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Speech preparation in adults with persistent developmental stuttering

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Abstract

Motor efference copy conveys movement information to sensory areas before and during vocalization. We hypothesized speech preparation would modulate auditory processing, via motor efference copy, differently in men who stutter (MWS) vs. fluent adults. Participants ($n=12/\text{group}$) had EEG recorded during a cue-target paradigm with two conditions: speech which allowed for speech preparation, while a control condition did not. Acoustic stimuli probed auditory responsiveness between the cue and target. MWS had longer vocal reaction times ($p<0.01$) when the cue-target differed (10% of trials), suggesting a difficulty of rapidly updating their speech plans. Acoustic probes elicited a negative slow wave indexing motor efference copy that was smaller in MWS vs. fluent adults ($p<0.03$). Current density responses in MWS showed smaller left prefrontal responses and auditory responses that were delayed and correlated to stuttering rate. Taken together, the results provide insight into the cortical mechanisms underlying atypical speech planning and dysfluencies in MWS.

Keywords

speech preparation; stuttering; motor efference copy; auditory event-related potentials

Introduction

Ordinary speech is the product of extraordinary cognitive processing. Speech preparation translates an abstract message into lexical and phonological representations, which in turn are transformed into motoric codes that guide the movement of vocal articulators (Indefrey & Levelt, 2004). A coalition of neural networks support speech planning and execution, with prominent interplay between perisylvian regions associated with speech motor control and auditory processing (Hickok, 2012; Indefrey, 2011).

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Given the rapidity and complexity of speech behavior it is not surprising that there are many opportunities for speech planning and production systems to become unstable, leading to moments of stuttering. Stuttering is characterized by disruptions in speech output, typically from repetitions, prolongations, and blocking (Bloodstein & Bernstein Ranter, 2008). The average age of stuttering onset is between ages 2 to 4 years, with the vast majority of children naturally recovering by age six (Bloodstein & Bernstein Ranter, 2008; Yairi & Ambrose, 2013). However, when stuttering continues through the teenage years and into adulthood the person is diagnosed with persistent developmental stuttering (Bloodstein & Bernstein Ranter, 2008).

Computational modeling suggests that precise temporal coordination between feedforward and feedback systems mediated by perisylvian speech regions are vital for speech fluency (Civier, Tasko, & Guenther, 2010; Guenther, 2006), and may contribute to developmental stuttering (Brown, Ingham, Ingham, Laird, & Fox, 2005; Max, Guenther, Ghosh, & Wallace, 2004). The feedforward system utilizes a motor efference copy to provide information about intended vocalizations to sensory areas, and provide online motor control (Crapse & Sommer, 2008; Sperry, 1950; Todorov, 2004; von Holst, 1954). The feedback system monitors auditory and somatosensory vocal output and identifies discrepancies between the intended and actual vocal output that need correction (Guenther, 2006; Perkell, 2012). Structural and functional MRI studies in people who stutter report differences in frontal premotor/motor and posterior temporal perisylvian speech areas that may, respectively, implement feedforward and feedback operations (Beal, Gracco, & Brettschneider, 2013; Beal, Gracco, Lafaille, & De Nil, 2007; Chang, Erickson, Ambrose, Hasegawa-Johnson, & Ludlow, 2008; Chang, Horwitz, Ostuni, Reynolds, & Ludlow, 2011; Foundas et al., 2004; Fox et al., 1996; Kikuchi, Ogata, Umesaki, & Yoshiura, 2011; Mock et al., 2012; Sommer, Koch, Paulus, Weiller, & Büchel, 2002). Also, a recent resting-state fMRI study suggests even in the absence of speech production demands, adults who stutter show anomalous resting-state networks within and between anterior and posterior perisylvian regions (Xuan et al., 2012).

Neurophysiological correlates of the feedforward system have been studied in humans using electrical (event-related potentials, ERPs) and magnetic field measures. It is well-established that ERPs or magnetic fields elicited by speech sounds are attenuated at ~100 ms latency (the N100/M100 component) when speaking relative to passive listening of recorded speech sounds (Curio, Neuloh, Numminen, Jousmäki, & Hari, 2000; Ford & Mathalon, 2004; Houde, Nagarajan, Sekihara, & Merzenich, 2002). Similar motor induced suppression is found in sensory responses to auditory and somatosensory stimuli during manual movements (Cohen & Starr, 1987; Schafer & Marcus, 1973), which suggests that N100 suppression to self-produced stimuli is not specific to vocal behaviors. The feedforward system has been hypothesized to be impaired in those who stutter (Brown et al., 2005; Civier, Bullock, Max, & Guenther, 2013; Civier et al., 2010; Max et al., 2004). However, during speech production in both children and adults who stutter motor induced suppression of the N100/M100 when speaking vs. passive listening is comparable to fluent controls (Beal et al., 2010, 2011; Liotti et al., 2010).

