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A

SPEECH PROSODY IN CEREBELLAR ATAXIA

by

Maureen Casper

A dissertation submitted to the Graduate Faculty in Speech and Hearing Sciences in partial fulfillment of the requirements for the degree of Doctor of Philosophy, The City University of New York

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Abstract

SPEECH PROSODY IN CEREBELLAR ATAXIA

by

Maureen Casper

Adviser: Professor Lawrence J. Raphael

The hallmark of cerebellar pathology is a disturbance in motor control known as ataxia. Ataxic movements of the limbs are characterized by poor coordination of muscles acting across different joints, as well as by defective scaling of movement, leading to the characteristic clinical findings of over- and undershoot (dysmetria), oscillatory movements in attempting to maintain stationary position, intention tremor, and irregularity of rapid alternating movements (dysdiadochokinesia). Ataxic speech, also known as "scanning" speech, is characterized perceptually by a relative loss of the normal intonational pattern which lends a sing-song quality to normal speech, as well as a tendency to blur the distinction between long and short syllables. Standard clinical analysis by speech-language pathologists is done on a perceptual basis, without quantification of the deficit. The present study sought an acoustic signature for the speech disturbance recognized in cerebellar degeneration.

Magnetic resonance imaging was used for a radiological rating of cerebellar involvement in six cerebellar ataxic dysarthric speakers. The speakers were matched pair-wise to normal controls by age, sex and dialectical status. The twelve speakers read aloud a series of sentences with the target [pɑp] syllable in question-answer format. The sentences were designed to elicit six different prosodic conditions and four contrastive prosodic events. Acoustic measures of the [pɑp] syllables in contrastive prosodic conditions and of normal vs. brain-damaged patients were used to further our understanding both of the speech degeneration that accompanies cerebellar pathology and of speech motor control and movement in general.

The direction and extent of jaw and tongue movements were inferred from vocal tract resonances: F1 and F2. In addition, the acoustic measures provided data on the frequency of vocal fold vibration: f_0 and the overall duration of the target [pɑp] syllable. Pair-wise comparisons of the prosodic conditions within the normal group showed statistically significant differences for all four prosodic contrasts. For three of the four contrasts analyzed, the normal speakers showed both longer durations and higher formant and fundamental frequency values in the more prominent first condition of the contrast. The direction of the contrast phrase-final accented vs. non-final accented showed a different direction of prominence from the other three contrasts for the normal speakers. Within this contrast, the first condition (phrase-final accented) had the longer duration while the three frequency values for that condition F2, F1 and f_0 were lower than the non-final condition. These data challenge spatio-temporal theory which explains movement as an artifact of time wherein longer durations predict more extreme movements and give further evidence for gestural internal dynamics of movement in which time emerges from articulatory events rather than dictating those events.

Differences between conditions and the direction of prominence across conditions within each contrast were examined. While the cerebellar speakers showed significant differences for the measure of syllable duration in the first contrast: phrase-final accented (+pf+a) vs. non-final accented (–pf+a) and for the measure of F1 in the fourth contrast: post-nuclear unaccented (-n-a) vs. reduced (red); the direction of these contrasts were reversed from the normal pattern. The +pf+a vs. –pf+a contrast was reversed from the normal pattern in the acoustic measures of F2 and F1. The direction of syllable reduction as measured in the prosodic contrast –n-a vs. red was reversed for the acoustic measures of syllable duration and F_0. In addition, nuclear accented (+n+a) vs. post-nuclear unaccented (-n-a) was reversed in F1 and non phrase-final accented (-pf+a) vs. non-phrase final unaccented (-pf-a) was reversed in f_0.

The acoustic measures of the normal prosodic contrast values were then used as a model to measure the degree of speech deterioration for individual cerebellar subjects. This estimate of speech deterioration as determined by individual differences between cerebellar and normal subjects' acoustic values of the four prosodic contrasts was used in correlation analyses with MRI ratings. Moderate correlations between speech deterioration and cerebellar atrophy were found in the measures of syllable duration and f_0. A strong negative correlation was found for F1.

Moreover, the normal model presented by these acoustic data allows for a description of the flexibility of task-oriented behavior in normal speech motor control. In normal speech motor control not all durations are the same. This model provides a sensitive index of cerebellar pathology with quantitative acoustic analyses. The findings presented here are discussed with regard to previous findings for cerebellar speech, normal motor control and cerebellar pathology in general.

ACKNOWLEDGMENTS

A main reason for writing this dissertation was to establish a more complete description of dysatrthria, one that may be useful to clinicians working with persons who have speech problems related to neurological disease. For presenting the question, and providing the enthusiasm and support to do this investigation, I wish to sincerely thank Katherine S. Harris, who inspired me to look at the bigger picture, describing the brain-behavior relationship.

For acoustic measurement, there were several hurdles to overcome. I wish to thank Lawrence Raphael for his valuable feed-back in the measurement phase of the study and for his time and efforts toward revising this work. I also wish to thank Fredericka Bell-Berti and Robert Goldfarb for their support.

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INTRODUCTION

1.1 Cerebellar pathology

Overall, the hallmark of cerebellar pathology is a disturbance in motor control known as ataxia, recognized as a profound loss of muscular coordination (Diener & Dichgans, 1992). Ataxia offers a fertile ground for research since this type of motor disorder has been directly correlated with the specific degeneration of the cerebellum. Thus, in ways not possible with other more systemic neuro-degenerative diseases, it is possible to associate an area of atrophy with a behavioral description. Although ataxia has long been recognized as a hallmark of cerebellar pathology (Diener & Dichgans, 1992), advances in both neuroimaging and behavioral analysis provide methods for a more complete description.

1.1.1 Limb movement in cerebellar ataxia

A loss of coordination in ataxia may take the form of the loss of balance, the inability to walk heel to toe, nystagmus (uncontrolled rapid eye movements), difficulty alternating sequences of movements ("supinating" palm up and "pronating" palm down) and "slurred speech," i.e. dysarthria (Diener, & Dichgans, 1992). Visually guided movements such as reaching for or pointing to an object are disordered in what is described as an over-reaching or "hypermetria" of movement. Physiological studies of hypermetria have shown atypical patterns of muscle activation (Hallet, Shahani, & Young, 1975 a, b). They found consistent patterns of muscle activation in normal subjects with regard to the duration and sequencing of the activation of agonist and antagonist muscles: The agonist, then the antagonist, and then once again the agonist become active in a step-input tracking task, such as tracking a jumping pattern with a cursor on a computer screen. A consistent pattern was not seen in cerebellar patients, while an extended duration of activity of the first agonist burst was found. Eccles (1977) considers this overshooting of targets a timing problem.

Investigations of human and primate motor control divide cerebellar disorders of coordination for limb movement into those of timing and those of motor learning (Rosenbaum, 1990). An example of motor learning is the adaptation of the vestibular ocular reflex, a compensatory response to maintain eye focus on an object when the head is turned.(Rosenbaum, 1990). This adaptation has been examined in primates with cerebellar damage and found to be absent (Robinson, 1976). In addition, primate studies have found that lesions to the cerebellum reduce the capacity for visuomotor anticipation (Sasaki, Gemba, & Mizuno, 1982).

1.1.2 Speech production in cerebellar ataxia

Kinematic/electromyographic (EMG) studies of speech have investigated both normal muscle activation patterns (Harris, 1978; Tuller, Harris, & Kelso, 1981) and muscle patterns in ataxic dysarthric speakers (Ackermann, Hertrich, Daum, Scharf, & Spieker, 1997; (Ackermannn, Hertrich, & Scharf, 1995). In addition, several investigations have focused on the possible similarities and differences between limb movements and speech movements in ataxia. Although some investigations suggest a difference between the abnormal tendon reflexes and the intact jaw reflex in Friedrich's ataxia (Gentil, Devanne, Maton, & Brice, 1992; Salisachs, 1979), other studies which probe the intentional task of speech production have found that similar muscle pattern abnormalities occur in both limb and speech movement for cerebellar patients (Akermann et al., 1995; Ostry, Keller, & Parush, 1983).

1.2 Perceptual descriptions of ataxic dysarthria

Ataxia is one of the disorders of speech included among the dysarthrias. The term dysarthria refers to abnormal speech caused by damage to either the central or peripheral nervous system that results in motor dysfunction. In early descriptions of dysarthria (Darley, Aronson & Brown, 1969 a., b), the authors claimed that distinct patterns of disrupted speech correlated with specific neurological syndromes. To gain a unified rationale for description, the authors considered the muscular dysfunction as a basis for their description. Thus, dysarthria was classified as flaccid, spastic, ataxic, hypokinetic, hyperkinetic, and mixed. These various dysarthrias were then mapped onto different neurological syndromes. The description of each type of dysarthria, however, was based solely on the perceptual judgments of several speech and voice characteristics. The 10 speech and voice characteristics of ataxic dysarthria (Darley et al., 1969a), typified by poor coordination of the articulators, were grouped into three clusters: 1. articulatory inaccuracy (imprecise consonants, irregular articulatory breakdown, and distorted vowels); 2. prosodic excess (excess and equal stress, prolonged phonemes, prolonged intervals, and slow rate); and 3. phonatory-prosodic insufficiency (monopitch, monoloudness, and harsh voice). Although subsequent studies have supplemented the description of ataxic dysarthria with acoustic analyses (Hertrich & Ackermann, 1999; Ziegler & Wessel, 1996; Ackermann & Hertrich, 1994; Ziegler, Hartmann, & Hoole, 1993; Bell-Berti, Gelfer, Boyle, & Chevrie-Muller,1991; Ziegler & von Cramon, 1983; Kent, Netsell, & Abbs, 1979), they have not specified the Darley prosodic descriptors in acoustic terms nor correlated their analyses with the results of findings of neurological imaging.

1.3 Acoustic descriptions of ataxic dysarthria

The thirty-six speech and voice characteristics used in the perceptually based work of Darley et al. (1969a,b) has been supplemented by acoustic descriptions of ataxic speech that have included measures of duration as well as the onset and offset frequencies of the first and second formants.

In the study by Kent et al. (1979), five ataxic adults, varying in severity, all demonstrated a longer overall duration, as well as a restricted range, of durations for CV and CVC production, when compared with normals. These extended durations were syllables containing lax vowels that were often as long or longer than syllables containing tense vowels. Because normal speech contains a variety of syllable durations, the reduced range of syllable durations in ataxic speech is striking. Thus, the investigators concluded "timing" control to be a major problem in ataxic dysarthria. The authors claim that in ataxic dysarthria there is not only a slowing of motor behavior but also a disturbance of the normal relative temporal relations among the syllables within phrases and, to an extent, the phonetic segments within syllables (as in the case of the unusually long lax vowels). They found that ataxic subjects do not reduce syllables when it is appropriate to do so. In normal speech, one expects a decrease in syllable duration as the number of syllables in a word increases (Crystal & House, 1990). Kent and colleagues (1979) found that ataxic speakers did not adjust syllable durations as the number of syllables increased. Therefore, the authors claimed that it is in the flexible and responsive capacity for the sequencing of motor commands, as in syllable reduction, that ataxic speakers are most challenged.

Although early acoustic analysis of dysarthria caused by cerebellar damage found a disproportionate lengthening of the segment to be a fundamental property of ataxic dysarthria (Kent et al., 1979), we do not yet know whether this is a property specific to ataxic dysarthria, or present in all or some other dysarthrias. Recent acoustic analyses of speech and voice characteristics[1] of patients across a range of neurological syndromes suggest that many of the speech characteristics that Darley et al. (1969b) attributed to one or another neurological group can be found in patients with other neurological conditions

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[1] Temporal variability of syllable duration for place of articulation and the production of stress was found to correlate with degree of severity rather than to specific neurological syndromes (Ziegler, Hartmann & Hoole, 1993).

such as pseudobulbar palsy and cerebral vascular accidents linked to various sites within the central nervous system (Ziegler et al.,1993).

Kent et al. (1979) suggested that the lengthened, weakly stressed vowels, found in the acoustic analysis of ataxic speech, caused either a lack of, or incorrect, focus as a result of the disruption of the "normal" stress and timing patterns of speech. In addition, this investigation reported normal formant values for vowels within isolated words. We do not yet know whether this finding will hold for words within a phrase, or for syllables or words within sentences. From the examination of narrow band spectrograms of sentences, Kent et al. (1979) suggested a syllable-level planning with a falling F_0 on each successive syllable. Based on the lengthening of segments and syllables, the investigators claimed a disordered prosody for cerebellar subjects. Whereas many dysarthrias are characterized by the dysprosody of slow rate, ataxic dysarthria is distinguished by a slow speaking rate that exhibits a peculiar pattern, variously called scanning, staccato, singsong or measured (Kent et al., 1979).[2]

In a more recent acoustic investigation, Bell-Berti et al. (1991) examined phrase-final lengthening as a way of further describing the temporal irregularities of patients diagnosed with Friedrich's ataxia. Although the investigators found that the Friedrich ataxic subjects in their study did not produce phrase-final lengthening, it is not clear whether this is a specific result of the type of neurological degeneration or a pattern typical of speech that is produced at an unnaturally slow rate. In a follow-up study,

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[2] Various meanings have been given to the term "scanning speech" depending on the observers' description of behavior. Walshe (cited in Darley , Aronson & Brown, 1975, p.157) claims this term refers to the deliberate and separate production of syllables by the speaker, whereas,

normal speakers failed to produce phrase-final lengthening if asked to speak at an unnaturally slow rate (Bell-Berti, Gelfer, & Boyle, 1995).

1.4 Prosody in normal speech production

Historically, a critical issue in the study of speech motor control concerns the possible mechanism/s involved in the temporal flow of serially ordered articulatory events. Early investigations that examined the relation between vowel duration and formant structure were directed by a two-stage model that proposed that temporal order came after the serially-ordered events were generated (Lashley, 1951). This model was recognized in the work of Lindblom (1963) and predicts that vowel duration and formant frequency are monotonically linked. To explain vowel reduction, Lindblom (1963) claimed that invariant serially-ordered unit commands, recognized linguistically as idealized phonemes, are neurologically triggered but not realized under certain conditions because of timing constraints on the physiological task. Regardless of changes in rate or stress, the same effects on the movements of the articulators would occur since the timing-command signal is constant and invariant. Lindblom's work is most known for its explanation of "articulatory undershoot" and predicts that any reduction in duration from normal values would be accompanied by an undershoot of the articulators.

Early studies aimed at defining the perceptual correlates of stress examined segmental duration with the assumption that duration is a parameter of speech production

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Scripture (cited in Darley et al., 1975, p.157) believes it applies to the exaggeration of both strong and weak syllables.

that may be directly controlled by the individual speaker. The first of these studies (Fry, 1955) examined trochees where the variations in syllable stress signaled a change in meaning for the listener. For example, the contrast between SUBject (noun) and subject (verb) was dependent on the ratio of durations of the first to the second syllable in each word. Fry found that the ratio of the durations of the first to the second syllable of the perceived noun form was greater than that of the perceived verb form. Thus, he considered duration a salient aid to the perception of lexical stress.[3]

In a later investigation of segmental timing, Klatt (1976) investigated a variety of segments and their durations in English. This work resulted directly in models for manipulating the parameter of duration for synthetic speech systems. By examining acoustic measures of segmental timing in an attempt to define what variables influence listeners to make linguistic decisions, Klatt maintained that duration serves as a primary perceptual cue for signaling stress. Speech timing in Klatt's model is specified at several different levels in sentence production and provides information about the semantics, syntax, and segmental composition of a sentence. These durational differences are described in Klatt's model as a system of rules that, on the phonetic level, provide a distinction between short and long vowels, voiced and voiceless fricatives, phrase-final and non-final syllables, voiced and voiceless post-vocalic consonants, and stressed and unstressed (reduced) vowels.

Later studies did not find that duration and formant frequency were monotonically linked with duration as an external control on articulatory events. For example, Harris

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[3] In a follow up study in which both f_0 and duration were manipulated, f_0 was found to have a significant effect on listeners' perception of stress (Fry, 1958).

(1978) found that, contrary to the predictions of Lindblom's model, when either rate or stress was manipulated, syllable duration and vowel formant frequency varied independently in a non-monotonic relation. In addition, EMG studies showed reduced orbicularis oris and genioglossus activity for syllables of reduced stress (Tuller et al., 1981; Harris, 1971,1978). The conclusion drawn from these physiological and acoustic data of normal speakers is that vowel reduction can be attributed not only to temporal overlap of motor programs, but to events at the muscular level. The speech mechanism is thus considered to be controlled partly at the level of muscular activation.

The attractiveness of such a model of speech production is in the testability of its predictions. One prediction of such a theory is that movement and duration are not monotonically linked, and that duration does not control the extent of articulator movement. Speech scientists have, thus, turned to the coordinative structure (Kelso, Tuller & Harris, 1983; Harris, 1986), task dynamic theory (Kelso, Saltzman, & Tuller, 1986; Saltzman, 1986), and gestural phonology models (Browman & Goldstein, 1990; 1992) in attempts to address the physiology of the system as a "prime mover" in the task of speech production. The specific predictions posed by these theories suggest that any change in rate or stress may result in independent variations of syllable duration and formant values, as Harris (1978) previously suggested.

Investigations of pathological speech have given further support for the independence of duration and articulatory target. Ziegler and von Cramon (1983) examined vowel centralization in a group of eight closed-head-injured male subjects with concomitant dysarthria. The study described how the area of the patients' vowel formant space changed over time, from a reduced space to a larger one as the patients recovered.

Although vowel duration in traumatic dysarthria was judged by auditory examination to be considerably increased (slow speech), the articulators did not attain their targets in the initial stages of recovery from closed-head injury. The authors therefore concluded that temporal explanations alone do not account for why initial consonantal and vowel targets are not adequately realized.

1.4.1 Physiological studies of prosody in normal speech production

Recent investigations of articulatory dynamics of lengthened syllables in either phrase-final position or in a condition of stress provide further support for independent mechanisms of control. Two alternative hypotheses have been offered to explain the effects of stress on syllables in speech production (deJong, Beckman, & Edwards, 1993). One, the sonority hypothesis, suggests a linguistic explanation for feature representation in stressed syllables in which a change in the vowel target directly creates a more open vocal tract attributed to degree of jaw opening, and thus, "sonority." In contrast, the hyper-articulation hypothesis suggests that the observed decrease in coarticulation in a stressed CVC syllable is not related to the vowel in the vowel space.By examining the effects of stress production on coarticulation, deJong et al. (1993) found that a stressed vowel has a more extreme range of phonetic features in general, not just sonority. Thus, stress may be interpreted, in part, as a reduction in coarticulation[4] within a stressed syllable, resulting in the increased duration of that syllable.

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[4] Coarticulation is defined as the physiological dynamic event of two or more articulators moving simultaneously for different speech segments. This definition is supported in the work of Bell-Berti & Krakow (1991); Bell-Berti & Harris (1981), among others.

In contrast to the hyper-articulation and lengthening of the segments for stressed syllables in general, Edwards, Beckman, and Fletcher (1991) claim a reduced vowel space and greater length for phrase-final syllables. Not only is there a non-monotonic relation between duration and articulator movement for pathological speech (Ziegler and von Cramon, 1983), but phrase-final syllables in the speech of normals show increased durations with increased centralization and demonstrate a non-monotonic relation between duration and extent of articulator movement (Edwards et al., 1991).

Investigations that have adopted measures of increasing sensitivity to the underlying mechanisms of durational effects show that speakers may vary in the displacement and velocity of articulator movement for the same acoustical durational measure. Cohen, Beckman, Edwards, and Fourakis (1995) proposed several alternative models of articulatory dynamics in an effort to describe more completely the underlying mechanisms that cause temporal variations in speech production. They examined the syllable [pap] across six varying conditions of accent and placement within a sentence. More extreme displacements and faster movement velocities were found in stressed syllables when compared to more weakly stressed syllables. Although having a similar lengthening as stressed syllables, those syllables in phrase-final position did not show the same extended displacements and faster movement velocities. The investigators explain these kinematic differences as representative of two different mechanisms of control for lengthening effects. One mechanism is the control of the end-point target and is evident in the relation between velocity and amplitude, whereas the other is the control of settling time and is captured in the relation between velocity and duration. Thus, whereas stressed syllables result in a pattern of hyper-articulation (control over end-point target), syllables in phrase-final position may be lengthened because of an overall slowing down of the speech mechanism (control over settling time).

1.4.2 Acoustic predictions for normal speech prosody

The components of prosody have been defined as the acoustic features of F_0, segment duration, amplitude and segmental quality or reduction, whose variation is said to signal constituent boundaries and syllable prominence. For an extensive review of issues in defining prosody see Shattuck-Huffnagel and Turk (1996).

The findings by Cohen et al. (1995) for six different conditions of prominence present a pattern that may be used as a basis for acoustic investigation. The investigators found that although a monotonic relation between duration and the displacement of the articulators (the duration and extent of jaw opening in the production of the syllable [pɑp]) may serve to describe the accented and unaccented contrast, this pattern does not describe the relation between phrase-final accented and non-phrase-final accented conditions. Moreover, the velocity of movement was distinctly different between conditions of phrase-final accented lengthening and non-phrase-final accented lengthening. The kinematic analysis suggests that a slowing of articulation occurs in phrase-final lengthening, whereas syllables of increased duration, like accented syllables in non-final position, show an increase in movement velocity and amplitude.

The six conditions of prosodic prominence include a range of values for syllable duration, with the greatest lengthening expected for phrase-final syllables and the shortest duration expected for the reduced syllable. It is expected that these durational differences will be found in the speech of the normal subjects of the proposed study. If we accept the kinematic findings of Cohen et al. (1995), the longest duration will be found in phrase-final accented syllables, followed by syllables in accented and unaccented positions with the following order: nuclear accented, non-phrase-final accented, non-phrase-final unaccented, post-nuclear unaccented, and the reduced syllable.

The formant measures for normal speakers are expected to reflect the prediction of a hyper-articulation for the more accented syllables, as represented in extreme values for F1 and F2. The more prominent [pɑp] syllables will be produced with higher F1 and F2 frequencies than the less prominent syllables. These differences in values are predicted based on the acoustic characteristics found in the production of the American English vowel [ɑ] (Hillenbrand, Getty, Clark, and Wheeler, 1995; Peterson, & Barney, 1952). What we may expect is a hyper-articulation vs. a centralization across syllables of different degrees of prominence: non-final accented (*-pf+a*) vs. non-final unaccented (*-pf-a*), nuclear accented (*+n+a*) vs. post-nuclear unaccented (*-n-a*), and post-nuclear unaccented (*-n-a*) vs. reduced (*red*) syllables.

Cohen and colleagues (1995) did not report on the fundamental frequency (f_0) values in their investigation of articulatory dynamics. Since the present study seeks an acoustic signature for prosodic differences between accented and unaccented syllables, phrase-final and non-final syllables, and unaccented and reduced syllables, measures of the fundamental frequency will be examined along with the measures of syllable duration and formant frequencies. The fundamental frequency is expected to correlate directly to the level of stress, but not necessarily to the amount of lengthening. For example, the lengthened phrase-final (*+pf+a*) syllable is expected to be longer with a lower f_0 than the slightly shorter but also lengthened accented (*-pf+a*) syllable, which is expected to have a higher f_0.

We predict, for comparison "a" (*+pf+a* vs. *-pf+a*) that the frequencies of F1, F2 and the fundamental frequency will be higher for *–pf+a* than for *+pf+a* and that the duration of *+pf+a* will be greater than that of *-pf+a.* For comparison "b" the frequencies of F1, F2 and the fundamental frequency will be higher and the duration greater for *-pf+a* than for *–pf-a.*. For comparison "c" the frequencies of F1, F2 and the fundamental frequency will be higher and the duration greater for *+n+a* than for *–n-a.* For comparison "d" the frequencies of F1, F2 and the fundamental frequency will be higher and the duration greater for *–n-a* than for *red.*

METHOD

2.1 Subjects

Two groups of subjects were selected for the study: Six subjects were individuals diagnosed by a neurologist as having cerebellar degeneration with accompanying ataxia of both limb and speech and six subjects were selected as normal matched controls. The six cerebellar subjects were also classified by neurological imaging as having cerebellar degeneration. Several different sub-types of cerebellar degeneration have been discussed in the literature. (See Plaitakias, Katoh & Huang 1992, for an extensive review of these sub-types.) The present study included three subjects with Friedreich's ataxia (JS, MC and ES), one subject with olivo-ponto cerebellar degeneration (WD), one subject with pure-recessive cerebellar degeneration (AH), and one subject with cerebellar degeneration of unknown etiology (PP). Thus, all six pathological subjects presented with cerebellar degeneration with accompanying limb ataxia and ataxic dysarthria, although of different etiologies. All twelve subjects spoke English before the age of twelve years. The normal subjects were pair-wise matched to the ataxic speaker by age, sex, dialect, and educational status

Ataxic speaker AH: This speaker was a 68-year-old Caucasian male diagnosed with a form of cerebellar degeneration known as pure-recessive cerebellar degeneration. The subject was born and raised in New York, and is a retired teacher with a graduate education. Fig. 2.1 through 2.3 present three images (midsagittal section, coronal section and transverse section) that were used for a rating of cerebellar degeneration. Table 2.1 presents the radiologist's rating of involvement for cerebellar vermis and hemispheres, brainstem and spinal cord. This subject was matched to normal control LJR (see Table 2.2).

Ataxic speaker PP: This speaker was a 36-year-old African-American male diagnosed with cerebellar syndrome of unknown etiology. The subject was raised in New York City after having been born in London. He is a high school graduate with two years of college education.. Fig. 2.4 through Fig. 2.6 present three images (midsagittal section, coronal section and transverse section) that were used for a rating of cerebellar degeneration. Table 2.1 presents the radiologist's rating of involvement for cerebellar vermis and hemispheres, brainstem and spinal cord. This subject was matched to normal control GC (see Table 2.2).

Ataxic speaker WD: This speaker was a Caucasian male, aged 72 years and diagnosed with olivo-ponto-cerebellar atrophy. The subject was born and raised in South Carolina and then moved to New York City at the age of 22 years. His education includes a graduate degree in Library Science. Fig. 2.7 through Fig. 2.9 present three images (midsagittal section, coronal section and transverse section) that were used for a rating of cerebellar degeneration. Table 2.1 presents the radiologist's rating of involvement for cerebellar vermis and hemispheres, brainstem and spinal cord. This subject was matched to normal control BW (see Table 2.2).

Ataxic speaker JS: This speaker was a Caucasian female ataxic speaker, aged 35 and diagnosed with Friedreich's ataxia. She was born and raised in Long Island, New York and is a sibling of ataxic speaker ES. Fig. 2.10 through Fig. 2.12 present three images (midsagittal section, coronal section and transverse section) that were used for a rating of cerebellar degeneration. Table 2.1 presents the radiologist's rating of involvement for cerebellar vermis and hemispheres, brainstem and spinal cord. This subject was matched to normal control YM (see Table 2.2).

Ataxic speaker ES: This speaker was a Caucasian female ataxic speaker aged 40 years and diagnosed with Friedreich's ataxia. The subject was born and raised in Long Island, New York. Her sibling is ataxic speaker JS. She is a lawyer and has been in intensive and ongoing speech therapy. Fig. 2.13 through Fig. 2.15 present three images (midsagittal section, coronal section and transverse section) that were used for a rating of cerebellar degeneration. Table 2.1 presents the radiologist's rating of involvement for cerebellar vermis and hemispheres, brainstem and spinal cord. This subject was matched to normal control LL (see Table 2.2).

Ataxic speaker MC: This speaker was a 29-year-old female of Italian-American decent and diagnosed with Friedreich's ataxia. She was born in Italy and moved to Brooklyn, N.Y. at the age of 3 and has spoken English since then. This subject is a high-school graduate. Fig. 2.16 through Fig. 2.18 present three images (midsagittal section, coronal section and transverse section) that were used for a rating of cerebellar degeneration. Table 2.1 presents the radiologist's rating of involvement for cerebellar vermis and hemispheres, brainstem and spinal cord. This subject was matched to normal sibling FC (see Table 2.2).

2.1.1 Magnetic resonance imaging data of cerebellar degeneration

Each of the cerebellar ataxic subjects received Magnetic Resonance Imaging (MRI) for analyses of the sites of atrophy within the nervous system. Sites of nervous system involvement judged to be affected in relatively different patterns across subjects included the cerebellar vermis and hemispheres, pons, medulla, and spinal cord. Figures 2.1 through 2.18 show differential involvement of these structures as captured through imaging and rated by radiologist Adam Silver, M.D., at Mt. Sinai Medical Center.

All six cerebellar subjects of this investigation presented with ataxia and cerebellar atrophy. Selected structures: cerebellar vermis, brainstem(pons and medulla), cerebellar hemispheres and cervical spinal cord were rated for degree of atrophy. Although all subjects rated were found to have significant involvement of the cerebellum, the pattern varied among subjects. Three of the subjects presented with cerebellar degeneration of different etiologies and three presented with Friedreich's ataxia (FA), a form of cerebellar degeneration with predominant atrophy to the cervical spinal cord. Plaitakis et. al (1992) suggest that for Friedreichs' sub-type a de-afferentation between the spinal cord and the cerebellum occurs with the progression of the disease. The imaging data reported here clearly show a rating scale difference for spinal cord involvement for those patients with FA and those diagnosed with cerebellar degeneration of the OPCA and pure recessive sub-types. All patients showed significant involvement of the cerebellar vermis.

Table 2.1 represents the radiological rating of cerebellar degeneration for six cerebellar subjects of this study. The degree of degeneration was determined by the degree of atrophy on a scale of 0 to 3 with 0 representing no involvement and 3 representing significant involvement. Three images were selected for each of the six patients (See Figures 2.1 through Fig. 2.18). The first of the three images for each subject is the midsagittal section. The vermis, brainstem and spinal cord were viewed and rated by a radiologist. Table 2.1 describes the value given for these areas for each of the six cerebellar subjects. The second image is a coronal section at the level of the 4th ventricle and the 3rd image is a transverse section at the level of the medulla. These last two images were used to provide a rating of hemispheric involvement.

Midsagittal image of the left hemisphere

The most apparent finding on the midsagittal section for all subjects is the atrophy of the cerebellar vermis. Although cerebellar atrophy is a consistent morphologic alteration, the magnitude and pattern varies from one syndrome to another. In Friedreich's ataxia the atrophy is more focally expressed whereas in olivo-ponto cerebellar atrophy the atrophy is more severe and diffuse. This atrophy primarily involves the central lobule, cumen, declive and tuber with some widening of the primary fissure. The pyramid and uvula remain relatively intact. Other structures labeled include the pons (Po), medulla (Md) and the 4th ventricle (4V).

Coronal

In the second view, a coronal section through the 4th ventricle, atrophy of the cerebellar hemispheres (Cer) is the most noticeable finding. Other structures labeled include the 4th ventricle (4V) and the lateral ventricle (LV).

Transverse

The third image is a transverse section through the medulla oblongata with a view of the cerebellar hemispheres, nodulus and vermis. This view was used by the radiologist to support the rating for cerebellar hemisphere involvement.

Table 2.1: Radiological rating of cerebellar degeneration: A scale of 0-3 was used to estimate cerebellar involvement of vermis and hemispheres as viewed on midsagittal, coronal and transverse sectioned MRI images. Brainstem and spinal cord were given + or – rating for involvement or non-involvement respectively.

	Etiology	**Degree of involvement for selected neural structures**			
Patient	Pure recessive cerebellar degeneration	**Cerebellar Vermis**	**Cerebellar Hemispheres**	**Brainstem**	**Spinal - cord**
AH	Unknown	2	2	+	-
PP	Olivo-ponto-cerebellar atrophy	3	3	-	-
WD	Friedreich's ataxia	3	2	-	-
JS	Friedreich's ataxia	2	2	-	+
ES	Friedreich's ataxia	2	2	+	+
MC		2	1	-	+

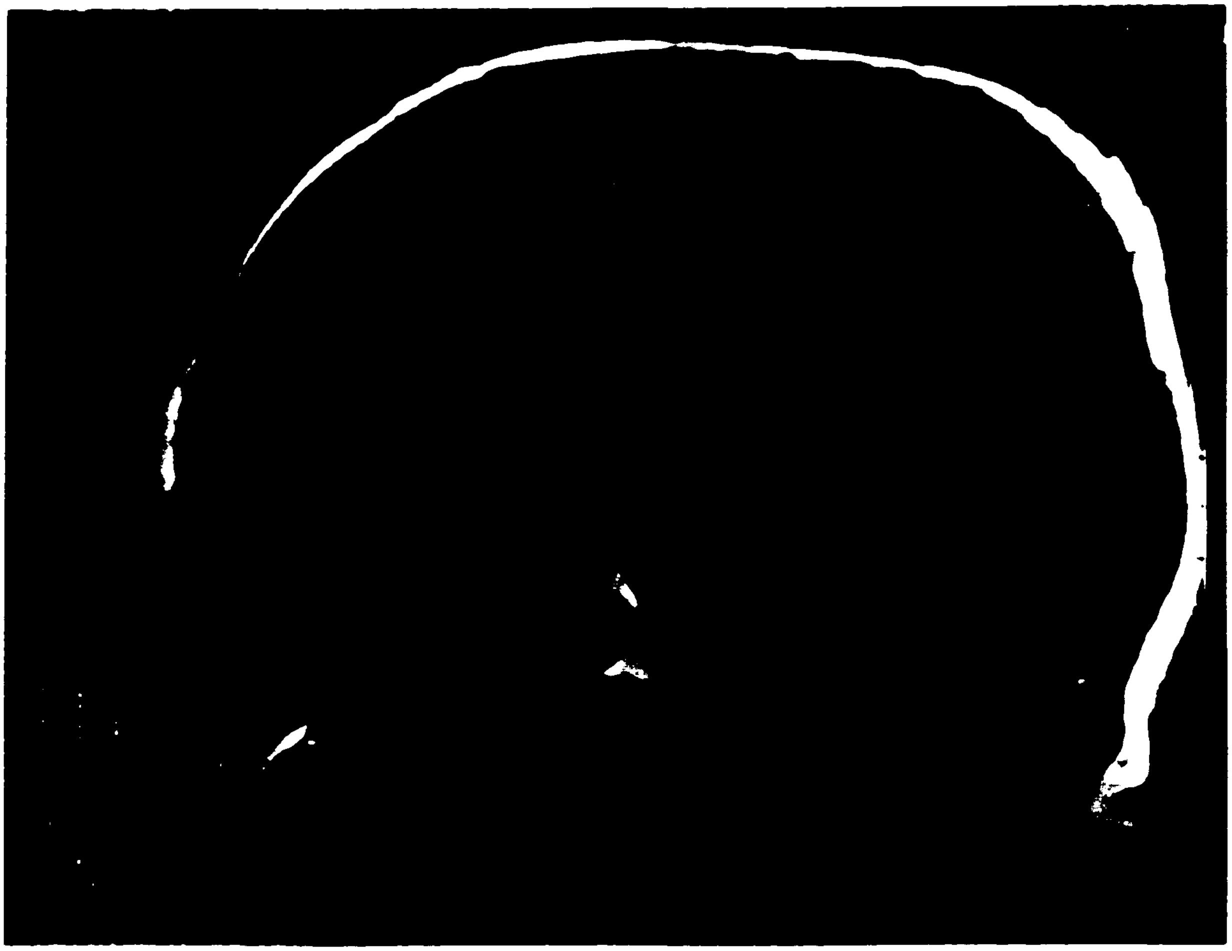

Fig.2.1: Midsagittal section of the left side of the brain in ataxic male subject AH. The most apparent finding is atrophy of the cerebellar vermis involving the primary degeneration of the central lobule (Ce), cumen (Cu), declive (D) and tuber (Tu). In addition there is a widening of the primary fissure (1F). The pyramid and uvula remain relatively intact. Other structures labeled include the pons (Po), medulla (Md) and the 4th ventricle (4V).

