Prosodic Disturbances in Speech Following Right Temporal Lobe Epileptic Seizure

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ABSTRACT

In this paper, we compare speech following the onset of epileptic seizure to reported data on speech of patients with identified ataxic dysarthria and right hemisphere lesions. In particular, we focus on speech produced by a male patient whose epileptic seizures originate in the right temporal lobe, and whose speech resembles that of patients with a diagnosis of ataxic dysarthria. Findings on measures of "scanning speech" pattern, vocal instabilities, and slow rate confirmed this perceptual impression. Results on the perceptual impression of "monopitch" were less clear. Speech following seizure onset showed pitch range expansion rather than compression. Pitch contours resembled a high plateau rather than the more normal declination pattern. These findings may be explained by a pattern of seizure activation spreading to cerebellar structures during the course of a seizure, thereby affecting the motor execution phase of speech production. More data is needed to determine if prosody planning in the right temporal lobe is also disrupted.

1 INTRODUCTION

Little is known about speech following epileptic seizure. Because seizures originate in specific areas of abnormality in the brain, there is a clear analogy with cases of lesion following stroke, focal trauma, etc. Thus, Ross and colleagues [1] have suggested that patients with left hemisphere seizure foci may show speech deficits following seizure that resemble speech deficits following left hemisphere stroke. Similarly, it has been suggested that patients with right hemisphere foci may show speech deficits that resemble those of patients with traumatic right hemisphere damage or stroke [2]. A disadvantage of stroke studies is that it is rarely possible to control for individual differences that may have existed before the injury. Thus, one compares the speech production of a brain-damaged patient to the average speech production characteristics of normal subjects rather than to the speech production of that patient before the injury occurred. A major advantage of epilepsy studies is that patients have periods in which they are seizure-free. Thus, a subject may be used as his or her "own control".

This paper began as an investigation into prosodic changes in the post-seizure speech of a male patient with a seizure focus in the right temporal lobe of the cerebral cortex. Patients with right temporal lobe damage are frequently described by clinicians as showing reduced or disturbed prosodic skills. We hypothesized that patients whose epileptic seizures originate in the right temporal lobe would show similar types of disturbed prosody. However, we became intrigued with the fact that experienced speech pathologists described our subject's post-seizure speech as sounding like ataxic dysarthria. This type of dysarthria is usually associated with a disruption of motor execution rather than semantic planning, and attributed to dysfunction of the cerebellum. We determined that an investigation of this epileptic patient's speech might shed light on: (a) the planning and execution of prosody in sentence production, and (b) the influence of epileptic seizures on these processes.

The speech characteristics ordinarily ascribed to ataxic speech are: (1) slow speech rate, (2) a "scanning" pattern, attributed to "excess and equal stress", (3) articulatory imprecision, (4) monotonous use of pitch and loudness, and (5) vocal instability, which may be described clinically as pitch breaks, vocal tremor, or pitch fluctuations [3], [4]. Although all of these characteristics tend to appear together in cases of ataxic dysarthria, each of these characteristics except the "scanning speech" pattern may also occur in other types of dysarthria. The perception of a "scanning speech" pattern is considered a key indicator of ataxic types of dysarthria. The cooccurrence of the "scanning" pattern with articulatory imprecision and vocal tremor is also a key aspect of the diagnosis, as it confirms the existence of a movement disorder general to all speech articulators (and thereby the cerebellum) rather than to specific nerves or muscles (and thereby cranial nerve branching points). Although the characteristics listed above definitely qualify as change in prosody from normal speech, ataxic dysarthrics are not ordinarily classified as having a disorder of prosody per sé, but rather a motor speech disorder. In other words, the distinction is between planning and execution. This fits well with the usual classification of the right hemisphere as involved in the planning of prosody, and of the cerebellum as involved in motor output. However, it becomes an interesting question when posed with regard to a single individual with an intermittent disorder such as epilepsy. Perhaps, for instance, our subject's speech productions sound ataxic because his seizures temporarily disrupt right-hemisphere functions such as prosody planning. Alternatively, perhaps our

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subject's productions sound ataxic because his seizure disorder affects motor execution in a way that mimics the dysfunction of ataxic dysarthria. A third possibility is that our subject's speech differs from his normal speech in some unpredicted way.