The above studies examined the effects of the feedforward system on auditory cortical activity in people who stutter during speech production. This project will examine feedforward influences during speech preparation in men who stutter (MWS), which could be particularly important given that over 90% of stuttering events occur on the initial sound/syllable of an utterance (Bloodstein & Bernstein Ranter, 2008; Sheehan, 1974). Our previous EEG study found that auditory processing is also subject to feedforward modulation before speaking, during the stage of speech preparation (Mock, Foundas, & Golob, 2011). In Mock et al. (2011) we used a delayed picture naming paradigm to (1) separate speech preparation from speech execution and (2) quantify auditory cortical responses by presenting auditory probes at multiple time points during speech preparation. The purpose of the acoustic probes was to assess auditory processing under conditions that encouraged vs. discouraged feedforward influences. This was done by comparing a speech condition that induced speech motor planning to a control condition where participants could not prepare a specific vocal response. The main behavioral result was that in the speech condition vocal reaction times to pictures were much faster when participants could prepare the appropriate vocal response to name the target. The auditory probes elicited ERPs that showed a long lasting negative shift (termed negative slow wave) that was larger in the speech vs. control condition and became greater as one got closer to speaking. The negative slow wave was suggested to index feedforward influences during speech preparation on auditory cortical processing.

We hypothesized that if feedforward processing is weaker in people who stutter (Brown et al., 2005; Civier et al., 2013; Max et al., 2004), then the auditory ERP negative slow wave would be smaller in MWS compared to fluent adults. Our paradigm can also examine the ability to update a speech motor program by comparing vocal reaction times to expected vs. unexpected targets (for details see Methods section below). Due to the MWS having anomalous networks within and between perisylvian regions (Chang et al., 2011; Xuan et al., 2012), we hypothesized even when speaking fluently, MWS will be slower than fluent adults at quickly updating to a different speech motor program.

Methods

Participants

Twelve MWS (age 34.8 ± 11.4 years, range 23–54 years, education 16.6 ± 2.9 years) were matched to 12 fluent men by age (± 2 years, avg. 33.7 ± 12.1 , range 23–55) and education (± 3 years, avg. 16.7 ± 2.7 years, range 12–20 years). All participants were right-handed, spoke English as their first and primary language, and reported normal neurological and psychiatric health. Hearing threshold tests (0.5–8.0 kHz) were in the normal range for all participants. For each MWS the number of stuttering-like dysfluencies (monosyllabic word repetitions, part-word repetitions, blocks and prolongations) within the first 300 syllables of a conversation and reading passage was quantified from a videotaped session. All stuttering-like dysfluencies were summed and divided by the total number of spoken syllables to obtain a percent of syllables stuttered. The percent of syllables stuttered for the conversation and reading passages were averaged to obtain a stuttering rate (mean = $9.8 \pm 1.3\%$, range = 3.5 to 19.5%). Stuttering rate was independently coded by the first author and a certified speech-

language pathologist, and was reliable (intraclass correlation coefficient = 0.89). Each participant signed a consent form, and all experimental procedures were performed in accordance with a protocol approved by the Tulane University Institutional Review Board that was consistent with the Declaration of Helsinki.

Experimental Design and Task

A schematic of the design and trials/condition are shown in Figure 1A. Participants sat in front of a computer monitor (distance = 100 cm) and viewed cue (letters 3 cm height) and target (picture 9×7 cm) stimuli in the center of the screen (500 ms duration). In the speech condition participants were instructed to use the cue word, which was one or two syllables, to get ready to verbally name the picture. Participants were asked to be fast but accurate, and to only use one word responses. The visual cue, in terms of meaning, matched the target picture on 90% of trials (termed “same trials”). For the remaining 10% of the trials the cue did not match the target, termed “different trials”. If the cue is used to prepare a speech motor program then vocal reaction times to the target should be faster on same vs. different trials. As expected, Mock et al. (2011) showed that reaction times were much faster on the same trials. This result showed that speech preparation occurred in this task, which is a prerequisite for a speech-related feedforward influence on auditory processing.