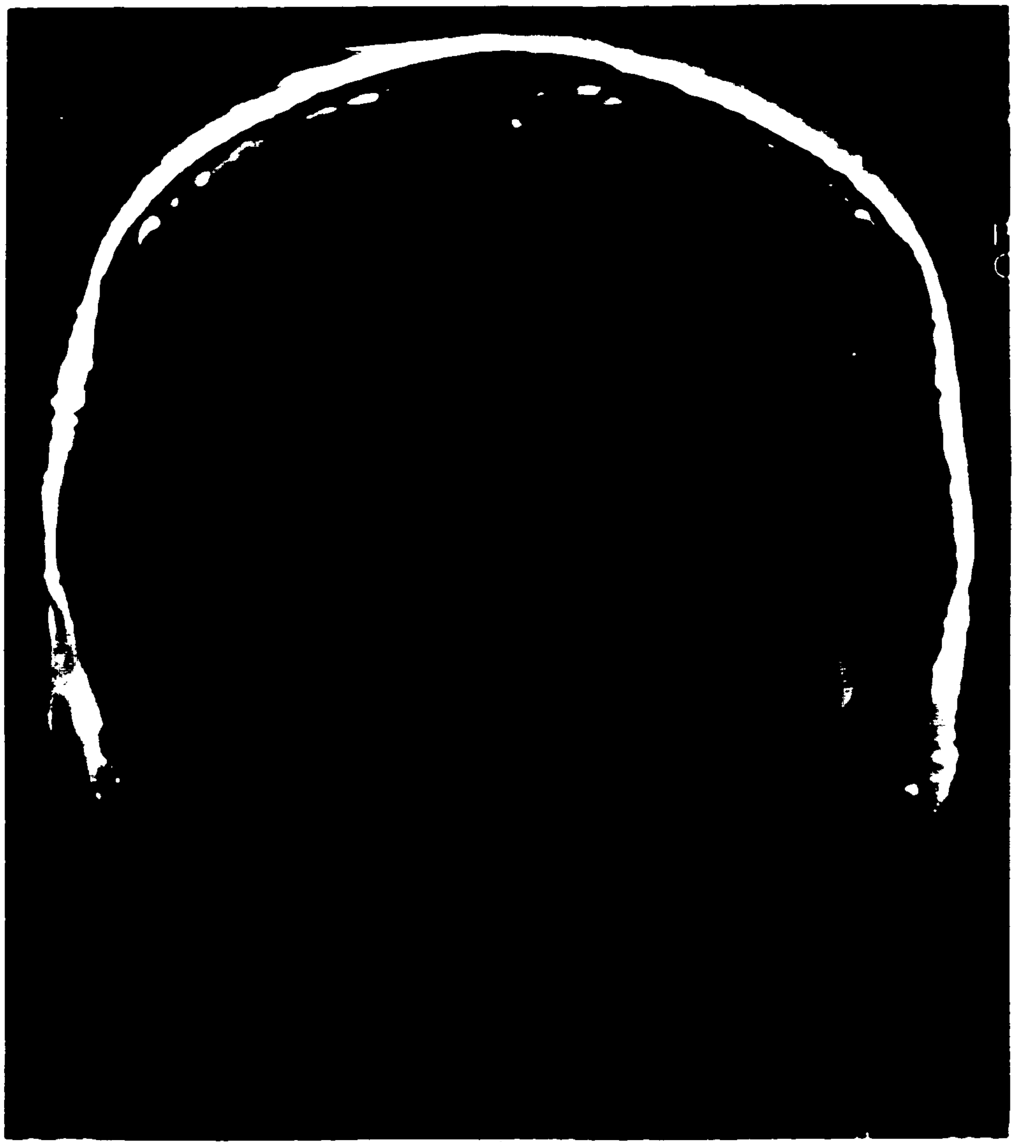

Fig. 2.2: Coronal section (T1) through the 4th ventricle in ataxic male subject AH. Atrophy of the cerebellar hemispheres (Cer) is the most noticeable finding. Other structures labeled include the 4th ventricle (4V) and the lateral ventricle (LV).

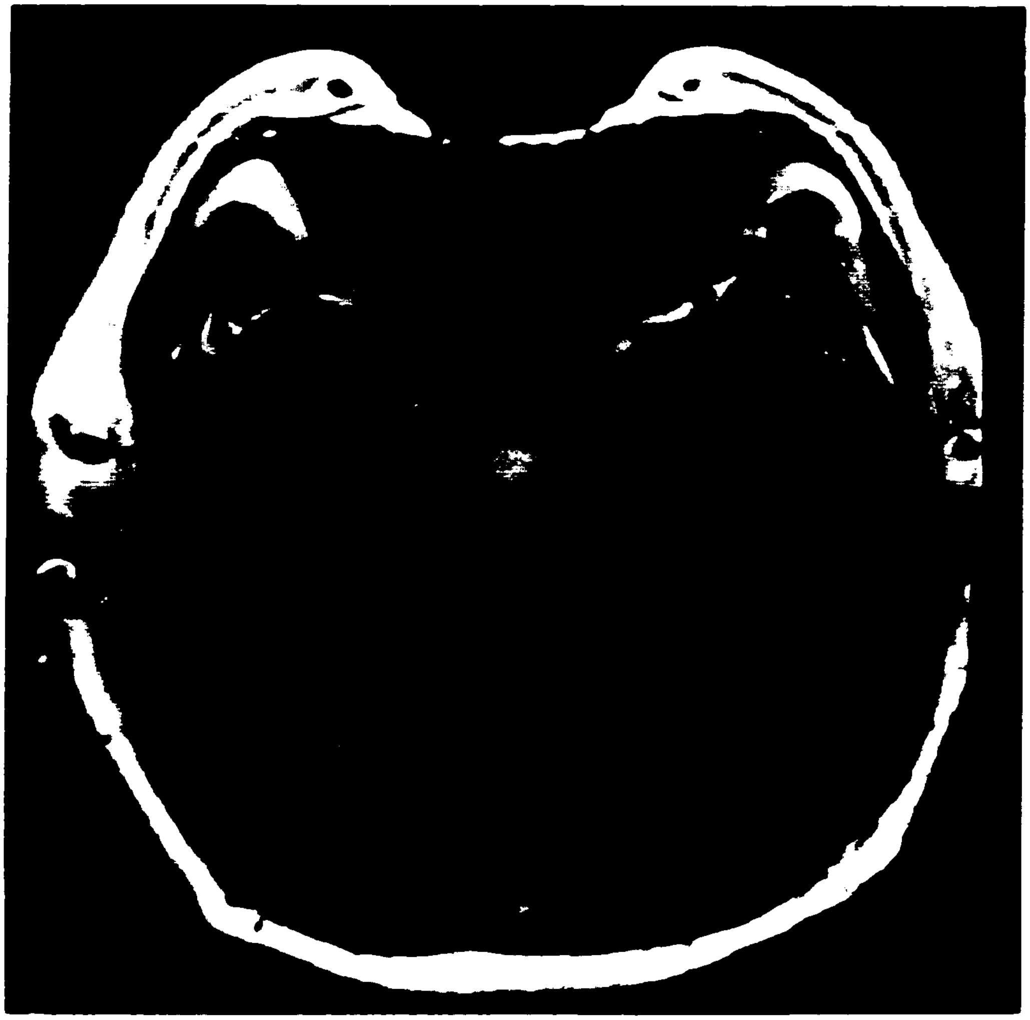

Fig. 2.3: Transverse section at the level of the medulla in ataxic male subject AH. Structures labeled include the medulla oblongata (Md), nodulus (No), vermis (Ver) and the cerebellar hemispheres (Cer).

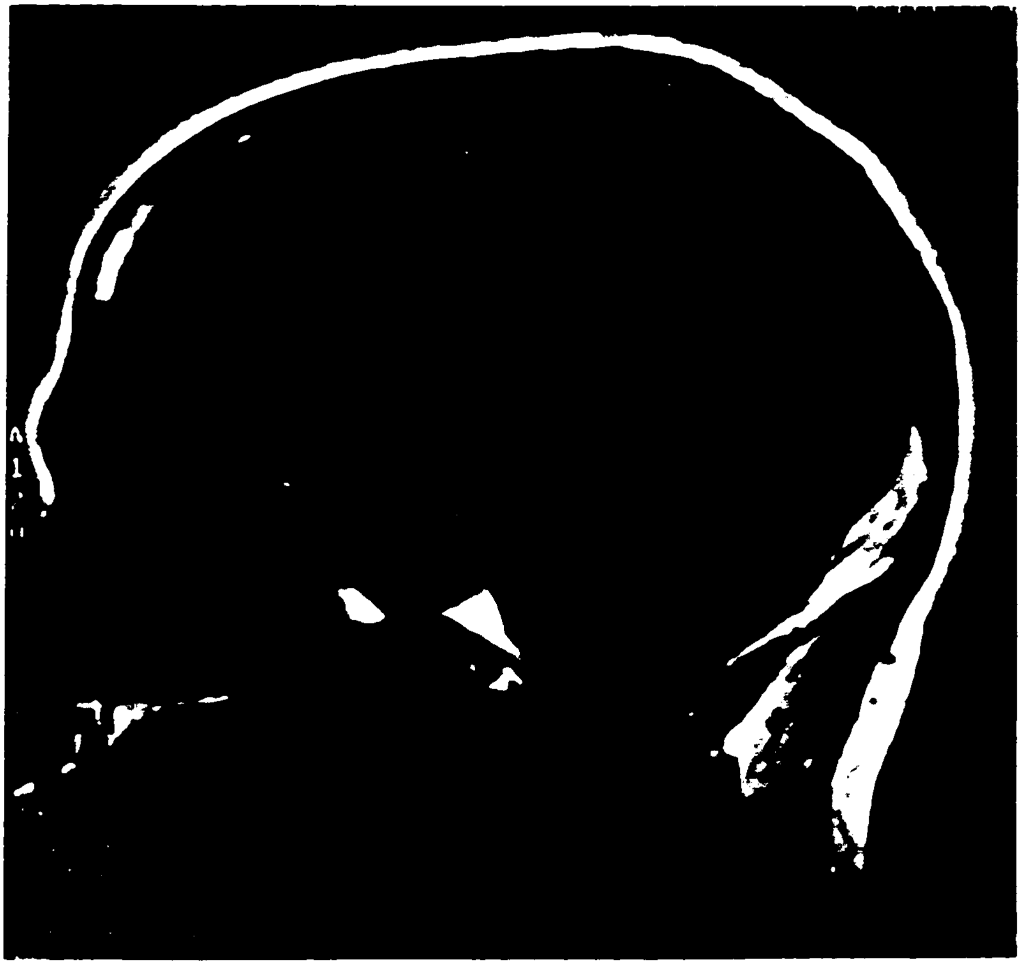

Fig.2.4: Midsagittal section of the left side of the brain in ataxic male subject PP. The most apparent finding is atrophy of the cerebellar vermis including primary degeneration of the central lobule (Ce), cumen (Cu), declive (D) and tuber (Tu). Other structures labeled include the pons (Po), medulla oblongata (Md) ,spinal cord (Sc) and the pyramid (P) and the uvula (U) of the vermis.

Fig. 2.5: Coronal section through the 4th ventricle (4V) of ataxic male subject PP. Atrophy of the cerebellar hemispheres (Cer), the most noticeable finding is of the "coarse comb" type. The lateral ventricle (LV) is also labeled.

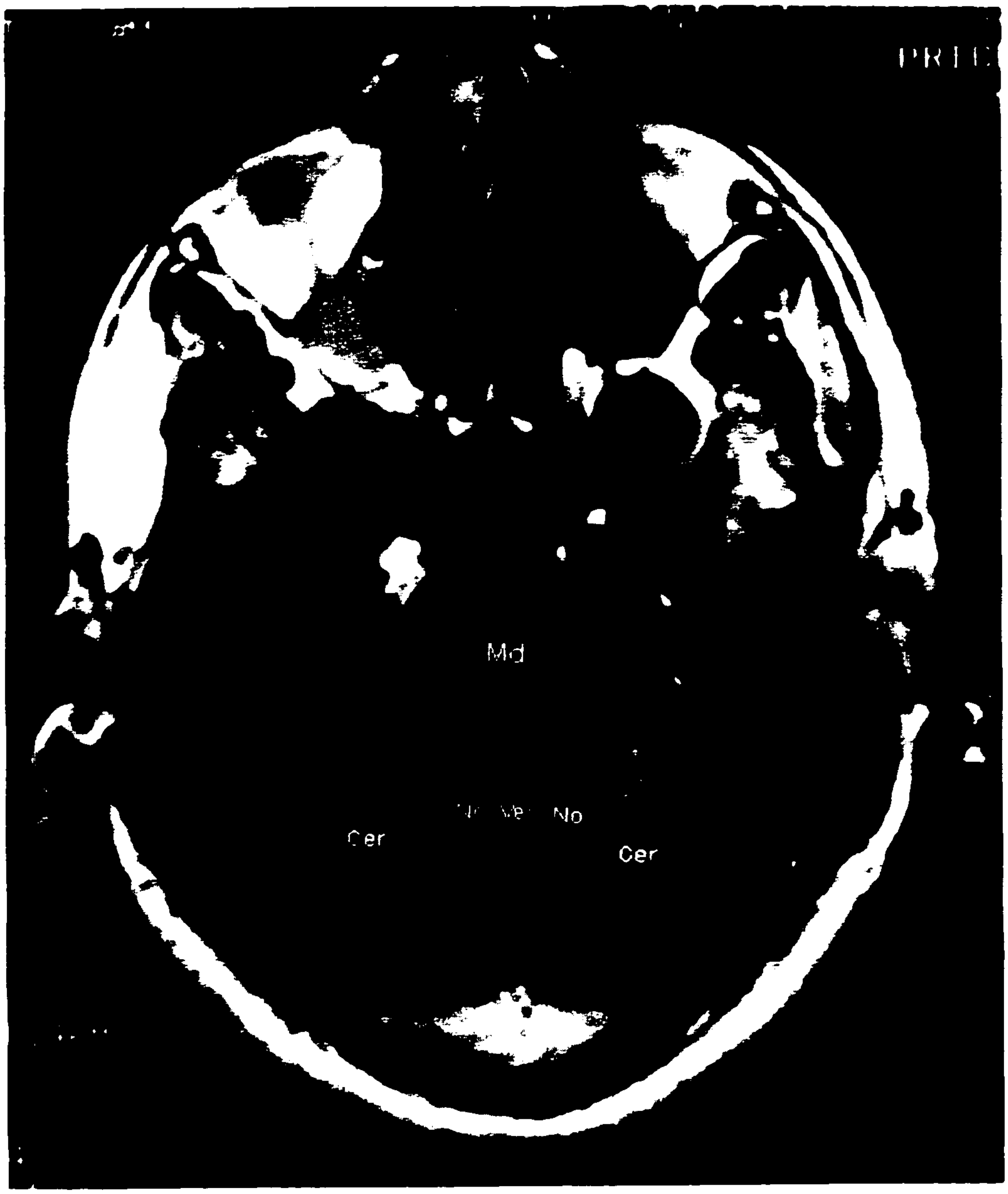

Fig.2.6 : Transverse section at the level of the medulla in ataxic male PP. Structures labeled include the medulla oblongata (Md), nodulus (No), vermis (Ver) and cerebellar hemispheres (Cer).

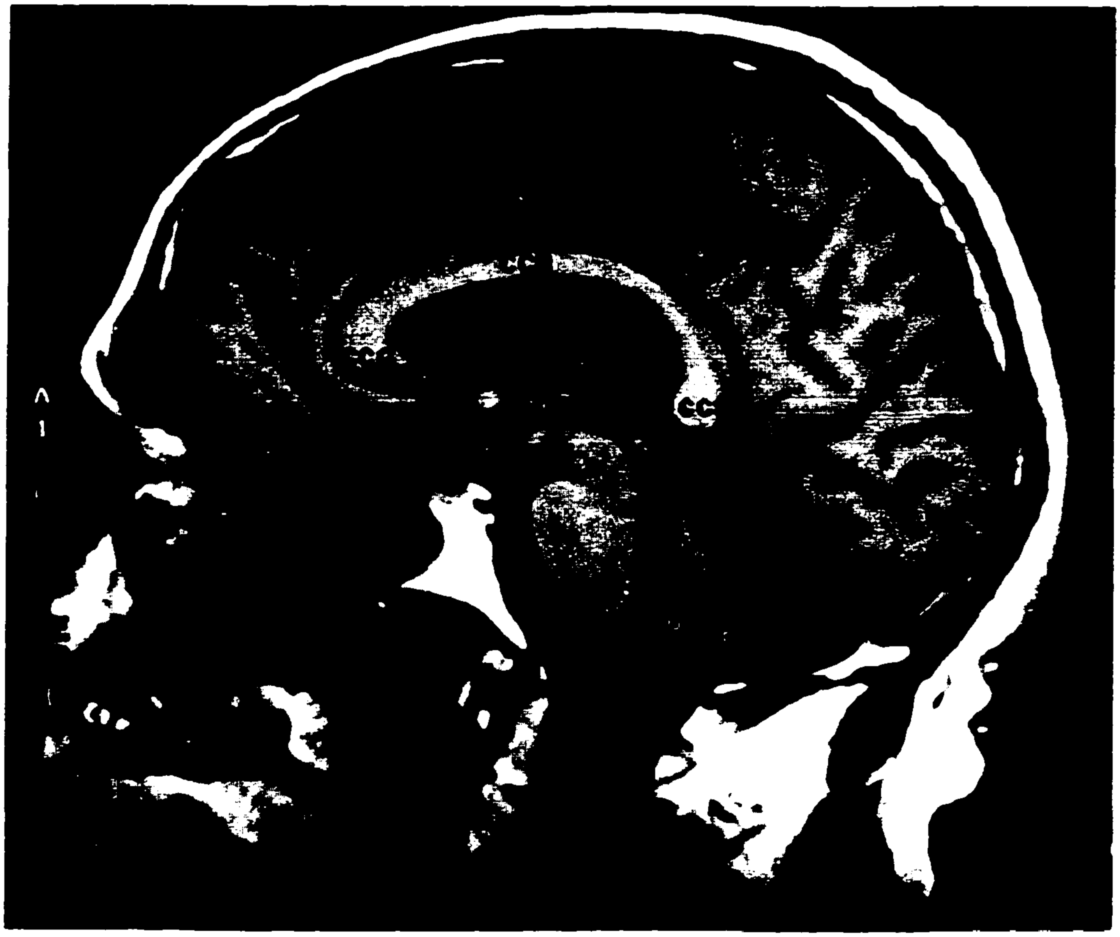

Fig. 2.7: Midsagittal section of the left side of the brain in ataxic male subject WD. The most apparent finding is atrophy of the cerebellar vermis including primary degeneration of the central lobule (Ce), cumen (Cu), declive (D) and tuber (Tu). In addition, there is a widening of the primary fissure (1F). The pyramid (P) and uvula (U) remain relatively intact. Other structures labeled include the pons (Po), medulla (Md), 4th ventricle (4V), corpus callosum: (genu (CCG), body (CCB) and splenium (CCS)), thalmus (Th) and hypothalmus (Hy Th).

Fig. 2.8: Coronal section (T2) through the 4th ventricle of ataxic male subject WD. Atrophy of the cerebellar hemispheres (Cer) is the most noticeable finding. Other structures labeled include the tonsils (To), 4th ventricle (4V) superior colliculi (Sc), inferior colliculi (Ic), and the hippocampus (Hi).

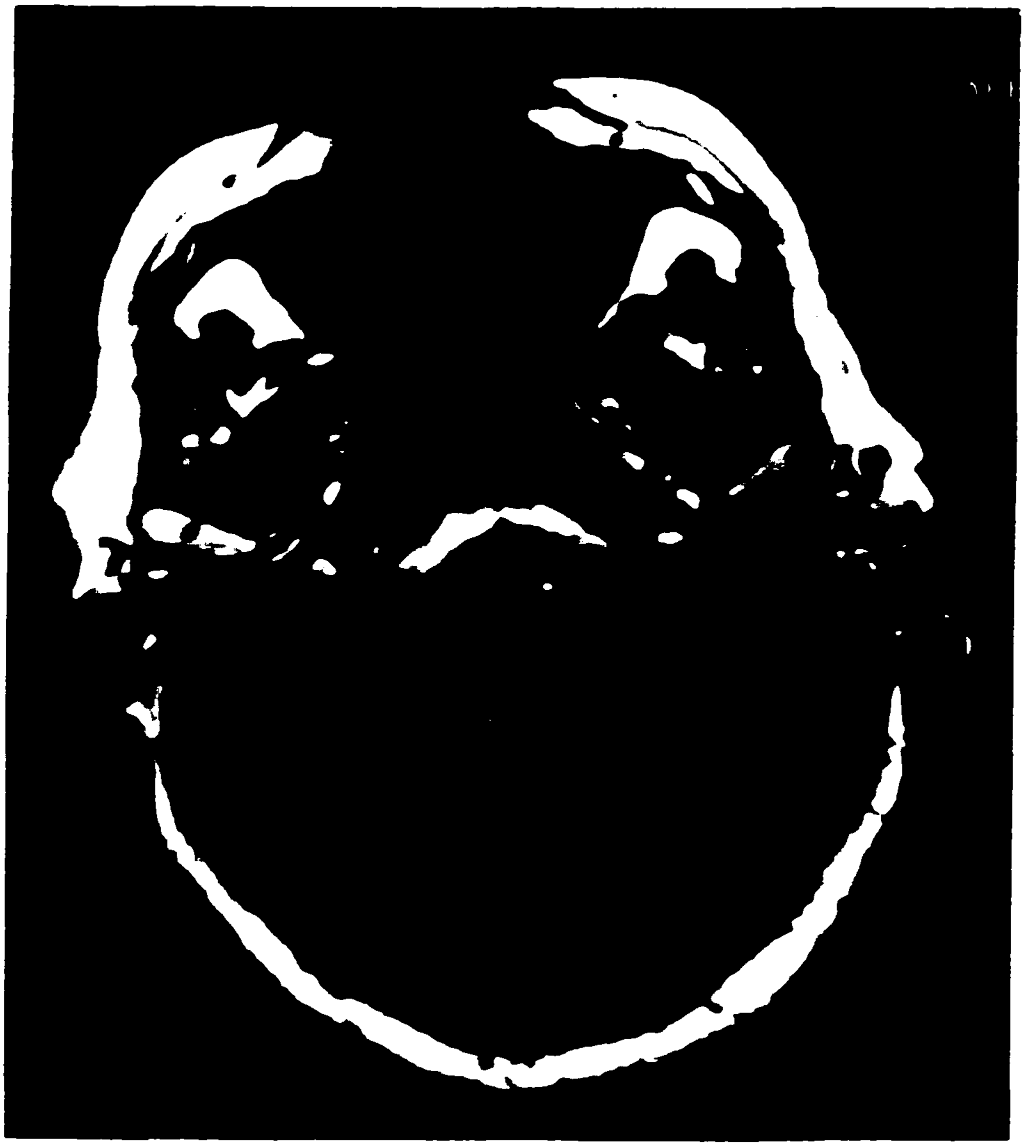

Fig. 2.9: Transverse section at the level of the medulla in ataxic male WD. Structures labeled include the medulla oblongata (Md), nodulus (No), Vermis (Ver) and the cerebellar hemispheres (Cer).

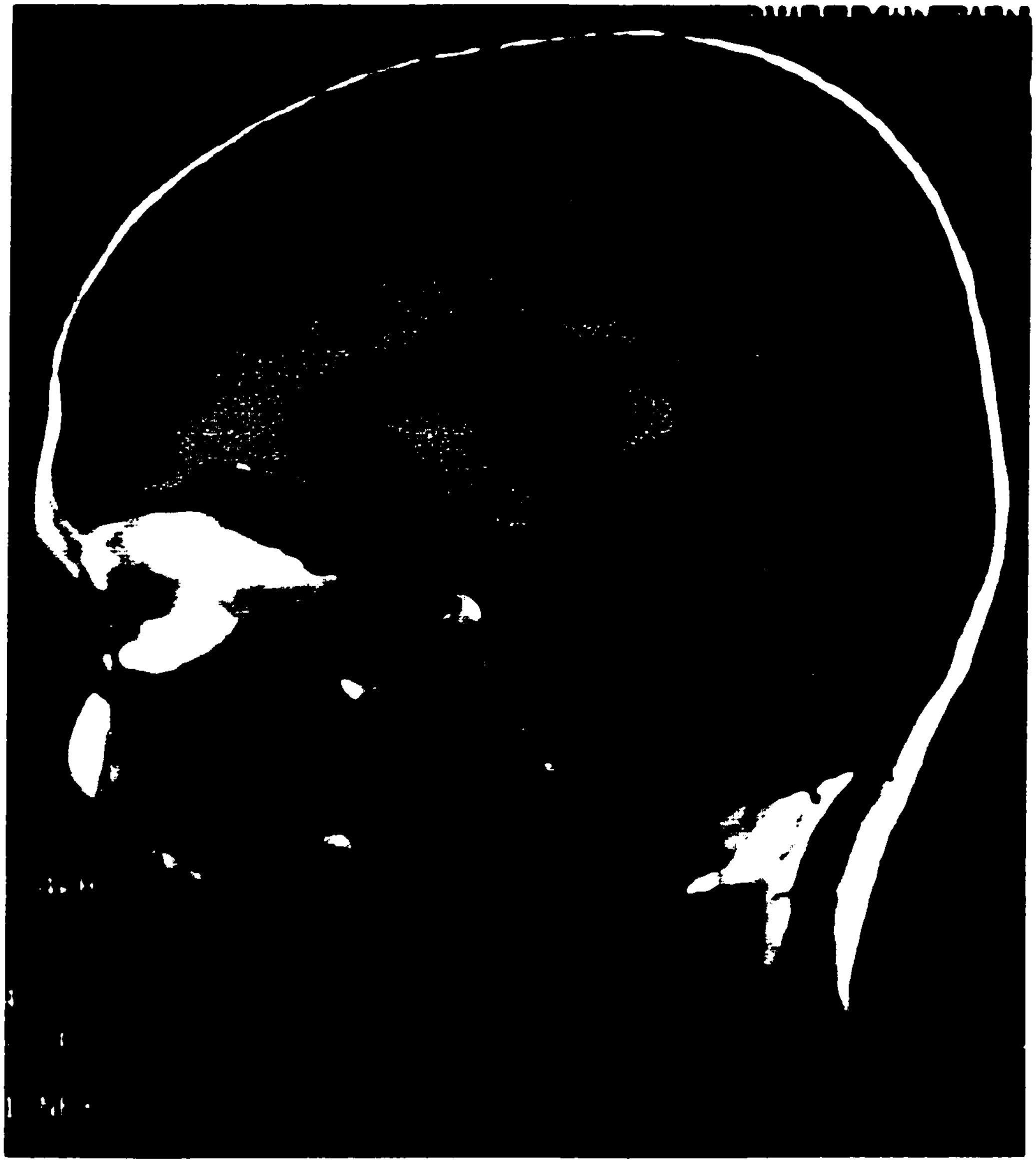

Fig.2.10: Midsagittal section of the left side of the brain in female ataxic, of the Friedreich's subtype, subject JS. The most salient feature is atrophy of the cervical cord. The cerebellar vermis shows atrophy primarily in the central lobule (Ce), cumen (Cu), declive (D) and tuber (Tu). Other structures labeled include the primary fissure (1F). the pons (Po), medulla (Md) and spinal cord (Sc).

Fig.2.11: Coronal section through the 4th ventricle of ataxic female subject JS. Atrophy of the cerebellar hemispheres is the most noticeable finding. There also appears to be cortical atrophy as evidenced by the increased amount of cerebrospinal fluid and and size of the lateral and fourth ventricle. Other structures labeled include the dentate nucleus (Den), 4th ventricle (4V) and the lateral ventricle (LV).

Fig.2.12: Transverse section of ataxic female JS through the 4th ventricle. Structures labeled include the cerebellar hemispheres, nodulus (No) and vermis (Ver).

Fig.2.13: Midsagittal section of the left side of the brain in ataxic subject (Friedreich's sub-type) ES. The most apparent finding is atrophy of the spinal cord. Atrophy of the cerebellar vermis is recognized in the degeneration of the central lobule (Ce), cumen (Cu), and declive (D). In addition, there is a widening of the primary fissure (1F). The pyramid (P) and the uvula (U) remain relatively intact. Other structures labeled include the pons (Po), medulla oblongata (Md) and the cervical spinal cord (Sc).

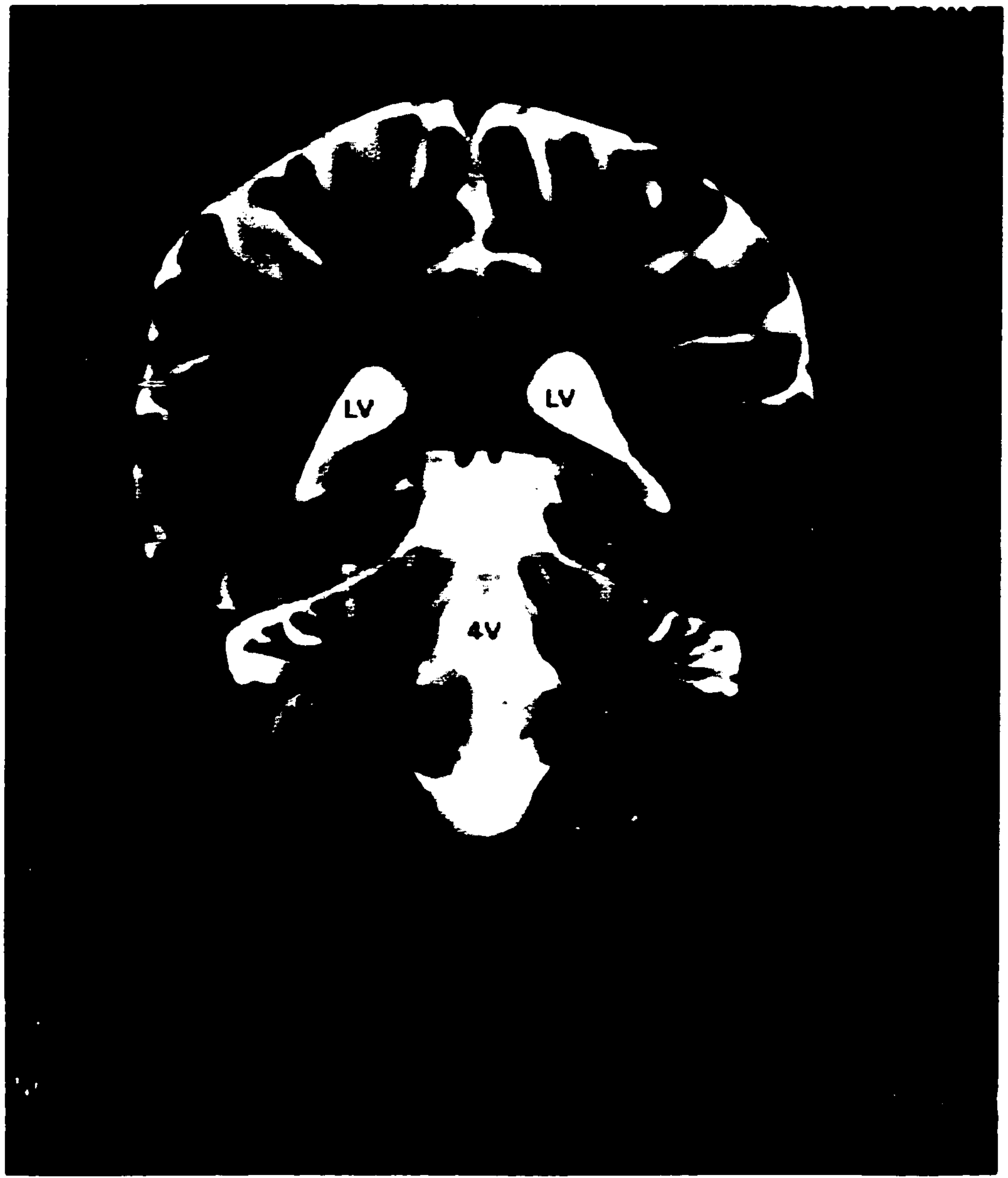

Fig. 2.14: Coronal section through the 4th ventricle of ataxic subject ES. Atrophy of the cerebellar hemispheres is the most noticeable finding. Other structures labeled include the lateral ventricle (LV) and the 4th ventricle (4V).

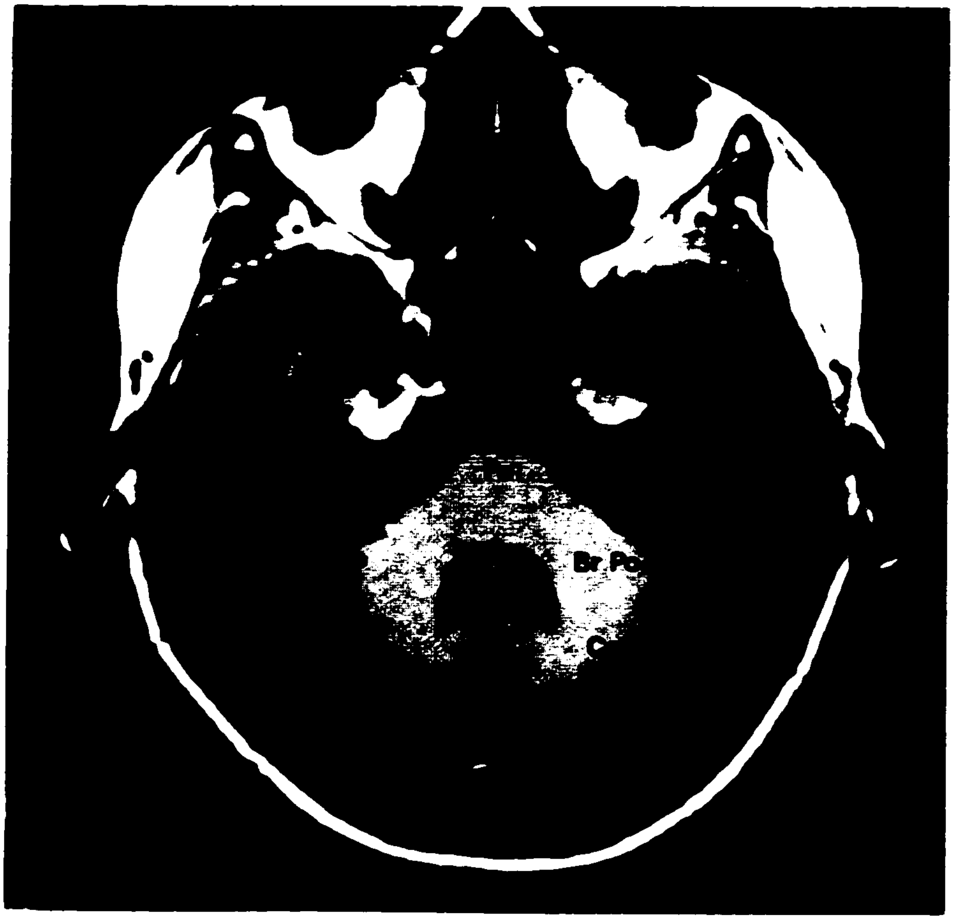

Fig.2.15: Transverse section through the 4th ventricle of ataxic female ES. Structures labeled include the cerebellar hemispheres (Cer), vermis (Ver), 4th ventricle (4V), brachium pontis (Br Po) and the pons (Po).

Fig.2.16: Sagittal section of the left side of the brain in ataxic subject MC. The most apparent finding is atrophy of the spinal cord (Sc). Atrophy of vermis is limited to the central lobule (Ce), cumen (Cu) and declive (D). The tuber (Tu) and pyramid (P) remain relatively intact. Other structures labeled include the primary fissure (1F), 4th ventricle (4V), pons (Po) and medulla oblongata (Md).

Fig. 2.17: Coronal section (T2) through the 4th ventricle (4V) of ataxic subject MC. Atrophy of the cerebellar hemispheres is the most noticeable finding. Other structures labeled include the superior colliculi (Sc) and the inferior colliculi (Ic).

Fig.2.18: Transverse section through the 4th ventricle of ataxic female MC. Structures labeled include the cerebellar hemispheres (Cer), vermis (Ver), 4th ventricle (4V), brachium pontis (Br Po) and the pons (Po).

Table: 2.2 **Age, sex , and neurological status of the normal and cerebellar ataxic subjects. N: normal subject ; A: cerebellar ataxic subject. Subjects designated by the same number formed a matched pair {eg.: AH (A1) & LJR (N1)}.**

Subject	**Sex**	**Age**	**Neurologic Classification**
AH (A1)	male	68 years	Pure cerebellar
PP (A2)	male	36 years	Cerebellar degeneration
WD (A3)	male	72 years	Olivo ponto cerebellar atrophy
JS (A4)	female	32 years	Friedreich's ataxia
ES (A5)	female	40 years	Friedreich's ataxia
MC (A6)	female	29 years	Friedreich's ataxia
LJR (N1)	male	61 years	Normal
GC (N2)	male	34 years	Normal
BW (N3)	male	70 years	Normal
YM (N4)	female	27 years	Normal
LL (N5)	female	35 years	Normal
FC (N6)	female	34 years	Normal

2.2 Materials

The speech materials used were a series of sentences presented on printed cards. The sentences read by the subjects were identical to those used by Edwards et al. (1991); Beckman and Edwards (1994) and Cohen et al., (1995). (See Table 2.3). Each subject read these sentences as a response to the experimenter's question. The target syllable CVC [pɑp] was embedded within each sentence. Each question sentence was designed to elicit a different prosodic response from the Ss as they produced this same target (CVC) syllable. The fifth sentence contained two samples of the syllable: *-n-a* and *red.* Ten sentences for each prosodic condition were pseudo-randomized for a total list of 50 sentences and 60 tokens of the target syllable.

Table 2.3. Five questions and response sentences used to elicit prosodic contrasts.

