials elicited to standard stimuli from those elicited to the deviant ones, for the four deviant types in the Frequency and Duration conditions. Fz (thick line) and right mastoid (RM; thin line) waveforms are plotted for persistent developmental stuttering (black line) and control (gray line) subjects in the same panel. The mismatch negativity (MMN) appears as negative deflection (pointing upward) at Fz, with its corresponding polarity reversal, i.e., positive deflection, at RM. Notice that the MMN increases in amplitude, in both groups similarly, as increasing the deviant–standard tone difference.

MMNs elicited similarly in both groups for the largely deviant stimuli in both conditions, i.e., for the 1,000- to 1,060-Hz and the 1,000- to 1,090-Hz frequency contrasts, and for the 200- to 80-millisecond and the 200- to 40-millisecond duration contrasts (see table 2 and figure 1). The similar brain responses to simple auditory stimulus contrasts in both groups were also indicated by the lack of significant differences in MMN mean amplitude between groups.

Mismatch negativity elicited in the Phoneme condition. Deviant phonemes, either prototypic or nonprototypic, elicited significant similar MMN in both groups, as shown in table 2 and figure 2. However, although the MMN amplitude for the Phoneme condition did not differ significantly between groups when compared at frontal and central scalp electrodes, striking group differences became evident when the factors MMN generator, cerebral hemisphere, and stimulus type were included in the ANOVA, as a strong interaction between groups, MMN generator (supratemporal vs frontal), and hemisphere (left vs right), for both the early [$F(1,23) = 7.08$, $p < 0.014$] and the late [$F(1,23) = 7.22$, $p < 0.013$] parts of MMN. As can be seen in figure 2, these interactions resulted from the MMN being larger at the LM for the two deviant phonemes in the PDS group, suggesting a much stronger activation of the left supratemporal MMN generator for phonetic contrasts in the PDS group vs control subjects.

Speech fluency and its relationship to electrophysiology. As expected, speech fluency as evaluated by means of the self-administrated questionnaire (CASAD), was clearly different between the stuttering and control subjects (mean score: PDS subjects = 39.83, controls = 8.46; $t_{23} = 7.79$, $p < 0.001$). To test whether there was any relationship between speech fluency and the electrophysiologic anomaly found in speech sound representation in the patients, a Pearson correlation coefficient was calculated between the score in CASAD and the amplitude of MMN elicited in the Phoneme condition at the LM in both groups. A positive correlation was found between these two factors (Pearson $r = 0.40$, $p = 0.043$), indicating, as shown in figure 3, that speech disfluency as rated by CASAD increased linearly with increasing the phonetic MMN LM amplitude.