In the control condition all cues were “XXXX” so the participants could not prepare a specific vocal response but did have information on the timing of the upcoming target. On 90% of the control trials the participants did not name the target picture but instead passively viewed the stimuli. To control for attentiveness, on 10% of the control trials a visual prompt was given 1,000 ms after the target that instructed the participant to name the previous target (catch trials). Thus within the control condition, speech preparation to name a specific target would not occur between the cue and target when auditory responsiveness was being probed.

The impact of speech motor planning on auditory cortical activity was examined by measuring auditory ERPs elicited by sounds that were delivered between the cue and target. The interval between cue and target was 1.5 sec, with an inter-trial interval (target to next cue) of 2.5 sec. In each trial one auditory probe (~60 dB nHL, ~200 ms duration) was presented between the cue and target, at either an early (600 ms) or late (1200 ms) probe onset relative to cue onset (50% probability for early/late probe onset). The auditory probes were either a consonant-vowel or pure tone (1,000 Hz, 5 ms rise/fall time). In the speech condition the consonant-vowel auditory probes were subdivided into two classes (match, mismatch). The only difference between the match and mismatch auditory probes was their relationship to the cue on a given trial. The sound of the match probes corresponded to the first consonant-vowel of the cue word, while the mismatch probes did not. Each sound type (match, mismatch, tone) was randomly presented in 20 trials/block (33% probability). A total of 60 different words and pictures (Snodgrass & Vanderwart, 1980) were used in each block, with 6 blocks/condition for a total of 360 trials/condition. All words began with a consonant-vowel, each block only had trials for one condition (speech or control), and the order of conditions was counterbalanced across participants.

Electrophysiological recordings

Experiments were conducted in a sound-attenuated, electrically shielded room. Acoustic stimuli were presented binaurally with etymotic headphones, and a microphone was placed 4 cm from the participant's mouth to record vocal responses. An electrode cap with 64 Ag / AgCl electrodes was placed on the scalp (reference between Cz and CPz, impedances < 10 k Ω , 500 Hz sampling rate, DC-100 Hz). Four electrodes were also used to monitor eye movements, one above and one below the left eye and one lateral to each eye. The electrodes were re-referenced in a linked mastoid configuration offline, and EEG was corrected for DC drift and eye blink artifacts (Gratton, Coles, & Donchin, 1983). Acoustic ERPs were epoched into 1,200 ms segments (–200 to 1000 ms relative to stimulus onset). All epochs were visually inspected for additional artifacts by looking for the abrupt onset of noise, such as movement artifacts, before being included in the ERP averages.

Data analysis

Vocal reaction times and ERPs—Vocal reaction times to pictures were manually defined by comparing the time of stimulus onset to the beginning of the vocal response waveform (2 ms resolution). All vocal responses that were errors, dysfluent, or had an outlier reaction time (> 3 SD) were excluded. All vocal reaction times were quantified by the first author. A subset ($n=4$, 2/group) was independently coded by a lab assistant and was shown to be reliable (intraclass correlation coefficient = 0.94). Auditory ERPs were digitally filtered (band pass 0.1–16 Hz, 12 dB down) to attenuate slow shifts, and baselined from –100 ms to 0 ms relative to stimulus onset. An auditory slow wave potential was quantified by measuring the mean amplitude from 100–400 ms after probe onset at fronto-central electrode sites. Peak amplitude and latency of the N100 and P200 were also quantified.

Current density analysis—Standardized low-resolution brain electromagnetic tomography (sLORETA, Pascual-Marqui, 2002) was used to estimate current density in response to the auditory probes (Curry 6.0, Neuroscan Compumedics, Charlotte, NC). The Montreal Neurological Institute standardized MRI template was used to construct a three-compartment boundary element model (skin, skull, cortex) using standard homogeneous conductivities. Current density (CD) responses to probes were quantified from 0 to 400 ms after sound onset in each participant and normalized across all data points using the formula $(CD - \text{Minimum CD}) / (\text{Maximum CD} - \text{Minimum CD})$. The four cortical locations with the largest cortical current density responses to auditory probes were selected; 3 locations in the superior temporal gyrus (anterior, middle, posterior) that had peaks ~ 100 ms and ~ 200 ms (Figure 4), and 1 location in ventral prefrontal cortex (Figure 6). Volume conduction from a discrete cortical source occurs almost instantaneously (Nunez & Srinivasan, 2006), therefore the unique ventral prefrontal current density response (one peak around 130 ms) indicates a cortical source distinct from the ~ 100 ms auditory cortex response. Exploratory analysis showed that the supplementary motor area site had identical current density responses to the auditory cortex (peaks ~ 100 and ~ 200 ms), which was not analyzed because it likely reflected volume conduction from temporal lobe sources. The current density response within the temporal-parietal junction was also investigated, but was small and unreliable as compared to the auditory and ventral prefrontal cortices.