Prosodic Condition	Question	Response
1. Phrase-final accented *(+pf+a)*	**Who** was opposing the question?	**Pop,** opposing the question strongly, refused to answer it.
2. Non-phrase-final accented *(–pf+a)*	**Who** was posing the question?	**Papa**, posing the question loudly, refused to answer it.
3. Non-phrase-final unaccented *(-pf-a)*	Who **posed** the question?	**Papa** posed the question loudly, and then refused to answer it.
4. Nuclear-accented *(+n+a)*	Did her **mama** pose a problem as far as their getting married?	Her **papa** posed a problem.
5. Post-nuclear-unaccented *(-n-a)* and Reduced *(red)*	Did **his** papa pose a problem as far as their getting married?	**HER** **pap/pa** posed a problem.

2.3 Instrumentation and recording protocols

Speakers were recorded using a Sony digital tape recorder (Model TCD-D7) and a Shure (Model SM10A) head-band unidirectional dynamic microphone. Recordings were made in an acoustically suitable conference room.

Subjects were presented with a consent form (Appendix C) with a general description and instructions about their participation in the experiment. After being read a set of instructions designed to elicit the prosodic contrasts, each subject was presented with a stimulus question.

After being asked the elicitation question the subject read the target sentence aloud. The questions and sentences were presented in a pseudo-randomized order. The question and subsequent response were repeated if the subject hesitated or misread the sentence.

To insure the optimum elicitation of the prosodic contrasts, the experimenter read the following instructions to each subject: "You are being asked to say a series of fifty sentences. I will say a number for record-keeping purposes and then I will ask you a question. Please listen carefully to the question. Then you will answer the question, the way it would come naturally, with the entire sentence that appears on the printed card." The stimulus questions were then presented with emphasis on the word in bold-face type (see Table: 2.3).

2.4 Acoustic measurements

All measures were made using the Kay Elemetrics Computerized Speech Lab (CSL). The signal was captured from the DAT recorder to the CSL with the sampling rate set at 20,000 Hz.. The displayed waveform was edited to display the target syllable [pɑp]. The syllable [pɑp] in six different conditions was measured for (1) the duration of [pɑp], (2) f_0 (fundamental frequency) and the frequencies of (3) F1 and (4) F2 of [ɑ] at mid-point of the vowel segment. A total of 4 measures was thus made for each syllable; this resulted in 240 measures per subject. and 2,880 measurements for the 12 subjects.

Bandwidth for wide-band spectrograms was determined on a "gender-specific" basis with the intent of using the narrowest appropriate bandwidth . The speech of the male speakers was analyzed with a bandwidth of 146 Hz. and that of the female speakers with a bandwidth of 293 Hz. Each utterance was edited by deleting the waveform after the word "opposing", "posed" or "posing" (See Table 2.3).

The acoustic measures were selected to provide information about the speech characteristics of subjects with cerebellar pathology and of normal subjects regarding:

I) the effect of cerebellar pathology on the production of prosody: the control of timing and the frequencies of F2, F1 and f_0 in syllable production in four pairs of contrastive conditions : 1. accented lengthening for syllables in phrase final (*+pf+a*) and non-final positions (*-pf+a*); 2. lengthening for stressed syllables in accented (*-pf+a*) and unaccented (*-pf-a*) contexts; 3. syllable duration for accented nuclear (*+n+a*) and unaccented post- nuclear (*-n-a*); and 4. vowel reduction for unaccented (*-n-a*) vs. schwa syllables (*red*);

II) the relation among durational changes and spectral dynamics and f_0.

2.4.1 Measurement of syllable duration

Duration measures of [pɑp] were made by hand from high-resolution gray-scale digital spectrograms using standard measurement criteria (Hillenbrand et al., 1995; Peterson and Lehiste, 1960). As the syllable structure of the CVC is /pVp/, visual inspection of the wide-band spectrograms provided a clear display of the bursts of the initial and final stops. The syllable durations were measured from the release burst of the initial consonant to the release burst of the following /p/ of the (CVC) for all conditions except the reduced vowel condition. Durations in this condition were measured from the release burst initiating the second syllable of "pa-pa" to the burst of the initial /p/ of the following word "posed": "papa posed." (Appendix B: Fig. A represents the segmentation of the CVC) .

2.4.2 Measurement of formant frequencies

The F1 and F2 frequencies were used as the basis for inferring vocal tract configuration with regard to mouth opening and degree and location of constriction. Formant frequencies were measured at the mid-point of the phonated segment of the syllable.

Fourier analysis was used on the acoustic signal using a combination of wide-band spectrographic analysis, Linear Predictive Coding (LPC) frequency response and Fast Fourier Transform (FFT) to resolve ambiguities: F1 and F2 were not always separated and there was a occasional overlap of F1 and f_0 at around 250-300 Hz for the female speakers.

Determination of mid-point of the vowel

Measurements of the F1 and F2 frequencies were made at the mid-point of the phonated segment of the CVC. The choice of mid-point is consistent with standard measuring procedure and recommended if reports on pathological speech are to be comparable with existing published data (Baken,1987). The CSL can display several windows simultaneously (e.g., waveform and spectrogram) so that the measurement of frequencies may be made at the same point in time from the waveform. The mid-point was determined by simultaneously viewing the waveform and the spectrogram. The onset of periodicity viewed from the waveform as increased amplitude served as the initial marker for a measure of the phonated segment of the syllable. In those cases where point of increased amplitude of periodicity in the waveform was difficult to determine, the spectrogram was used to estimate the onset of the phonated segment by viewing the presence of resonance expressed in formant bands. The cursors on the waveform and the spectrogram were then time-linked at the location of the initial point of the phonated segment. The duration of the phonated section was calculated from the waveform as the difference between the initial point and the point of critical damping, that served as the end-point marker of phonation offset. The duration of the phonated segment, in ms., was then halved and used to calculate the mid-point.

A wide-band power spectrum at the calculated mid-point was generated using Linear Predictive Coding (LPC). The LPC frequency response method proved inadequate for the analysis of the vowel [ɑ]: Because of the proximity of the first and second formants, this analysis often showed only one peak. This peak could be interpreted as either the F1 or F2. To resolve the ambiguous LPC and the wide-band spectrogram, a narrowband Fast Fourier Transform (FFT) display was generated. This analysis displayed the amplitudes of the harmonics at the calculated midpoint. The decision was made to select the two harmonic peaks as a discrete measure of the first and second formants as this would be the most consistent form of measurement across subjects (L.J. Raphael, personal communication). This method was used for all subjects, even those whose first and second formants did not merge in the LPC display.

There were several problems in deciding on a system of measurement for the vowel [ɑ] in the syllable [pɑp]. Although both Peterson and Barney, 1952 and Hillenbrand et al., 1995 (see Table: 2.4) have reported acoustic measures for this vowel on a large data base, those studies examined [ɑ] within the syllable "hod" [hɑd] rather than [pɑp].

Furthermore, there remains a large amount of variability reported not only for age and gender but also within individual speakers (Peterson & Barney, 1952; Hillenbrand et al., 1995). The present study examined the same vowel in different conditions of stress.

Thus, if differences were to be measured and found significant the procedure of measurement had to be consistent both within and across subjects as well as a measure of the desired event: F1 and F2 resonance.

The FFT analysis was used to resolve ambiguities in the LPC and wide-band spectrogram displays. The decision to view this spectrum was made based on the understanding that emphasis of different harmonics results from vocal tract resonance response. Baken (1987) defines formants as a local maximum in the vocal tract transfer function. It is the single frequency at which the vocal tract transmission is more efficient than at nearby frequencies.

> ...a formant can be operationally defined as a peak in the displayed amplitude spectrum that is not due to source spectrum properties. ...the vocal signal which is the wave usually being transmitted, has a line spectrum. That is, it has significant energy only at discrete (harmonic) frequencies. So the formant becomes that point on the frequency scale where a harmonic has greater amplitude than its neighbors. ... the frequency of the harmonic does not necessarily fall at exactly the true frequency of a formant peak. ...Individual harmonics serve as "samples" of the vocal tract's resonant responses. (Baken, 1987: p.354).

Table: 2.4. Acoustic averages for the values of f_0, F1 and F2 for [ɑ] within the syllable "hod".

Peterson & Barney, 1952				Hillenbrand et al., 1995		
Sex	**f_0**	**F1**	**F2**	**f_0**	**F1**	**F2**
Male	124 Hz.	730 Hz.	1090 Hz.	123 Hz.	768 Hz.	1333 Hz.
Female	212 Hz.	850 Hz.	1220 Hz.	215 Hz.	936 Hz.	1551 Hz.

The measurement of the F1 frequency was taken as the frequency of the first harmonic peak in the narrow band FFT display. The protocol for measurement of the F2 frequency was to select the next highest hamonic peak after the one used to determine F1. For most speakers, the harmonics' amplitudes dipped at frequencies above the F1 harmonic and then rose again. This helped to identify a pattern that, when present, was used to determine the point of measurement for the F2 (see Appendix B). On a subject-by-subject basis the harmonic pattern varied. In a few cases, a high amplitude harmonic would occur in close proximity to the first harmonic peak, with frequency values in the expected F1 frequency range. In these cases, the next highest harmonic peak was selected. A check of both wide-band and narrow band spectrograms, was used to find a range that would support the choice for the frequency of this harmonic peak. For consistency in measurement the cursor was always placed at the left edge of the peak harmonic bar in the FFT display.

2.4.3 Fundamental frequency: f_0

Fundamental frequency (f_0) was calculated with the "pitch extraction" routine of the of Kay Elemetrics Computerized Speech Lab (CSL). f_0 was measured at the mid-point of the phonated segment of the vowel.

2.4.4 Brain imaging data

All six cerebellar subjects received magnetic resonance imaging (MRI) within one month of the recording of the acoustical sample. This brain imaging technique allows more accurate and differential diagnoses among different neurological syndromes, as well as a comparative measure of involvement within a particular syndrome. Previously, classification of cerebellar disorders was based on post-mortem examination or behavioral symptoms. Neuroimaging allows a description of morphologic changes at various stages of disease in living patients, thus providing a better understanding and evaluation of disease processes. A patient who presents with ataxia may now be linked to a description of the underlying neuroanatomical site of pathology. For cerebellar patients, the exact site and degree of atrophy may vary from one individual to another. The imaging data provide information that contributes to definitive diagnoses not possible before. For example, atrophy of the cerebellum and its pathways has been found to be significantly greater in patients with Friedreich's ataxia than normal control subjects (Junck , Gilman, Gebarski, Koeppe, Kluin & Markel, 1994). Moreover, brain imaging techniques have clearly shown patterns of atrophy that allow a differential diagnosis to be made among different cerebellar disorders, including Friedreich's ataxia and Olivo-ponto-cerebellar atrophy (Plaitakis & Huang, 1992).

2.5 Statistical design

The design was one between and one within-subject repeated measures analysis of variance. Given the independent variables of diagnosis and prosodic condition, there are three effects that can be tested: the two main effects of diagnosis (D) and stress (S), and one second-order interaction. These are outlined in Table 2.5 . The between-subjects factor, diagnostic condition, has two values, cerebellar damage vs. normal. The within-subjects or repeated measures factor, "prosodic stress condition," comprises 6 "conditions": (1) *+pf+a*, (2) *–pf+a*, (3) *–pf-a*, (4) *+n+a*, (5) *–n-a* and (6) *red* (see Table 2.7).

The dependent variables (Tables 2.8, 2.9, 2.10 and 2.11) were average durations of the CVC, the average F1 and F2 frequencies at the midpoints of the phonated vowel segment of the CVC utterance, and average f_0 at midpoints of the CVC. In addition to testing for the main effects of each of these variables as well as their interaction, there was a series of "focused comparisons" or "sub-effects" that were examined using selected levels of the repeated measures factor. More specifically, these comparisons involved the following linguistic stress conditions: (1) *+pf+a* vs. *–pf+a*, (2) *–pf+a* vs. *–pf-a*, (3) *+n+a* vs. *-n-a*, and (4) *–n-a* vs. *red* (Table 2.6). Moreover, differences between the cerebellar damage and normal subjects were compared across the particular levels of prosodic dominance implied by these four comparisons. Stated somewhat differently, in addition to the two main effects and their interaction, a subset of first-order interaction effects or "simple interactions" was tested.

Pearson rank-order correlations were used to investigate the relation between cerebellar degeneration and speech deterioration. An estimate of the degree of cerebellar involvement for each of several areas: cerebellar, brainstem and spinal cord as viewed on anatomical MRIs were rated by a radiologist (Table 2.1) Numerical ratings ranging from 0 (least involvement) to 3 (most involvement) were used as an estimate of cerebellar (vermis and hemispheres) involvement and + or – symbol was used to assess involvement of the brainstem and spinal cord. A value for the average score of cerebellar hemispheres and vermis was then calculated for each subject (Table 3.33). For the correlation analyses the average measure as calculated from the ratings for vermis and hemispheres was used. The brainstem and spinal cord ratings were not considered.

The data from the cerebellar subjects were also analyzed for a measure of speech deterioration. As an individual measure of speech deterioration, a "D" statistic was generated for each of the cerebellar subjects. This statistic, also known as the Euclidean Distance Metric, was used for an overall rating of speech deterioration for each of the six cerebellar subjects. The degree of deterioration as represented by the "D" score was based on the differences among all four contrast values (*+pf+a* vs. *–pf+a; -pf+a* vs. *–pf-a; +n+a* vs. *–n-a; -n-a* vs. *red*) between each cerebellar speaker and his/her normal matched control:

Equation #1: **Euclidean Distance Score - D**

Cer.Subj.$_D$=(Cer: [+*pf*+*a*] – [–*pf*+*a*] – Nor:[+*pf*+*a*] – [-*pf*+*a*])^2 + (Cer:[-*pf*+*a*] – [-*pf*-*a*] – Nor: [-*pf*+*a*] – [-*pf*-*a*])^2 + (Cer:[+n+a] – [-n-a] – Nor: [+*n*+*a*]- [-*n*-*a*])^2 + (Cer: [-*n*-*a*] – [*red*]– Nor: [-*n*-*a*]– [*red*])^2 = x ; $\sqrt{\ }$ x = D

In addition, a sub-analysis **for each contrast, the "d" score, was also measured for degree of difference between each subject and his/her matched control.**

Equation #2: **Raw Distance Score - d**

Cer. Subj.$_d$ =(Cer:[+*pf*+*a*] – [–*pf*+*a*]) – (Nor:[+*pf*+*a*] – [-*pf*+*a*]) = d

Cer. Subj.$_d$ =(Cer:[-*pf*+*a*] – [-*pf*-*a*]) - (Nor:[-*pf*+*a*] – [-*pf*-*a*]) = d

Cer. Subj.$_d$ =(Cer: [+*n*+*a*] – [-*n*-*a*]) – (Nor: [+*n*+*a*] – [-*n*-*a*]) = d

Cer. Subj.$_d$ =(Cer: [-*n*-*a*] – [*red*]) –(Nor: [-*n*-*a*] – [*red*]) = d

Table 2.5. One between x one within-repeated measures anova

Source of variance	**df**	**Error term**	**df**
Between- Subjects Factor **Diagnostic condition**	1	**Subject group**	10
Within- Subjects Factors **Stress condition**	5	**S X subj. (groups)**	50
Interaction between **stress** and **diagnostic** condition	5	**S X subj. (groups)**	50

Table 2.6. **Within-subject comparisons of prosodic conditions**

Phrase-final lengthening	*+pf+a* [pɑp] vs.–*pf+a* [pɑp]
Accented lengthening	*-pf+a* [pɑp] vs. –*pf-a* [pɑp]
Syllable duration	*+n+a* [pɑp] vs. *-n-a* [pɑp]
Reduction	*-n-a* [pɑp] vs. *red* [pəp]

Table 2.7. **Between-subject comparisons of prosodic conditions of stress within the intonational hierarchy**

Cerebellar	*+pf+a*	*-pf+a*	*+n+a*	*-pf-a*	*-n-a*	*red*
Normal						

Notation Explanation:
+pf+a= **phrase-final accented**
+n+a = **nuclear accented**
-pf+a= **non phrase-final accented**
-pf-a = **non phrase-final unaccented**
-n-a =**post-nuclear unaccented**
red= **reduced**

Table 2.8. Mean F1 values (Hz) of

+pf+a /pɑp/ [ɑ] $^{\text{medial}}$
+n+a /pɑp/ [ɑ] $^{\text{medial}}$
-pf+a /pɑp/ [ɑ] $^{\text{medial}}$
-pf-a /pɑp/ [ɑ] $^{\text{medial}}$
-n-a /pɑp/ [ɑ] $^{\text{medial}}$
red [pəp] [ə]$^{\text{medial}}$

Table 2.9. Mean F2 values (Hz) of :

+pf+a /pɑp/ [ɑ] $^{\text{medial}}$
+n+a /pɑp/ [ɑ] $^{\text{medial}}$
-pf+a /pɑp/ [ɑ] $^{\text{medial}}$
-pf-a /pɑp/ [ɑ] $^{\text{medial}}$
-n-a /pɑp/ [ɑ] $^{\text{medial}}$
red [pəp] [ə]$^{\text{medial}}$

Table 2.10. Mean f0 values of:

+pf+a /pɑp/ [ɑ] $^{\text{medial}}$
+n+a /pɑp/ [ɑ] $^{\text{medial}}$
-pf+a /pɑp/ [ɑ] $^{\text{medial}}$
-pf-a /pɑp/ [ɑ] $^{\text{medial}}$
-n-a /pap/ [ɑ] $^{\text{medial}}$
red [pəp] [ə]$^{\text{medial}}$

Table 2.11. **Mean duration values (ms.) of:**

phrase-final accented: *(+pf+a*) [pɑp]
nuclear accented: *+n+a* [pɑp]
non-final accented: *-pf+a* [pɑp]
non-final unaccented: *-pf-a* [pɑp]
post-nuclear unaccented: *-n-a* [pɑp]
reduced: red [pəp]

Notation Explanation: [pɑp] = release burst of initial stop to the release burst of the final stop of the CVC. The reduced [pəp] was measured from the burst of the second syllable of "pa-pa" to the burst of the initial segment of the following word "posed": "papa posed".

RESULTS

The purpose of the present study was to investigate, acoustically, the effects of cerebellar pathology, as identified by neurological diagnoses and neuroimaging techniques, on certain aspects of speech prosody. This study compares selected acoustic features of syllables under various conditions of accent and phrase position for two groups of subjects: normals and those with cerebellar disorder.

Measures of syllable duration, formant frequency, and fundamental frequency were made to gain an understanding of the properties of the CVC syllable [5] in speakers with cerebellar pathology. These data were then compared to those obtained from the analysis of speech produced by normal speakers.

The following abbreviations are used in the presentation of the study:
Ner= neurological status; Pros. = prosodic conditions; *+pf+a* = phrase-final accented; *-pf+a* = non-final accented; *-pf-a* = non-final unaccented; *+n+a*= nuclear accented; *-n-a* = post-nuclear unaccented and *red* = reduced vowel; CVC = consonant-vowel-consonant syllable; Cer.= cerebellar ataxic speakers; Nor.= normal speakers.

Two questions asked are: 1. Do syllable duration, fundamental frequency, and prosodically conditioned formant frequencies, as a function of syntactically determined position, differ between normals and patients diagnosed with cerebellar ataxia? 2. If they differ, what is the nature and degree of the difference between:

a. accented syllables in phrase-final (*+pf+a*) vs. non-phrase final (*-pf+a*) position;

b. accented (*-pf+a*) vs. unaccented syllables (*-pf-a*) in non phrase-final positions;

c. nuclear accented (*+n+a*) vs. post-nuclear unaccented (*-n-a*) syllables; and

d. post-nuclear unaccented (*-n-a*) vs. reduced (*red*) CVC syllables (i.e., full versus reduced vowels)?

Section 3.1 examines the effects of prosodic condition on syllable duration for both normal and cerebellar ataxic speakers. Section 3.2 examines the effects of prosodic condition on F1 and F2 for normal and cerebellar ataxic speakers. Section 3.3 examines the effect of prosodic condition on fundamental frequency for normal and cerebellar ataxic speakers. Section 3.4 examines the correlations between cerebellar degeneration and speech deterioration.

For each section, the statistical analysis of the acoustic data is divided into three analyses of variance. The first study examines the three main effects: the effect of neurological status, the effect of prosodic condition, and the interaction between these two.

In the second analysis, the interaction is studied with pair-wise comparisons focused on mean differences of neurological status and then mean differences of prosodic conditions. The third and final analysis looks at the first order interaction effects or simple interactions as found in four focused comparisons of the six prosodic conditions and the effect of neurological status. Each of the four contrasts is examined for

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[5] In this study the term syllable is used to refer to the CVC sequences in the speakers' production of "pop", "papa" and "papa posed".

the four acoustic measures of syllable duration, F1, F2 and f_0 frequencies at the mid-point of the phonated vowel segment.

3.1 The effects of prosodic condition on syllable durations

Durations for the sequence [pɑp] produced by the twelve subjects across six different prosodic conditions were examined. Three main effects were examined for the measure of syllable duration: the effect of neurological condition, the effect of prosodic condition, and the interaction of these conditions. Main effects were found to be significant for all of the measures of syllable duration (Table 3.1): the effect of neurological status: $p=.003<.01<.05$; prosodic conditions: $p=.000<.001<.05$ and the interaction between neurological status and prosodic condition: $p=.000 < .001<.05$. The significant main effect found for the interaction of neurological status with prosodic condition is the most important finding. Because the interaction of neurological condition and prosodic condition was found to be significant, the pair-wise comparisons are therefore examined in detail.

Table 3.2 presents the findings of neurological status for the acoustic measure of syllable duration. The mean value for syllable for duration and the mean difference between the two groups is presented. The cerebellar group had a mean syllable duration of 395.122 ms., while the normal group had a mean syllable duration of 257.700 ms. The mean difference between the two groups was 137.422 ms. The significance of this difference in syllable duration was $p=.003<.01$.

Tables 3.3a and 3.3b present the statistical analysis of the interaction between prosodic condition and neurological status. The findings for each of the mean values of syllable duration for each of the six prosodic conditions when compared across neurological status were statistically significant (See also, Fig.3.1). That is, for each prosodic condition the normal group differs significantly from the cerebellar group at least at the .05 level of significance (Table 3.4b). Moreover, the unaccented conditions, *-pf-a, –n-a* and the reduced syllable, showed significant differences between groups at the greater than .01 level.

Table 3.4 presents the contrasts between prosodic conditions for each subject group: cerebellar and normal. Although the mean differences between several conditions were found to be significant within the normal group, results are discussed exclusively for the four prosodic contrasts.

Table 3.4 presents the data for the prosodic contrasts within each group. For the cerebellar group the difference between *+pf+a* and *–pf+a* conditions was significant while the remaining three contrasts were not. The normal group showed significant differences for all of the four prosodic contrasts: *+pf+a* vs. *–pf+a*; *-pf+a* vs. *–pf-a* ; *+n+a* vs. *–n-a* and *–n-a* vs. red.

In Figure 3.1 the normal and cerebellar ataxic groups are contrasted for the pattern of durational changes across prosodic conditions. The six cerebellar subjects were compared to their normal matched controls for a qualitative description of durational differences. In Fig.3.2 normal and cerebellar ataxic groups are compared with regard to durational differences across prosodic contrasts. Fig. 3.3 through Fig. 3.8 illustrate the differences for the durations in these prosodic conditions and contrasts for each of the six ataxic speakers his/her normal matched controls.

Although the group graph (Fig.3.1) presents a comparison between groups, examination of each group exclusive of the other group showed the greatest degree of lengthening across conditions for the phrase-final position. However, a comparison between groups shows that the duration of the reduced syllable for the ataxic speakers (400 ms) is longer than even the phrase-final accented condition of the normal speakers (364 ms). Table 3.3a presents the mean values for each of the prosodic conditions for both groups. When we examine the individual paired-subject graphs (Figures 3.3 through 3.8) it appears that the ataxic speakers produce relatively longer syllable durations than their normal controls for all conditions, as well as a more extensive contrast of length between phrase-final and non-final positions than the normal speakers with the exception of one normal speaker of southern dialect (BW: Fig.3.5) who remains outside the range of durations of the other five normal speakers. The contrasts presented in Fig. 3.2 show a consistent pattern of longer to shorter values from accented to unaccented conditions for the normal speakers; whereas except for the *+pf +a* vs. *–pf+a* contrast, this pattern was not evident for the cerebellar group. The cerebellar speakers tend to neutralize or reverse the pattern found in normals for the contrasts at the phrase level: *-pf+a* vs. *–pf-a* and *+n+a* vs. *–n-a* and at the lexical level in contrast *–n-a* vs. *red.* All six cerebellar subjects showed neutralization for phrase accent in the contrasts *–pf+a* vs. *–pf-a* and *+n+a* vs. *–n-a*.

Table 3.1: **Within-Subjects Effects: Duration**

Source	**Type III Sum of Squares**	**df**	**Mean Square**	**F**	**Sig.**
Neurological status	**339926.082**	**1**	**339926.082**	**29.503**	**p<.003****
Prosodic condition	**57609.006**	**5**	11521.801	11.271	p<.001***
Interaction between neurological status & prosodic condition.	122624.913	**5**	**24524.983**	**6.795**	**p<.001*****

Table 3.2: The marginal means for each group and mean difference (pair-wise comparison) of syllable duration between groups for the acoustic measure of syllable duration.

Neurological Status	Mean	Standard Error	95% Confidence Interval		Sig.
	Mean Difference		Lower Bound	Upper Bound	
Cerebellar	395.122	27.773	323.730	466.514	
Normal	257.700	5.953	242.397	273.004	
Cerebellar - Normal	137.422	25.300	72.386	202.458	p=.003**

Based on the estimated marginal means the mean difference between groups is significant at the .01 level.**

Table 3.3a: Marginal means of syllable durations for the interaction between prosodic condition and neurological status.

Prosodic Condition	Neurological Status	Mean	Standard Error	95% Confidence Interval	
				Lower Bound	Upper Bound
+pf+a	Cerebellar	637.531	91.466	402.411	872.652
	Normal	363.700	24.650	300.336	427.064
-pf+a	Cerebellar	347.493	21.437	292.387	402.598
	Normal	270.589	9.257	246.792	294.386
-pf-a	Cerebellar	321.561	23.277	261.724	381.398
	Normal	241.113	7.248	222.480	259.746
+n+a	Cerebellar	334.817	19.647	284.312	385.321
	Normal	263.217	9.308	239.288	287.145
-n-a	Cerebellar	329.341	23.497	268.939	389.742
	Normal	238.917	8.884	216.081	261.753
red	Cerebellar	399.991	55.721	256.755	543.227
	Normal	168.667	9.515	144.208	193.125

Table 3.3b: Pair-wise Comparisons of mean syllable durations for the interaction between prosodic condition and neurological status.

Prosodic Condition	Neurological Status		Mean Difference	Stand. Error	Sig.[a]	95% Confidence Interval	
						Lower Bound	Upper Bound
+pf+a	Cer	Nor	273.831	74.892	.015*	81.316	466.347
-pf+a	Cer	Nor	76.904	22.772	.020*	18.367	135.440
-pf-a	Cer	Nor	80.448	21.468	.013**	25.264	135.632
+n+a	Cer	Nor	71.600	21.128	.019*	17.290	125.910
-n-a	Cer	Nor	90.424	19.016	.005**	41.542	139.307
red	Cer	Nor	231.324	49.601	.006**	103.820	358.828

Based on estimated marginal means (Table 3.3a):

* The mean difference is significant at the .050 level; ** significant at the .01 level.

[a] Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

Table 3.4: Pair-wise comparisons (mean syllable duration differences between prosodic conditions) within each subject group: normal and cerebellar.

Neurological Status	Prosodic Contrast	Mean Difference	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
Normal	+pf+a vs. –pf+a	93.111*	31.129	.030*	13.091	173.132
	-pf+a vs. –pf-a	29.587*	7.964	.014*	9.004	49.948
	+n+a vs. –n-a	24.300**	5.566	.007**	9.992	38.608
	-n-a vs. red	70.250**	14.002	.004**	34.257	106.243
Cerebellar	+pf+a vs. –pf+a	290.039*	83.212	.018*	76.135	503.943
	-pf+a vs. –pf-a	25.931	11.465	.073	-3.540	55.403
	+n+a vs. –n-a	5.476	10.584	.627	-21.730	32.682
	-n-a vs. red	-70.650	67.583	.344	-244.326	103.026

* The mean difference is significant at the .05 level; **significant at .01 level.

[a] Adjustment for multiple comparisons: Least significant difference (equivalent to no adjustments).

Fig. 3.1: **Mean syllable durations in ms. across the six prosodic conditions for cerebellar ataxic speakers and normals. The differences between group means is represented when significant by *(p<.05) and **(p<.01).**

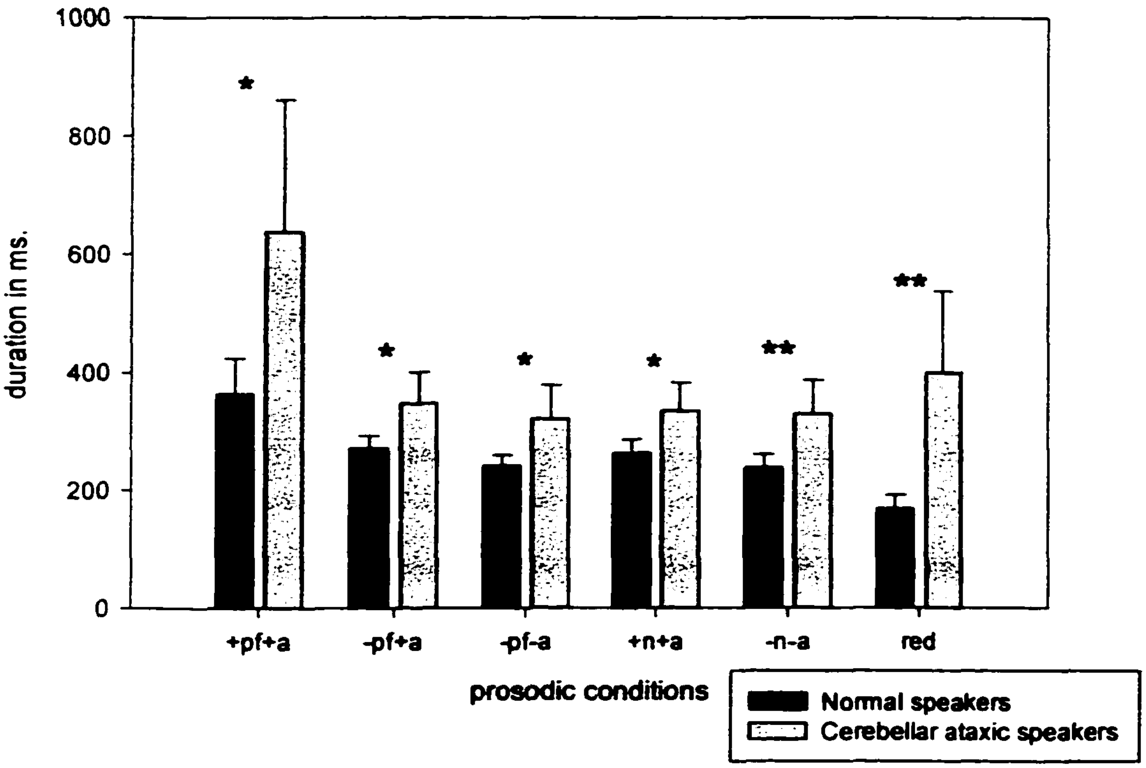


Fig. 3.2: Mean syllable durations in ms for each of the four prosodic contrasts for cerebellar ataxic speakers and normals. The difference in means within **each** group is represented as significant by *($p<.05$ and **($p<.01$). (Table 3.4) The mean difference **between** groups for each of four contrasts is also marked for significance on the x axis:+pf+a -pf+a (Tables 3.5 a,b) and -n-a red (Tables 3.8 a,b).

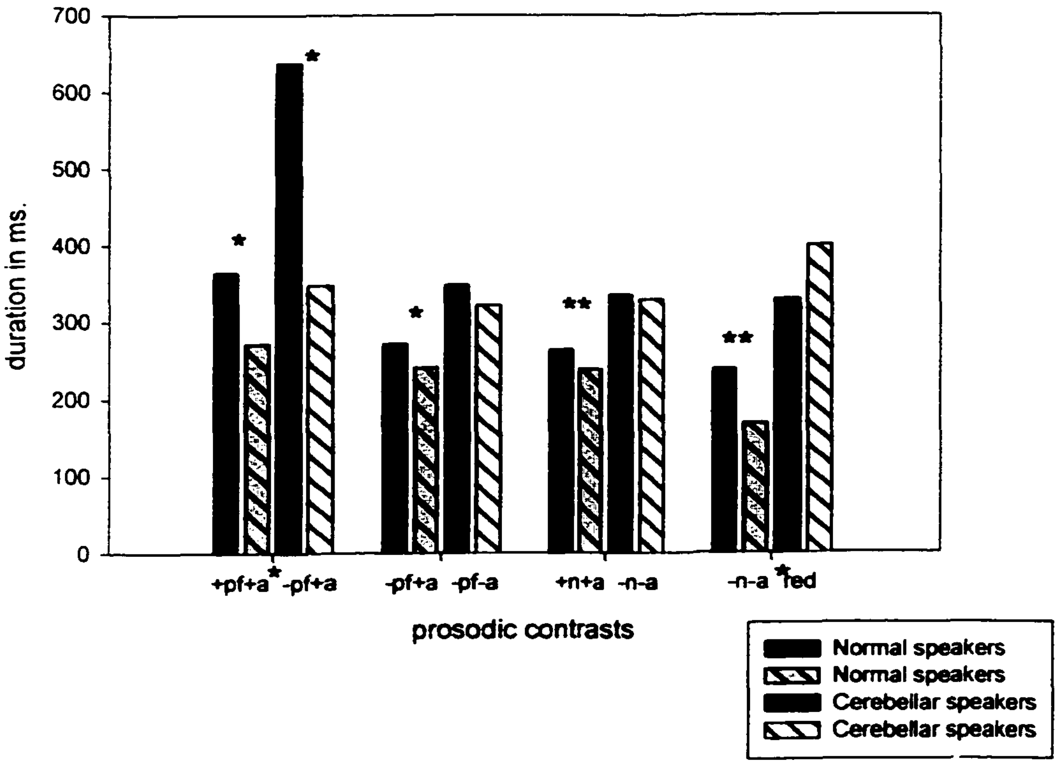


Fig. 3.3 :Mean syllable duration in ms for six prosodic conditions for cerebellar ataxic speaker AH (D = 454.69; MRI = 2.0) and normal matched control LJR.

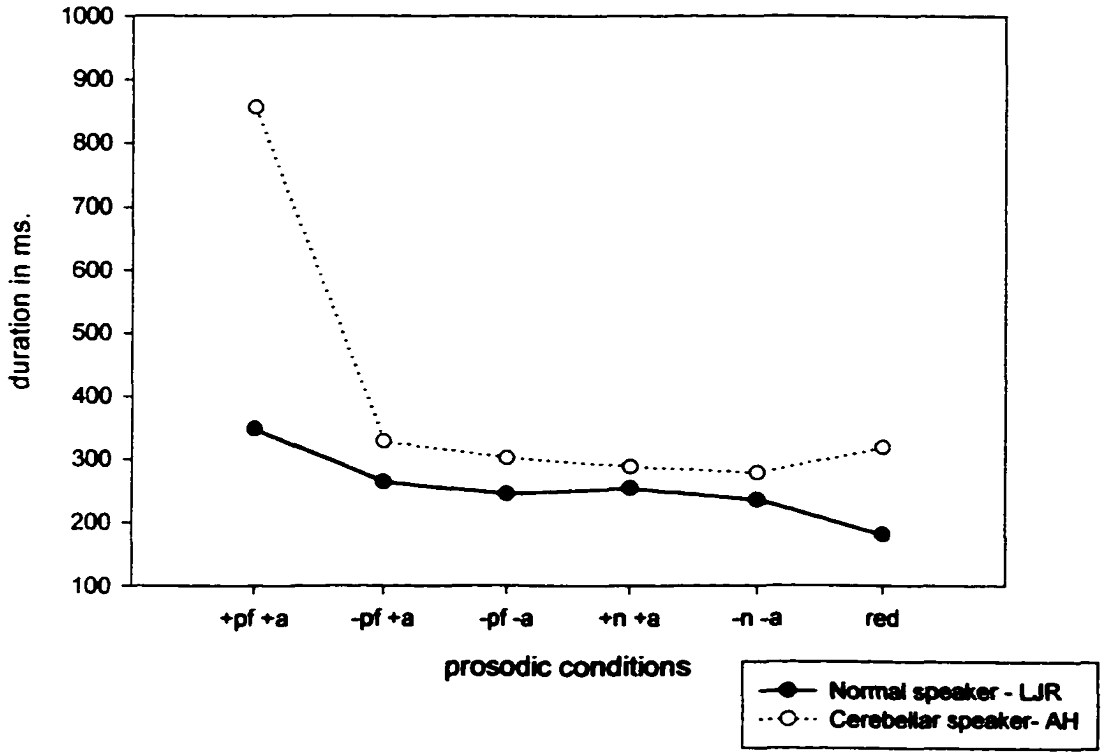


Fig. 3.4: Mean syllable duration in ms for six prosodic conditions for cerebellar ataxic speaker PP (D= 267.90; MRI: 3.0) and normal matched control GC.