In this paper, then, we set out to compare characteristics of our epileptic speaker's seizure speech with reported characteristics of patients with ataxic dysarthria. We compared our subject's seizure speech with his own speech recorded in a seizure-free control condition. Because the characteristics of normal speech at slow rates are not well described, we also recorded ten normal male speakers at normal and slow rates. The recordings of the epileptic subject were made as part of a routine protocol administered to patients considering surgical intervention in the Epilepsy Treatment Program at University Hospital in Cincinnati. The patient's seizures were monitored via scalp EEG, and as soon as seizure activity began, he was asked to read the sentence "They heard him speak on the radio last night" from a printed card. The subject began speaking as soon as he was able to respond to the instructions, and continued to repeat the sentence as requested past the point where EEG activity ceased. The subject was diagnosed with complex partial seizures originating in the right temporal lobe. As is common with this type of epilepsy, he was able to produce speech during the period of time in which EEG activity was diminishing. The subject was right-handed. All speech was in English and involved the same sentence. The experimental data were thus as follows:

Male epileptic speaker (codename RUDLAD)
Five (5) control utterances
Ten (10) sequential seizure utterances

Ten (10) normal male speakers
Five (5) repetitions at fast rate
Five (5) repetitions at slow rate

The following measurements were obtained:
(a) Total duration of the sentence
(b) Duration of all syllables in the sentence
(c) Pitch range (Peak F0 - minimum F0)
(d) Duration from seizure onset to utterance beginning
(e) Number of pitch fluctuations in the sentence
(f) Scanning speech index (see below)

We used total duration (including pauses) as an index of speech rate. For our measure of "scanning" speech, we used the SI index suggested by Hertrich & Ackermann [5], computed on the six syllables of "speak on the radio". The index is constructed to equal 1 when perfect isochrony prevails (s = syllable duration).

$$\text{SI index} = \frac{s1 * s2 * s3 * s4 * s5 * s6}{[(s1 + s2 + s3 + s4 + s5 + s6)/6]^6}$$

We measured vocal instabilities visually from 1024 point narrow-band spectrograms as the number of short-duration fundamental frequency modulations over time. We measured F0 range as the difference between F0 peak and F0 minimum in the same sentence. For this paper, we did not measure articulation per sé. Overall, we hypothesized that the collective results of our measures would resemble those found by other investigators for ataxic dysarthric speakers [3-8].

2 RESULTS

The epileptic speaker's control utterances were somewhat slow compared to the average of the normal male speakers, but his utterances after the onset of seizure were considerably slower than his control utterances. Fig. 1 shows RUDLAD's seizure utterances as a function of time post seizure onset. Control utterances are shown as occurring at the same point in time on the graph but time is not relevant for these.

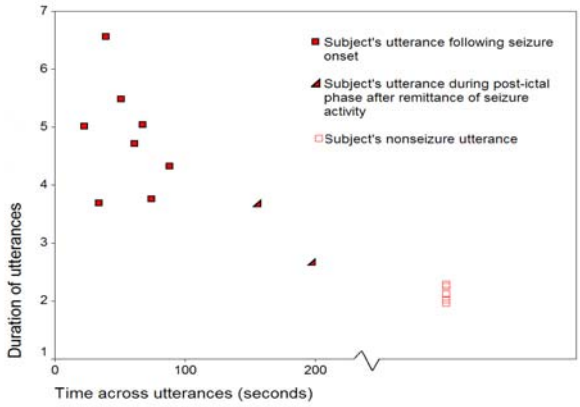

Fig. 1. Utterance Duration vs. Time

As the figure shows, RUDLAD's speech rate is relatively slow for productions immediately post seizure onset. While there is considerable variability, it is also clear that his speech rate tends to be faster as a function of time post seizure onset. Note that while seizure-related EEG activity continued during the first eight utterances post seizure onset, seizure activity was no longer identifiable via EEG during production of the last two utterances. These are accordingly labeled with different symbols.

We next looked at whether RUDLAD exhibited "scanning speech" patterns in his post-seizure utterances, in comparison to his control utterances. Fig. 2 shows the SI index calculated for each utterance as a function of time post seizure. It is clear that RUDLAD's immediate post-seizure utterances have a high SI index, and that the trend is for the SI index to decrease over time. In a one-way ANOVA, the comparison between all of RUDLAD's post-seizure utterance SI's (mean SI = 0.52) with those of his control utterances (mean SI = 0.37) was significant ($DF = 1,13$, $F = 12.2$, $p < 0.01$). We conclude that RUDLAD shows some scanning speech behavior following seizure, and that this behavior is related to the seizure activity itself.