Statistical analysis

Behavioral, ERP, and current density analyses used repeated measures analysis of variance (ANOVA; $p < .05$), with the Greenhouse-Geisser correction for any violations of sphericity. Pearson's correlation coefficient (r) was used to test the correlation between stuttering rate and specific neurophysiological components. The group of MWS had a mean stuttering rate of 9.8%, which is comparable in severity to other studies (Bloodstein & Bernstein Ranter, 2008; Bloodstein, 1944; Ingham, Grafton, Bothe, & Ingham, 2012).

The behavioral, ERP (peak and window measures), and current density analyses combined the sound types (match, mismatch, and tone) within each auditory probe onset time because preliminary analyses showed the group differences were consistent across sound types. Behavioral data in the speech condition (vocal reaction time, accuracy) were analyzed with a 2 (group: fluent, MWS) $\times$ picture-word agreement (same, different) $\times$ 2 (probe onset: early, late) ANOVA tests. Scalp ERP measures at midline sites included within-subject variables of condition (speech, control) and probe onset, and group was a between-subjects factor. Hemispheric analyses for ERP and current density measures included within-subject variables of condition, probe onset, and hemisphere (left, right), with group as the between-subjects factor.

Auditory ERPs were analyzed at fronto-central electrode sites (FC5 (left), FCz (midline), FC6 (right)). The main ERP difference between conditions was a long lasting slow wave in the speech vs. control condition that was quantified by the mean amplitude between 100–400 ms. Current density data within the auditory cortex (superior temporal gyrus) were quantified by peak measures (100 ms peak, 200 ms peak). Prefrontal current density peaks were quantified by using a 100–160 ms window.

Results

Behavior

A 2 (group) $\times$ 2 (picture-word agreement) $\times$ 2 (probe onset) ANOVA test showed a main effect of picture-word agreement ($F_{(1,22)} = 280.8$, $p < 0.0001$), with much longer vocal reaction times on different relative to same trials (Figure 1B). The slower responses on trials where the cue and picture did not agree served as a validity check that both groups used the cue to prepare their responses. There was a group $\times$ picture-word agreement interaction ($F_{(1,22)} = 11.7$, $p = 0.002$). Compared to fluent men the MWS had comparable reaction times on the same trials but longer reaction times on the different trials ($p = 0.048$). Both groups were extremely accurate and had few excluded trials. Out of 4,320 speech trials/group, the MWS had 282 excluded responses (212 dysfluent, 40 naming errors, 30 > 3 SD) while the fluent controls had 73 excluded responses (15 naming errors, 58 > 3 SD). During the experimental trials, three out of the 12 MWS did not register any stuttering events, and another 4 out of 12 MWS had < 6 total stuttering events (out of 360 speech trials/participant).

Auditory event-related potentials

Auditory ERPs were analyzed using a 2 (group) $\times$ 2 (condition) $\times$ 2 (probe onset) ANOVA test at midline (FCz) followed by a separate analysis with hemisphere as a factor (FC5: left, FC6: right).

ERP 100–400 ms window

The slow wave amplitude (FCz: 100–400 ms window) showed an effect of condition ($F_{(1,22)} = 28.7, p = 0.001$; speech < control) that was qualified by a group $\times$ condition interaction ($F_{(1,22)} = 5.4, p = 0.029$). The fluent men had a larger difference between conditions than MWS (fluent = $-1.3 \pm 0.3\mu\text{V}$, MWS = $-0.5 \pm 0.2\mu\text{V}$), with the most pronounced group differences in the control condition. See Figure 2. Replicating our previous finding in young adults (Mock et al., 2011), there was a condition $\times$ probe onset interaction ($F_{(1,22)} = 7.9, p = 0.010$) where the difference between conditions was greater for the late vs. early probe onset. The difference between conditions (speech – control: negative slow wave) was not correlated to stuttering rate.