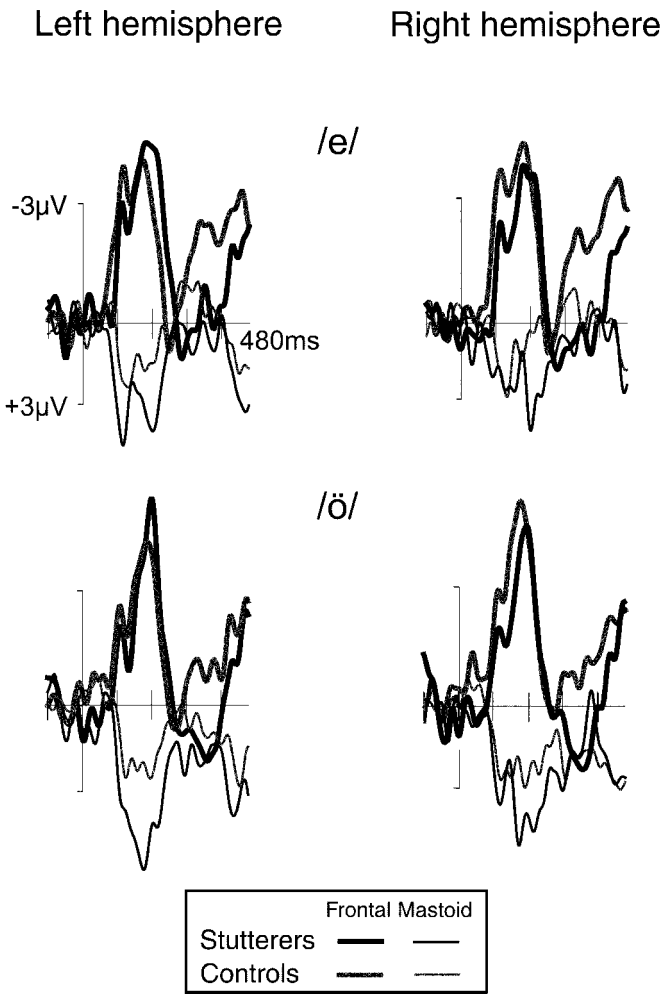

Figure 2. Difference waves (deviant minus standard event-related brain potentials) in the Phoneme condition (upper panel, prototypic /e/; lower panel, nonprototypic /ö/). The figure follows the same scheme as figure 1, i.e., the frontal (F3, F4; thick line) and mastoid (left mastoid, right mastoid; thin line) waveforms are plotted for persistent developmental stuttering (black line) and control (gray line) subjects in the same panel. As in figure 1 for the Frequency and Duration conditions, the mismatch negativity appears as a negative deflection at frontal electrodes and as a positive deflection at mastoid locations. Notice, however, that whereas the mismatch negativity was of identical amplitude in both groups at frontal electrodes, it was larger at mastoid locations for the persistent developmental stuttering group, particularly over the left hemisphere.

Discussion. Three distinctive features characterized our results: first, the similar MMN amplitudes in PDS and control subjects to simple sound contrasts, and their parallel increase with increasing deviance in both groups,[19,20] indicating that the perceptual analysis of simple sound features and their representation in neural traces within the auditory cortex was preserved in PDS; second, the enhanced left-mastoid MMN amplitude elicited to prototypic and nonprototypic phonetic contrasts in the PDS group vs controls; and third, the fact that the enhanced LM MMN amplitude correlated positively with speech disfluency as self-assessed by the study subjects. Because the MMN originates from supratemporal bilateral generators,[10,12-15] with the activity recorded at each mastoid electrode reflecting the activation of the ipsilateral auditory cortex,[12-17] our results indicate abnormal activation of the left supratemporal generator of the MMN. Moreover, because the supratemporal MMN generators purely reflect the perceptual neural representation of the auditory features that is violated by the incoming deviant sound,[10-12,16,17,24,25,27] our results strongly support abnormal speech sound representation within the auditory cortex of the left hemisphere. However, interpretation of these results must be cautious given that one of the working hypotheses in developmental stuttering involves atypical cerebral dominance[3,4,6,8,35,36] and that our PDS group included both right-handed and a few left-handed individuals. However, abnormalities encompassing the left posterior speech-related regions[3] and other brain regions,

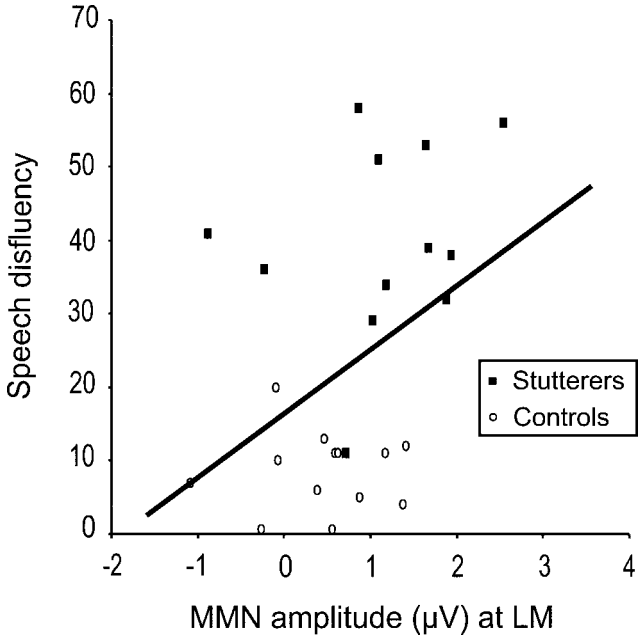

Figure 3. Self-assessed speech disfluency as a function of mismatch negativity (MMN) amplitude in the Phoneme condition. Notice that most persistent developmental stuttering subjects clustered to the high speech disfluency/large MMN amplitude quadrant, whereas control subjects clustered to the small MMN amplitude at left mastoid (LM) and low speech disfluency quadrant, resulting in a significant phonetic MMN amplitude and speech disfluency correlation.

such as the prefrontal and occipital lobes,[36] have been described in both left- and right-handed stuttering adults.