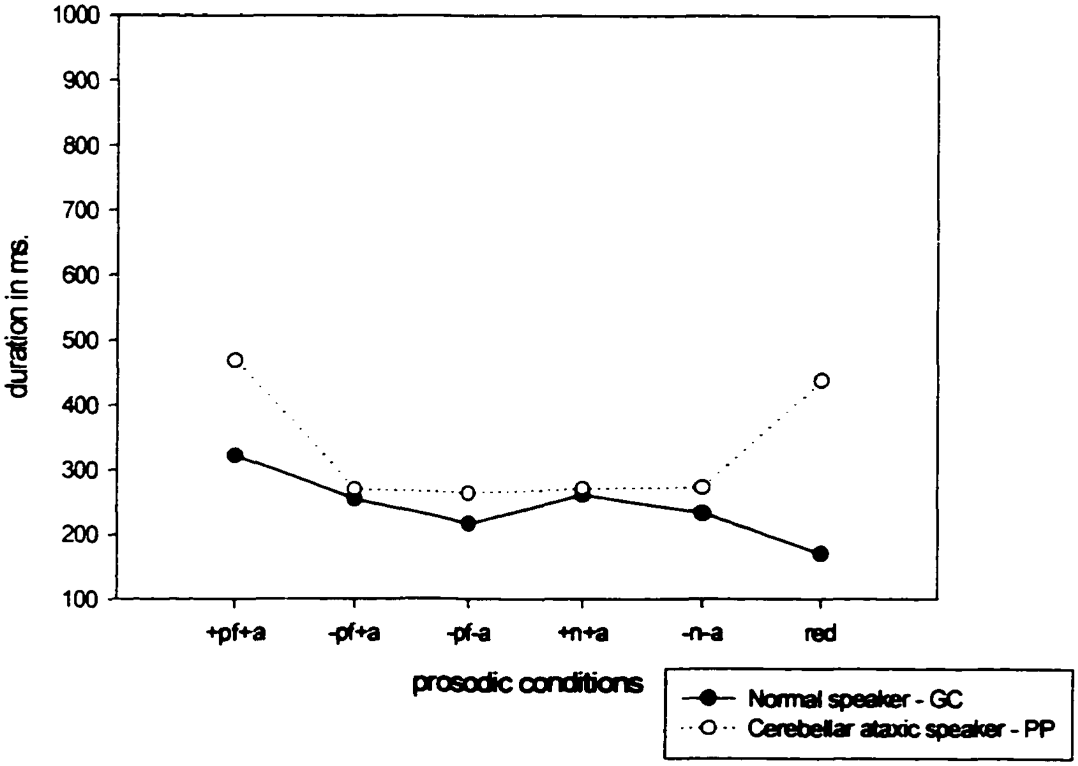


Fig. 3.5: Mean syllable duration in ms for six prosodic conditions for cerebellar ataxic speaker WD (D =487.81; MRI = 2.5) and normal matched control BW.

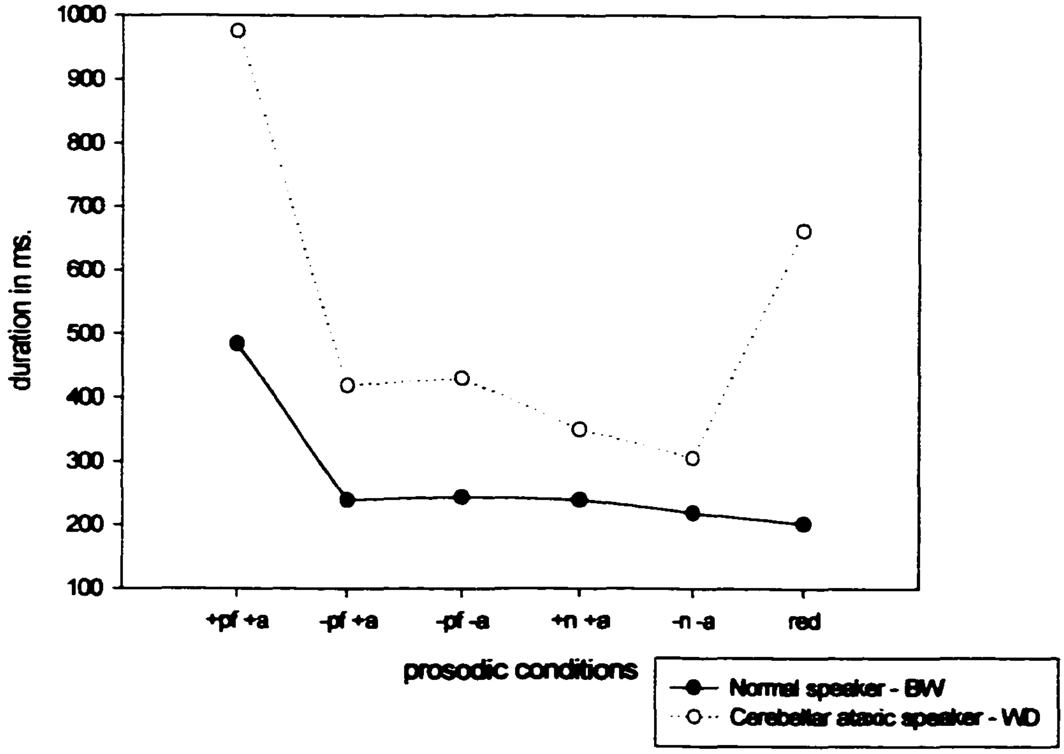


Fig. 3.6: Mean syllable duration in ms for six prosodic conditions for cerebellar ataxic speaker JS (D= 205.5 ; MRI= 2.0) and normal matched control YM.

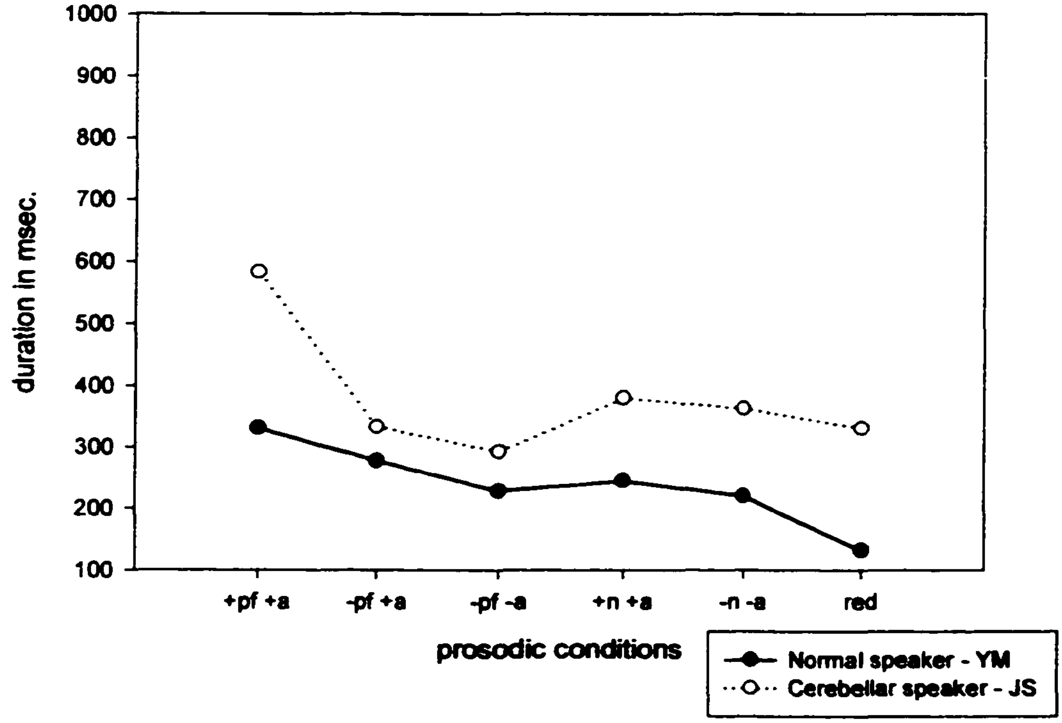


Fig. 3.7: **Mean mid-point syllable duration in ms. for six prosodic conditions for cerebellar ataxic ES (D= 85.62; MRI = 2.0) and normal matched control LL**

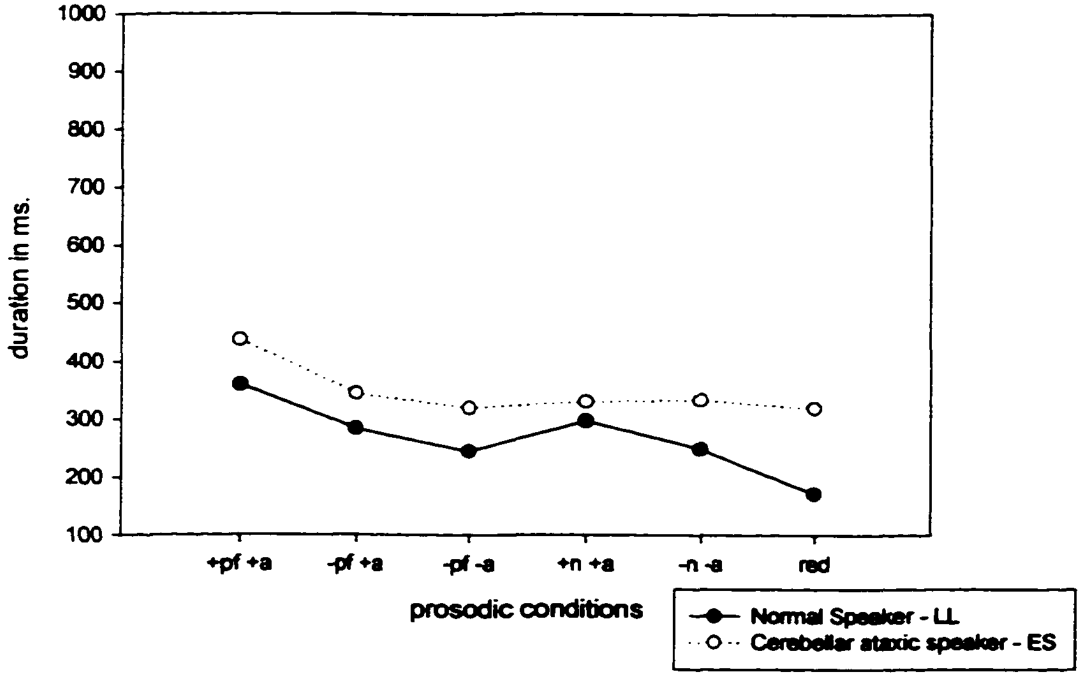
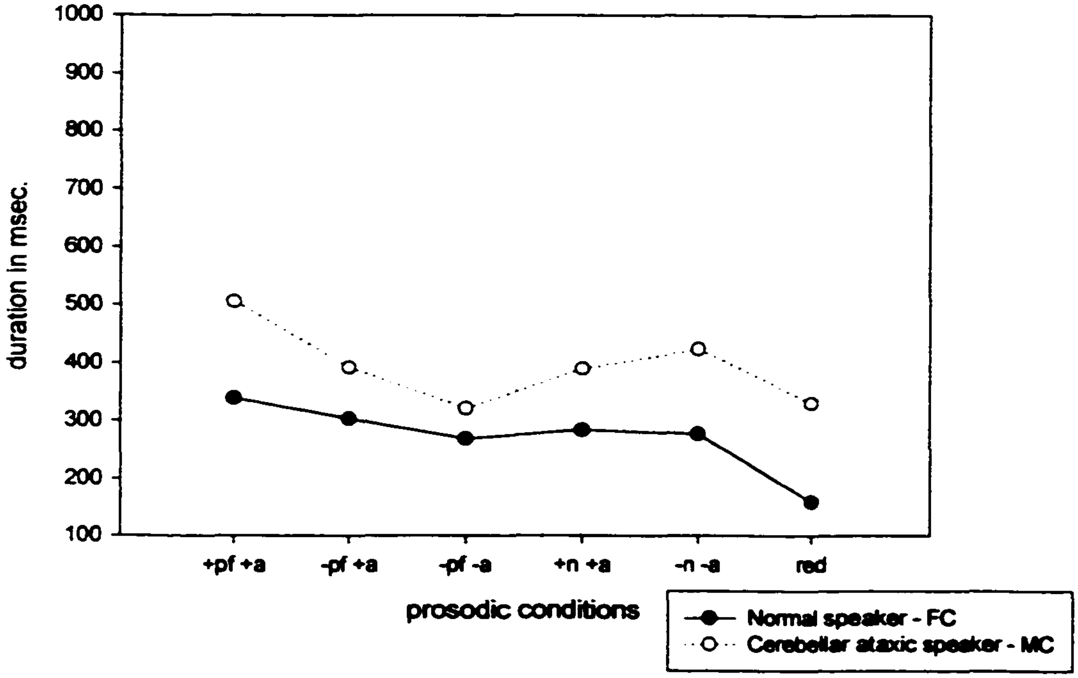


Fig. 3.8: Mean syllable duration in ms for six prosodic conditions for cerebellar ataxic speaker MC (D= 98.38; MRI = 1.5) and normal matched control FC.



3.1.1 The effects of cerebellar disturbance on the lengthening of accented syllables (+a) in phrase-final (+pf) and non-phrase-final (-pf) positions : Contrast 1: +pf+a vs. –pf+a

This section compares the duration of phrase-final accented (+pf+a) and non-phrase-final accented (*-pf+a*) syllables. The purpose of this comparison is to consider the differences in duration between these two conditions as a function of neurological status/speech production. The statistical analysis of variance and t test revealed a significant difference p=.028<.05 between the two groups (Table 3.5a,b). The group differences are illustrated in Fig. 3.2. Both groups show a contrast between conditions with the *+pf+a* relatively longer than the *–pf+a* condition. As well as being longer in duration for these two conditions, the extent of the contrast is significantly greater in the cerebellar group than the normal group. In addition, the analysis of prosodic contrasts (Table 3.4) presented in the pair-wise comparisons for each of the groups showed that both normals and cerebellar ataxics differ significantly between these conditions. Normals: p=.030<.05 and Cerebellar speakers: p= .018 < .05. This is the only contrast that cerebellar speakers produce with a marked contrast greater than that of the normal speakers.

able 3.5a: Tests of Significance for Contrast 1: +pf+a vs. –pf+a: Unique Sum of Squares:

Analysis of Variance :	Contrast 1= +pf+a vs. –pf+a : cerebellar vs. normal				
Source of Variation	SS	DF	MS	F	Sig. of F
Within Cells	124555.31	5	24911.06		
Constant	232683.27	1	232683.27	9.34	p=.028*

Table 3.5 b:Estimates for Contrast 1: +pf+a vs. –pf+a: Cerebellar vs. Normal:

Parameter	Coefficient	Std. Error	t-value	Sig. T	95% Level of Confidence	
					Lower	Upper
1	196.927767	64.43480	3.05623	.028*	31.29536	362.56018

3.1.2 The effects of cerebellar disturbance on accented (+a) and un-accented (-a) syllables in non-final (-pf) position: Contrast 2: -pf+a vs. –pf-a

This section compares the durations of accented versus un-accented syllables in non-final positions. Unlike the first contrast (*+pf+a* vs. *–pf+a*), the position in the sentence for *–pf+a* and *–pf-a* is the same while the phrase accent differs (*+a* and *–a*). The purpose of this comparison is to consider the difference in accented and un-accented syllables as a function of neurological status. The analysis of variance and t test did not reveal a significant difference between the two groups (Table 3.6 a & b).

Although the between-group differences were not significant for this contrast an examination of the pair-wise comparisons within each group reveals a different pattern for each group. The normal group showed a significant difference ($p=.014<.05$) between the prosodic conditions of *-pf+a* and *–pf-a*, whereas the cerebellar group did not (Table 3.4). Thus, it appears the production of this contrast may be a characteristic of normal speech motor dynamics, but not of cerebellar motor dynamics.

Table 3.6a: Tests of **Significance for Contrast 2: –pf+a vs. -pf-a : Unique Sum of Squares**

Analysis of Variance : Contrast 2 = -pf+a vs. -pf-a : cerebellar vs. normal					
Source of Variation	SS	DF	MS	F	Sig. of F
Within Cells	2726.65	5	545.33		
Constant	75.38	1	75.38	.14	p=.725

Table 3.6 b: Estimates **for Contrast 2: -pf+a** vs. **–pf-a : Cerebellar** vs.**Normal**

Parameter	**Coefficient**	**Std. Error**	**t-value**	Sig. T	**95% Level of Confidence**	
					Lower	Upper
1	-3.5444463	9.53354	-.37179	.725	-28.05082	20.96193

3.1.3 The effects of cerebellar disturbance on syllable duration for accented (+a) and un-accented (-a) syllables in nuclear (+n) and post-nuclear (-n) positions: Contrast 3: +n+a vs. –n-a

The section compares the third contrast of accented versus un-accented syllables, both in nuclear (+*n*+*a)* and post-nuclear (-*n*-*a*) positions. The nuclear accented (+*n*+*a*) syllable receives the speaker's primary focus and therefore the greatest prominence of the sentence. The purpose of this comparison is to consider the difference between nuclear accented and post-nuclear unaccented syllables as a function of neural status. The analysis of variance and t test did not reveal a significant difference between the two groups (Table 3.7a and Table 3.7b). However the pair-wise comparisons (Table 3.4) within each group showed again that normal speakers durations differed significantly ($p=.007<.01$) between these two conditions whereas those of the cerebellar speakers did not.

Table 3.7a: Tests of **Significance for Contrast 3: +n+a vs. –n-a: Unique Sum of Squares**

Analysis of Variance : Contrast 3= +n+a vs. –n-a : cerebellar vs. normal					
Source of Variation	SS	DF	MS	F	Sig. of F
Within Cells	3703.78	5	740.76		
Constant	2126.08	1	2126.08	2.87	p= .151

Table 3.7b: **Estimates for Contrast 3: +n+a vs. –n-a: Cerebellar vs. Normal**

Parameter	Coefficient	Std. Error	t-value	Sig. T	95% Level of Confidence	
					Lower	Upper
1	-18.824076	11.11122	-1.69415	.151	-47.38595	9.73779

3.1.4 The effects of cerebellar disturbance on syllable duration for un-accented (-a) post-nuclear (-n) and reduced (red) syllables: Contrast 4: -n-a vs. red

This section examines the contrast between unaccented post-nuclear (-*n*-a) and reduced (*red*) syllables. The purpose of this comparison is to examine differences at the word level, as opposed to the phrase level, between un-accented and reduced syllables as a function of neural status. The group statistical analysis of variance and t test approached significance for the difference between the two groups (p=.051) for this contrast (Tables 3.8 a and b).

Once again the pair-wise comparisons within each group showed that the durations differed significantly (p= .004<.01) whereas those of the cerebellar speakers did not (Table 3.4).

Table 3.8a: Tests of Significance for Contrast 4: -n-a vs. red : Unique Sum of Squares

Analysis of Variance : Contrast 4= -n-a vs. red : cerebellar vs. normal					
Source of Variation	SS	DF	MS	F	Sig. of F
Within Cells	90735.48	5	18147.10		
Constant	119116.85	1	119116.85	6.56	p= .051

Table 3.8b:Estimates for Contrast 4: -n-a vs. red: Cerebellar vs. Normal

Parameter	Coefficient	Std. Error	t-value	Sig. T	95% Level of Confidence	
					Lower	Upper
1	-140.89999	54.99560	-2.56202	.051	-282.26853	.46855

3.1.5 Duration: Discussion and conclusions

Each of the six prosodic conditions investigated here showed a significant difference between the normal and the cerebellar groups for the measure of syllable duration. The four contrasts, when examined within each group, showed one significantly different contrast (*+pf+a* vs. *–pf+a*) for the cerebellar group whereas the normal group showed significant differences for all of the four contrasts investigated. Between-group differences in the production of contrasts, were significant (*+pf+a vs.-pf+a*) and nearly significant for (*–n-a vs. red)*. These two contrasts present evidence for different patterns in accented lengthening and syllable reduction across groups.

The graphic displays for individual subjects and their matched controls (Figures 3.3 through 3.8) reveal what is perhaps the most salient property of speech of those with neurological speech motor disorders: durations are greater than those of normals. Furthermore, the findings reported here statistically support the claim that ataxic speakers are not able to reduce syllables in the way that normal speakers do. All of the normal speakers investigated here show a significant reduction in the duration of the reduced syllable ($p= .004<.01$: Table 3.4); the ataxic speakers did not show this reduction. Two of the ataxic speakers (JS: Fig. 3.6 and ES: Fig. 3.7) equalized the durations of the reduced and post-nuclear unaccented syllables; three of the subjects (AH: Fig. 3.3, PP: Fig. 3.4 and WD: Fig. 3.5) produced "reduced" syllables of greater duration than even some of the accented syllables investigated.

3.2 The effects of prosodic condition on formant frequencies

The F2 and F1 mid-point frequencies were examined for the syllable [pɑp] produced by the twelve subjects across six different prosodic conditions.

Table 3.9 and Table 3.13 present the results for the significance of three main effects for measures of F2 and F1 frequency: the effect of neurological condition, the effect of prosodic condition and the interaction of these conditions. The main effect of the interaction of the F2 measure was significant (p=.05), whereas the other two main effects were not (Table 3.9). For the measure of F1, two main effects were found to be significant: prosodic condition and the interaction of neurological status and prosodic condition (Table: 3.13).

3.2.1 The effects of prosodic condition on F2 frequency

Table 3.10 examines the main effect of neurological status on F2 mid-point frequency values averaged over the six prosodic conditions. The mean across conditions does not differ significantly between groups. The average value for the cerebellar subjects falls at 1202 Hz. and the normal group at 1219 Hz. However, The values among conditions, however, vary significantly in both groups and so the difference between means is not a true indication of the difference between the patterns revealed by closer analysis of the data.

Tables 3.11a and 3.11b reflect the interaction between neurological status and prosodic conditions. An examination of the means for each of the prosodic conditions revealed differences between groups. Normal speakers showed a distinct pattern of differences across conditions (Table 3.11a and Fig. 3.9). The lowest mean F2 frequency for the normal group occurred in the reduced condition (1074 Hz). In contrast, the highest mean F2 frequency was in the non-phrase-final accented condition *–pf+a* (1296 Hz). This does not conform to the prediction that the highest value would be found in the nuclear accented condition (Chapter 2). Normal speakers consistently produced higher mean F2 frequencies for the two conditions of *-pf+a* (1296 Hz). and *+n+a* (1289 Hz), while the *–a* conditions had relatively lower mean F2 frequencies (*–pf-a*: 1205 Hz. and *–n-a*: 1217 Hz). The mean F2 frequency (1235 Hz) in the *+pf+a* condition was lower than in the other *+a* conditions and close to the *–a* conditions.

Cerebellar ataxic speakers did not produce the range of F2 frequency differences seen in normals: (Table 3.11a and Fig. 3.9). F2 values for cerebellar speakers ranged from 1161 Hz. to 1225 Hz. (a 64 Hz. difference) whereas normal speakers ranged from 1074 Hz. to 1296 Hz. (a 222 Hz. difference). The two highest values for cerebellar speakers across the prosodic conditions were the *red* condition at 1222 Hz. and the *+pf+a* condition: 1225 Hz., while the *+n+a* was among the lower values. Therefore, the cerebellar speakers did not distinguish accented and unaccented conditions as did the normals. In addition, the highest mean F2 frequency for the cerebellar speakers was found in the *+pf+a* condition, while the value for this condition was among the lower F2 frequencies for the normal speakers. When we consider the extended syllable durations for *+pf+a* in both groups, the relative difference in F2 frequency between groups may reflect different underlying mechanisms of control. It may be that in the *+pf+a* condition, the cerebellar speakers increase articulatory effort while normals respond with an overall slowing down.

The only condition to show a significant difference between groups for the mean F2 frequency was the reduced condition: $p=.022<.05$ (Table 3.11b).

The four contrasts between prosodic conditions were analyzed within each subject group (Table 3.12 and Fig. 3.10). The cerebellar group showed no significant differences between the mean F2 frequencies for any of the four prosodic contrasts. In contrast, the normal group showed significant differences for three of the four prosodic contrasts: *+pf+a* vs. *-pf+a* ($p= .039<.05$); *+n+a* vs. *–n-a* ($p=.018<.05$) and *–n-a* vs. *red* ($p=.006<.01$). Although the *+pf+a* vs. *–pf+a* difference was not statistically significant, an examination of differences between groups suggests different dynamics of lengthening within that contrast (Fig.3.10). The normal speakers, as a group, showed a relatively higher average F2 frequency for the accented syllable in non-final position as compared to the accented syllable in phrase-final position. Cerebellar speakers as a group did not show this pattern.

Figures 3.11 through 3.16 illustrate the differences for mean F2 frequencies of the six prosodic conditions for each of the six ataxic speakers and their normal matched controls. While the normal speakers consistently lowered the F2 frequency for the *–a* conditions relative to the *+a* conditions in the comparisons of *-pf+a* vs. *–pf-a*, *+n+a* vs. *–n-a* and *–n-a* vs. *red*, the cerebellar speakers did not. Five of the six cerebellar speakers (AH, Fig. 3.11; PP, Fig. 3.12; JS, Fig. 3.14; ES, Fig. 3.15; and MC, Fig. 3.16) all had higher F2 frequencies in the *–a* condition in the contrast *+n+a* vs. *–n-a*. For the *–n-a* vs. *red* contrast two of the cerebellar speakers (WD, Fig. 3.13; and JS, Fig. 3.14) increased the F2 frequency in the reduced condition and one (MC, Fig. 3.16) had fairly equal F2 frequencies across conditions. Three of the six cerebellar speakers (AH, Fig. 3.11; ES, Fig. 3.15; and MC, Fig. 3.16) showed increased F2 frequencies for the *–a* condition and one speaker (PP, Fig. 3.12) showed equalization of F2 frequency in the *–pf+a* vs. *–pf-a* contrast.

Table 3.9: **Within-Subjects Effects: F2 frequency**

Source	**Type III Sum of Squares**	**df**	**Mean Square**	**F**	**Sig.**
Neurological status	**5666.491**	**1**	**5666.491**	**.263**	**p=.630**
Prosodic condition	**75516.058**	**5**	**15123.212**	**1.535**	**p=.215**
Interaction between neurological status & prosodic condition	**132727.328**	**1.298**	**102233.721**	**5.456**	**p=.050***

Table 3.10: **The marginal means for each group and mean difference (pair-wise comparison) of mean F2 frequency at vowel mid-point between groups.**

Neurological Status	Mean	Standard Error	95% Confidence Interval		Sig.
	Mean Difference		Lower Bound	Upper Bound	
Cerebellar	1201.570	33.015	1116.703	1286.437	
Normal	1219.313	64.676	1053.058	1385.568	
Cerebellar - Normal	-17.743	34.615	-106.724	71.238	p=.630

Based on the estimated marginal means the mean difference between groups is not significant at the .05 level.

Table 3.11a: **Means of mid-point F2 frequency for the interaction between prosodic condition and neurological status.**

Prosodic Condition	Neurological Status	**Mean**	**Standard Error**	**95% Confidence Interval**	
				Lower Bound	**Upper Bound**
+pf+a	Cerebellar	1225.133	43.569	1113.136	1337.130
	Normal	1235.217	69.950	1055.405	1415.028
-pf+a	Cerebellar	1207.104	29.732	1130.676	1283.532
	Normal	1295.576	75.238	1102.171	1488.981
-pf-a	Cerebellar	1193.250	52.662	1057.879	1328.621
	Normal	1204.919	74.718	1012.852	1396.987
+n+a	Cerebellar	1161.817	31.962	1079.655	1243.978
	Normal	1288.767	78.416	1087.192	1490.341
-n-a	Cerebellar	1200.404	37.096	1105.046	1295.761
	Normal	1216.917	61.378	1059.140	1374.693
red	Cerebellar	1221.715	78.898	1018.901	1424.528
	Normal	1074.483	60.587	918.740	1230.227

Table 3.11b: Pair-wise comparisons of mid-point F2 frequency for the interaction of prosodic condition and neurological status.

Prosodic Condition	Neurological Status		Mean Difference	Standard Error	Sig.[a]	95% Confidence Interval	
						Lower Bound	Upper Bound
+pf+a	Cer	Nor	-10.083	52.478	.855	-144.981	124.815
-pf+a	Cer	Nor	-88.472	59.944	.168	-229.711	52.766
-pf-a	Cer	Nor	-11.669	44.451	.803	-125.934	102.595
+n+a	Cer	Nor	-126.950	68.800	.124	-303.807	49.907
-n-a	Cer	Nor	-16.513	28.098	.582	-88.742	55.716
red	Cer	Nor	147.231*	45.023	.022*	31.497	262.966

Based on estimated marginal means

* The mean difference is significant at the .050 level.

[a] Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

Table 3.12: **Pair-wise comparisons (mean mid-point F2 frequency differences between prosodic conditions) for each group: normal and cerebellar.**

Neurological Status	**Prosodic Contrast**	**Mean Difference**	**Std. Error**	**Sig.[a]**	**95% Confidence Interval for Difference[a]**	
					Lower Bound	**Upper Bound**
Normal	+pf+a vs. –pf+a	-60.359	48.828	.271	-185.875	65.156
	-pf+a vs. –pf-a	90.656*	32.639	.039*	6.756	174.557
	+n+a vs. –n-a	71.850*	20.837	.018*	18.288	125.412
	-n-a vs. red	142.433*	31.385	.006**	61.755	223.111
Cerebellar	+pf+a vs. –pf+a	18.030	28.393	.553	-54.956	91.016
	-pf+a vs. –pf-a	13.854	41.288	.751	-92.280	119.987
	+n+a vs. –n-a	-38.587	25.173	.186	-103.297	26.123
	-n-a vs. red	-21.311	69.881	.773	-200.946	158.324

* The mean difference is significant at the .050 level; ** = significance at .01 level.

[a] Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments.)

Fig. 3.9: Mean F2 frequency in Hz across the six prosodic conditions for cerebellar ataxic speakers and normals. The differences between group means is represented as significant with *(p<.05).

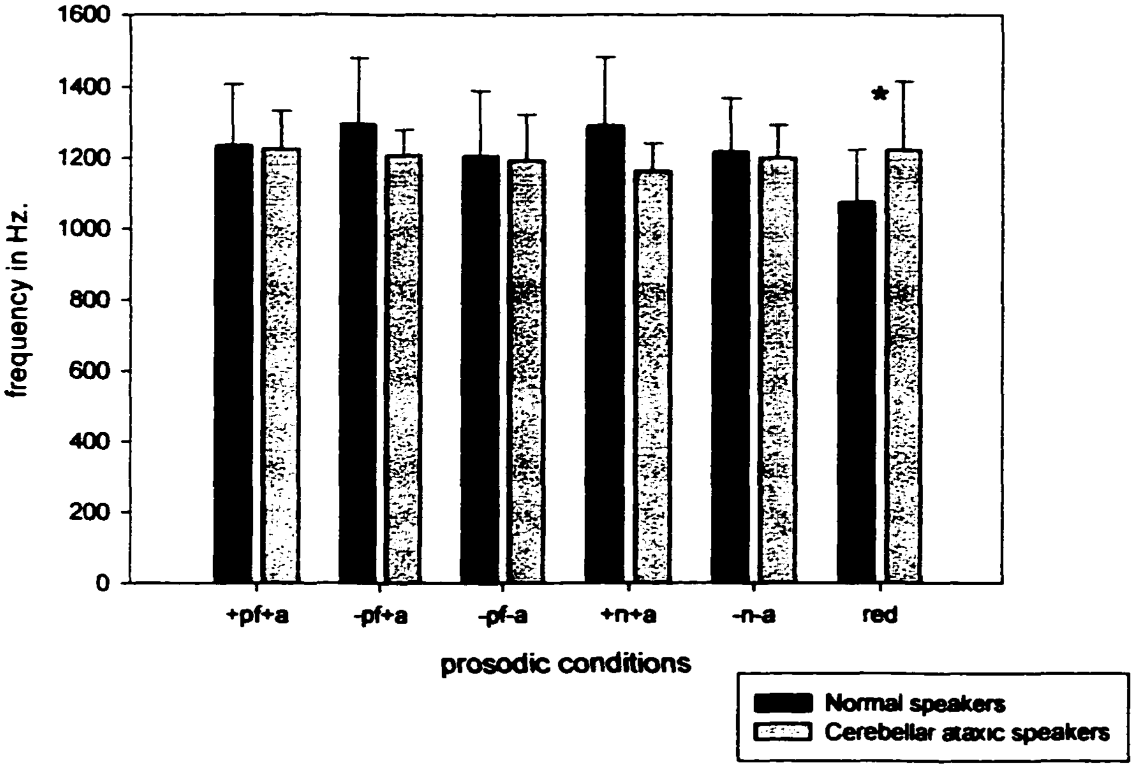


Fig. 3.10: Mean F2 frequency in Hz for each of the four prosodic contrasts for cerebellar ataxic speakers and normals. The mean contrasts for each group is represented as significant by *(p<.05) and **(p<.01)(Table 3.12). The mean difference between groups for each of the four contrasts is marked for significance on the x axis: -pf+a -pf-a (Tables 3.19 a,b) and -n-a red (Tables 3.23 a,b).

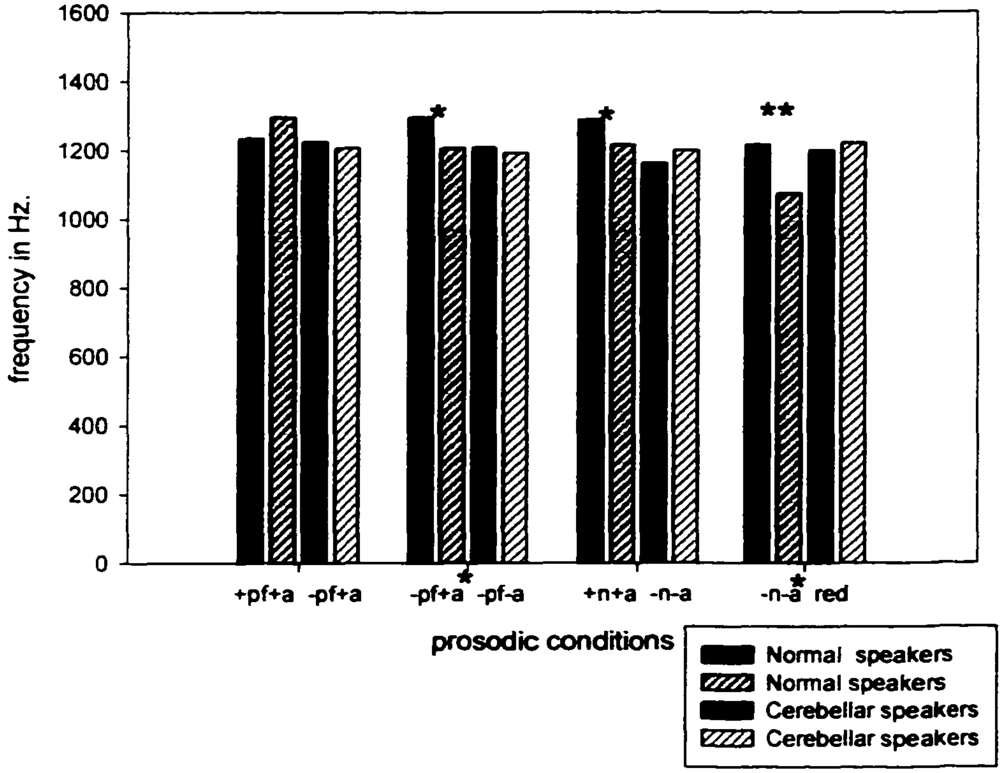


Fig. 3.11: Mean F2 frequencies at mid-point in Hz across six prosodic conditions for cerebellar ataxic speaker AH (D = 132.72; MRI = 2.0) and normal matched control LJR.