Lateral electrode sites used to examine hemisphere effects found a condition $\times$ probe onset $\times$ hemisphere interaction ($F_{(1,22)} = 8.3, p = 0.009$). Across groups the early auditory probes showed a left lateralized condition effect (left: $p = 0.01$, right: $p = 0.37$) while the late auditory probes showed a bilateral condition effect (left: $p = 0.0001$, right: $p = 0.001$); replicating our previous findings in young fluent adults (Mock et al., 2011).

ERP N100 peak amplitude and latency

Main effect of probe onset ($F_S = 13.8, p = 0.001$; early = $-6.8 \pm 0.6\mu\text{V}$, late = $-8.1 \pm 0.5\mu\text{V}$) was found for the N100 peak amplitude. No group effects were found. At lateral electrodes no group $\times$ hemisphere interactions were found.

For the N100 peak latency, no effects of condition were found and the effect of group did not reach significance ($p = 0.13$; MWS = 117 ± 2 ms, fluent = 112 ± 2 ms). See Figure 3. There were no significant group $\times$ hemisphere interactions at lateral electrode sites.

ERP P200 peak amplitude and latency

A main effect of condition ($F_{(1,22)} = 21.8, p = 0.0001$; speech < control) was found for P200 peak amplitude. There was a trend towards a condition $\times$ probe onset interaction ($p = 0.054$). No Group effects were found. At lateral electrodes, a condition $\times$ hemisphere interaction was found ($F_{(1,22)} = 17.0, p = 0.0004$) due to the P200 amplitude being smaller in the left vs. right hemisphere within the speech ($p = 0.007$) but not control condition ($p = 0.5$). No group $\times$ hemisphere interactions were found.

For the P200 peak latency there was a main effect of group; the MWS had longer peak latencies than fluent controls ($F_{(1,22)} = 11.1, p = 0.003$). See Figure 3. There was also a main effect of condition ($F_{(1,22)} = 14.4, p = 0.001$) with peak latencies being shorter in the speech condition (control = 212 ± 4 ms, speech = 207 ± 4 ms). At lateral electrodes, no group $\times$ hemisphere interactions were found.

In summary, auditory probes delivered between the cue and target elicited a negative slow wave in the speech relative to control condition. However, the slow wave difference between conditions was about half that in MWS compared to fluent controls; supporting the initial hypothesis of aberrant auditory modulation via a motor efference copy in MWS. Also the MWS appear to show anomalous auditory processing by showing a delayed P200 peak latency across conditions.

Current Density: Auditory and ventral prefrontal cortex

The probes elicited auditory cortex current density peaks at ~100 and ~200 ms, which parallels the timing of the N100 and P200 auditory ERP components (Figure 4). One peak with a source in ventral prefrontal cortex was also examined (~130 ms, See Figure 6). Current density was analyzed for each measure (auditory peaks ~100 and ~200 ms; PFC window 100–160ms) using factors of group, condition, probe onset and hemisphere.

Auditory cortex - 100 ms current density peak

No overall group differences in amplitude or latency were found. However, the 100 ms current density peak was positively correlated stuttering rate in the speech ($r = .70$, $p = 0.012$), but not control condition ($r = .22$, $p = 0.5$). There was also an effect of probe onset ($F_{(1,22)} = 14.4$, $p = 0.001$) due to larger auditory current density responses at the late vs. early onset (early = 0.17 ± 0.02 , late = 0.24 ± 0.03).

Auditory cortex - 200 ms current density peak

A condition $\times$ probe onset interaction was found ($F_{(1,22)} = 14.0$, $p = 0.001$) (see Figure 4 and 5B). The interaction was due to both groups having a condition effect (speech < control) at the late ($p < 0.001$) but not early probe onset time, which replicated a previous finding (Mock et al., 2011). Stuttering rate was not correlated to the 200 ms current density peak response.

Analysis of the 200 ms peak latencies revealed a group effect ($F_{(1,22)} = 26.2$, $p < 0.001$), with longer latencies in MWS (213 ± 3 ms) vs. fluent controls (194 ± 3 ms). See Figure 4 and 5C. There were also effects of condition ($F_{(1,22)} = 5.2$, $p = 0.032$; speech < control) and hemisphere ($F_{(1,22)} = 17.4$, $p = 0.001$; left > right hemisphere).