Taken together, all of the current findings reveal that stuttering adults have a specific auditory perceptual deficit, restricted to the left lateralized auditory processing of speech sounds that seems to underlie their speech production deficits. Previous anatomic PDS studies have provided evidence of an anomalous anatomy in perisylvian speech and language areas of the left cerebral cortex.[3,4] Moreover, PDS individuals show reduced left anterior and superior temporal phonologic activation and a deactivation of a verbal fluency circuit encompassing the left frontal and left temporal cortices.[5,6] These findings apparently contradict our current observation of an increased activation of the left supratemporal generator of MMN. However, the increased activity of the left supratemporal region was revealed here with an electrophysiologic method of high temporal resolution and was restricted to a narrow latency range expanding 100 to 200 milliseconds from stimulus onset. It is therefore possible that this phasic increase in activation might become override when measuring the activity of a large anomalous anatomic region[3,4] with neuroimaging methods of a large time constant, such as PET or fMRI. Nevertheless, when taken together with the previous findings, our current results suggest a functional relationship between the anomalous anatomy of speech-related circuits and their dysfunctional activation in PDS, by revealing a specific auditory perceptual deficit restricted to speech sound processing, in agreement with recent results.[9]

In contrast with other findings showing larger MMN to prototypic than to nonprototypic phonetic contrasts,[25,27] we obtained similar MMN amplitudes for both prototypic and nonprototypic phonetic contrasts in these stuttering adults. This suggests that PDS subjects have a similar anomalous processing of both native and nonnative speech sounds. According to recent theoretical proposals based on MMN,[18,24,25,27] speech perception relies on language-specific phonetic neural traces of the posterior part of the left auditory cortex, which represent the invariance of the acoustic input critical for phoneme discrimination. Therefore, our results indicate that adults with developmental stuttering have difficulties in discriminating nonnative from native sounds properly, suggesting abnormal auditory processing of all kind of speech-like signals. One hypothesis explaining this generalized speech processing deficit is that stuttering adults lack the ability for processing fast temporal changes of auditory signals. Indeed, speech sounds are characterized by a harmonically complex structure constantly changing over time and thus requiring continuous and rapid analysis. Hence, the ability to identify properly the speech components of sound depends on the ability to track rapidly changing acoustic information,[37-39] an ability of the left superior temporal cortex.[40,41] Therefore, based on the left perisylvian abnormalities found in PDS subjects,[3,4] their impairment for properly recognize speech-like sounds may result from a defect in processing fast temporal changes of auditory signals. In fact, difficulties in processing rapidly changing information have been reported in dyslexia,[42,43] and some dyslexic children show poor articulatory skills when speaking,[44] suggesting that an altered speech perception at some stage of language acquisition might be a high risk factor for developing a range of language disorders. Hence, deficits in speech perception in early ages should be considered as a critical issue in which diagnostic attention and future studies on language disorders should focus.

On a broader context, our results also suggest that the neural sound representation underlying MMN generation might be involved, in addition to the subjective perception of speech sounds,[10,18-20,24,25,27,29] to their production, as supported by the positive correlation between a deficient speech sound representation and the increasing severity of disfluency. This is in agreement with new integrated models on the functional anatomy of language highlighting an auditory–motor interface located in the posterior part of the sylvian fissure, at the boundary between the parietal and temporal lobes, referred to as Spt (sylvian–parietal–temporal).[41,45] Therefore, the results obtained in the current study suggest that, underneath speech production deficits, there must be at least some degree of speech perception impairment.

However, establishing which deficits arise from which is an issue that remains to be clarified in future studies. Speculating, we suggest that the language-specific neural traces in PDS are not normally developed in the left auditory cortex as a result of relying on an ongoing deficit in processing speech-like signals, so that at some stage of speech production, the auditory system might fail in retrieving the proper phonemic trace, i.e., in activating a neural perceptual model of the sound to be produced. This is consistent with the well-described effects of auditory feedback in alleviating the stuttering behavior,[1,4,9] in which external clues help to synchronize neural activity in auditory areas related to the speech sound in play. It is also consistent with our findings, where the altered MMN instead of being absent of reduced (as expected in clinical populations) was abnormally enlarged, suggesting an overexcited response of the auditory cortex to specific speech sounds. A large body of evidence indicates that the development of stimulus representation is accompanied by reductions in associated neural activity, resulting in more tuned neural populations to the features of the eliciting stimulus.[46] In this way, adults who stutter would have neural populations less tuned to the phonetic components of the auditory input, which in turn would result in deficient models for speech articulation.

The overall picture outlined above led us to propose that specific impairments in language perception must not be forgotten in speech disorders, so that such impairments might influence speech production. Despite that the current pathophysiologic hypotheses of stuttering are based on disturbed timing of activation within the speech production network,[4,7,47] our findings are in agreement with recent studies emphasizing anomalies in auditory perceptual processing in PDS.[7-9] In this way, our results open the door to the idea of stuttering rehabilitation by means of auditory perceptual programs in addition to production programs. Because deficits in speech processing seems now to be one of the features of stuttering, specialized training programs could be designed and implemented to remediate the auditory processing skills in individuals who stutter. As in a study showing that reading skills were improved in dyslexic children by using an audiovisual training without linguistic material,[48] new training methods could be designed, such as having stutterers accurately learn to discriminate speech sound elements, to lead to improvements in speech production.

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