Assisted and unassisted recession of functional anomalies associated with dysprosody in adults who stutter


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A B S T R A C T

Purpose: Speech in persons who stutter (PWS) is associated with disturbed prosody (speech melody and intonation), which may impact communication. The neural correlates of PWS’ altered prosody during speaking are not known, neither is how a speech-restructuring therapy affects prosody at both a behavioral and a cerebral level.

Methods: In this fMRI study, we explored group differences in brain activation associated with the production of different kinds of prosody in 13 male adults who stutter (AWS) before, directly after, and at least 1 year after an effective intensive fluency-shaping treatment, in 13 typically fluent-speaking control participants (CP), and in 13 males who had spontaneously recovered from stuttering during adulthood (RAWS), while sentences were read aloud with ‘neutral’, instructed emotional (happy), and linguistically driven (questioning) prosody. These activations were related to speech production acoustics.

Results: During pre-treatment prosody generation, the pars orbitals of the left inferior frontal gyrus and the left anterior insula were activated less in AWS than in CP. The degree of hypoactivation correlated with acoustic measures of dysprosody. Paralleling the near-normalization of free speech melody following fluency-shaping therapy, AWS normalized the inferior frontal hypo-activation, sooner after treatment for generating emotional than linguistic prosody. Unassisted recovery was associated with an additional recruitment of cerebellar resources.

Conclusions: Fluency shaping therapy may restructure prosody, which approaches that of typically fluent-speaking people. Such a process may benefit from additional training of instructed emotional and linguistic prosody by inducing plasticity in the inferior frontal region which has developed abnormally during childhood in PWS.

1. INTRODUCTION

Prosody – the melody and intonation of speech – is an important carrier of human communication. It is a constituent of both
language perception and production that permits the emission and perception of linguistic and emotional information on contrast, focus, or other elements of language that may not be encoded by grammar or vocabulary. Persons interpret and respond to other persons’ speech prosody and produce adequate prosodic cues themselves. Prosody is the most important and ontogenetically earliest processed feature of language development emerging prenatally. It provides access to early phoneme discrimination and phonotactic learning (Friederici, 2005).

Prosody is characterized by several suprasegmental acoustic voice and language parameters, such as the modulation of fundamental frequency; the rhythm, stress, and rate of speech; the sound or syllable duration, intensity, formant frequencies; and pauses between different phrases (intonational phrase boundaries). Beyond isolated acoustic parameters, voice-specific information plays an important role in communication. Prosody details depend on the specific phonological, grammatical, and tonal characteristics of a language but also on social, cultural, status-, gender-, and age-related circumstances.

Speech prosody may be sub-classified into emotional (or affective) and linguistic prosody. Processing of emotional information, for example of happiness, irony, sadness, fear, anger, or sarcasm, may be conveyed by various means of communication, such as propositional content, speech intonation, facial expression, and gestures (Ethofer et al., 2006). Both emitting and perceiving emotional information is central to social communication and evolutionarily relevant for immediate reactions. The processing of linguistic prosodic information enables a listener to decide whether an utterance is a statement, a question, or a command. It requires integrated processing of linguistic and prosodic aspects of speech, such as syntax-prosody mapping and constant adaptation to semantic information, to ensure disambiguity (Anderson & Carlson, 2010).

Dysprosodic or apraxic speech production occurs in a variety of neurological and psychiatric disorders, such as autism, schizophrenia, cortical and subcortical brain damage, Parkinson’s disease, cerebellar ataxia, post-traumatic stress disorder, multiple sclerosis, Alzheimer disease, alcohol abuse, fetal alcohol-exposure (Arnold et al., 2013; Casper et al., 2007; Hesling et al., 2010; Monnot et al., 2002; Ross & Monnot, 2011; Paulmann, Pell, & Kotz, 2009; Paulmann, Seifert, & Kotz, 2010; Paulmann & Pell, 2010; Rusz, Cmejla, Ruzickova, & Ruzicka, 2011; Wymer, Lindman, & Booksh, 2002), and in family members with variants of FOXP2 (Shriberg et al., 2006). Speech in persons who stutter (PWS) is also dysprosodic (Jäncke, Bauer, & Kalveram, 1996; Packman, Onslow, Richard, & Van Doorn, 1996). PWS speak with a flattened speech melody; even during stuttering-free speech PWS produce a significantly smaller range of their fundamental frequency (F0) than persons who do not stutter (PWNS) (Bossardt, Sappok, Knipschild, & Hölscher, 1997; Healey, 1982). In addition, speech rhythm parameters are altered in the speech of PWS, even in fluent utterances (Maruthy, Venugopal, & Parakh, 2016). PWS have been reported to experience difficulties in placing sentence accent correctly; their stuttering episodes are located mainly on stressed syllables, a fixed timing pattern of speech enhances their fluency, and the intervals between stressed syllables are more variable in their speech, even in symptom-free passages, than in the speech of PWNS (Bergmann, 1986).

