AN ACOUSTIC INVESTIGATION OF PITCH ACCENT CONTRASTS IN THE SPEECH OF A NORWEGIAN PATIENT WITH THE FOREIGN ACCENT SYNDROME

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ABSTRACT

In 2005 a middle aged Norwegian man became aphasic as a result of a left hemisphere stroke. After a few months his aphasic condition had improved. He was mildly agrammatic with word finding problems and what sounded like a foreign accent. Deviant prosody was an important feature of his foreign sounding speech, in particular the lack of a clear distinction between the two Norwegian word tones (pitch accents). Acoustic analysis of his speech revealed limited F0 variation at word and utterance level and a similar F0 pattern on the two word tones. His deviant prosody is assumed to be the result of reduced ability to produce appropriate F0 variation, a dysarthric condition. There was no clear indication of apraxia of speech.

Keywords: Foreign accent syndrome, aphasia, apraxia of speech, dysarthria, pitch accents

1. INTRODUCTION

Brain damage which results in aphasia and/or apraxia of speech frequently causes deviant articulation; and in a limited number of cases the patients' speech takes on characteristics normally associated with a dialect that is not their own, or it resembles the performance of a non-native speaker of the language. This condition is referred to as the foreign accent syndrome (FAS). Although there are a relatively large number of reported cases of FAS, there is little agreement about the syndrome. There is disagreement about whether it is a syndrome in its own right or whether it is a subtype of apraxia, aphasia, or dysarthria. There is disagreement about whether it can be accounted for in terms of a single underlying mechanism, and there is disagreement about its neurological underpinnings [1]. We are still at the stage where careful individual case studies are needed in order to clarify why a neurological disorder results in speech which the listener perceives as a foreign accent.

The clinical impression is that deviant prosody is almost universally present in FAS. And this impression is supported by acoustic analyses which have found deviant fundamental frequency contours, deviant intensity, and deviant segmental duration in the speech of the patients [1].

The present study looks at the production of word prosody (F0) in the speech of a Norwegian stroke patient with FAS. Norwegian is a pitch accent language where every accented syllable has the pitch pattern of one of two possible accents. The patient does not distinguish between these two patterns, and this is a deviant feature which strongly contributes to the 'foreignness' of his accent in the ears of the listener.

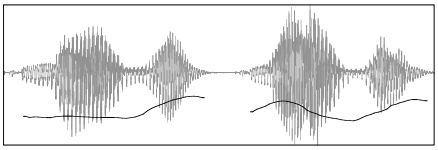
2. THE NORWEGIAN PITCH ACCENT

In Norwegian every accented syllable will carry the pitch pattern of one of two possible tones, referred to as Accent1 and Accent2. The choice between these accents is normally lexically determined. That is, the accent must be listed in the lexicon together with the word's segmental phonological structure. There are a number of minimal pairs differing only in tone, the pitch pattern of their accented syllables, for instance the following pairs (the superscripts [1] and [2] indicate Accent1 and Accent2, respectively): *vannet* /[1]vane/ (the water) - *vanne* /[2]vane/ (to water); *skuffen* /[1]skufen/ (the drawer) - *skuffen* /[2]skufen/ (the shovel).

Both accents are associated with a low pitch level in East Norwegian. Accent2 involves a rise in pitch followed by a fall on the first, stressed, syllable. The pitch reaches its lowest level in the beginning of the second, unstressed, syllable. Accent1 has a low level pitch on the accented syllable [2]. The pitch pattern may be described as

high-low in Accent2 syllables and as low in Accent1 syllables. The pitch pattern of the syllable(s) following the accented syllable varies. This variation is not part of the tonal distinction, but belongs to the domain of sentence intonation and signals differences in the information structure of the utterance. The pitch patterns in Figure 1 represent two words spoken in isolation, with focal accents. The final pitch patterns are therefore rising. When the accents are not focal, the pitch pattern will end in a more moderate rise or in a low level. When the accented syllable is not followed by an unaccented one, there is no tonal opposition. The fundamental frequency contour in these cases is similar to Accent1.