Prefrontal cortex current density peak (100 – 160 ms)

The ventral prefrontal sites showed a group $\times$ hemisphere interaction ($F_{(1,22)} = 4.5$, $p = 0.045$). The MWS had a smaller left hemisphere current density amplitude ($p = 0.02$) while the right hemisphere response was comparable between groups. See Figure 6A, B. Across groups, an effect of condition ($F_{(1,22)} = 4.7$, $p = 0.042$; speech < control) was found but superseded by a condition $\times$ probe onset interaction ($F_{(1,22)} = 4.9$, $p = 0.037$). The late probes showed a condition effect (speech < control, $p = 0.002$) while the early probe did not ($p = 0.96$). Stuttering rate was not correlated to the prefrontal cortex current density measure.

Discussion

This study tested the hypothesis that the influence of speech motor preparation on auditory responsiveness is diminished in MWS. The hypothesis was supported by the finding that a negative slow wave (speech – control) previously associated with motor efference copy was smaller in MWS relative to fluent adults. A corollary was that MWS would be slower at updating to a new speech motor plan. This prediction was supported by the finding that vocal reaction times were comparable among groups for expected targets, but MWS had longer vocal reaction times relative to controls when the cue and target did not match. The current density measures in MWS differed from fluent controls in both conditions, with a smaller left ventral prefrontal cortex response (~130 ms) and a delayed bilateral auditory cortical response (~200 ms). Also, the auditory cortex current density responses ~100 ms correlated to stuttering rate in the speech, but not control condition. Overall, these results point to differences in cortical activity in MWS that are associated with speech preparation, and occur well before the onset of speech behavior.

Group differences in vocal reaction times

Vocal reaction times were longer in MWS vs. fluent adults only when the target did not match the cue, which suggests updating to a new speech motor plan takes longer in MWS. To our knowledge this is the first study to test how rapidly individuals who stutter can override a speech motor plan and assemble a new one. Comparable vocal reaction times among groups were observed when the cue and target matched. This shows MWS were not slower overall at making verbal responses in this paradigm and neurophysiological differences among groups are not secondary to performance differences. Past studies have found longer manual and vocal reaction times to uncued visual and auditory stimuli in people who stutter relative to fluent controls (for review see Bloodstein and Bernstein-Ranter, 2008). The current design appears to have encouraged fluent responses by only requiring one word responses and/or by correctly cueing in advance on most trials the target word to be spoken. Only 4.9% of speech trials in the MWS were rejected due to dysfluent responses whereas the average assessed pre-experiment stuttering rate was 9.8%.

Slow wave ERPs to probes and motor efference copy

Both the fluent adults and the MWS replicated our previous study in young adults of a fronto-central negative shift in amplitude (termed negative slow wave) to probes in the speech vs. control condition (Mock et al., 2011). The slow wave amplitude lasted from ~100–400 ms after probe onset, and was left lateralized for the early probes and bilateral for the late probes. The negative slow wave indexes the modulation of auditory cortical responses to sounds when speech planning is typically present vs. absent, and is thought to reflect the influence of motor efference copy on auditory processing (Mock et al., 2011). The main new finding was the negative slow wave amplitude in MWS was about half the size of fluent men; supporting the idea that impaired motor efference copy may be one mechanism for dysfluencies in adults who stutter (Brown et al., 2005; Max et al., 2004). The group difference was not directly reflective of fluency abilities as the negative slow wave amplitude (speech - control) was not correlated to stuttering rate.

Although the main point of interest is the contrast between speech and control conditions, the group differences in amplitude between 100 and 400 ms were driven by the control condition. The MWS had more negative amplitudes in the control condition than fluent men, and therefore had less of a difference relative to the speech condition. Although speculative, this suggests that it may be worthwhile to test the idea that in MWS motor efference copy may be engaged under conditions where it is unnecessary.