Because of the variability of stuttering symptoms with multifaceted combinations of syllable repetitions, prolongations, blocks, circumventions of utterances, or stressed pauses, the disturbance of prosody is highly variable and not easy to parameterize. Nevertheless, an effective stuttering therapy, in particular if it is aimed at speech fluency, should normalize the disturbed speech prosody. Some stuttering treatments, for example speech restructuring approaches such as fluency shaping (Neumann et al., 2016), deal explicitly with prosodic tools, for example, by practicing soft voice onsets or speech bows, thus impacting speech melody. Other treatments, with their focus on speech naturalness, for example stuttering modification (Van Riper, 1973), imply that prosody normalizes with the reduction of the frequency or severity of disfluencies.

Producing emotional and linguistic prosody activates the bilateral inferior frontal gyrus, anterior insulae, and large parts of the temporal cortex, together with the striatum and cerebellum (Aziz-Zadeh, Sheng, & Gheytenchi, 2010; Pichon & Kell, 2013). Interestingly, most of these regions are also structurally or functionally altered in PWS compared with non-stuttering control participants (CP) (for example Beal et al., 2010; Beal, Gracco, Lafaille, & De Nil, 2007; Brown, Ingham, Ingham, Laird, & Fox, 2005; Chang, Erickson, Ambrose, Hasegawa-Johnson, & Ludlow, 2008; Cykowski et al., 2007; De Nil & Kroll, 2001; Giraud et al., 2008; Kell et al., 2009; Neumann et al., 2005; Watkins, Smith, Davis, & Howell, 2008). Prosody production in PWS has not yet been addressed with functional neuroimaging. Here our goal was to identify neural correlates of disturbed speech melody, as one aspect of dysprosody, in untreated adults who stutter (AWS) during the generation of instructed emotional and linguistic prosody. Additionally, we investigated whether a fluency-shaping therapy, consisting of an intensive computer-assisted treatment and two to three refresher weekends (for a description see Euler, Wolff von Gudenberg, Jung, & Neumann, 2009), modified intonation. We studied its associated changes in brain activity directly after a three-week in-patient intensive treatment and at least one year later.

The treatment modifies prosody during neutral speech by an initial reduction of speech tempo, by soft voice onsets, continuous phonation, and smooth bows between utterances. This speech-restructuring technique produces an initially unusual and new speech pattern which prevents the occurrence of disfluencies. Soft voice onsets are trained as raising intonation and continuous phonation is exercised by speech bows linking utterances softly to each other. Hence, speech melody is modulated constantly and voluntarily. Patients control these modulations by the computer feedback they get from the envelopes of both the aimed and the realized acoustic speech signals in the time domain by using templates. These changes of speech pattern initially impinge on emotional prosody by a voluntarily changed intonation that is not affect-related. Later on, a more natural sounding speech is targeted by combining both the emotional aspects of speech and the partially automatized new speech pattern. Furthermore, linguistic prosody is also altered by the treatment because the initial artificially-sounding speaking manner is paralleled by unusual phrase boundaries and a voluntarily falling and raising intonation, which in later treatment phases have to be merged with the linguistic speech structure. The treatment thus acts on prosodic speech properties, particularly on speech melody, even if it does not explicitly focus on instructed emotional or linguistic prosody, the facets that are studied here.

Speech rhythm and sound duration are other prosodic aspects that undergo changes during the treatment, in particular by slowing
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