



Short communication

Delayed auditory feedback simulates features of nonfluent primary progressive aphasia



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ABSTRACT

The pathophysiology of nonfluent primary progressive aphasia (nfvPPA) remains poorly understood. Here, we compared quantitatively speech parameters in patients with nfvPPA versus healthy older individuals under altered auditory feedback, which has been shown to modulate normal speech output. Patients ($n = 15$) and healthy volunteers ($n = 17$) were recorded while reading aloud under delayed auditory feedback [DAF] with latency 0, 50 or 200 ms and under DAF at 200 ms plus 0.5 octave upward pitch shift. DAF in healthy older individuals was associated with reduced speech rate and emergence of speech sound errors, particularly at latency 200 ms. Up to a third of the healthy older group under DAF showed speech slowing and frequency of speech sound errors within the range of the nfvPPA cohort. Our findings suggest that (in addition to any anterior, primary language output disorder) these key features of nfvPPA may reflect distorted speech input signal processing, as simulated by DAF. DAF may constitute a novel candidate pathophysiological model of posterior dorsal cortical language pathway dysfunction in nfvPPA.

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1. Introduction

During normal speech production, auditory feedback provides sensory information that is used to fine-tune vocal motor output: where access to this feedback is limited (as in the speech of hearing impaired individuals), speech distortions tend to emerge. In experimental settings, synthetically altered auditory feedback (AAF) has been shown to modulate speech output when applied to a speaker's air-conducted voice [21]. Two forms of AAF, namely delayed auditory feedback (DAF; [10]) and frequency altered feedback [37] have been most extensively studied. Individuals with intrinsically normal speech fluency often show loss of fluency, distorted prosody or articulatory errors under AAF [7], whereas AAF has been used therapeutically in stutterers [3, 24]. Functional brain imaging studies have demonstrated a distributed cortical substrate for AAF in bilateral posterior superior temporal and inferior parietal areas that form part of the dorsal cortical stream for processing speech and other sounds [18,35]. While a number of detailed accounts of dorsal cortical auditory pathway function have been proposed [19,20,26,32,41], these generally emphasise intimate sensorimotor linkages between speech perception and production. More

particularly, perceptual control of speech production may engage a mechanism in the posterior superior temporal plane (STP) that links auditory vocal representations with articulatory gestures via the dorsal language pathway [41].

Progressive non-fluent aphasia (the nonfluent/agrammatic variant of primary progressive aphasia, nfvPPA) is a canonical neurodegenerative syndrome characterised by slow, effortful, hesitant speech marred by errors of grammar and articulation [13,14,27]. It is generally considered a disorder of language output programming, though the pathophysiology of nfvPPA is incompletely understood. Neuroanatomically, nfvPPA is linked to damage in peri-Sylvian cortical regions associated with the dorsal language pathway [1,25,30]. The speech disturbance in nfvPPA bears certain similarities to that induced in healthy individuals by AAF: in particular, slowing of speech rate, dysprosody and emergence of articulatory errors. Moreover, patients with nfvPPA have additional deficits in processing complex sounds, including prosody, accents, pitch patterns, voices and environmental noises [11,12,15,16,28], aligning this syndrome with the wider spectrum of progressive aphasia syndromes [38]. This suggests that AAF and nfvPPA might disrupt language network function by at least partly convergent pathophysiological mechanisms, whereby disordered processing of vocal sensory input contributes to impaired speech output via the dorsal language pathway. AAF techniques have been used to assess mechanisms and to rehabilitate dysarthria and dysphasia in stroke, Parkinson's disease and various other neurodegenerative disorders [4,6,9,17,39] but have not been applied

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previously in nfvPPA. Here, we compared quantitatively the speech produced by healthy older individuals under AAF and by patients with nfvPPA. We hypothesised that healthy participants under AAF would show slowing of speech rate and emergence of speech sound errors similar to those exhibited by patients with nfvPPA.

2. Material and methods

2.1. Participants

The healthy participant group ($n = 17$; nine males, mean age 67 years, range 50–78 years) comprised older native English speakers with no previous history of developmental dysfluency, stuttering or hearing deficits. Patients with nfvPPA ($n = 15$; 12 males, mean age 77 years, range 66–84 years) were recruited consecutively from a specialist cognitive disorders clinic; all fulfilled current consensus criteria for nfvPPA [13] and general neuropsychological performance profiles corroborated the syndromic diagnosis in all cases [27]. The nfvPPA and healthy participant groups did not differ in gender composition ($\chi^2 = 0.467$; $p = 0.545$), however the nfvPPA group was on average significantly older than the healthy participants (Mann–Whitney $U = 134.000$; $p = 0.03$).

Ethical approval for the study was obtained from the Local Research Ethics Committee, and all participants gave written informed research consent.