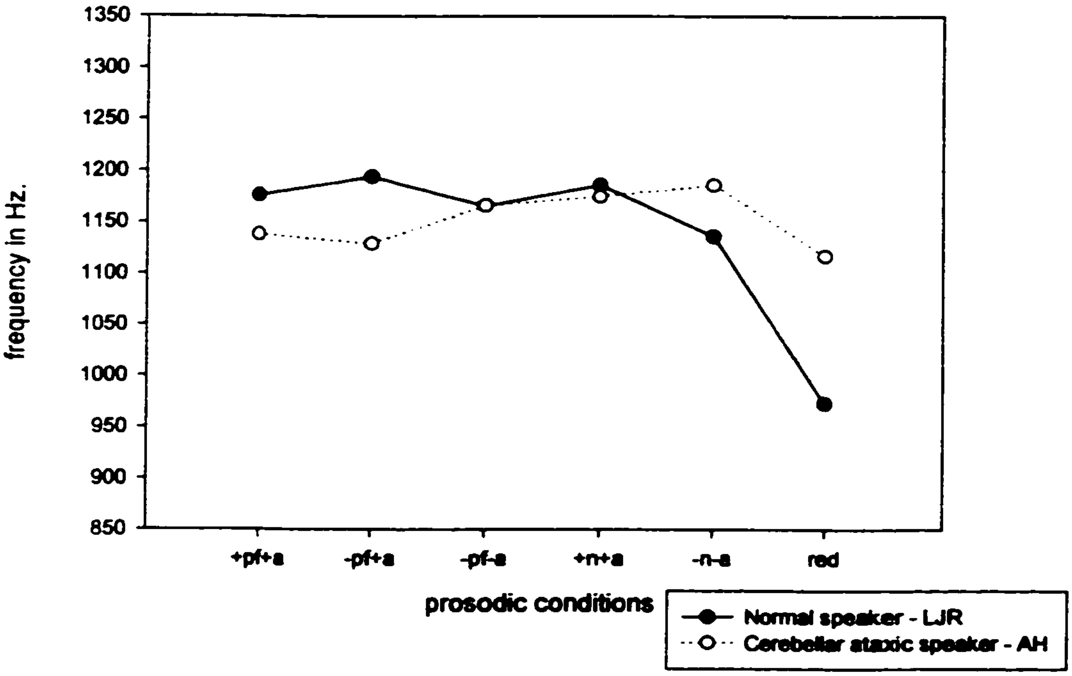


Fig. 3.12: Mean F2 frequencies at mid-point in Hz. across the six prosodic conditions for cerebellar ataxic speaker PP (D= 177.59; MRI = 3.0)and normal matched control GC.

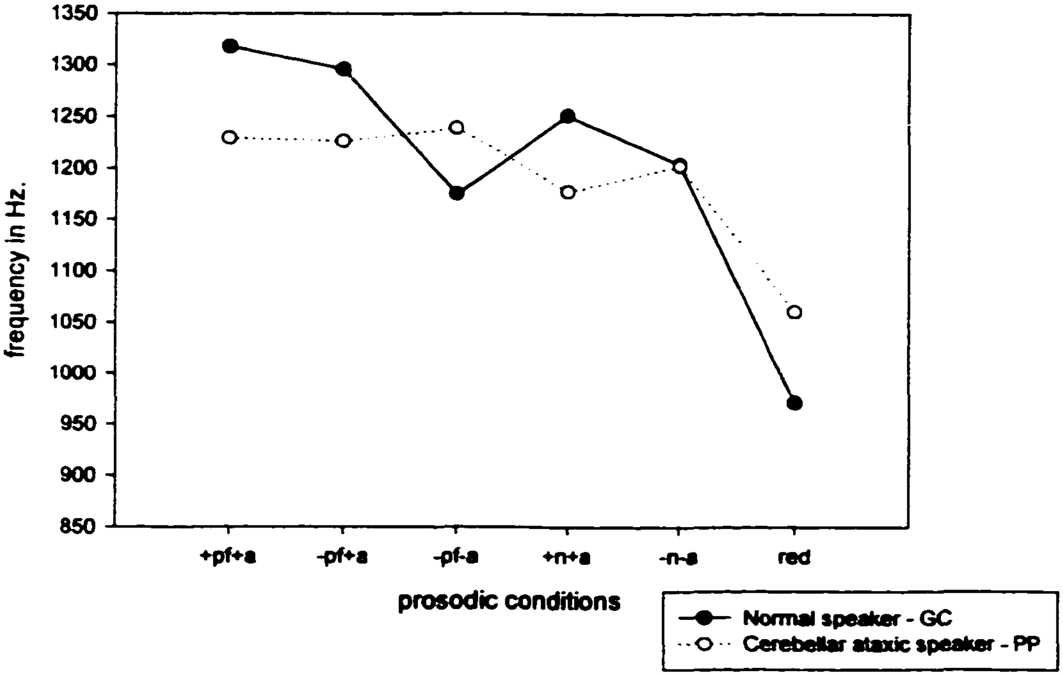


Fig. 3.13: Mean F2 frequenciesat mid-point in Hz. across the six prosodic conditions for cerebellar ataxic speaker WD (D= 162.60; MRI = 2.5) and normal matched control BW.

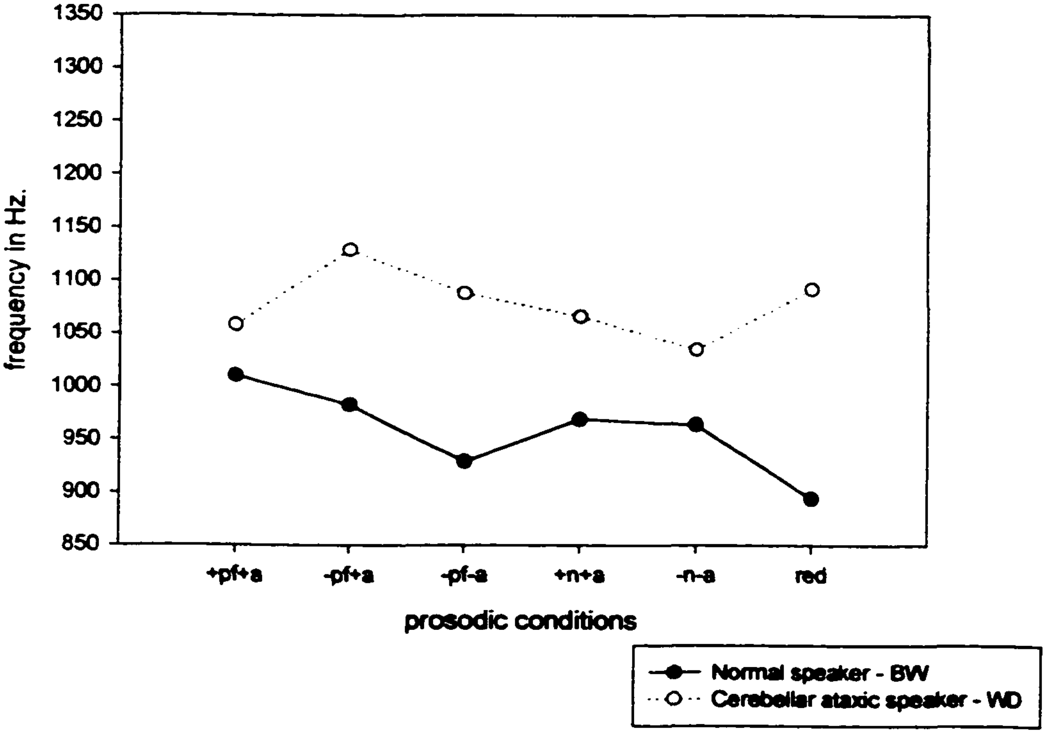


Fig. 3.14: **Mean F2 frequencies at mid-point in Hz across the six prosodic conditions for cerebellar ataxic speaker JS (D= 621.52; MRI = 2.0) and normal matched control YM.**

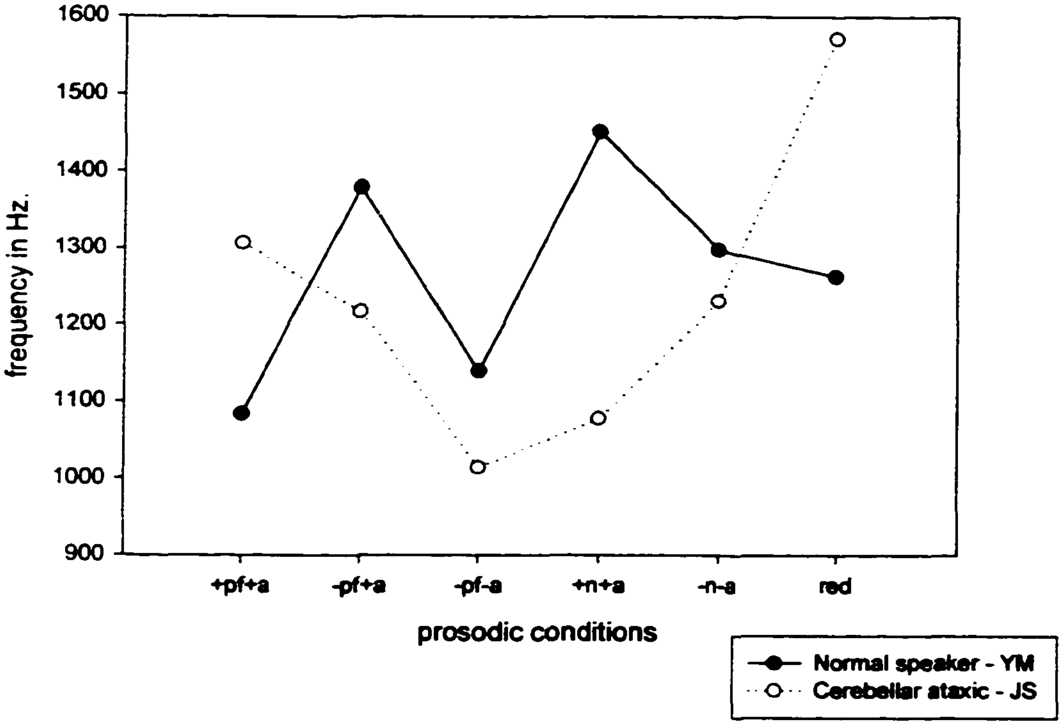


Fig. 3.15: Mean F2 frequencies at mid-point in Hz across the six prosodic conditions for cerebellar ataxic speaker ES (D = 274.48; MRI =2.0) and normal matched control LL.

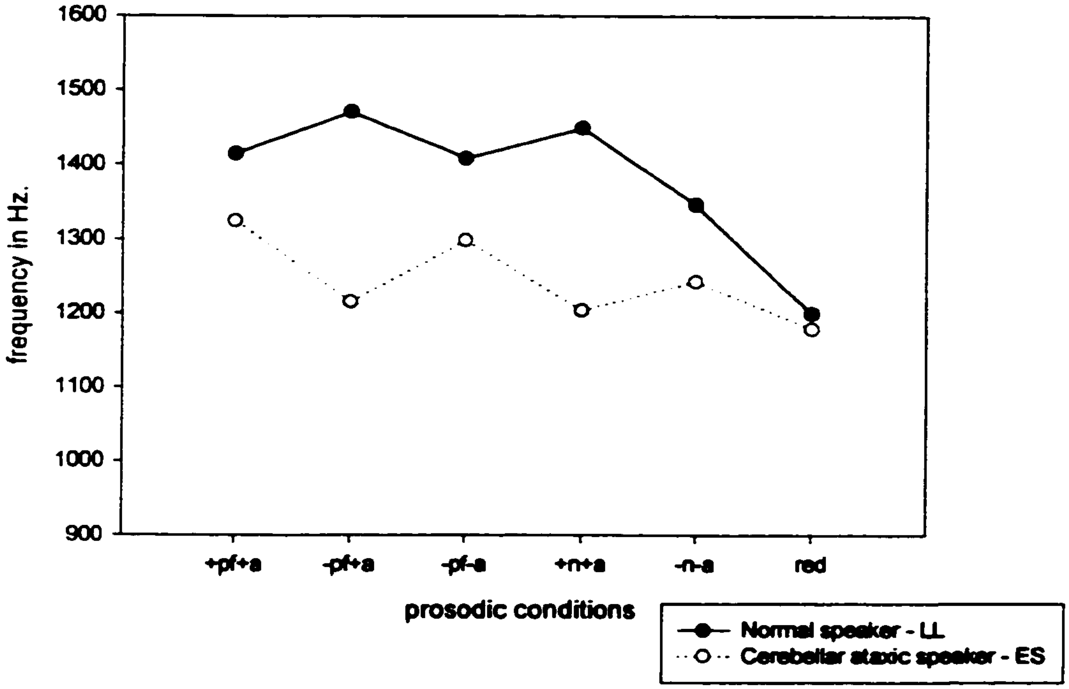
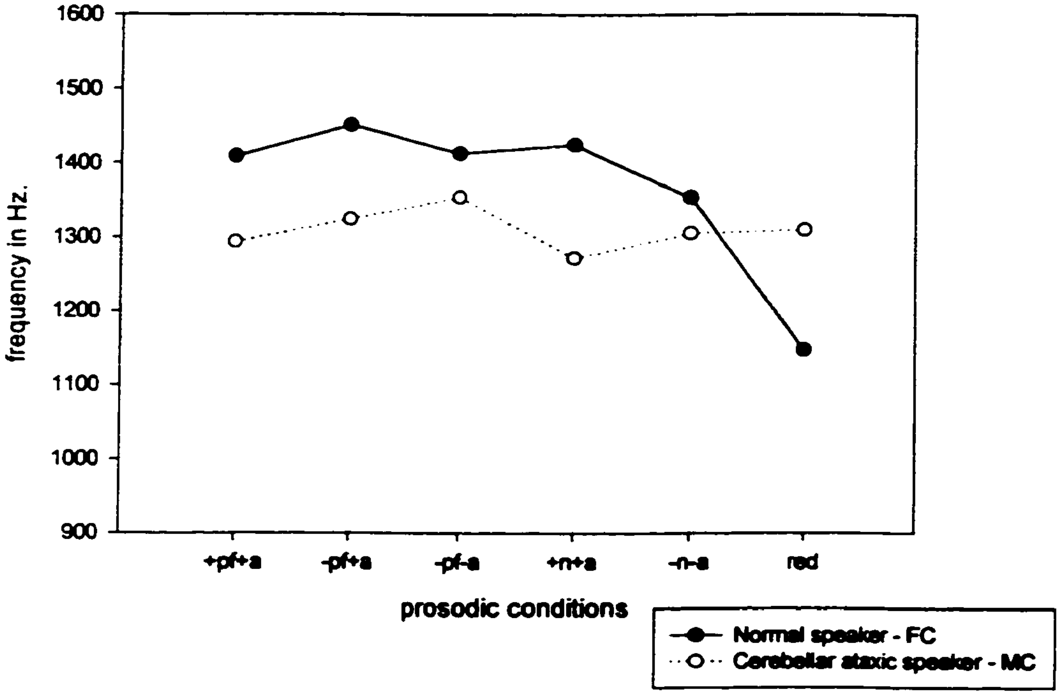


Fig. 3.16: Mean F2 frequencies at mid-point in Hz across the six prosodic conditions for cerebellar ataxic speaker MC (D = 245.78; MRI = 1.5) and normal matched control FC.



3.2.2 The effects of prosodic condition on F1 frequency

Table 3.13 describes the three main effects of neurological status, prosodic condition, and the interaction between neurological status and prosodic condition for the acoustic measure of F1 frequency. The main effects of prosodic condition ($p=.000<.001$) and of the interaction between prosodic conditions and neurological status ($p=.000<.001$) were significant.

Table 3.14 examines the main effect of neurological status on F1 mid-point frequency across the six prosodic conditions. The means across conditions for the vowel [ɑ] in the syllable [pɑp] did not differ significantly between groups (Cerebellar: 774 Hz and Normal: 784 Hz).

Tables 3.15a and 3.15b and Fig. 3.17 show the interaction between neurological status and prosodic conditions. As with F2 frequency, an examination of F1 means each of the prosodic conditions revealed differences between groups. For normal speakers the lowest mean F1 frequency was in the *red* (518 Hz.). In contrast, the highest mean value was in the *–pf+a* condition (866 Hz.). Normal speakers consistently produced higher frequency values for the two accented conditions of *-pf+a* (866 Hz.) and *+n+a* (864 Hz.), while the unaccented conditions had relatively lower mean F1 frequencies (*–pf-a* 808 Hz. and *–n-a*, 795 Hz.). One accented condition, *+pf+a* (853Hz.) was lower than the other two phrase and accent positions (*–pf+a* and *+n+a*).

The range of mean F1 frequencies for cerebellar ataxic speakers was 192 Hz (848 Hz – 656 Hz), which was smaller than that for the normal speakers which was 358 Hz (866 - 578 Hz). The two highest mean F1 frequencies for cerebellar speakers were *–n-a* (797 Hz.) and *+pf+a* (848 Hz.). As with F2 frequency, the cerebellar speakers did not show a pattern of F1 frequencies that distinguish accented (*+a*) from unaccented (*–a*) as did the normal subjects.

Only the *red* condition showed a significant difference between groups for F1 frequency ($p= .001 \leq .001$) (Table 3.15b).

The four contrasts between prosodic conditions were analyzed within each subject group (Table 3.16). The cerebellar group showed a significant difference for mean F1 frequencies only in the *-n-a* vs. *red* contrast ($p= .01 \leq .01$). The normal group however, showed significant differences for three of the four prosodic contrasts: *-pf+a* vs. *–pf+a* ($p= .025 < .05$) ; *+n+a* vs. *–n-a* ($p=.01=.01$) and *–n-a* vs. *red* ($p=.000<.001$) (Fig. 3.18).

Figures 3.19 through 3.24 illustrate the differences in mean F1 frequencies for each of the prosodic conditions for each of the six ataxic speakers and their normal matched control. While the normal speakers consistently lowered the F1 frequency for the unaccented (*–a*) conditions relative to the *+a* conditions in the *-pf+a* vs. *–pf-a, +n+a* vs. *–n-a and –n-a* vs. *red* contrasts, the cerebellar speakers did not follow this pattern. Indeed, three of the six cerebellar speakers (JS, Fig.3.22; ES, Fig. 3.23; and MC, Fig. 3.24) all increased the *–a* condition in the *+n+a* vs. *–n*-a contrast.

Table 3.13: **Within-Subjects Main Effects: F1 frequency**

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Neurological status	1776.466	1	1776.466	.193	p=.679
Prosodic condition	560260.076	5	112052.015	63.011	p<.001***
Interaction between neurological status & prosodic condition	97985.795	5	19597.159	9.577	p<.001***

Based on estimated marginal means

*** The mean difference is significant at the .001 level

Table 3.14: **The marginal means for each group and mean difference (pair-wise comparison) of mean F1 at vowel mid-point between groups.**

Neurological Status	Mean	Standard Error	95% Confidence Interval		Sig.
	Mean Difference		Lower Bound	Upper Bound	
Cerebellar	774.102	30.136	696.635	851.570	
Normal	784.037	39.698	681.990	886.084	
Cerebellar - Normal	-9.934	22.633	-68.115	48.246	p=.679

Based on the estimated marginal means the mean difference between groups is not significant at the .05 level.

Table 3.15a: **Means of mid-point F1 for the interaction between prosodic condition and neurological status.**

Prosodic Condition	Neurological Status	Mean	Standard Error	95% Confidence Interval	
				Lower Bound	Upper Bound
+pf+a	Cerebellar	847.917	51.816	714.719	981.114
	Normal	852.750	43.418	741.140	964.360
-pf+a	Cerebellar	779.922	25.435	714.540	845.304
	Normal	866.494	44.802	751.328	981.661
-pf-a	Cerebellar	774.980	27.073	705.387	844.573
	Normal	808.344	38.033	710.576	906.111
+n+a	Cerebellar	789.220	18.908	740.615	837.826
	Normal	863.833	44.238	750.117	977.550
-n-a	Cerebellar	796.761	45.482	679.846	913.677
	Normal	795.017	45.534	677.976	912.066
red	Cerebellar	655.815	30.819	576.593	735.036
	Normal	517.783	35.805	425.743	609.824

Table 3.15b: Pair-wise comparisons of mean F1 at mid-point for the interaction of prosodic condition and neurological status.

Prosodic Condition	Neurological Status		Mean Difference	Standard Error	Sig.[a]	95% Confidence Interval	
						Lower Bound	Upper Bound
+pf+a	Cer	Nor	-4.833	30.297	.879	-82.715	73.048
-pf+a	Cer	Nor	-86.572	36.398	.063	-180.137	6.993
-pf-a	Cer	Nor	-33.364	28.316	.292	-106.153	39.425
+n+a	Cer	Nor	-74.613	42.529	.140	-183.938	34.712
-n-a	Cer	Nor	1.744	33.960	.961	-85.553	89.042
red	Cer	Nor	138.031	21.850	.001***	81.865	194.198

Based on estimated marginal means

*** The mean difference is significant at the .001 level.

[a] Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

Table 3.16: **Pair-wise comparisons of mean mid-point F1 frequency differences between prosodic conditions for each group: normal and cerebellar.**

Neurological Status	**Prosodic Contrast**	**Mean Difference**	Std. Error	Sig.[a]	**95% Confidence Interval for Difference[a]**	
					Lower Bound	Upper Bound
Normal	+pf+a vs. –pf+a	-13.744	27.165	.634	-83.575	56.086
	-pf+a vs. –pf-a	58.151*	18.454	.025*	10.712	105.590
	+n+a vs. –n-a	68.817*	17.107	.010**	24.842	112.792
	-n-a vs. red	277.233*	28.367*	.000***	204.314	350.152
Cerebellar	+pf+a vs. –pf+a	67.994	28.711	.064	-5.809	141.798
	-pf+a vs. –pf-a	4.943	9.248	.616	-18.829	28.714
	+n+a vs. –n-a	-7.541	34.228	.834	-95.526	80.445
	-n-a vs. red	140.946*	34.902	.010**	-230.665	-51.227

Based on the estimated marginal means

* The mean difference is significant at the .050 level, ** p=.01,*** p<.001.

[a] Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

Fig. 3.17: Mean F1 frequency at mid-point in Hz for the six prosodic conditions for cerebellar ataxic speakers and normals. The differences between group means is represented when significant with ***(p=.001).

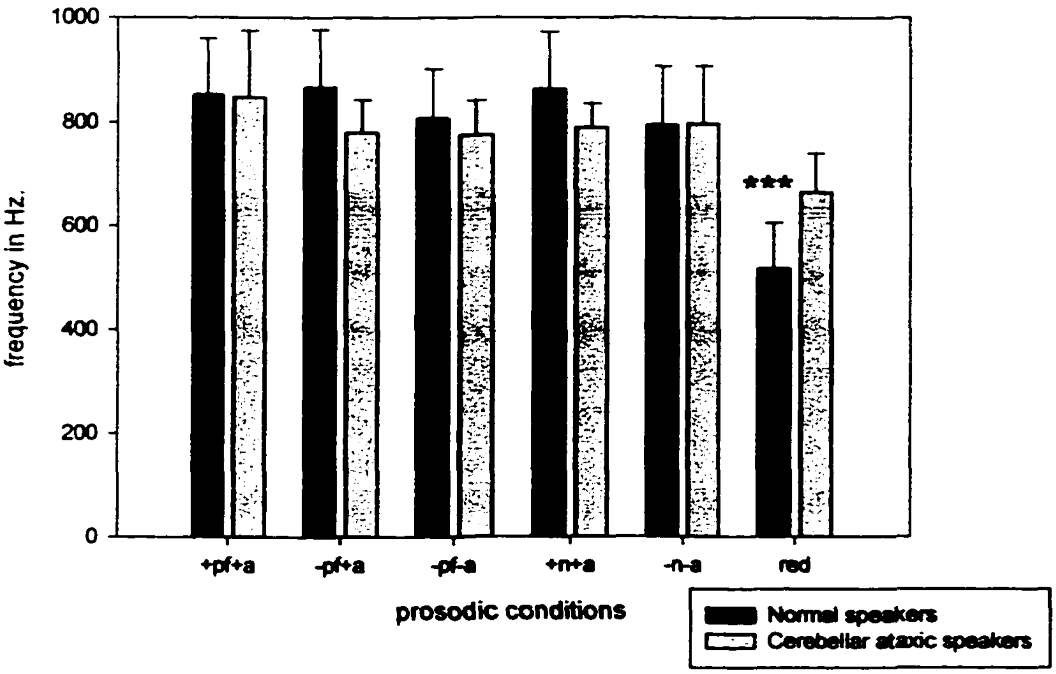


Fig. 3.18: Mean F1 frequencies at mid-point in Hz for each of the four prosodic contrasts for cerebellar ataxic speakers and normals. The differences in means within **each** group is represented as significant by *(p<.05) and **(p<.01 and ***(p<.001). (Table 3.16). The mean difference **between** groups for each of the four contrasts is marked for significance on the x axis -pf+a -pf-a (Tables 3.20 a,b) and -n-a red (Tables 3.24 a,b).

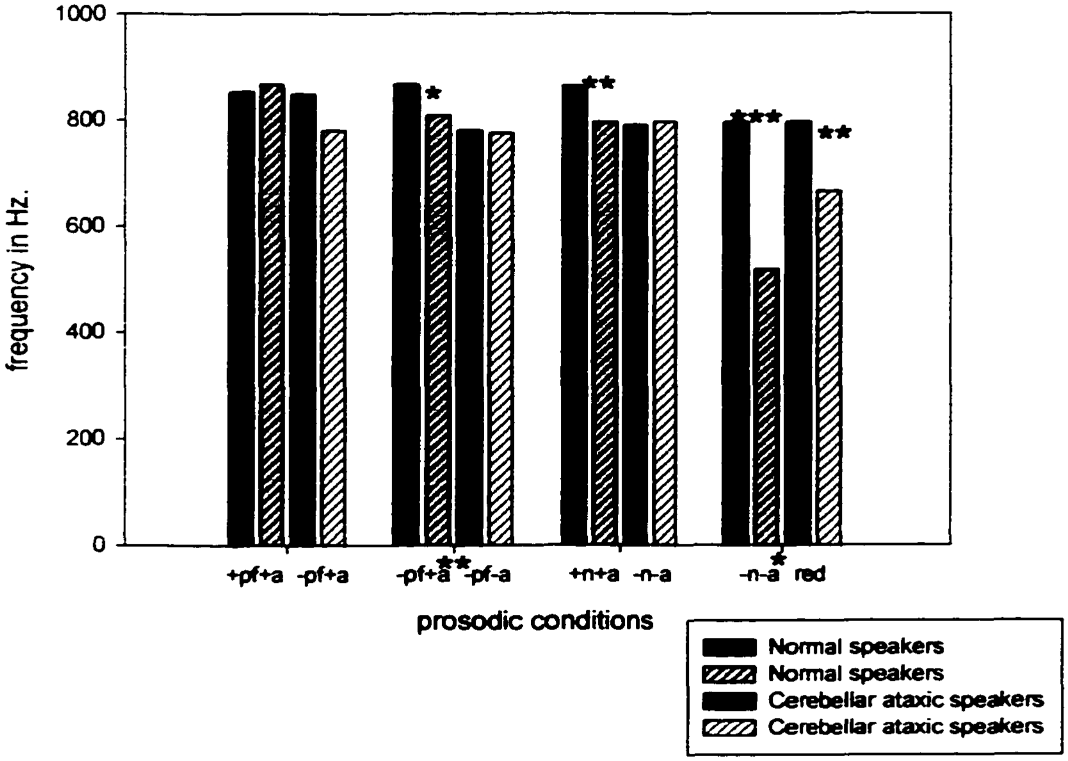


Fig. 3.19: **Mean F1 frequencies at mid-point in Hz for the six prosodic conditions for cerebellar ataxic speaker AH (D= 188.05; MRI = 2.0) and normal matched control LJR.**

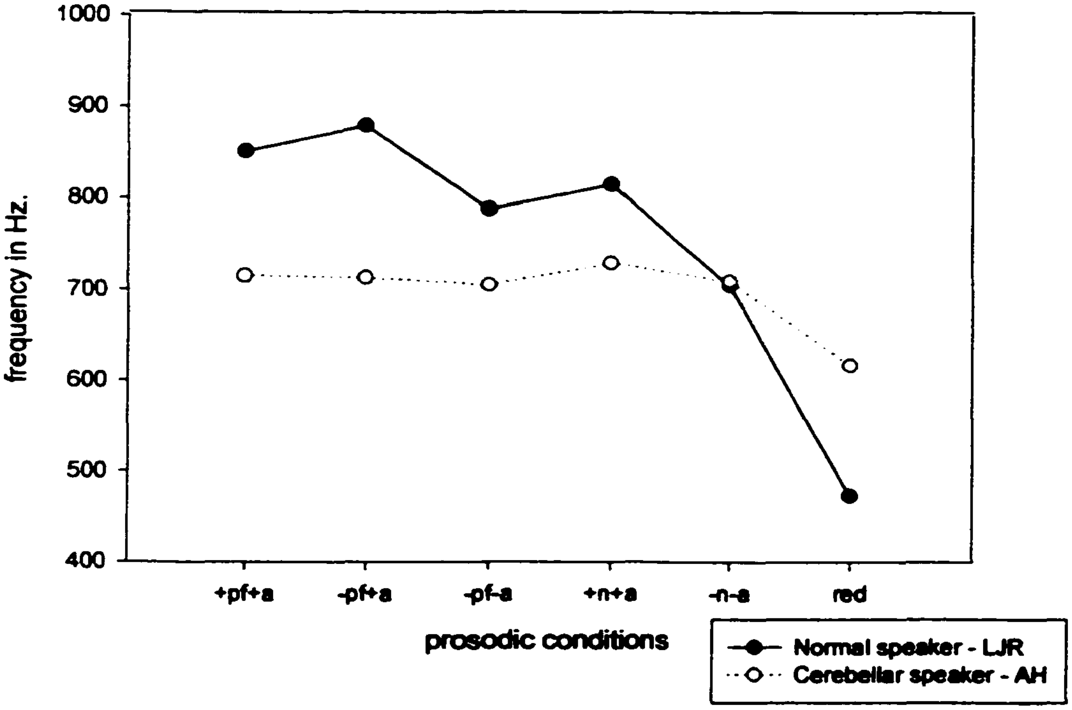


Fig. 3.20: **Mean F1 frequencies at mid-point in Hz for the six prosodic conditions for cerebellar ataxic speaker PP (D=91.32; MRI = 3.0) and normal matched control GC.**

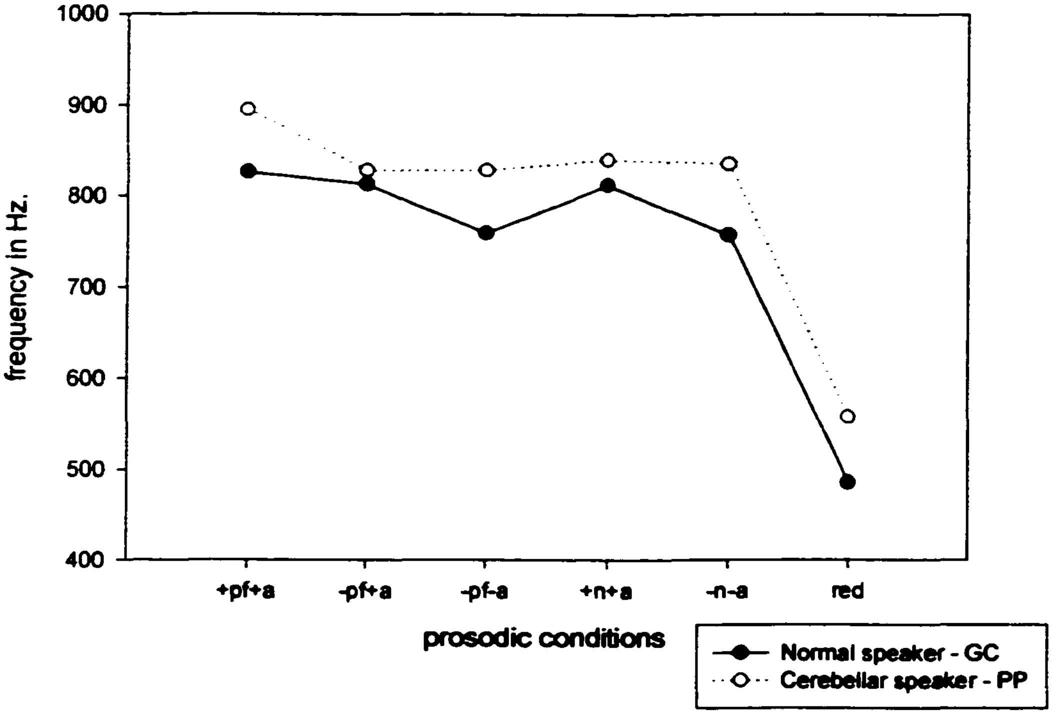


Fig. 3.21: **Mean F1 frequencies at mid-point in Hz for the six prosodic conditions for cerebellar ataxic speaker WD (D= 176.10; MRI = 2.5) and normal matched control BW.**

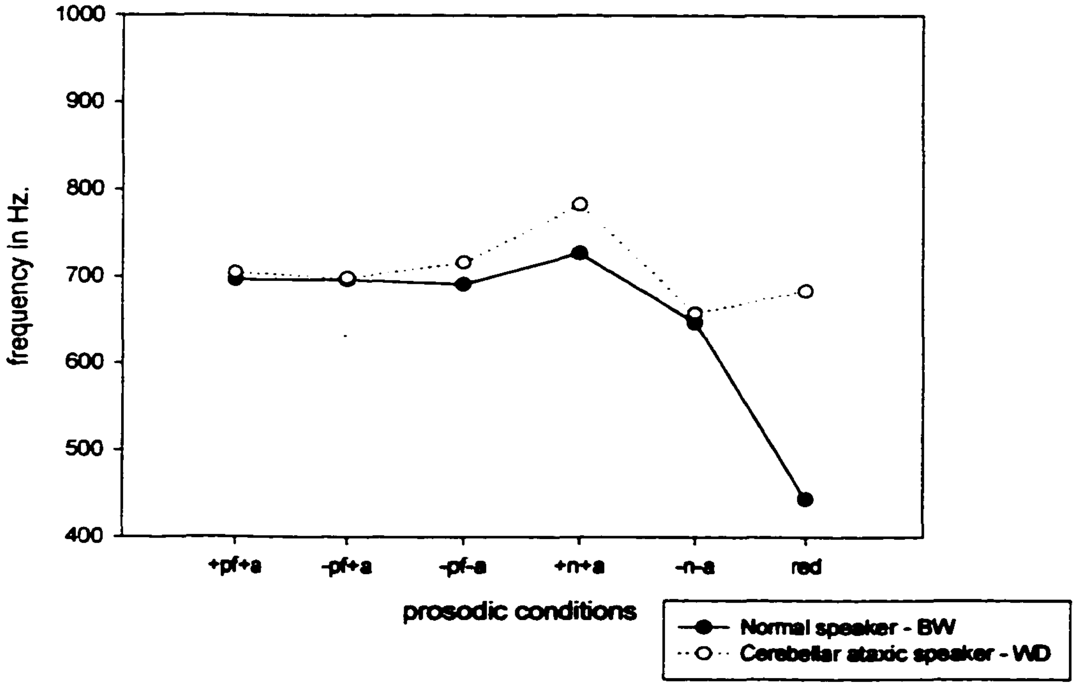


Fig. 3.22: Mean F1 frequencies at mid-point in Hz for the six prosodic conditions for cerebellar ataxic speaker JS (D= 373.98; MRI = 2.0) and normal matched control YM.

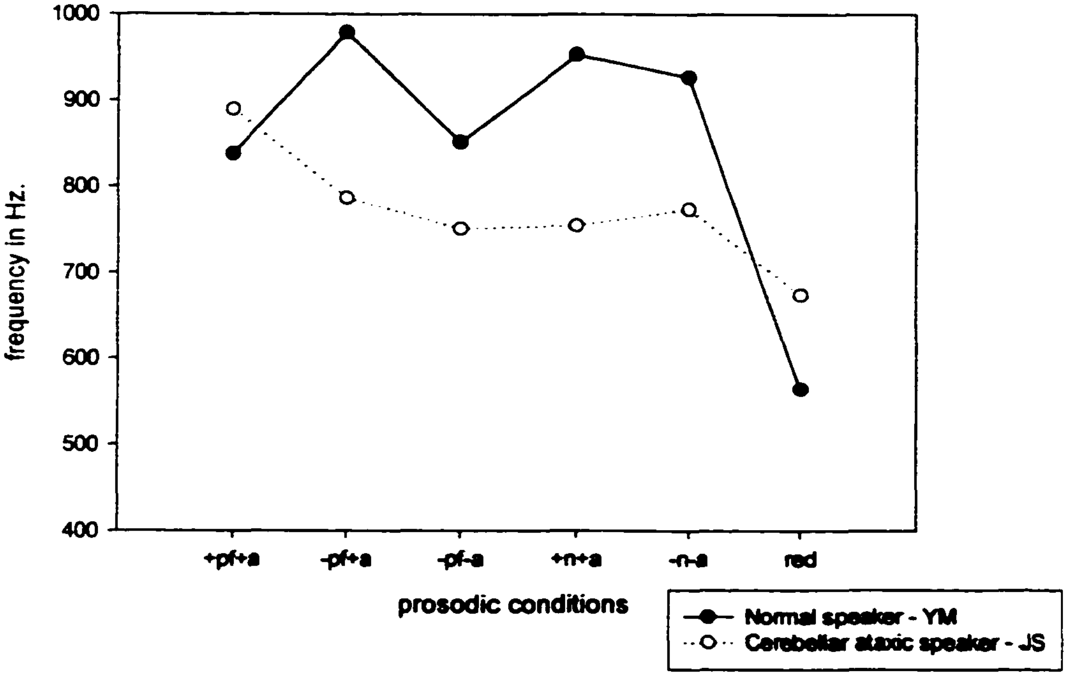


Fig. 3.23: Mean F1 frequencies at mid-point in Hz for the six prosodic conditions for cerebellar ataxic speaker ES (D= 296.67; MRI = 2.0) and normal matched control LL.