One question that arises is whether RUDLAD's "scanning speech" behavior is related to his slowed speech rate after seizure. To test this, we calculated the SI index for normal speakers at self-selected normal and slow rates. Interestingly, the SI index for normal speakers was found to

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be significantly higher (DF = 1,98, F = 24.7, $p < 0.01$) for slow (mean SI = 0.46) vs. normal speech rate (mean SI = 0.32). **It appears that normal speech at slow rates is more isochronous than normal speech at faster rates.** Thus, some portion at least of the perception of "scanning speech" in dysarthric speakers may be due to their typically slow rate.

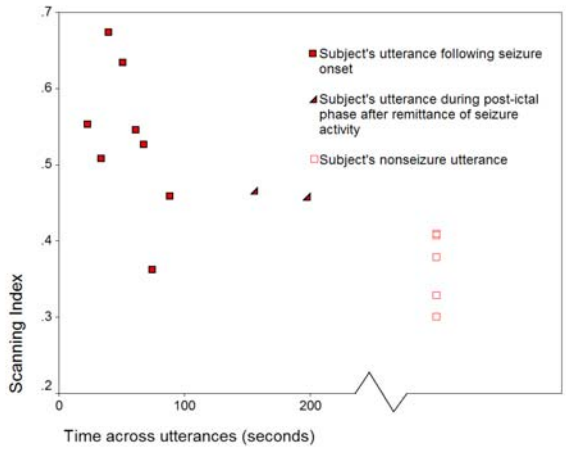

Fig. 2. Scanning Index vs. Time

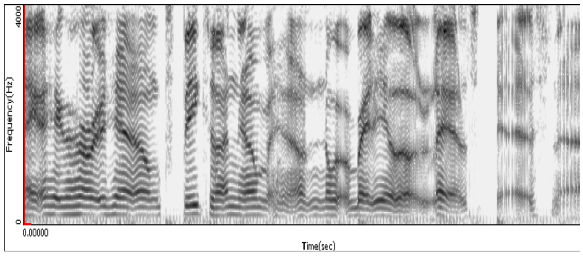

Fig. 3. Narrow-band spectrogram of RUDLAD's fourth utterance after seizure

Examination of narrow-band spectrograms of RUDLAD's post-seizure speech revealed a high incidence of localized changes in vocal fold vibratory mode, including vocal fry, pitch breaks, and tremor-like oscillations. Fig. 4 shows the number of such pitch instabilities as a function of time post seizure onset. It is evident that speech produced after seizure onset contains many vocal instabilities (mean = 15.8 per sentence, with a maximum of 28). This measure shows little downward trend. RUDLAD's two post-seizure utterances with no associated EEG activity have only a few more vocal instabilities compared to his normal utterances (mean of 0.6). The difference in number of instabilities between RUDLAD's post-seizure and control utterances is significant (DF = 1,13, F = 17.2, $p < 0.01$). It appears that RUDLAD's post-seizure speech contains vocal instabilities such as are reported for ataxic dysarthria.

Monopitch has been described as a symptom both in ataxic dysarthria [4] and in cases of right hemisphere stroke [9]. [In the case of dysarthria, it is attributed to dysfunctional execution; in the case of right hemisphere stroke it is attributed to a disruption of the prosody planning process.] As noted above, we measured pitch range as the difference between peak and minimum F0. The high incidence of vocal instabilities in RUDLAD's utterances, including vocal fry, resulted in a high number of octave errors in the output of the pitch algorithm. Fig. 5 shows a wide-band spectrogram with F0 track for RUDLAD's fourth utterance post seizure in which octave errors generated by the pitch algorithm are evident in the pitch contour below the spectrogram. To prevent erroneous minimum F0 values, we corrected for these octave errors before calculating F0 range.

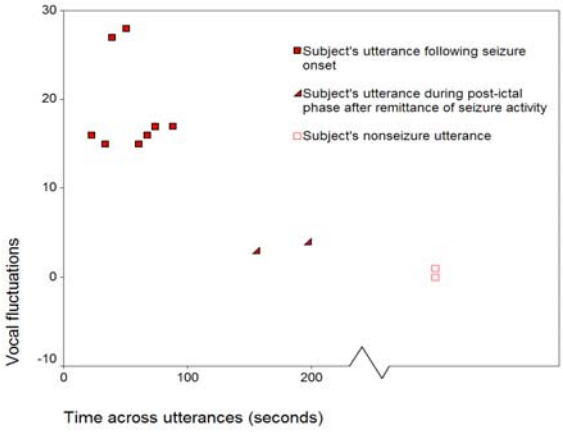

Fig. 4. Vocal Instabilities vs. Time

Contrary to the hypothesis of monopitch, RUDLAD's productions following seizure showed a greater average pitch range than his control utterances (71 Hz vs. 29 Hz). This contrasts with the very minor difference shown for slow (mean of 62) vs. normal (mean of 58) rate productions by normals. [Due to technical difficulties, four of the ten normal subjects were not measured for pitch range.] The difference was significant for RUDLAD's control vs. seizure utterances (DF = 1,13, F = 27.5, $p < 0.01$), but not for normal subjects according to rate (DF =1,58, F = 0.229, $p = 0.63$). Thus, RUDLAD appears to increase his pitch range in the post-seizure condition. Further, his post-seizure pitch range is slightly larger than the average for normals.