The present results agree with other studies that observed group differences in auditory ERPs to tones and passive listening to speech at latencies between ~200 and 400 ms, which is within the time period of our slow wave measures (Hampton & Weber-Fox, 2008; Liotti et al., 2010). Convergent evidence is also provided for functional imaging studies showing anomalous deactivation patterns within auditory cortical areas in adults who stutter (Braun et al., 1997; Fox et al., 1996). It should be noted the term “negative slow wave” is a very general descriptor used in ERP experiments to describe relatively prolonged negative amplitude shifts. The functional interpretation of a given slow wave depends on the specific cognitive processes and task that are being studied. Various negative slow waves have been identified, including the readiness potential before voluntary movement (Brunia, 1988), the contingent negative variation (Walter, Cooper, Aldridge, McCallum, & Winter, 1964), slow waves related to attention (Okita, 1981), and slow waves following the P300 in oddball and recognition memory tasks (Mecklinger, Kramer, & Strayer, 1992). The negative slow wave observed in the present study has similar properties (i.e. latency range and polarity) to a slow wave seen in auditory selective attention tasks (Naatanen, Michie, & Näätänen, 1979; Okita, 1981). This supports the possibility that topdown mechanisms may be influencing auditory cortical processing similarly in both speech preparation and auditory selective attention tasks. Indeed, the importance of preparation and action plays a major role in several theories of attention (Allport, 1989; Corbetta & Shulman, 2002; Rizzolatti, Riggio, Dascola, & Umiltà, 1987).

P200 latency ERP

The current study found that P200 latencies were delayed in MWS across conditions. We also found the P200 latencies to be shorter in the speech vs. control condition, showing that preparing to speak influenced latencies similarly between groups. Even though multiple studies have recorded evoked responses in people who stutter, very few have found latency differences. One previous study found adults who stutter to have (1) earlier M100 latencies in the right vs. left hemisphere during the processing of speech feedback and (2) delayed M50 and M100 peak latencies during passive listening (Beal et al., 2010). The author interpreted these group differences in latency as (1) compensation for deficits in a left hemisphere speech networks and (2) representing a fundamental difference in how adults who stutter process auditory stimuli relative to fluent adults. The delayed P200 peak latencies support the notion that even in the absence of speech preparation, adults who stutter process auditory stimuli differently than fluent adults.

Auditory cortex current density responses

The auditory cortex current density response to probes both replicated results from the scalp ERPs and uncovering effects not observed within the scalp ERPs. For instance, both

measures showed (1) larger 100 ms amplitudes too late vs. early onset probes, (2) shorter 200 ms latencies in the speech vs. control condition, (3) smaller 200 ms amplitudes in the speech vs. control condition right before target onset, and (4) longer 200 ms latencies in MWS vs. fluent controls. However, the auditory cortex current density response did reveal a difference not seen in the scalp ERPs. The 100 ms current density peak from the auditory cortex positively correlated to stuttering rate in the speech condition. This finding suggests that understanding the neuronal processing within the cue-target paradigm might provide insight into factors relating to stuttering severity. We note that the 100 ms current density peak is an estimate of supratemporal plane and lateral auditory cortex activity that is based on a biophysical model. However, convergent evidence for supratemporal plane generators with a pronounced peak at ~100 is provided by intracranial (Godey, Schwartz, de Graaf, Chauvel, & Liégeois-Chauvel, 2001) and magnetoencephalographic (Pantev et al., 1995; Zouridakis, Simos, & Papanicolaou, 1998) recordings.

Multiple top-down cognitive factors have been shown to influence the N100 such as attention (Bennett, Golob, & Starr, 2004; Hillyard, Hink, Schwent, & Picton, 1973), short-term memory (Conley, Michalewski, & Starr, 1999; Golob & Starr, 2004), and motor efference copy when speaking (Heinks-Maldonado, Mathalon, Gray, & Ford, 2005; Houde et al., 2002). Also previous studies found hemispheric differences around 100 ms within the auditory cortex of adults who stutter (Beal et al., 2010; Liotti et al., 2010; Salmelin R, Schnitzler A, Schmitz F, Jancke L, Witte OW et al., 1998), whereas the 100 ms group differences found in the current study appear to be bilateral.

Across groups the 200 ms auditory cortex current density peak was attenuated in the speech vs. control conditions for late probes, replicating our previous findings (Mock et al., 2011). This result is similar to work in both adults and children who stutter that found normal attenuation of auditory cortical responses when speaking (Beal et al., 2010, 2011; Liotti et al., 2010). Consequently, the negative slow wave group difference seen in the scalp ERPs did not reach significance within the auditory cortex current density response, suggesting the slow wave group difference recorded from the scalp is generated by multiple cortical areas. The condition differences from the auditory cortex current density response contribute only to the early portion of the negative slow wave recorded at the scalp. This is expected because at any given time scalp electrodes are measuring the sum of synchronized activity from multiple cortical regions (Jackson & Bolger, 2014).

