



Identification of neural structures involved in stuttering using vibrotactile feedback

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ABSTRACT

Feedback delivered over auditory and vibratory afferent pathways has different effects on the fluency of people who stutter (PWS). These features were exploited to investigate the neural structures involved in stuttering. The speech signal vibrated locations on the body (vibrotactile feedback, VTF). Eleven PWS read passages under VTF and control (no-VTF) conditions. All combinations of vibration amplitude, synchronous or delayed VTF and vibrator position (hand, sternum or forehead) were presented. Control conditions were performed at the beginning, middle and end of test sessions. Stuttering rate, but not speaking rate, differed between the control and VTF conditions. Notably, speaking rate did not change between when VTF was delayed versus when it was synchronous in contrast with what happens with auditory feedback. This showed that cerebellar mechanisms, which are affected when auditory feedback is delayed, were not implicated in the fluency-enhancing effects of VTF, suggesting that there is a second fluency-enhancing mechanism.

1. Introduction

Stuttering interrupts the forward flow of speech (Howell, 2010). Meta-analyses have identified three replicable and distinctive anomalous neural patterns that are associated with stuttering (Belyk, Kraft & Brown, 2014; Brown, Ingham, Ingham, Laird & Fox, 2005; Budde, Barron & Fox, 2014): (1) activity in auditory cortex is reduced; there is over-activity in (2) the right frontal operculum and/or anterior insula; and (3) the cerebellar vermis. Stimulating these affected brain regions could have an impact on stuttering (Howell & Lu, 2016). One way of doing this is to deliver exteroceptive sensory inputs that transmit to the regions where activity is anomalous and determine what effects these modulations have on the fluency of PWS. Applying this logic, auditory stimulation with metronome clicks (Howell & El-Yaniv, 1987) or masking noises (Cherry & Sayers, 1956) ameliorated stuttering. Presenting a changed version of the speaker's own voice (altered auditory feedback, AAF) by delaying (DAF) it or altering its spectral content (FSF) also affected the fluency of PWS (Howell, El-Yaniv & Powell, 1987). These improvements under AAF have been replicated and extended in research (for reviews: Howell, 2004; Kalinowski & Saltuklaroglu, 2006) and clinical settings (for review: Lincoln, Packman, & Onslow, 2006). A prosthetic device that delivers DAF and FSF whilst PWS speak is available (Stuart et al., 2003).

A related issues concerns whether stuttering changes when non-auditory stimuli are presented to PWS whilst they speak. These inputs

employ transmission pathways that by-pass or transmit over additional afferent pathways to those involved in audition. These properties provide a way of determining the role that the included and excluded neural structures play in stuttering. The most extensively investigated non-auditory modality is vibration. In one procedure, the speaker's vocal output drives vibration transducers attached to the body (vibrotactile feedback, VTF). VTF and AAF are similar in that they both: (1) change speech feedback from its normal state; and (2) improve the fluency of PWS (Kuniszzyk-Jozkowiak, Smolka & Adamczyk, 1996; Snyder, Blanchet, Waddell, & Ivy, 2009; Waddell, Goggans & Snyder, 2012). VTF and AAF are transmitted over different neural pathways, at least to levels below the thalamic nuclei (see Hendelman (2005) and Juliano and McLaughlin (1999) respectively for reviews of the auditory and vibratory pathways). Consequently, VTF pathways bypass the sub-thalamic afferent auditory pathways that project from the cochlea to the cerebellum and auditory cortex.

The present study exploited the known differences between auditory and vibratory afferent architectures and the changes to speech control that occur under AAF and VTF in PWS, to investigate neural mechanisms for facilitating fluency. As background: (1) the pathways to somatosensory cortex that VTF takes are described and compared with the auditory pathways; (2) the ways VTF influences the fluency of PWS are reviewed; (3) a neural model, that proposes how auditory inputs improve the fluency of PWS, is described. One way in which the model could be revised to account for how VTF affects the speech of PWS by

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incorporating the observations about projection pathways is considered; and (4) the predictions for the current study that derive from the revised model are presented.

1.1. Vibratory and auditory pathways

Two types of mechanoreceptor in the skin are responsible for detecting vibration: Meissner's corpuscles are sensitive to frequencies between 30 and 50 Hz; Pacinian corpuscles detect vibrations between 100 and 400 Hz (peak sensitivity of 250 Hz) (Griffin, 1990; Siegel & Sapru, 2006).

The main sensory trigeminal cranial nerve (CN) and posterior column-medial lemniscal (non-CN) pathways transmit vibrotactile information to primary somatosensory cortex. The trigeminal CN enters the brainstem at the level of the mid-pons and ascends via the trigeminal lemniscus, to the thalamus, and from there to primary somatosensory cortex (Juliano & McLaughlin, 1999). This pathway carries information about discriminative touch for the face and top of the head. The fasciculus gracilis, which forms part of the posterior column-medial lemniscal pathway, provides information about vibration from the upper part of the body (t6 and above, including the arms) to the primary somatosensory cortex via the medulla and thalamus. Neither vibratory pathway transmits via the cerebellum.

The afferent auditory system projects from the cochlear nucleus to the inferior colliculus and then via the medial geniculate nucleus to auditory cortex (Hendelman, 2005). The medial geniculate nucleus is part of the thalamic relay system and auditory projections after this may meet up with the post-thalamic vibration pathways. Auditory stimulation activates cerebellar pathways: There are direct connections from the cochlear nucleus to the vermis area of the cerebellum (part of its medial division) in mammals (Huang, Liu & Huang, 1982; Niemer & Cheng, 1949; Zhang, Sun & Jen, 1990). The anatomy of all parts of the cerebellum is similar (Edge, Marple-Horvat & Apps, 2003), allowing the inputs to medial cerebellum to activate the lateral cerebellum directly.