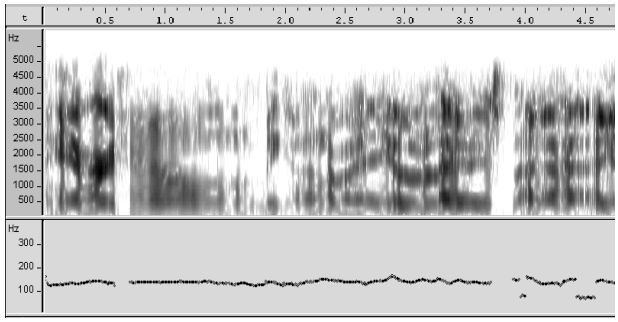

Fig. 5. Wide-band spectrogram of RUDLAD's third utterance after seizure, showing octave errors by pitch tracker.

The pitch range measured for RUDLAD's control utterances is about half of the normal range, even for speech read aloud in a neutral environment [10]. This may be an artifact of the small corpus of sentences, or it may

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reflect RUDLAD's habitual pitch range. RUDLAD's normal speech patterns may also have been affected by changes to basal ganglia seen in chronic epilepsy [11]. Note also that the pitch contours used by RUDLAD in his post-seizure speech are intonationally quite peculiar. When normal speakers read an emotionally neutral sentence aloud, they routinely follow a pitch contour in which the highest F0 value is early in the sentence, while their lowest F0 is close to the end of the sentence [12]. This tendency is correlated with the physiologically determined loss of respiratory volume for sentences produced in a single breath. In spite of their restricted range, RUDLAD's control utterances follow the standard pattern. In contrast, all of his post-seizure utterances showed an anomalous pattern in which the lowest F0 occurred unpredictably in early or middle portions of the utterance, 80% of pitch values were in the upper portion of his pitch range, and the sentence terminated with a flat or rising contour. Pitch contours of this type require considerable effort in countering the effects of reduced air pressure. Perceptually, they sound unfinished. This anomalous pattern does not fit the conventional definition of monopitch, but, because of the consistent high pitch, might well be described that way by a listener.

3 CONCLUSION

The results of this study are fairly clear for the parameters of slow rate, isochrony, and vocal instabilities (see Table 1). Each of these measures is noted in ataxic dysarthric speech. For RUDLAD, the changes in the parameters of slow rate and isochrony diminish linearly over time relative to the seizure onset, suggesting that the size of the effect is regulated by a linear decay process set in motion by the seizure itself. Vocal instabilities appear to vanish with the offset of EEG activity in RUDLAD's final 2 utterances.

Measure	**Ataxic**	**RUDLAD**
slower rate	yes	yes
isochrony	yes	yes
monopitch	Not clear	Not clear
vocal instabilities	yes	yes

Table 1. Summary of Results

These results suggest that seizure activity is disrupting areas of the brain that are also disrupted in ataxic dysarthria. The finding of anomalous pitch contours in RUDLAD's post-seizure speech is in some ways more interesting. Our results are consistent with reported results on pitch range in ataxic dysarthria [4],[6],[12], but differences in measurement make direct comparison difficult. Physiologically, the maintenance of high pitch can be accounted for as a simple result of increased tension in the intrinsic muscles of the larynx. Increased overall tension might also explain the increased incidence of vocal instabilities, including vocal fry. Both phenomena may certainly result from disruption of motor execution. At the same time, the use of unusual pitch contours may also be attributed to the prosody-planning phase of speech production. As such, RUDLAD's anomalous pattern may result from disruption at the point of his seizure locus, causing him to select an inappropriate intonation pattern for his post-seizure speech. Interestingly, his pattern is quite different from that reported by Blonder et al. [9] for a patient tested six months after a stroke affecting the right fronto-temporal region and right basal ganglia. Due to the paucity of studies in this area, we leave the question open at this time. Clearly, more data on the speech production of patients with identified lesion sites is needed in order to pursue the comparison with speech production in seizure disorders.

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