The importance of the auditory system in stuttering is supported by structural and functional neuroimaging studies (Beal et al., 2007; Braun et al., 1997; Foundas et al., 2004; Fox et al., 1996), and the positive impact of acoustic interventions on speech fluency (Foundas, Mock, Corey, Golob, & Conture, 2013; Lincoln, Packman, & Onslow, 2006). Previous experiments in people who stutter have examined auditory processing while covertly reading (Salmelin R, Schnitzler A, Schmitz F, Jancke L, Witte OW et al., 1998), in response to ones' own voice when speaking (Beal et al., 2010, 2011; Liotti et al., 2010) and during picture recognition and delayed production (Maxfield, Huffman, Frisch, & Hinckley, 2010; Maxfield, Pizon-Moore, Frisch, & Constantine, 2012). The studies by Maxfield et al. (2010, 2012) found that long latency (> 400 ms) components related to semantic and phonological activation differed between adults who stutter and fluent controls. Taken together with the

above observations, our new results indicate that auditory cortical processing can distinguish fluent men from those who stutter, and support the notion that atypical auditory processing is a prominent feature in stuttering.

Ventral prefrontal cortex current density response

There was a prominent bilateral current density response to probes that was estimated to be generated in the ventral prefrontal cortex at a latency of ~130 ms. Regardless of condition or probe onset time the left hemisphere response was smaller in MWS relative to controls. We are interpreting the ~130 ms prefrontal response as distinctly different from the ~100 ms auditory cortex response. Volume conduction from a discrete cortical current source occurs almost instantaneously (Nunez & Srinivasan, 2006), which shows the ~130 ms prefrontal peak is not mirroring the ~100 ms auditory response. However, it is a possibility these two regions are directly modulating each other around this time range as single-unit recordings in primates have shown that neurons in dorsal and ventral prefrontal cortex responding to auditory stimuli at a comparable latency (Vaadia, Benson, Hienz, & Goldstein, 1986). The smaller left ventral prefrontal response in MWS might be related to structural and functional anomalies found within the same region in people who stutter (Beal et al., 2013; Chang et al., 2008, 2011; Mock et al., 2012; Sommer et al., 2002; Watkins, Smith, Davis, & Howell, 2008). The current density analysis modeling prefrontal and auditory cortices suggests that auditory processing in bilateral auditory and left prefrontal networks differs in adults who stutter relative to fluent speakers.

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Highlights

- Auditory responsiveness during speech preparation is different in men who stutter.
- Men who stutter are slower than fluent adults at updating a speech motor plan.
- Men who stutter have atypical left prefrontal and bilateral auditory responses.

Figure 1. (A) A schematic depicting the cue – target paradigm by condition (control, speech) and probe onset (early, late). (B) Vocal reaction times as a function of picture-word agreement (same, different) separated by group (MWS, fluent). Picture-word agreement indexes whether the visual cue and target stimuli matched (in terms of meaning), which occurred in 90% of the speech trials (termed “same trials”). In 10% of the speech trials, the meaning of the cue and target did not match (termed “different trials”).

Figure 2.

(A) Comparison of auditory ERPs from electrode FCz separated by condition (speech, control), probe onset (early, late) and group (MWS, fluent). (B) Topographic representations of the auditory ERP amplitudes from 100–400 ms post auditory onset collapsed across the early and late probes separated by condition and group. Also shown is the topographic distribution of the negative slow wave (difference between conditions) as seen under Speech - Control.

Figure 3. (A) Comparison of auditory ERPs averaged across condition and probe onset but separated by group (fluent, MWS). (B) Bar graph showing the MWS had longer P200 peak latencies than fluent adults.

Figure 4.

Time plot of normalized current density responses within the auditory cortex from 0 to 400 ms post auditory onset for each condition (control, speech) and separated by group (fluent, MWS). ● = location where auditory cortex current density responses are shown.

Figure 5.

Normalized current density responses within the auditory cortex represented by: (A) Scatterplot showing the 100 ms response to auditory probes within the speech condition was positively correlated to stuttering rate. (B) Bar graph collapsed across groups showing the attenuated 200 ms response to the late auditory probes when preparing to speak. (C) Bar graph showing the MWS had longer 200 ms latencies than fluent men.

Figure 6.

(A) Time plot of normalized current density responses within the prefrontal cortex from 0 to 400 ms post auditory onset separated by group (fluent, MWS) and averaged across condition and probe onset. (B) Bar graph showing the MWS had smaller left prefrontal current density responses from 100–160 ms post auditory onset. ● = location where prefrontal current density response was measured.