Figure 1: F0 patterns of the two East Norwegian pitch accents, the words *løvet* and *løve*



/¹l ø: v e / /²l ø: v e /

3. PATIENT CHARACTERISTICS

The patient ZZ is a 59 year old physician who in October 2005 suffered a cerebrovascular accident and developed right hemiparesis, facial paresis, tongue deviation, dysphagia, dysarthria, and aphasia. While an initial CT-scan was without pathology, rescan after six hours showed changes in accordance with left parietal infarction. ZZ was treated with thrombolytic therapy resulting in an almost complete restitution of sensorimotor function. He regained independence with regard to activities of daily living, but the communication disorder prevailed. Besides the communication disorder which is described in detail below, neuropsychological screening revealed reduced psychomotor tempo and sustained attention. Furthermore, a memory disorder was diagnosed. Especially impaired were storage and recall of oral verbal items together with recall of visual stimuli. In addition, working memory was somewhat reduced in comparison to the presumed premorbid level. The patient lived for several years in Germany as a medical student. He had a fluent command of German and English in addition to his native Norwegian. Listeners characterise his speech as having an unidentified foreign accent, possibly of East European origin. None of the listeners have associated this with a German or an English accent. In other words, there is no indication that this is a case of deviant speech associated with polyglot aphasia.

The patient has been tested with the Norwegian Aphasia Battery and with the Norwegian standardised version of the Frenchay dysarthria assessment. At the time of the present investigation there were no clear indications of apraxia of speech, but a rest of oral apraxia. The dysarthria assessment showed voice problems, reduced phonation time and reduced pitch variation.

3.1. General speech characteristics

The patient speaks slowly with deviant articulation of individual segments. His prosody is monotonous. He has word finding problems, and he occasionally makes syntactic mistakes.

3.2. The patient's production of pitch accents

3.2.1. Test battery

Test 1: The patient was asked to read twelve words in a syntactic frame, *Det var . . . jeg sa* (It was . . . I said) and to put emphatic stress on the target words. Each target word was presented by a drawing with the sentence written beneath the drawing. The target words were chosen to form pairs, though not minimal pairs, of Accent1 and Accent2 words with the same, or similar, segmental structure in the accented syllable.
Test 2: The patient was presented with stimuli consisting of six minimal pairs of words differing only in accent type. The minimal pairs were illustrated by drawings, with the target word written below each drawing. The patient was asked to read each word. The members of the same pair were read consecutively. Digital recordings of both tests were made in a quiet room in the hospital.

3.2.2. Auditory impression

The recordings of Test 2 were presented to five normal subjects who were asked to identify the targets by pointing to the corresponding drawing. The subjects were either uncertain as to which word was produced or identified both words of a

minimal pair as the word with Accent1. None of the subjects identified a test word as Accent2.

3.2.3. Acoustic analysis

Acoustic analyses confirm the auditory impression: F0 curves of the two accents in Test 2 are similar and resemble Accent1. Figures 2a and 2b show F0 contours of the words [1]*kammer* (chamber) and [2]*kammer* (combs) as spoken by the patient and by an age matched control.

Figure 2a: F0 traces of [1]*kammer* (thin line) and [2]*kammer* (heavy line) spoken by the control subject

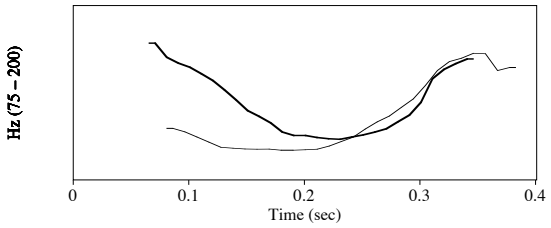
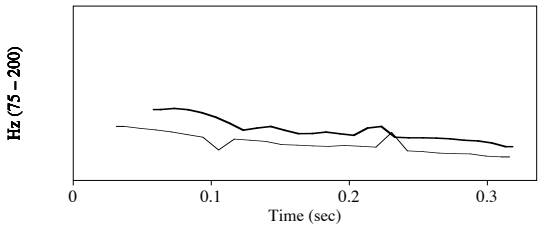


Figure 2b: F0 traces of [1]*kammer* (thin line) and [2]*kammer* (heavy line) spoken by the patient



The tendency to produce a pitch contour similar to that of Accent1 on both accents is also present in Test 1 where the words are not minimal pairs. And both tests indicate that the patient does not differentiate between a focal accent, an accent ending in a rise, and a non-focal accent which does not end in a rise. Figures 3a,b show the F0 contours of the words [1]*bilen* (the car) and [2]*biler* (cars) in the frame *Det var _ jeg sa* spoken by the patient and by the control. In this context a focal accent is the appropriate one on the words *bilen* and *biler*. And the two accents both end in a rise in the F0 contours of the control, but not in those of the patient.

Although the patient's fundamental frequency curves for the two accents are similar, there are indications that he may try to distinguish between the accents by other means than F0 variation, by variation in segmental duration or variation in air pressure. In the production of the minimal pair [2]*drar til* (hits) [1]*drar til* (goes to) read consecutively with the Accent2 word first, he lengthens the accented syllable in the Accent1 word and in that way makes a distinction between the two words (see Figure 4). It is noteworthy that there may be a durational difference between the accented syllables in normal speech, but the difference will then be that Accent2 is slightly longer than Accent1 [2].