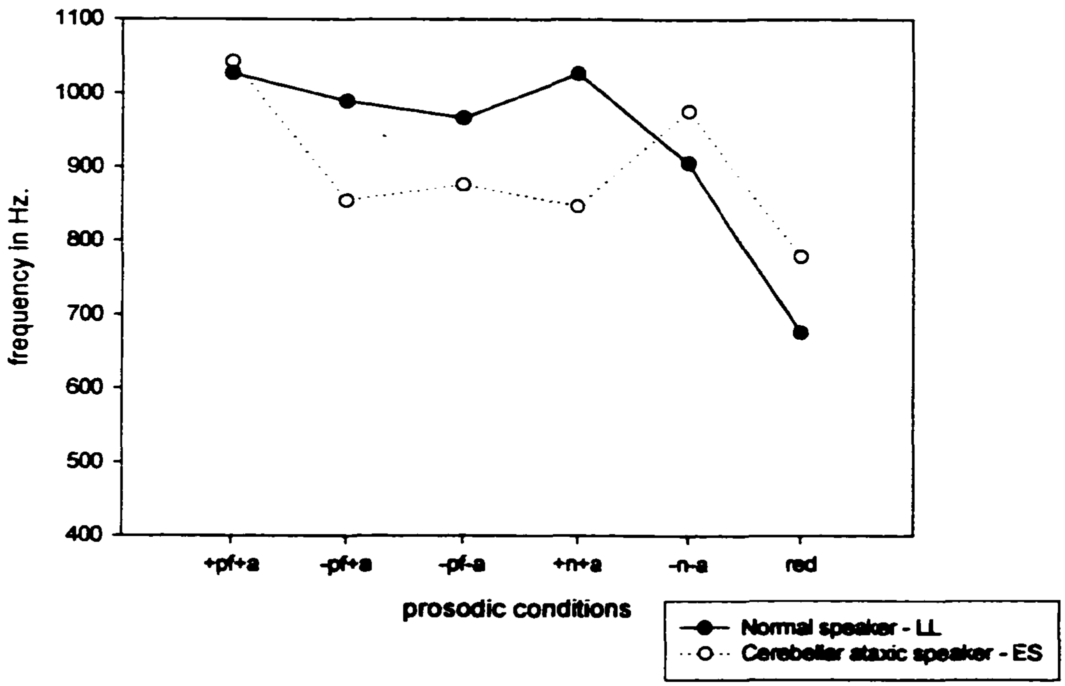
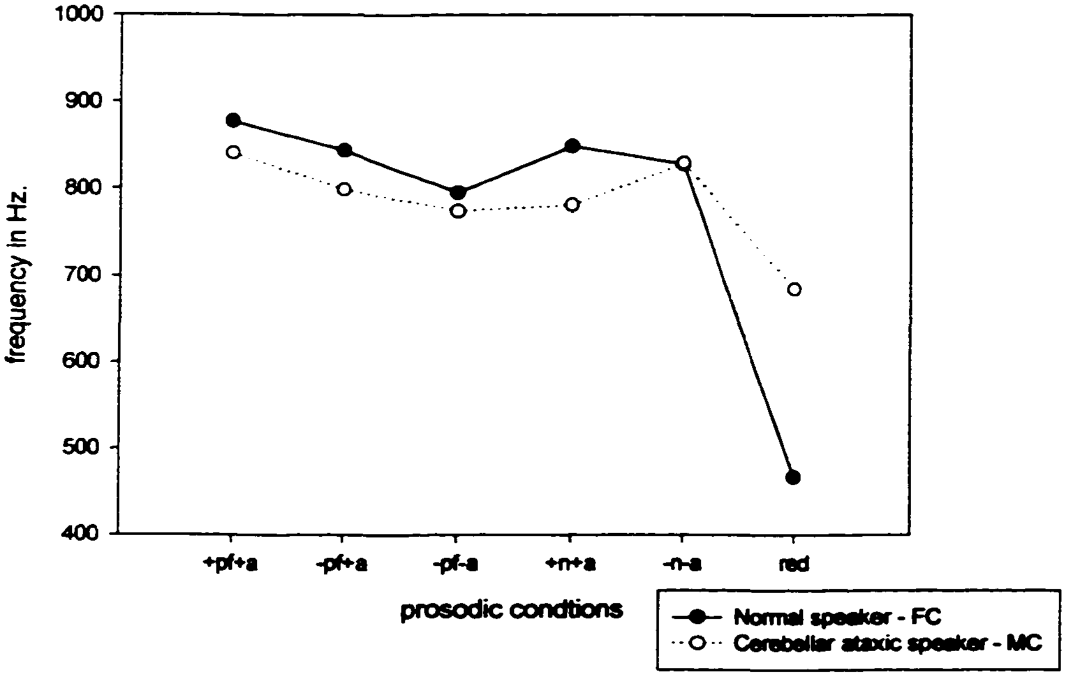


Fig. 3.24: Mean F1 frequencies at mid-point in Hz for the six prosodic conditions for cerebellar ataxic speaker MC (D= 228.81; MRI = 1.5) and normal matched control FC.



3.2.3 Simple interaction effects of prosodic contrast and neurological status

The final F1 and F2 formant frequency analysis examined the simple interaction effects for the four prosodic contrasts as a function of neurological status (Tables 3.17 a,b through 3.24 a,b). The two contrasts that were significantly different between groups for both F2 and F1 were *–pf+a* vs. *–pf-a* ($p= .016<.05$) and *-n-a* vs. ***red*** ($p= .017 <.05$).

3.2.3.1. The effects of cerebellar disturbance on F2 and F1 frequencies in lengthened syllables: contrast 1: +pf+a vs. –pf+a.

The purpose of this comparison is to consider the articulatory differences in these two accented conditions as a function of neural status. The ANOVAs and t tests for this contrast did not reveal a significant difference between the two groups for either F1 or F2 frequency (Tables 3.17 a,b and Tables:3.18 a,b). The pair-wise comparisons of conditions for each group also did not show a significant difference for either the normal or the cerebellar group (F2: Table 3.12; F1: Table 3.16). Figure 3.10 (F2) and Fig.3.18 (F1) show the differing trends of the two groups for this contrast: the normal group tends to have higher F2 and F1 frequencies for the *–pf+a* condition as compared to the *+pf+a* condition, whereas the cerebellar group tends to have lower F2 and F1 frequencies for the *–pf+a* condition in this contrast. .

Contrast 1: +pf+a vs. –pf+a: F2

Table 3.17a: Tests of Significance for Contrast 1: +pf+a vs. –pf+a: Unique Sum of Squares

Analysis of Variance :	Contrast 1= +pf+a F2 – -pf+a F2 : Cerebellar vs. Normal				
Source of Variation	SS	DF	MS	F	Sig. Of F
Within Cells	149453.18	5	29890.64		
Constant	36868.89	1	36868.89	1.23	p=.317

Table 3.17b: Estimates for Contrast 1: +pf+a F2 – -pf+a F2 :Cerebellar vs. Normal

Parameter	Coefficient	Std. Error	t-value	Sig. T	95% Level of Confidence	
					Lower	Upper
1	78.3888652	70.58167	1.11061	.317	-103.04435	259.82208

Contrast 1: +pf+a vs. –pf+a: F1

Table 3.18a: Tests of Significance for Contrast 1: +pf+a vs. –pf+a: Unique Sum of Squares

Analysis of Variance :	Contrast 1: +pf+a F1 – -pf+a F1: Cerebellar vs. Normal				
Source of Variation	SS	DF	MS	F	Sig. Of F
Within Cells	457778.92	5	9155.78		
Constant	40087.51	1	40087.51	4.38	p=.091

Table 3.18b:Estimates for Contrast 1: +pf+a F1 – -pf+a F1: Cerebellar vs. Normal

Parameter	Coefficient	Std. Error	t-value	Sig. T	95% Level of Confidence	
					Lower	Upper
1	81.7389221	39.06359	2.09246	.091	-18.67571	182.15355

3.2.3.2 The effects of cerebellar disturbance on F2 and F1 for accented and un-accented syllables in non-final position: contrast 2: -pf+a vs. –pf-a.

The purpose of this comparison is to consider the artculatory differences between accented and unaccented syllables as a function of neural status. The ANOVAs and t tests for this contrast revealed a significant difference at the .05 level between the two groups for F2 ($p=.016<.05$) and at the .01 level for F1($p=.007<.01$) (Tables 3.19 a,b and Tables 3:20 a,b).

Within group analysis revealed a significant difference between the *–pf+a* and *–pf-a* conditions for F2 ($p = .039 < .05$) and F1($p= .025$) (Table 3.12 and Table 3.16) but not for the cerebellar group (F2: $p= .751 >.05$; F1: $p= .616 >.05$). Thus, it appears the production of this contrast may be a characteristic of normal speech motor dynamics that is not shared by cerebellar speakers. The normal speakers display lower F2 and F1 values in the unaccented condition as compared to the accented condition while the cerebellar speakers did not. Within the group of cerebellar speakers, some had increased frequencies for unaccented syllables in this phrase accent contrast [6] and others had equal frequencies[7] for this phrase accent contrast.

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[6] AH (F2): Fig.3.11; WD (F1): Fig. 3.19; ES (F2;F1):Fig. 3.15; Fig.3.23 and MC (F2;F1): Fig.3.16; Fig. 3.24;

[7] AH (F1): Fig. 3.19 and PP (F2; F1) Fig. 3.12 and Fig. 3.20.

Contrast 2: -pf+a vs. –pf-a: F2

Table 3.19a: Tests of **Significance for Contrast 2: -pf+a vs. –pf-a: Unique Sum of Squares**

Analysis of Variance :	**Contrast 2= -pf+a F2 – -pf-a F2 : Cerebellar vs. Normal**				
Source of Variation	SS	DF	MS	F	Sig. of F
Within Cells	14001.05	5	2800.21		
Constant	35391.95	1	35391.95	12.64	p=.016*

Table 3.19b:**Estimates for Contrast 2 : -pf+a F2 – -pf-a F2: Cerebellar vs. Normal**

Parameter	**Coefficient**	Std. Error	t-value	Sig. T	95% Level of Confidence	
					Lower	Upper
1	-76.802724	21.60328	-3.55514	.016*	-132.33488	-21.27057

Contrast 2: -pf+a vs. –pf-a: F1

Table 3.20a: Tests of **Significance for Contrast 2 : Unique Sum of Squares**

Analysis of Variance :	**Contrast 2: -pf+a F1– -pf-a F1: Cerebellar vs. Normal**				
Source of Variation	SS	DF	MS	F	Sig. of F
Within Cells	4347.13	5	869.43		
Constant	16986.78	1	16986.78	19.54	p=.007**

Table 3.20 b:**Estimates for Contrast 2: -pf+a F2 – -pf-a F2: Cerebellar vs. Normal**

Parameter	**Coefficient**	Std. Error	t-value	Sig. T	95% Level of Confidence	
					Lower	Upper
1	-53.208364	12.03762	-4.42017	.007**	-84.15158	-22.26515

3.2.3.3 . The effects of cerebellar disturbance on F2 and F1 for accented and unaccented syllables in nuclear and post-nuclear positions: contrast 3: +n+a vs. –n-a..

The nuclear accented (+n+a) syllable contains the speakers' primary focus and therefore the greatest prominence in the sentence, whereas the phrase-accented syllable may be expected to have less prominence. The purpose of this comparison is to consider the difference between F2 and F1 frequencies in nuclear accented (+n+a) and post-nuclear unaccented (-n-a) syllables as a function of neurological status. The analysis of variance did not reveal a significant difference between the two groups.

Within-group analyses revealed significant differences between the *+n+a* and *–n-a* conditions for the normal group for F2 ($p = .018 < .05$) and F1 ($p= .010 \leq .01$) but not for the cerebellar group: F2: $p=.186 > .05$; F1: $p= .834$. (See Table 3.12 and Table 3.16 respectively). Once again, the production of this contrast appears to be a characteristic of normal speech motor dynamics that is not used by cerebellar speakers. Three of the cerebellar speakers, JS, ES and MC showed increased F2 and frequencies for the unaccented syllable in the post nuclear position (Figures 3.14, 3.15, 3.16 for F2 values and Figures 3.22, 3.23 and 3.24 for F1 values).

Contrast 3: +n+a vs. –n-a: F2

Table 3.21a: Tests of Significance for Contrast 3: +n+a vs. –n-a : Unique Sum of Squares

Analysis of Variance :	Contrast 3= +n+a F2– -n-a F2 : Cerebellar vs. Normal				
Source of Variation	SS	DF	MS	F	Sig. of F
Within Cells	61832.68	5	12366.54		
Constant	73178.03	1	73178.03	5.92	p= .059

Table 3.21b: Estimates for Contrast 3: +n+a F2– -n-a F2: Cerebellar vs. Normal

Parameter	Coefficient	Std. Error	t-value	Sig. T	95% Level of Confidence	
					Lower	Upper
1	-110.43703	45.39922	-2.43258	.059	-227.13768	6.26361

Contrast 3: +n+a vs. –n-a: F1

Table 3.22a: Tests of Significance for Contrast 3: +n+a vs. –n-a : Unique Sum of Squares

Analysis of Variance:	Contrast 3= +n+a F1– -n-a F1 : Cerebellar vs. Normal				
Source of Variation	SS	DF	MS	F	Sig. of F
Within Cells	47005.23	5	9401.05		
Constant	34982.71	1	34982.71	3.72	p=.112

Table 3.22b: Estimates for Contrast 3 : +n+a F1 vs. -n-aF1: Cerebellar vs. Normal

Parameter	Coefficient	Std. Error	t-value	Sig. T	95% Level of Confidence	
					Lower	Upper
1	-76.357391	39.58334	-1.92903	.112	-178.10807	25.39328

3.2.3.4. The effects of cerebellar disturbance on F2 and F1 for unaccented post-nuclear and reduced syllables: contrast 4: -n-a vs. red.

The purpose of this comparison is to examine the differences between unaccented (*-a*) and reduced syllables (*red*) as a function of neurological status. The group analysis of variance revealed a significant difference between the two groups for F2 ($p=.017< .05$) and F1($p= .024 < .05$) for this contrast. See (Tables:3.23 a,b and 3.24 a,b).

Within-group analyses (Tables 3.12 and 3.16) revealed significant differences in this contrast for the normal group for both F2 ($p = .006<.01$) and F1 ($p=.000 < .001$) frequency, whereas the cerebellar group showed a significant difference only for F1($p=.010 \leq .01$) frequency. Although both subject groups showed a significant difference between conditions for this contrast in the F1 measure, normal speakers produced a greater difference between conditions than the cerebellar speakers.

An examination of patterns for individual subjects (F2: Figures 3.11 to 3.16 and F1: Figures 3.19 to 3.24) showed that one subject (AH: Fig. 3.11; Fig. 3.19) reversed the pattern found in his normal control for both acoustic measures of F2 and F1 while another J.S.(Fig. 3.14) showed an extreme reversal from her normal control for this contrast in F2 but not in F1. M.C. (Fig. 3.16) showed equivalent values for the measure of F2. The pattern found for the other three ataxic speakers for the measurement of F2 and F1 was simply a less extreme contrast between conditions than the normal controls.

Contrast 4: -n-a vs. red: F2

Table 3.23a: Tests of **Significance for Contrast 4 : -n-a vs. red: Unique Sum of Squares**

Analysis of Variance:	**Contrast 4= -n-a F2 vs. red F2 : Cerebellar vs. Normal**				
Source of Variation	SS	DF	MS	F	Sig. of F
Within Cells	66031.69	5	13206.34		
Constant	160873.48	1	160873.48	12.18	p= .017*

Table 3.23b: **Estimates for Contrast 4 – -n-a F2 vs. red F2: Cerebellar vs. Normal**

Parameter	Coefficient	Std. Error	t-value	Sig. T	95% Level of Confidence	
					Lower	Upper
1	-163.74446	46.91542	-3.49021	.017*	-284.34254	-43.14637

Contrast 4: -n-a vs, red: F1

Table 3.24a: Tests of **Significance for Contrast 4:-n-a vs. red: Unique Sum of Squares**

Analysis of Variance:	**Contrast 4= -n-a F1 vs. red F1 : Cerebellar vs. Normal**				
Source of Variation	SS	DF	MS	F	Sig. of F
Within Cells	54308.72	5	10861.74		
Constant	111444.93	1	111444.93	10.26	p=.024*

Table 3.24b: **Estimates for Contrast 4: -n-a F1 vs. red F1: Cerebellar vs. Normal**

Parameter	Coefficient	Std. Error	t-value	Sig. T	95% Level of Confidence	
					Lower	Upper
1	-136.28703	42.54751	-3.20317	.024*	-245.65723	-26.91684

3.2.4 Discussion and conclusions: the effects of cerebellar degeneration on formant frequencies: F1 and F2.

When we examine the differences between different conditions of stress for the normal speakers' productions of [ɑ] in the syllable [pɑp], what appears is a pattern of increased frequency for both F2 and F1 in those syllables that are more prominent. Following the three parameter model of Stevens and House (1955), an increase in F2 resonance may be effected by tongue fronting whereas an increase in F1 may be effected by a narrowing in the pharyngeal cavity that may accompany jaw lowering and/or tongue backing.

The parameters specified by Stevens and House (1955) present a model for an estimating the acoustic effects of articulatory events. A high first formant is associated with a narrow tongue constriction near the glottis and an un-rounded large mouth opening. When the first formant is low the mouth opening is small and rounded or there is a narrow tongue constriction near the mouth opening. F2 generally increases in frequency as the point of constriction moves forward from the glottis.

The F1 and F2 frequencies of each of the six cerebellar subjects were compared to those of their normal matched controls for four prosodic contrasts *(+pf+a* vs. *–pf+a, -pf+a* vs. *–pf-a, +n+a* vs. *–n-a and –n-a* vs. *red*). See (Figures. 3.11 through 3.16 and Fig. 3.19 through 3.24). The ataxic speakers showed markedly different patterns of formant frequencies from those of the normal speakers. The normal speakers, as a group, follow a consistent pattern of both F2 and F1 values for three of the four prosodic contrasts examined here.

Those syllables with greater prosodic prominence in a particular contrast show higher frequency values for both F2 and F1. This pattern is consistent with the literature that claims that more stressed syllables are hyper-articulated, although the pattern is not necessarily consistent with the claims that stressed syllables are produced with less co-articulation (deJong et al., 1993). The relatively higher F1 values for accented syllables as compared to unaccented syllables may be taken to reflect the more open oral cavity and narrow pharynx found in the production of the vowel [ɑ]. The relatively higher F2 frequencies for accented syllables may be explained in part by the forward position of the tongue.

A significant difference between subject groups for both F2 and F1 frequencies was found in the reduced prosodic condition. See (Fig. 3.9 and Fig. 3.17, respectively). A significant difference was found within the cerebellar group only for F1 frequency for the *-n-a* vs. *red* contrast, with F1 frequency being lower in the reduced condition. Within the normal group, significant differences were found for both F2 and F1 frequencies for three of the four contrasts: *-pf+a* vs. *–pf-a, +n+a* vs. *–n-a,* and *–n-a* vs. *red* with both F2 and F1 frequencies lower in the less prominent condition. The two groups differed significantly on the *-pf+a* vs. *–pf-a* and *–n-a* vs. *red* contrasts.

. The formant patterns in the relatively more prominent syllables may be explained not necessarily by relatively less coarticulation in un-accented syllables but rather by a lack of coarticulatory constraint between the lingual and labial articulatory mechanism (F.Bell-Berti, personal communication). Since hyper-articulation is generally understood as a reduction in coarticulation for stressed syllables (de Jong et al. 1993), the explanation of articulatory events for the more prominent syllables as presented here is better represented as an expression of formant extremum than of hyper-articulation.

3.3 The effects of prosodic condition on fundamental frequency

Table 3.25 presents the three main effects of neurological status, prosodic condition and the interaction between neurological status and prosodic condition for fundamental frequency. Two of the three main effects were found to be significant: the effect of prosodic condition ($p=.000<.001$) and the interaction between neurological status and prosodic condition ($p=.000< .001$).

Table 3.26 presents the f_0 means as a function of neurological status. The mean f_0 across conditions for each group and the mean difference between the two groups is presented. The mean difference between the groups was only 3.421 Hz, which does not represent a significant difference. However, further analysis of the prosodic conditions and contrasts will reveal that the values for conditions vary significantly both within and between groups.

Tables 3.27a and 3.27b present the statistical analysis of the interaction between prosodic condition and neurological status. A significant difference between groups was found for the *red* condition ($p= .041< .05$). In addition, an examination of the means for each of the prosodic conditions revealed a tendency for differences between groups. As found in the measures of F2 and F1 frequency, normal speakers showed a distinct pattern of differences across conditions (Fig. 3.25), with the lowest mean f_0 for the normal group occurring in the *red* condition with speakers averaging 130.8 Hz. In contrast, the highest value was *+n+a* condition at 212.5 Hz. Normal speakers produced higher frequency values for the accented (*+a*) conditions (*-pf+a*: 203.3 Hz. and *+n+a* 212.5 Hz.), while the unaccented (*–a*) conditions (*-pf-a*: 198.1 Hz., *-n-a*: 143.1 Hz.) were relatively lower in frequency. However, the *+pf+a* condition (190.8 Hz.) was lower than even the *–pf-a* (198.1 Hz.) Taken together with the measures of F1 and F2 frequency described above give one pause in considering the different underlying mechanism of control for *+pf+a* syllables. It is evident that normal speakers produce f_0 in the *+pf+a* condition distinctly differently from other accented syllables. The relatively lower f_0 for an accented syllable in phrase-final position supports findings for a different mechanism of control for syllables in phrase-final position (Cohen et al.,1995; Beckman & Edwards, 1994; Harris, 1978).

The cerebellar speakers did not produce the range of f_0 differences seen in normals: (Fig. 3.25). Cerebellar speakers' f_0 values ranged from 169 Hz. to 185 Hz. (16 Hz. difference) whereas normal speakers' f_0 values ranged from 131 Hz. to 213 Hz. (82 Hz. difference) across prosodic conditions. The highest f_0 for cerebellar ataxic speakers was the *+pf-a* condition (185 Hz.) and the lowest value was for the *–n-a* condition at (169 Hz.)

The only condition to show a significant difference between groups for the measure of f_0 was the *+n+a* vs. *–n-a* condition ($p=.009<.01$).

The four contrasts between prosodic conditions were analyzed for each subject group (Table 3.28 and Fig. 3.26). The cerebellar group showed no significant differences in the mean f_0 for any of the four prosodic contrasts. In contrast, the normal group showed significant differences for three of the four prosodic contrasts: *+pf+a* vs. *–pf+a* ($p=.036<.05$*); *+n+a* vs. *–n-a* (p=.002<.01**) and *-n-a* vs. *red* (p=.045<.05*). The one contrast that was not significanct for the normal group was the *–pf+a* vs. *–pf-a*.

Across the four acoustic measures investigated, it appears that significant differences for syllable durations, formant measures and for fundamental frequency measures occur more often in normal speakers' data than in those speakers with cerebellar pathology. The normal speakers showed significant differences for final lengthening (*+pf+a* vs. *–pf+a*) in the measure of f_0 but not in the formant frequencies. The F1 and F2 frequencies showed significant differences for *+a* in the contrast *–pf+a* vs. *–pf-a*, while the f_0 difference was not significant for this contrast (Tables 3.4, 3.12, 3.16, 3.28 and 3.29).

Table 3.25: **Within-Subjects Effects: f_0**

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Neurological status	210.647	1	210.647	.090	p=.777
Prosodic condition	23674.229	5	4734.846	16.750	p<.001***
Interaction between neurological status and prosodic condition	12547.678	5	2509.536	6.632	p<.001***

Table 3.26: **The marginal means for each group and mean difference (pair-wise comparison) between groups for the acoustic measure of f_0**

Neurological Status	Mean	Standard Error	95% Confidence Interval		Sig.
	Mean Difference		Lower Bound	Upper Bound	
Cerebellar	176.338	19.502	126.206	226.470	
Normal	179.759	11.999	148.914	210.605	
Cerebellar - Normal	-3.421	11.426	-32.791	25.949	p=.777

Based on the estimated marginal means the mean difference between groups is not significant at the .05 level.

Table 3.27a: **Means of mid-point f_0 for the interaction between prosodic condition and neurological status.**

Prosodic Condition	Neurological Status	Mean	Standard Error	95% Confidence Interval	
				Lower Bound	Upper Bound
+pf+a	Cerebellar	176.617	18.355	125.432	219.801
	Normal	190.750	13.508	156.025	225.475
-pf+a	Cerebellar	181.302	19.534	131.089	231.515
	Normal	203.259	14.100	167.014	239.505
-pf-a	Cerebellar	184.528	20.532	131.748	237.308
	Normal	198.135	19.656	147.608	248.661
+n+a	Cerebellar	180.344	17.016	136.603	224.086
	Normal	212.517	10.149	186.427	238.607
-n-a	Cerebellar	169.172	22.419	111.543	226.802
	Normal	143.083	11.748	112.884	173.283
red	Cerebellar	170.068	24.162	107.957	232.179
	Normal	130.812	13.119	97.088	164.536

Table 3.27b: Pair-wise comparisons of mid-point f_0 for the interaction of prosodic condition and neurological status.

Prosodic Condition	Neurological	Status	Mean Difference	Standard Error	Sig.[a]
+pf+a	Cerebellar	Normal	-18.133	14.038	.253
-pf+a	Cerebellar	Normal	-21.957	13.507	.165
-pf-a	Cerebellar	Normal	-13.607	18.896	.504
+n+a	Cerebellar	Normal	-32.172	13.790	.067
-n-a	Cerebellar	Normal	26.089	16.796	.181
red	Cerebellar	Normal	39.256*	14.321	.041*

Based on estimated marginal means

* The mean difference is significant at the .050 level.

[a] Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

Table 3.28: Pair-wise comparisons (mean mid-point f_0 differences between prosodic conditions) for each group: normal and cerebellar.

Neurological Status	Prosodic Contrast	Mean Difference	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
Normal	+pf+a vs. –pf+a	-12.509	4.378	.036*	-23.764	-1.255
	-pf+a vs. –pf-a	5.125	6.025	.434	-10.363	20.612
	+n+a vs. –n-a	69.433**	11.521	.002**	39.817	99.050
	-n-a vs. red	12.271*	4.603	.045*	.440	24.103
Cerebellar	+pf+a vs. –pf+a	-8.685	3.739	.068	18.296	.926
	-pf+a vs. –pf-a	-3.226	4.971	.545	-16.004	9.552
	+n+a vs. –n-a	10.277	16.124	.552	-31.170	51.724
	-n-a vs. red	-.895	9.116	.926	-24.329	22.538

Based on the estimated marginal means

* The mean difference is significant at the .050 level; ** at the .01 level.

[a] Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

Table 3.29: Summary table of significance in the four acoustic measures for pair-wise comparisons (mean differences between prosodic conditions) for each group: normal and cerebellar.

Neurological Status	Prosodic Contrast	**Syllable Duration**	**F2**	**F1**	**f_0**
Normal	+pf+a vs. –pf+a	*			*
	-pf+a vs. –pf-a	*	*	*	
	+n+a vs. –n-a	**	*	**	**
	-n-a vs. red	**	**	***	*
Cerebellar	+pf+a vs. –pf+a	*			
	-pf+a vs. –pf-a				
	+n+a vs. –n-a				
	-n-a vs. red			**	

Based on the estimated marginal means

* The mean difference is significant at the .050 level, ** significant at the .01 level and *** significant at the .001 level.

Fig. 3.25: Mean f0 at mid-point in Hz across the six prosodic conditions for cerebellar ataxic speakers and normals. The mean difference between groups is represented as significant by *(p<.05).

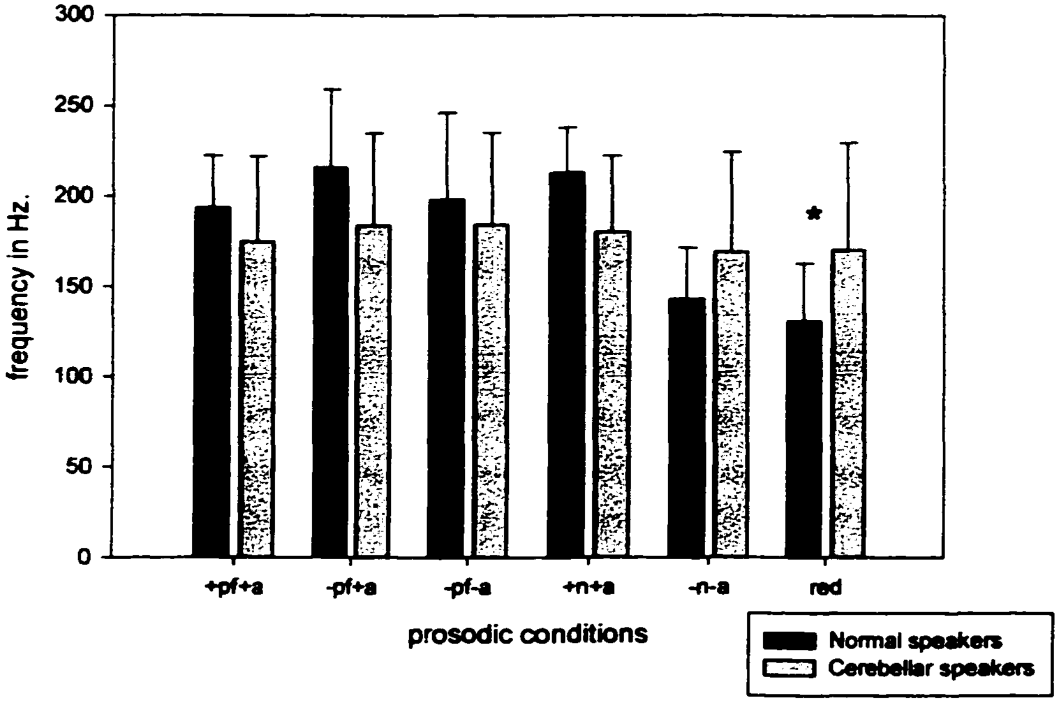


Fig. 3.26: **Mean f0 at mid-point in Hz for each of the four prosodic contrasts for cerebellar ataxic speakers and normals. The difference in means within each group is represented as significant by *(p<.05) and **(p<.01). (Table 3.28). The mean difference between groups for each of the four contrasts is marked for signicance on the x axis: +n+a -n-a (Tables 3.31 a,b).**

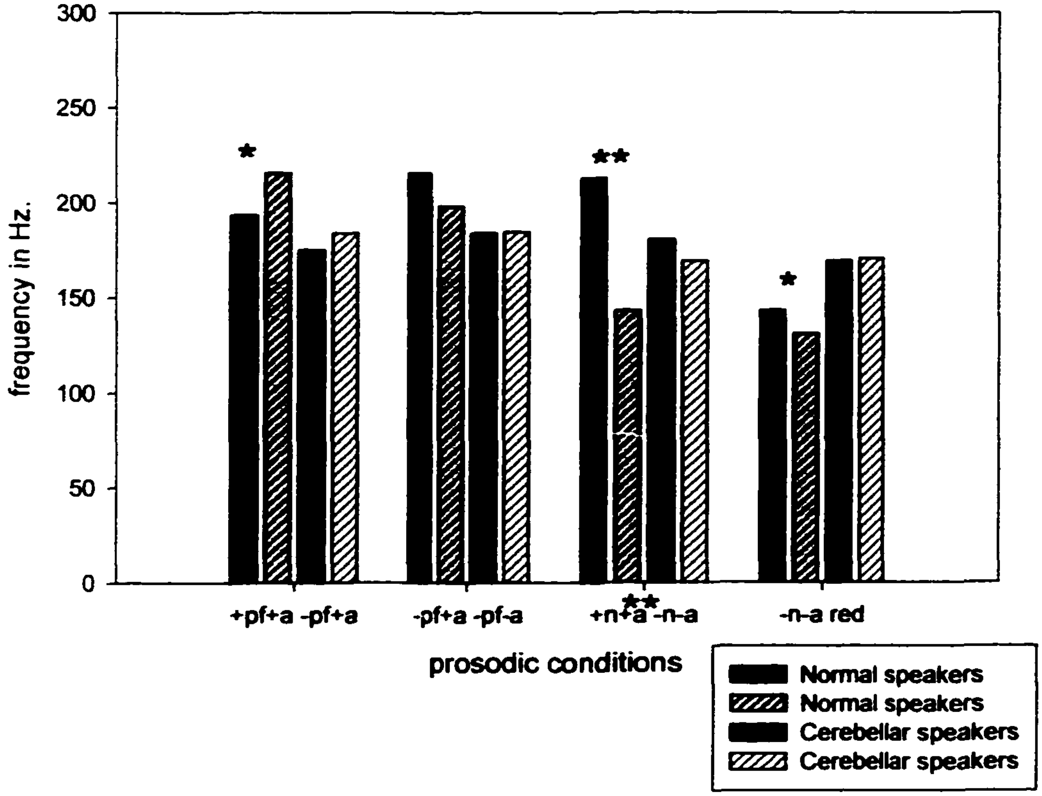


Fig. 3.27: Mean f0 at mid-point for the six prosodic conditions for cerebellar ataxic speaker AH (D = 90.54; MRI = 2.0) and normal matched control LJR.

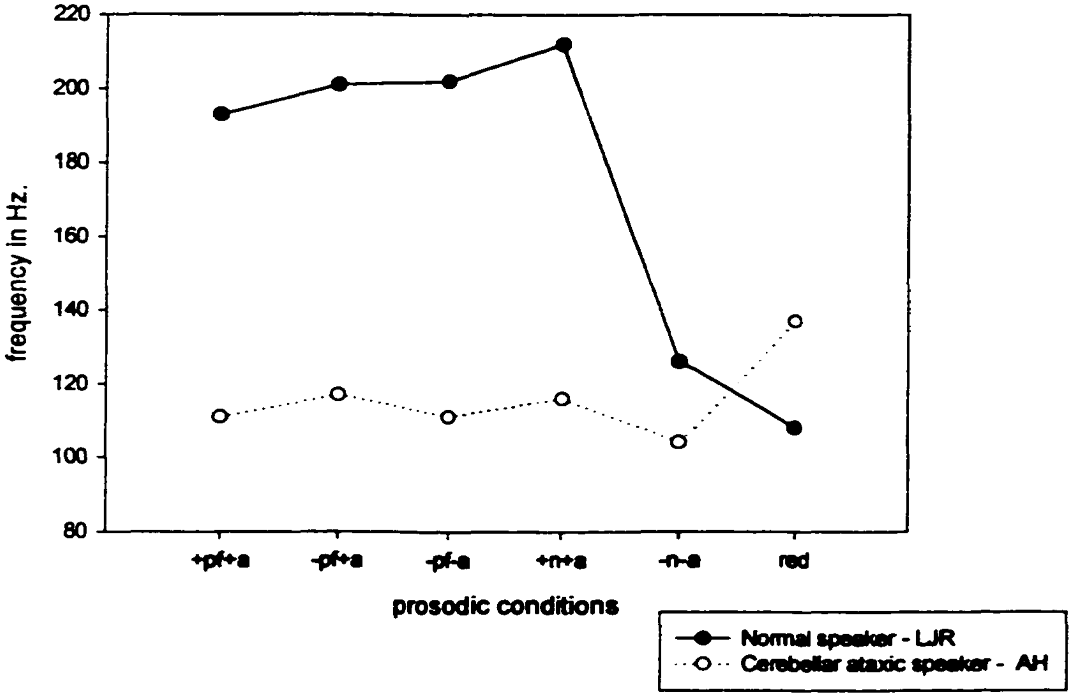


Fig.3.28: Mean f0 at mid-point for the six prosodic conditions for cerebellar ataxic spaker PP (D = 87.85; MRI = 3.0)and normal matched control GC.