A MEG study by Caetano and Jousmaki (2006) showed that 200 Hz vibrotactile stimulation activated primary somatosensory as well as auditory cortex and activation of the latter was not due to sound artefacts. Consequently, vibration and audition may share a pathway which, anatomical considerations suggest, would have to lie above the thalamus (Hendelman, 2005; Juliano & McLaughlin, 1999). The post-thalamic structures could be responsible for the fluency-enhancing effects of VTF and any form of AAF that is transmitted to them. In contrast, any cerebellar mechanisms involved in fluency-enhancement could only be accessed by auditory inputs because they transmit to this structure whereas vibrotactile stimulation by-passes them.

1.2. Effects of VTF on PWS

Kuniszzyk-Jozkowiak et al. (1996) investigated how synchronous and asynchronous auditory, vibratory and visual speech feedback affected fluency (percentage syllables stuttered, %SS) and speaking rate (number of syllables uttered per second). Synchronous (0 ms delay) and asynchronous (delays of 50, 100 and 160 ms) feedback were investigated in all modalities. %SS and speaking rate reduced significantly as feedback delay increased for all modalities. Both measures reduced more under Delayed Auditory Feedback (DAF) than under delayed VTF.

PWS in Snyder et al.'s (2009) study held their thyroid cartilage between thumb and index finger. This synchronous VTF reduced stuttering frequency by 72% when compared to a condition where there was no VTF. Speaking rate was not examined.

Waddell et al. (2012) picked up vibration on the throat either by microphone or by accelerometer and used this to deliver synchronous VTF to tactile stimulators held between the index finger and thumb.

Stuttering frequency reduced significantly and depended on whether a microphone or an accelerometer was used to pick up the vibration; accelerometer-driven feedback led to most improvement.

1.3. Model for processing auditory inputs that are asynchronous with vocalization and modifications for incorporating VTF effects

Auditory and motor inputs are synchronous when speech is fluent. For example, an efferent copy of fluent speech plans has the same time pattern as the corresponding speech output (Howell & Sackin, 2002). Differencing the timing patterns of these synchronous inputs would null them and absence of activity could signal that speech is fluent and no change to speech control is necessary (Howell, 2010).

DAF creates a situation where the sound of the speaker's voice is asynchronous with the speech plans and speech output. Asynchronous events can also occur in normal listening conditions when speech is dysfluent. For example, stuttered speech may arise because PWS initiate speech before planning is complete (Howell, 2010). Planning can continue during the time that the speech is uttered. If an efferent copy of the plan was taken at the point at which speech was initiated, then this would be asynchronous with the eventual auditory output. Differencing the timing patterns of asynchronous inputs (DAF and stuttered speech) would not cancel. Speakers may use the signal that is selectively generated when inputs are asynchronous as an indication to reduce speaking rate. Consistent with this, DAF slows the speaking rate of fluent speakers, as first reported by Lee (1950). DAF has been used to slow the speaking rate of PWS (Goldiamond, 1965) as a fluency intervention that would allow time to complete speech-planning. The reduction in speaking rate would remove the condition that led to the fluency problem and inputs that were asynchronous would reinstate to synchrony.

Two observations support the view that slowing speaking rate puts plans and outputs back into synchrony and allows fluency to be regained (Howell, 2010): (1) Empirical reports show that the reduced speaking rate under DAF improves fluency (Brendel, Lowit & Howell, 2005; Goldiamond, 1965; Howell, Wingfield & Johnson, 1988; Ryan, 1974). Whilst PWS can achieve fluency gains when they increase their overall speaking rate under AAF (Howell et al., 1987; Kalinowski, Armson, Stuart, and Gracco (1993)), speaking rate is low in the vicinity of segments that have a high likelihood of attracting stuttering (Howell & Sackin, 2000). For instance, DAF always leads to stressed vowels being prolonged under different speaking rates and DAF delays (Kalveram, 2001; Kalveram & Jäncke, 1989); (2) voluntary reductions in speaking rate in normal listening conditions enhance the fluency of PWS (Adams, Lewis & Besozzi, 1973; Andrews, Howie, Dozsa & Guitar, 1982; Janssen & Wieneke, 1987; Perkins, Bell, Johnson, & Stocks, 1979; Vanryckeghem, Glessing, Brutton & McAlindon, 1999).

The cerebellum has been proposed as the site of timing control structures that determine whether inputs during vocalization are synchronous, and if they are not, to initiate a slowing response. Howell and Sackin (2002) used Wing and Kristofferson's (1973) task to investigate how the cerebellum operates under DAF (i.e. when inputs are asynchronous). In the task, participants attempt to produce motor outputs isochronously (i.e. at equally timed intervals). Wing and Kristofferson (1973) decomposed the variance in the responses into motor (Mv) and timekeeper (clock variance, Cv). Ivry (1997) showed that these variance components are mediated by different parts of the cerebellum since lesions in the lateral or the medial parts of the cerebellum led to deficits in Cv or Mv respectively. Speakers in Howell and Sackin (2002) produced syllables isochronously under normal (synchronous) and DAF (asynchronous) feedback conditions. Only Cv increased under DAF compared to normal feedback. From this, Howell and Sackin (2002) proposed that the timekeeper in the lateral cerebellum detected when inputs were asynchronous. The Watkins', Smith, Davis, and Howell

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