The minimal pair [1]bokser (a dog), read first, and [2]bokser (an athlete) both sound like an Accent1 word in the patient's speech, but the sound pressure is stronger on the Accent2 than on the Accent1 word (see Figure 5).

Figure 3a: F0 traces [1]*bilen* (thin line)[2]*biler* (heavy line) spoken by the control subject

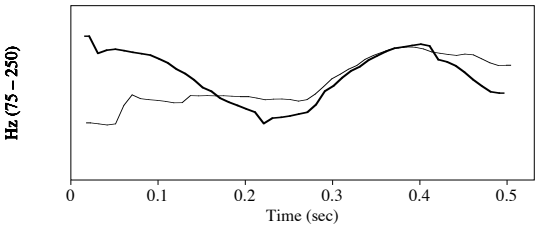


Figure 3b: F0 traces [1]*bilen* (thin line)[2]*biler* (heavy line) spoken by the patient

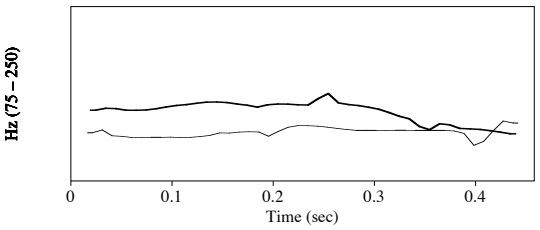


Figure 4: F0 traces and spectrogram of the patient's pronunciation of *drar til* with Accent2 and Accent1

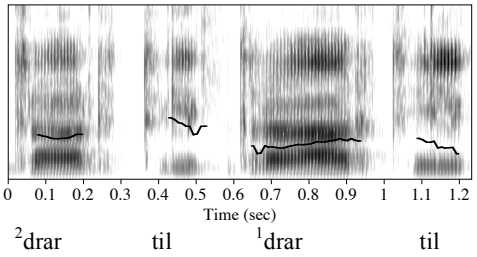
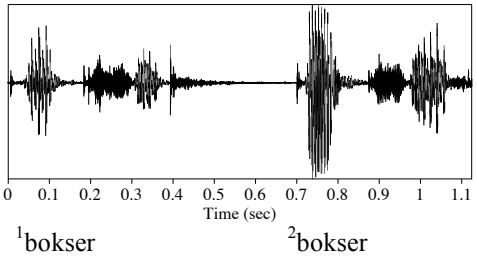


Figure 5: Speech oscillogram of the words *[1]bokser* and *[2]bokser* spoken by the patient



4. DISCUSSION

Brain damage may lead both to phonological and phonetic deviations in clinical speech. In the case of patient ZZ his Accent2 contours are similar to his Accent1 contours and both contours are similar to the Accent1 contours in normal speech. This may be interpreted as a case of replacement of Accent2 with Accent1, a phonological problem, a frequent phenomenon in aphasic speech. However, although ZZ's articulation of individual segments may be deviant, the deviations are not of a type to indicate phonological substitutions. The correct target phoneme is always recognizable. Therefore, since there are no indications of segmental phonological substitutions in the patient's speech, and since he clearly has a problem with varying his fundamental frequency, it is more likely that his deviant accent production reflects an articulatory problem than a phonological one. Since Accent1 involves less variation in fundamental frequency than Accent2, a lack of fundamental frequency variation can easily lead to an acoustic similarity between the two accents. The interpretation that the similarity between the two accents reflects an articulatory rather than a phonological problem, is also supported by the fact that he apparently, when reading minimal pairs, tries to make a distinction between the two accents by other means than F0 variation, by variation in segmental duration or variation in sound pressure. It should be mentioned, however, that the patient's ability to distinguish auditorily between the two accents has not yet been tested.

There are suggestions in the literature that FAS may be a subtype of apraxia of speech [3]. The present case offers no firm support for this hypothesis. Clinical tests have indicated that the patient has problems with voice control and oral apraxia, but there are no definite indications of apraxia of speech. It must, however, be kept in mind that the distinction between apraxia of speech and oral apraxia is not always a clear cut one.

This case confirms the general impression that deviant prosody is an important factor when clinical speech sounds like a foreign accent. This patient's prosody, though, differs from that of the majority of reported FAS cases in having a flat intonation rather than excessive rises and falls [1]. It resembles other Norwegian cases of FAS in showing reduced ability to distinguish between the two Norwegian pitch accents [5] [4].

In addition to deviant prosody the patient also has deviant segmental features in his speech. The segmental deviations have not yet been analysed. It is therefore premature to discuss the possibility of a single underlying mechanism as the cause of his foreign sounding accent.

5. REFERENCES

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