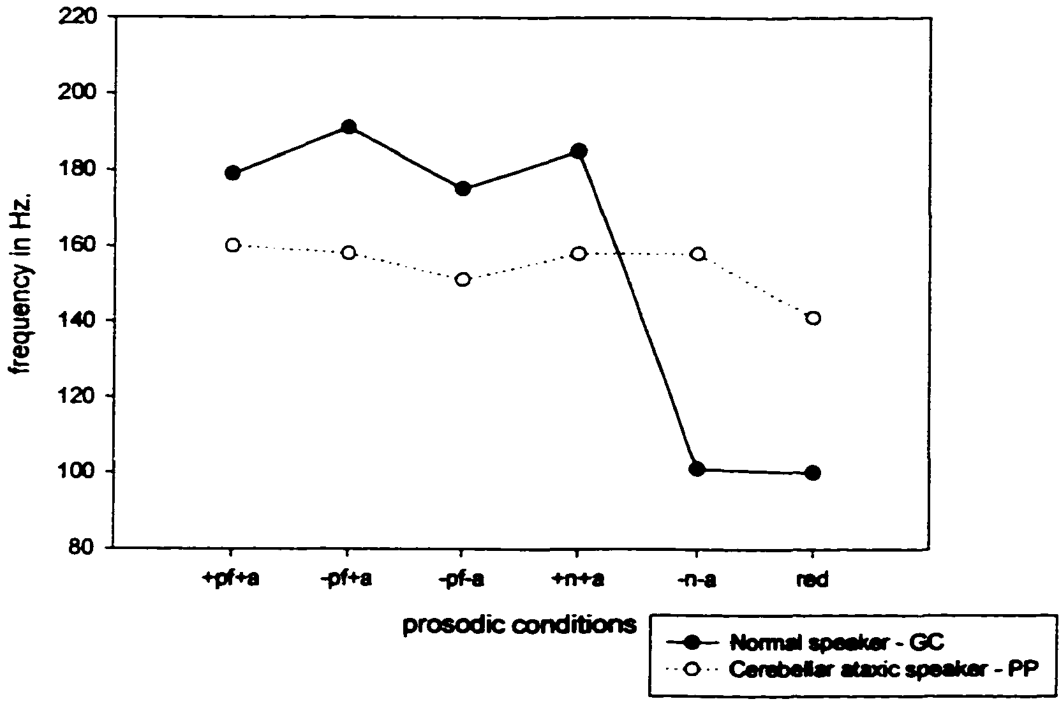


Fig. 3.29: Mean f0 at mid-point for the six prosodic conditions for cerebellar ataxic speaker WD (D = 54.60; MRI = 2.5) and normal matched control BW.

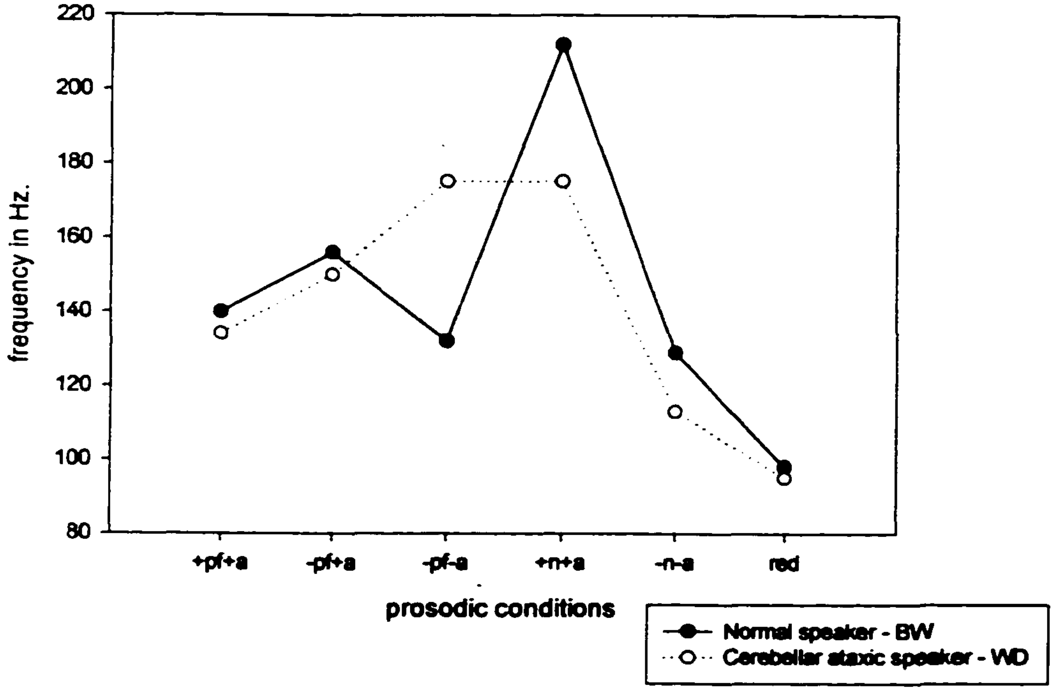


Fig.3.30: Mean f0 at mid-point for the six prosodic conditions for cerebellar ataxic speaker JS (D= 102.40; MRI = 2.0) and normal matched control YM.

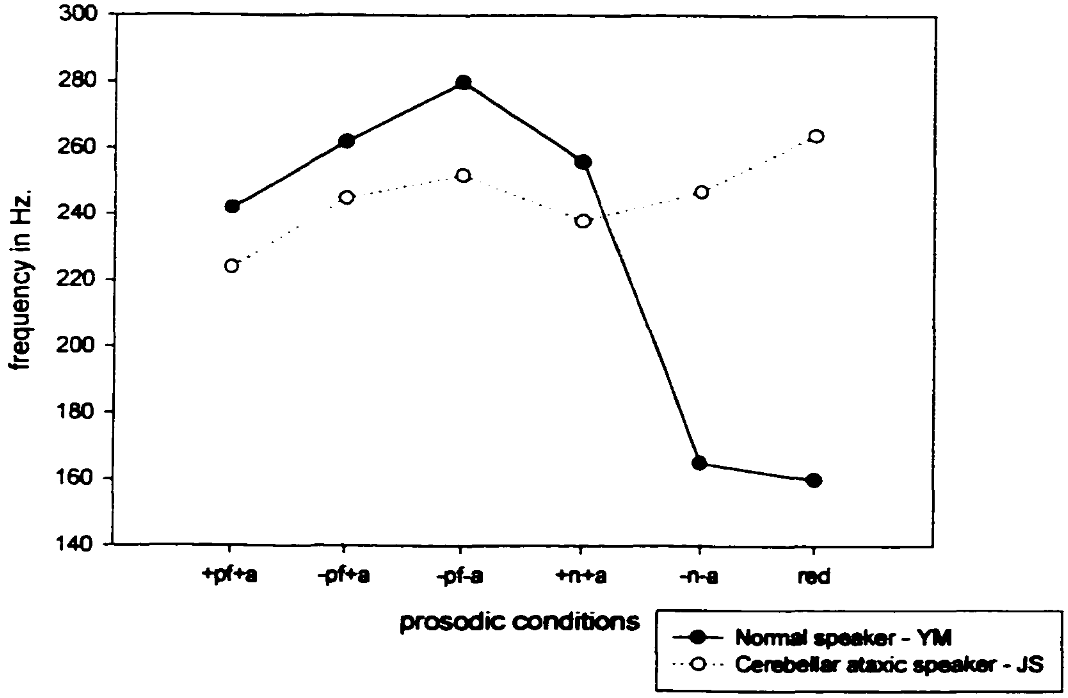


Fig.3.31: Mean f0 at mid-point for the six prosodic conditions for cerebellar ataxic speaker ES (D = 58.45; MRI = 2.0) and normal matched control LL.

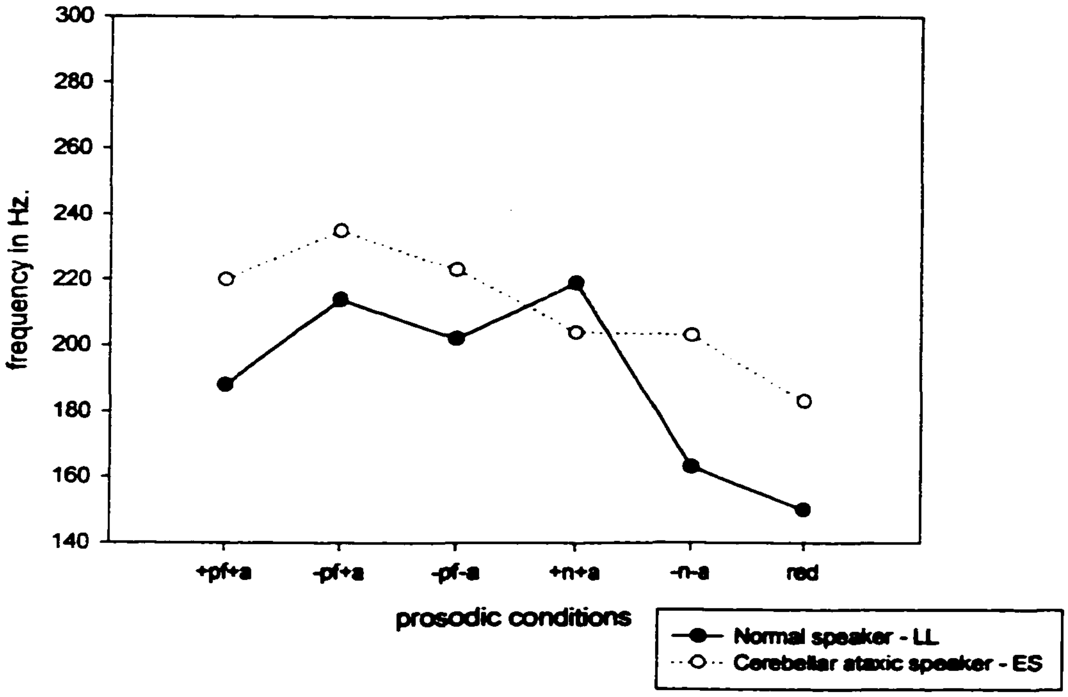
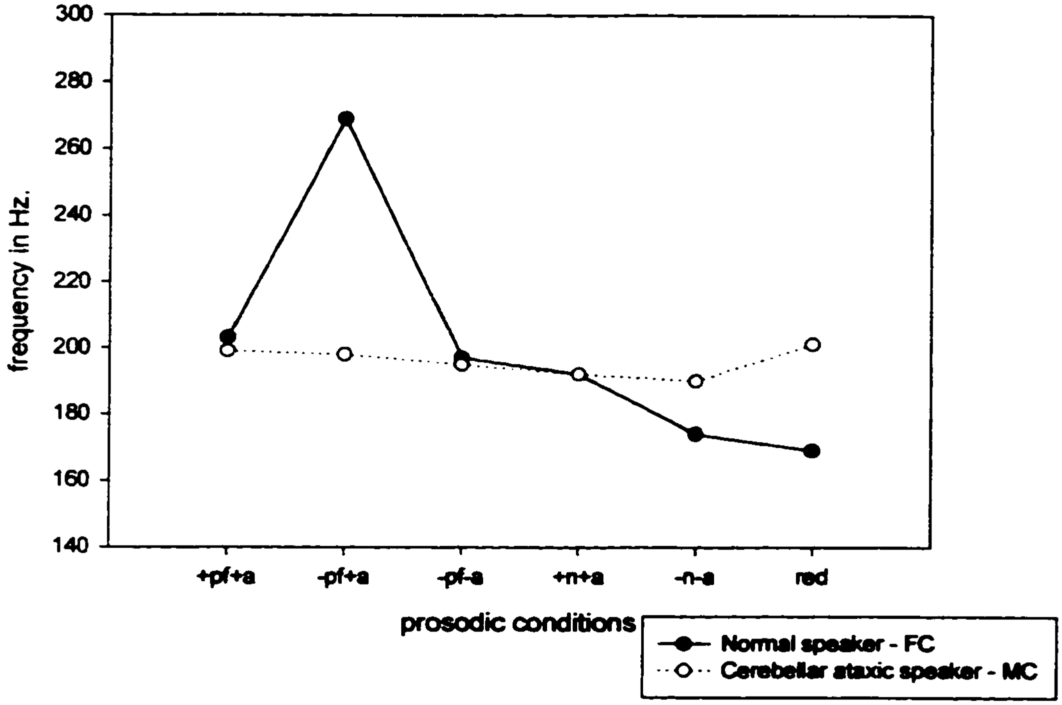


Fig.3.32: Mean f0 at mid-point for the six prosodic conditions for cerebellar ataxic speaker MC (D = 23.50 ; MRI = 1.5) and normal matched control FC.



3.3.1 The effects of cerebellar disturbance on f_0 for phrase-final lengthening and non-final lengthening of accented syllables: Contrast 1: +pf+a vs. –pf+a

The final analysis examined the simple interaction effects for the four prosodic contrasts on f_0 as a function of neurological status. This section examines the effect of the first contrast, phrase-final accented syllables versus non-phrase-final accented syllables on f_0.

The group statistical analysis of variance did not reveal a significant difference at the .05 level between the two groups for this contrast ($p = .288 > .05$). (See Table: 3.30).

Within-group analysis revealed a significant difference ($p = .036 < .05$) between the prosodic conditions of *+pf+a* and *–pf+a* for the normal speakers but not for the cerebellar group($p = .068 > .05$). Once again the production of this contrast appears to be a characteristic of normal speech motor dynamics that is not used by cerebellar speakers. When we examine vocal fold dynamics for the prosodic contrasts investigated here, we find that normal speakers have a tendency to use a lower f_0 in the phrase-final condition, whereas the cerebellar speakers are inconsistent in the direction of f_0 difference.

Contrast 1: *+pf+a vs. –pf+a*: f_0

Table 3.30a: Tests of Significance for Contrast 1: +pf+a vs. –pf+a : Unique Sum of Squares

Analysis of Variance:	Contrast 1: +pf+af_0 vs. -pf+af_0 : Cerebellar vs. Normal				
Source of Variation	SS	DF	MS	F	Sig. of F
Within Cells	310.92	5	62.18		
Constant	87.74	1	87.74	1.41	p=.288

Table 3.30b: Estimates for Contrast 1: +pf+a f_0 vs. -pf+a f_0: Cerebellar vs. Normal

Parameter	Coefficient	Std. Error	t-value	Sig. T	95% Level of Confidence	
					Lower	Upper
1	3.82407761	3.219	1.8786	.288	-4.45127	12.09942

3.3.2 The effects of cerebellar disturbance on f_0 for accented and un-accented syllables in non-final position: Contrast 2: -pf+a vs. –pf-a

Section 3.3.2 examines f_0 for the contrast of accented versus un-accented syllables; in non-final positions. Unlike the contrast of *+pf+a* vs. *–pf+a*, the position of the syllable in the sentence for *–pf+a* and *–pf-a* is the same while the phrase accent is different. The purpose, then, of this comparison is to consider the difference between accented and unaccented syllables as a function of neurological status. The analysis of variance did not reveal a significant difference between the two groups for this contrast ($p= .395>.05$). (See Table: 3.31 a,b). Neither were there significant differences between conditions for either group considered alone (Table 3.28). It is interesting to note that this is the only acoustical parameter for which the normal subjects did not show a significant difference for this contrast. An examination of Fig. 3.26 shows a small, although not statistically significant difference in f_0 for the normal speakers (with lower f_0 in the unaccented (*–a*)) condition. The cerebellar speakers do not show this difference.

A closer examination of the individual speaker data for f_0 shows that both cerebellar and normal speakers (Fig. 3.27 to Fig. 3.32) are inconsistent in their use of f_0 to mark this contrast. .

Contrast 2: -pf+a vs. –pf-a

Table 3.31a: Tests of **Significance for Contrast 2:-pf+a vs. –pf-a : Unique Sum of Squares**

Analysis of Variance:	**Contrast 2: -pf+a f_0 vs. -pf-a f_0: Cerebellar vs. Normal**				
Source of Variation	SS	DF	MS	F	Sig. of F
Within Cells	2418.92	5	483.78		
Constant	418.38	1	418.38	.86	p=.395

Table 3.31 b: **Estimates for Contrast 2: -pf+a f_0 vs. -pf-a f_0: Cerebellar vs. Normal**

Parameter	Coefficient	Std. Error	t-value	Sig. T	95% Level of Confidence	
					Lower	Upper
1	-8.3504639	8.97946	-.92995	.395	-31.43256	14.73163

3.3.3 The effects of cerebellar disturbance on f_0 for accented and un-accented syllables in nuclear and post-nuclear positions: Contrast 3: +n+a vs. –n-a

Section 3.3.3 examines the contrast, accented versus un-accented syllables, in nuclear and post-nuclear positions. The group statistical analysis of variance revealed a significant difference at the between the two groups for this contrast ($p=.009<.01$). See (Tables: 3.32 a,b).

When analyzed separately, the normal speakers showed a significant difference ($p = .002 < .01$) on this contrast whereas the cerebellar group did not (Table 3.28). Thus, it appears the production of this contrast again may be a characteristic of normal speech motor dynamics that is not shared by the cerebellar speakers. When we examine f_0 across these two conditions (Fig.3.27 through Fig. 3.32), we see that the normal speakers had a tendency to use a lower f_0 for the unaccented condition in post-nuclear position. The cerebellar speakers did not show a consistent difference between conditions for this contrast. Although two of the cerebellar speakers (AH, Fig. 3.27 and WD, Fig. 3.29) followed the f_0 pattern of their normal matched controls, the size of the differences between the f_0 values in this contrast is far less than those for the normal controls. Three cerebellar speakers (PP.Fig. 3.28; ES, Fig. 3.31 and MC, Fig. 3.32) did not mark this contrast using f_0, while one speaker (JS, Fig. 3.30) showed a reversal, with f_0 in the *–n-a* condition higher than the *+n+a* condition.

Contrast 3: +n+a vs. –n-a

Table 3.32a: Tests of Significance for Contrast 3: +n+a vs. –n-a : Unique Sum of Squares

Analysis of Variance:	**Contrast 3= +n+a f_0 – -n-a f_0: Cerebellar vs. Normal**				
Source of Variation	SS	DF	MS	F	Sig. of F
Within Cells	5909.08	5	1181.82		
Constant	20366.15	1	20366.15	17.23	p= .009**

Table 3.32b: Estimates for Contrast 3 : +n+a f_0 vs. -n-a f_0: Cerebellar vs. Normal

Parameter	Coefficient	Std. Error	t-value	Sig. T	95% Level of Confidence	
					Lower	Upper
1	-58.261116	14.03458	-4.15126	.009**	-94.33760	-22.18463

3.3.4 The effects of cerebellar disturbance on f_0 for un-accented post-nuclear and reduced syllables: Contrast 4: -n-a vs. red

Section 3.3.4 examines the fourth contrast, unaccented post-nuclear vs. reduced syllables. The analysis of variance did not reveal a significant difference between the two groups for this contrast.

When analyzed separately, the normal group showed a significant difference ($p = .045 < .05$) for this contrast, whereas the cerebellar group did not (Table 3.28). Once again it seems the production of this contrast may be a characteristic of normal speech motor dynamics but not of cerebellar motor dynamics. Normal speakers reduce f_0 for the *red* condition, whereas the cerebellar speakers use f_0 inconsistently to mark the contrast. Thus, two of the six cerebellar speakers (WD, Fig.3.29 and ES, Fig. 3.31) followed the f_0 pattern of their normal matched controls, three speakers (AH, Fig. 3.27; JS, Fig. 3.30 and MC, Fig. 3.32) reversed the direction for this contrast. In one case, a normal speaker(GC, Fig. 3.28) did not use f_0 to mark the contrast, while a cerebellar speaker (PP) used a lower f_0 for the less prominent *red* syllable.

Contrast 4: -n-a vs. red

Table 3.33a: Tests of Significance for Contrast 4:-n-a vs. red: Unique Sum of Squares

Analysis of Variance:	Contrast 4=: -n-a f_0– red f_0: Cerebellar vs. Normal				
Source of Variation	SS	DF	MS	F	Sig. of F
Within Cells	2798.17	5	559.63		
Constant	1040.17	1	1040.17	1.86	p = .231

Table 3.33b: Estimates for Contrast 4: -n-a f_0 vs. red f_0: Cerebellar vs. Normal

Parameter	Coefficient	Std. Error	t-value	Sig. T	95% Level of Confidence	
					Lower	Upper
1	-13.166658	9.65777	-1.36332	.231	-37.99236	11.65904

3.3.5 Discussion and conclusions: The effects of cerebellar degeneration on f_0

Of the six prosodic conditions investigated, the only condition to show a significant difference between the normal and cerebellar groups was the reduced syllable: *red* (Table 3.27b). While normal speakers produced significant differences (Table 3.28) between conditions for three of the four contrasts analyzed (*+pf+a* vs. *-pf+a, +n+a* vs. *-n-a and -n-a* vs. *red*), the cerebellar speakers did not produce significant differences or any of the four contrasts. The range of f_0 (Table 3.27a) for the normal speakers (82 Hz) was clearly greater than that for the cerebellar speakers (16 Hz). In addition, the cerebellar speakers produced the highest mean f_0 in an unaccented condition: *-pf-a* (185 Hz). While the normal speakers produced a systematic pattern for prosodically distinct syllables (Fig. 3.26), the cerebellar group did not.

Perhaps the most significant finding for the measure of f_0 was the difference found in normal speakers (Fig. 3.26, Table 3.28) for the contrast *+pf+a* vs. *-pf+a*. The normal speakers produced a significant difference in this contrast with the f_0 lower in the *+pf+a* than in the *-pf+a* condition. Although there was no significant difference between conditions for the cerebellar speakers for the contrast *+pf+a* vs. *-pf+a* (Table 3.28), the pattern for the production of this contrast for cerebellar speakers was similar to that of the normal speakers in that the cerebellar speakers as a group show a lower f_0 for the *+pf* condition (Fig. 3.26). The individual data show that four of the six cerebellar speakers (AH, Fig. 3.27; WD, Fig. 3.29; JS, Fig. 3.30; and ES, Fig. 3.31) use a lower the f_0 in *+pf* for the contrast *+pf+a* vs. *–pf+a* while two speakers (PP, Fig. 3.28 and MC, Fig. 3.32) showed little difference in f_0 between these conditions.

3.4 Correlations between cerebellar degeneration and speech disturbance

Three views of anatomical MRIs (midsagittal, coronal and transverse) were used to estimate the degree of cerebellar pathology. See (Figures 2.1 through 2.18). Table 2.1 presents the radiologist's ratings assigned to several selected structures (cerebellar vermis , cerebellar hemisphere, brainstem and spinal cord) to indicate the degree of neurological involvement for each of the six cerebellar ataxic subjects.

A rating from 0 (no involvement) to 3 (greatest involvement) was used to estimate the degree of cerebellar pathology for the vermis and cerebellar hemispheres. An average was then taken of the ratings of these two areas to form a rating level for each of the six cerebellar ataxic subjects. The brainstem and spinal cord were not included in this average but given a rating for of (+) for involvement and (-) for no involvement.

The cerebellar subjects were also analyzed for a measure of speech deterioration. As an individual measure of speech deterioration, each cerebellar subject was compared to his/her normal matched control for a combined value of all four prosodic contrasts: The "D" score.

In addition, a sub-analysis calculated the degree of difference between each subject and his/her matched control ("d" score) for each of the four prosodic contrasts. See (Chapter 2, pp. 73-74). The following section presents the post-hoc analyses for correlations between speech disturbance and cerebellar degeneration. using the "D" and "d" scores as measures.

Table 3.34 presents the D scores, MRI ratings and Pearson-correlations between those measures for each of the six cerebellar ataxic speakers. Figures 3.33 through 3.36 graphically represent these correlations.

Table 3.39 presents the raw "d" score values for each of the four prosodic contrasts (*+pf+a* vs. *–pf+a*, *-pf+a* vs. *-pf-a*, *+n+a* vs. *–n-a*, *-n-a* vs. *red*) for syllable duration, F2, F1 and f_0 in the six cerebellar ataxic speakers. These values are graphically represented with each icerebellar speaker and the matched control in Figures 3.3 through 3.8(for syllable duration); Figures 3.11 through 3.16 (for F2); Figures 3.19 through 3.24 (for F1) and Figures 3.27 through 3.32 (for f_0). A correlation analysis for the four prosodic contrasts in each of the four acoustic measures is shown in Tables 3.35 through 3.38 and Figures 3.37 through 3.40.

3.4.1 Correlations between measures of syllable duration and cerebellar degeneration

Table 3.34 and Fig. 3.33 show that, a moderate positive correlation ($r = .444$) between speech disturbance and cerebellar degeneration was found for the measure of syllable duration.

An examination of the data (Table 3.34 and Fig. 3.33) showed that the three subjects with the more severe speech deterioration are the subjects with cerebellar degeneration of etiology other than Friedreich's ataxia. The subjects WD (D = 488), AH (D=455) and PP (D= 268) had more deterioration as measured in syllable duration than the three subjects with Friedreich's ataxia JS (D=206), ES(D = 86) and MC (D = 98). Thus, the pattern among the cerebellar subjects differed depending on the relative involvement of the cerebellum and the etiology of cerebellar degeneration.

The three subjects with Friedreich's ataxia, a neuro-degenerative condition with primary spinal cord involvement (JS, Fig. 3.6; ES, Fig. 3.7 and MC, Fig. 3.8) produced their shortest durations on the reduced syllable, whereas the three subjects with primary cerebellar involvement (AH,Fig. 3.3; PP, Fig. 3.4; and WD, Fig. 3.5) had longer durations for the reduced syllable than any other condition except the *+pf+a* syllable. All six subjects showed a neutralization for phrase accent as seen in the contrasts *–pf+a* vs. *–pf-a* and *+n+a* vs. *–n-a* contrasts. One subject with Friedreich's ataxia did show a longer duration for the unaccented condition, but in the contrast *+n+a* vs. *–n-a.*

For the contrast analysis "d " scores (Table 3.39), strong negative correlations for syllable duration were found for two of the four contrasts: *-pf+a* vs. *–pf-a* (r = -.834); (p=.039<.05) and –n-a vs. red (r = -.755). See (Table 3.34 and Fig. 3.37).

These negative values were not derived from the "D" statistic but from a raw score difference measure ("d"). The distance criteria used for the D statistic were modified to a simple difference measure "d" and negative correlations in these cases represent significant speech deterioration, in the speaker's neutralization and/or reversal for the direction of the given prosodic contrast, with a strong correlation to degree of cerebellar atrophy (See Equation #2).

3.4.2 Correlations between measures of F2 and cerebellar degeneration

Table 3.34 shows a mild negative correlation (r = -.285) for the measure of F2 was found between speech disturbance and cerebellar degeneration. Three of the six cerebellar subjects with Friedreich's ataxia (JS, Fig. 3.14; D =621.52; MRI =2.0), (ES:Fig. 3.15; D=274.48: MRI=2.0), (MC: Fig. 3.16; D=245.78; MRI=1.5) showed more deterioration in the measure of F2 than the three subjects with cerebellar degeneration of other etiologies (AH: Fig. 3.11; D=132.72; MRI = 2.0), (PP: Fig. 3.12; D=177.59; MRI =3.0), (WD: Fig. 3.13; D=162.60; MRI = 2.5).

Table 3.36 and Fig. 3.38 show the "d" score correlations for each of the four prosodic contrasts. Two contrasts *+n+a* vs. *–n-a* and *–n-a* vs. *red* show relatively stronger relationship between speech deterioration and cerebellar pathology than the other two contrasts. When we examine the individual scores for each contrast in Table 3.39 we see that the three speakers with Friedreich's ataxia show more severe speech deterioration on this measure.

3.4.3 Correlation between measures of F1 and cerebellar degeneration

Table 3.34 and Fig. 3.35 show a strong negative correlation for the measure of F1 frequency (r= -.630) between speech disturbance and cerebellar degeneration. As with F2, but to a greater degree, the three subjects with Friedreich's ataxia, (JS, Fig.3.22; D=373.98; MRI =2.0), (ES, Fig.3.23; D=296.67; MRI = 2.0), (MC: Fig. 3.24; D=228.81; MRI = 1.5) showed greater speech deterioration in the measure of F1 than the three subjects with cerebellar degenerations of other etiologies (AH, Fig. 3.19; D=188.05; MRI:2.0), (PP, Fig. 3.20; D= 91.32; MRI = 3.0) and (WD, Fig. 3.21; D=176.10; MRI= 2.5).

The difference measure for each individual contrast showed a strong positive correlation (r= .615) in syllable reduction: *-n-a* vs. *red* (Table 3.37 and Fig. 3.39). Given the raw score value for this measure the interpretation of this correlation is reversed and so directs a view of more severe disturbance in the subjects with relatively less cerebellar degeneration.

The F1 frequencies for this contrast can be seen in Figures 3.19 through 3.24 and Table 3.39 shows the individual "d" scores for this contrast (AH, Fig. 3.19: d= -140.68; MRI:2.0), (PP, Fig. 3.20; d=5.40; MRI = 3.0), (WD, Fig.3.21; d=-168.50, MRI = 2.5), (JS, Fig.3.22; d= -263.84; MRI = 2.0), (E,:Fig. 3.23; d= -33.80; MRI = 2.0) and (MC, Fig. 3.24; d=-70.20; MRI = 1.5).

3.4.4 Correlation between measures of f_0 and cerebellar degeneration

Table 3.34 and Fig. 3.36 show a moderate positive correlation (r= .446) for the measure of f_0, between speech disturbance and cerebellar degeneration. The three subjects with cerebellar degeneration of etiology other than Friedreich's (AH, Fig.3.27; D=90.54; MRI = 2.0), (PP, Fig. 3.28; D=87.85; MRI=3.0), (WD, Fig.3.29; D=54.60; MRI =54.60) showed more speech deterioration for the measure of f_0 than the Friedreich's ataxic subjects (JS, Fig.3.30; D=102.40; MRI = 2.0), (ES, Fig. 3.31; D= 58.45; MRI = 2.0) and (MC, Fig.3.32; D= 23.50; MRI = 1.5).

An examination of "d" scores (Table 3.38 and Fig. 3.40) showed two moderate positive correlations: (*+pf+a* vs. *–pf+a*: r = .588), (*-n-a* vs. *red*: r = .524) and one moderate negative correlation (*-pf+a* vs. *–pf-a*: r = -.444).

3.4.5 Discussion and conclusions: Correlations between cerebellar degeneration and speech disturbance

Of the four acoustic measures investigated, syllable duration and fundamental frequency showed moderate positive correlations between speech disturbance and cerebellar degeneration using the distance criterion ("D" score: See pp. 73-74). F1 showed a strong negative correlation and F2 a mild negative correlation between cerebellar degeneration and speech disturbance.

In this investigation, a post-hoc analysis found that the six cerebellar subjects divided into two sub-groups of cerebellar pathology: those with Friedreich's ataxia and those with cerebellar degeneration of other etiologies (pure recessive cerebellar , olivo-ponto-cerebellar atrophy and unknown cerebellar degeneration). The degree of cerebellar degeneration tended to be more severe in the three subjects of other etiologies (AH: MRI:2.0, PP: MRI:3.0 and WD:MRI: 2.5). And so it is interesting that acoustic measures of syllable duration and f_0 showed a positive correlation to cerebellar degeneration while F1 and F2 showed a negative correlation. Although these findings were not statistically significant, given the small number of subjects, it is possible that these acoustic measures may be useful in describing differences among subjects with cerebellar pathology.

Each of the four contrasts was also analyzed separately ("d" score) for correlations between cerebellar degeneration and speech disturbance. A strong positive correlation (pp. 73-74) between cerebellar degeneration and speech disurbance in the measure of syllable duration was found in *–pf+a* vs. *–pf-a* (r= -.834*) (p=.039<.05*) and *–n-a* vs. *red* (r= -.755). In the measure of F1 a strong negative correlation for the contrast *–n-a* vs. *red* (r=.615) was found. Fundamental frequency showed both positive and negative moderate correlations in three of the four contrasts investigated (*+pf+a* vs. *–pf+a* (r = .588), *-pf+a* vs. *–pf-a* (r=-.444) and *–n-a* vs. red(r= .524).

Table 3.34: Average MRI rating of cerebellar involvement as compared to individual subject D score metric with with the Pearson rank correlation "r" to MRI rating of cerebellar atrophy.for each of the four acoustic measures: duration, F2, F1 and f_0.

Cerebellar subject	Cerebellar syndrome	MRI ratings	Euclidean Distance Metric : D scores			
			Duration	F2	F1	f_0
AH	pure recessive	2.0	454.69	132.72	188.05	90.54
PP	unknown	3.0	267.90	177.59	91.32	87.85
WD	OPCA	2.5	487.81	162.60	176.10	54.60
JS	Friedreich's	2.0	205.52	621.52	373.98	102.40
ES	Friedreich's	2.0	85.62	274.48	296.67	58.45
MC	Friedreich's	1.5	98.38	245.78	228.81	23.50
Pearson correlation			r= .440	r = -.285	r= -.630	r=.446

Fig.3.33: Pearson Correlation (r=.440) between the average MRI rating for cerebellar involvement vs. the D statistic as a measure of speech deterioration for each of the six cerebellar ataxic speakers for the measure of syllable duration.

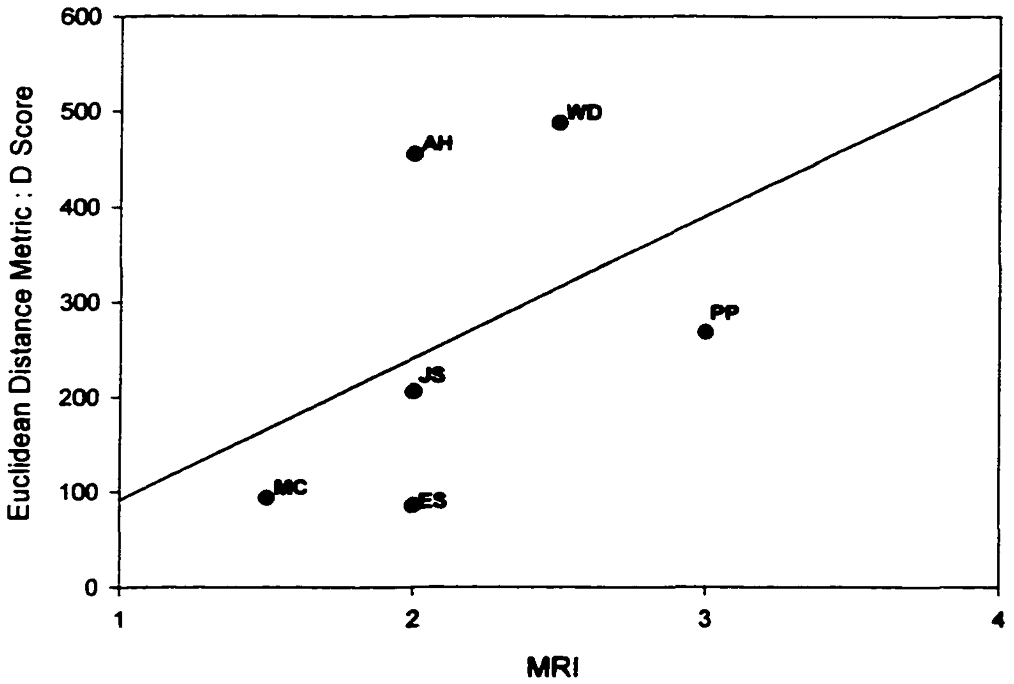


Fig. 3.34: Pearson Correlation (r=-.285) between the average MRI rating for cerebellar involvement vs. the D statistic as a measure of speech deterioration for each of the six cerebellar ataxic speakers for the measure of F2.