2.2. Experimental procedures

The “Grandfather Passage” ([40]; Supplementary Fig. S1) was chosen as a standardised, representative inventory of English phonemes. Three AAF conditions were created using a commercially available software package, Fluency Coach® (<http://www.fluencycoach.com/>). A short-latency DAF condition was set at 50 ms, corresponding approximately to the minimum delay at which modulation of fluency has been shown in studies of stuttering [22]; a long-latency DAF condition was set at 200 ms, corresponding approximately to the duration of a syllable in conversational spoken English and associated with maximal fluency disruption in previous work [33]; and a combined AAF condition was set at 200 ms plus an upward pitch shift of 0.5 octaves.

The AAF conditions were administered to healthy participants via Sennheiser® (HD265 Linear) headphones at a comfortable listening level (at least 70 dB) in a quiet room. Participants were instructed to read the passage aloud as naturally as possible. Speech samples were recorded as digital wavefiles using Goldwave® software onto a laptop computer with a built-in microphone, for analysis off-line. Before recording commenced, healthy participants were first familiarised with the AAF procedure and set-up. The order of presentation of AAF conditions was randomised between participants, however the baseline (no AAF) condition was always administered last, to reduce any rehearsal effects; participants were blind to condition order.

Speech wavefiles were initially edited manually to remove any extraneous noise sources or pauses. Mean speech rate for each AAF condition in the healthy participant group and for the nfvPPA group was calculated as the mean number of words produced per second, as determined using a customised programme in MATLAB®. The mean total number of errors for each AAF condition in the healthy participant group and for the nfvPPA group was determined from an acoustic analysis of the speech recordings: errors were further subclassified according to whether they were speech sound errors (syllable duplications, omissions or misarticulations), or grammatical errors (errors of morphology or syntax).

2.3. Statistical and qualitative analyses

Statistical analyses were performed using SPSSv17®. Multivariate analyses of variance (MANOVAs) were used to assess the effect of

group membership (healthy vs nfvPPA) on behavioural performance in each AAF condition. Age, gender and reverse digit span (an index of auditory working memory potentially relevant to monitoring of speech output under AAF) were incorporated as covariates in group comparisons. MANOVAs were also performed to assess the effect of DAF condition (independent variable: baseline, short-latency DAF, long-latency DAF) on behavioural performance of healthy participants (dependent variables: speech rate, total errors, duplications, misarticulations, omissions); post hoc pair-wise comparisons between conditions using Bonferroni’s correction were carried out if significant overall correlations were found. For all tests, results were considered statistically significant at a threshold $p < 0.05$.

In addition, in order to qualitatively assess the confusability of healthy individuals’ speech under AAF with speech produced by patients with nfvPPA, speech samples from the nfvPPA group and the healthy group under DAF were classified according to group membership by an experienced cognitive neurologist (PW) blinded to group membership.

3. Results

3.1. Group data on reading task

For the reading aloud task, the healthy participant group showed a significantly faster mean speech rate than the nfvPPA group at baseline ($F(1,27) = 57.7$, $p < 0.0001$) and this difference remained (but was attenuated) under the short-latency DAF ($F(1,27) = 17.9$, $p < 0.0001$), long-latency DAF ($F(1,27) = 8.77$, $p = 0.006$) and combined AAF ($F(1,27) = 6.34$, $p = 0.018$) conditions. The mean total error score and scores for error subcategories did not differ significantly between the healthy participant and nfvPPA groups at baseline nor under any of the AAF conditions; this was likely attributable to the wide variation in error scores within the nfvPPA group (see Fig. 1). In both the healthy participant and nfvPPA groups, the most frequent speech sound error types were phonemic duplications and misarticulations.

Significant main effects of DAF condition on speech rate ($F(2,43) = 29.95$, $p < 0.0001$), total error score ($F(2,43) = 10.35$, $p < 0.0001$) and duplication ($F(2,43) = 8.05$, $p = 0.001$) and misarticulation ($F(2,43) = 6.63$, $p = 0.003$) error scores were found. Speech rate was significantly slower on short-latency and long-latency DAF than on baseline ($p < 0.0001$). Duplication errors were significantly more frequent in the long-latency DAF condition than at baseline or in the short-latency DAF condition ($p < 0.05$) and misarticulation errors were significantly more frequent in the long-latency DAF condition than at baseline ($p = 0.002$).

3.2. Individual data: healthy individuals acquiring speech features of nfvPPA under AAF

A proportion of healthy individuals (Fig. 1) showed slowing of mean speech rate and total error rates within the range of patients with nfvPPA. The proportion of healthy participants acquiring these characteristics rose with increasing DAF latency: at a DAF latency of 200 ms, 4/17 (24%) of healthy participants developed a mean speech rate within the nfvPPA range and 6/17 (35%) developed a total error score within the nfvPPA range. Main effects of gender and age on error rates were observed: healthy male participants produced significantly more duplication errors than healthy female participants overall ($F(1,43) = 5.88$, $p = 0.020$), and healthy participants made significantly more frequent misarticulation errors with advancing age ($F(1,43) = 7.83$, $p = 0.008$).

When speech samples from the nfvPPA group and the healthy participant group under DAF (latency 200 ms) were classified (nfvPPA or healthy) by an experienced cognitive neurologist blinded to group membership, 2/17 (12%) of healthy participant speech samples were misclassified as nfvPPA while all nfvPPA samples were classified correctly.

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