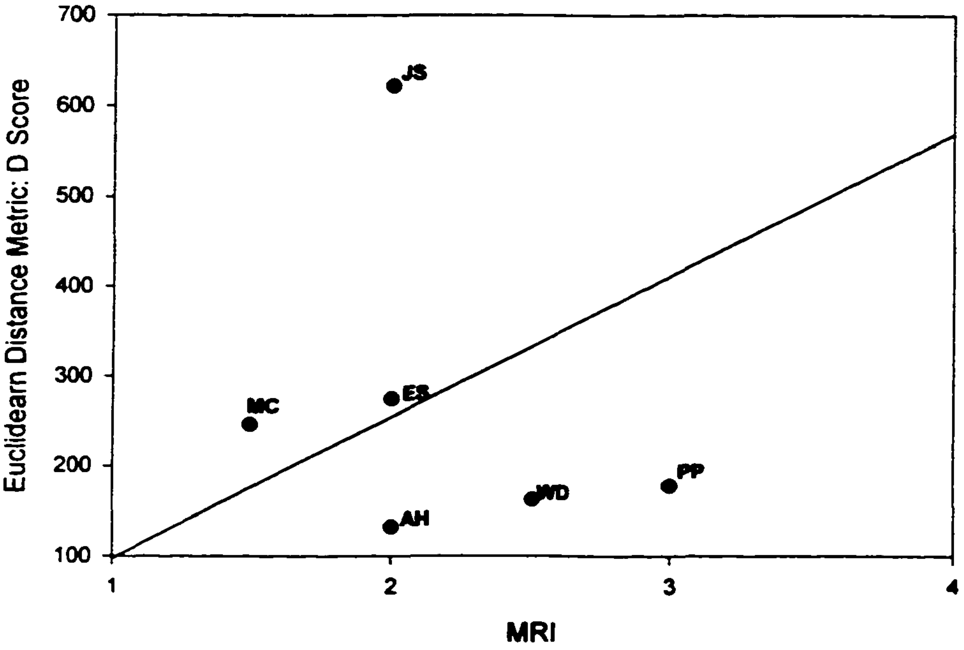


Fig. 3.35: **Pearson Correlation (r=-.630) between the average MRI rating for cerebellar involvement vs. the D statistic as a measure of speech deterioration for each of the six cerebellar ataxic speakers for the measure F1.**

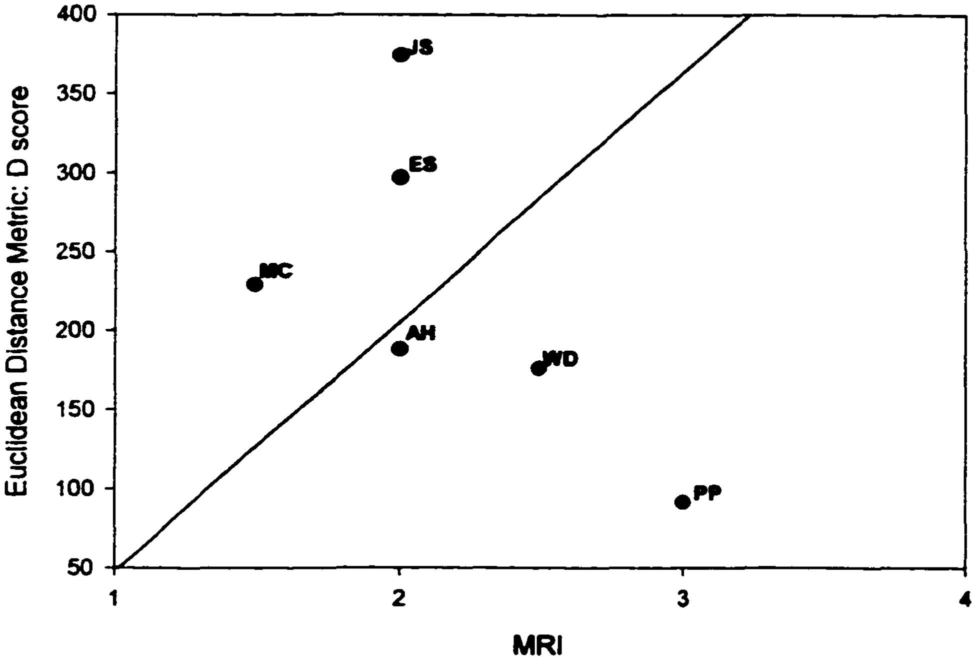


Fig. 3.36: **Pearson Correlation (r=.446) between the average MRI rating for cerebellar involvement vs. the D statistic as a measure of speech deterioration for each of the six cerebellar ataxic speakers for the measure of f_0.**

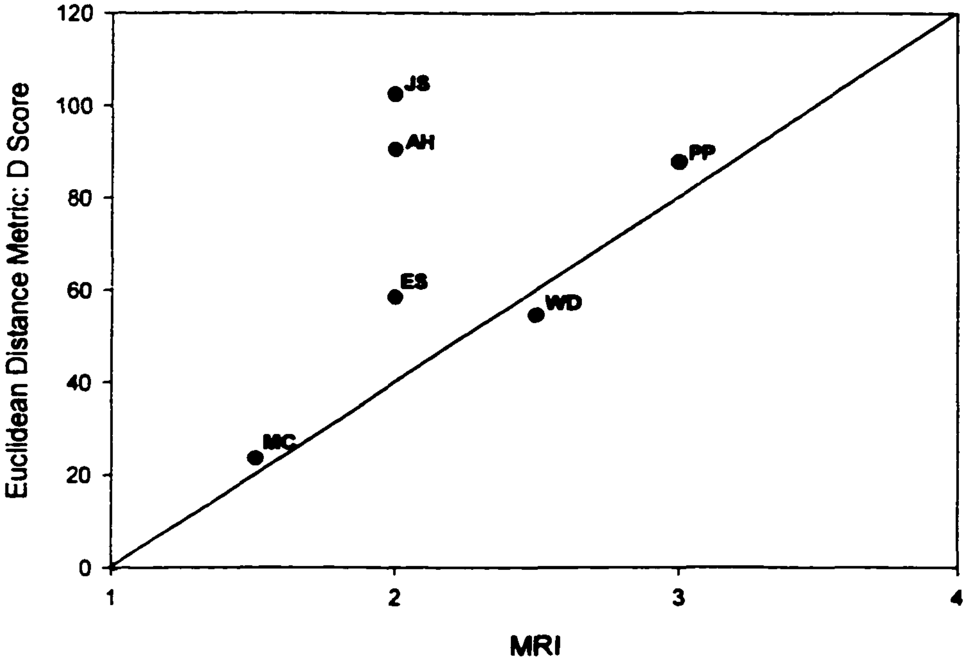


Table 3.35: Correlations between the average MRI rating of cerebellar degeneration and speech disturbance in syllable duration for each of the four contrasts "d".

Prosodic Contrasts	Correlations d score vs. MRI	
+pf+a : -pf+a	Pearson Correlation	.128
	Sig. (2-tailed)	.810
-pf+a: -pf-a	Pearson Correlation	-.834
	Sig. (2-tailed)	.039*
+n+a:-n-a	Pearson Correlation	.314
	Sig. (2-tailed)	.544
-n-a: red	Pearson Correlation	-.755
	Sig. (2-tailed)	.083

Fig. 3.37: Pearson correlation of average MRI rating and speech disturbance "d" across each of the four prosodic contrasts for the acoustic measure of syllable duration. * =p<.05.

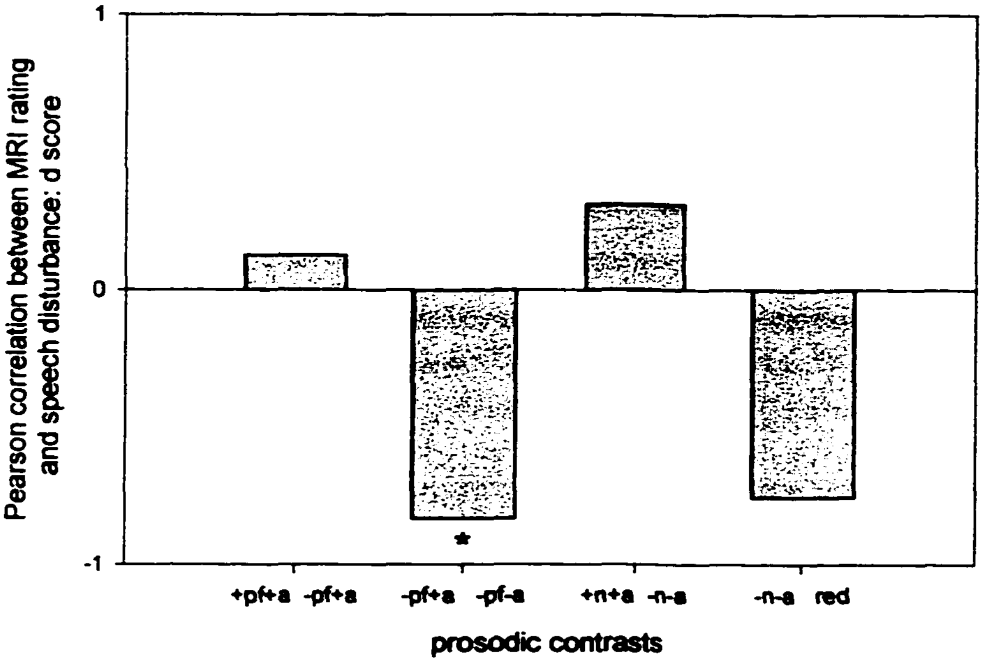


Table 3.36: Correlations between the average MRI rating of cerebellar degeneration and speech disturbance in F2 for each of the four contrasts “d”.

Prosodic Contrasts	Correlations d score vs. MRI	
+pf+a : -pf+a	Pearson Correlation	-.340
	Sig. (2-tailed)	.510
-pf+a: -pf-a	Pearson Correlation	-.212
	Sig. (2-tailed)	.887
+n+a:-n-a	Pearson Correlation	.358
	Sig. (2-tailed)	.486
-n-a: red	Pearson Correlation	.391
	Sig. (2-tailed)	.443

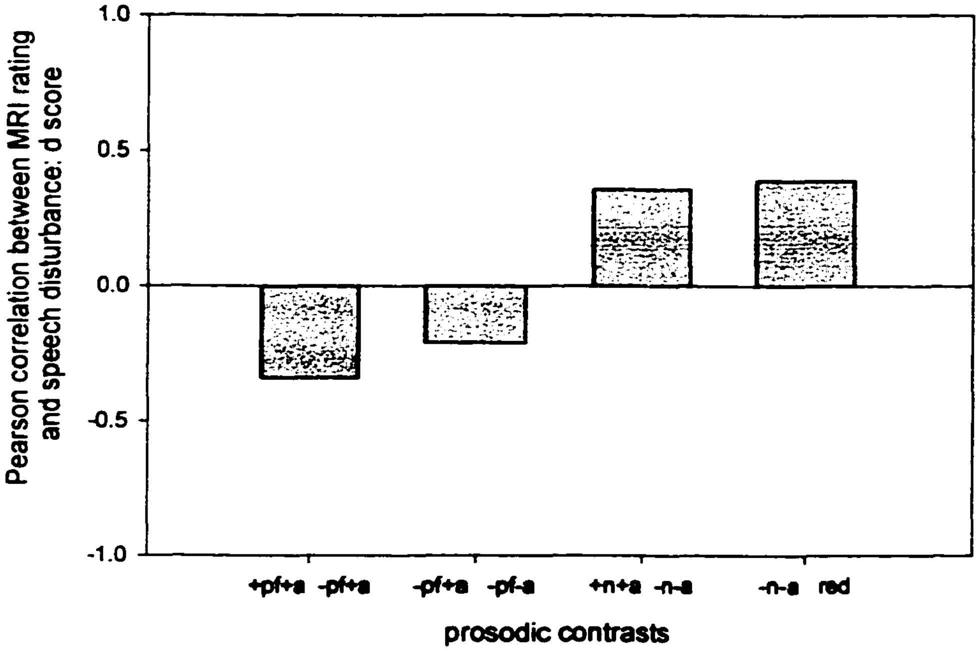


Fig. 3.38: Pearson correlation of average MRI rating and speech disturbance "d" across each of the four prosodic contrasts for the acoustic measure of F2.

Table 3.37: **Correlations between the average MRI rating of cerebellar degeneration and speech disturbance in F1 each of the four contrasts "d".**

Prosodic Contrasts	Correlations d score vs. MRI	
+pf+a : -pf+a	Pearson Correlation	-.119
	Sig. (2-tailed)	.823
-pf+a: -pf-a	Pearson Correlation	.002
	Sig. (2-tailed)	.996
+n+a:-n-a	Pearson Correlation	.337
	Sig. (2-tailed)	.514
-n-a: red	Pearson Correlation	**.615**
	Sig. (2-tailed)	.193

Fig. 3.39: Pearson correlation of average MRI rating and speech disturbance "d" across each of the four prosodic contrasts for the acoustic measure of F1.

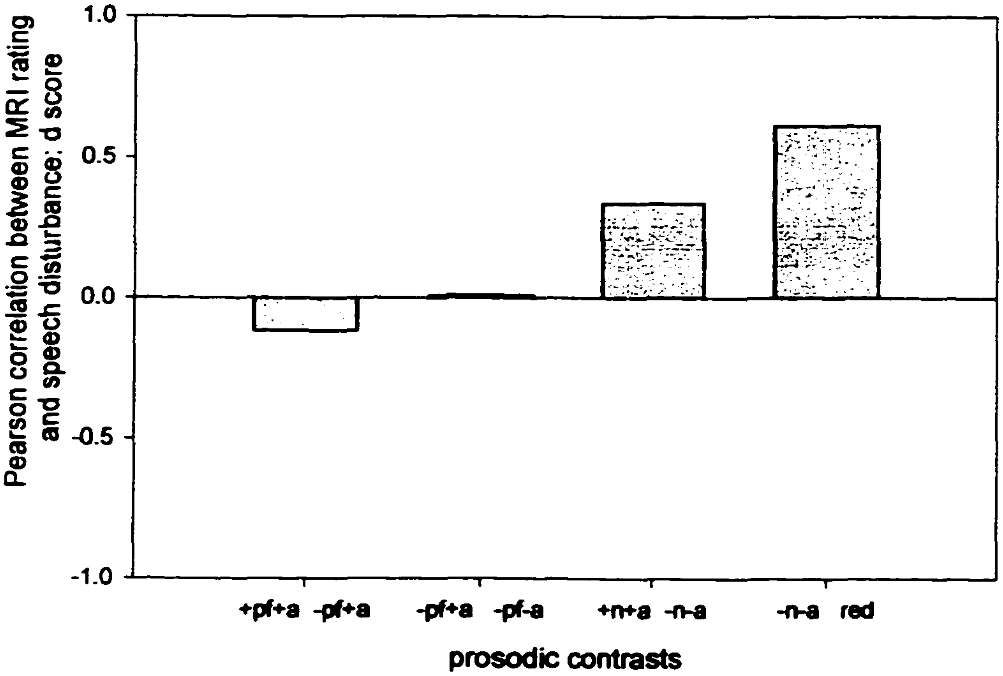


Table 3.38: **Correlations between the average MRI rating of cerebellar degeneration and speech disturbance for f_0 each of the four contrasts "d".**

Prosodic Contrasts	**Correlations d score vs. MRI**	
+pf+a : -pf+a	Pearson Correlation	**.588**
	Sig. (2-tailed)	.219
-pf+a: -pf-a	Pearson Correlation	**-.444**
	Sig. (2-tailed)	.378
+n+a:-n-a	Pearson Correlation	-.322
	Sig. (2-tailed)	.534
-n-a: red	Pearson Correlation	**.524**
	Sig. (2-tailed)	.286

Fig. 3.40: Pearson correlation of average MRI rating and speech disturbance "d" across each of the four prosodic contrasts for the acoustic measure of f_0.

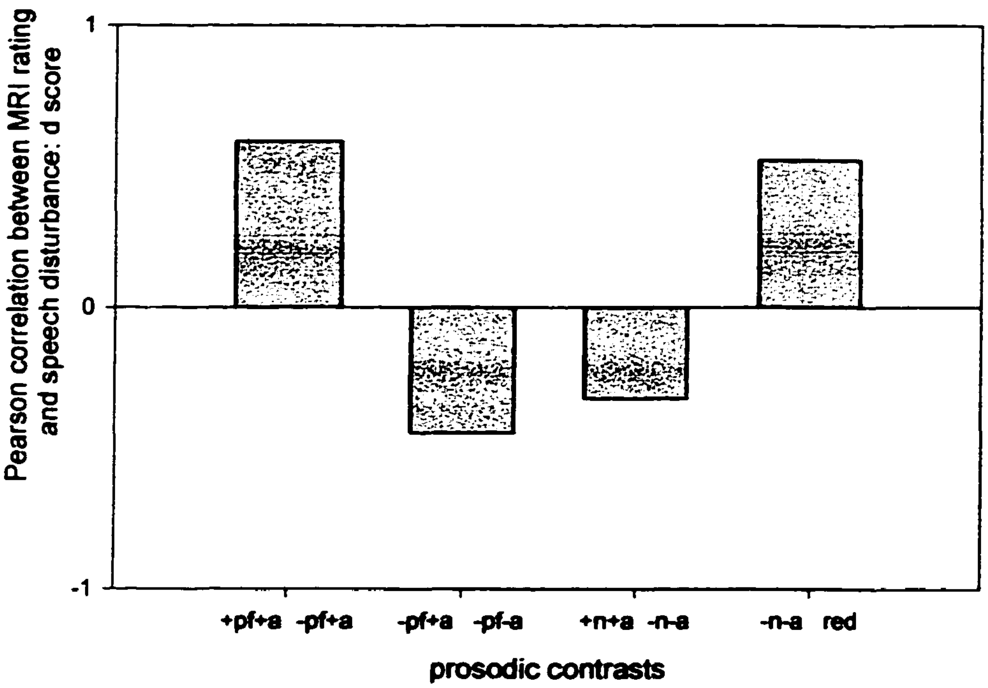


Table 3.39: The individual differences "d" between contrast values for the cerebellar ataxic speaker and their normal matched controls as an estimate of speech disturbance across the four acoustic measures for each of the four prosodic contrasts with Pearson correlations "r" of MRI rating with each of the four contrasts across the four acoustic measures.

			d scores															
Cerebellar Subject	Cerebellar syndrome	MRI	Duration				F2				F1				F0			
			+pf +a -pf+a	-pf+a -pf-a	+n+a –n-a	-n-a red	+pf +a -pf+a	-pf+a -pf-a	+n+a -n-a	-n-a red	+pf +a -pf+a	-pf+a -pf-a	+n+a –n-a	-n-a red	+pf +a -pf+a	-pf+a -pf-a	+n+a –n-a	-n-a red
AH	pure recessive	2.0	444.44	7.06	-9.04	-95.28	28.68	-65.08	-62.62	-93.52	28.97	-82.97	-88.97	-140.68	1.19	7.01	-74.63	-50.76
PP	unknown	3.0	132.20	-32.70	-29.60	-228.80	-18.90	-133.20	-72.70	-90.30	53.80	-53.90	-50.10	5.40	13.80	-8.00	-84.70	17.02
WD	OPCA	2.5	311.30	-6.20	24.80	-374.70	-98.00	-12.35	25.60	-126.60	5.50	-22.15	45.80	-168.50	-.40	-49.03	-20.20	13.00
JS	Friedreich's	2.0	197.30	-9.97	-8.00	-56.12	385.30	-36.70	-307.00	-377.14	244.20	-92.88	-44.58	-263.84	-.90	11.43	-99.33	-22.07
ES	Friedreich's	2.0	18.52	-15.56	-50.20	-65.00	164.56	-146.29	-141.20	-83.20	149.67	-43.86	-250.10	-33.80	13.66	-13.62	-54.80	6.40
MC	Friedreich's	1.5	77.80	36.10	-40.90	-25.50	10.70	-67.20	-104.70	-211.70	8.30	-23.90	-70.20	-216.30	-4.40	2.10	-15.90	-16.60
Pearson Correlation r			.128	**-.834**	.314	**-.755**	-.340	-.212	.358	.391	-.119	.002	.337	**.615**	**.588**	**-.444**	-.322	**.524**

4.1 THE EFFECTS OF CEREBELLAR DEGENERATION ON SPEECH PROSODY

4.1.1 The production of prosodic prominence as a dynamic response and hallmark speech motor control:

Measures of the acoustic parameters that signal prosodic differences, provide a means of describing behavioral adaptation. This adaptation, provides a quantitative description of the relative integrity of the speech motor system in its response to various conditions of linguistic stress. The present study examined systematic prosodic changes across conditions of varying patterns of prominence, and showed that the acoustic parameters that play the more prominent role in signaling a particular prosodic contrast vary between normal and cerebellar speakers. In other words the cerebellar speakers adapted differently to the demands of stress placement than did the normals. In order to better describe such adaptation, it is necessary to begin with a discussion of the normal speakers studied.

In normal prosody, a flexible response occurs, and this response is represented in systematically varying degrees of prominence. The acoustic measurement of this prominence, as found in the present study, suggests that both the context of the target syllable as well as the context of the surrounding syllables: (phrase and/or sentence) lead to a systematic variability in acoustic parameters across the normal speakers. This systematic variability is recognized statistically within the pair-wise comparisons of prosodic contrasts produced by the normal speakers and further supported in part by the sub-analysis of the normal group (Appendix A).

4.1.2 Prosodic prominence as a representation of qualitative invariance co-occurring with quantitative changes across acoustic parameters of syllable duration, F2, F1 and f_0

Inherent in the concept of dynamic principles of coordination is the prediction for dissociation between time and movement amplitude. The present study investigated syllable duration, formant frequencies and fundamental frequency. For the measure of syllable duration, all four contrasts showed a significant difference in the pair-wise comparisons for normal speakers (Table 4.1). For each of the frequency measures, F1 and F2 and f_0, three of the four contrasts showed significant differences; although, those contrasts yielding a significant difference varied across measures. Both F1 and F2 frequencies showed significant differences for *-pf+a* vs. *–pf-a; +n+a* vs. *–n-a* and *–n-a* vs. *red*; whereas, f_0 showed significanct differences for *+pf+a* vs. *–pf+a, +n+a* vs. *–n-a and –n-a* vs. *red*. It is interesting to note that F1 and F2 yielded significant differences for all contrasts except the phrase-final lengthening contrast (*+pf+a* vs. *–pf+a*), while f_0 yielded significanct differences for all contrasts except phrase accent (*-pf+a* vs. *–pf-a*). These findings were then tested by a subsequent analyses within the normal group (Appendix A).

In those analyses, two groups were generated for the sub-analysis of normals: one was the speaker LJR, and the other comprised the remaining five normal speakers. Thus LJR assumed the variability of the group, although he remained a single subject. Although there are considerable limits to such a design, the purpose was to explore further the suggestion of patterns within the normal group. Support for the initial findings, within the pair-wise comparisons was found only for the measures of F1 and f_0. Both LJR and the group showed significant differences for *+n+a* vs. *–n-a* and *–n-a* vs. *red* for F1, and *+n+a* vs. *–n-a* for f_0.

The main effects were also explored in the subsequent analysis and significant differences for the prosodic effect were found for each of the acoustic measures: syllable duration, F1, F2 and f_0, while the group effect remained insignificant. (See Appendix A) . Combined, these analyses support a suggestion of prosodic invariance in normal speakers that is best measured for the production of [ɑ] by F1 and f_0 and most sensitive to nuclear accent (*+n+a* vs. *–n-a*) and syllable reduction (*-n-a* vs. *red*). Based on these findings, phrase accent contrasts (*-pf+a* vs. *–pf-a*) and final lengthening contrasts (*+pf+a* vs. *–pf+a*) did not generate clear differences in the acoustic measures.

Thus, the patterns of dissociations between formant extremum and syllable duration predicted for the normal speakers and found in the pair-wise comparisons of the first study for *+pf+a* vs. *-pf+a* were not found in the second sub-analysis and so their usefulness as an acoustic measure for individual subjects is doubtful.

Previous kinematic studies of the effects of final lengthening in normals

(Cohen et al., 1995) showed the measure of velocity to be the descriptor of different underlying dynamics, so that all lengthenings are not the same. For the analysis of normals presented in the pair-wise comparisons (Table 3.28), a significant difference ($p=.036<.05$) for f_0 was found for the*–pf+a* vs. *+pf+a* contrast. Furthermore, both the values and graphic representation of this difference show higher F2 and F1 frequencies for the prosodic condition *–pf+a* then *+pf+a*. Although the contrast *+pf+a* vs. *–pf+a* did not further yield significance for F1 and F2, an examination of results on a speaker-by-speaker basis shows that some speakers produce more extreme formant values with relatively shorter durations. In addition, the analyses of normal speakers also suggest that phrase accent (*-pf+a* vs. *–pf-a*) and nuclear accent (*+n+a* vs. *-n-a*) are realized differently. Whereas phrase accent is shown by significant differences between conditions for F1 and F2, nuclear accent is produced with a significant difference between conditions for f_0.

Table 4.1: Significance of pair-wise comparisons for normal group. * p<. 05 level, ** p<.01 level and ***p< .001 level. The direction of the contrast is marked from longer to shorter > and shorter to longer < for duration and higher to lower > and lower to higher < for frequency (Tables 3.4, 3.12, 3.16 and 3.28).

Prosodic Contrast	**Duration**		**F2**		**F1**		**F_0**	
+pf+a vs. –pf+a	*	>		<		<	*	<
-pf+a vs. –pf-a	*	>	*	>	*	>		>
+n+a vs. –n-a	**	>	*	>	**	>	**	>
-n-a vs. red	**	>	**	>	***	>	*	>

4.1.3 Prosodic conditions and contrasts and the effects of cerebellar pathology

The six prosodic conditions investigated here were examined in relation to the four acoustic parameters of syllable duration, F2 and F1 and f_0. Although the measure of syllable duration (Table 3.1) yielded significant differences for each of the six prosodic conditions across groups, the reduced syllable condition was found to be the sole condition to yield significant differences for the remaining three measures: F2, F1 and f_0 (Tables 3.9, 3.17 and 3.25).

When examined for significance between groups, the four prosodic contrasts yielded several patterns across the acoustic measures reviewed here (Table 4.3): First, whereas the *+pf+a* vs. *–pf+a* contrast was found significantly diferent for syllable duration, the *-pf+a* vs. *–pf-a* contrast was significantly different for F2 and F1 frequencies. The *-n-a* vs. *red* contrast was significantly different for both syllable duration and F2 and F1 frequencies. Finally, *+n+a* vs. *–n-a*, the one contrast not significantly different for either syllable duration or F2 and F1 frequencies was significantly different in f_0.

These differences between groups lead to a view consistent with those previous studies that found syllable reduction and phrase final lengthening dynamics compromised in cerebellar ataxic speakers (Kent, 1979; Bell-Berti et al., 1991). However, the results for F1 and F2 are not as previously reported (Kent, 1979) within the normal limits. The data presented here show that unlike normal speakers, cerebellar speakers do not systematically reduce F1 and F2 frequencies for unaccented conditions within a prosodic contrast. In addition, the F1 and F2 frequency values were reversed in direction from those of the normal speakers (Table 4.2) for the *+pf+a* vs. *–pf+a* contrast and for F1 frequency in the *+n+a* vs. *–n-a* contrast.

The cerebellum has long been thought of as the coordinator of movement within the central nervous system. This study investigated cerebellar function with a sensitive behavioral analysis. The production of prosodically normal speech requires rapid adaptability within an utterance. The acoustic measures used to quantify this normal adaptation clearly show that cerebellar speakers, although intelligible, demonstrate a deterioration in speech that is acoustically measurable. Cerebellar speech is markedly slow and fails in the rapid adaptation to reduce frequency and shorten length for syllables that are produced with less prominence in the normal utterance.

Table 4.2: Significance of pair-wise comparisons for cerebellar group. * p< .05 level, **p<.01 level . The direction of the contrast is marked from longer to shorter > and shorter to longer < for duration and higher to lower > and lower to higher < for frequency (Tables 3.4, 3.12, 3.16 and 3.28). # represents a reversal in direction from the normal direction (Table 4.1).

Prosodic Contrast	**Duration**		**F2**		**F1**		**F0**	
+pf+a vs. –pf+a	*	>		> #		> #		<
-pf+a vs. –pf-a		>		>		>		<#
+n+a vs. –n-a		>		>		< #		>
-n-a vs. red		< #		>	**	>		<#

Table 4.3: Cerebellar group vs. normal group. *p<.05 level ; **p<.01 level.

Prosodic Contrast	**Duration**	**F2**	**F1**	**F0**
+pf+a vs. –pf+a	*			
-pf+a vs. –pf-a		*	**	
+n+a vs. –n-a				**
-n-a vs. red	*	*	*	

Appendix A

Table A1: Within-Normal Subjects Effects: Duration

Source	Type III Sum of Squares	Df	Mean Square	F	Sig.
Prosodic condition	59527.372	5	11905.474	10.295	.000***
Pros*Normals	550.434	5	110.087	.095	.992

Table A2: Within-Normal Subjects Contrasts: Duration

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Prosodic condition	47372.489	1	47372.489	49.187	.002**
Pros*Normals	275.104	1	275.104	.286	.621

Table A3: Within-Normal Subjects Effects: F2

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Prosodic condition	107951.485	5	21590.297	3.207	.028*
Pros*Normals	4142.767	5	828.553	.123	.986

Table A4: Within-Normal Subjects Contrasts: F2

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Prosodic condition	36714.977	1	36714.977	9.856	.035*
Pros*Normals	47.830	1	47.830	.013	.915

Table A5: Within-Normal Subjects Effects: F1

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Prosodic condition	321605.659	5	64321.132	46.470	.000***
Pros*Normals	8184.217	5	1636.843	1.183	.353

Table A6: Within-Normal Subjects Contrasts: F1

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Prosodic condition	200851.099	1	200851.099	148.616	.000***
Pros*Normals	5184.285	1	5184.285	3.836	.122

Table A7: Within-Normal Subjects Effects: f_0

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Prosodic condition	24399.777	5	4879.955	11.110	.000***
Pros*Normals	721.388	5	144.278	.328	.890

Table A8: Within-Normal Subjects Contrasts: f_0

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Prosodic condition	13600.296	1	13600.296	16.687	.015*
Pros*Normals	504.000	1	504.000	.618	.476

Table A9: **Pairwise Comparisons**
Acoustic **Measure: Duration**

Normals	Prosodic Contrast	Mean Difference	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
LJR	+pf+a vs. –pf-a	84.200	85.111	.379	-152.107	320.507
	-pf+a vs. –pf-a	18.600	20.981	.425	-39.652	76.852
	+n+a vs. –n-a	18.600	14.920	.281	-22.825	60.025
	-n-a vs. red	55.500	37.485	.213	-48.576	159.576

Normals	Prosodic Contrast	Mean Difference	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
Normals	+pf+a vs. –pf-a	94.893	38.063	.067	-10.788	200.573
	-pf+a vs. –pf-a	31.651*	9.383	.028*	5.600	57.702
	+n+a vs. –n-a	25.440*	6.673	.019*	6.914	43.966
	-n-a vs. red	73.200*	16.764	.012*	26.656	119.744

Based on the estimated marginal means

* The mean difference is significant at the .050 level.

[a] Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

Table A10: **Pairwise Comparisons**
Acoustic Measure: F2

Normals	Prosodic Contrast	Mean Difference	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
LJR	+pf+a vs. –pf-a	-18.300	131.721	.896	-384.016	347.416
	-pf+a vs. –pf-a	28.400	82.625	.748	-201.004	257.804
	+n+a vs. –n-a	51.800	55.996	.407	-103.671	207.271
	-n-a vs. red	162.300	85.260	.130	-74.420	399.020

Normals	Prosodic Contrast	Mean Difference	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
Normals	+pf+a vs. –pf-a	-58.771	58.907	.308	-232.324	94.782
	-pf+a vs. –pf-a	103.108*	36.951	.049*	.515	205.700
	+n+a vs. –n-a	75.860*	25.042	.039*	6.631	145.389
	-n-a vs. red	138.460*	38.130	.022*	32.595	244.325

Based on the estimated marginal means

*. The mean difference is significant at the .050 level.

[a] Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

Table A11: **Pairwise Comparisons**
Between-"Subjects" Effects – Normals & Prosodic Condition
Acoustic Measure: F1

Normals	Prosodic Contrast	Mean Difference	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
LJR	+pf+a vs. –pf-a	-27.700	74.001	.727	-233.160	177.760
	-pf+a vs. –pf-a	90.900	47.249	.127	-40.286	222.086
	+n+a vs. –n-a	109.700	41.152	.056*	-4.555	223.955
	-n-a vs. red	231.900*	73.610	.035*	27.525	436.275

Normals	Prosodic Contrast	Mean Difference	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
Normals	+pf+a vs. –pf-a	-10.953	33.094	.757	-102.838	80.931
	-pf+a vs. –pf-a	51.601	21.131	.071	-7.067	110.269
	+n+a vs. –n-a	60.640*	18.404	.030*	9.544	111.736
	-n-a vs. red	-286.300*	39.920	.001**	-377.700	-194.900

Based on the estimated marginal means

* The mean difference is significant at the .050 level.

[a] Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

Table A12: Pairwise Comparisons
Between-“Subjects” Effects – Normals & Prosodic Condition
Acoustic Measure: f0

Normals	Prosodic Contrast	Mean Difference	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
LJR	+pf+a vs. –pf-a	-7.700	11.697	.548	-40.176	24.776
	-pf+a vs. –pf-a	-1.100	16.143	.949	-45.921	43.721
	+n+a vs. –n-a	85.900*	30.236	.047*	1.952	169.848
	-n-a vs. red	18.200	12.180	.209	-15.818	52.016

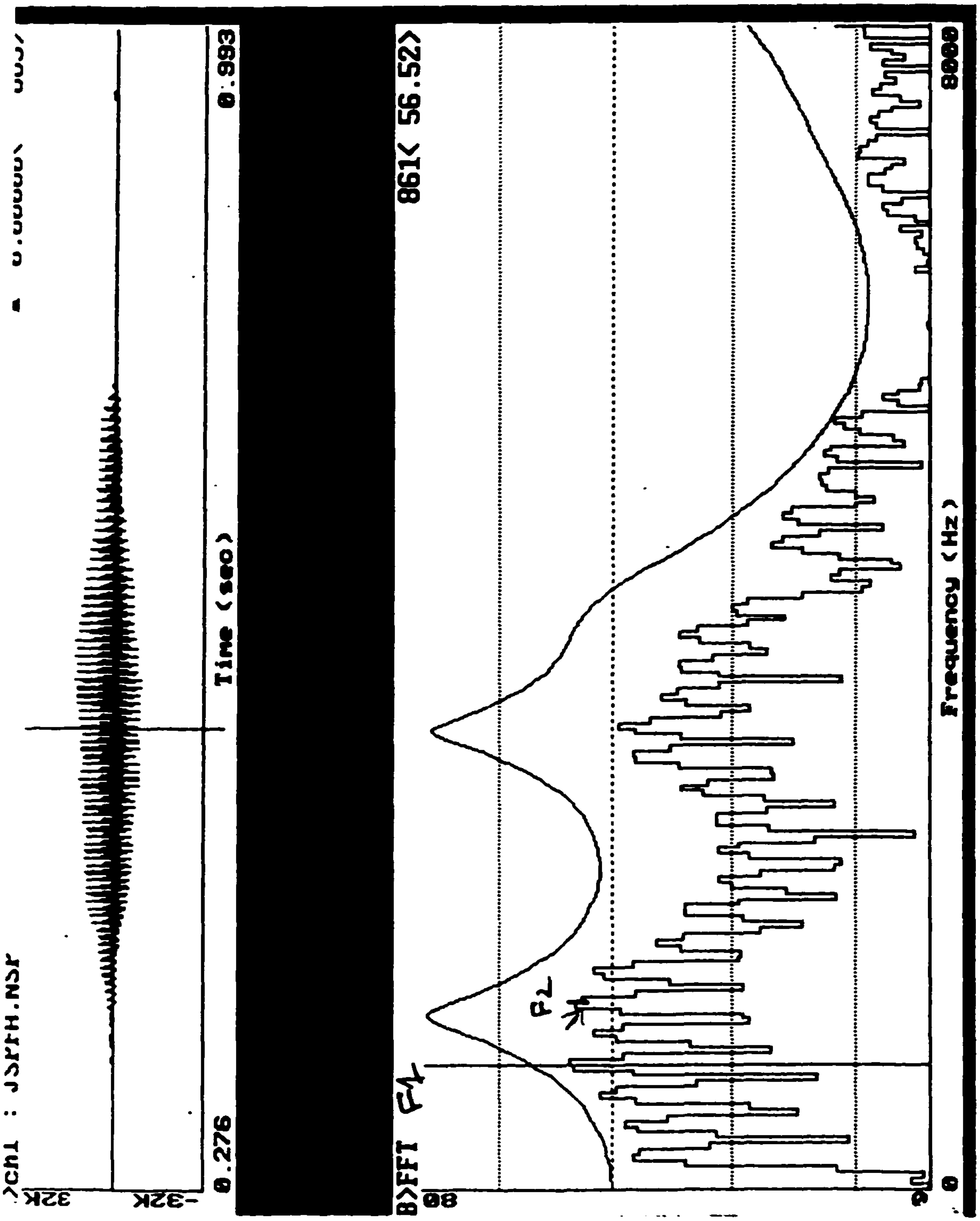
Normals	Prosodic Contrast	Mean Difference	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
Normals	+pf+a vs. –pf-a	-13.471	5.231	.082	-27.995	1.053
	-pf+a vs. –pf-a	6.369	7.220	.427	-13.675	26.414
	+n+a vs. –n-a	66.140*	13.522	.008**	28.597	103.683
	-n-a vs. red	11.086	5.447	.112	-4.037	26.209

Based on the estimated marginal means

* The mean difference is significant at the .050 level.

[a] Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

Appendix B
Acoustic Measurement



Appendix C
Magnetic Resonance Imaging: Midsagittal
Normal

Fig. : Midsagittal section of the left side of a brain in a normal. Structures labeled include corpus callosum: genu (CCG), body (CCB), splenium (CCS), the pons (Po), medulla oblongata (Md), cerebellar vermis: central lobule (Ce), cumen (Cu), declive (D), tuber (Tu), pyramid (P), uvula (U), 4th ventricle (4V) and primary fissure (1F). In addition the thalmus (Th) and hypo-thalmus (Hy Th) are labeled.

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